ESTABLISHING CLINICAL NORMATIVE DATA FOR NEURODIAGNOSTIC AUDITORY BRAINSTEM RESPONSE TESTING FOR THE KORLE-BU TEACHING HOSPITAL

SESI COLLINS AKOTEY
(10373996)

THIS DISSERTATION IS SUBMITTED TO THE UNIVERSITY OF GHANA, LEGON IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE AWARD OF MSc AUDIOLOGY DEGREE

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JULY, 2013

DECLARATION

I, SESI COLLINS AKOTEY hereby declare that this dissertation which is being submitted in partial fulfillment of the requirements for Master of Science (MSc) in Audiology is the result of my own independent research project or investigation and that, except where otherwise other sources are acknowledged with explicit references and are included in the reference list, this work has not previously been accepted in substance for any degree and neither is it being concurrently submitted for any degree.

Signed ..... Date 16th January 2014

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Most of all, I thank God for His abundant grace and love.

DEDICATION

To the Lord God Almighty do I dedicate this work for you have brought me this far and I am perpetually grateful.

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ABSTRACT

Background: Auditory Brainstem Response are series of scalp recorded electrical potentials of neural activity generated within the auditory nerve, nuclei and tracts of the lower brainstem during the first 10 ms after a click or tone pip stimulus presentation.

Aim: To develop normative data for the Intelligent Hearing Systems Smart EP systems (IHS) at the Korle-Bu Teaching Hospital's Hearing Assessment Center (KBTHHAC).

Objective: The study was to discover the relationship between the clinically established normative values with the normative data provided by the IHS manufacturer and also find out the effect of filter change on normative data generation

Methodology: A prospective study design was adopted and normative data was collected from 50 normal hearing individuals (25 males and 25 females) between the ages of 18-35 years using the IHS. Data was analyzed using Statistical Package for Social Sciences (SPSS), one sample independent t-test, mean, and standard deviation with the statistical significance set to $p<0.05$ to determine how the clinical norms compared with the manufacturer's norm.

Results: Analyzed data showed that the clinically established latencies by the study were significantly delayed compared with IHS and Hood's normative data, however, the data fulfilled the required standards for absolutes latencies based on how much variation is allowed for the upper or lower limits for normative data to be valid or invalid.

Conclusions: Results from the study were found to be appropriate for neurodiagnostic purposes most importantly, for adult ABR at the KBTHHAC taking into consideration the testing protocols and the testing environment.

Key words: Auditory brainstem response; waves; intensity; latency

CHAPTER ONE

INTRODUCTION

1.0 BACKGROUND

Auditory Brainstem Response (ABR) represents a series of neuroelectric potentials recorded from electrodes placed on the scalp which measure response latencies and their anatomical origin often labeled with Roman numerals (I to VII) (Amedofu, 1985; Hood, 1998). ABR testing has assumed a prominent and optimistic role as an objective method of assessing hearing sensitivity in the young and/or difficult-to-test patients that could not otherwise be evaluated or are incapable of responding behaviorally (Amedofu, 1985; Burkard and Secor, 2002; Gelfand, 2009; Atcherson, 2012).

Clinically, ABR tests are routinely conducted to assess the integrity of the auditory function from the peripheral auditory system to the level of the lower brainstem (Arnold, 2007; Ness, 2009). In recent years, ABR has become one of the most popular tests for hearing screening, threshold estimation and early detection of hearing impairment in high-risk and multiple or neuro-developmentally handicapped children, and also facilitates habilitation as soon as possible in many health centers (Burkard and Secor, 2002; Gelfand, 2009).

Hearing assessment at the Korle-Bu Teaching Hospital's Hearing Assessment Center (KBTH HAC) over the years has been limited to pure tone audiometry, tympanometry measures and otoacoustic emission (OAE) testing making it difficult to test some children and the difficult-to-test population. These challenges can be overcome via the use of Intelligent Hearing Systems Smart EP systems (IHS) which facilitate ABR testing of the otherwise difficult-to-test patients and/or patients who cannot respond

behaviorally, as well as enhancing the diagnostic test battery used by physicians in identification and diagnosis of retrocochlear pathologies such as acoustic neuromas. This is observed if the administration of the ABR testing and the interpretations carried out conform to acceptable clinical norm/standards and test protocols. Consequently, the recent installation of an IHS device at KBTH HAC has significantly aided the clinical test battery.

1.1 STATEMENT OF THE PROBLEM

Generally, ABR consists of eliciting and recording waveforms that represent neural activities generated at several anatomical sites from the auditory nerve region to the lower brainstem. Subsequently, comparing the quality of waveforms is made with normative data to determine normal versus abnormal responses (Hood, 1998; Ness, 2009; Gelfand, 2009). Although there are a number of standardized normative data available worldwide, it is important for each audiology clinical facility to develop its own set of ABR norms for each piece of test equipment (Hall 1992 and 2007; Ness, 2009; Gelfand, 2009).

The KBTH HAC is currently implementing ABR testing using published normative data for comparison and has yet to develop its own instrument-specific clinical norms for the IHS. Additionally, the manufacturer's high pass filter used for normative data is 3000 Hz, compared to 1500Hz for the equipment varies. This raises technical questions regarding variations of the waveform latencies obtained during clinical application. There is the possibility that locally obtained data would vary compared with normative data provided by the manufacturer since filtering and filter types may significantly affect waveform latencies and amplitude (Hood, 1998; Hall, 2007).

Furthermore, there has been considerable pressure to begin clinical evaluation before

adequate numbers of normal subjects are tested for determination of clinical norms as suggested by Hall (1992). This leads to a condition where there are no in-house instrument-specific or clinically-determined norms for comparing test results for effective and valid diagnosis exist. A crucial factor in accurate ABR interpretation is the consistency in the criteria used in waveform analysis during generation of a normative data base versus those used in clinical analysis of the response (Hall, 1992).

In light of these considerations, it is therefore imperative for clinical normative data to be collected from persons with normal hearing to establish the standard for comparison, for clinical application and for diagnosis to be made from test results obtained using the IHS at the KBTH HAC.

1.2 AIM OF THE STUDY

The aim of the study was to develop an in-house normative data for neurodiagnostic ABR testing for the KBTH HAC for purposes of providing a comparative standard for making informed diagnostic decisions on patient's auditory function using the IHS.

1.3 OBJECTIVES OF THE STUDY

The following specific objectives were used to achieve the aim of the study:

1. Discover the relationship between clinically established normative values with the standardized normative data provided by the IHS manufacturer
2. Find out the effect of filter change on normative data generation
3. Establish the normative data for waves I, III, V and inter-wave latencies I-III, I-V and III-V for the KBTH HAC.

1.4 HYPOTHESIS

The following hypotheses were used:

1. H_0: There were no significant differences between the latencies of waves I, III, and V of the clinical established normative values compared to the standard normative values provided by the IHS manufacturer at α level of significance.

2. H_0: The inter-wave latencies of wave I-III, I-V, III-V of the clinical established normative values compared to the standard normative values provided by the manufacturer did not show any significant difference at α level of significance.

1.5 SIGNIFICANCE OF THE STUDY

The normative data established by this study will be very useful and significant for the following reasons:

- The data will provide a reference point for valid and reliable comparison of wave latencies and inter-wave latencies for purposes of early identification of patients with hearing difficulties.
- The data will facilitate the diagnosis of retrocochlear lesions in the KBTH HAC for early remediation.
- The data also will provide a reference point for differential diagnosis.

CHAPTER TWO

LITERATURE REVIEW

2.0 INTRODUCTION

This chapter presents related literature of earlier studies conducted on ABR. The literature was reviewed from research articles, journals, books on ABR and normative data for ABR. The areas to be discussed include origin of ABR, testing parameters, factors that affect ABR results, noise, studies on establishing normative data and clinical applications of ABR.

2.1 AUDITORY BRAINSTEM RESPONSE

Auditory brainstem response is known by other names such as Brainstem Evoked Response (BSER), Brainstem Auditory Evoked Response (BAER), and Brainstem Auditory Evoked Potential (BAEP). According Hall and Mueller (1997), Arnold (2000), and Atcherson (2012), in 1967, Sohmer and Feinmesser were the first to publish that ABRs could be recorded with surface electrodes non-invasively in humans and described the response as cochlear action potentials. However, it was Jewett and colleagues in 1970 who correctly described the sequence of ABR wave form components as responses arising from the auditory nerve and various auditory brainstem structures (Hall and Mueller, 1997; Arnold, 2000; Hall, 2007; Gelfand, 2009; Atcherson, 2012). Following the initial work by Jewett and colleagues in 1970, other investigators have demonstrated strong relationships between abnormalities of the ABR in neurological disorders. Hall and Mueller (1997), Atcherson (2012) noted that in 1974, Hecox and Galambos showed that ABR could be used for threshold estimation in adults and infants. They further observed that Starr and Achor in 1975 were the first to report of the effects of central nervous system pathology on ABR (pathology that restricted the brainstem).

ABR is highly influenced by changes in the stimulus parameters such as the effect of intensity level on the overall amplitudes of all waves (Hall, 2007; Gelfand, 2009; Atcherson, 2012).Various pathologies as well affect the ABR morphology differently. Physiological measurements provide powerful diagnostic tools that supplement information obtained from patient's intake history and behavioral tests, and make it possible to test young patients or otherwise incapable of responding behaviorally. Gelfand (2009) noted that activities of the nervous system produce electrical signals that can be picked by electrodes placed on the head and subsequently displayed on the screen of a recording device and/or paper. However, with the presentation of stimuli (such as sound) there is a change in neural activities which also produces a change in the electrical signals picked up by the electrodes (Gelfand, 2009). Gelfand further noted that these electrical responses of the nervous system elicited by a stimulus are called ***evoked potentials*** or ***auditory evoked potentials*** (AEPs) when the stimulus is sound. The ABR is an example of AEPs.

ABR is an objective, early-latency neurologic test of the auditory brainstem function in response to auditory stimuli. Amedofu (1989) and Ness (2009) defined ABR as a series of scalp recorded electrical potentials of neural activity generated within the auditory nerve, nuclei and tracts of the lower brainstem during the first 10 ms after a click or tone pip stimulus presentation. ABR audiometry refers to an evoked potential generated by a brief click or tone pip transmitted from an acoustic transducer in the form of an insert earphone or headphone. The elicited waveform response is measured by surface electrodes typically placed at the vertex of the scalp and ear lobes (Silman and Silverman, 1991). ABRs assess the auditory function from the peripheral auditory system to the level of the lower brainstem.

The waveform peaks of ABR are labelled I-VII (Amedofu, 1985; Silman and Silverman, 1991; Hall, 1992; Gelfand, 2009). Clinically, the most significant ABR peaks are designated wave I, III, and V (Stapells et al., 2005). These waveforms normally occur within a 10 ms time period after a stimulus is presented at high intensities (70-90 dB normal hearing level [nHL]). ABR typically uses a click stimulus (Gelfand, 2009) that generates a response from the basilar region of the cochlea. The signal travels along the auditory pathway from the cochlear nucleus complex proximally to the inferior colliculus. Although the ABR provides information regarding auditory function and hearing sensitivity, it is not a substitute for a formal hearing evaluation. ABR waveform results should be used in conjunction with behavioral audiometry whenever possible (Hall, 2007; Gelfand, 2009).

2.2 WAVEFORM COMPONENTS

A typical ABR waveform shown in Fig. 2.1 represents waves I, II, III and V as generated from various sections of the auditory pathway. Wave I of an ABR represents the action potential of the acoustic nerve arising ipsilaterally to stimulation, from the distal portion of cranial nerve (CN) VIII (auditory nerve) within the inner ear (Rowe, 1981; Amedofu, 1989; Hood, 1998; Atcherson, 2012). The response is believed to originate from afferent activities of the CN VIII fibres (first-order neurons) as they exit the cochlea into the internal auditory canal. The generators of wave I are highly resistant to the effects of hypoxia or ischemia and may persist after total post anoxia brain death (Rowe, 1981).

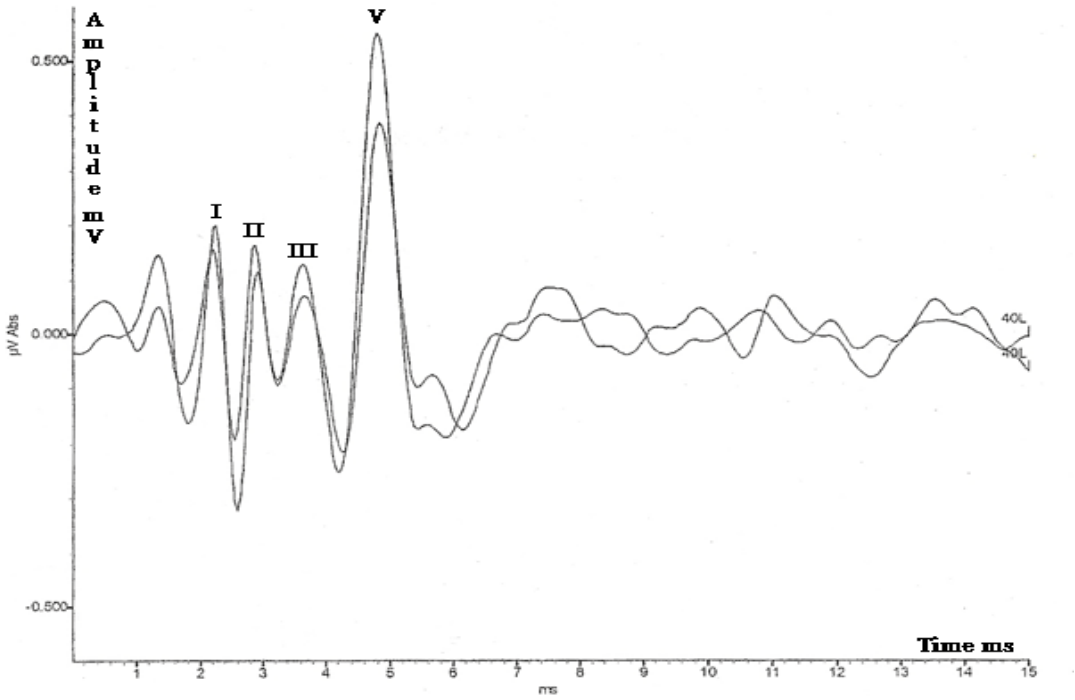

Fig. 2.1: A typical ABR waveform

The ABR wave II is generated by the proximal portion of the auditory nerve (VIII nerve) (brainstem termination) (Amedofu, 1989; Hood, 1998; Atcherson, 2012). Wave III arises in the region of the cochlear nucleus, with Hood (1998) suggesting that the generation is mainly by neurons in the cochlear nucleus. Wave IV from midline brainstem structures (perhaps acoustic strai, trapezoid bodies, and the superior olivary complex) (Atcherson, 2012). The generation of wave V is believed to originate from the termination of lateral lemniscus on the contralateral side.

2.3 ABR Testing Parameters

2.3.1 Intensity

ABRs express full complements of waves at moderately high intensity (~70 to 90 dBnHL) levels and can show up to seven waves in some cases. The reason for these relatively high intensity levels is to produce the largest amplitude and shortest latency waves possible (Atcherson, 2012). These high intensities have the effect of maximally

stimulating numerous auditory nerve fibers (i.e., high neural synchrony), to the extent that decrease in intensity levels subsequently cause reductions of wave amplitudes and prolong latencies when estimation of behavioral thresholds on an audiogram is desired. In particular, earlier waves (e.g., waves I and III) tend to drop out first when intensity levels are decreased, while wave V often remains and exhibits prolonged latencies when all other waves have disappeared (Atcherson, 2012).

2.3.2 Amplitude

Amplitude measures for ABR are often the least useful because of their sensitivity to signal-to-noise ratio (SNR) conditions and the artifact rejection process (Atcherson, 2012). Atcherson noted that although absolute peak and peak-to-trough measures can be easily obtained for any of the prominent ABR waves for general normative comparison, the wave V to I (or V/I) amplitude ratio may have some neurodiagnostic significance. The mathematical calculation is provided to divide the wave V amplitude by the wave I amplitude; i.e., a ratio of 1 is obtained if amplitudes for waves V and I are equal; otherwise the ratio < 1 if wave V is smaller than I. Typically, wave V is larger than wave I, so a significantly smaller wave V is suspected for retrocochlear lesion, but not always. A criterion of 0.75 or less has been recommended for an abnormal V/I amplitude ratio (Atcherson, 2012). The amplitudes of ABRs decrease as signal intensity decreases and there is considerable inter-subject variability in amplitudes (Silman and Silverman, 1991). The most common use of ABR amplitude is found in tracking various waves with each decrease in intensity for threshold estimation purpose, without actually observing the precise amplitude value. As the threshold is approached, wave V is often the last of the ABR complex to remain visible in the recording.

2.3.3 Latency

Latency is defined as the time taken for the response to occur following a stimulus presentation. It is measured in milliseconds (ms) and is the most widely used ABR measure (Hall, 2007; Gelfand, 2009; Don and Kwong, 2009; Ness, 2009). According to Ness (2009) peak latencies are determined by how the stimulus travels through the structures of the ear and the brainstem. Noting that if there is a clear pathway from the outer ear through to the brainstem, latency values should occur within a designated time period. However, if any of the structures mentioned above are disordered, a prolongation of the latency can occur and occasionally waveforms can disappear all together.

Clinically, latency values are analyzed by calculating the absolute latency values, inter-wave latency values, and interaural latency difference. Don and Kwong (2009) noted that latencies are used clinically because they are robust measures and are virtually unaffected by variations in the positions of the recording electrodes. Ness (2009) and Atcherson (2012) also noted that, the absolute latency is the time taken for each waveform to occur after a stimulus is presented to the ear. More significantly, Ness (2009) found that when using click stimuli at high intensity levels (70 – 90 dBnHL), wave I occur at approximately 1.5 ms after the stimulus is presented to the ear. In addition, Ness (2009) and Atcherson (2012) noted that waveforms II, III, IV, and V occur at approximately 1.0 ms intervals of waveform I, 2.5 ms for wave II, 3.5 ms for wave III, 4.5 ms for wave IV, and 5.5 ms for wave V, and that any absolute latency that exceeds 2 standard deviations is considered diagnostically significant.

The inter-peak latency (IPL) value is the time difference in absolute latency between waveforms. The waves I – III, III – V, and I – V IPLs are calculated when all

waveforms are labeled in terms of absolute latency. These values are then compared to normative values to determine if they occur within normal limits (Ness, 2009). On the approximate IPL values, Ness (2009) and Atcherson (2012) posited that the IPL of waveforms I – III should be approximately 2.0 ms, 2.0 ms for waveforms III – V, and 4.0 ms for waveforms I – V. According to Atcherson (2012) any IPL that exceeds 2 standard deviations is considered diagnostically significant. The IPL should not be more than ± 0.4 ms from the norm to be considered normal as reported by Hood (1998).

Gelfand (2009) and Don and Kwong (2009) also noted that as the stimulus intensity gets lower, the peak latencies become longer and their amplitudes becomes smaller. According to Gelfand (2009), the latency shift is seen most vividly by the rightward shift of wave V as the intensity drops progressively.

2.4 FACTORS AFFECTING ABR RESULTS - PARTICIPANT FACTORS

2.4.1 Age and Gender

The effect of age on ABR amplitude is inconclusive (Silman and Silverman, 1991). Maturation has a significant effect on overall morphology of the ABR, particularly for patients younger than 2 to 3 years of age. Each ABR peak has a slightly different maturational time course, and infant ABRs are characteristically lower in spectral content compared with matured brainstems (Atcherson, 2012). According to Sininger and Hyde (2009), wave I achieves adult latency at about 3 months of age, wave III is mature between 8 and 16 months, while wave V latency achieves adult values from 18 to 36 months of age. Anatomical, physiologic, and functional changes in adults have always been speculated to result in longer click-evoked latencies, but the data are generally mixed.

Gelfand (2009) reported that among adults ABR is affected by gender and aging. However, Silman and Silverman (1991) observed that inter-peak latencies in adult males exceeded those of adult females. In addition, the peak latencies and the wave I-III inter-peak latencies increased with age as shown in comparison of 25 young (17 – 33 years) and 25 older (51 – 74 years) adults. Furthermore, Silman and Silverman (1991) also found that the peak latency of wave V increased by approximately 0.1ms per decade. Jerger and Hall (1980) reported that the peak latency of wave V increased by 0.02 ms, on average, over the age range from 25 to 55 years. However, the peak amplitude of wave V decreased, on average, by about 0.05μV over the same age range. Silman and Silverman (1991) noted that age effects were present in both males and females, and that the average peak latencies increased by approximately 0.20 ms in their older subjects compared with their young normal-hearing subjects.

Silman and Silverman (1991) reported that as hearing sensitivity at 4000 Hz increased, the absolute latency of wave V remained constant in females and increased in males. This gender effect in contrast with young adults was reduced in the normal-hearing older adults. Gelfand (2009) contended that ABR is present but not adult-like in newborns, and its characteristics changes with the infant's maturation. He further explained that although waves I, III, and V are observable in newborns, the absolute latencies of waves III and V are prolonged relative to adult values, as are the inter-wave latencies. In particular, as the infant matures, the other peaks emerge, the latencies of the waves shorten, and their amplitudes change, eventually achieving adult characteristics by 18 months of age.

Silman and Silverman (1991) and Hall (1992) conceived that since some studies reported a slight age effect (on the order of 0.2 ms), then age effects must be

considered when collecting normative data, stating that separate norms should be obtained for subjects under 50 years of age and subjects over 50 years of age.

2.4.2 Body Temperature

Silman and Silverman (1991) associated hypothermia with increased inter-peak latencies and explained that decreased body temperatures were commonly observed during surgical procedures involving cardiopulmonary bypass or total circulation arrest, coma and drug intoxication cases. Furthermore, the ABR may be affected when increased body temperature is induced in patients suspected of having multiple sclerosis. Arnold (2007) explained that a decrease in body temperature below normal causes an increase in ABR inter-peak latency; this effect is thought to be due to slowed neural conduction velocity and synaptic transmission speed in hypothermia. Because low birth weight infants and comatose patients are prone to hypothermia, temperature should be measured prior to an ABR evaluation in these patients.

In effect, body temperature does not affect the ABR unless it drops to or below 36^0C. The correction factor is obtained by subtracting of 0.15 ms from the obtained I-V IPL for every degree Celsius when the body temperature falls below 36^0C (Silman and Silverman, 1991).

2.4.3 Hearing Loss

The effect of hearing sensitivity on the ABR is an important clinical consideration. Hearing sensitivity also creates difficulties in analysis and interpretation since hearing losses have the effect of reducing the perceived intensity level of the stimuli. This could cause prolongation of all or most waves of the ABR as if the clinician were decreasing the physical intensity level (Atcherson, 2012). Indeed, persons with rapidly sloping, high-frequency sensorineural hearing loss (with normal low

frequency thresholds) can produce ABRs with prolonged wave V, giving impressions that an acoustic tumor is causing this shift. In addition, when the pure tone threshold at 2000 Hz is not greater than 40 dB HL, and the audiometric average of three frequencies, 500, 1000 and 2000 Hz, is not greater than 50 dB HL, it has been reported that 80% of ABRs can be normal (Atcherson, 2012).

Gelfand (2009) also noted that typical wave V latency-intensity functions associated with normal hearing, conductive losses, and sensorineural losses of cochlear origin are quite different, and further explained that conductive losses reduced the amount of signal intensities reaching the cochlear resulting in latency-intensity functions. These are essentially horizontally displaced to the right (higher latency levels) by more or less the amount of the conductive loss. However, Gelfand (2009) cautioned that such patterns depend on the configuration of the hearing loss because the ABR relies heavily on the basal (high frequency) portion of the cochlea. Gelfand explained that the different latency-intensity functions associated with conductive and cochlear impairment allow the ABR to help discriminate between these two kinds of hearing losses. However, conductive losses may have to exceed 35 dB to be reliably distinguishing from sensorineural impairments with the ABR (Gelfand, 2009). In cochlear impairments, wave V latencies typically are elevated at slightly above threshold and then converge to the normal latency range as the click intensity is raised (Gelfand, 2009).

In effect, Atcherson (2012) suggested a technique to correct for hearing loss by subtracting 0.1 ms from every 10 dB of hearing greater than 50 dB at 4000 Hz. This technique can be used with the wave V inter-latency difference neurodiagnostic criterion, but it is generally not recommended clinically unless there is a chance that a

unilateral tumor is greater than 1 cm.

2.4.4 Medication

Literature has shown that sedation does not affect ABR or cases of drug-induced coma. Anticonvulsants such as dilantin, administered at therapeutic levels, essentially have an adverse effect on ABR (Silman and Silverman, 1991; Gelfand, 2009), since they rather reduce muscle artifact, thereby enhancing the ABR waveform. Additionally, Silman and Silverman (1991), and Gelfand (2009) have stated that since anesthesia has a minimal effect on ABRs, intraoperative monitoring using ABRs has been increasingly employed.

2.5 STIMULUS FACTORS

2.5.1 Stimulus Type

The ABR can be evoked by virtually any stimulus that is abrupt in nature, such as a 100-µsec clicks with an essentially instantaneous onset, tone bursts with rise time of a few milliseconds, and even gaps in broadband noise with fall times of a few milliseconds (Atcherson, 2012). Hall (2007) and Atcherson (2012) further suggested that the abruptness of these various stimuli causes a synchronous discharge of numerous auditory nerve fibers. Clicks presented to the ears by way of transducers are said to broadly stimulate the cochlea because of their instantaneous rise causing a spectral splattering of frequencies.

Additionally, Burkard and Secor (2002) opined that the two most common transient stimuli used clinically are clicks and tone bursts. Tone bursts used with the ABR are short duration sinusoids, usually no more than ~4 to 5 cycles long at typical octave audiometric frequencies from 500 to 4000 Hz (Atcherson, 2012). According to

Atcherson, the brevity of tone burst stimuli in ABR recordings is necessary for the ABR's dependency on neural synchrony.

Higher frequencies in the click acoustic spectrum are responsible for generating the ABR in the normal ear (Hall, 2007). An ABR evoked by a moderately intense (60 dBnHL) click stimulus and delivered with insert earphones reflects activation of the high-frequency regions (1000 through 4000 Hz) of the cochlea (Hall, 2007). However, Hall (2007) observed that there are two general principles to keep in mind in considering stimuli for evoking auditory responses. First, frequency specificity of a stimulus (i.e., the concentration of energy in a specific frequency region) is indirectly related to duration (Hall, 2007). With very brief stimuli, energy tends to be distributed over more frequencies, whereas stimuli with longer duration (including rise/fall times and plateau time) are spectrally constrained. Second, there is generally a direct relationship between duration of the response and duration of the stimulus (Hall, 2007). Hall further explained that, slower responses (longer latency) are activated best by slower (lower rate of stimulation, and longer onset and duration) stimuli; whereas faster (shorter latency) responses require faster (higher rate of stimulation and shorter onset and duration) stimuli.

2.5.2 Stimulation Rate

Rate is a stimulus parameter that must be selected by the operator in auditory evoked response (AER) measurement and can be manipulated to permit the fastest data collection in the least amount of time, thus either saving test time or permitting a thorough AER assessment in the time available (e.g., while a small child sleeps after sedation) (Hall, 2007). There is no single correct rate, one that is appropriate for all patients under all test circumstances. Hall (1992 and 2007) noted that the effects of

rate are distinct for each of the AER types, particularly the shorter electrocochleography (ECochG) and longer ABR responses. For each AER type, rate effects are a product of the interactions among rate, a variety of subject characteristics (such as age, body temperature, and drugs), and various other stimulus parameters (such as intensity and duration) (Hall, 1992 and 2007). He further explained that, rate appears to be a factor in the diagnostic power of certain ABRs. That is, rate may interact also with neuropathology and a simple, yet statistically and clinically significant relationship exists between rate for transient stimuli and behavioral auditory threshold.

The general rule with stimulation rate is that there can be no more than one stimulus presented in the same analysis time window, or else an overlapped and time-shifted response will be averaged (Arnold, 2007; Atcherson, 2012). This means that, during signal averaging, the stimulus must be presented at a specified rate, which must be slow enough to prevent the overlapping of responses that will occur if a new stimulus is presented before the response to the previous stimulus has been completed. For example, if a 10 ms window is used, the click stimuli could be presented at a rate of 1 click every 10 ms, or 100/s (Arnold, 2007; Atcherson, 2012). However, Arnold (2007) noted that a slower stimulus rate produces the most clearly defined waveform, but increases the amount of time required to obtain a single average. A higher stimulus rate, on the other hand, reduces test time, but it decreases the amplitude of the ABR, particularly the early components of the waveform.

In conventional ABR recordings, stimuli are generally presented at rates ~10 to 40/s, usually with odd and decimal numbers (e.g., 27.7) to avoid multiples of the 60-Hz line noise (Atcherson, 2012). However, Arnold (2007) noted that another clinical strategy

is to use a slow rate (<20 per second) when clear definition of all waveform components is required, as for otoneurological assessment, and to use a higher rate (eg 39 per second) when measuring wave V latency - intensity function for threshold estimation because wave V is most resistant to the effects of high stimulus rates.

In some neurodiagnostic protocols (particularly for vestibular schwannoma detection), Atcherson (2012) noted that, both low and high rate click presentations are used, with the latter being used to "stress" the auditory system and see how well it performs. Whether the high-rate addition to the protocol adds another neurodiagnostic criterion is a matter of some continued speculation (Atcherson, 2012) because most ABRs are likely to miss tumors smaller than 1 cm and will depend on which groups of auditory nerve fibers are affected as well as the type of disorder.

From a stimulus rate of 5/sec to a stimulus rate of 80/sec, threshold is enhanced by 5 dB (measured in peak SPL) (Hall, 2007). The rate-versus-intensity relation itself is in turn influenced by frequency. Significantly less threshold improvement with increasing rate is observed for high- versus low-frequency stimuli. Silman and Silverman (1991) pointed out that as the click rate (number of clicks per second, also referred to as the repetition rate) increases, the absolute latencies of all the ABR components and the inter-peak latencies increase.

Nevertheless, the absolute latency of wave V is not substantially prolonged until repetition rate exceeding 30 Hz are obtained (Silman and Silverman, 1991). These investigators also noted that when click rate is increased, the absolute latencies of waves I and V are increased with wave V prolonged to greater extent than wave I. In effect, the wave I-V inter-peak latencies increases but the increase in IPL with stimulus rate increase is smaller at moderate (50 dB SL) that at high (70 dB SL)

intensities. This is so because wave I is more affected than wave V by the rate increase at moderate than at higher intensities. According to Hall (1992), in the selection of stimulus rate for any AEP measurement, it is best to avoid numbers that are evenly divided into 60 Hz, in order to minimize the possibility of power-line interference; hence, rates such as 21.1/sec or 37.7/sec, as opposed to 20/sec, are routinely used clinically for ABR. Stimulus rate must be considered in collecting normative AER data (Hall, 2007).

2.5.3 Stimulus Polarity (phase)

Rarefaction (R), condensation (C), or alternating (A) polarity (phase) signals are employed in ABR assessment (Silman and Silverman, 1991; Hall, 2007; Atcherson, 2012). According to Hall (2007) with a positive electrical pulse or signal and movement of the transducer diaphragm toward the tympanic membrane, a click signal with a positive pressure wave is generated. Movement in a positive direction, or a positive polarity, is also known as "condensation polarity." A pressure wave in a negative direction (negative polarity), produced by a movement of the transducer diaphragm away from the tympanic membrane, is called "rarefaction polarity." Alternating polarity is a switching between condensation and rarefaction polarities at subsequent stimulus presentations (Silman and Silverman, 1991; Hall, 2007; Atcherson, 2012).

Silman and Silverman (1991) indicated that rarefaction clicks tend to be associated with shorter absolute peak latencies than condensation (C) presented at 70 dB SL. Silman and Silverman (1991) reported that click phase is an important factor contributing to inter-subject variability in amplitude, morphology, and inter-wave latencies of the ABRs. Increase in inter-peak latency with increase in stimulus rate is

more pronounced for rarefaction than for condensation clicks (Silman and Silverman, 1991). On alternating polarity, Silman and Silverman (1991) posited that some clinicians use alternating polarity clicks when the electrical artifact and the cochlear microphonic impinge on wave I; this effect is more apparent at high stimulus presentation levels.

However, Atcherson (2012) noted that the latencies do not change much with the different polarities. The waveform morphology may change in the same individual and produce different waveform morphologies in different individuals. Atcherson (2012) explained that a comparison of click-evoked ABR with both polarities (condensation and rarefaction) using insert earphones show no significant polarity-related differences in patients seen for unilateral acoustic neuroma. However, significant differences may appear with TDH-type headphones.

2.5.4 Clicks

The most common stimulus used for neurodiagnostic purposes is the unfiltered click. The rise of a click is sufficiently rapid to synchronize the discharges of a large population of neurons, and thus, to yield a large, well-formed ABR with its component wave (Fowler and Durrant, 1994). According to Arnold (2000) most commercial evoked potential instruments allow a choice of rarefaction, condensation, or alternating rarefaction and condensation phase for the click onset. Arnold indicated that there is presently no consensus regarding which polarity is the best to use; however, most researchers recommend using either rarefaction or alternating phase. Rarefaction is the phase that stimulates the afferent dendrites of the auditory nerve and has been shown to produce shorter latencies and larger amplitudes of the major ABR waveform components in most individuals (Arnold, 2000). Alternating phase is

useful in reducing stimulus artifact, which may interfere with identification of wave I. The stimulus artifact follows the phase of the stimulus and therefore cancels out when opposite polarities are added together. However, alternating phase may degrade the clarity of the waveform, especially in the case of high-frequency hearing loss (Arnold, 2000).

2.5.5 Tone Bursts

The difference between rarefaction and condensation phase is less important for tone bursts than clicks, because the stimulus rise time will include excursions of both polarities. Alternating phase can be used to cancel out the cochlear microphonic and stimulus artifact. For low frequency tone burst, alternating phase will broaden the ABR peaks and may degrade clarity of the waveform (Arnold, 2000).

2.5.6 Transducer Type

According to Atcherson (2012), the ABR can be recorded using either supra-aural or insert (tubal) earphones. The rubber tube of the insert earphone introduces a 0.8- to 0.9-msec acoustic delay between the transducer box and the foam insert that is coupled to the patient's ear. Atcherson explained that the purpose of this acoustic delay is to separate in time the stimulus-related artifact produced by the transducer and the desired ABR. Another advantage of insert earphones for ABR is the increased interaural attenuation (Hall, 2007; Atcherson, 2012).

One disadvantage of the insert earphones is that the sound pressure level differences are greater between infant and adult ears when compared with circumaural and supra-aural earphones (Atcherson 2012). This is an especially important concern for pure tone threshold estimation when calibration standards are based on the thresholds of adults with normal hearing.

2.5.7 Noise

For most auditory evoked potential (AEP) recordings in human subjects, the amplitude is small relative to the amplitude of the background noise (Burkard and Secor, 2002). Electrodes pick up not only ABR, but any physiological potential from multiple sources (Sokolov, Kurtz, Steinman, Long, and Sokolova, 2005). According to Burkard and Secor (2002) all unwanted electrical activities are generically termed noise, which is composed of both periodic and aperiodic activity, both of biologic and non-biologic origin. For example, muscle activity from the subject, the brain – electroencephalogram (EEG), the eyes - electrooculogram (EOG) and electronystagmogram (ENG), the heart - electrocardiogram (ECG) and the skeletal muscles - electromyogram (EMG) (Sokolov et al., 2005). The conventional ABR systems require the patient's eyes to be closed and motionless (Sokolov et al., 2005) since electro-oculogram (EOG) generates very strong and harmful artifacts for ABR by saturating the preamplifier.

Muscular activity generates very strong artifacts with the strongest coming from the facial and neck muscles. This is always present in non-relaxed patients and normally disappears as patient sleeps, particularly under sedation (Sokolov et al., 2005). Some muscles in relaxed and even sedated patients may move and generate artifacts, while in some patients such artifacts may be present even without visible body movements. Also importantly, muscular activity frequencies range form 30-500 Hz and are well within the ABR frequency range explaining why they pass the ABR band-pass filter. In conventional systems their effect can be reduced only by artifact rejection (Sokolov et al., 2005).

Electric and magnetic fields exist in any clinical environment most especially, strong electromagnetic fields are found in non-shielded rooms: operating rooms (OR), intensive care units (ICU), neonatal intensive care units (NICU), hospital wards, and doctor's offices (Sokolov et al., 2005). These fields may introduce electro-magnetic interferences (EMI) in conventional ABR systems making ABR testing very difficult or impossible (Sokolov et al., 2005).

2.6 RECORDING FACTORS

2.6.1 Filters

The purpose of a filter is to eliminate the contaminating effects of electromyographic noise and to reduce the amplitude of the ongoing electroencephalographic activity without significantly affecting the auditory-evoked potentials (Silman and Silverman, 1991; Gelfand, 2009). Hall (2007) also explained that filters selectively remove part of the bioelectrical activity from the total bioelectrical activity plus electrical activity arising from sources outside of the brain. In ABR measurement, filters reject electrical energy at certain frequencies and pass energy at other frequencies. Gelfand (2009) explained that differential amplification is used to boost the level of the evoked potential response while at the same time removing noise. Additionally, Silman and Silverman (1991) state that as the low-frequency cutoff of the bandpass filter is increased to 300 Hz, the latencies of all the waves decrease and the amplitude of wave V decreases relative to that of wave IV. The effect on the amplitude of wave V is most marked as the low frequency cutoff is increased from 100 to 300 Hz (Silman and Silverman, 1991).

On the high-frequency cutoff of the bandpass filter, Silman and Silverman (1991) noted as the frequency is increased from 300 to 3000 Hz, the latencies of all the

waves decrease and there is improvement in the resolution of waves IV and V. As the high-frequency cutoff increases beyond 3000 Hz, the resolution of wave IV and V is not improved further as high noise is added to the waveform.

2.6.2 Signal Averaging

Signal averaging is a technique that allows the response (evoked potential) to be extracted from the noise and is a central principle of many physiological methods (Gelfand, 2009). The ABR is very small, and even with filtering, it is buried in a background of noise. Therefore, signal averaging helps to reduce this noise so that the signal, in this case the ABR, can be detected (Arnold, 2000). Signal averaging is possible because the ABR is time-locked to stimulus onset, whereas the noise interference occurs randomly. That is, the signal occurs at the same points in time following onset of the eliciting stimulus, but the noise has no regular pattern (Arnold, 2000). Arnold posited that in signal averaging, a large number of stimuli are presented, and the responses to each of the individual stimulus presentations (termed sweeps) are averaged together to obtain a final averaged waveform. That is, by averaging, the random noise tends to cancel out, whereas the evoked potential is retained because it is basically the same in each sweep.

The greater the number of stimulus presentations used, the greater the improvement in signal-to-noise ratio and the more clearly the ABR can be visualized in the final averaged waveform. For ABR recording, between 1000 and 2000 sweeps are typically used. However, for efficient use of test time, averaging may be terminated before the specified number of sweeps is reached, as soon as a clear waveform is visualized in the averaged response (Arnold, 2000). Conversely, a large number of sweeps may be needed (e.g., 6000) near threshold where the amplitude of the response is small or

when background noise is particularly high.

These notwithstanding, the patient must be reclining or lying in a relaxed state without moving, or else interference from muscle activity will make identification of the ABR difficult if not impossible. Therefore, for infants and young children, this generally requires that they are tested while they are as sleep or sedated (Arnold, 2000).

2.7 STUDIES ON NORMATIVE DATA

Several studies have been conducted in the area of establishing normative data for ABR testing. According to Sininger (1992) there are no established guidelines for acquiring normative data clinically. Sininger observed that, although the American Electroencephalographic (EEG) Society has published recommended guidelines for extensive studies of normative data, the society suggests that new laboratories can utilize published data with proper precautions. This is as a result of the fact that the type of hardware and clinical needs of each laboratory differs. In the generation of normative data, many subject, stimulus and recording factors influences the ABR. These factors include age, gender, hearing loss, electrode placement and as well as several others. Sininger (1992) posited that in the establishment of an ABR, the investigator may choose to evaluate one or more response parameters including general waveform morphology, absolute latencies, inter-peak latencies, absolute amplitude and relative peak amplitudes.

Sininger (1992) and Hall (2007) identified some steps for the establishing of norms for ABR:

1. Choose a set of recording and stimulus parameters according to the type(s) of patients to be evaluated and the information that is desired.

2. Conduct a literature search for published databases.
3. Establish normalized hearing levels using the stimulus parameters and behavioral hearing thresholds for each stimulus to be used. Perform a careful threshold determination procedure carried out in a quiet environment such as a hearing test booth.
4. Conduct ABR tests on a group of young adults who have normal hearing and no history of neurologic or otologic disorder to validate the recording procedure.
5. Compare your measured response parameters to those published. Data for most "normal" subjects should fall within 2 standard deviations of published data. Any major discrepancies (more than 5% outside this range) must be explored (Sininger, 1992; Hall, 2007).

According to Hall (1992; and 2007) the number of subjects selected in generating normative data for ABR clinically is probably determined more often on the basis of convenience or convention rather than on the basis of statistics. Silman and Silverman (1991) noted that most clinical laboratories based their normative data on a small sample size of 10 – 30 subjects. On the same issue of subject size, Hall (1992) observed that, due to time and subject availability constraints, normative data are often collected from a small group of presumably normal hearing subjects, such as 10 males and 10 females using a single protocol.

Hall (1992) provided useful statistical guidelines for determining the smallest adequate number of subjects for a normative data base indicating that the group (sample) of normal subjects should be large enough to produce standard error of the mean (SEM) values that are less than the time resolution of the response. SEM is

defined as the standard deviation of the means of all samples of the population. Literature searches show that for ABR I-V latency interval, the SEMs for several progressively larger normal samples sizes were as follows: *n* = 6, 0.08 ms; *n* = 10, 0.05 ms; *n* = 14, 0.05 ms; *n* = 18, 0.04 ms; *n* = 22, 0.03 ms (Hall, 1992). He noted that a sample (normal group) size of 22 is necessary to produce an SEM smaller than the time resolution for ABR latency, stating that if time resolution demands were only 0.1 ms (less stringent), then as few as 6 subjects would be needed. Silman and Silverman (1991); and Hall (1992) also posited that it would be quite feasible for a laboratory to maintain ongoing SEM calculation as AEP data for each normal subject was added to the data base until the time (or amplitude) resolution criterion was met for a given AER component.

2.7 CLINICAL APPLICATION OF ABR

ABR as a test protocol is a very useful tool in the diagnostic battery in clinical settings (Gelfand, 2009). The test is used clinically both in the estimation of auditory sensitivity and in otoneurological assessment (that is, to detect lesions along the auditory nerve and brainstem pathways) in individuals who are not readily testable by conventional behavioral audiometric procedures (Arnold, 2000). Arnold (2000), Gelfand (2009) and Sininger and Hyde (2009) noted that, these individuals include infants, developmentally delayed children, multiple handicapped children and adults, autistic individuals, and persons suspected of pseudohypoacusis. The ABR is also an effective screening tool used in the evaluation of suspected retrocochlear pathology such as an acoustic neuroma or vestibular schwannoma.

In addition to retrocochlear pathologies, several other factors may influence ABR results. These factors include the degree of hearing loss, asymmetry of hearing loss,

test parameters, and other patient factors (Hood, 1998; Gelfand, 2009). These influences must be factored in when performing and analyzing an ABR result.

According to Gelfand (2009) some findings are suggestive of retrocochlear pathology; and may include anyone or more of the following:

- Prolonged latency for wave V.
- Prolonged inter-wave latency for I to V (as well as for I to III and / or III to V).
- Interaural latency differences.
- An ABR waveform that is not replicable.
- Abnormally low V: I amplitude ratio.
- Absence of an ABR even though hearing is normal or only mildly impaired.

In general, ABR exhibits a sensitivity of over 90% and a specificity of approximately 70-90%. Sensitivity for small tumors is not as high. For this reason, a symptomatic patient with a normal ABR result should receive a follow-up audiogram in 6 months to monitor for any changes in hearing sensitivity or tinnitus. The ABR may be repeated if indicated. Alternatively, MRI with gadolinium enhancement, which has become the new criterion standard, can be used to identify very small (3-mm) vestibular schwannomas.

CHAPTER THREE

METHODOLOGY

3.0 INTRODUCTION

This chapter presents the methods and techniques used in carrying out the study and include research approach, research design, population, sample, sampling technique, research instrument, data collection procedure, and data analysis.

3.1 RESEARCH METHOD

According to McMillan and Schumacher (1997), a quantitative approach emphasizes objectivity and quantification of a phenomenon. As a result, it maximizes objectivity by using numbers and statistics structure. Creswell (2005) also views the quantitative approach to studies as a method which provides for decisions on what to study, asking of specific, narrow questions, collection of numeric (numbered) data, statistical analysis of numbers, and conducting inquiries in an unbiased, objective manner. In this study, a quantitative approach was adopted because the study yielded numeric data which were analyzed using the Statistical Package for the Social Sciences (SPSS) version 16.0 and descriptive statistics (mean, median, standard deviation, and standard error).

3.2 RESEARCH DESIGN

The research adopted a prospective study design. According to the Mosby's Medical dictionary (2009) prospective study is an analytical study designed to determine the relationship between a condition and a characteristic shared by some members of a group. According to the Medical dictionary (209), the population selected for the study is healthy at the beginning; that is, participants in this case, did not have any hearing loss or history of ear pathology and had hearing thresholds within the normal

range according to the standards of KBTH. The design therefore facilitated the in-depth examination of peak latencies of wave I, III and V; and inter-wave latencies of wave I-III, I-V and III-V and the relationship between the clinically established normative data and that of the manufacturer. The design also enabled the exploration of normative values for clinical comparison and diagnosis.

3.3 POPULATION

The population for the study consisted of normal, otherwise healthy adolescents and young adults (18 – 35 years) in and around KBTH. This population was chosen since the determination of the clinical norms required individuals with hearing thresholds within normal limits and their accessibility.

3.4 SAMPLE SIZE AND SAMPLING TECHNIQUES

The sample size was 50 participants put into two categories: 25 males and 25 females. A combination of convenience sampling technique and purposive sampling techniques were used to select the participants. According to Cohen and Morrison (2007); Vanderstoep and Johnson (2009) convenience sampling – otherwise called, accidental or opportunity sampling – involves choosing the nearest individuals to serve as respondents and continuing that process until the required sample size has been obtained. Therefore, participants for the study were drawn from the student population at the KBTH and individuals who were readily available.

Additionally, the final targeted population was purposively selected based on the inclusion criteria requirement. According Cohen and Morrison (2007), purposive sampling allows handpicking the cases to be included in the sample on the basis of typicality or possession of required particular characteristics. In this way, samples are

built to satisfy specific needs. Therefore, the final 50 participants were handpicked from the general population based on the inclusion criteria requirement used.

3.5 INCLUSION AND EXCLUSION CRITERIA

3.5.1 Inclusion Criteria

All subjects for the study met the following criteria: a thorough otosocpic examination was performed on the day of testing and all subjects had clearly visible and normal tympanic membranes bilaterally. Additionally, pure tone air conduction thresholds at the standard audiometric frequencies (250, 500, 1000, 2000, 4000, 8000 Hz) were measured using a modified Hughson-Westlake (10-dB down, 5-dB) method. A calibrated Interacoustics AC 33 clinical audiometer was used in a calibrated test booth. All subjects had pure tone behavioral thresholds ≤20 dB HL on the day of testing. Furthermore, 226-Hz tympanometry was performed to ensure the subjects had peak compensated static admittance and ear canal volumes within normal limits ruling out any middle ear pathology.

3.5.2 Exclusion Criteria

The following criteria were used to exclude non-participants:

- Individuals aged outside the age bracket of 18 to 35 years.
- Participants with histories of any ear or hearing related pathology.
- Participants with hearing threshold outside the normal limit of ≤ 20 dBHL at the time of testing
- Participants whose tympanometric results show no normal peak compensated static admittance and ear canal volumes.

3.6 INSTRUMENTATION

According to Vanderstoep and Johnson (2009), physiological measures provide bodily measures with precision of data of the instruments. Physiological measures using IHS was therefore employed for data collections. A schematic diagram of the testing instrument is shown in Fig 3.1

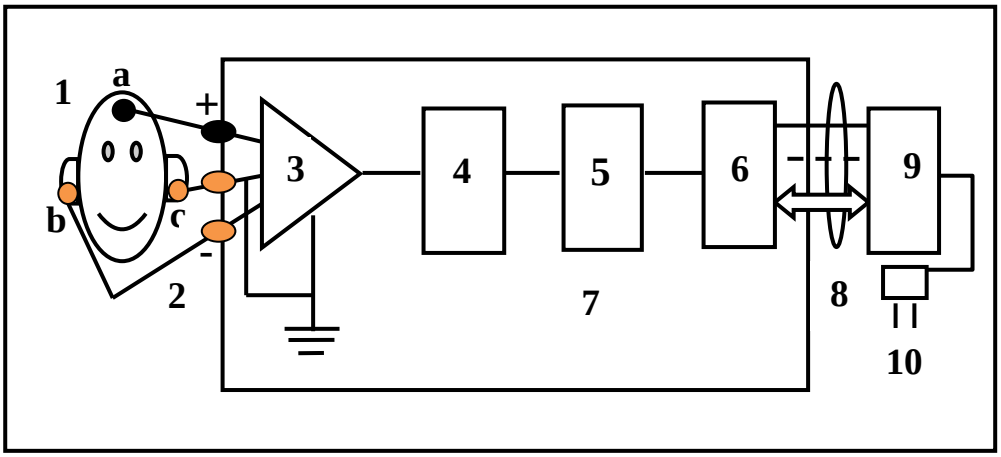

Fig. 3.1: Conventional ABR recording system

1a – non-inverting (+) electrode (shown placed on higher forehead), b – ground electrode (shown placed on right ear lobe), c – inverting electrode (shown paced on the left ear lobe), 2 – electrode lead wires (leads), 3 – differential preamplifier, 4 – band-pass filter (typically 30-1500 Hz for ABR), 5 – power amplifier, 6 – analog-to-digital (A/D) converter, 7 – interface module, 8 – interface cable, 9 – personal computer (typically a laptop), 10 – power cord. (Courtesy Sokolov et al., 2005).

All the equipment used for the testing of participants to was calibrated. These included the AC 33 audiometer for the pure tone air testing, and tympanometer for middle ear status testing. The IHS equipment for the ABR had already been calibrated. These factors will ensure that the test results from this equipment are valid and reliable.

3.7 PROTOCOL FOR TESTING AND DATA COLLECTION PROCEDURE

3.7.1 Testing Protocol

The following protocol described in Table 3.1 was used for testing and data collation.

Table 3.1: Testing Protocol

Stimulus	**0.1 milliseconds click**
Rate	21.1/sec
Polarity	Rarefaction
Transducer	Insert Earphones
Filters	30-1500 Hz
Intensity	80 dB HL down to 10 dB HL
Amplification	100x
Runs	2
Analysis time window	12.8 milliseconds
Sweeps	2048
Electrode montage	Ipsilateral

ABR waveform identification after collection was done by visual inspection of the resulting waveforms for the presence of waves (I, III, V) at KBTH HAC. A minimum of two replications were required for the repetitiveness of the waveform. Wave I identification is very important in neurological testing because it sets the reference point for the identification of all other waves; and must therefore be differentiated from the cochlear microphonic and stimulus artifact. To ensure accuracy of identification, a high intensity and a relatively slow stimulus rate were used to obtain a clearly visible and increased wave I amplitude. The stimulus polarity was changed from rarefaction to condensation to cancel cochlear microphonic in cases where wave I was not easily identified. Wave V amplitude identification was done taking into consideration the trough that occurs after the wave V amplitude. Again, the confusion in the fusion of wave IV and V peaks was resolved by lowering the intensities. This

procedure split wave V from wave IV. For the ABR data acquisitions, the presentation level was reduced in 10 dB decrements from 80 dB HL to 10 dB HL.

The IHS is the electrophysiologic equipment for ABR testing of patients for the KBTH HAC and was used for participants and data collection. Data for the study were collected by setting every subject up for a one-channel differential recording where the non-inverting and reference electrodes were common to both channels. The non-inverting/positive (+) electrode was placed on the high forehead (Fig.3.1) while the inverting (-) reference electrode was placed on one ear lobe (C7 vertebra) and the ground placed on the other ear lobe (Fig.3.1). The surface of the skin was prepared in a conventional manner in order to reduce the impedance between electrodes. All impedances were required to be <3 kOhm (the resolution of the measuring equipment). All testing was conducted in a sound-attenuated room with the subject lying on a bed. The participants were instructed to relax and sleep to reduce myogenic interference. Insert headphones (Etymotic Research ER-3) were used for stimulus presentation.

3.7.2 Validity and reliability of Data Collected

Double waveform collections were made to ensure repeatability of the waveforms for the validity and reliability of the data generated, most especially in the identification and labeling of the waveform peaks at each intensity. This was done to ensure repeatability of the waveform for the identification of each waveform amplitude and latency. To avoid bias and mistakes, the various latency values were also automatically generated by the equipment once the waveform peaks are identified and labeled.

3.8 DATA ANALYSIS

Descriptive statistics was used to describe, present and interpret the data in light of the hypotheses of the study. Cohen et al., (2007) explained that descriptive statistics do exactly what they say; that is, they describe and present data. The latencies of the waveforms were measured from the onset of the stimulus to the most prominent peaks of waves I, III and V. The mean and standard deviations were calculated for the latency values of waves I, III and V. Composite data were also computed for the determination of waves I, III and V as a function of stimulus intensity. The inter-wave latencies of waves I-III, III-V and I-V were established and compared to the standard normative values. All the data were computed and analyzed using SPSS and the one sample independent t-test (Z- score or standard score) was used to analyze the data with the statistical significance set to $p < 0.05$.

3.9 ETHICAL CONSIDERATIONS

Ethical clearance was obtained from the Ethics and Protocol Review Committee of the School of Allied Health Sciences before the commencement of the data collection (Appendix III). Permission to commence the data collection was granted by the KBTH HAC (Appendix IV). Participation of subjects conformed to the required ethical guidelines regarding the use of human subjects. Written informed consent was sought from each participant before the collection of data (Appendix II). All participants were made aware of the objectives and methods of the study (Appendix I) and the testing process duly explained. Additionally, participants were assured of strict confidentiality with regards to their bio-data and any data generated by the study.

CHAPTER FOUR

RESULTS

4.0. INTRODUCTION

The aim of the study was to develop in-house normative data normative data for neurodiagnostic ABR testing for the KBTH HAC for purposes of providing a comparative standard for making informed diagnostic decisions on ABR-based patients' hearing test. This chapter is divided into two sections; the first section provides the results of the absolute mean and standard deviation from data as collected from the participants and the second section presents the results for the study in regards to the hypotheses of the study. Refer to appendices VI to IX for the participants' raw data.

4.1. WAVE LATENCIES

Data was collected from 50 participants, 25 males, and 25 females between the ages of 18-35 years. ABR testing was conducted on the participants, their waveforms labeled and the various latency values collected and analyzed. The absolute wave latencies for wave I, III, and V; and the inter-wave latencies, I-III, III-V and I-V were collected at intensities of 80 dB to 30 dB with a 10 dB decrement for all participants and their respective means and standard deviations computed. A typical waveform from the participants is shown below in Fig.4.1. The rest are shown in Appendix V. The mean latencies and standard deviations for male, female and combined group are presented in Tables 4.1-3

The results shown in Tables 4.1-3 establish an inverse relationship between the latency values and the intensities which is consistent with the literature. Additionally, comparing the wave latencies of the various waveforms to the corresponding

waveform latencies of the male values (Table 4.1), it can be seen that the values of the male latencies are slightly longer or delayed compared to the female data (Table 4.2). This is also consistent with literature.

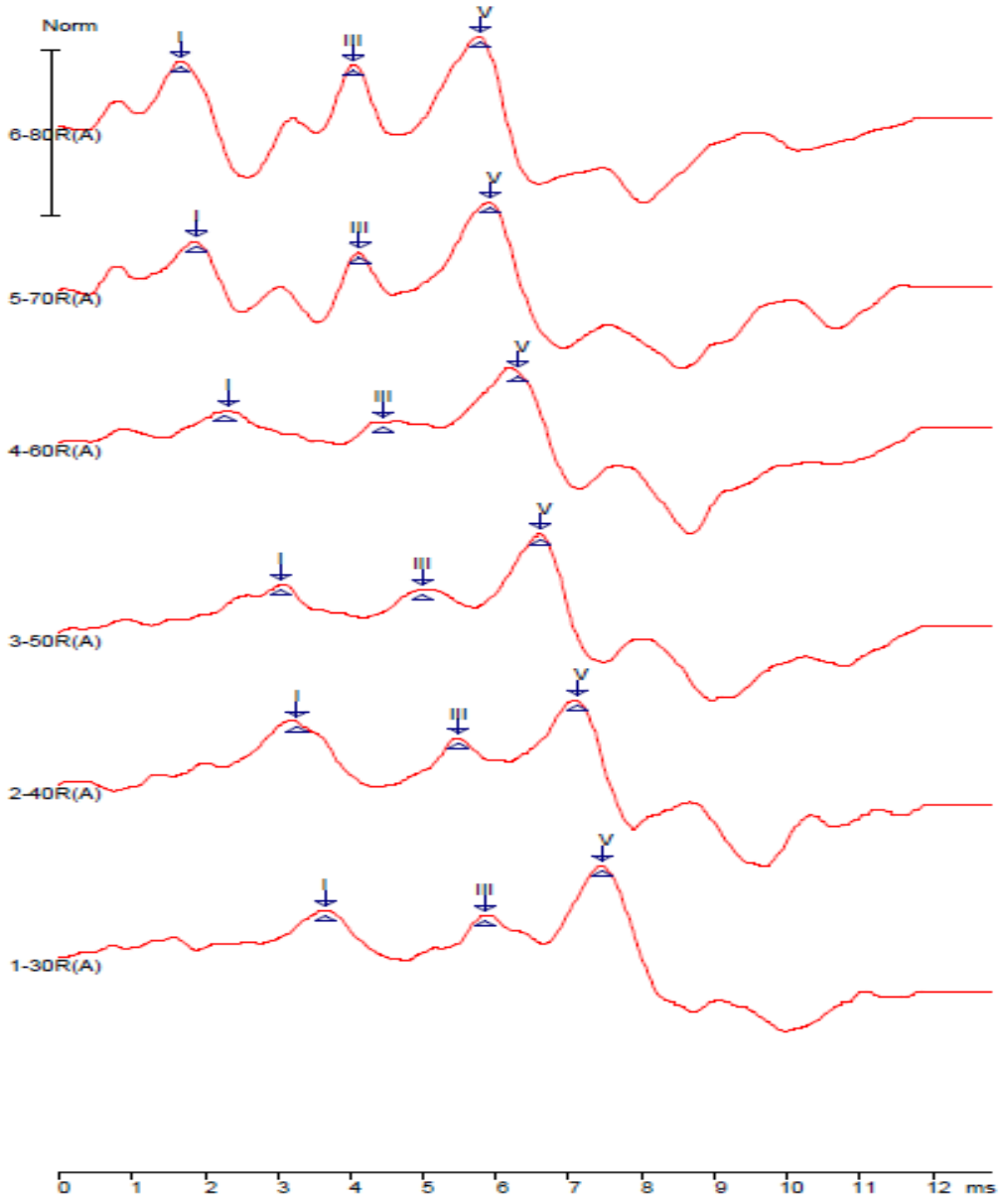

Fig. 4.1: A typical ABR recording from participants

Table 4.1: Mean latencies and standard deviations for male participants

Intensity (dB HL)	Wave I (ms)		Wave III (ms)		Wave V (ms)		Inter-wave I-V (ms)		Inter-wave I-III (ms)		Inter-wave III-V (ms)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
80	1.80	0.18	4.01	0.19	6.00	0.21	4.20	0.29	2.21	0.27	1.99	0.18
70	2.04	0.17	4.20	0.23	6.17	0.24	4.13	0.24	2.16	0.24	1.97	0.21
60	2.29	0.16	4.43	0.25	6.39	0.25	4.09	0.30	2.14	0.26	1.95	0.27
50	2.61	0.23	4.79	0.38	6.76	0.27	4.16	0.34	2.14	0.35	1.96	0.29
40	3.04	0.33	5.19	0.42	7.20	0.46	4.03	0.43	2.17	0.45	1.90	0.30
30	3.55	0.28	5.62	0.42	7.62	0.43	3.90	0.52	2.01	0.35	1.97	0.32

Table 4.2: Mean latencies and standard deviations for female participants

Intensity (dB HL)	Wave I (ms)		Wave III (ms)		Wave V (ms)		Inter-wave I-V (ms)		Inter-wave I-III (ms)		Inter-wave III-V (ms)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
80	1.75	0.20	3.82	0.27	5.75	0.22	4.00	0.30	2.07	0.29	1.93	0.26
70	1.98	0.15	4.03	0.27	5.93	0.28	3.95	0.29	2.05	0.25	1.90	0.25
60	2.29	0.24	4.27	0.31	6.17	0.29	3.90	0.31	1.99	0.23	1.90	0.25
50	2.62	0.33	4.65	0.37	6.48	0.26	3.89	0.33	2.03	0.24	1.84	0.27
40	3.00	0.45	5.01	0.41	6.92	0.28	3.92	0.42	2.02	0.18	1.87	0.31
30	3.51	0.45	5.55	0.31	7.35	0.32	3.95	0.41	2.07	0.37	1.86	0.22

Table 4.3: Mean latencies and standard deviations of combined data

Intensity (dB HL)	Wave I (ms)		Wave III (ms)		Wave V (ms)		Inter-wave I-V (ms)		Inter-wave I-III (ms)		Inter-wave III-V (ms)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
80	1.77	0.19	3.92	0.25	5.88	0.25	4.10	0.31	2.14	0.28	1.96	0.22
70	2.01	0.17	4.12	0.27	6.05	0.29	4.04	0.28	2.11	0.24	1.93	0.24
60	2.29	0.20	4.35	0.29	6.28	0.29	4.00	0.32	2.07	0.26	1.93	0.26
50	2.62	0.29	4.72	0.38	6.62	0.30	4.01	0.35	2.08	0.29	1.90	0.28
40	3.02	0.39	5.10	0.43	7.06	0.40	3.97	0.42	2.09	0.33	1.88	0.30
30	3.53	0.36	5.59	0.37	7.48	0.40	3.92	0.46	2.04	0.35	1.92	0.27

4.2 Testing of Hypotheses 1

H_0: There is no significant difference between the latencies of waves I, III, and V of the clinically established normative values compared to the standard normative values provided by the IHS manufacturer at α level of significance.

The combined data from participants for absolute wave latencies for waves I, III and V was compared with the published normative data provided by the IHS manufacturer for each intensity respectively. The normative data provided for the IHS by the manufacturer for latencies of wave I, III and V are presented in Table 4.4 while results generated from comparisons of the mean latencies of normative data generated with those obtained via the IHS published normative data at an intensity level of 80 dB HL are shown in Table 4.5. The IHS published normative data for adults was established using a stimulus level of 80 dBnHL through to 30 dBnHL at a 10 dB decrement and a click stimulus.

Table 4.4 Published normative data for IHS system

Intensity (dB HL)	**Wave I (ms)**		**Wave III (ms)**		**Wave V (ms)**	
	Mean	SD	Mean	SD	Mean	SD
80	1.60	0.26	4.10	0.31	6.27	0.44
70	1.79	0.32	4.26	0.34	6.50	0.33
60	1.93	0.31	4.46	0.40	6.34	0.27
50	2.28	0.41	4.75	0.37	6.68	0.41
40	2.49	0.48	4.93	0.37	6.86	0.33
30	2.86	0.61	5.08	0.50	7.20	0.44

Table 4.5: Compared mean latencies for wave I, III and V at 80 dBnHL

Waves/Parameters	*n*	df	Mean	SD	SEM	T	*p*-value
Wave I	50	49	1.7734	.18759	.02653	6.913	.000
Wave III	50	49	3.9172	.25318	.03581	7.742	.000
Wave V	50	49	5.8756	.24641	.03485	8.770	.000

σ = standard error of the mean

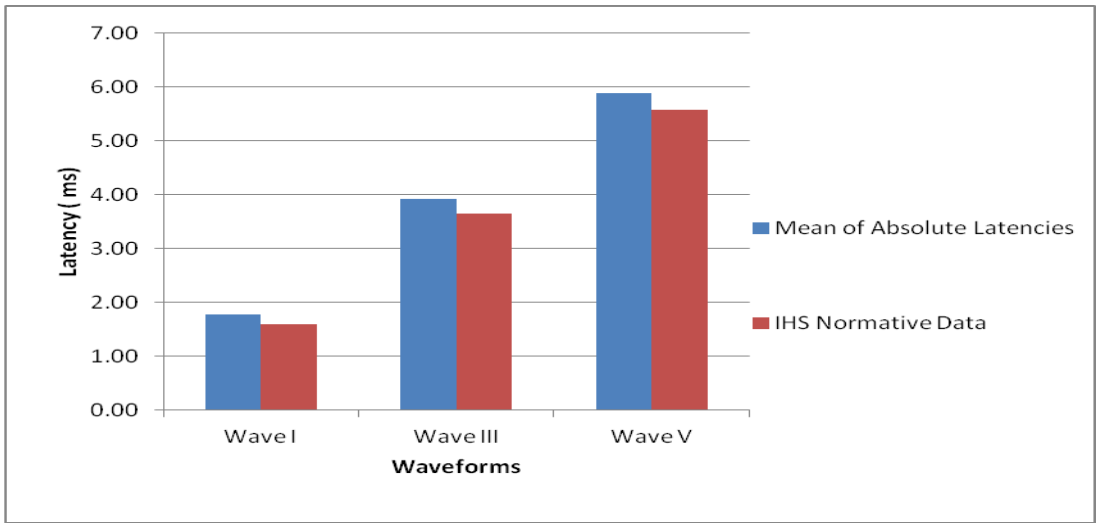

Fig. 4.2: Graphical representation of comparison between clinical data with IHS published values at 80 dBnHL

The wave I latency at 80 dBnHL from the study (*n*=50, *M*=1.78, *SD* = 0.18) compared to that of the IHS normative data (*M*=1.59, *SD*=.24) showed the *p*-value is 0.000. There was significant difference between the clinically established normative data and the IHS normative data at the $p<0.05$ level for the three conditions. In effect, the latency of the clinical normative data was slightly delayed (0.19 ms) compared with that of the IHS normative data.

The wave III latency at 80 dBnHL from the study (*n*=50, *M*=3.92, *SD*=.25) compared

to that of the IHS normative data (*M*=3.64, *SD*=.17), showed the *p*-value is 0.000. There was significant difference between the clinically established normative data and the IHS normative data at the $p<0.05$ level for the three conditions. This is indicative of variation between the clinically established data and the IHS normative data with the clinical data showing a delay of 0.28 ms.

The wave V latency at 80 dBnHL from the study (*n*=50, M=5.87, SD=.25) compared to that of the IHS normative data (M=5.57, SD=.16), showed the *p*-value is 0.000. This shows that the established clinical data was delayed by 0.3 ms as compared with the IHS normative data and therefore, there was a significant difference between the clinically established normative data and the IHS normative data with $p<0.05$ level for the three conditions. Figure 4.2 compares the clinically established normative data at 80 dBnHL to that of the IHS normative data.

A comparison of the generated mean latencies of normative data results with the IHS published normative data at an intensity level of 70 dB HL are shown in Table 4.6. The wave I latency at 70 dBnHL from the study (*n*=50, *M*=2.00, *SD*=.16) compared to that of the IHS normative data (*n*=50, *M*=1.75, *SD*=.21), showed the *p*-value is 0.000. The wave III latency also at 70 dBnHL from the study (*n*=50, M=4.11, SD=.26) compared to that of the IHS normative data (M=3.86, SD=.23) showed the *p*-value is 0.000 and the wave V latency at the same intensity (*n*=50, *M*=6.04, *SD*=.28) compared to that of the IHS normative data (*M*=5.67, *SD*=.15) showed the *p*-value is 0.000.

These showed that there was significant difference between the clinically established normative data and the IHS normative data at the $p<0.05$ level for the waves I, III and V. In effect, the latencies of the clinical normative data at the intensity of 70 dBnHL

are all delayed as compared with those of the IHS normative data. In effect the wave latencies of the clinical data are delayed by the following margin, wave I, 0.25 ms; wave III, 0.25 ms and wave V, 0.37 ms.

Fig. 4.3 displays the graphical comparison of the clinically established normative data at intensity 70 dBnHL to those of the IHS normative values.

Table 4.6: Compared mean latencies for wave I, III and V at 70 dBnHL

Waves/Parameters	*n*	df	Mean	SD	SEM	T	*p*-value
Wave I	50	49	2.0090	.16548	.02340	11.067	.000
Wave III	50	49	4.1156	.26619	.03765	6.790	.000
Wave V	50	49	6.0486	.28503	.04031	9.392	.000

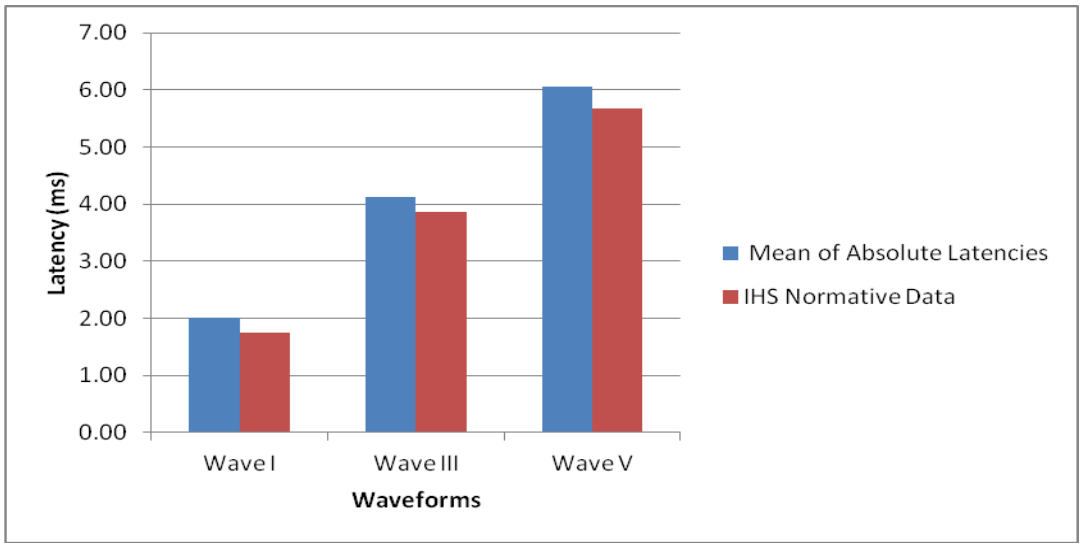

Fig. 4.3: Graphical representation of comparison between clinical data with IHS published values at 70 dBnHL

Table 4.7 below is the summarized SPSS results generated by comparing the mean

latencies of normative data generated by the study to that of the IHS published normative data at an intensity level of 60 dB HL and Fig. 4.4 show the graphical representation.The wave I latency at 60 dBnHL from the study (*n*=50, M=2.29, SD=0.20) compared to that of the IHS normative data (M=1.88, SD=.27), showed the *p*-value is 0.000. Values for wave III latency at the same intensity from the study (*n*=50, M=4.35, SD=0.29) compared to that of the IHS normative data (M=4.11, SD=.20) also showed the *p*-value is 0.000. Additionally, the wave V latency from the study at 60 dBnHL (*n*=50, M=6.27, SD=.29) compared to that of the IHS normative data (M=5.81, SD=.27) showed the *p*-value is 0.000.

These values are indicative of a significant difference between the clinically established normative data and the IHS normative data at $p<0.05$ level for the waves I, III and V at 60 dBnHL which show that the clinical data was significantly delayed compared to the IHS normative data.

Table 4.7: Compared mean latencies for wave I, III and V at 60 dBnHL

Waves/Parameters	***n***	**df**	**Mean**	**SD**	**σ**	**T**	***p*-value**
Wave I	50	49	2.2924	.20170	.02853	14.457	.000
Wave III	50	49	4.3540	.28867	.04082	5.977	.000
Wave V	50	49	6.2798	.28965	.04096	11.469	.000

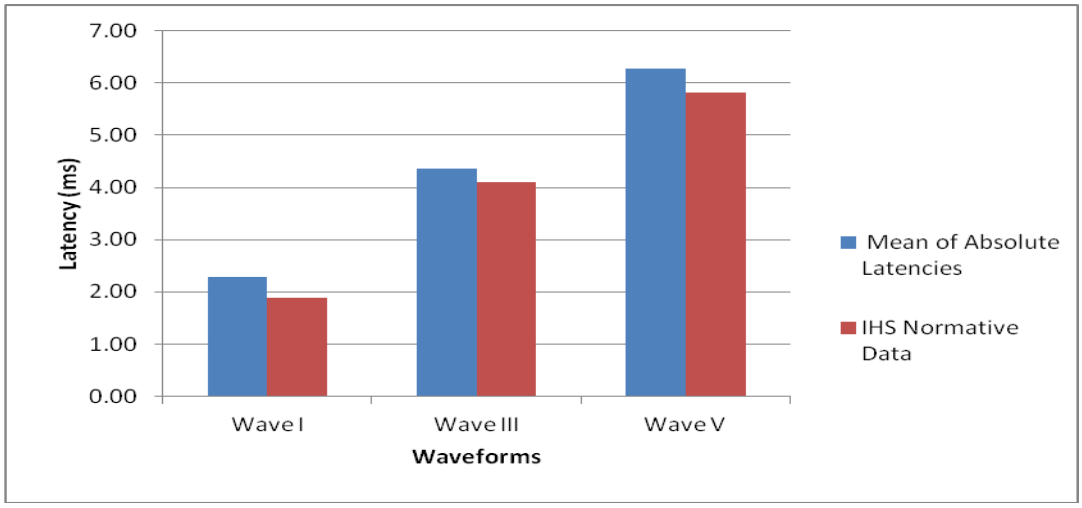

Fig. 4.4: Graphical representation of comparison of clinical data with IHS published values at 60 dBnHL

The wave I latency at 50 dBnHL from the study (n=46, M=2.61, SD=0.28) compared to that of the IHS normative data (M=2.23, SD=.34) showed the p-value is 0.000. There was significant difference between the clinically established normative data and the IHS normative data at the p<0.05 level. In effect, the clinical normative data exhibit a significant delay compared with those of the IHS normative data.

The wave III latency at 50 dBnHL from the study (n=49, M=4.71, SD=0.37) compared to that of the IHS normative data (M=4.49, SD=0.18), showed the p-value is 0.000. There was significant difference between the clinically established normative data and the IHS normative data at the p<0.05 level. This is the clinically established data by the study at this intensity was delayed compared with the IHS normative data.

The wave V latency at 50 dBnHL from the study (n=50, M=6.62, SD=0.29) compared to that of the IHS normative data (M=5.81, SD=0.22), showed the p-value is 0.000. This shows that the latency of wave V of the established clinical data at this intensity was delayed compared with the IHS normative data because there was significant

difference between the clinically established normative data and the IHS normative data at the $p<0.05$ level. Figure 4.5 and Table 4.8 compares the clinically established normative data at 50 dBnHL to that of the IHS normative data.

Table 4.8: Compared mean latencies for wave I, III and V at 50 dBnHL

Waves/Parameters	*n*	df	Mean	SD	σ	T	*p*-value
Wave I	46	45	2..6185	.28580	.04214	9.219	.000
Wave III	49	48	4.7171	.37662	.05380	4.222	.000
Wave V	50	49	6.6236	.29928	.04233	11.898	.000

σ = standard error of the mean

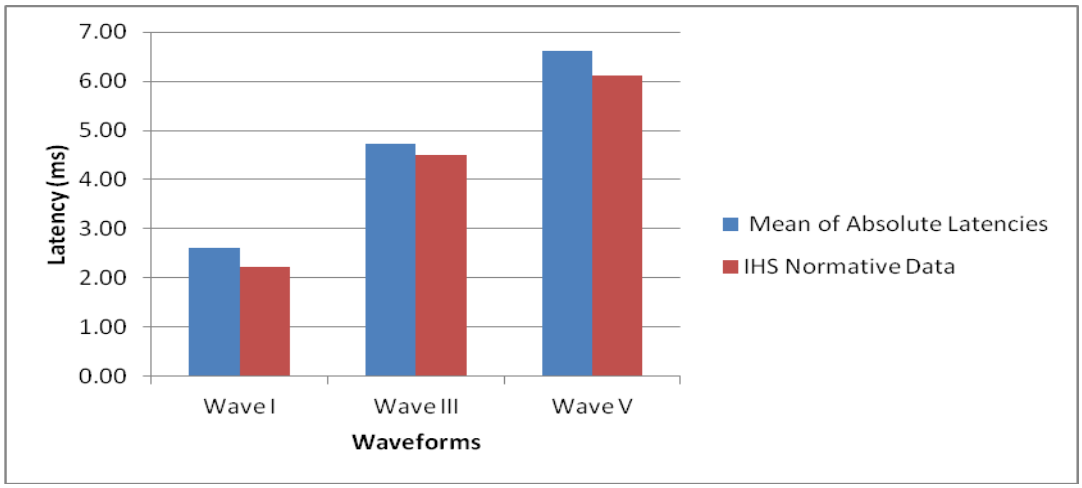

Fig. 4.5 Graphical representation of comparison between clinical data with IHS published values at 50 dBnHL

A comparison of the generated mean latencies of normative data results with the IHS published normative data at an intensity level of 40 dB HL are shown in Table 4.9. The wave I latency at 40 dBnHL from the study (n=38, M=3.01, SD=.39) compared to that of the IHS normative data (M=2.46, SD=.31), showed the p-value is 0.000. The wave III latency also at 40 dBnHL from the study (n=47, M=5.10, SD=.42)

compared to that of the IHS normative data (M=4.55, SD=.26) showed the *p*-value is 0.000 and the wave V latency at the same intensity (*n*=50, M=7.05, SD=0.40) compared to that of the IHS normative data (M=6.29, SD=.27) showed the *p*-value is 0.000.

These showed that there was significant difference between the clinically established normative data and the IHS normative data at the $p<0.05$ level for the three conditions for the waves I, III and V at the intensity of 40 dBnHL. In effect, the clinical normative data at this intensity are significantly delayed compared with those of the IHS normative data.

Fig. 4.6 displays the graphical comparison of the clinically established normative data at intensity 40 dBnHL to those of the IHS normative values.

Table 4.9: Compared mean latencies for wave I, III and V at 40 dBnHL

Waves/Parameters	*n*	df	Mean	SD	σ	T	*p*-value
Wave I	38	37	3.0189	.38988	.06325	8.838	.000
Wave III	47	46	5.1004	.42445	.06191	8.890	.000
Wave V	50	49	7.0580	.40149	.05678	13.526	.000

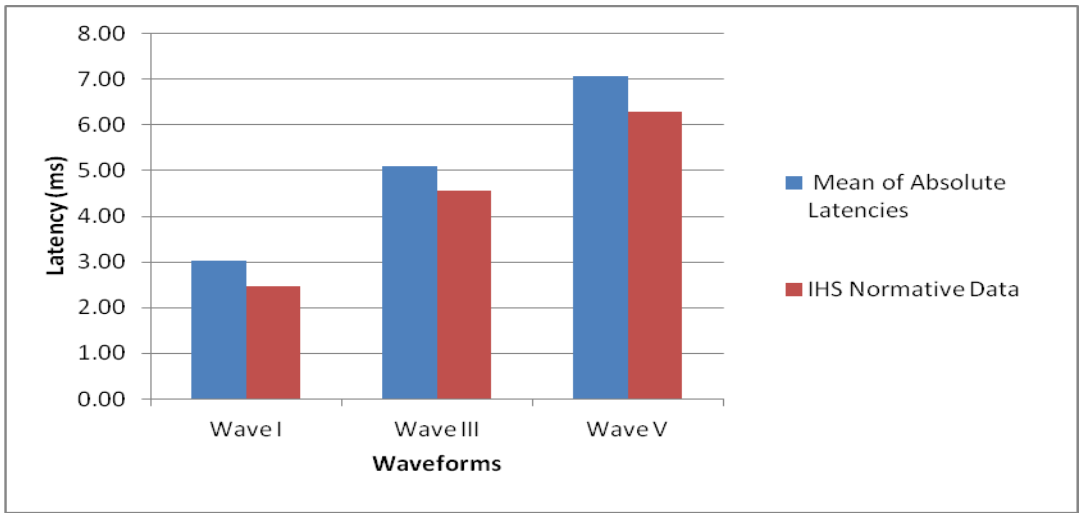

Figure 4.6 Graphical representation of comparison of clinical data with IHS published values at 40 dBnHL

Table 4.10 below is the summarized SPSS results generated by comparing the mean latencies of normative data generated by the study to that of the IHS published normative data at an intensity level of 30 dBnHL. The wave I latency at 30 dBnHL from the study (n=19, M=3.53, SD=0.35) compared to that of the IHS normative data (M=2.83, SD=0.36), showed the p-value is 0.000. There was significant difference between the clinically established normative data and the IHS normative data at the p<0.05 level. In effect, the clinical normative was delayed compared with those of the IHS normative data.

Wave III latency at 30 dBnHL from the study (n=38, M=5.58, SD=0.36) compared of the IHS normative data (M=5.00, SD=0.31), showed the p-value is 0.000. There was significant difference between the clinically established normative data and the IHS normative data at the p<0.05 level. In effect, the clinical normative data was delayed compared with those of the IHS normative data.

The wave V latency at 30 dBnHL from the study (n=48, M=7.48, SD=.39) compared

to that of the IHS normative data (M=6.69, SD=.30), *p*-value is 0.000. There was significant difference between the clinically established normative data and the IHS normative data at the $p<0.05$ level. In effect, the clinical normative data was delayed compared with those of the IHS normative data. Figure 4.7 displays the graphical comparison of our data to the normative data at Intensity 30 dBnHL.

Table 4.10: Compared mean latencies for wave I, III and V at 30 dBnHL

Waves/Parameters	*n*	df	Mean	SD	σ	T	*p*-value
Wave I	19	18	3.5316	.35637	.08176	8.581	.000
Wave III	38	37	5.5876	.36787	.05968	9.847	.000
Wave V	48	47	7.4815	.39842	.05751	13.763	.000

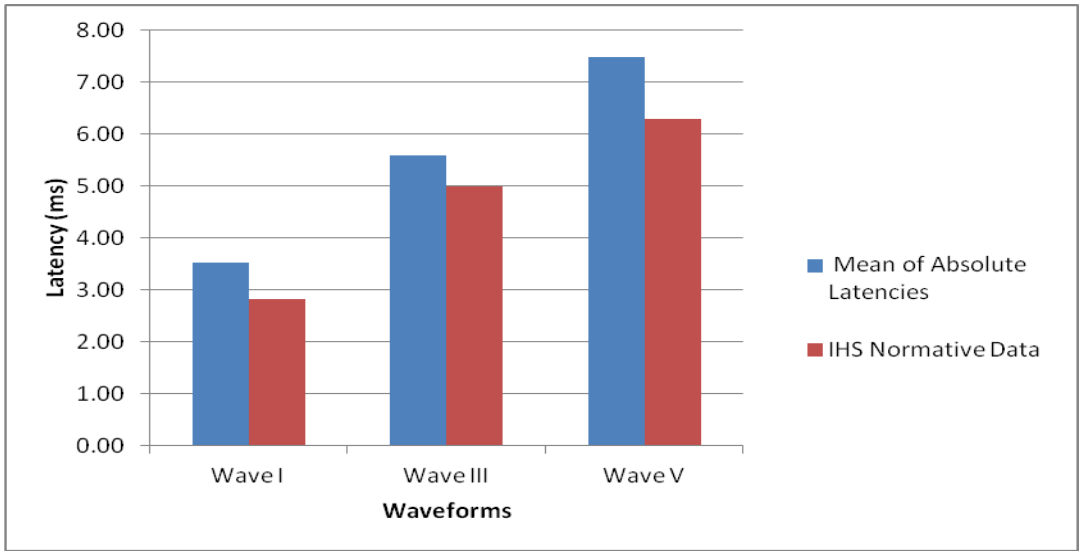

Fig. 4.7: Graphical representation of comparison between clinical data with IHS published values at 30 dBnHL

4.3 Testing of Hypotheses 2

H_0: The inter-wave latencies of wave I-III, I-V, III-V of the clinically established normative values compared to the standard normative values provided by the manufacturer does not show any significant difference at α level of significance.

Table 4.11 is the summarized SPSS results generated by comparing the mean of the clinically generated normative data from the study to those of the IHS normative data at the inter-wave latency (Wave I – V). At 80 dBnHL, the clinically generated normative data (n=50, M=4.10, SD=.31) compared to that of the IHS normative data (M=3.98, SD=.25), showed the p-value is 0.007. There was significant difference between the clinically established normative data (wave I-V) and to the IHS normative data at the p<0.05 level for the three conditions. In effect, the clinical normative data exhibit a significant delay compared with those of the IHS normative data.

Comparing the clinically generated normative data at 70 dBnHL, the results showed that the clinical data (n=50, M=4.04, SD=.27) compared to that of the IHS normative data (M=3.92, SD=.22) had a p-value of 0.004. The result is indicative that there was significant difference between the clinically established normative data (wave I-V) and to the IHS normative data at the p<0.05 level for the three conditions. In effect, the latency of the clinical normative data was slightly delayed as compared to those of the IHS normative data.

However, at 60 dBnHL, the clinically generated normative values for the inter-wave latency (n=50, M=3.99, SD=0.32) compared to that of the IHS normative data (M=3.93, SD=.22), showed the p-value is 0.133. This means that there was no significant difference between the clinically established normative data (inter-wave I-

V at 60 dBnHL) and the IHS normative values at the $p>0.05$ level. In effect, the clinical normative data does not exhibit any significant delay compared with those of the IHS normative data at this intensity.

At 50 dBnHL unlike at 60 dBnHL, the inter-wave latency I-V of the clinically generated normative data (n=46, M=4.01, SD=0.35) was delayed compared with that of the IHS normative data (M=3.89, SD=.24). The p-value for the comparison was 0.026 showing that there was significant difference between the clinically established normative data (wave I-V) and the IHS normative data at the $p<0.05$ level.

Similarly, at 40 dBnHL, the inter-wave latency I-V of the clinically generated normative data (n=38, M=3.96, SD=.42) compared to that of the IHS normative data (M=3.83, SD=.15), showed the p-value is 0.050. There was significant difference between the clinically established normative data (wave I-V) and the IHS normative data at the $p<0.05$ level. In effect, the clinical normative data was delayed compared with those of the IHS normative data at intensity 40 dBnHL.

In contrast at 30 dBnHL, the inter-wave latency I-V of the clinically generated normative data (n=19, M=3.92, SD=.45) does not exhibit any significant delay compared with that of the IHS normative data (M=3.86, SD=.30), with the p-value of 0.553. In effect, there was no significant difference between the clinically established normative data (inter-wave I-V at 30 dBnHL) and the IHS normative data at the $p>0.05$ level. Figure 4.8 displays the graphical comparison of our data to the normative data.

Table 4.11: Compared inter-wave latency's mean (I-V)

Wave I – V	*n*	df	Mean	SD	σ	T	p-value
Intensity 80	50	49	4.1026	.30773	.04352	2.817	.007
Intensity 70	50	49	4.0400	.27686	.03915	3.065	.004
Intensity 60	50	49	3.9986	.31766	.04492	1.527	.133
Intensity 50	46	45	4.0100	.35325	.05208	2.304	.026
Intensity 40	38	37	3.9682	.41973	.06809	2.029	.050
Intensity 30	19	18	3.9232	.45534	.10446	.605	.553

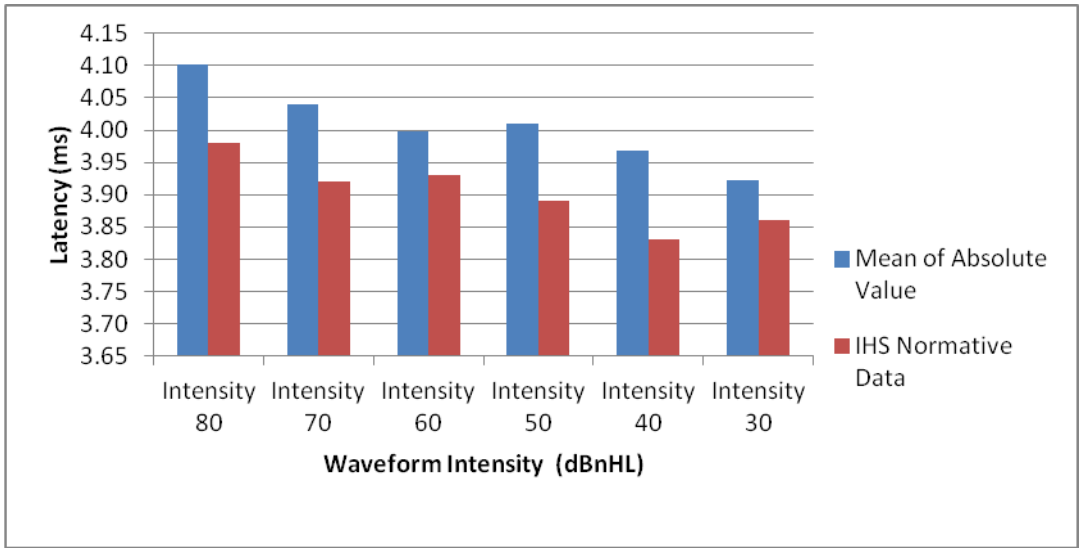

Fig. 4.8: Graphical representation of comparison between inter-wave (I-V) latencies of clinical data with IHS published values

Table 4.12 shows the mean inter-wave values of waves I-III and III-V, as well as the means and standard deviations of the inter-wave latencies at various intensities and how they vary with intensity decrease.

Table 4.12: Mean of inter-wave latencies (I-III, III-V) and standard deviations of combined data

Intensity	Inter-wave I-III		Inter-wave III-V	
(dB HL)	Mean	SD	Mean	SD
80	2.14	0.28	1.96	0.22
70	2.11	0.24	1.93	0.24
60	2.07	0.26	1.93	0.26
50	2.08	0.29	1.90	0.28
40	2.09	0.33	1.88	0.30
30	2.04	0.35	1.92	0.27

These values were not compared with the IHS normative data because the IHS normative data does not have figures or values for the inter-wave latencies and standard deviations for waves I-III and III-V. However, since these inter-wave latencies also play an important role in the diagnostic process, the researcher compared these inter-wave latencies with Hood (1998) published normative data to find out how the values compared to them. Table 4.13 below is the inter-wave latencies (I-III and III-V) from Hood (1998).

Table 4.13 Hood's normative data

Intensity (dB HL)	Wave I-III (ms)		Wave III-V (ms)	
	Mean	SD	Mean	SD
80	2.06	0.11	1.79	0.09
70	2.03	0.11	1.79	0.12
60	2.02	0.12	1.72	0.10
50	2.02	0.19	1.56	0.18
40	1.85	0.14	1.71	0.14
30			1.74	0.26

Table 4.14 is the summarized SPSS results generated by comparing the mean of the clinically generated normative data from the study to those of the Hood's normative data at the inter-wave latency (Wave I – III).

At 80 dBnHL, the clinically generated normative data (n=50, M=2.14, SD=0.28) compared to that of Hood's normative data (M=2.06, SD=0.11), showed a p-value of 0.041. There was statistical significant difference between the clinically established normative data (wave I-III) and Hood's normative data at the p<0.05. In effect, the clinical normative data exhibit significant delay of latency compared with those of the Hood's normative data.

Comparing the clinically generated normative data at 70 dBnHL, the results showed that the clinical data (n=50, M=2.10, SD=0.24) compared to that of Hood's normative data (M=2.03, SD=0.11) there was significant difference between the clinically established normative data (wave I-III) and the Hood's normative data at the p<0.05 (p-value = 0.032). In effect, the latency of the clinical normative data was slightly

delayed as compared to those of Hood's normative data.

However, at 60 dBnHL, the clinically generated normative values (n=50, M=2.06, SD=0.25) compared to that of the Hood's normative data (M=2.02, SD=0.12), showed there was no statistical significant difference between the clinically established normative data (inter-wave I-III at 60 dBnHL) and the Hood's normative values at the p>0.05 level (p-value = 0.205). In effect, the clinical normative data does not exhibit any statistical significant delay compared with those of Hood's normative data at this intensity.

At 50 dBnHL, the inter-wave latency I-III of the clinically generated normative data (n=46, M=2.08, SD=0.29) compared to that of Hood's normative data (M=2.02, SD=0.19). The p-value for the comparison was 0.156 showing that there was no statistical significance difference between the clinically established normative data and the Hood's normative data at the p<0.05.

However at 40 dBnHL, the inter-wave latency I-III of the clinically generated normative data (n=38, M=2.09, SD=0.33) compared to that of the Hood's normative data (M=1.85, SD=0.14), showed the p-value is 0.000. There was a significant difference between the clinically established normative data (wave I-III) and the Hood's normative data at the p<0.05 level. In effect, the latency of clinical normative data was delayed compared with that of Hood's normative data at intensity 40 dBnHL.

At the intensity of 30 dBnHL, there no comparison was done for the inter-wave latency for waves I-III because Hood's normative data does not have any value for that intensity. Figure 4.9 displays the graphical comparison of the clinical data to

Hood's normative data.

Table 4.14: Compared inter-wave latency's mean (I-III)

Wave I – III	*n*	df	Mean	SD	σ	T	p-value
Intensity 80	50	49	2.1440	.28278	.3999	2.100	.041
Intensity 70	50	49	1.9344	.23552	.03331	4.335	.000
Intensity 60	50	49	2.0666	.25632	.03625	1.286	.205
Intensity 50	46	45	2.0826	.29455	.04343	1.442	.156
Intensity 40	38	37	2.0900	.32964	.05347	4.488	.000
Intensity 30			.	.	.	.	.

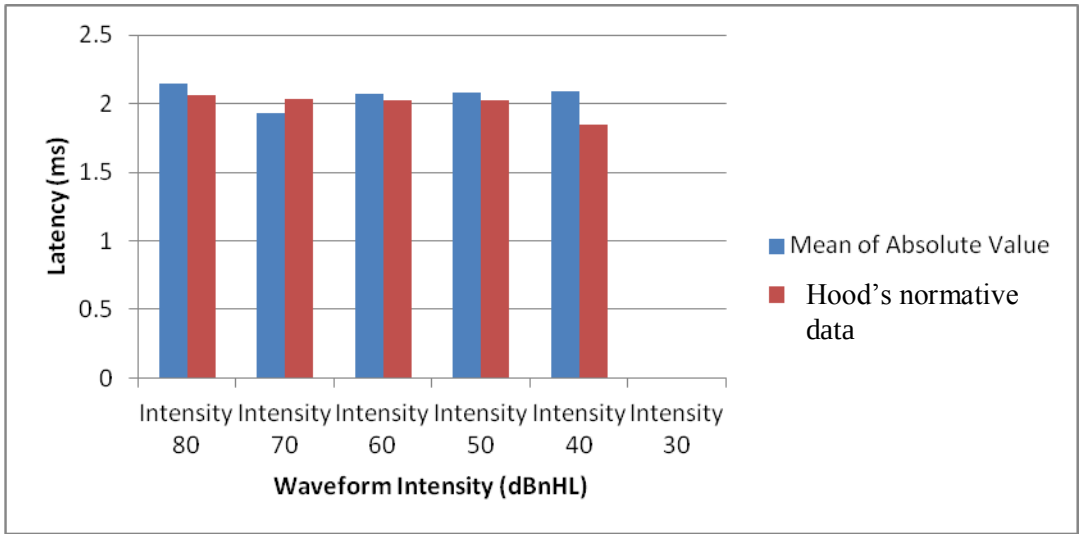

Fig. 4.9: Graphical representation of comparison between inter-wave (I-III) latencies of clinical data with Hood's normative values.

Table 4.15 is the summarized SPSS results generated by comparing the means of the clinically generated normative data from the study to those of Hood's normative data for the inter-wave latency III – V. At 80 dBnHL, the clinically generated normative data (*n*=50, M=1.96, SD=0.22) compared to that of the Hood's normative data (M=1.79, SD=0.09), showed the *p*-value is 0.000. This is indicative a statistical significant difference between the clinically established normative data (wave III-V)

and to Hood's normative data at the $p<0.05$. In effect, the latency of the clinical normative data exhibit a significant delay compared with those of Hood's normative data.

Comparing the inter-wave latency of the clinically generated normative data at 70 dBnHL, the results showed that the clinical data (n=50, M=1.93, SD=0.23) compared to that of the Hood's normative data (M=1.79, SD=0.12) there was a statistical significant difference between the clinically established normative data and Hood's normative data at the $p<0.05$ (p-value of 0.000). In effect, the latency of the clinical normative data was delayed significantly compared to that of Hood's normative data.

Similarly, at 60 dBnHL, the clinically generated normative values for the inter-wave latency (n=50, M=1.92, SD=0.25) compared with Hood's normative data (M=1.72, SD=.10), showed the p-value is 0.000. This means that there was statistical significant difference between the clinically established normative data (inter-wave III-V at 60 dBnHL) and Hood's normative values at the $p<0.05$. In effect, this is indicative that the latency of the clinical normative data exhibit a significant delay compared with Hood's normative data at this intensity.

Additionally, at 50 dBnHL the inter-wave latency III-V of the clinically generated normative data (n=49, M=1.90, SD=0.28) was delayed compared with that of the Hood's normative data (M=1.56, SD=.18). The p-value for the comparison was 0.000 showing that there was significant difference between the clinically established normative data (wave III-V) and Hood's normative data at the $p<0.05$.

Furthermore, at 40 dBnHL, the inter-wave latency III-V of the clinically generated normative data (n=47, M=1.18, SD=.30) compared to that of the Hood's normative

data (M=1.71, SD=.14), showed the *p*-value is 0.000. Also, at 30 dBnHL the clinically generated normative data (n=39, M=1.91, SD=.27) exhibits a significant delay compared with that of the Hood's normative data (M=1.74, SD=.26), with a *p*-value of 0.000. These are indicative of statistical significant difference between the clinically established normative data (wave III-V) at these intensities compared with Hood's normative data at the $p<0.05$. In effect, the clinical normative data was delayed compared with those Hood's normative data at intensities 40 and 30 dBnHL.

Table 4.15: Compared inter-wave latency's mean (III-V)

Wave III – V	*n*	df	Mean	SD	σ	T	p-value
Intensity 80	50	49	1.9600	.22246	.03146	5.404	.000
Intensity 70	50	49	1.9344	.23552	.03331	4.335	.000
Intensity 60	50	49	1.9268	.25893	.03662	5.648	.000
Intensity 50	49	48	1.9010	.28259	.04037	8.447	.000
Intensity 40	47	46	1.8845	.30348	.04427	3.941	.000
Intensity 30	39	38	1.9167	.27413	.04390	4.025	.000

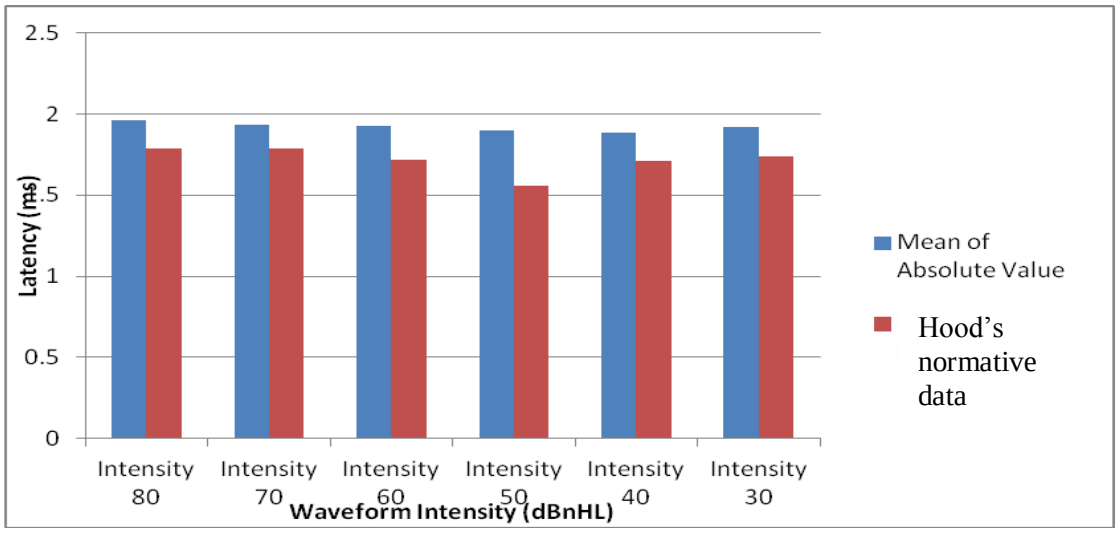

Fig. 4.10: Graphical representation of comparison between inter-wave (III-V) latencies of clinical data with Hood's normative values.

CHAPTER FIVE

DISCUSSION

5.1 INTRODUCTION

The results of this study based on the hypotheses set for the research work are presented here.

5.2 HYPOTHESES

5.2.1 Hypotheses 1

H_0: There was significant difference between the latencies of waves I, III, and V of the clinical established normative values compared to the standard normative values provided by the IHS manufacturer at the α level of significance.

Results of the clinical normative data generated by this research project from the 50 participants for the waves I, III and V latencies across the intensities range of 80 dBnHL to 30 dBnHL suggest that the normative data compared to those of the IHS system were significantly delayed. Hood (1998) and Sininger and Hyde (2009) suggested that the manipulations of test stimuli affect both the latency and the amplitude of early evoked potentials. One of such stimulus factor is the filter setting. Filtering and filter type affect ABR results since changes in frequency band through which the ABR is filtered affect waveform latency and amplitude, i.e., increasing the high-pass filter cutoff frequency from 30 Hz to 100 Hz or 150 Hz results in decrease in amplitude and latency of response (Hood, 1998; Sininger and Hyde, 2009). Additionally, as the low pass filter is increased from 300 to 3000 Hz, the latencies of all the waves decrease and there is improvement in the resolution of waves I and V (Silman and Silverman, 1991). Therefore, due to the variability of filter change between the clinical data of the study (30 to 1500 Hz) and that used by the

manufacturer (30 to 3000 Hz), there is the likelihood of significant differences between the two data. According to Hall (1992), interpretation of auditory evoked responses using specific normative data base may be invalid after filter settings have been significantly altered.

Due to the variability of testing environment and its acoustic characteristics, there is the tendency that wave latencies would show significant differences. These, in effect explain the significant delay in the latencies of the clinically established data compared to those of the IHS normative data. However, on the issues of determining the significance of absolute latency value for a normative data or otherwise for clinical use, Hood (1998) and Atcherson (2012) suggested that the absolute latency of wave I should occur at approximately 1.6 ms after stimulation onset, wave III at about 3.7 ms, and wave V at about 5.6 ms for clicks presented at an intensity of approximately 75 dB above normal threshold. They further noted that the normal latency limit range should be within either two or three standard deviations from the mean values. This means that wave I would be 1.6 ms ± 0.2 ms, wave III, 3.7 ms ± 0.2 ms and wave V, 5.6 ms ± 0.2 ms.

In a related study, Ness (2009) citing Hall (2006) on published normative data reported that the absolute latencies for normal hearing adults wave I was 1.65 ms (2 SD of 0.28 ms), 3.80 ms (2 SD of 0.36 ms) for waveform III, and 5.64 ms (2 SD of 0.46 ms) for waveform V. With these reference points, using the IHS normative data as another reference point, then, all the clinically established data for the absolute latencies fell favorably within the ±0.2 ms standard deviation allowance. In effect, the clinically established normative data are appropriate and can be used for diagnosis purposes.

5.3 Hypotheses 2

H_0: The inter-wave latencies of wave I-III, I-V, III-V of the clinically established normative values compared to the standard normative values provided by the manufacturer show significant difference at α the level of significance.

Results from the comparison of the clinically established data with the IHS normative data for the inter-wave latencies for waves I-V across the intensities used for the study (80 to 30 dBnHL) suggest that out of the six intensities, four (80, 70, 50 and 40 dBnHL) showed significant delays, while two the latencies at two intensities (50 and 30 dBnHL) did not show any significant delay. Additionally, results from comparison between the clinically established data for inter-waves I-III and III-V showed that the latencies were significantly delayed compared to that of Hood's normative data.

According to Hood (1998) and Atcherson (2012), the inter-wave latencies used in clinical interpretation of ABR waveforms pertain to waves I to III, waves III to V and waves I to V, and opined that the inter-wave intervals for waves I-III and III-V should approximate 2.0 ms and the wave I-V interval, about 4.0 ms taking into account a standard deviation of ± 0.4 ms for the normal limits of the I-V interval. In a related study, Ness (2009) reported mean inter-wave latency values of 2.15 ms with a 2 SD of 0.28 for wave I-III, 1.84 ms with a 2SD of 0.28 ms for waves III-V, and 3.99 ms with a 2 SD of 0.40 ms for waves I-V. However, it is observed that the inter-wave latency is usually considered abnormal if it is greater than 2 or 2.5 standard deviations above the mean. In effect, the inter-wave latencies of the clinically established normative values must not be greater than 2 or 2.5 SD above their respective mean in order to be considered normal. Based on these findings, the inter-wave normative data generated by this research project are consistent with the conditions mentioned above.

In light of the aforementioned discussions, the results of this study are consistent with studies on normative data based on the standard deviation allowance for any data to be considered significant or not. In contrast, this is not so with data provided by the manufacturer for the IHS system. This signifies that the data generated by this study are appropriate and can be used for determining the possible presence of hearing difficulties and retrocochlear pathologies during neurodiagnostic ABR testing and evaluation. This is particularly relevant for the KBTH HAC taking into account the testing protocol and the testing environment. Pathological disorders prolong ABR latencies. Accordingly, Ness (2009) noted that the upper latency limit is determined by applying +2 or +2.5 SD to the mean values. On the lower limits of the applied standard deviation range, Ness (2009) observed that it is not used to delineate between a normal auditory system and an auditory system with a retrocochlear pathology.

CHAPTER SIX

CONCLUSIONS AND RECOMMENDATIONS

6.0 INTRODUCTION

This section presents the summary of the research findings, conclusion and appropriate recommendation.

6.1 SUMMARY OF THE STUDY

It was the aim of this study to develop in-house normative data for neurodiagnostic ABR testing at the KBTHHAC. Based on this, the study specifically sought to find out:

- The relationship between the clinically established normative data with the standardized normative data provided by the manufacturer of the IHS.
- The effect of filter change on the ABR results.
- What the normative data for waves I, III, V and inter-wave latencies I-III, I-V and III-V were for the KBTHHAC.

The study involved 50 participants comprised of 25 males and 25 females between the ages of 18 and 35 years. Convenience and purposive sampling were used for the selection of the participants based on the inclusion criteria requirement. The main instrument used for the study was the IHS (an electrophysiological measure) for the collection auditory click evoked response (waveforms) from each participant. The responses were graphically recorded on a screen representing each participant's auditory brainstem response. The physiological measure provided precision in data collection devoid of any tester or participant bias. The means and standard deviations of all the data on the various waveforms were computed and the results compared to standardized normative data to determine if there was any significant difference.

Below are the findings from this study:

- The absolute latencies of waves I, III, and V for the normative data established by this research project at all test intensities were significantly delayed compared with the IHS normative data provided by the manufacturer. However, the data fulfilled the required standards for absolutes latencies based on how much variation is allowed for the upper or lower limits for normative data to be valid or invalid.
- The inter-wave latencies of waves I-III, III-V, and I-V of the normative data established by this study were significantly delayed compared with the manufacturer's normative data. However, comparing the data with other related studies, the entire waveform values were found to be consistent with and conforming to agreed standards.
- The effect of filter change is very critical in ABR testing and altering the filters can significantly affect the ABR results and hence the interpretation.
- The data collected by this research were appropriate and can be used for determining the possible presence of hearing difficulties and retrocochlear pathologies during neurodiagnostic ABR testing and evaluation.

6.2 CONCLUSION

Results from the study are very appropriate for neurodiagnostic purposes most importantly, for the KBTHHAC taking into consideration the testing protocols and the testing environment. That is, for adult ABR testing and diagnosis purposes, the protocol used for the study and the normative data established are very appropriate and highly recommended. This represents what clinical standards are and the latency and inter-wave data are for the various intensities.

6.3 RECOMMENDATION

Based on the findings of the study, the following recommendations were made:

- The clinically established normative data by the study is appropriate and should be used for neurodiagnostic purposes for the KBTH HAC.
- The results of the study is very conducive for adult population ABR testing, therefore, there is the need for the development of clinical normative data for neonates and infants for the KBTH HAC.
- It is further recommended that a determination of amplitude values and amplitude ratios for patients at the KBTH HAC be made to enhance the diagnostic ability of the clinician.

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APPENDIX I

PARTICIPANT INFORMATION FORM

School of Allied Health Sciences

College of Health Sciences

University of Ghana

Department of Audiology

Title of research: Establishing Clinical Normative Data for Neurodignostic Auditory Brainstem Response Testing For The Korle-Bu Teaching Hospital

Principal Investigator: Sesi Collins Akotey
Department of Audiology
Professional MSc Audiology
Mob: 0244954391; email: scakotey@yahoo.com

General Information about Research

Under the supervision of Professor Amedofu, G.K., and Dr Anim Sampong of the University of Ghana School of Allied Health Sciences, I, Sesi Collins Akotey, a graduate student in research of the Department of Audiology. I am conducting research on establishing clinical normative data for neurodiagnostic auditory brainstem response testing for Korle-Bu Teaching Hospital. The purpose of the study is to collect data by conducting ABR testing on normal hearing participants for establishing clinical normative data for the Korle-Bu Teaching Hospital for neurodiagnostic ABR testing. The process will involve checking your hearing status and connecting you to an ABR testing equipment and measuring your hearing sensitivity with regards to the ABR testing. This data will later be analyzed with other data for the establishment of the clinical norms.

Possible Risks and Discomforts

There are minimal risks for participation in this study since the testing equipment and procedure does give any side effect.

Possible Benefits

Participating in the study provides you with the opportunity of knowing your hearing status and the presence or not of any hidden hearing problem without any cost.

Alternatives to Participation

In the event of any noticed problem the participant will be referred for follow-up for further testing and the necessary action as needed.

Confidentiality

All information provided will remain confidential and will only be reported as group data with no identifying information. All data, including test results will be kept in a secure location and only those directly involved with the research will have access to them.

Compensation

Participants who commute to have the testing done will have their transportation fares catered for. Those who do not will receive a small token from the principal investigator. There shall be no additional cost to be borne by participants who participate in the study.

Voluntary Participation and Right to Leave the Research

Participation in this research study is voluntary. You have the right to withdraw at any time or refuse to participate entirely without any jeopardy to you whatsoever.

Contacts for Additional Information

For any information, clarification or questions about the study, please contact the principal investigator, Sesi Collins Akotey on 0244954391.

Your rights as a Participant

This research has been reviewed and approved by the Ethics and Protocol Committee of the School of Allied Health Sciences, College of Health Sciences, university of Ghana. If you have any questions about your rights as a research participant you can contact the EPC Office between the hours of 8am-5pm through the landline +233-302-687974/5 or postal addresses: Box KB 143, Korle-Bu, Accra.

APPENDIX II

VOLUNTEER AGREEMENT

The document describing the benefits, risks and procedures for the research: Establishing Clinical Normative Data For Neurodignostic Auditory Brainstem Response Testing For The Korle-Bu Teaching Hospital has been read and / or explained to me. I have been given an opportunity to have any questions about the research asked and answered to my satisfaction. I agree to participate as a volunteer.

_____ _____

Date Signature or mark of volunteer

If volunteers cannot read the form themselves, a witness must sign here:

I was present while the benefits, risks and procedures were read to the volunteer. All questions were answered and the volunteer has agreed to take part in the research.

_____ _____

Date Signature of Witness

I certify that the nature and purpose, the potential benefits, and possible risks associated with participating in this research have been explained to the above individual.

_____ _____

Date Signature of Person Who Obtained Consent

APPENDIX III

SCHOOL OF ALLIED HEALTH SCIENCES
COLLEGE OF HEALTH SCIENCES
UNIVERSITY OF GHANA
ACADEMIC AFFAIRS

Phone: +233-0302-687974/5
Fax: +233-0302-688291

My Ref. No. SAHS/ 10231377
Your Ref. No.

P. O .Box KB 143
Korle Bu
Accra
Ghana

19th April, 2013.

Mr. Collins Sesi Akotey,
Dept. of Audiology,
SAHS,
Korle Bu.

Dear Mr. Akotey,

ETHICS CLEARANCE

Ethics Identification Number: SAHS – ET. /10373996/AA/3A/2012-2013.

Following a meeting of the Ethics and Protocol Review Committee of the School of Allied Health Sciences held on Friday 10th April, 2013, I write on behalf of the Committee to approve your research proposal as follows:

TITLE OF RESEARCH PROPOSAL: **"Establishing Clinical Normative Data for Neurodiagnostic Auditory Brainstem Response Testing For the Korle-Bu Teaching Hospital"**

This approval requires that you submit six-monthly review reports of the protocol to the Committee and a final full review to the Committee on completion of the research. The Committee may observe the procedures and records of the research during and after implementation.

Please note that any significant modification of the research must be submitted to the Committee for review and approval before its implementation.

You are required to report all serious adverse events related to this research to the Committee within seven (7) days verbally and fourteen (14) days in writing.

As part of the review process, it is the Committee's duty to review the ethical aspects of any manuscript that may be produced from this research. You will therefore, be required to furnish the Committee with any manuscript for publication.

Please always quote the ethical identification number in all future correspondence in relation to this protocol.

Thank you.

Yours sincerely,

Dr. (Maj. Rtd.) George Asare
(Chairman, Ethics and Protocol Review Committee)

cc Dean
Co-ordinator, Dept. of Audiology
Senior Assistant Registrar

APPENDIX IV

SCHOOL OF ALLIED HEALTH SCIENCES
COLLEGE OF HEALTH SCIENCES
UNIVERSITY OF GHANA
DEPARTMENT OF AUDIOLOGY

Phone: +233-0302-687974/5
Fax: +233-0302-688291

P O Box KB 143,
Korle Bu

My Ref. No. SAHS/

Your Ref. No.

March 21, 2013

The Head
Hearing Assessment Centre, KBTH
Korle Bu Teaching Hospital

Dear Sir,

PERMISSION TO CARRY MSc RESEARCH PROJECT AT THE HEARING ASSESSMENT CENTRE, KORLE BU TEACHING HOSPITAL

Mr. Sesi Collins Akutey is a 2nd year MSc Audiology student in the Department of Audiology of the University of Ghana School of Allied Health Sciences (SAHS).

He is carrying out a MSc research dissertation project on establishing normative data for neurodiagnostic ABR for KBTH under the supervision of Prof. G.K. Amedofu (KNUST) and Dr. S. Anim-Sampong (SAHS). The SAHS Ethical and Protocols Review Committee has reviewed and passed his work as meeting all ethical requirements.

The Department would be most grateful if you could kindly grant him permission to carry out this research project from April – June 2013 for the common good of the University and the hospital. Thank you.

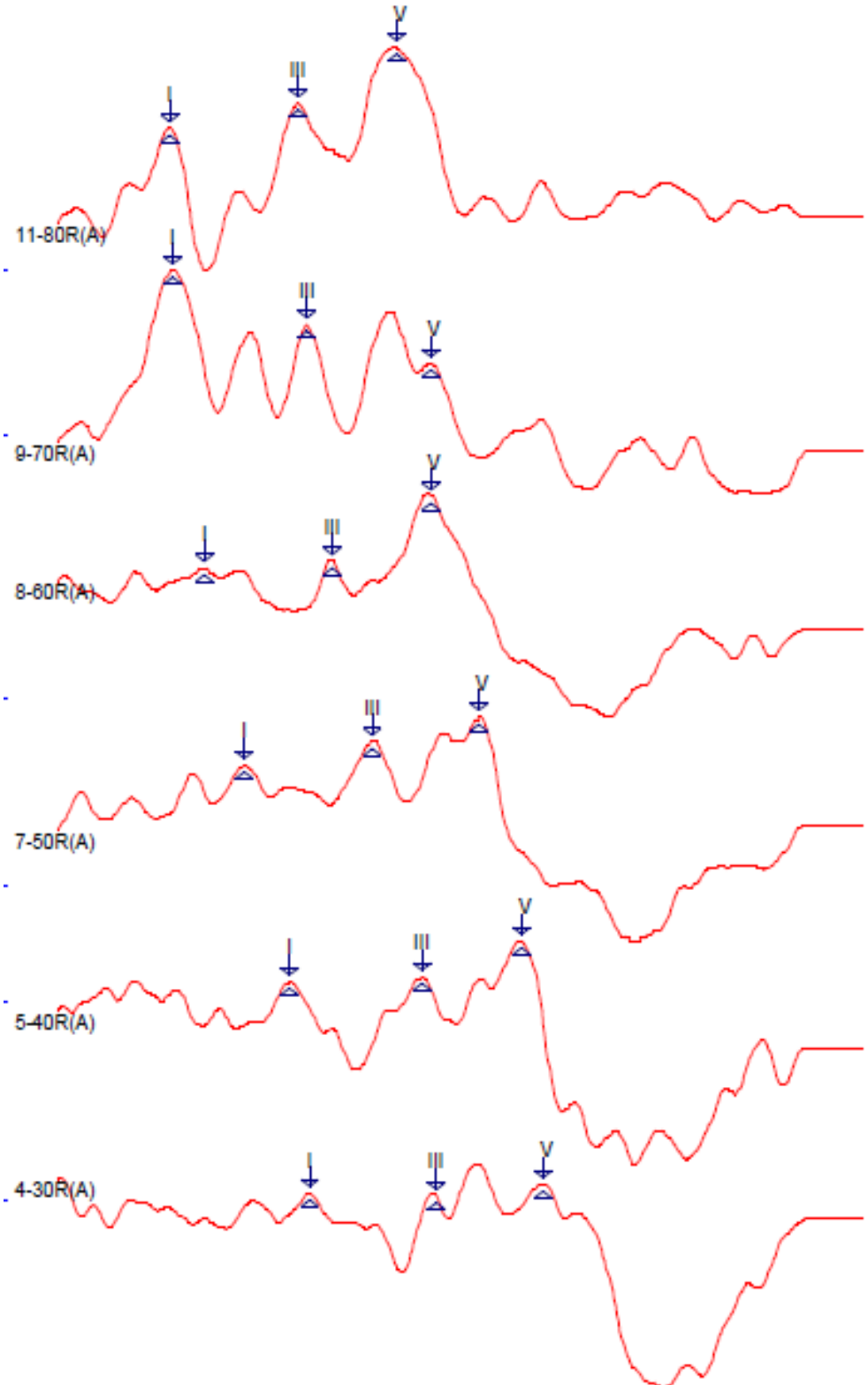
Yours faithfully,

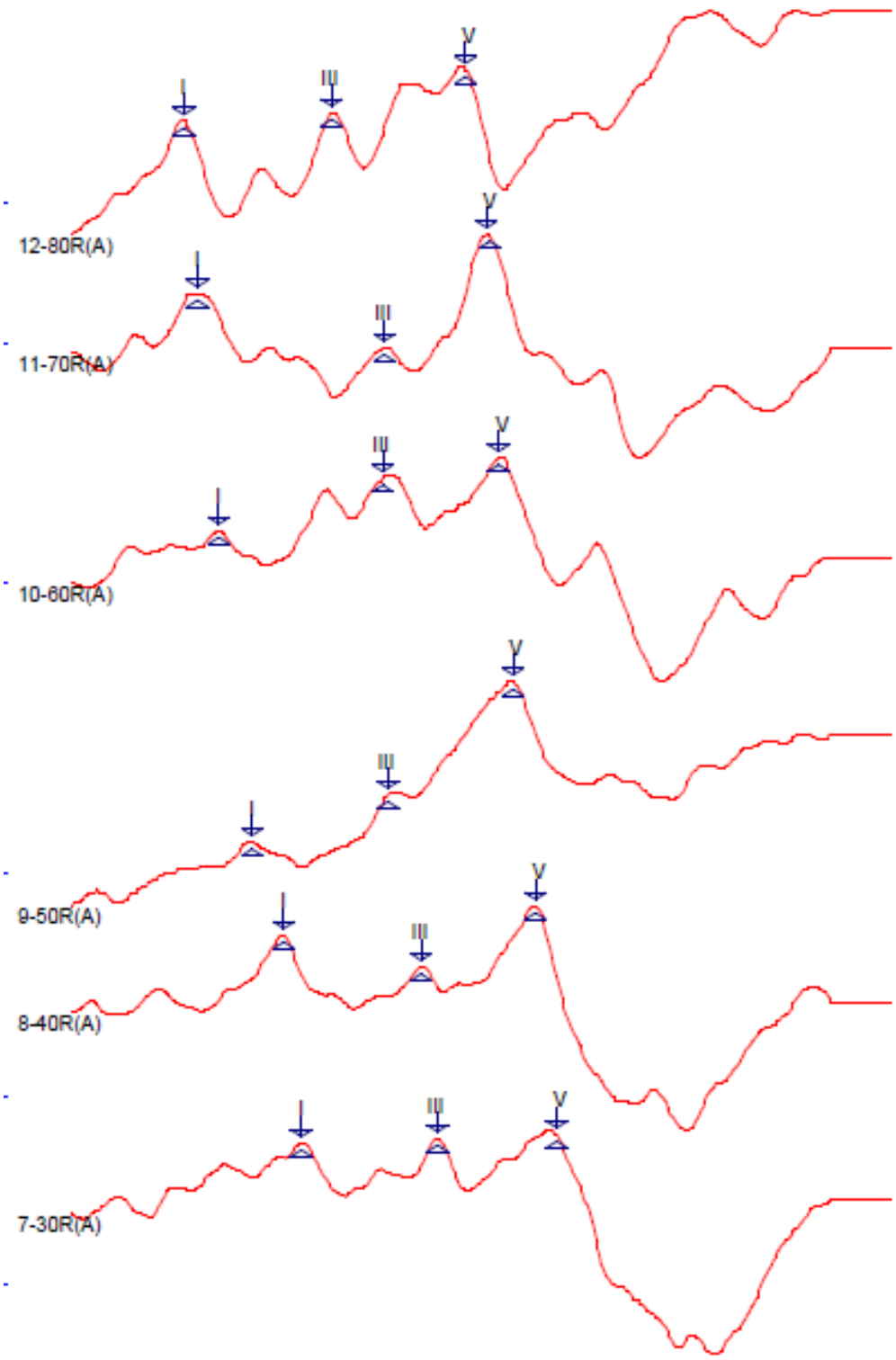
Dr. S. ANIM-SAMPONG
(Academic Coordinator)

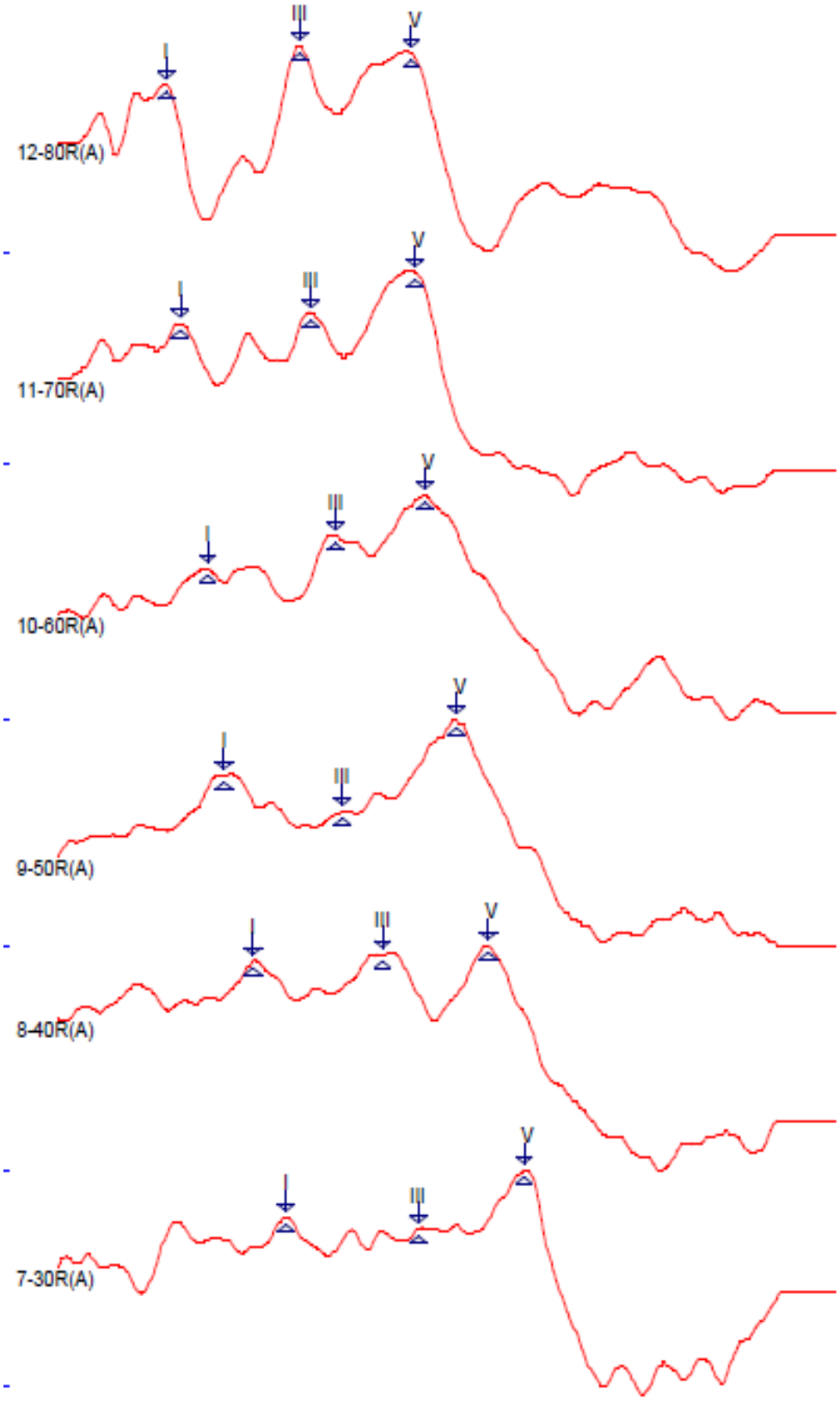
cc Dean (SAHS) Vice-Dean (SAHS) Dr. E.D. Kitcher (ENT, KBTH)

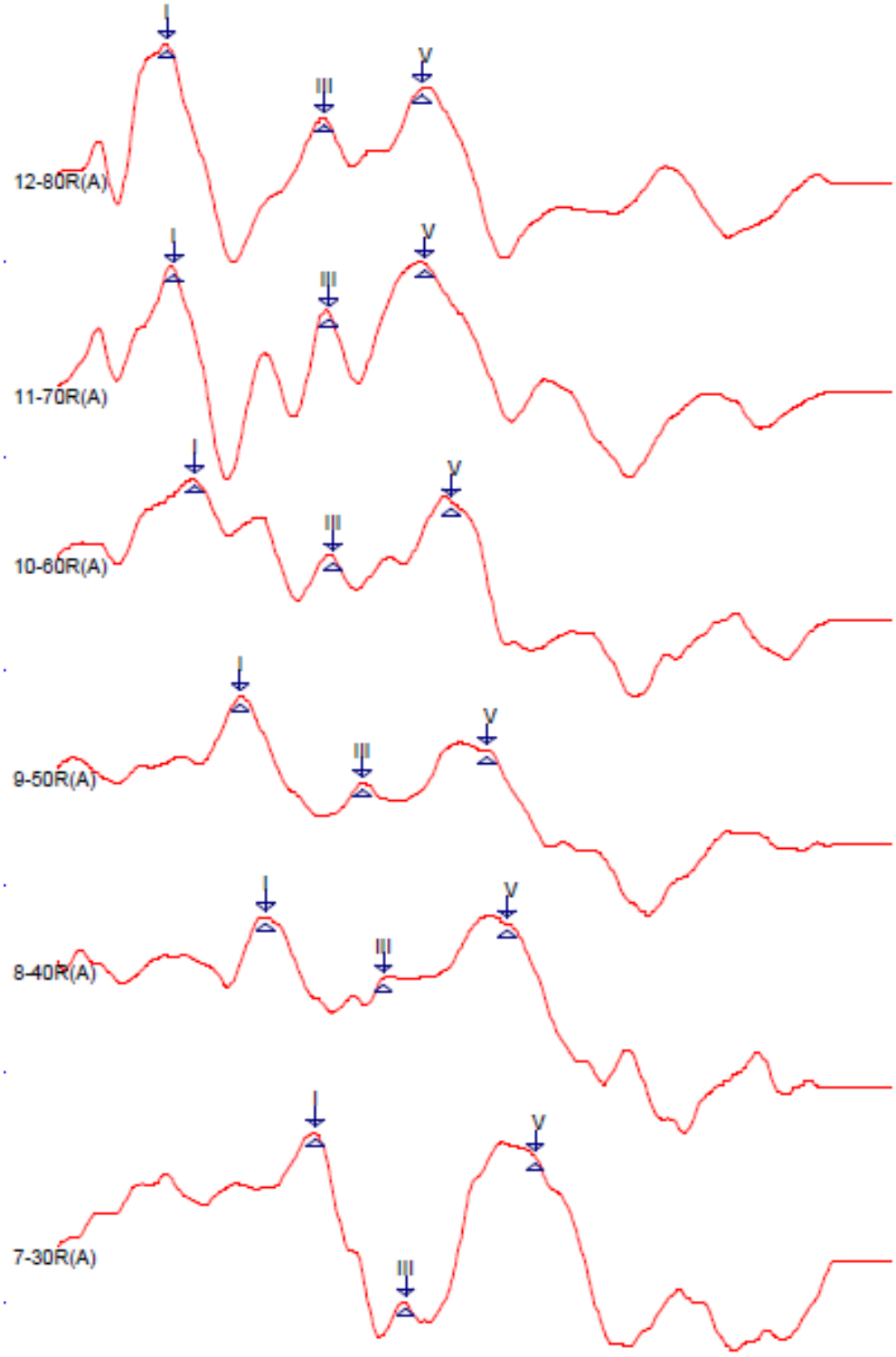
APPENDIX V

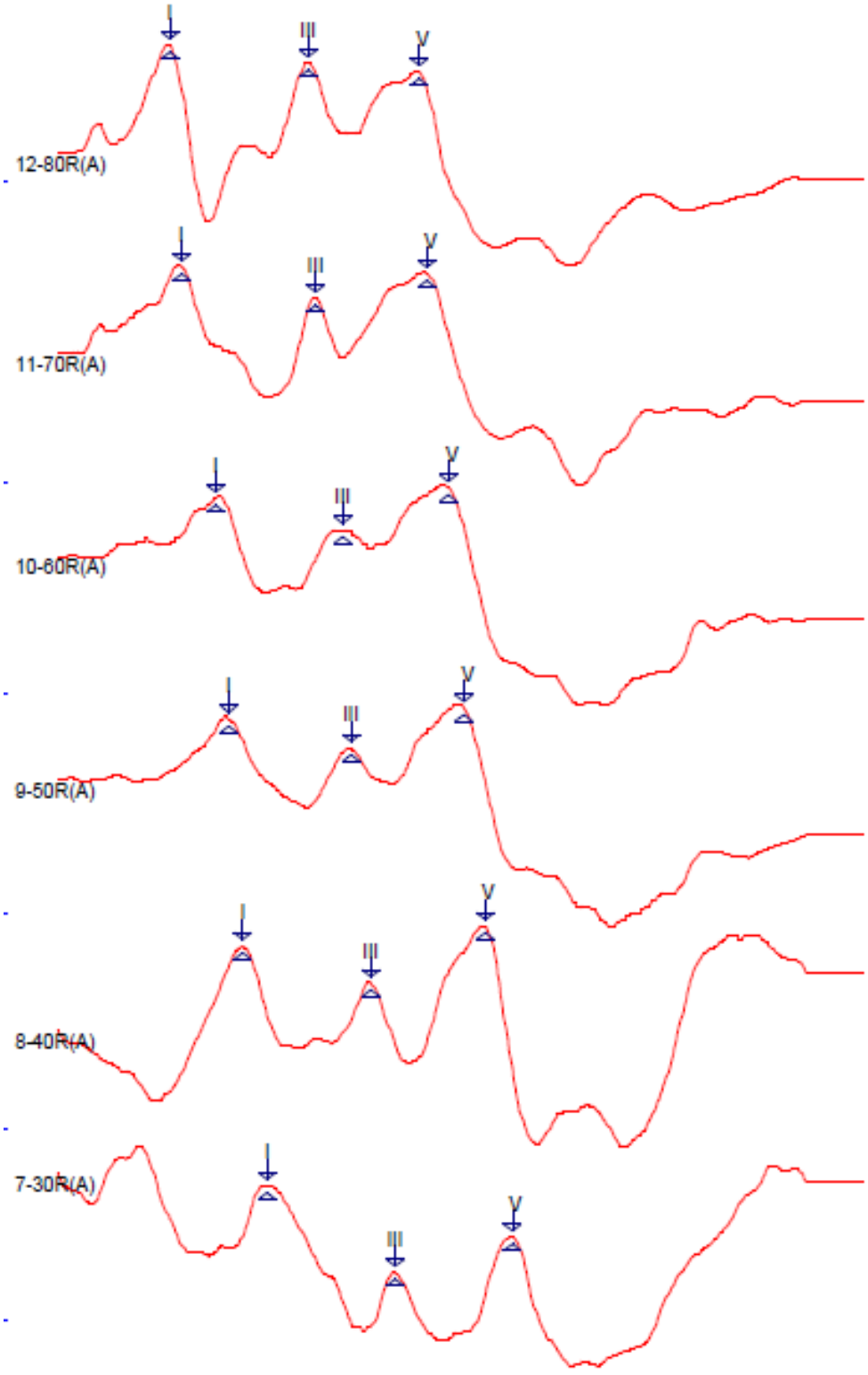
RECORDED WAVEFORMS

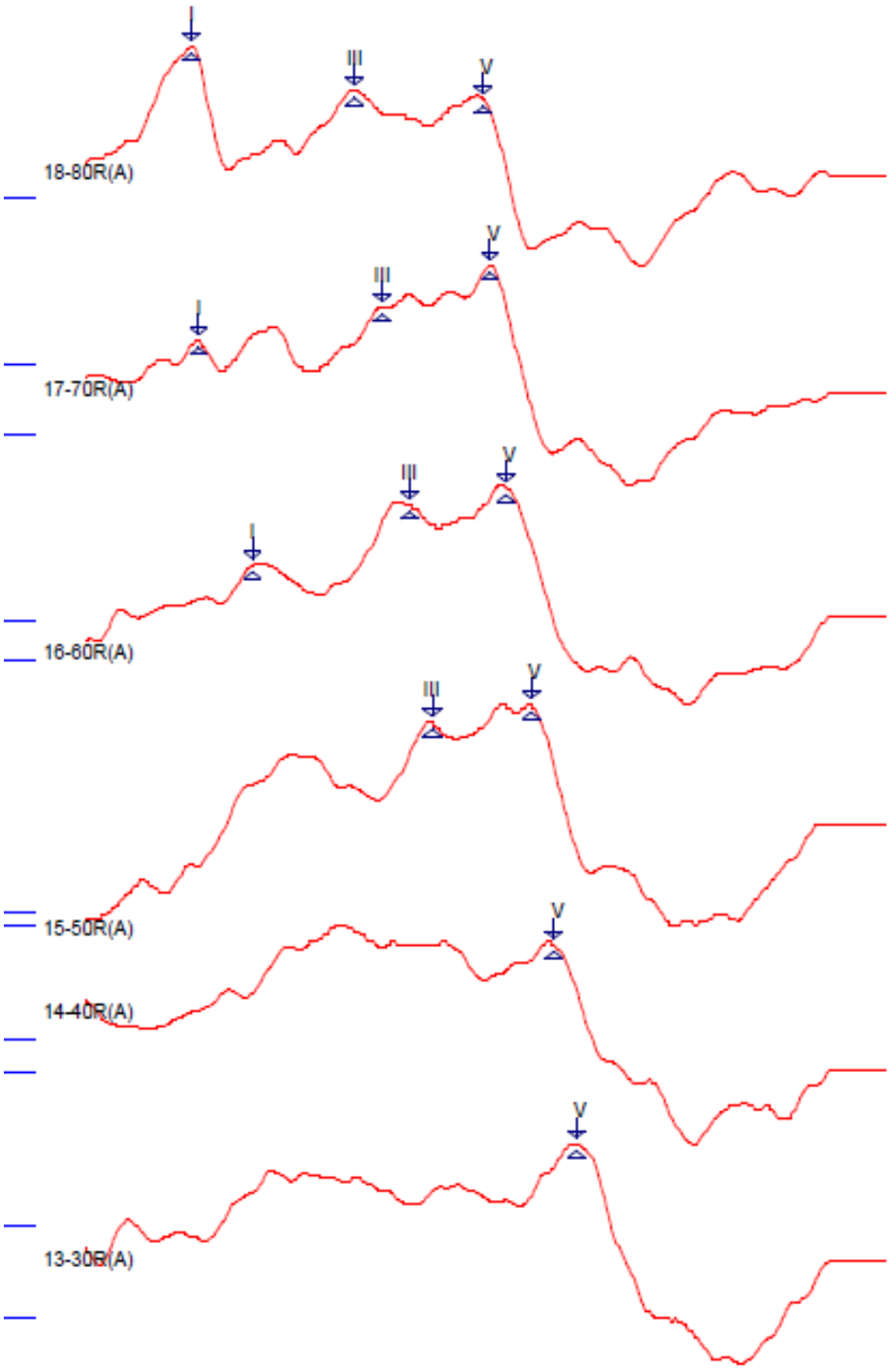


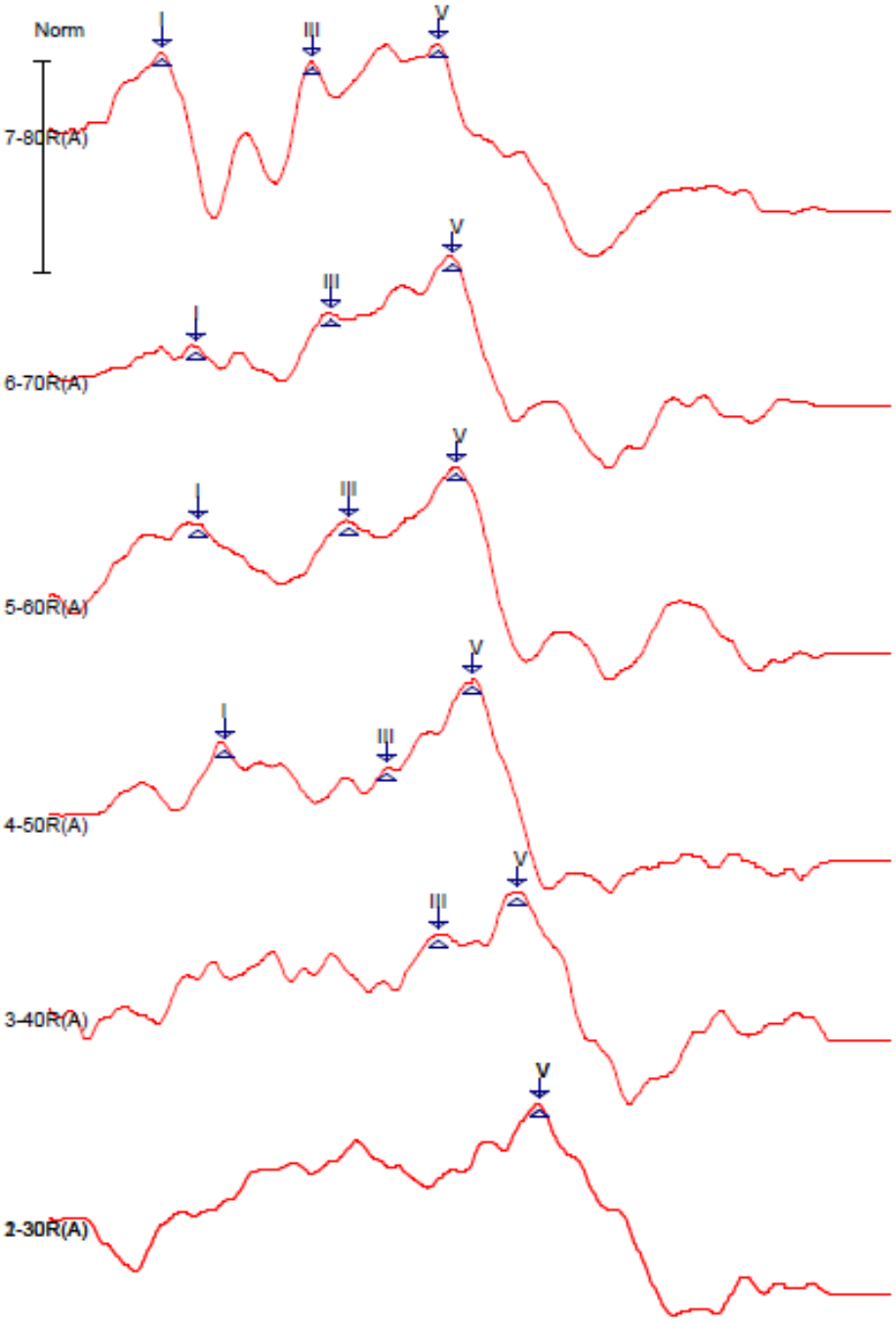


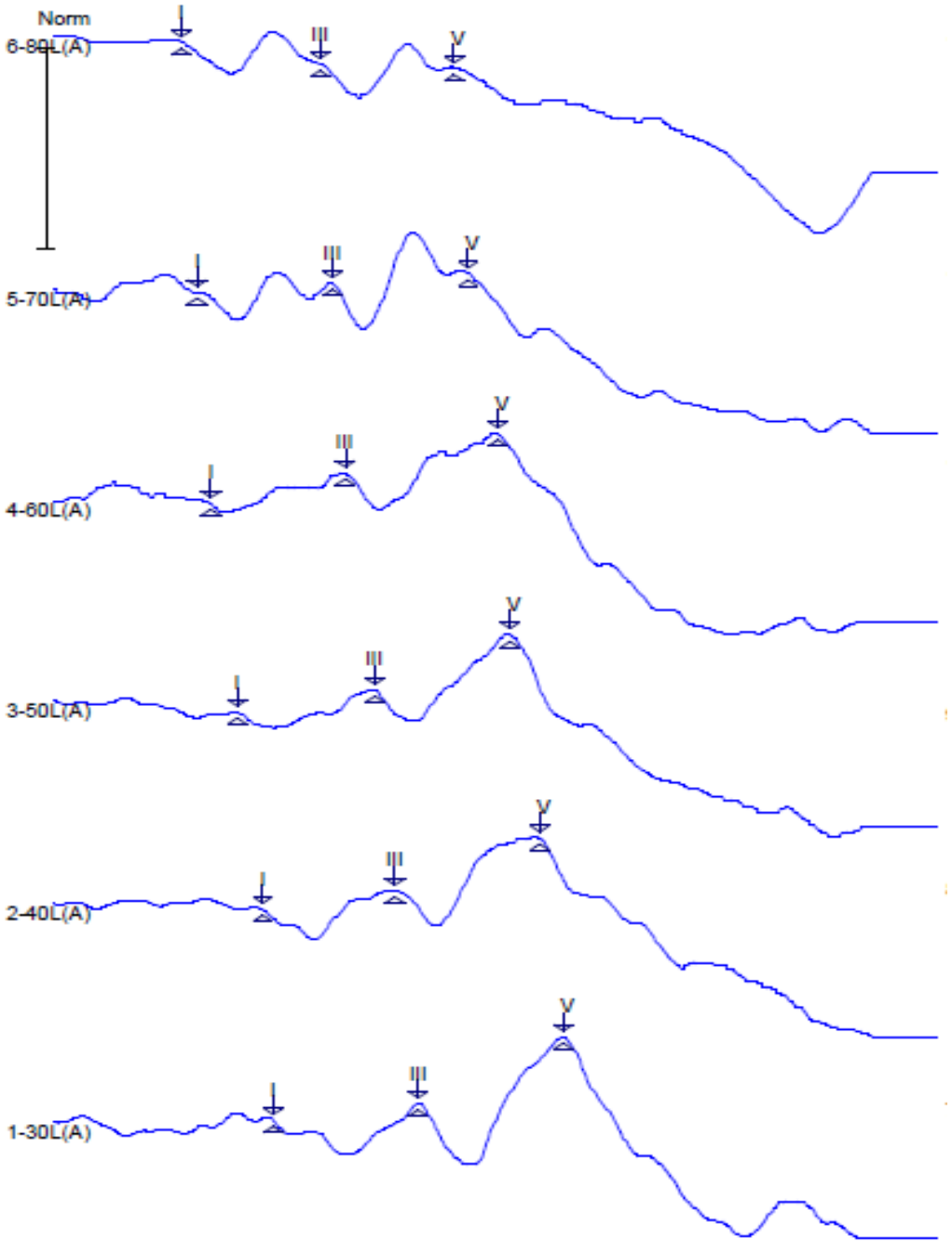


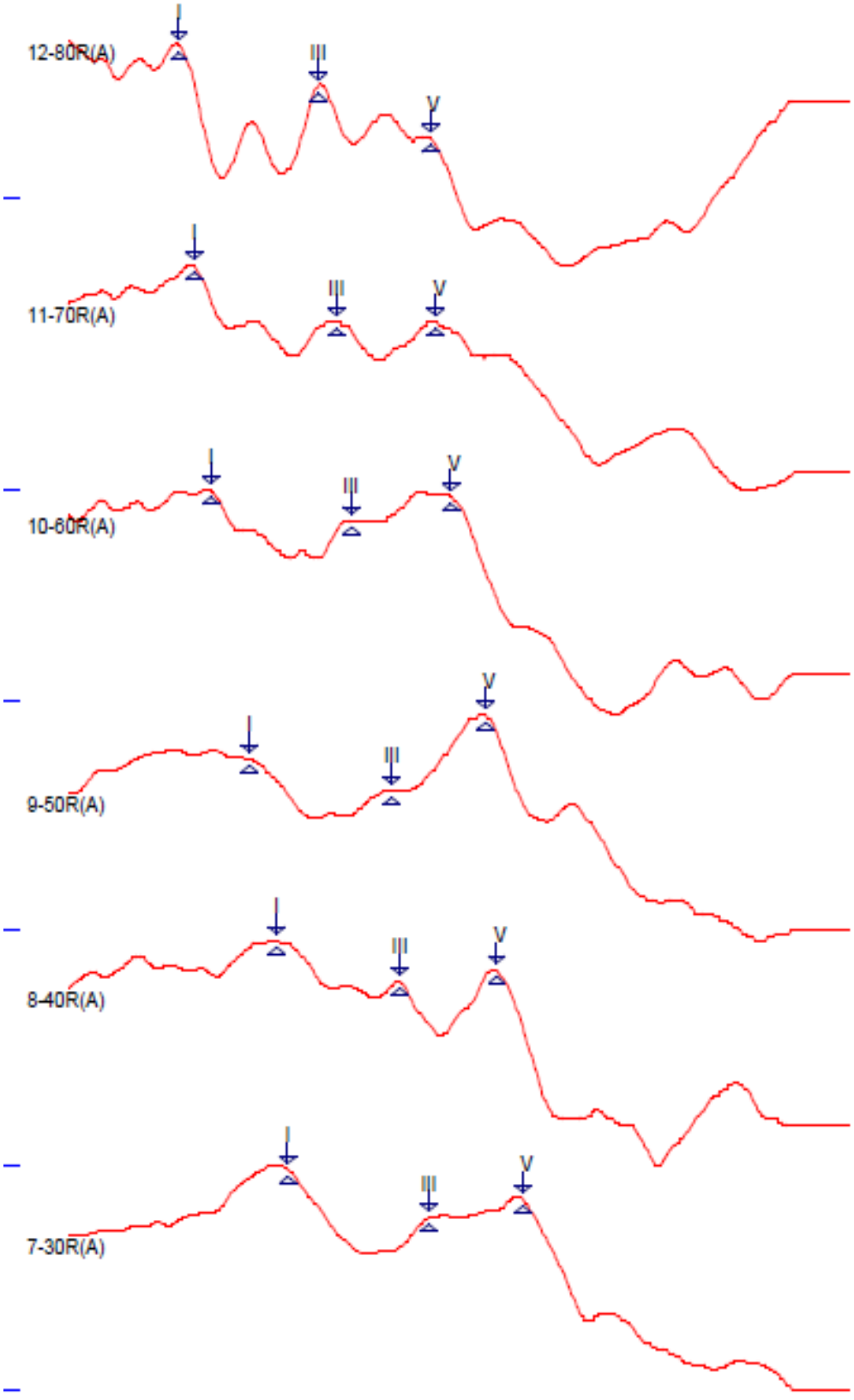


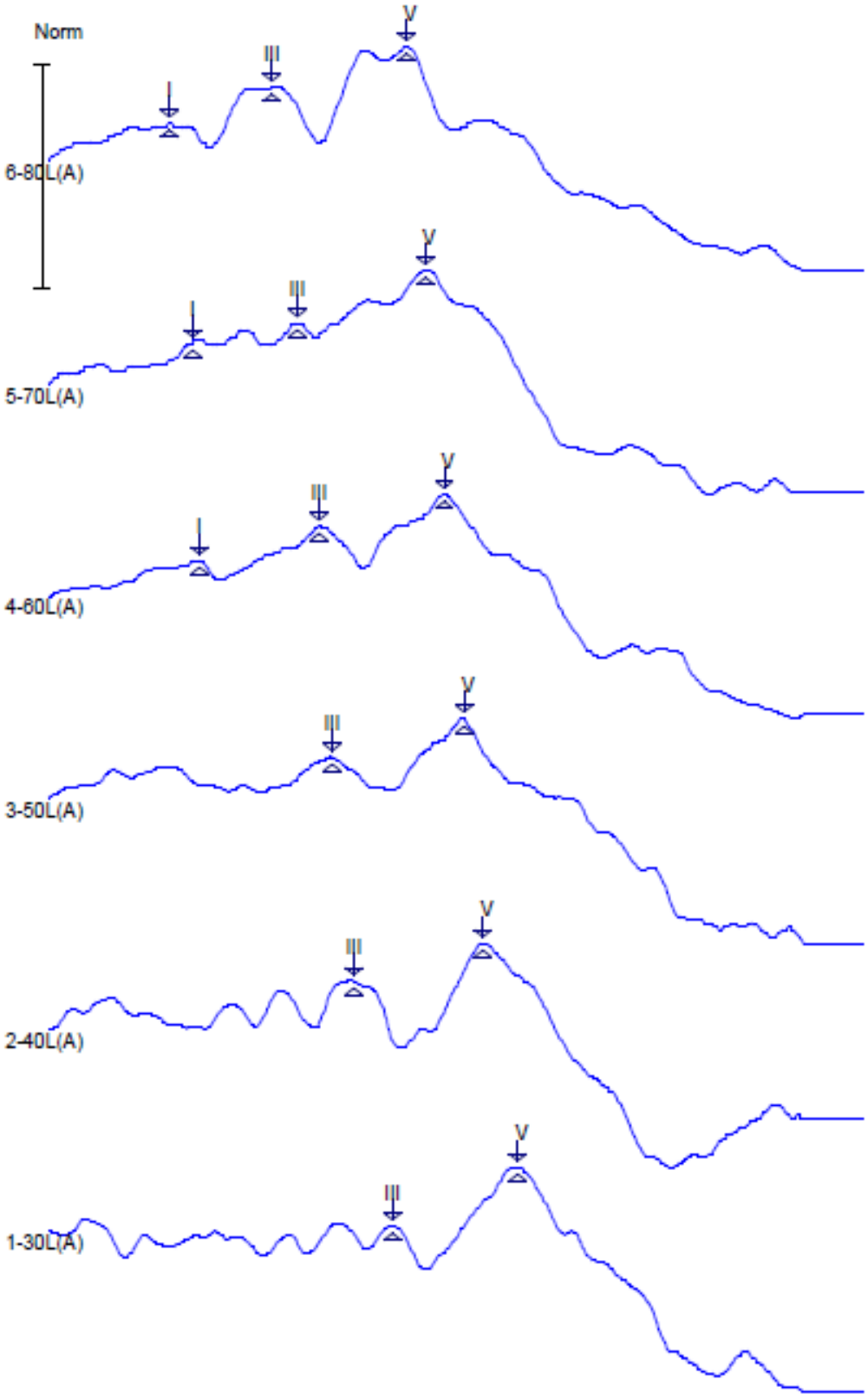


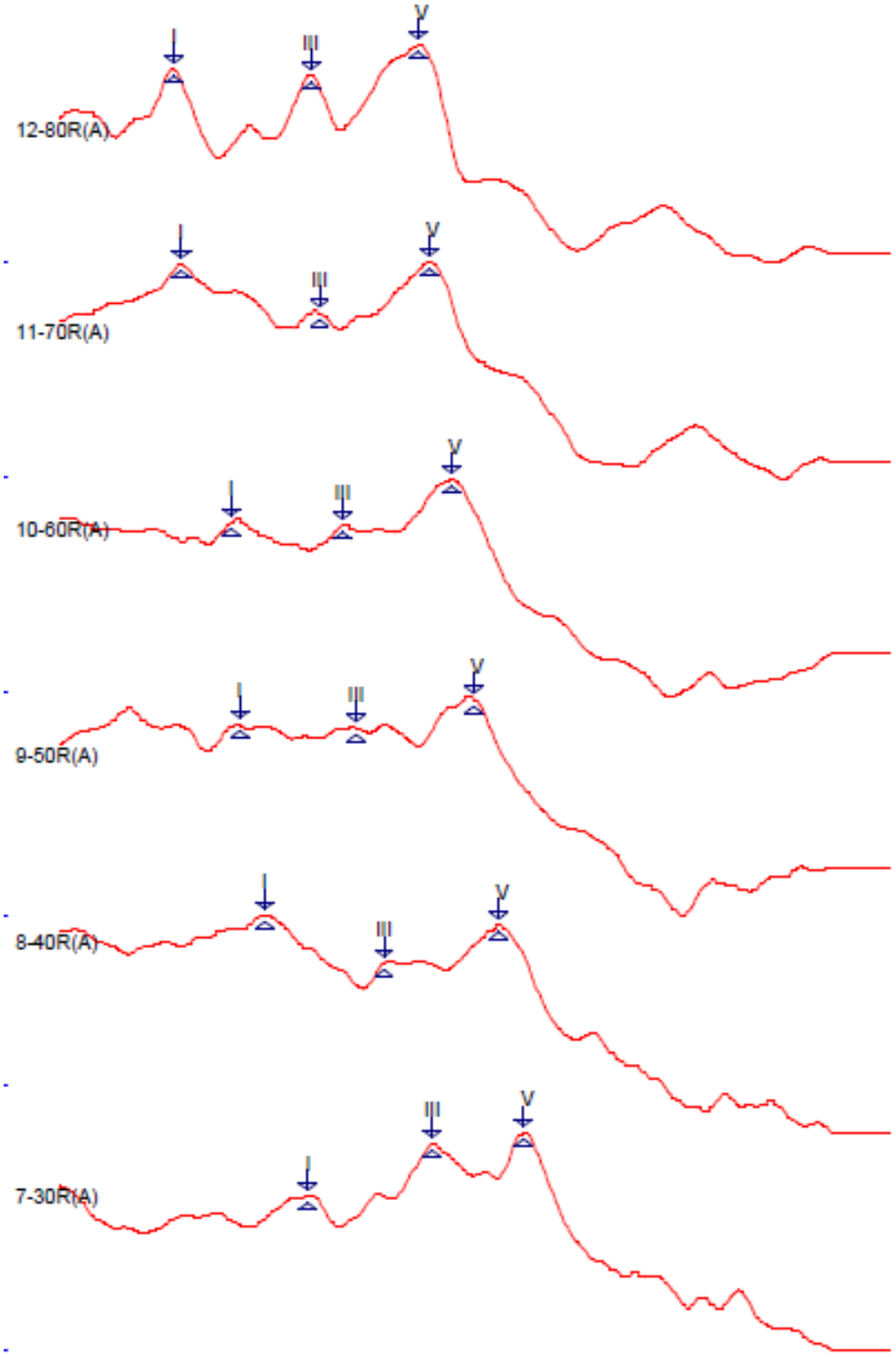


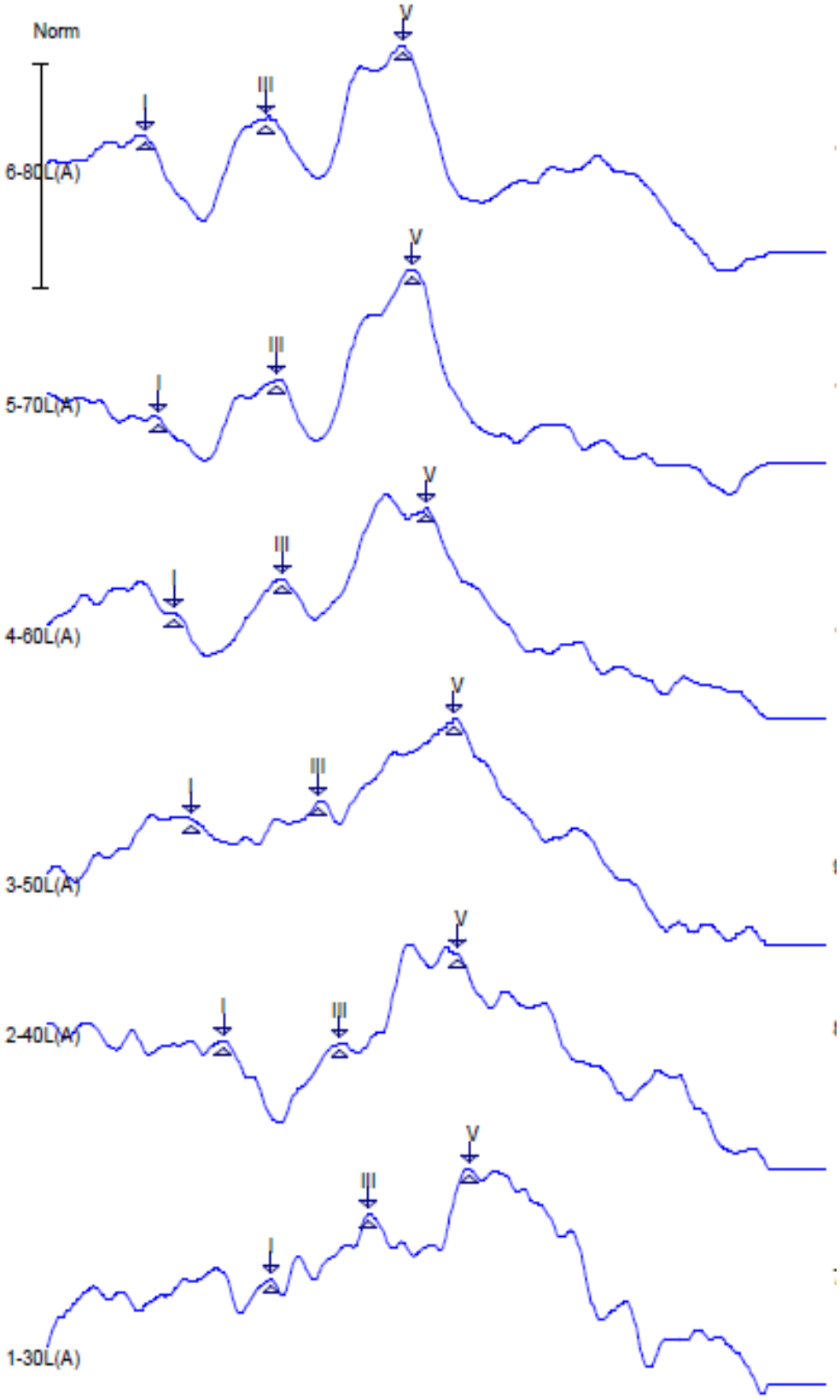


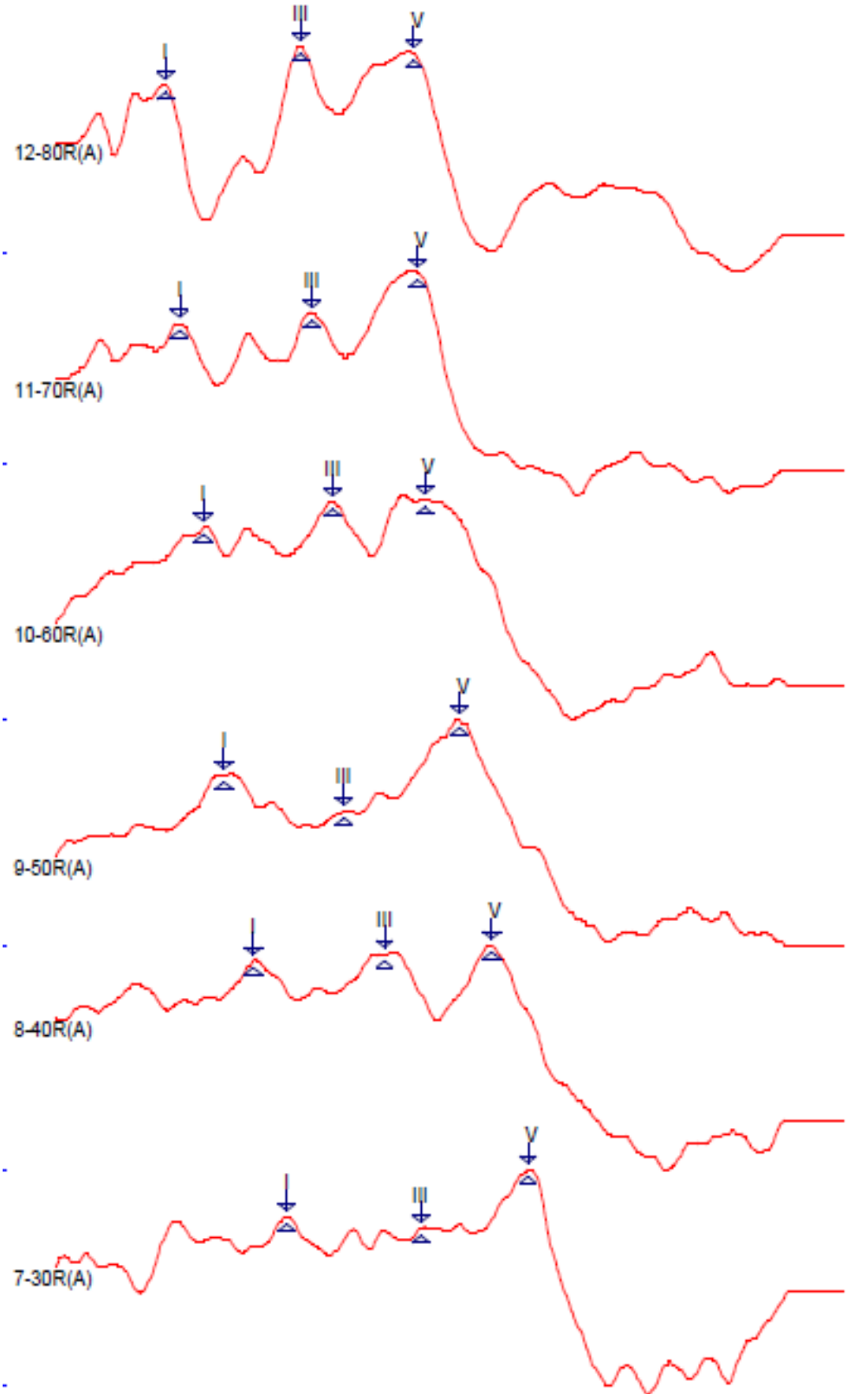


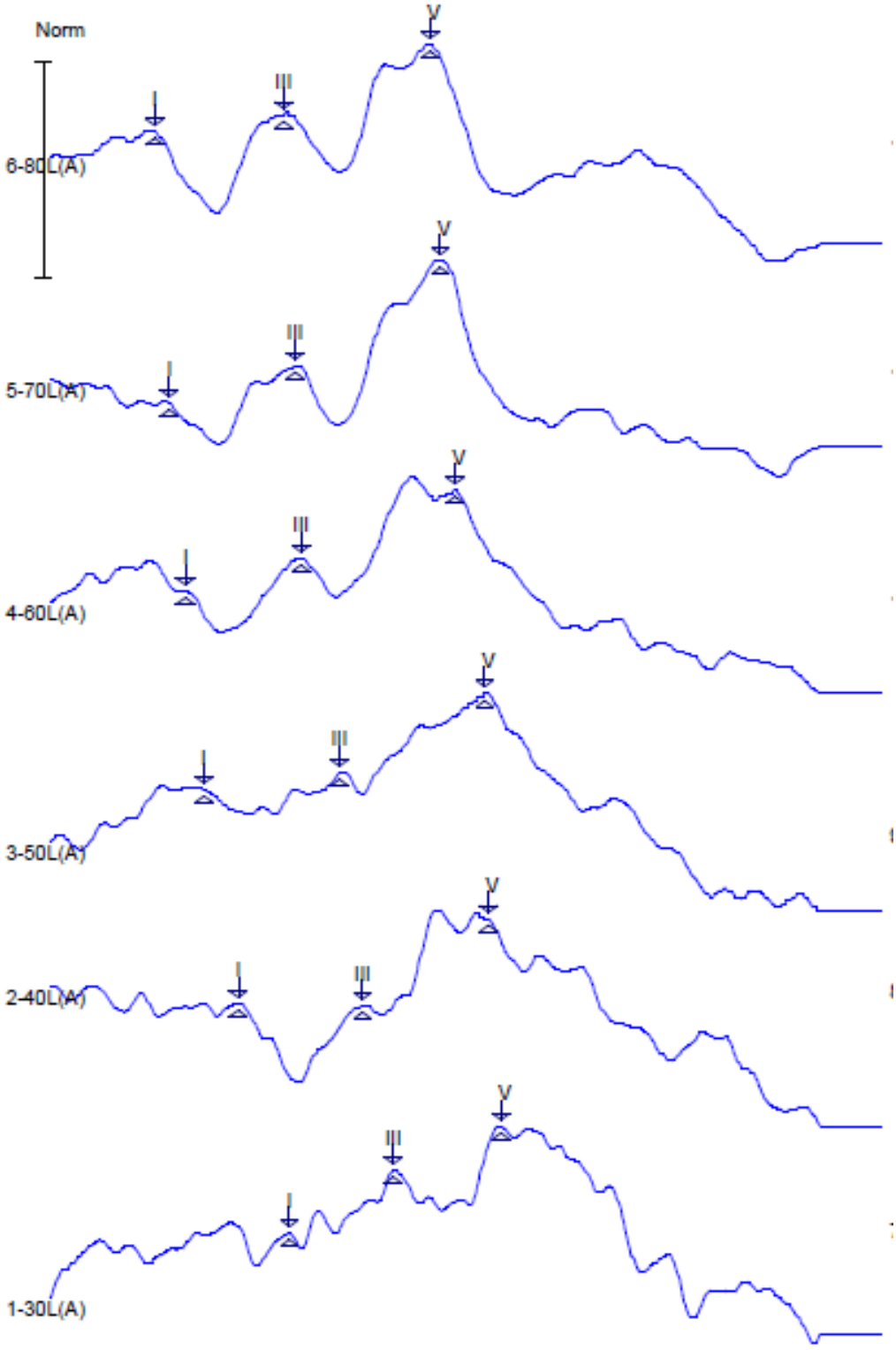


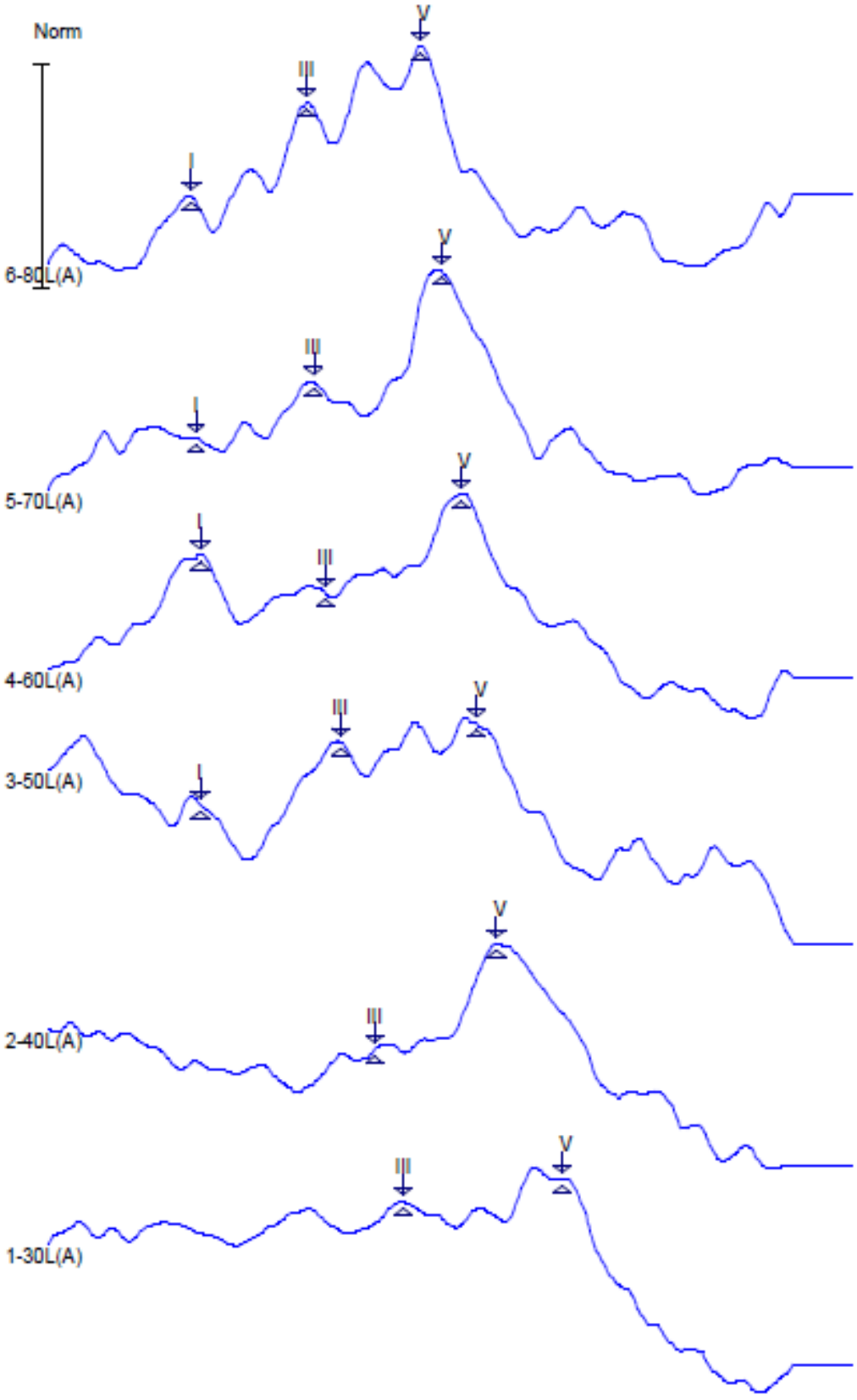


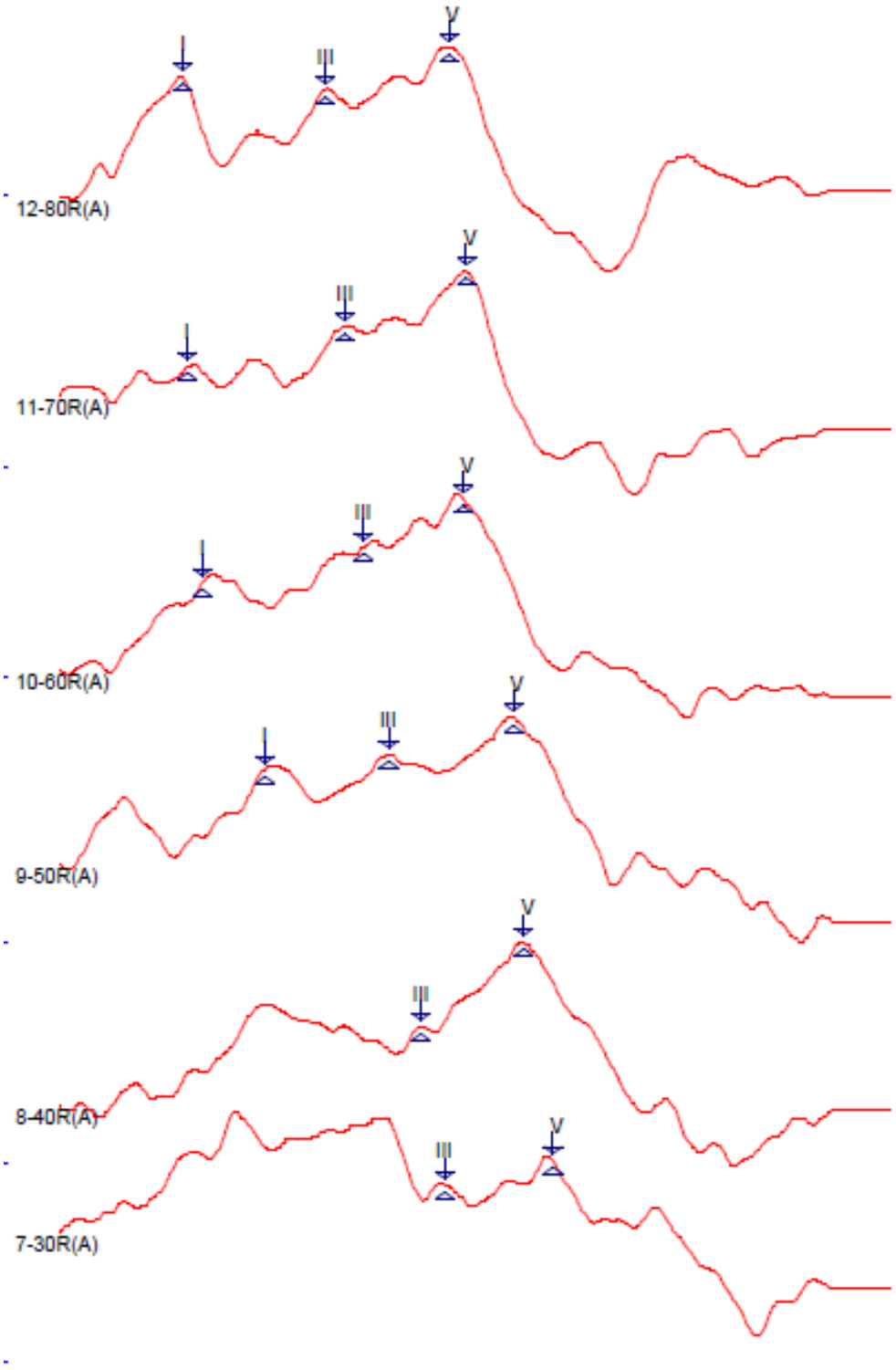


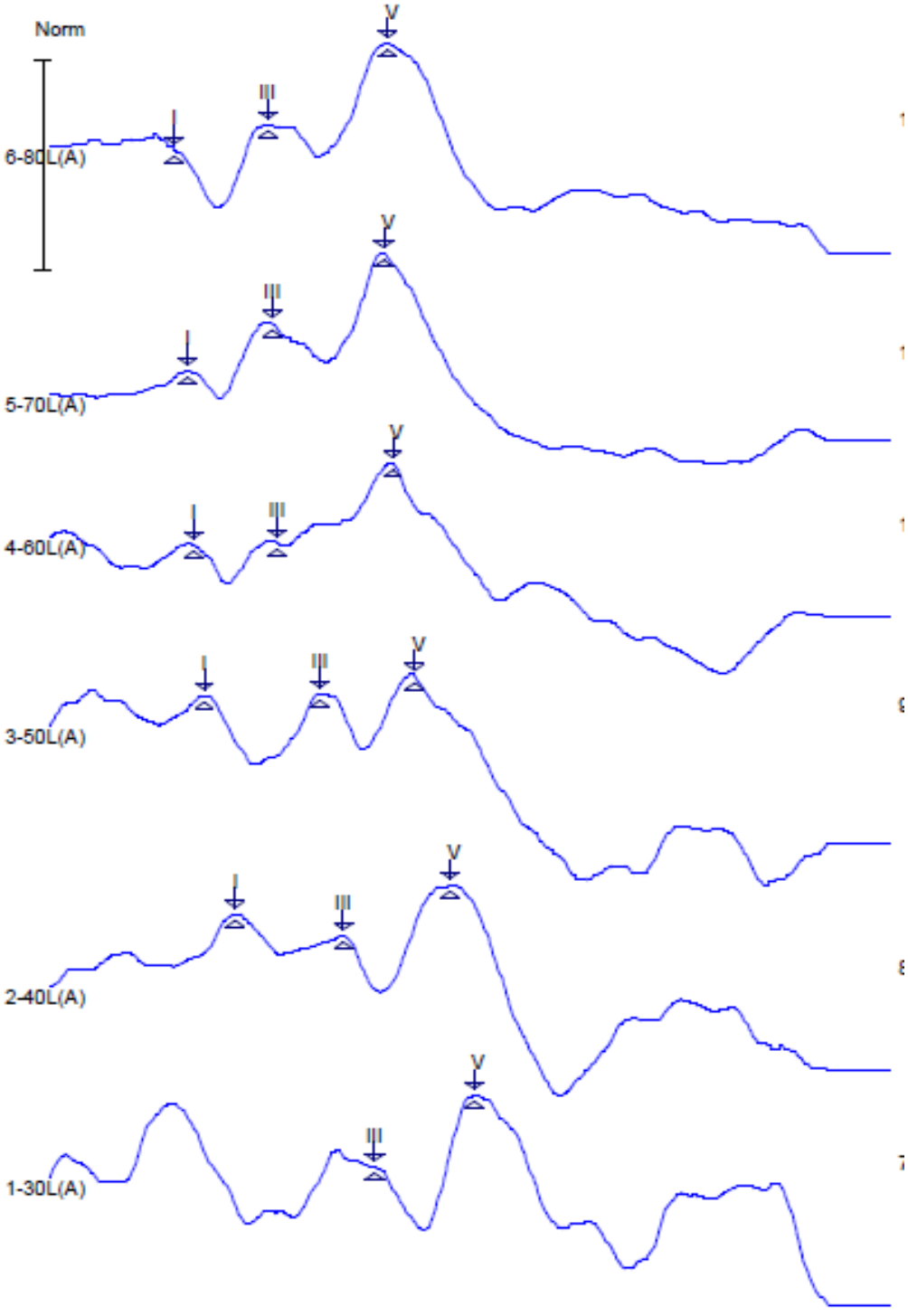


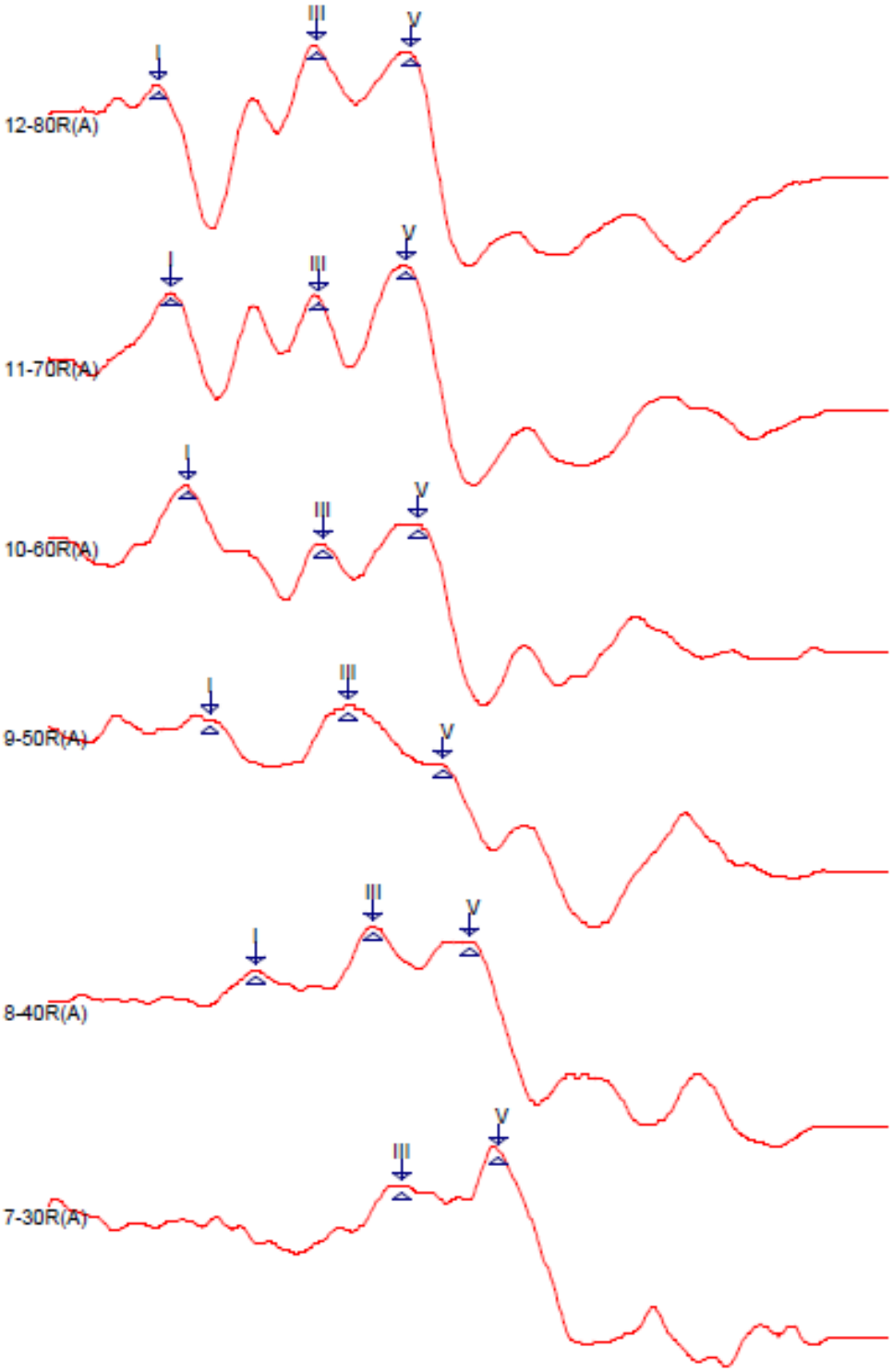


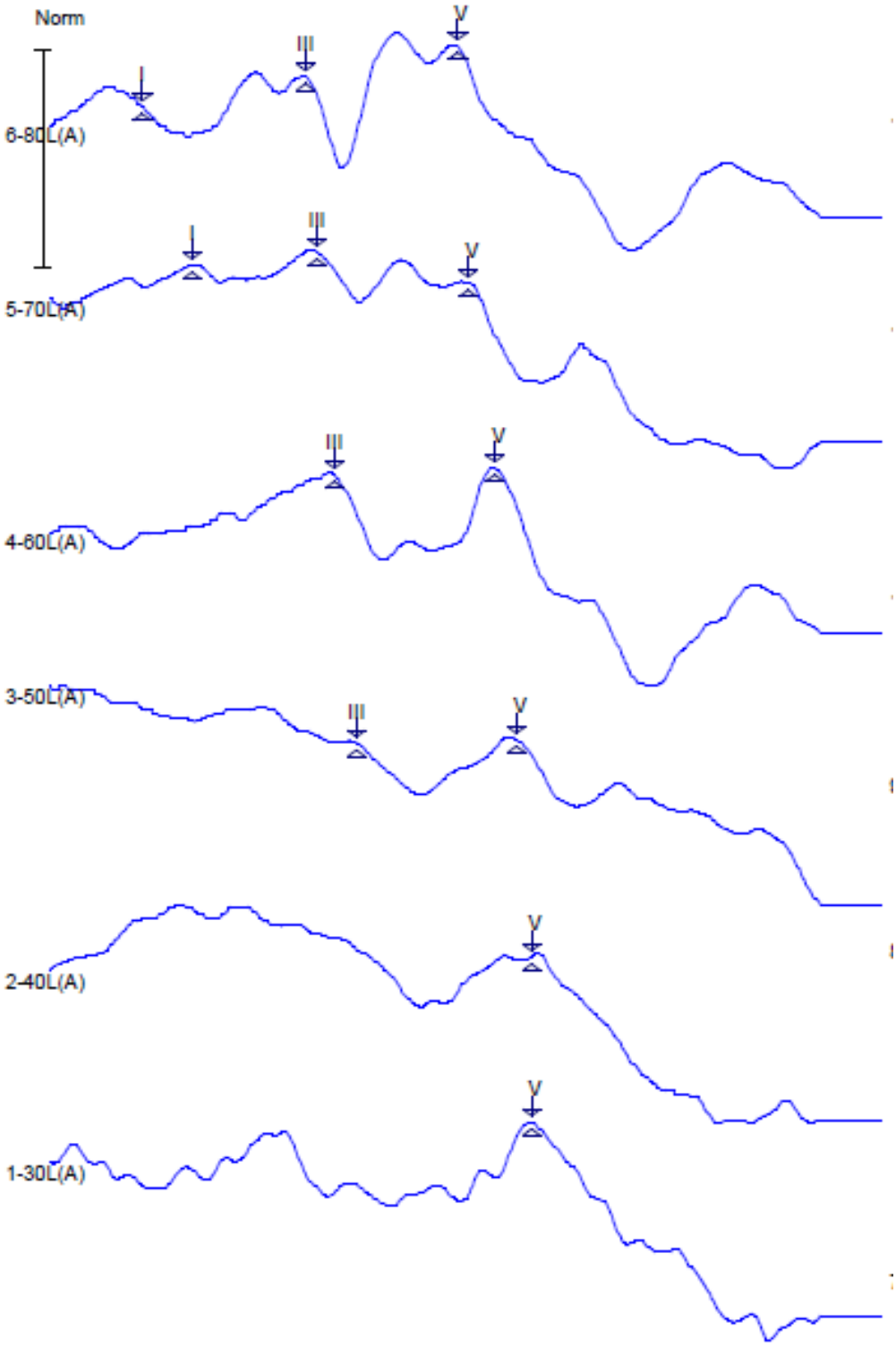


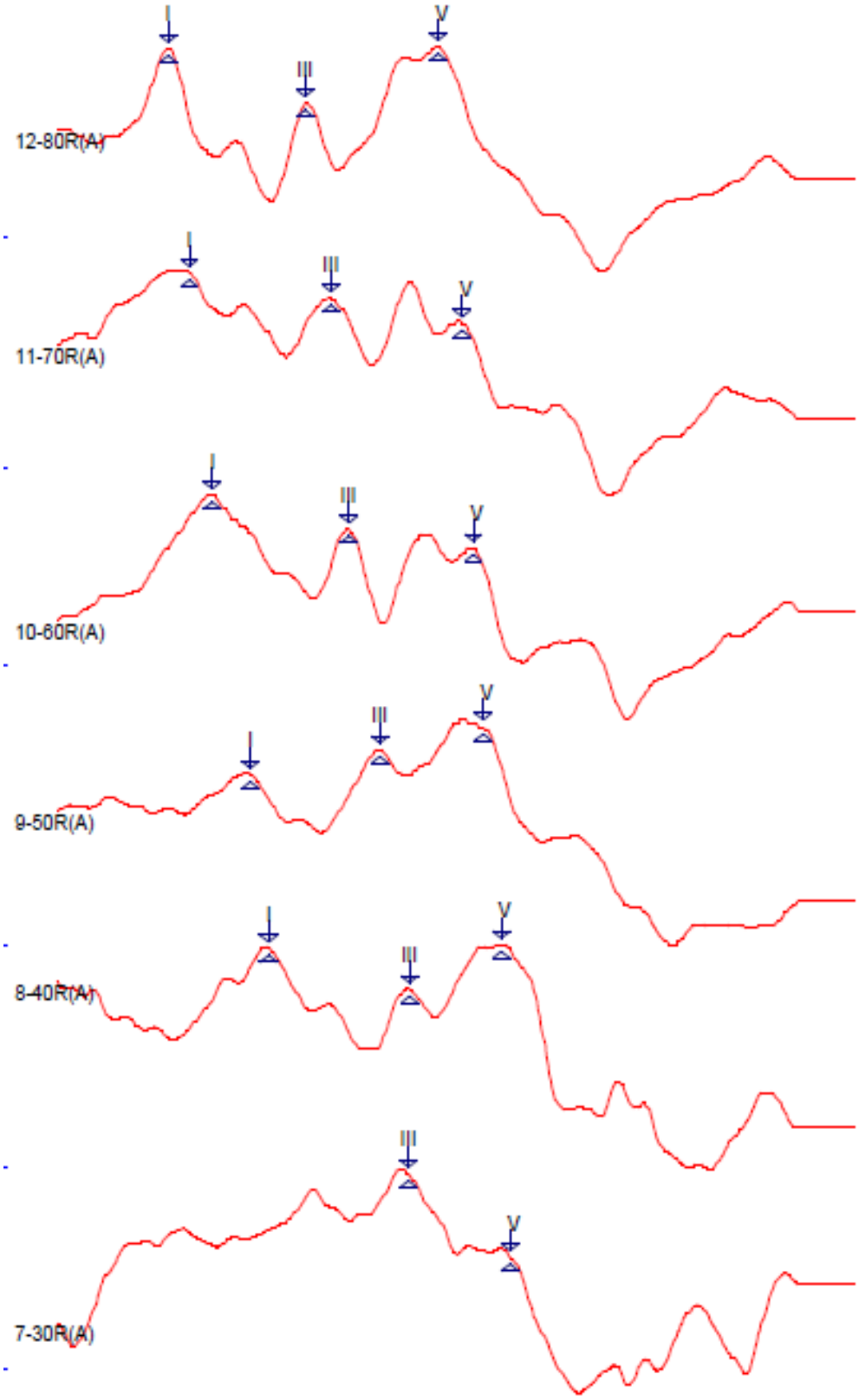


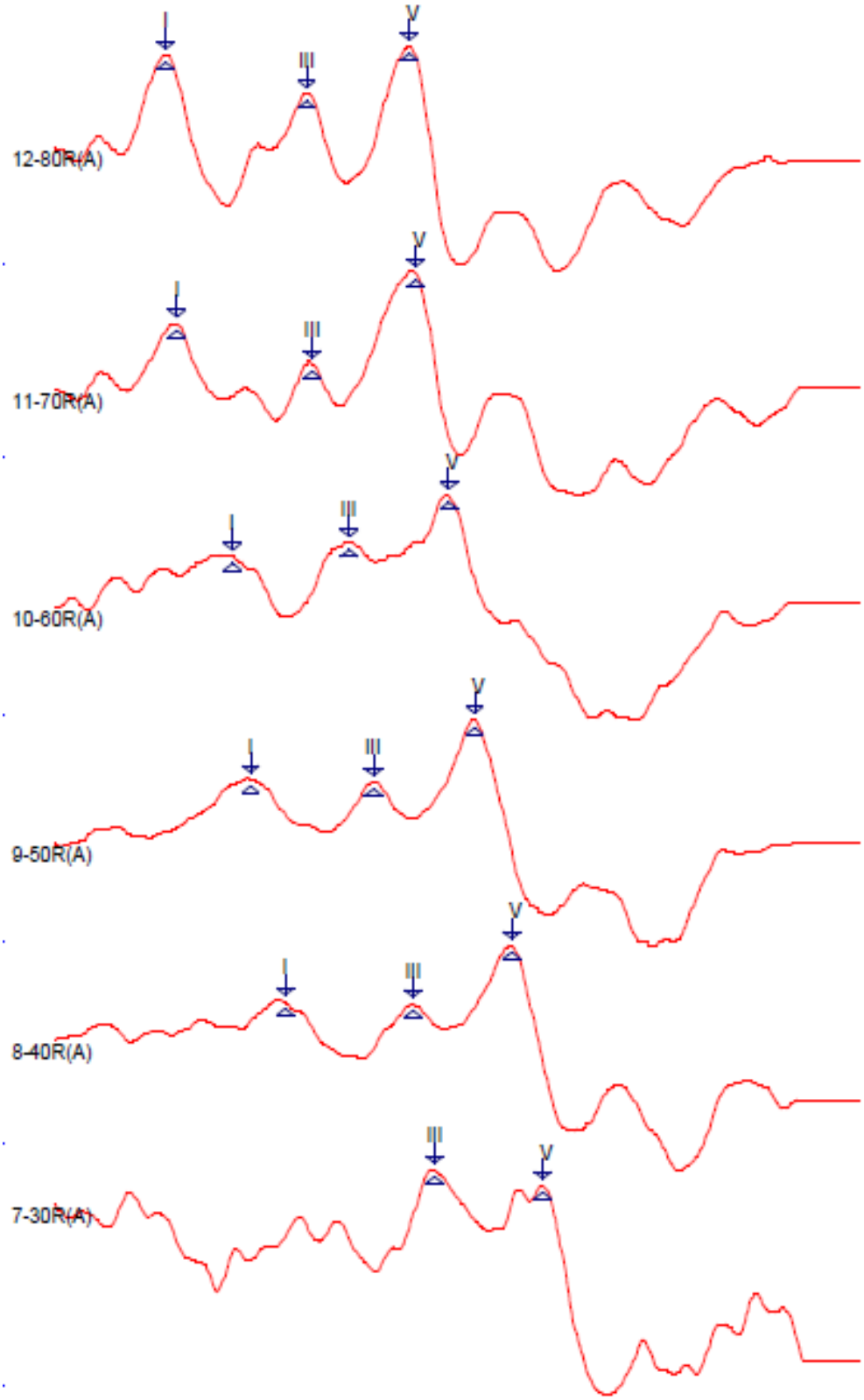


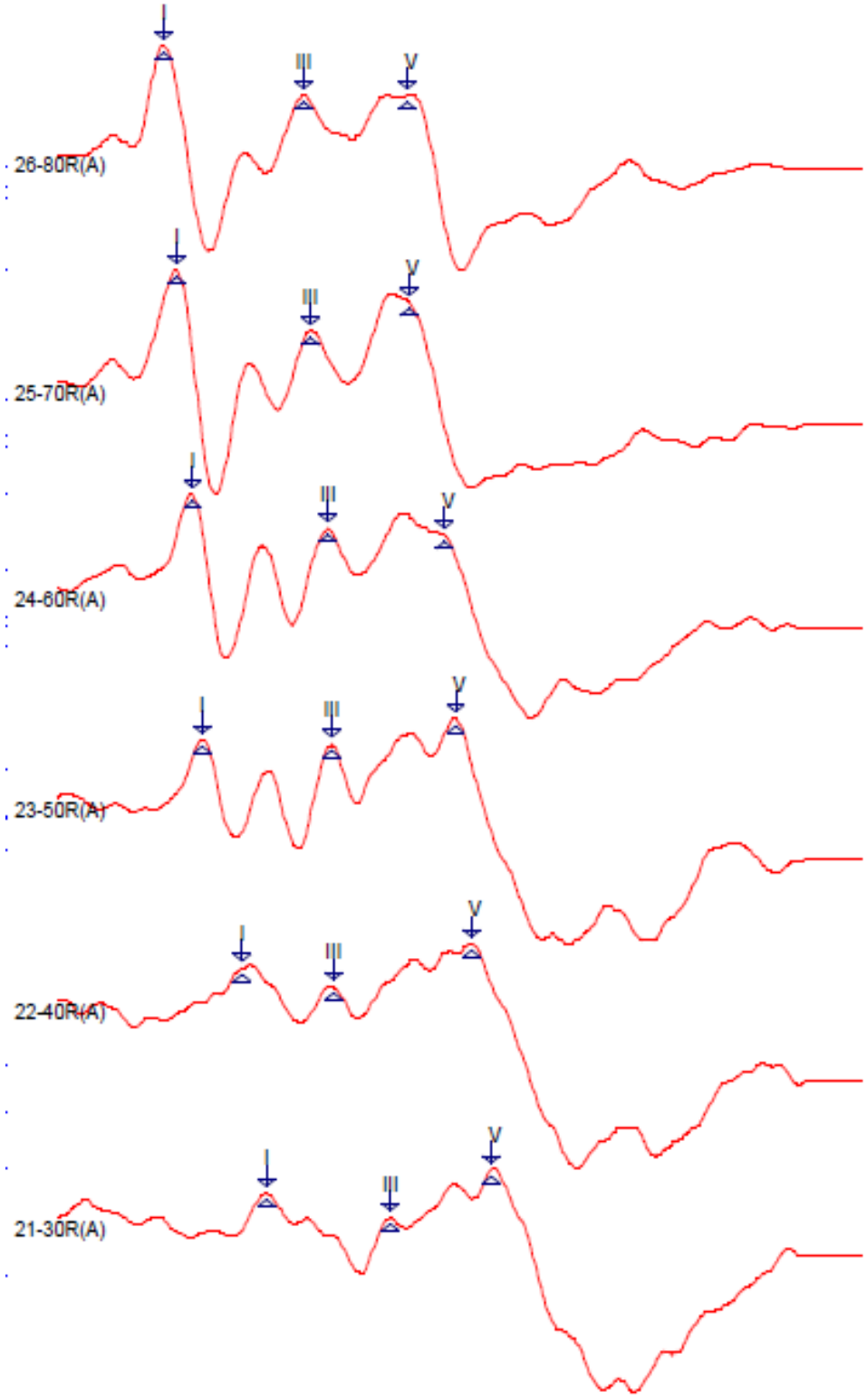


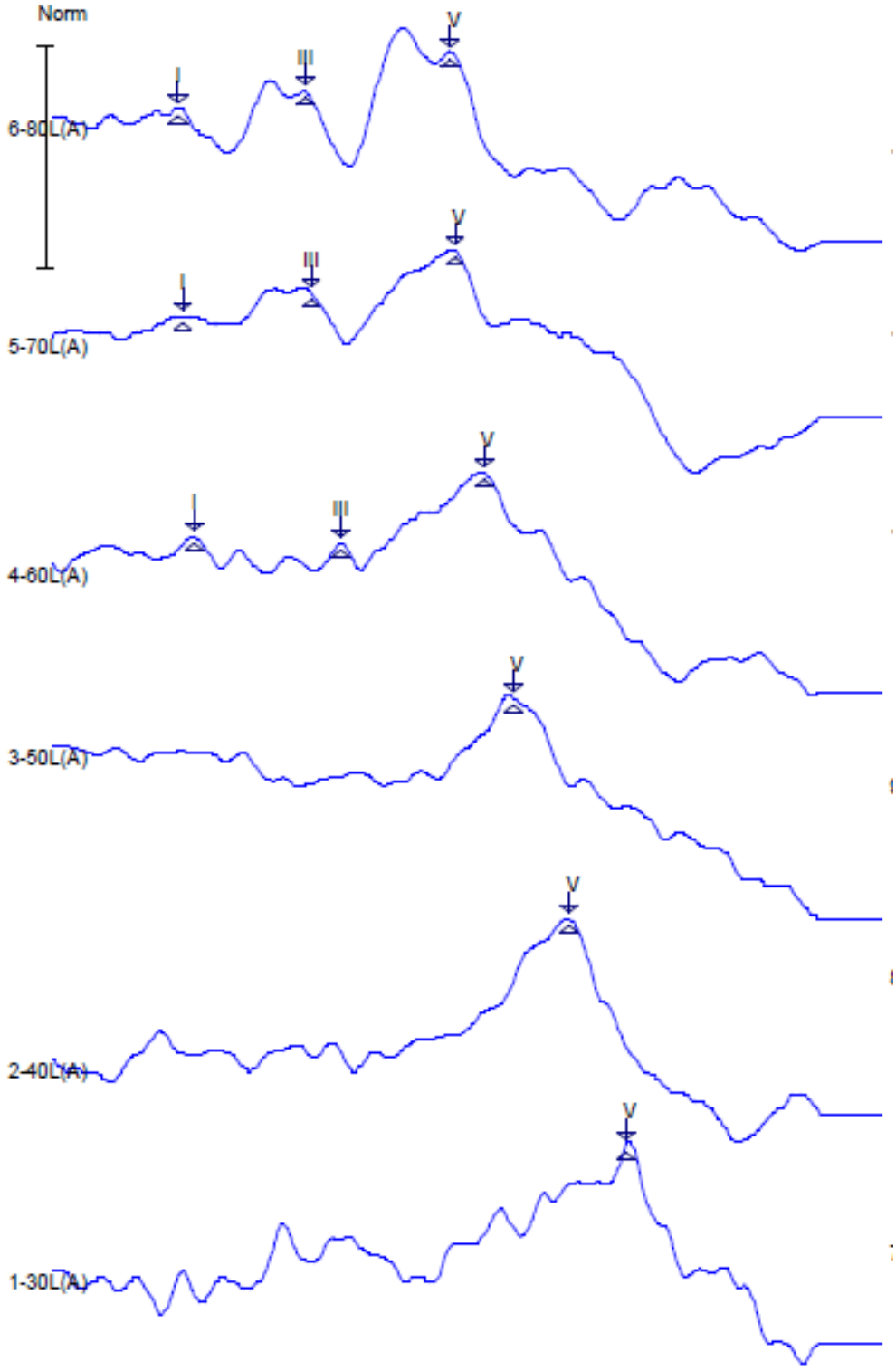


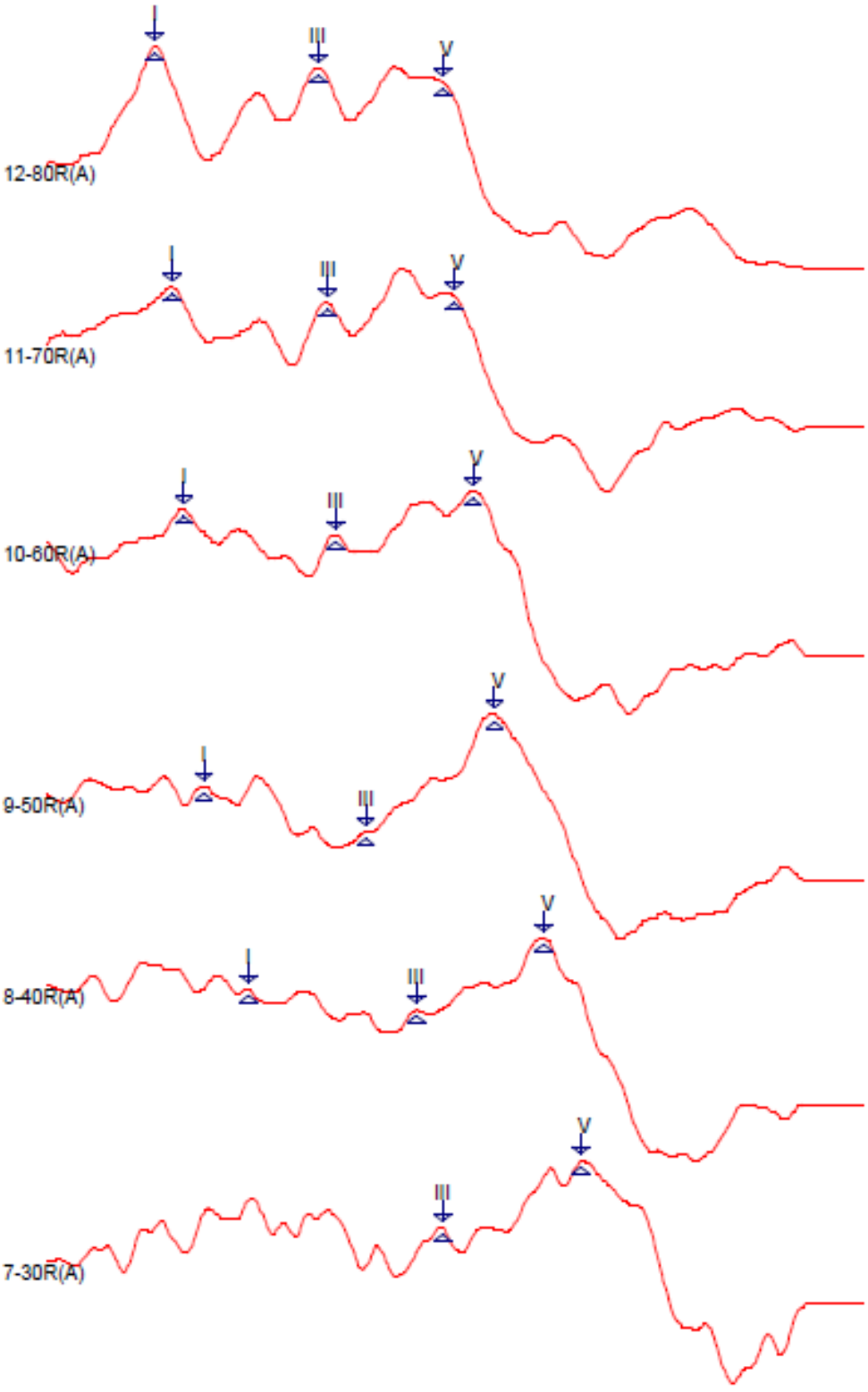


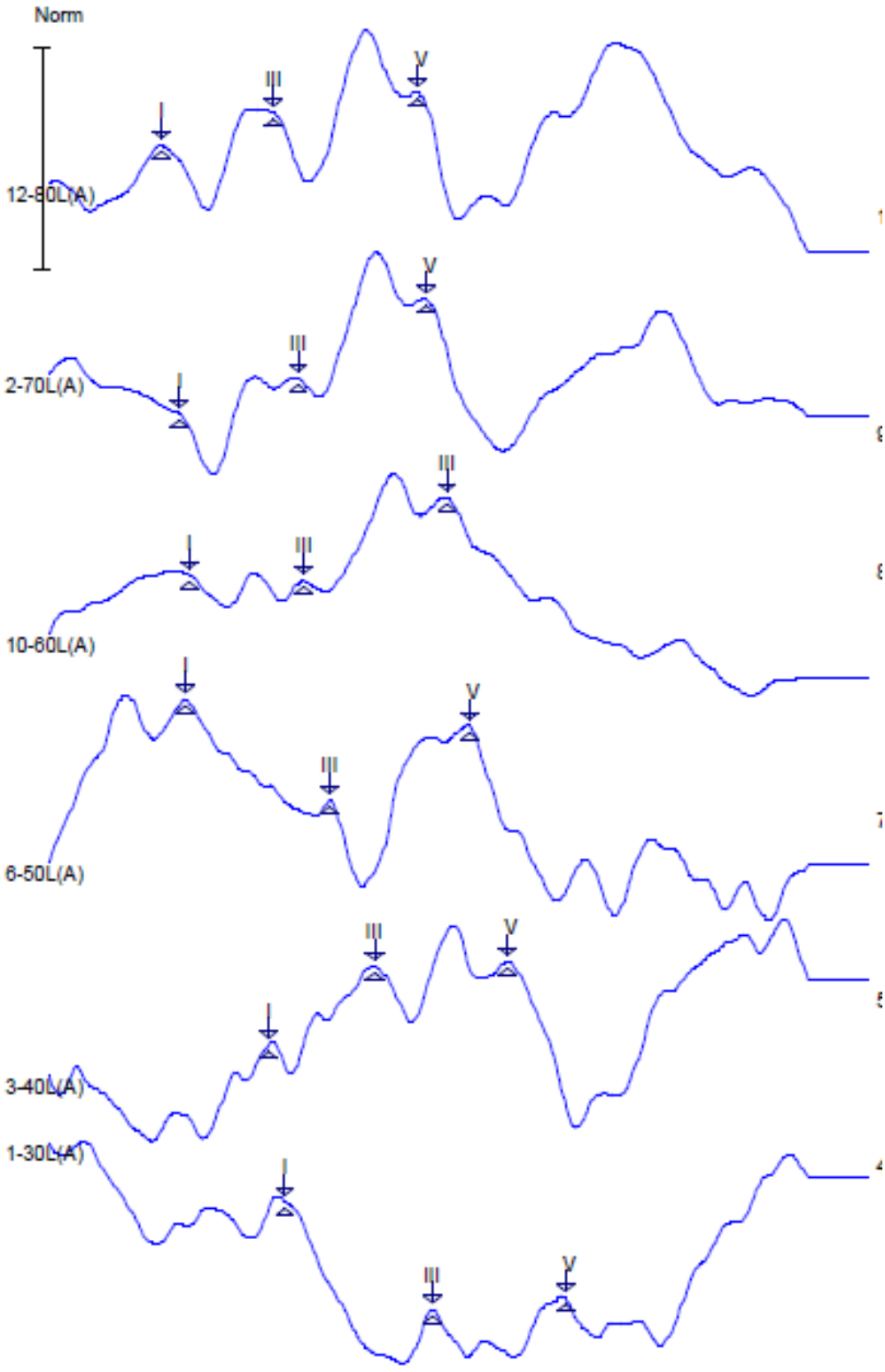


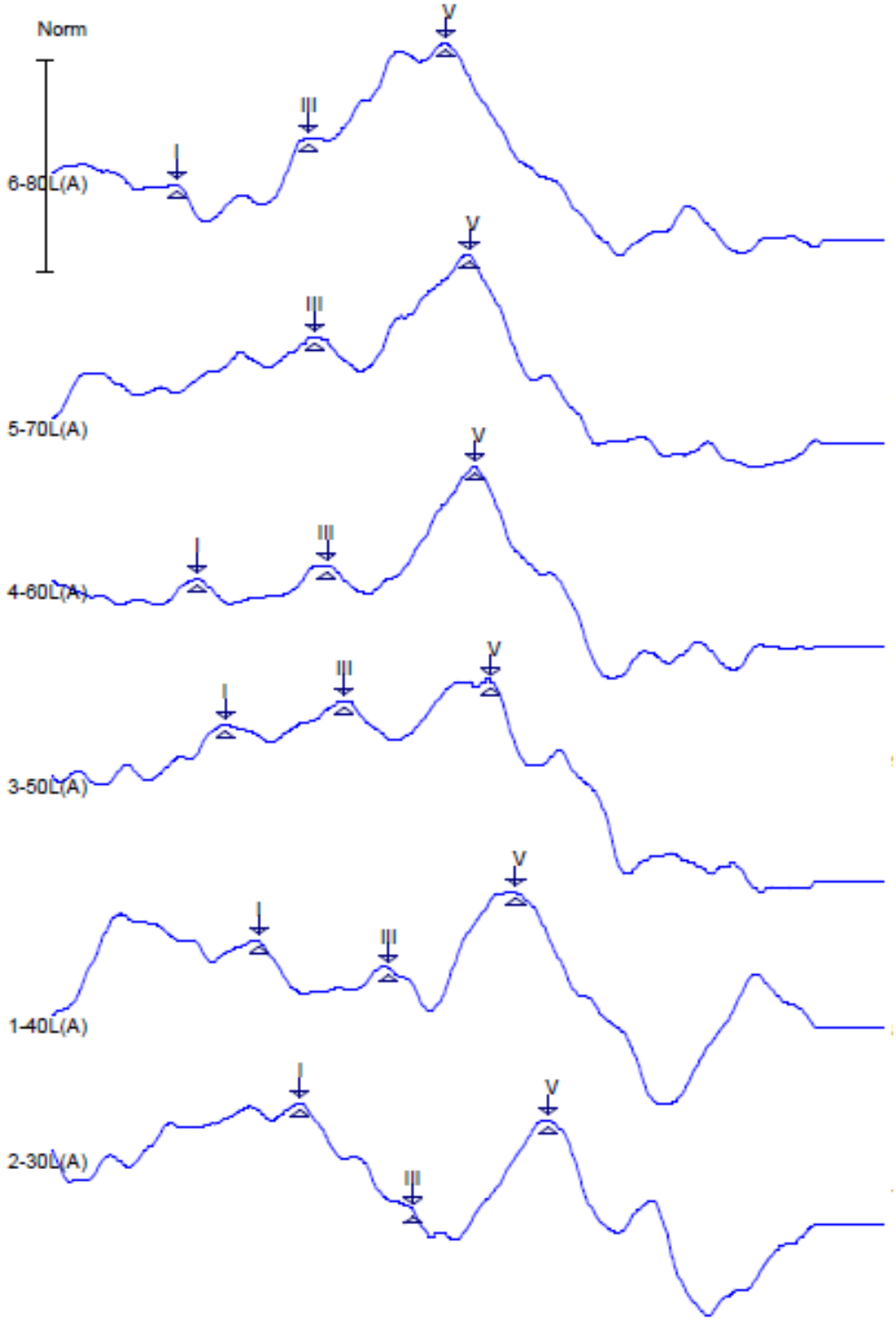


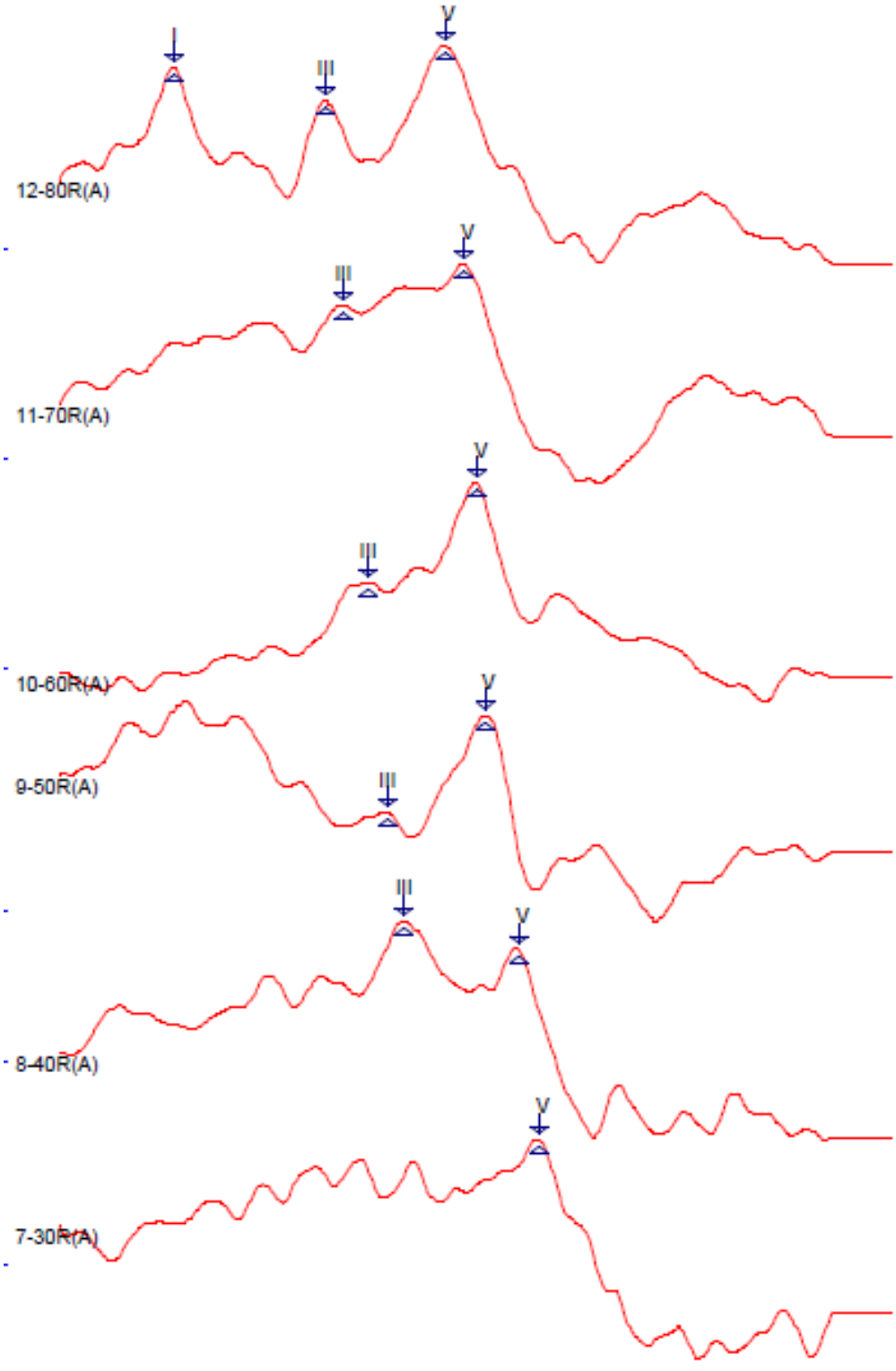


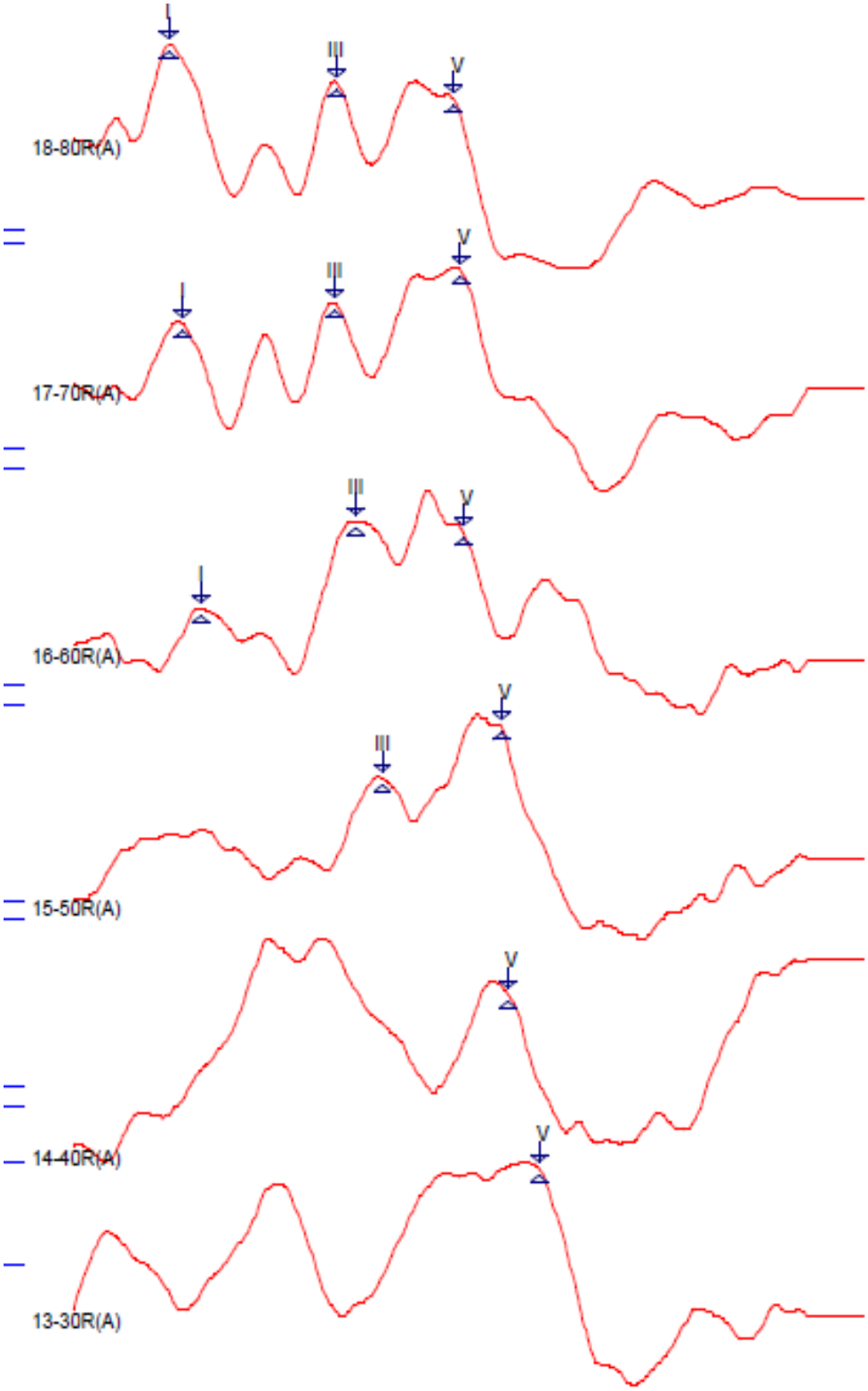


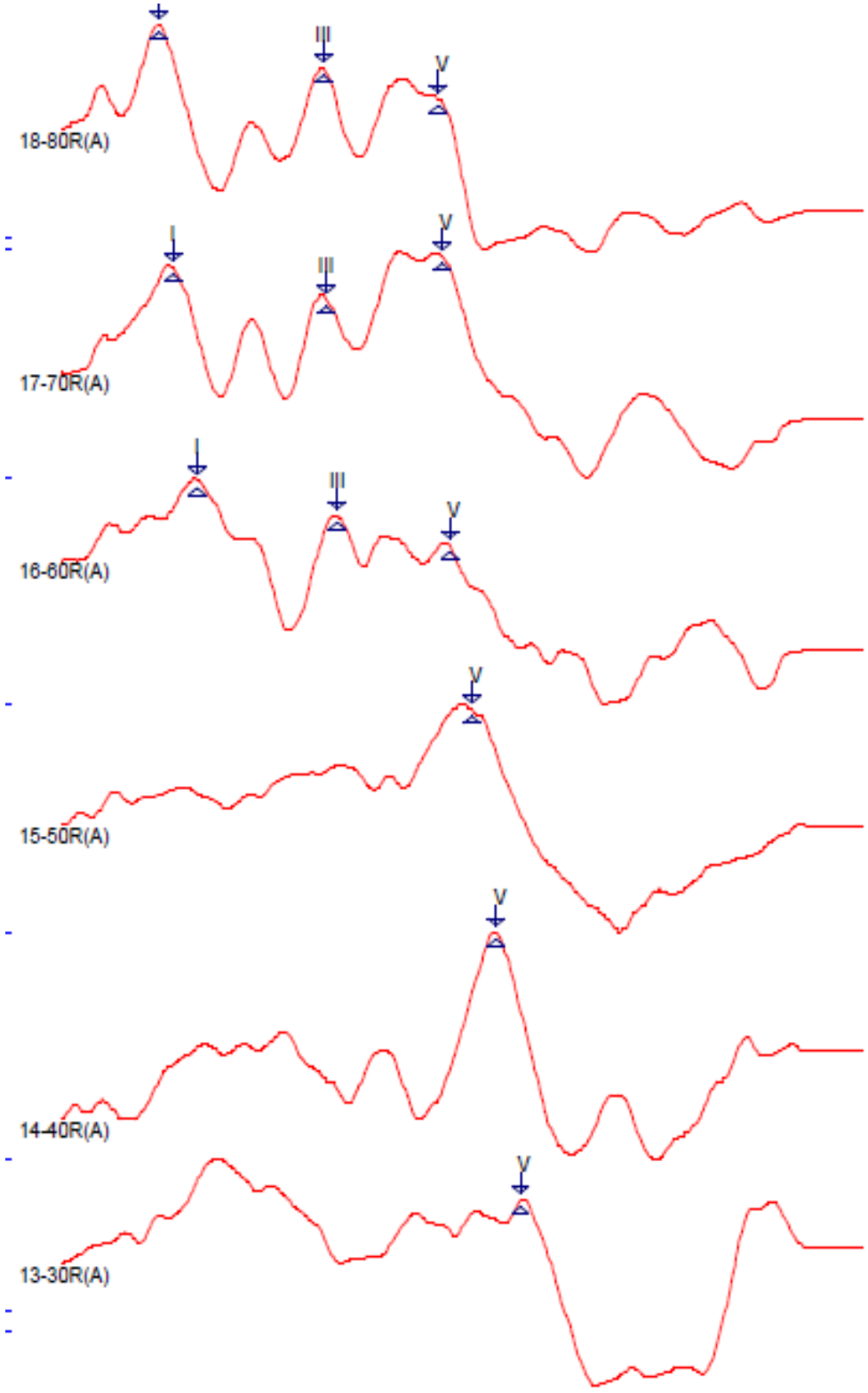


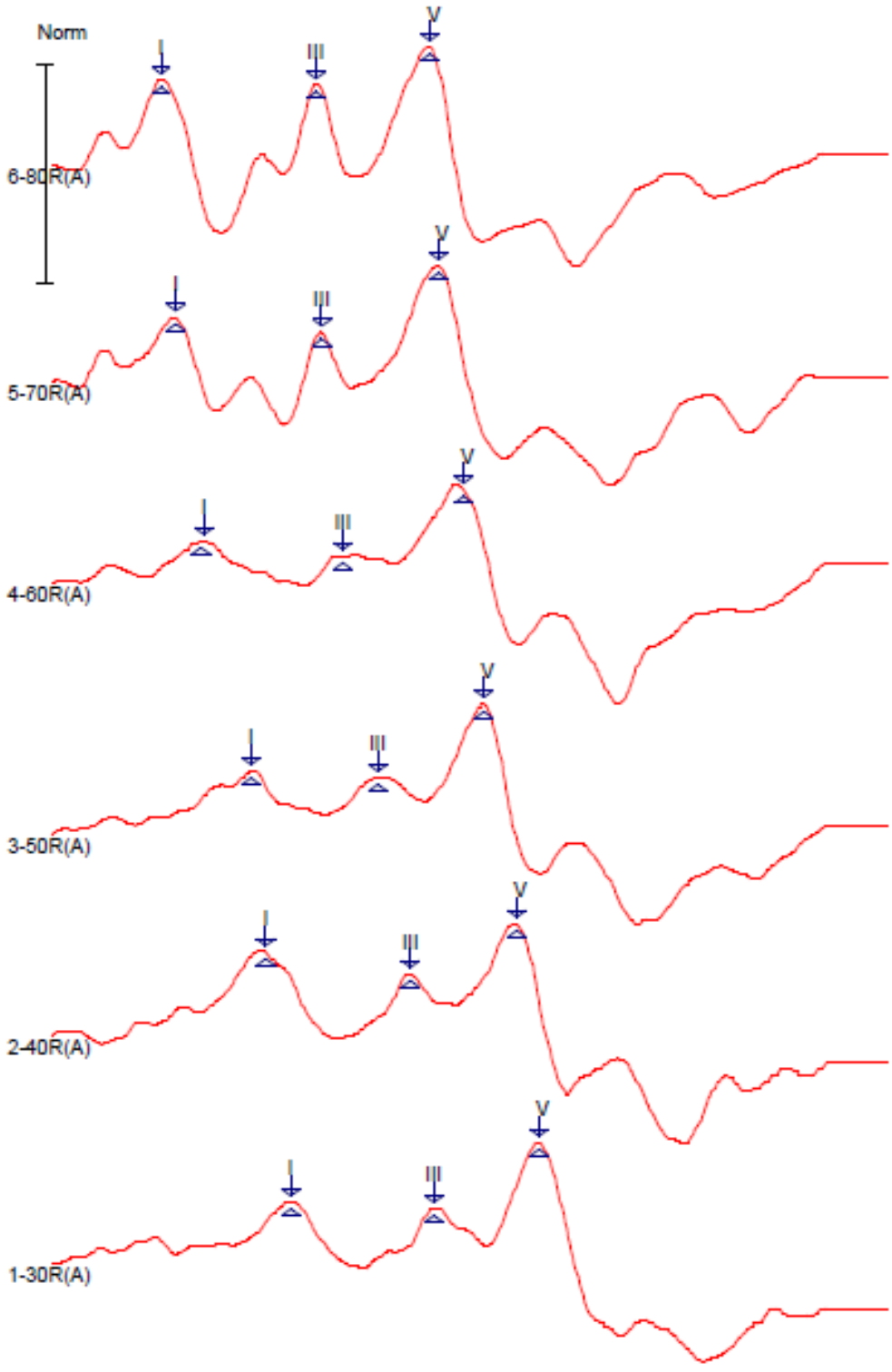


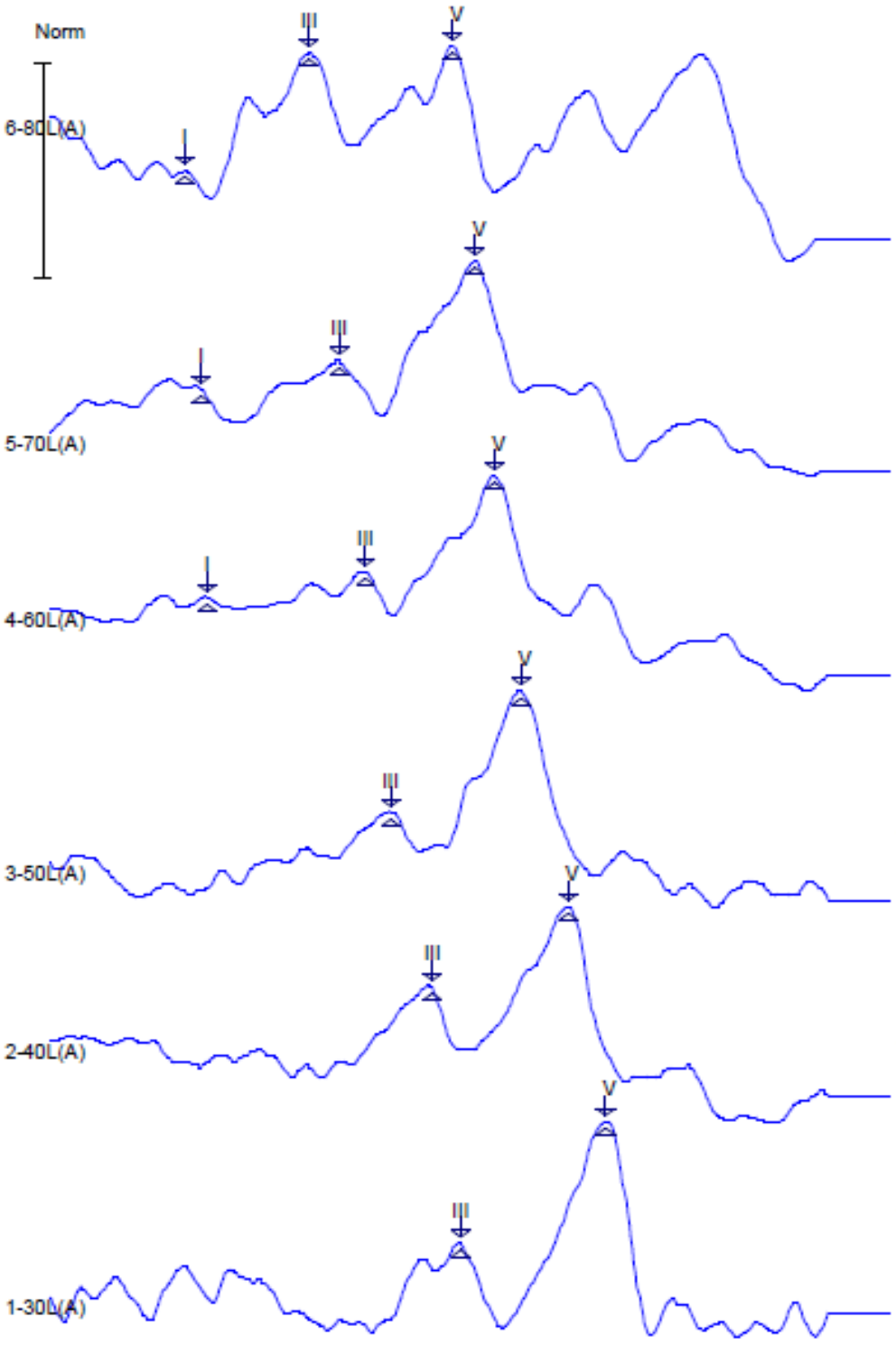


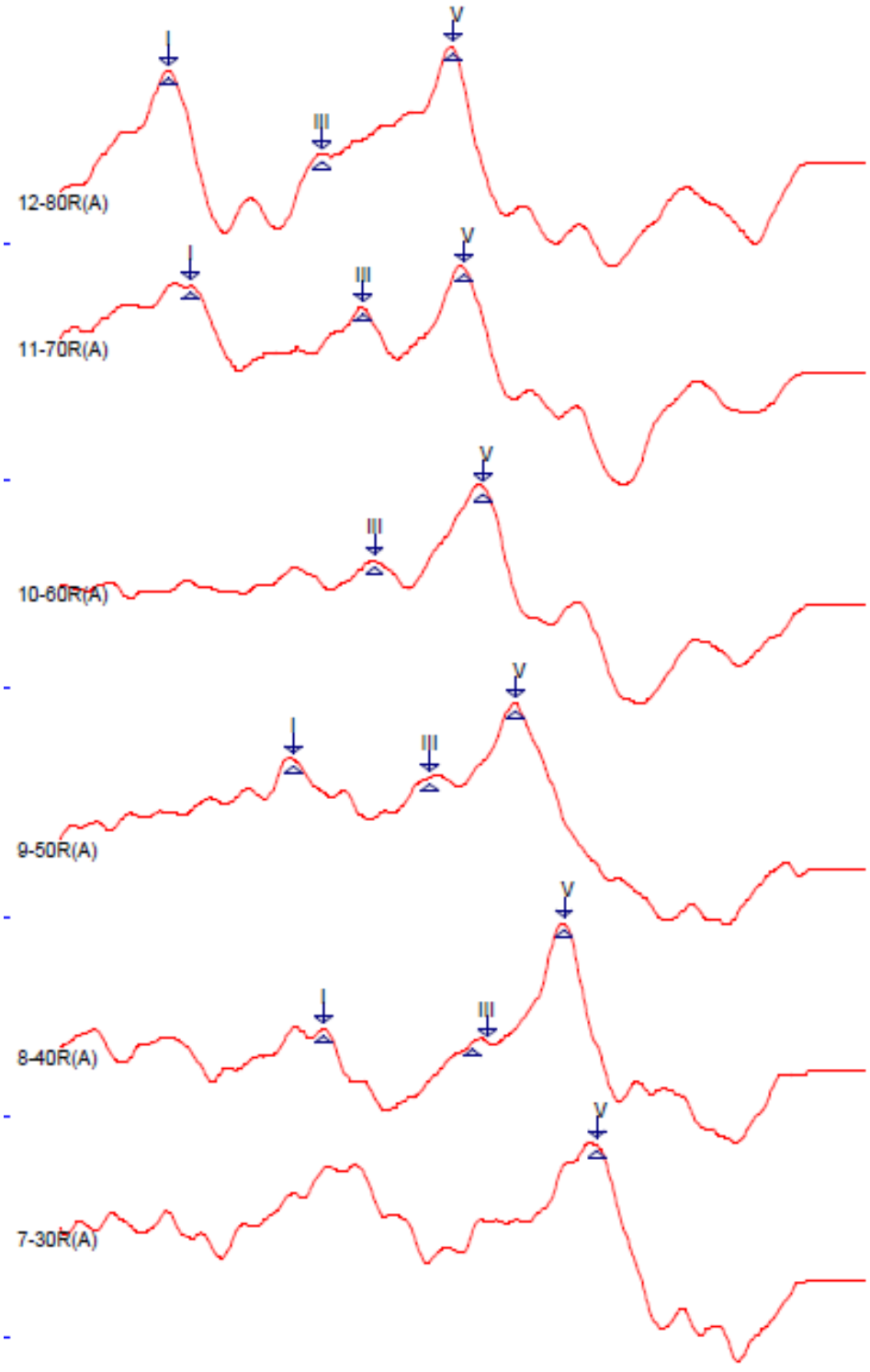


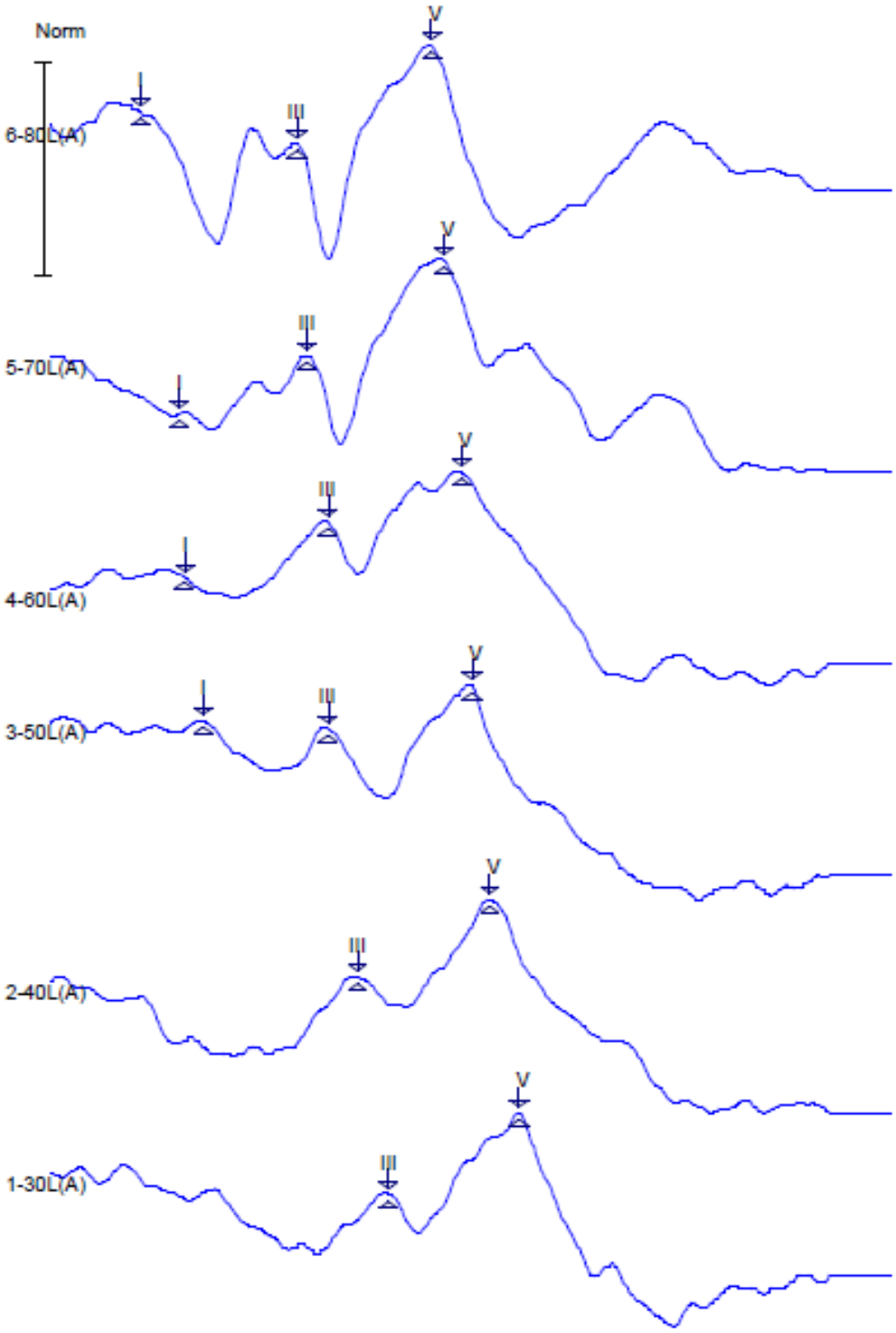


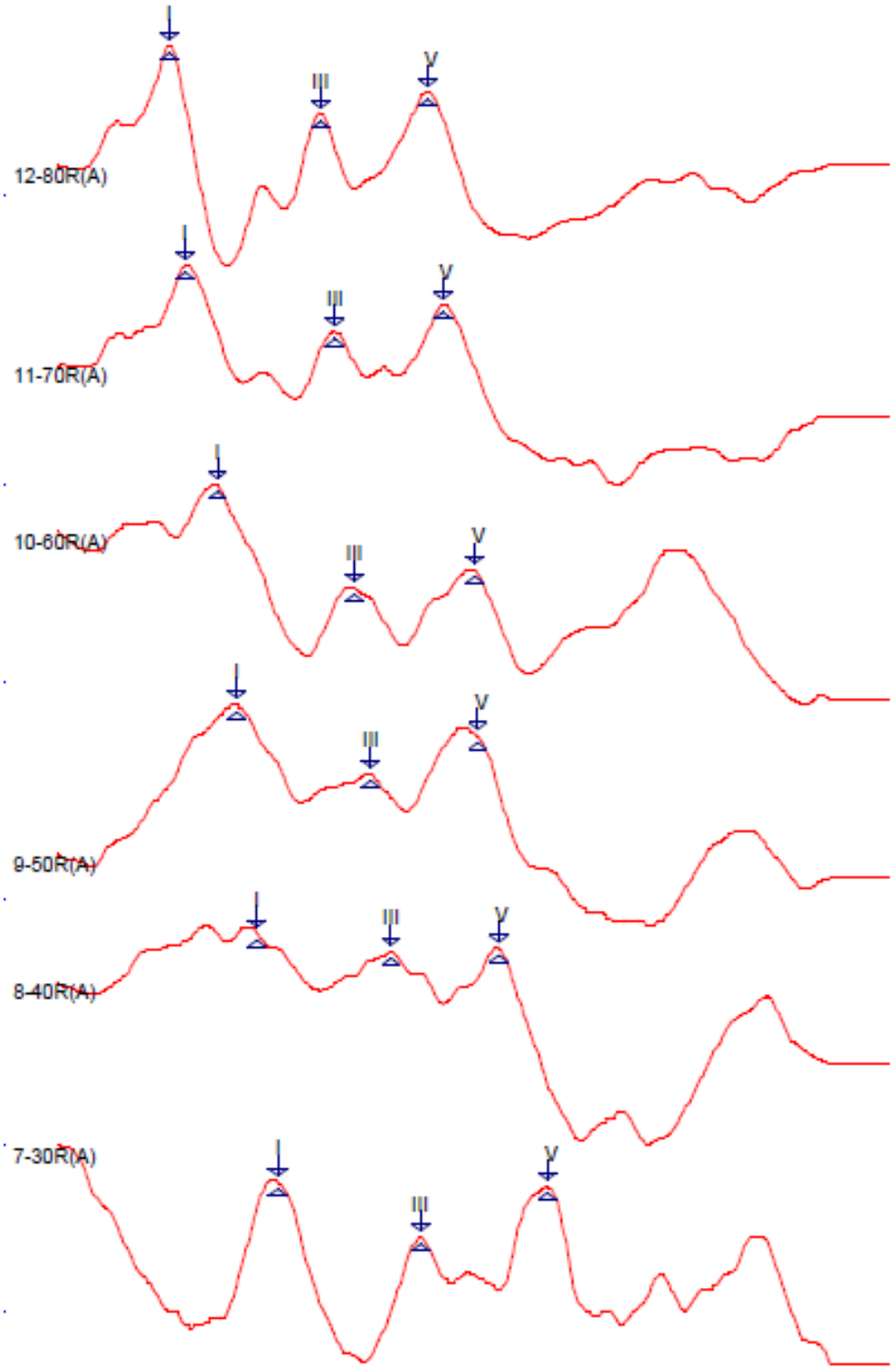


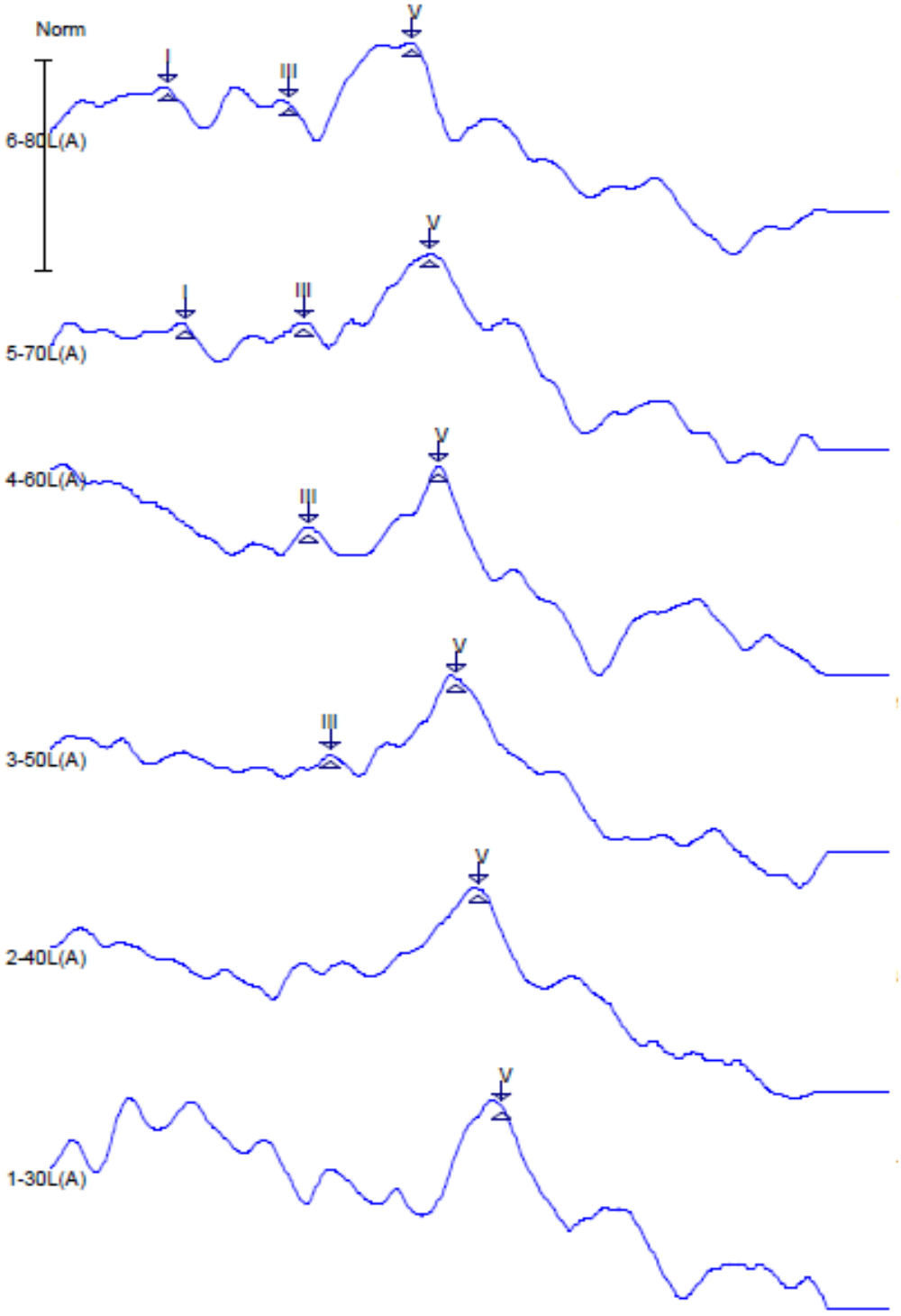


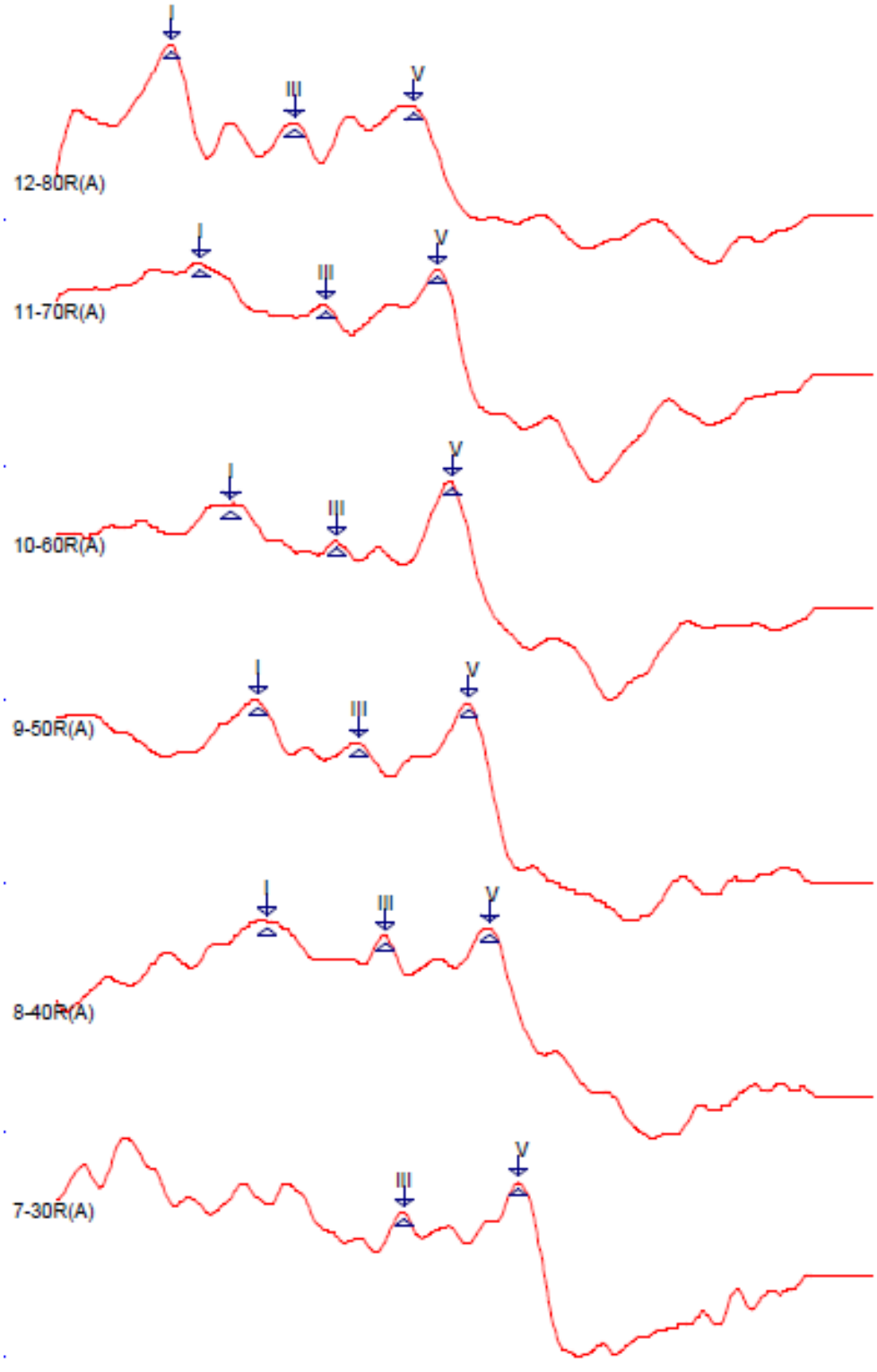


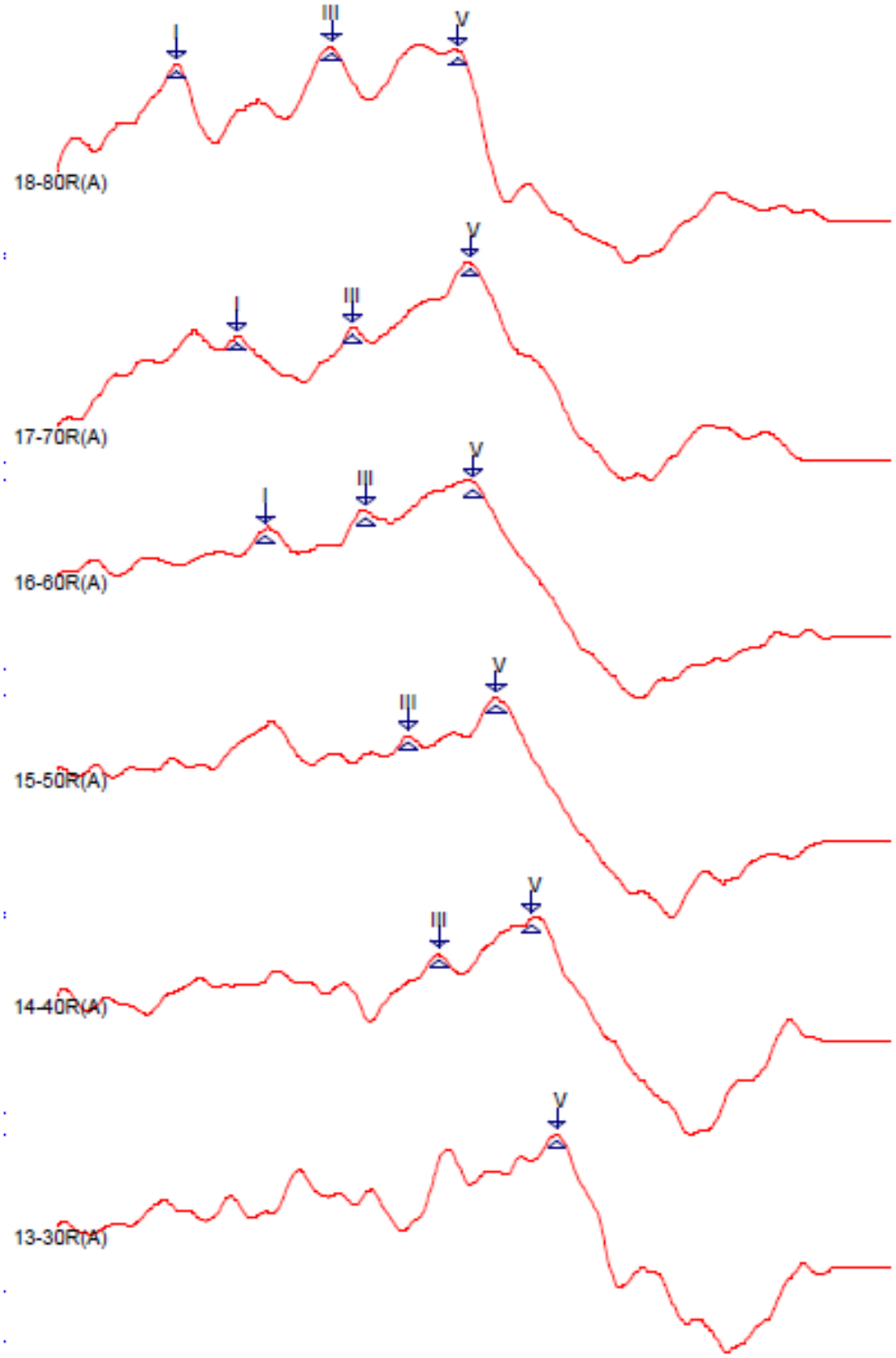


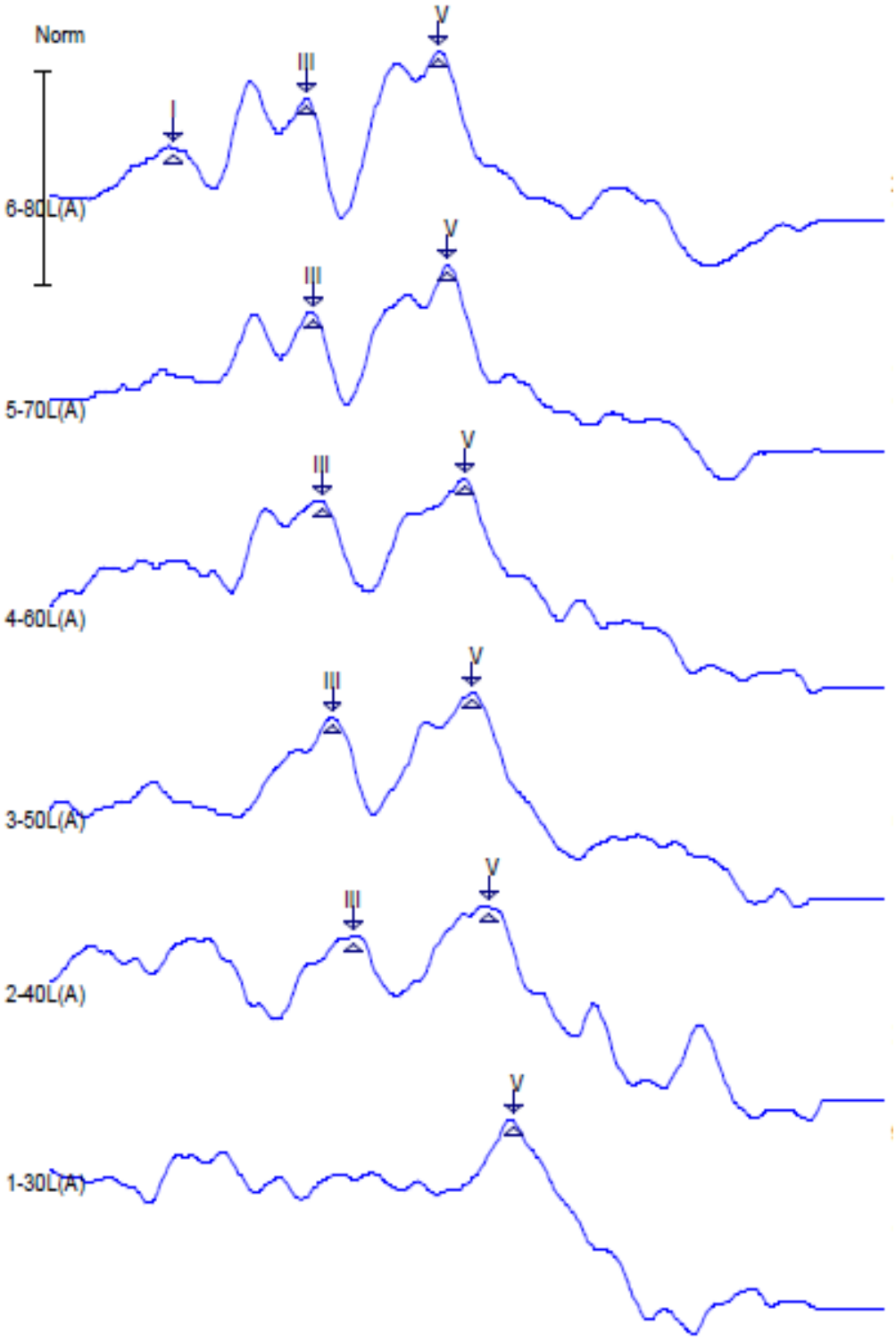


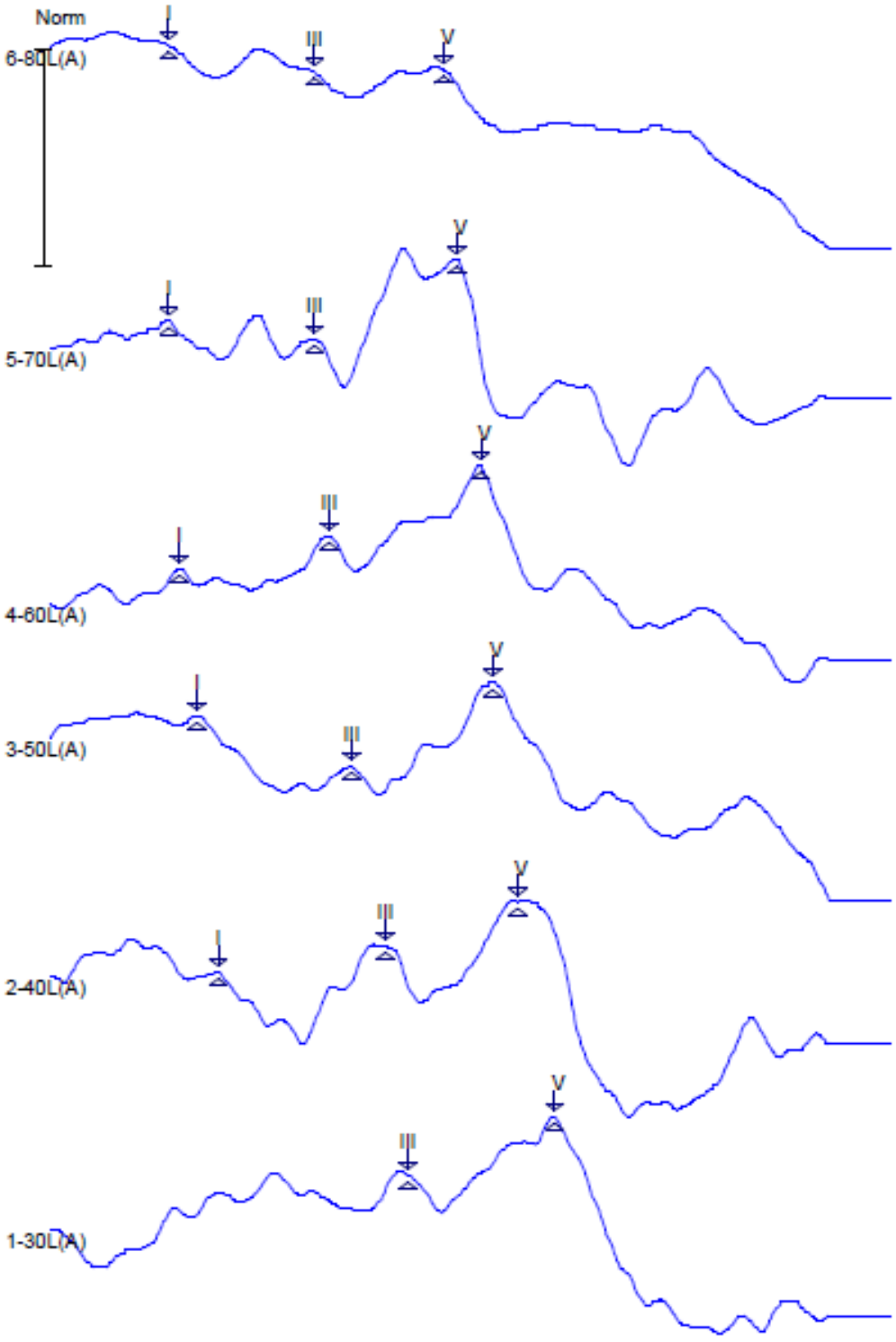


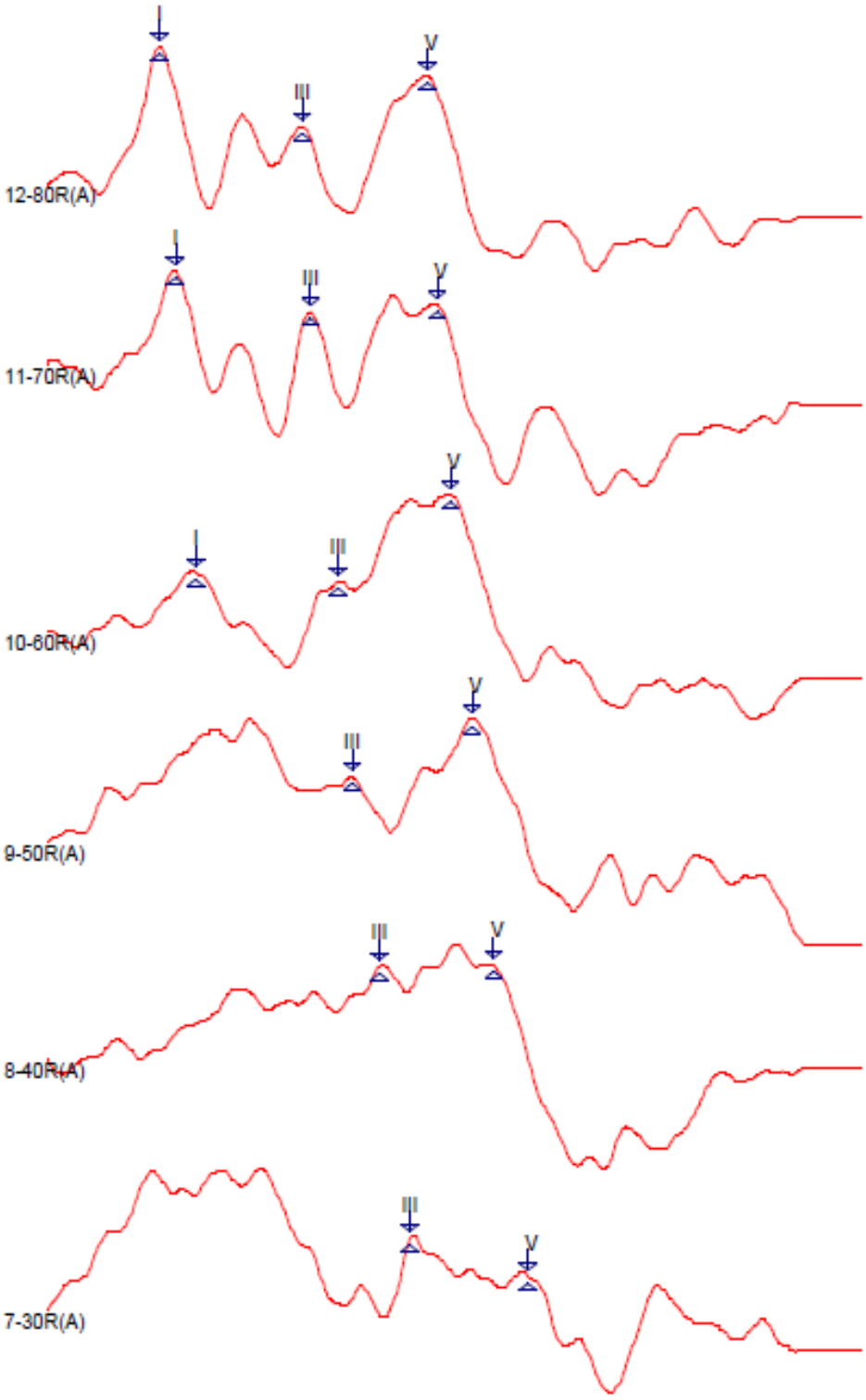


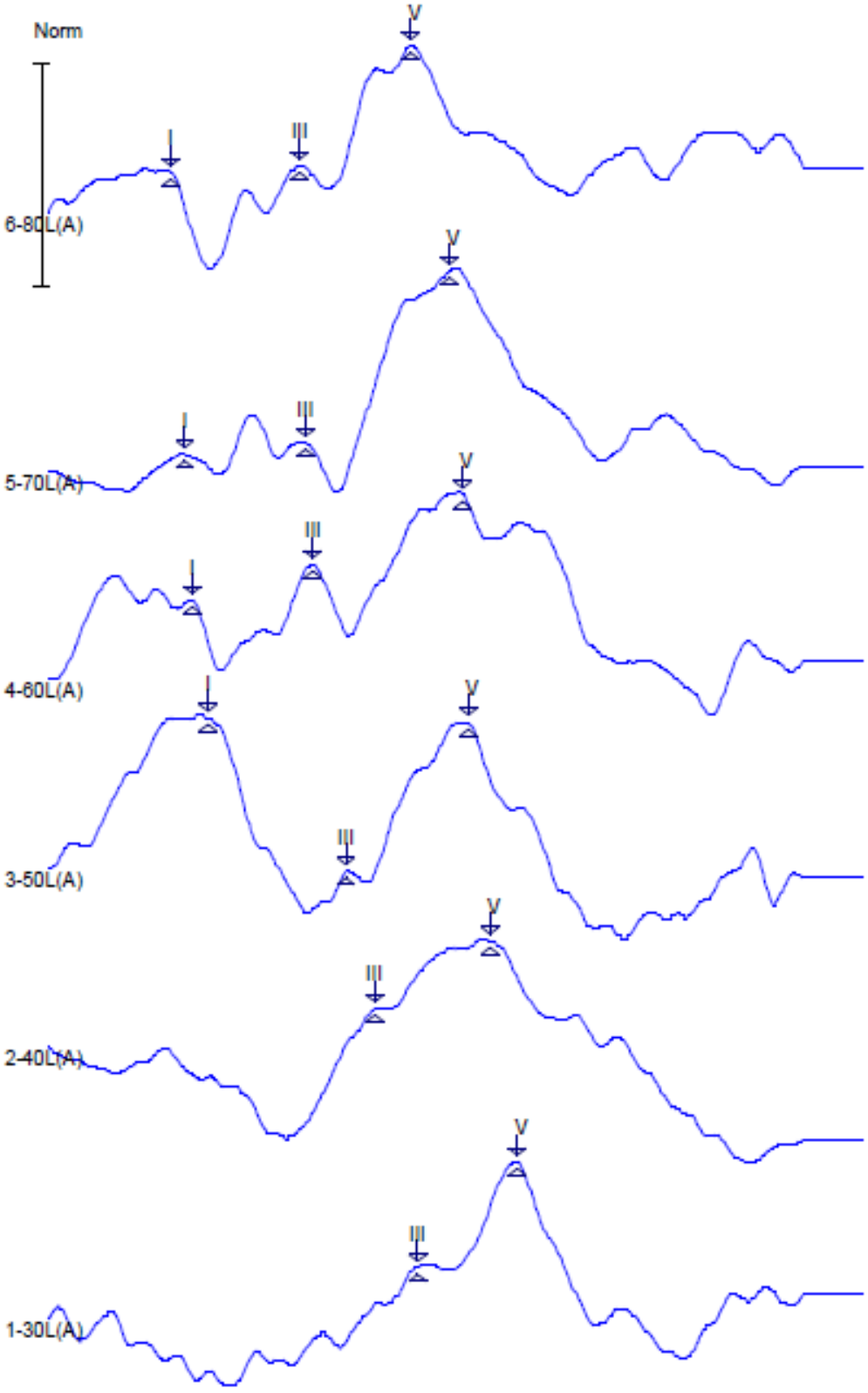


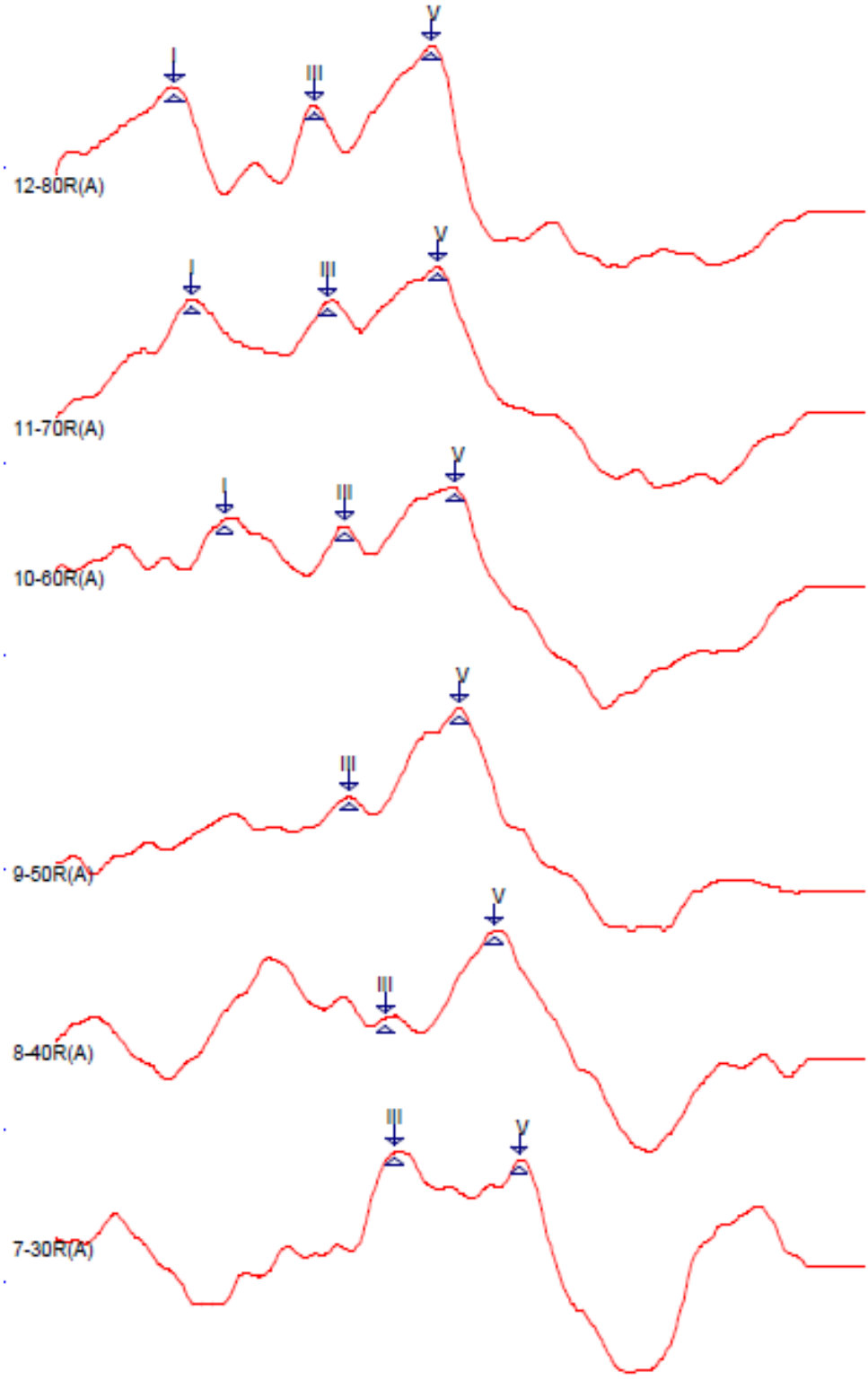


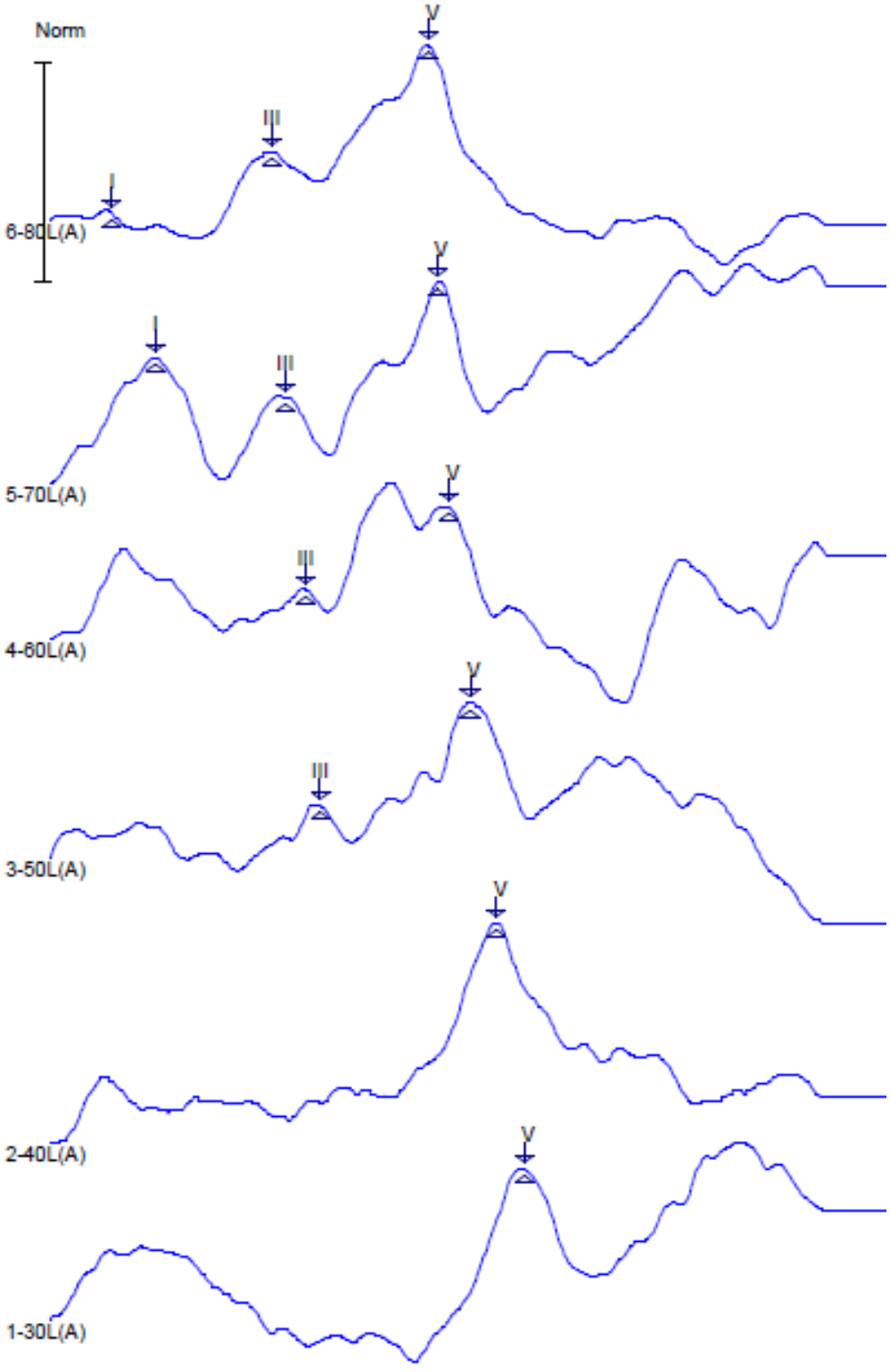


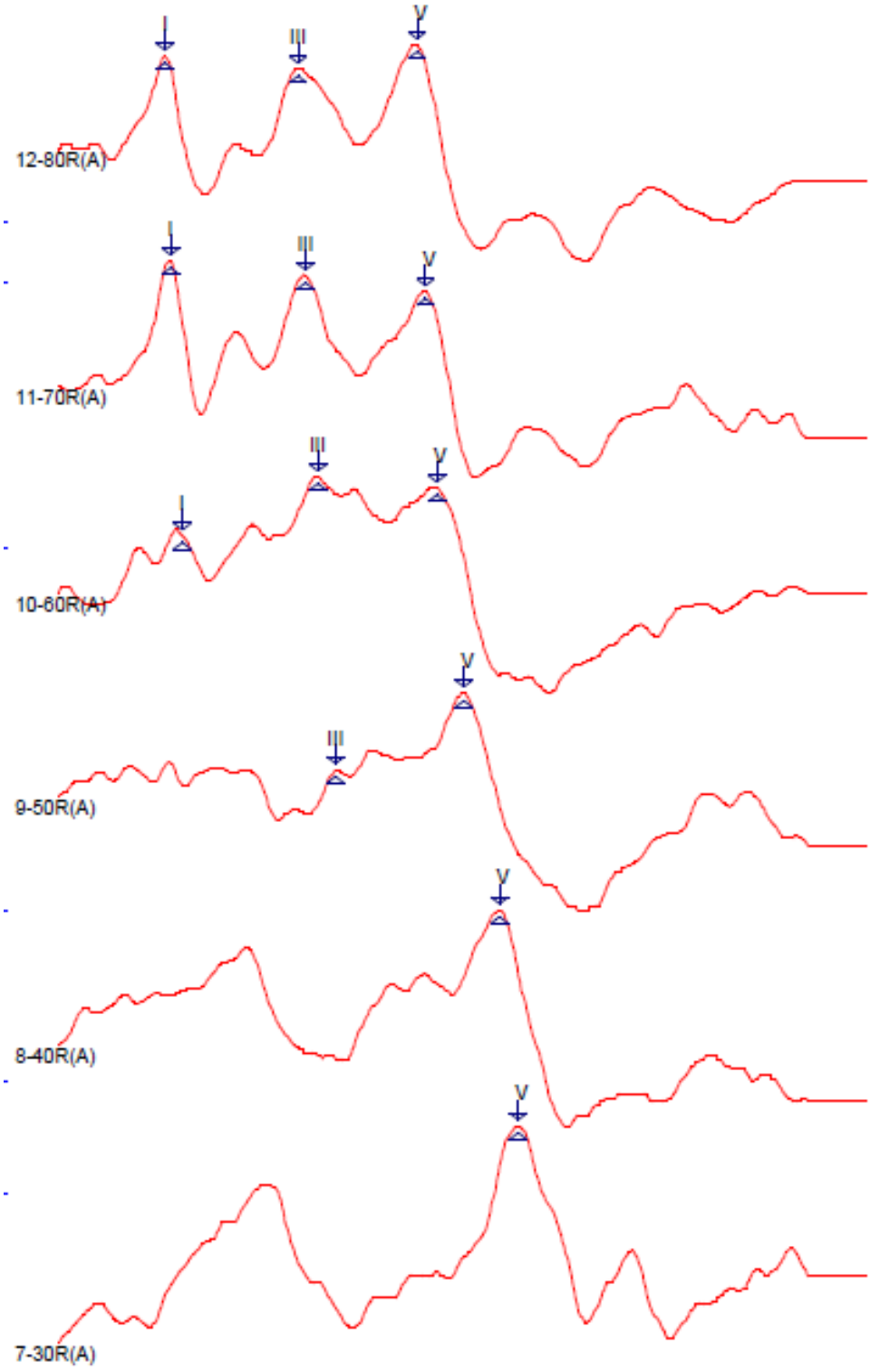


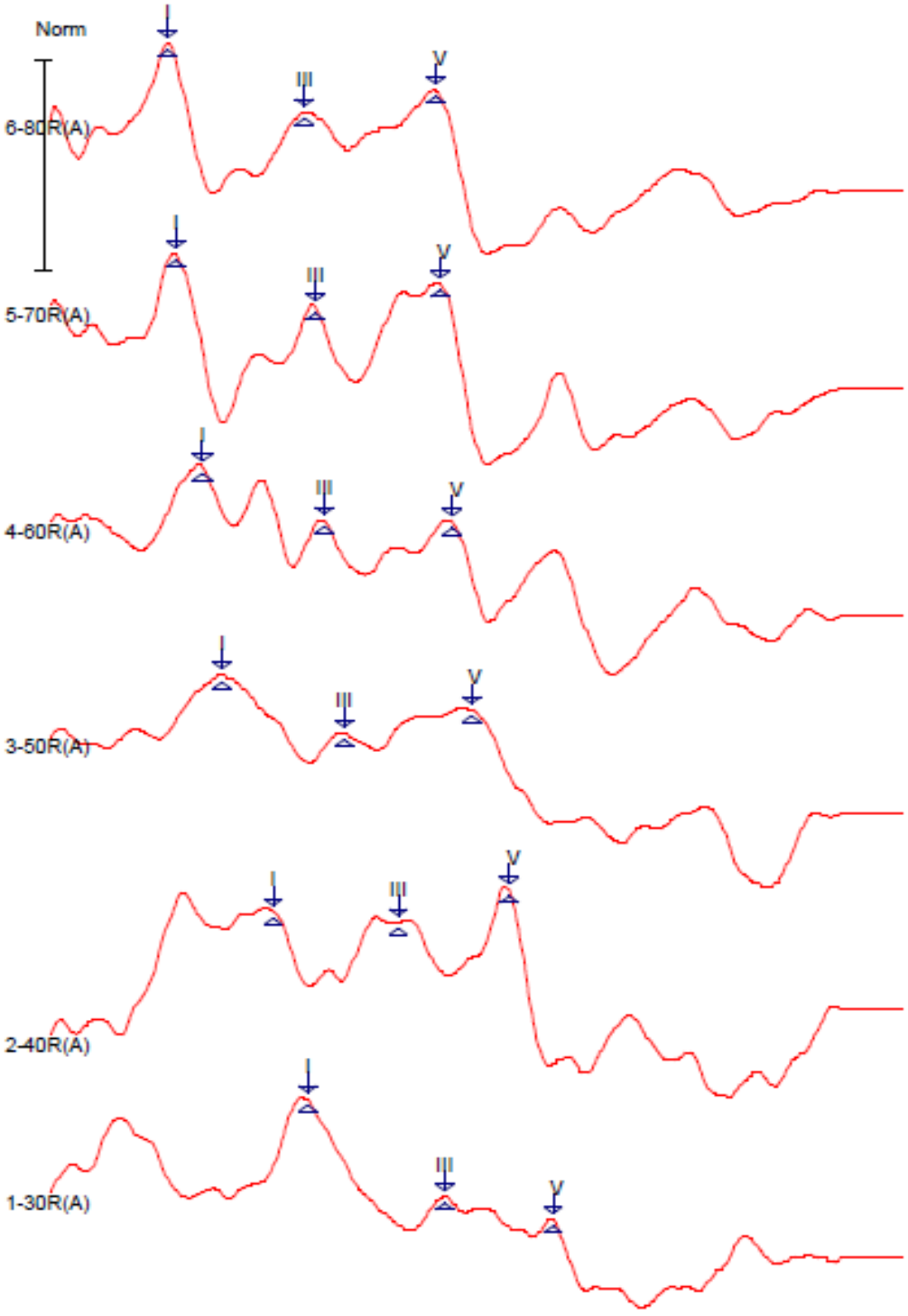


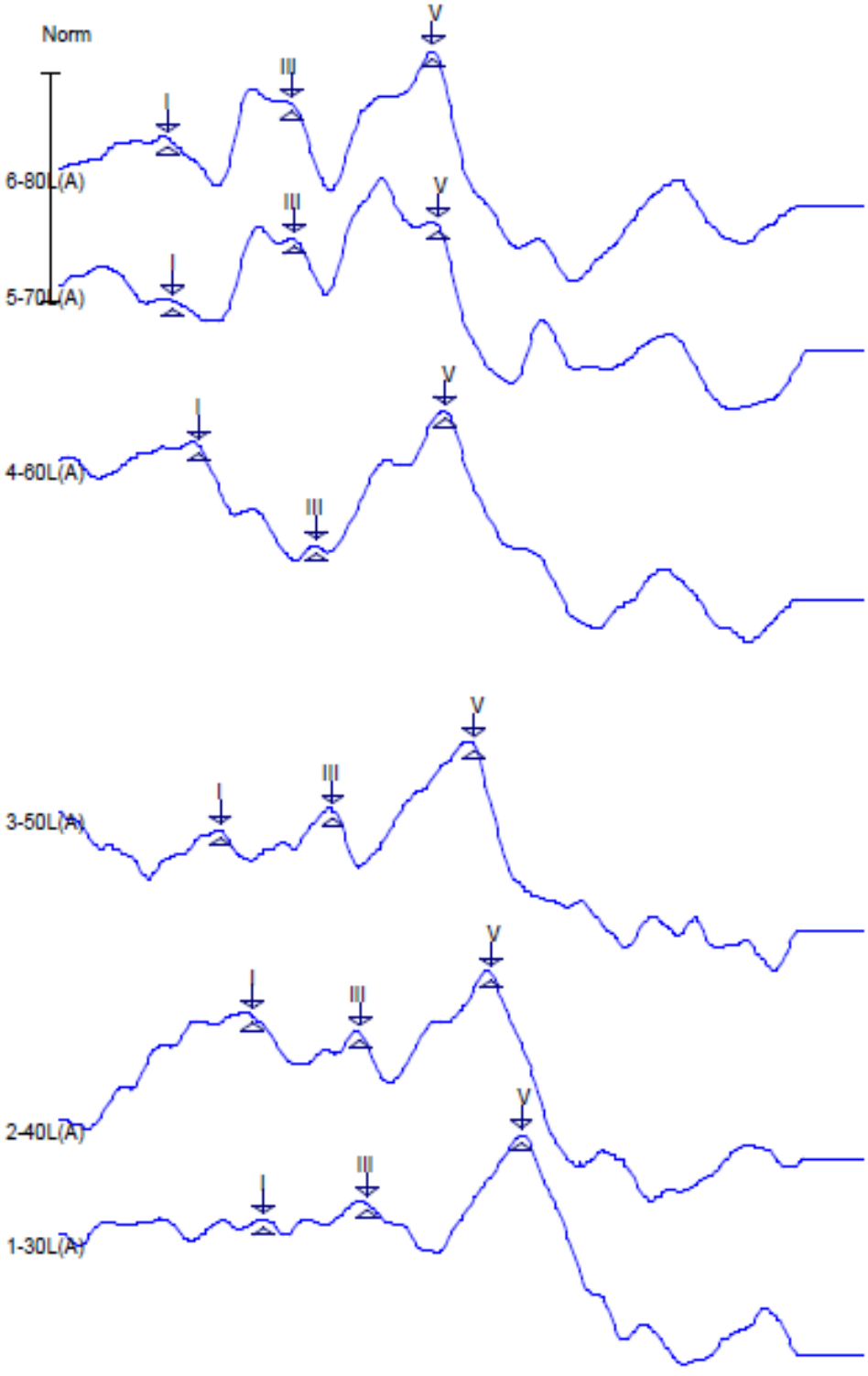


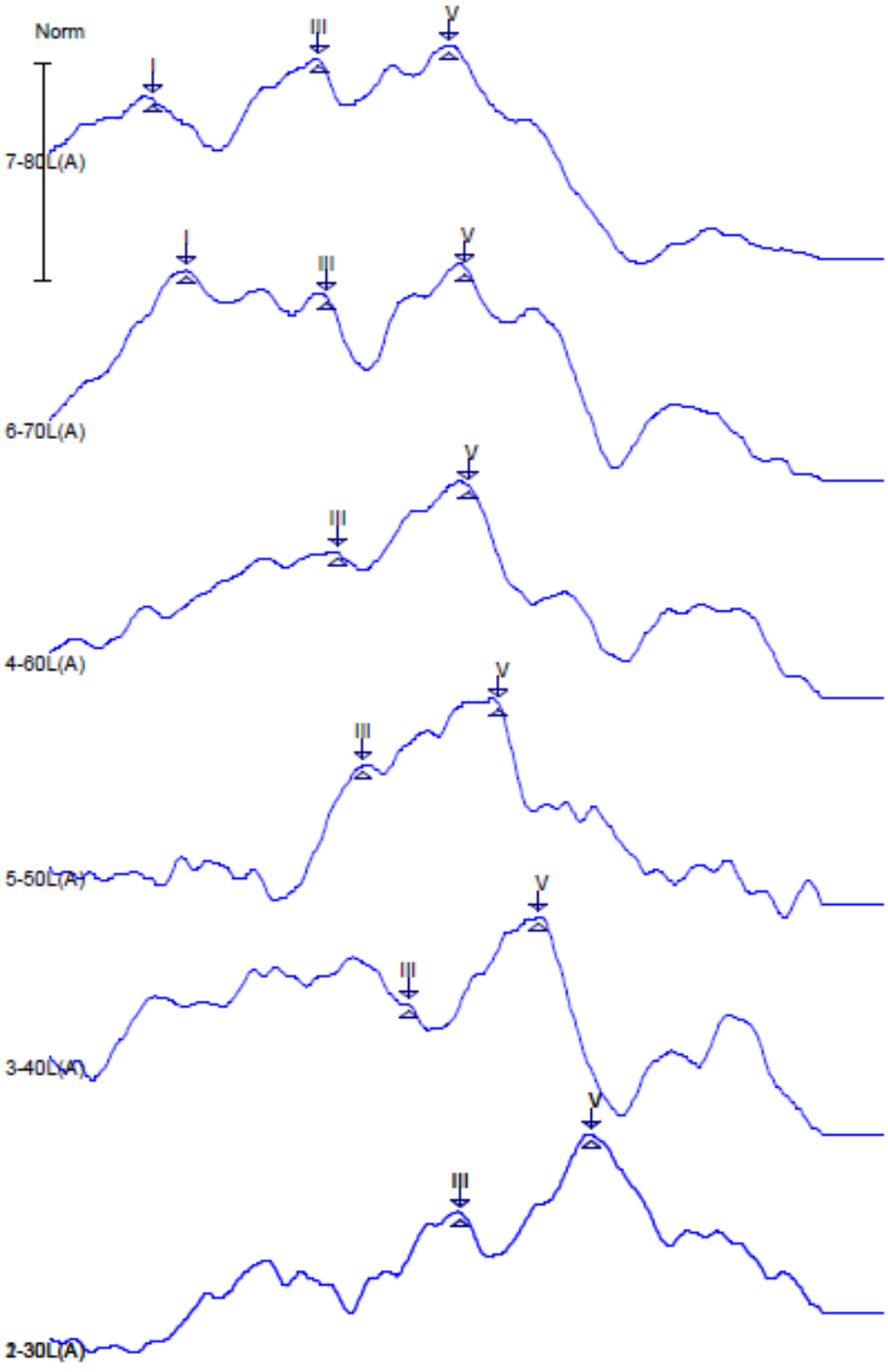


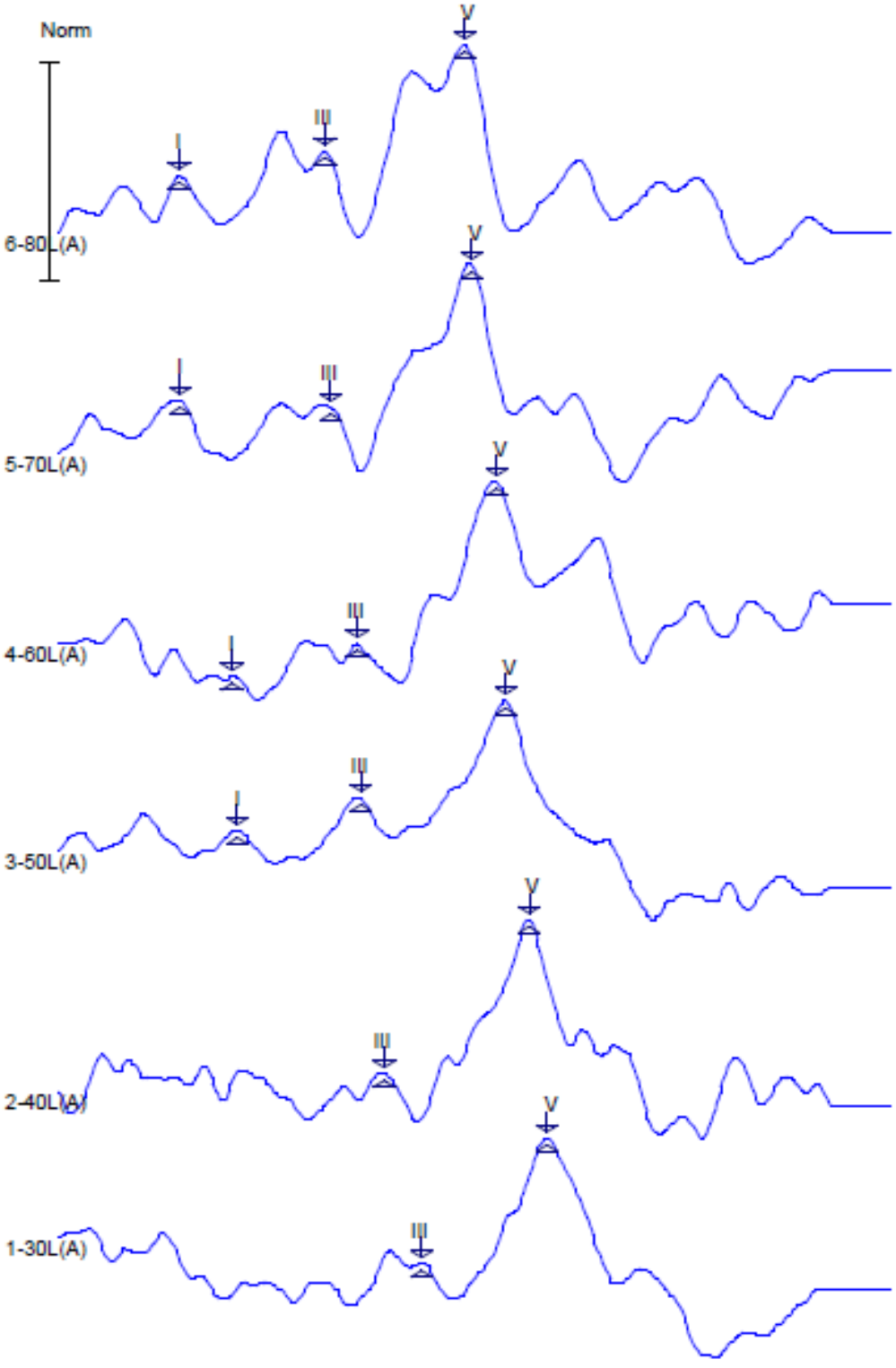


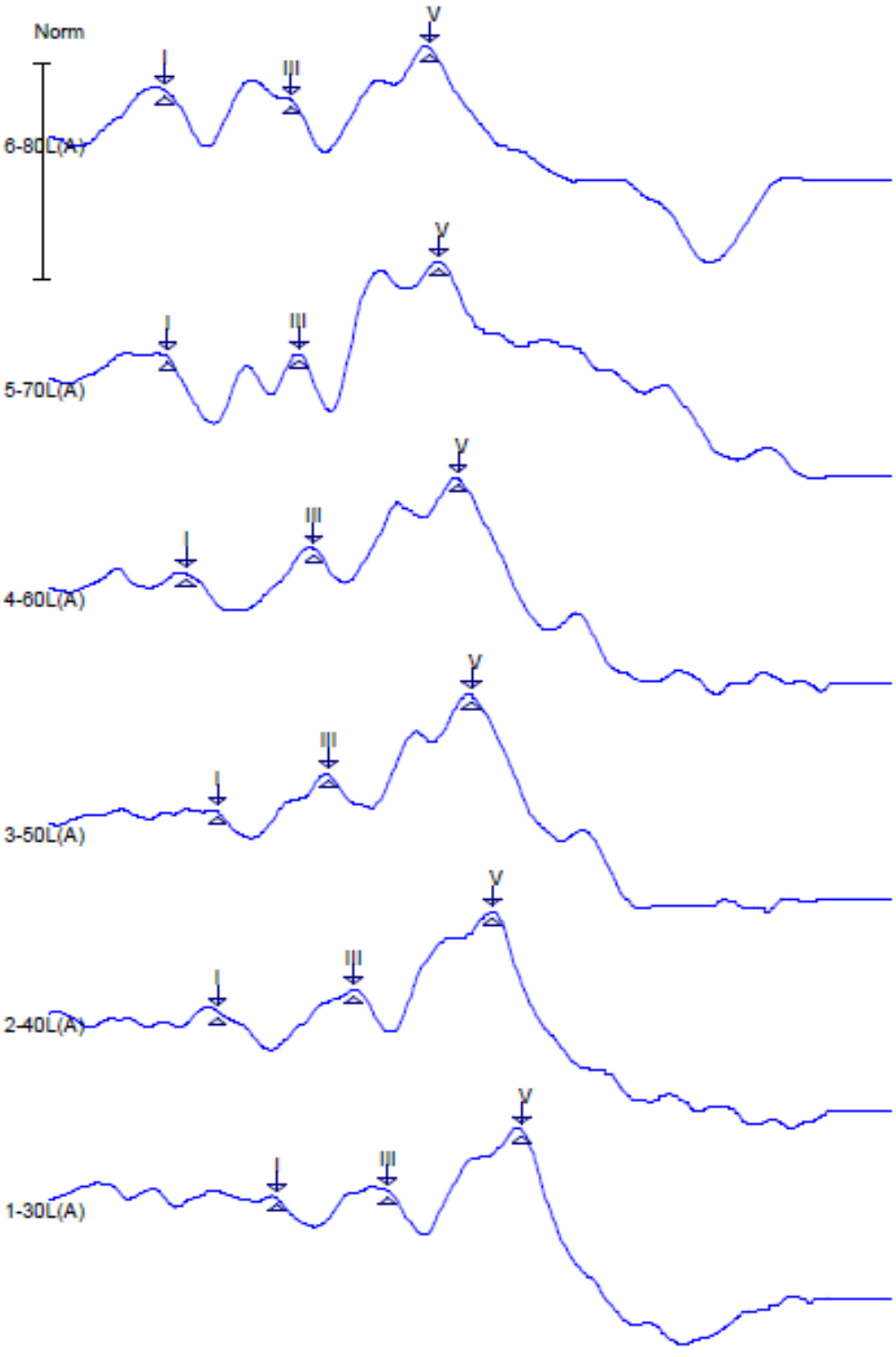


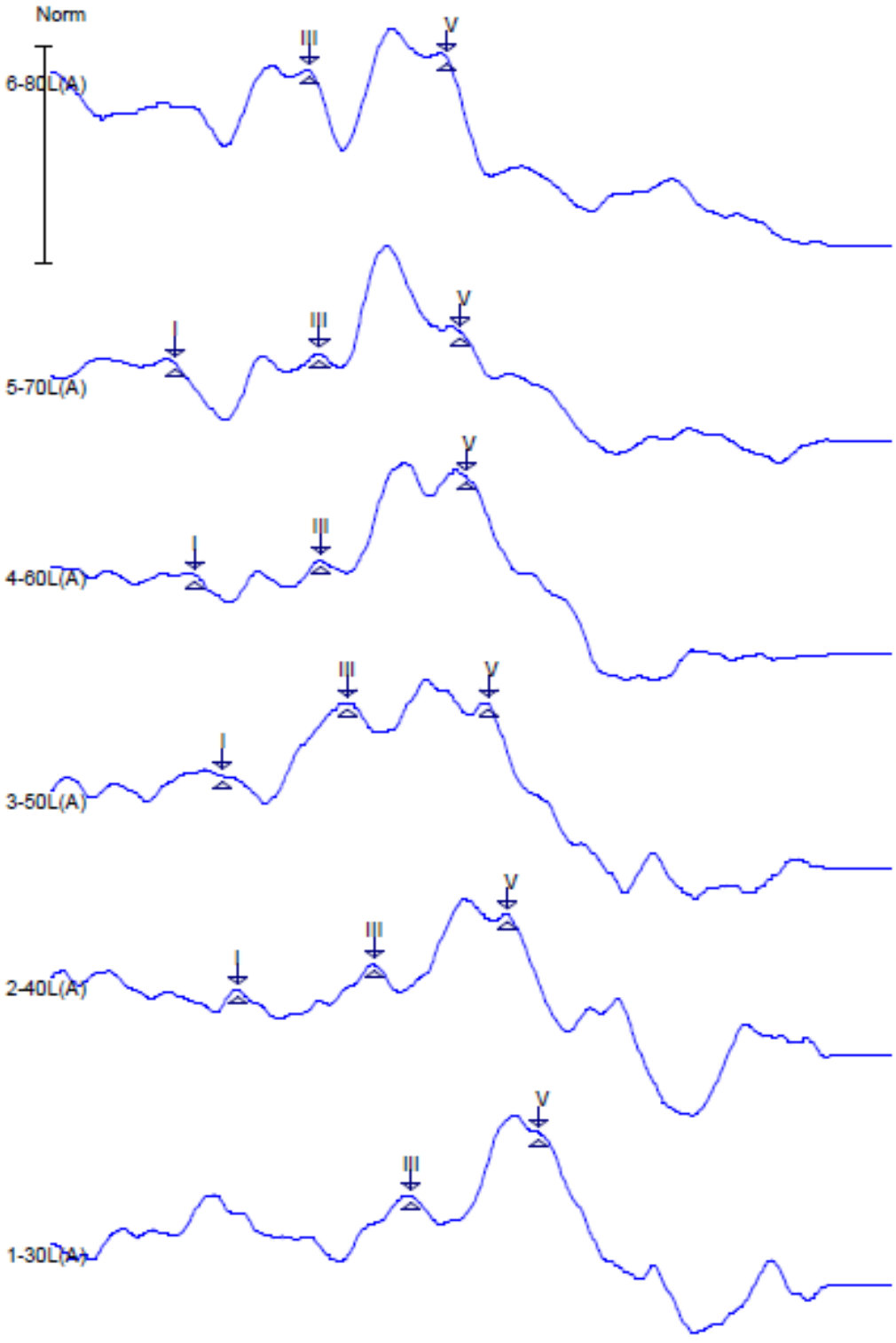












APPENDIX VI (a)

ABSOLUTE LATENCY DATA FOR MALE PARTICIPANTS – 80 to 60 dBnHL

	Intensity 80 dB			Intensity 70 dB			Intensity 60 dB		
ID	**Wave I**	**Wave III**	**Wave V**	**Wave I**	**Wave III**	**Wave V**	**Wave I**	**Wave III**	**Wave V**
AA L	1.73	3.70	5.95	1.82	3.75	6.03	2.23	4.10	6.15
AS	1.77	3.85	5.80	1.90	4.00	5.88	2.30	4.13	6.05
AZAG	1.60	4.15	6.15	2.13	4.28	6.40	2.24	4.45	6.42
AZ	1.75	4.03	5.95	2.25	4.33	6.17	2.27	4.58	6.20
DC	1.88	4.13	6.25	1.90	4.20	6.38	2.70	4.60	6.75
BB	1.77	4.10	6.17	2.13	4.29	6.42	2.31	4.14	6.41
SA	1.55	4.28	6.17	1.77	4.25	6.28	2.08	4.58	6.33
SE	1.57	4.20	6.03	1.80	4.25	6.10	2.20	4.42	6.20
MA	1.93	3.95	5.97	2.12	4.08	6.13	2.32	4.20	6.40
MK	1.90	4.13	5.95	2.20	4.35	6.03	2.24	4.40	6.17
MM	2.08	3.98	6.17	2.33	4.42	6.50	2.42	4.83	6.80
MR	1.75	4.17	6.25	2.10	4.83	6.42	2.34	5.03	6.72
QQ	2.30	4.13	5.95	2.38	4.25	6.28	2.45	4.42	6.60
CC	1.93	4.10	6.00	2.00	4.40	6.28	2.23	4.70	6.22
DA	1.85	4.22	6.17	2.10	4.55	6.35	2.30	4.75	6.40
EA	1.82	4.05	6.00	1.82	4.05	6.20	2.00	4.28	6.58
EA 1	1.77	4.03	5.97	2.02	4.15	6.15	2.27	4.38	6.30
POO	1.63	3.63	5.88	1.85	3.80	6.03	2.10	3.88	6.25
ST	1.93	3.52	5.65	2.27	3.92	5.95	2.40	4.28	6.22
ST4	1.77	3.88	5.53	1.90	4.03	5.72	2.67	4.38	6.05
PO	1.80	4.00	5.83	2.02	4.17	5.90	2.48	4.58	6.05
IA	1.73	3.92	5.58	1.93	4.05	5.60	2.17	4.33	6.17
SS	1.73	4.28	6.20	2.00	4.42	6.40	2.17	4.55	6.67
SSS	1.98	3.92	6.17	2.02	4.03	6.22	2.23	4.47	6.67
BQ	1.45	3.95	6.30	2.23	4.13	6.45	2.23	4.40	6.88
Mean	**1.80**	**4.01**	**6.00**	**2.04**	**4.20**	**6.17**	**2.29**	**4.43**	**6.39**
SD	**0.18**	**0.19**	**0.21**	**0.17**	**0.23**	**0.24**	**0.16**	**0.25**	**0.25**

APPENDIX VI (b)

ABSOLUTE LATENCY DATA FOR MALE PARTICIPANTS – 50 to 30 dBnHL

	Intensity 50 dB			Intensity 40 dB			Intensity 30 dB		
ID	**Wave I**	**Wave III**	**Wave V**	**Wave I**	**Wave III**	**Wave V**	**Wave I**	**Wave III**	**Wave V**
AA L	2.58	4.35	6.60	3.08	4.80	6.88	3.27	4.90	7.38
AS	2.60	4.42	6.35	3.38	5.25	6.90	3.88	5.95	7.58
AZAG	2.75	4.83	6.90	3.20	5.53	7.53		6.33	8.32
AZ	2.70	5.15	6.45	3.65	5.95	7.13		5.21	7.47
DC	2.77	4.67	6.90	3.12	5.03	7.25	3.58	5.60	7.53
BB	2.85	4.97	6.92	3.35	5.50	7.28	3.63	5.75	7.60
SA	2.23	5.03	6.92	2.63	5.90	7.05		5.80	7.55
SE	2.77	4.70	6.58	3.42	5.35	6.95		5.45	7.35
MA	2.48	4.35	6.50	2.59	4.67	6.75			
MK	2.55	4.34	6.55	2.59	4.63	6.72	3.02	4.82	7.20
MM	2.53	5.22	7.20	3.02	5.85	7.92	3.45	6.28	8.50
MR	2.85	5.90	7.25			8.53			
QQ	2.45	4.67	6.85		5.22	7.15		5.67	8.20
CC	3.17	5.08	7.00		5.26	7.15			7.60
DA		5.40	6.75		5.03	7.30			7.67
EA	2.25	4.60	6.75	2.58	5.13	7.13		5.47	7.70
EA 1		4.80	6.67		5.25	7.03		5.72	7.58
POO	2.40	4.47	6.70	2.92	4.83	6.78	3.70	5.30	6.95
ST		4.47	6.55		4.80	6.85		5.42	7.38
ST4	2.80	4.58	6.40	3.17	5.03	6.78	3.85	5.75	7.15
PO	2.75	4.70	6.58	3.23	5.35	7.10	3.77	5.95	7.67
IA	2.33	4.38	6.35	2.95	4.40	6.60	3.35	5.30	6.90
SS	2.48	5.03	7.03	3.17	5.80	7.80		6.20	8.38
SSS			7.13			8.00			8.20
BQ	2.56	4.75	7.20	2.63	4.92	7.42			7.42
Mean	**2.61**	**4.79**	**6.76**	**3.04**	**5.19**	**7.20**	**3.55**	**5.62**	**7.62**
SD	**0.23**	**0.38**	**0.27**	**0.33**	**0.42**	**0.46**	**0.28**	**0.42**	**0.43**

APPENDIX VII (a)

INTER-WAVE DATA FOR MALE PARTICIPANTS –80 to 60 dBnHL

	Intensity 80 dB			Intensity 70 dB			Intensity 60 dB		
ID	I-III	III-V	I-V	I-III	III-V	I-V	I-III	III-V	I-V
AA L	1.98	2.25	4.23	1.93	2.28	4.20	1.87	2.05	3.93
AS	2.08	1.95	4.02	2.10	1.88	3.98	1.83	1.92	3.75
AZAG	2.55	2.00	4.55	2.15	2.13	4.28	2.21	1.97	4.18
AZ	2.28	1.92	4.20	2.08	1.85	3.92	2.30	1.63	3.93
DC	2.25	2.13	4.38	2.30	2.17	4.48	1.90	2.15	4.05
BB	2.32	2.08	4.40	2.16	2.13	4.29	1.83	2.27	4.10
SA	2.73	1.90	4.62	2.48	2.03	4.50	2.50	1.75	4.25
SE	2.63	1.83	4.45	2.45	1.85	4.30	2.22	1.78	4.00
MA	2.03	2.02	4.05	1.96	2.05	4.01	1.88	2.20	4.08
MK	2.23	1.83	4.05	2.15	1.68	3.83	2.16	1.77	3.93
MM	1.90	2.20	4.10	2.10	2.08	4.17	2.40	1.97	4.38
MR	2.42	2.08	4.50	2.73	1.60	4.32	2.69	1.70	4.38
QQ	1.83	1.83	3.65	1.88	2.03	3.90	1.97	2.17	4.15
CC	2.17	1.90	4.07	2.40	1.88	4.28	2.48	1.52	4.00
DA	2.37	1.95	4.32	2.45	1.80	4.25	2.45	1.65	4.10
EA	2.22	1.95	4.18	2.22	2.15	4.38	2.28	2.30	4.58
EA 1	2.25	1.95	4.20	2.13	2.00	4.13	2.10	1.92	4.02
POO	2.00	2.25	4.25	1.95	2.23	4.18	1.77	2.38	4.15
ST	1.60	2.13	3.73	1.65	2.03	3.68	1.88	1.95	3.82
ST4	2.10	1.65	3.75	2.13	1.70	3.82	1.70	1.67	3.38
PO	2.20	1.83	4.03	2.15	1.73	3.88	2.10	1.47	3.57
IA	2.20	1.65	3.85	2.12	1.55	3.67	2.15	1.85	4.00
SS	2.55	1.92	4.48	2.42	1.98	4.40	2.38	2.13	4.50
SSS	1.95	2.25	4.20	2.00	2.20	4.20	2.25	2.20	4.45
BQ	2.50	2.35	4.85	1.90	2.33	4.23	2.17	2.47	4.65
Mean	**2.21**	**1.99**	**4.20**	**2.16**	**1.97**	**4.13**	**2.14**	**1.95**	**4.09**
SD	**0.27**	**0.18**	**0.29**	**0.24**	**0.21**	**0.24**	**0.26**	**0.27**	**0.30**

APPENDIX VII (b)

INTER-WAVE DATA FOR MALE PARTICIPANTS – 50 to 30 dBnHL

	Intensity 50 dB			Intensity 40 dB			Intensity 30 dB		
ID	I-III	III-V	I-V	I-III	III-V	I-V	I-III	III-V	I-V
AA L	1.77	2.25	4.02	1.72	2.08	3.80	1.63	2.47	4.10
AS	1.82	1.92	3.75	1.88	1.65	3.53	2.08	1.63	3.70
AZAG	2.08	2.08	4.23	2.33	2.00	4.33		2.00	
AZ	2.45	1.30	3.75	2.30	1.17	3.48		2.26	
DC	1.90	2.23	4.13	1.91	2.22	4.13	2.02	1.93	3.95
BB	2.12	1.95	4.07	2.15	1.78	3.93	2.13	1.85	3.97
SA	2.80	1.90	4.69	3.27	1.15	4.42		1.75	
SE	1.93	1.88	3.81	1.93	1.60	3.53		1.90	
MA	1.87	2.15	4.10	2.08	2.08	4.16			
MK	1.79	2.21	4.00	2.04	2.09	4.13	1.80	2.38	4.18
MM	2.39	1.98	4.67	2.83	2.08	4.90	2.83	2.22	5.05
MR	3.05	1.35	4.40		2.33				
QQ	2.22	2.17	4.40		1.93			2.52	
CC	1.90	1.92	3.83		1.89				
DA		1.35			2.27				
EA	2.35	2.15	4.50	2.55	2.00	4.55		2.23	
EA 1		1.88			1.78			1.85	
POO	2.07	2.23	4.30	1.90	1.95	3.85	1.60	1.65	3.25
ST		2.08			2.05			1.95	
ST4	1.78	1.83	3.60	1.85	1.75	3.60	1.90	1.40	3.30
PO	1.95	1.88	3.83	2.12	1.75	3.87	2.18	1.72	3.90
IA	2.05	1.97	4.02	1.45	2.20	3.65	1.95	1.60	3.55
SS	2.55	2.00	4.55	2.63	2.00	4.63		2.17	
SSS									
BQ	2.19	2.45	4.64						
Mean	**2.14**	**1.96**	**4.16**	**2.17**	**1.90**	**4.03**	**2.01**	**1.97**	**3.90**
SD	**0.35**	**0.29**	**0.34**	**0.45**	**0.30**	**0.43**	**0.35**	**0.32**	**0.52**

APPENDIX VIII (a)

ABSOLUTE LATENCY DATA FOR FEMALE PARTICIPANTS – 80 to 60 dBnHL

	Intensity 80 dB			Intensity 70 dB			Intensity 60 dB		
ID	**Wave I**	**Wave III**	**Wave V**	**Wave I**	**Wave III**	**Wave V**	**Wave I**	**Wave III**	**Wave V**
BF	1.80	4.00	6.13	2.15	4.40	6.50	2.50	4.70	6.67
EE	1.93	3.35	5.17	2.13	3.40	5.10	2.23	3.48	5.25
BC	1.70	4.10	5.53	1.90	4.13	5.47	2.15	4.20	5.65
FM	1.70	4.08	5.80	1.93	4.15	5.95	2.35	4.47	6.33
JK	1.88	3.88	5.83	2.10	4.08	6.03	2.30	4.25	6.45
JJ	1.82	4.10	5.95	2.08	4.40	6.03	2.35	4.65	6.28
JO	1.80	3.67	5.72	1.90	3.65	5.72	2.28	4.25	6.08
JOG	1.77	4.03	5.65	1.98	4.10	5.75	2.85	4.70	6.28
LO	1.93	3.77	6.08	1.93	4.10	6.25	2.23	4.13	6.35
OO	1.70	4.10	5.60	1.80	4.17	5.65	2.13	4.25	6.05
LTD	1.77	3.70	5.80	1.82	3.83	5.95	2.10	4.05	6.22
TD	1.80	4.00	5.75	2.00	4.10	5.88	2.52	4.55	6.20
VV	1.40	3.77	5.83	2.00	3.92	6.00	2.12	4.25	6.28
RV	1.75	4.08	5.72	2.00	4.30	5.95	2.48	4.58	6.42
DT	0.97	3.40	5.80	1.63	3.63	5.95	1.92	3.92	6.13
TT	1.73	3.83	5.70	1.80	3.92	5.83	2.00	4.13	6.00
SAA	1.80	3.65	5.53	2.08	3.88	5.80	2.25	3.95	5.95
SA	1.82	3.75	5.63	2.27	4.25	5.97	2.75	4.40	6.20
BM	1.95	3.98	5.72	2.17	4.08	6.33	2.27	4.17	6.53
BM I	1.90	4.10	5.95	2.17	4.33	6.05	2.70	4.58	6.33
DA	1.85	4.22	6.17	2.10	4.55	6.35	2.23	4.75	6.40
DT	1.63	3.40	5.80	1.82	3.63	5.95	1.98	3.92	6.13
DT3	1.73	3.83	5.70	1.80	3.92	5.83	2.00	4.13	6.00
JJ	1.77	3.52	5.78	2.05	3.90	5.92	2.23	4.00	6.20
JM	1.80	3.25	5.40	1.85	3.98	5.95	2.35	4.38	5.95
Mean	**1.75**	**3.82**	**5.75**	**1.98**	**4.03**	**5.93**	**2.29**	**4.27**	**6.17**
SD	**0.20**	**0.27**	**0.22**	**0.15**	**0.27**	**0.28**	**0.24**	**0.31**	**0.29**

APPENDIX VIII (b)

ABSOLUTE LATENCY DATA FOR FEMALE PARTICIPANTS – 50 to 30 dBnHL

	Intensity 50 dB			Intensity 40 dB			Intensity 30 dB		
ID	**Wave I**	**Wave III**	**Wave V**	**Wave I**	**Wave III**	**Wave V**	**Wave I**	**Wave III**	**Wave V**
BF	3.13	5.20	6.68	3.40	5.67	7.13		5.65	7.28
EE	2.38	4.13	5.58	2.85	4.50	6.13			6.47
BC	2.48	4.58	6.03	3.17	4.97	6.42		5.40	6.88
FM	3.08	5.03	6.65	3.30	5.50	7.13	3.67	5.88	7.47
JK	2.70	4.67	6.65	3.05	4.97	7.08	3.20	5.30	7.40
JJ	2.98	5.30	6.85	3.42	5.45	7.03	3.63	5.92	7.45
JO	2.40	4.65	6.53		5.05	7.00			7.67
JOG	3.15	5.08	6.67	3.67	5.72	7.28		6.05	7.78
LO	2.65	4.55	6.67	2.88	4.95	6.97		5.50	7.45
OO	2.83	4.70	6.60	3.20	5.03	6.90	3.98	5.35	7.35
LTD	2.58	4.28	6.42	2.58	4.65	6.75	3.48	5.17	7.20
TD	2.73	4.67	6.45	2.95	5.00	6.80	3.35	5.38	7.22
VV	2.35	4.25	6.42		4.70	6.70		5.17	7.15
RV	2.77	4.83	6.47	3.10	5.15	6.80		5.60	7.55
DT	2.12	4.15	6.42	2.45	4.53	6.85			7.28
TT	2.25	4.40	6.42	2.40	4.53	7.00			7.30
SAA	2.37	4.30	6.20	2.48	4.45	6.55			6.90
SA	3.17	4.78	6.47	3.33	5.17	6.80		5.47	7.25
BM	2.52	4.70	6.63		5.15	6.95		5.83	7.38
BM I	2.80	4.65	6.40		5.22	6.95		5.38	7.35
DA	2.60	5.40	6.75			7.30			7.67
DT	2.22	4.15	6.42	2.34	4.46	6.85	2.56	5.20	7.28
DT3	2.21	4.40	6.42	2.32	4.52	7.00		5.18	7.30
JJ	2.15	4.40	6.60	3.45	5.10	7.17	3.70	6.00	8.08
JM	2.98	5.03	6.70	3.70	5.80	7.38	4.03	6.03	7.72
Mean	**2.62**	**4.65**	**6.48**	**3.00**	**5.01**	**6.92**	**3.51**	**5.55**	**7.35**
SD	**0.33**	**0.37**	**0.26**	**0.45**	**0.41**	**0.28**	**0.45**	**0.31**	**0.32**

APPENDIX IX (a)

INTER-WAVE DATA FOR FEMALE PARTICIPANTS – 80 to 60 dBnHL

	Intensity 80 dB			Intensity 70 dB			Intensity 60 dB		
ID	I-III	III-V	I-V	I-III	III-V	I-V	I-III	III-V	I-V
BF	2.20	2.13	4.32	2.25	2.10	4.35	2.20	1.97	4.17
EE	1.43	1.82	3.25	1.27	1.70	2.97	1.25	1.77	3.02
BC	2.40	1.43	3.83	2.23	1.35	3.57	2.05	1.45	3.50
FM	2.38	1.72	4.10	2.23	1.80	4.03	2.12	1.85	3.98
JK	2.00	1.95	3.95	1.98	1.95	3.93	1.95	2.20	4.15
JJ	2.27	1.85	4.13	2.33	1.63	3.95	2.30	1.63	3.93
JO	1.87	2.05	3.92	1.75	2.07	3.82	1.97	1.83	3.80
JOG	2.25	1.63	3.88	2.12	1.65	3.77	1.85	1.58	3.43
LO	1.85	2.30	4.15	2.17	2.15	4.32	1.90	2.22	4.12
OO	2.40	1.50	3.90	2.37	1.48	3.85	2.13	1.80	3.95
LTD	1.93	2.10	4.02	2.00	2.13	4.13	1.95	2.17	4.12
TD	2.20	1.75	3.95	2.10	1.78	3.88	2.02	1.65	3.68
VV	2.38	2.05	4.43	1.92	2.08	4.00	2.13	2.03	4.16
RV	2.33	1.65	3.97	2.30	1.65	3.95	2.10	1.85	3.95
DT	2.42	2.40	4.82	2.00	2.33	4.33	2.00	2.20	4.21
TT	2.10	1.88	3.98	2.12	1.90	4.03	2.13	1.88	4.00
SAA	1.85	1.88	3.73	1.80	1.92	3.72	2.00	2.00	4.21
SA	1.93	1.88	3.80	1.98	1.72	3.70	1.65	1.80	3.45
BM	2.03	1.75	3.77	1.90	2.25	4.15	1.90	2.35	4.25
BM I	2.20	1.85	4.05	2.15	1.72	3.88	1.88	1.75	3.63
DA	2.37	1.95	4.32	2.45	1.80	4.25	2.52	1.65	4.17
DT	1.77	2.40	4.17	1.81	2.32	4.13	1.94	2.21	4.15
DT3	2.10	1.88	3.98	2.12	1.90	4.03	2.13	1.88	4.00
JJ	1.75	2.25	4.00	1.85	2.02	3.88	1.77	2.20	3.97
JM	1.45	2.15	3.60	2.13	1.98	4.10	2.02	1.58	3.60
Mean	**2.07**	**1.93**	**4.00**	**2.05**	**1.90**	**3.95**	**1.99**	**1.90**	**3.90**
SD	**0.29**	**0.26**	**0.30**	**0.25**	**0.25**	**0.29**	**0.23**	**0.25**	**0.31**

APPENDIX IX (b)

INTER-WAVE DATA FOR FEMALE PARTICIPANTS – 50 to 30 dBnHL

	Intensity 50 dB			Intensity 40 dB			Intensity 30 dB		
ID	I-III	III-V	I-V	I-III	III-V	I-V	I-III	III-V	I-V
BF	2.08	1.65	3.72	2.27	1.45	3.73		1.63	
EE	1.75	1.45	3.20	1.65	1.63	3.27			
BC	2.10	1.45	3.55	1.80	1.45	3.25		1.47	
FM	1.95	1.63	3.58	2.20	1.63	3.85	2.20	1.60	3.80
JK	1.97	1.98	3.95	1.92	2.10	4.03	2.10	2.10	4.20
JJ	2.32	1.55	3.87	2.03	1.58	3.60	2.30	1.53	3.83
JO	2.25	1.88	4.13		1.95				
JOG	1.93	1.60	3.52	2.05	1.55	3.60		1.73	
LO	1.90	2.13	4.02	2.08	2.02	4.10		1.95	
OO	1.88	1.90	3.77	1.83	1.88	3.70	1.37	2.00	3.37
LTD	1.70	2.15	3.85	2.07	2.13	4.17	1.70	2.03	3.73
TD	1.95	1.78	3.73	2.05	1.80	3.85	2.02	1.85	3.87
VV	1.90	2.17	4.07		2.00			1.98	
RV	2.05	1.65	3.70	2.05	1.65	3.70		1.95	
DT	2.03	2.27	4.30	2.08	2.32	4.40			
TT	2.13	2.02	4.17	2.13		4.60		2.18	
SAA	2.03	1.90	4.30	2.08	2.32	4.40			
SA	1.60	1.70	3.30	1.85	1.63	3.47		1.78	
BM	2.18	1.92	4.10		1.80			1.55	
BM I	1.85	1.75	3.60	2.27	1.73	4.00		1.97	
DA	2.80	1.35	4.15		1.62				
DT	1.93	2.27	4.20	2.12	2.39	4.51	2.64	2.08	4.72
DT3	2.19	2.02	4.21	2.20	2.48	4.68		2.12	
JJ	2.25	2.20	4.45	1.65	2.08	3.72	2.30	2.07	4.37
JM	2.05	1.67	3.73	2.10	1.58	3.67	2.00	1.70	3.70
Mean	**2.03**	**1.84**	**3.89**	**2.02**	**1.87**	**3.92**	**2.07**	**1.86**	**3.95**
SD	**0.24**	**0.27**	**0.33**	**0.18**	**0.31**	**0.42**	**0.37**	**0.22**	**0.41**