

THE ANALYSIS OF AUDITORY EVOKED BRAIN POTENTIALS  
IN RECURVE ARCHERY

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THE ANALYSIS OF AUDITORY EVOKED BRAIN POTENTIALS  
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Approval of the Graduate School of Social Sciences

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## **ABSTRACT**

### **THE ANALYSIS OF AUDITORY EVOKED BRAIN POTENTIALS IN RECURVE ARCHERY**

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Archery can be described as a static sport requiring strength and endurance of the upper body, in particular the shoulder girdle (Mann, 1984; Mann & Littke, 1989). To get a good record in an archery competition, one requires well-balanced and highly reproducible movements during the shooting (Nishizono, 1987). The bowstring is released when audible impetus is received from a device called “clicker”. As the fall of the clicker is an acoustic stimulus, it may evoke a sequence of potentials that can be recorded from the scalp of an archer. Auditory Evoked Potentials (AEPs) occur at different latencies and with various relations to the auditory stimuli. Therefore, the present study aims at investigating the Long-latency Auditory Evoked Potentials in Recurve Archery. Research questions can be stated briefly as follows: (1) What kind of Brain Potentials are Evoked by the Event (Fall of Clicker) during Archery Shooting? (2) Is there any significant difference between the characteristics of the potentials measured in laboratory conditions and during archery shooting? (3) Is there any significant difference between the successful and unsuccessful shots in terms of Auditory Evoked

Brain Potentials? (4) Does Archery Shooting session have any effect on Auditory Evoked Brain Potentials?

The subjects of the present study were 10 non-archers (N=6 males; N=4 females) for control trials and 15 archers (N=9 males; N=6 females) for archery shooting experiments. All subjects reported normal hearing, had medical histories free of significant neurological problems, and were not taking medication known to affect brain activity. Six different control paradigms have been created. Archery shootings were performed from 18 m that is official competition distance with target face. AEBPs were recorded 200 ms before and 800 ms after the trigger (fall of the clicker) over the vertex during the shots of each subject. Paradigm 1 and 5 was conducted just before and after the archery shooting to test the effect of archery shooting on AEBPs. The hit-area is defined as the rectangle between  $(x_1, y_1)$ ,  $(x_1, y_2)$ ,  $(x_2, y_1)$ ,  $(x_2, y_2)$  and the miss-area is the outer part of the hit-area on the target face.

The preliminary analysis has shown that fall of the clicker evokes long latency auditory brain potentials with the latency of 100 msec and 200 msec. These responses are called as N1-P2 components. The means and standard deviations of both N100 and P200 amplitudes were as follows: N100 =  $27,73 \pm 16,82$ , P200 =  $-21,89 \pm 20,46$ . The latencies of given brain responses were also summarized as: N100 =  $141,93 \pm 41,46$ ; P200 =  $211,8 \pm 43,97$ . N1 amplitude was significantly different in archery shooting than that of control conditions ( $p < 0.05$ ) except for trial 3, N1 latency was significantly different than that of trial 2 – 5 ( $p < 0.05$ ). P2 amplitude is significantly different in archery shooting than that of trial 6 ( $p < 0.05$ ). However, there was no significant difference in terms of P2 latency between archery shooting and control conditions ( $p > 0.05$ ). There was no significant difference between successful and unsuccessful shots in terms of N1-P2 components ( $p > 0.05$ ). An archery shooting session did not create any difference between these components recorded before and after the shot ( $p > 0.05$ ).

Having higher N1 amplitudes during archery shooting can be explained by the known multi-component structure of this wave. Different lobes and regions of the brain can be

active during the time of the scalp-recorded N1 and simultaneous involvement of several of these areas may be contributing to the electrical field recorded at scalp in the archery shooting paradigm.

Keywords: Archery, Auditory Evoked Potentials, Brain, Clicker

## ÖZ

### OKÇULUKTA İŞİTSEL UYARILMIŞ BEYİN POTANSİYELLERİNİN İNCELENMESİ

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Okçuluk, üst vücut bölgesinde ve özellikle de omuz çevresinde kuvvet ve dayanıklılığa ihtiyaç duyulan statik bir spordur (Mann, 1984; Mann & Littke, 1989). Bir okçuluk yarışmasında yüksek puan atabilmek için dengeli ve tekrar edilebilir hareketler ortaya konulmalıdır (Nishizono, 1987). Kiriş, “klikır” adı verilen bir cihazdan uyarana geldiği zaman bırakılır. Klikır’ın düşmesi işitsel bir uyarana neden olması sebebiyle bir takım beyin potansiyeli uyarması mümkündür. İşitsel Uyarılmış Beyin Potansiyelleri (İUBP) belli gecikmelerle ve işitsel uyarana bir takım ilişkiler sergileyerek oluşurlar. Bu nedenle mevcut araştırma da okçulukta uzun gecikmeli İşitsel Uyarılmış Beyin Potansiyellerinin incelenmesi amaçlanmıştır. Araştırma soruları şu şekilde sıralanmıştır: (1) Klikır uyarani ile ne tür beyin potansiyelleri uyarılmaktadır? (2) Laboratuvar ortamı ve ok atışı sırasında ölçülen beyin potansiyelleri arasında fark var mıdır? (3) Başarılı ve başarısız atışlar karşılaştırıldığında İşitsel Uyarılmış Beyin Potansiyellerinde bir farklılık

oluşmaktadırmı? (4) Bir antrenman birimi İşitsel Uyarılmış Beyin Potansiyelleri üzerinde bir etkiye sahipmidir?

Bu araştırmaya control denemeleri için okçu olmayan 10 kişi (N=6 erkek; N=4 bayan) ve ok atışı denemeleri için aktif okçuluk yapan 15 kişi (N=9 erkek; N=6 bayan) katılmıştır. Denekler, normal işitme özelliklerine sahip olan, nörolojik sebeplerle ve beyin aktiviteleri üzerinde etkisi olan herhangi bir ilaç kullanmayan kişilerden oluşmuştur. Altı farklı control paradigması oluşturulmuştur. Ok atışları ise resmi yarışma mesafesi olan 18 m'den hedef kağıtlı olarak yapılmıştır. İUBP 200 ms uyarın öncesi ve 800 ms uyarın sonrası olmak üzere toplam 1000 ms olarak atış sırasında kafatası üzerinden kaydedilmiştir. 1 ve 5. paradigmlar atış antrenmanının etkisini araştırmak amacıyla atış öncesi ve sonrası kaydedilmiştir. İsbetli atışlar  $(x_1, y_1)$ ,  $(x_1, y_2)$ ,  $(x_2, y_1)$ ,  $(x_2, y_2)$  dikdörtgeni içinde kalan atışlardan oluşurken bu dikdörtgenin dışında kalan atışlar isabetsiz atışlar olarak sınıflandırılmıştır.

Ön ölçümler, klikir'ın düşüşünün 100 ve 200 ms gecikmelerle ortaya çıkan uzun gecikmeli beyin potansiyellerini uyardığını göstermiştir. Bu tepkiler N1-P2 bileşeni olarak tanımlanmaktadır. Bu bileşenlerin genlikleri; N100=  $27,73 \pm 16,82$ , P200=  $-21,89 \pm 20,46$  ve gecikmeleri ise; N100=  $141,93 \pm 41,46$ ; P200=  $211,8 \pm 43,97$  şeklinde ortaya çıkmıştır. Ok atışı sırasında ölçülen genlikler ile 3. deneme hariç tüm denemeler arasında N1 genliği bakımından anlamlı farklılık gözlenmiştir ( $p < 0.05$ ). N1 gecikmesi ile de sadece 2 ve 5. denemeler arasında farklılık gözlenmiştir ( $p < 0.05$ ). Ok atışı sırasında ki P2 genliği ile sadece 6. deneme arasında anlamlı farklılık gözlenirken ( $p < 0.05$ ), P2 gecikmesi konusunda ok atışı ile hiçbir deneme arasında anlamlı fark gözlenmemiştir ( $p > 0.05$ ). N1-P2 bileşelerinin genlik ve gecikmeleri konusunda isabetli ve isabetsiz atışlar arasında anlamlı farka rastlanmamıştır ( $p > 0.05$ ). Ayrıca atış antrenmanının beyin potansiyelleri üzerinde bir etki yaratmadığı gözlenmiştir ( $p > 0.05$ ).

Ok atışı sırasında daha yüksek genlikli potansiyellerin ölçülmesi atış sırasında bir çok farklı beyin bölgesinin aktive edilmesi ile açıklanabilir. Farklı beyin lobları ve bölgelerinden birkaç tanesi aynı anda aktif hale getirilmiş olabilir ve bu nedenle de ok

atışı sırasında N1 genliğinde laboratuvar ortamındakinden daha yüksek bir değere ulaşılmış olabilir.

Anahtar Kelimeler: Okçuluk, İşitsel Uyarılmış Beyin Potansiyelleri, Klikır

To My wife Nihan and  
My son Ufuk Tan

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# CHAPTER I

## INTRODUCTION

### 1.1. Background of the Study

Archery can be described as a static sport requiring strength and endurance of the upper body, in particular the shoulder girdle (Mann, 1984; Mann & Littke, 1989). To get a good record in an archery competition, one requires well-balanced and highly reproducible movements during the shooting (Nishizono, 1987). Both elite and beginner archers use equipment of the same general quality; differences between their performances must be highly dependent upon their own controlled actions (Stuart, 1990). An archer pushes the bow with an extended arm, which is statically held in the direction of the target, while the other arm exerts a dynamic pulling of the bowstring from the beginning of the drawing phase, until the release is dynamically executed (Leroyer et al., 1993). The release phase must be well balanced and highly reproducible to achieve commendable results in a competition (Nishizono, 1987).

The bowstring is released when audible impetus is received from a device called “clicker”. Each arrow can be drawn to an exact distance and a release can be obtained and maintained by this device. The clicker is reputed to improve the archer’s score and is used by all target archers (Leroyer et al., 1993). The archer should react to the clicker as quickly as possible, and synchronize the muscle activity of the whole body to attain eventual optimal accuracy. In particular, there should be a repeated contraction and relaxation of archery specific muscle groups during archery training and competitions according to the high number of arrows. When the clicker signal is heard, the archer relaxes the flexor group muscles of the forearm and actively contracts the extensor group muscles for producing the release (Ertan et al., 2003; Hennesy et al., 1990; Clarys et al., 1990; Nishizono et al., 1987).

As the fall of the clicker is an acoustic stimulus, it evokes a sequence of potentials that can be recorded from the scalp of an archer using computer-averaging technique. Auditory Evoked Potentials (AEPs) occur at different latencies and with various relations to the auditory stimuli. The most commonly used classification of AEPs is based upon the latency of the response and upon the response's temporal relationship to the auditory stimulus. The latency of response is loosely categorized as first, fast, middle, slow, or late. Transient responses occur following a change in a stimulus. Sustained responses occur throughout the duration of a continuing stimulus. Steady-state responses are evoked or "driven" by rapidly repeating stimuli. AEPs can also be classified as "exogenous" or "endogenous". Exogenous EPs are determined by physical characteristics of a stimulus, whereas endogenous EPs are determined by the physiological significance of the stimulus to the subject (Picton, 1982).

Analysis of the auditory stimulus (the fall of the clicker) and the response (releasing the bowstring) may enhance the current knowledge on archery by measuring the AEPs. The factors affecting the latency, amplitude, and scalp distribution of the archery response will also enlighten the understanding of archery. Therefore, the present study aims to investigate the Long-latency Auditory Evoked Potentials in Recurve Archery.

## **1.2. Purpose of the Study**

This study is conducted in order to analyze the Long-latency Auditory Evoked Brain Potentials in Recurve Archery.

Research questions can be stated briefly as follows:

1. What kind of Brain Potentials are Evoked by the Event (Fall of Clicker) during Archery Shooting?
2. Is there any significant difference between the characteristics of the potentials measured in laboratory conditions and during archery shooting?

3. Is there any significant difference between the successful and unsuccessful shots in terms of Auditory Evoked Brain Potentials?
4. Does Archery Shooting session have any effect on Auditory Evoked Brain Potentials?

### **1.3. Significance of the Study**

An archer is supposed to react to the stimulus with optimal accuracy. In the stimulus-response chain, processing of the click sound should play an important role. Researchers will be able to study the time gap between the stimulus (fall of the clicker) and the brain's response (i.e., accomplishment of certain phase of sound processing of the click) in achieving different scores according to Federation International Tir el'Arc (FITA). This will enhance the understanding of archery response by both scientists and coaches in the field. Scientists may have chance to make further researches on the basis of the current project. On the other hand, coaches may improve the technical qualifications of their archers accordingly. Thus, the findings of the current project will possibly add new theories to the scientific arena. It is therefore expected that the coaches make use of the results of this basic research.

The neurophysiological findings related with AEPs are general neurophysiological findings, which are not specific to archery. An archer shoots about 400 arrows during a competition, which means that the athlete responds to the auditory stimulus 400 times. Moreover, they train about 6 days/week and shoot about 200 arrows in a single training session. This means that, they also train their responses to the specific stimuli. There is lack of knowledge in the literature related with the characteristics of the archers' responses to the fall of the clicker. This archery specific and well-trained response should be defined to clarify the unknown part of archery shooting.

As it is mentioned before, there is a device called 'clicker' that causes an audible stimulus and it is used by all of the recurve archers. Hence, this stimulus most

probably evokes some longer-latency brain potentials in an archer's brain. Most of the late potentials are known to be related to the psychological significance of the stimulus to the subject, and are thus named as endogenous. Changing the intensity and frequency of the stimulus may significantly alter the latency and amplitude of the response. Besides, increasing or decreasing arousal, attention, and anxiety of the subject may alter the endogenous components. All these findings would help to come over trait and state anxiety in order to teach to archers how to calm or relax themselves. Moreover, these results may supply some more effective motivation, concentration and attention methods related with archery shooting.

As it is widely accepted, there are some archers having some problems with the fall of the clicker. First group of problems is to be not able to pull the arrow beyond the clicker in the given time gap and the second one is not to be able to appropriately respond to the fall of the clicker. Archers who are having these kinds of clicker related problems may have different types of brain responses in terms of latency and amplitude than that of normal archers not having clicker related problems. Differentiating the both group of archers in terms of brain neural activity during archery specific response may also help to cure the archers with clicker phobia.

#### **1.4. Definitions of Terms**

*Federation International Tir el'Arc (FITA)*: International Archery Federation is the governing body of the all archery activities world wide.

*Clicker*: A spring-loaded lever that produces an audible impetus to the archer that the arrow has been drawn to a fixed distance.

## **CHAPTER II**

### **REVIEW OF LITERATURE**

In this chapter, the research literature relevant to the purpose of this study is presented. The sport archery is defined from different aspects like, muscular contraction strategies, heart rate changes during archery shooting and classification of archery skill in order to give general view of sport archery. Besides, studies in general on Event Related Brain Potentials and more specifically on attention related components of Auditory Evoked Brain Potentials.

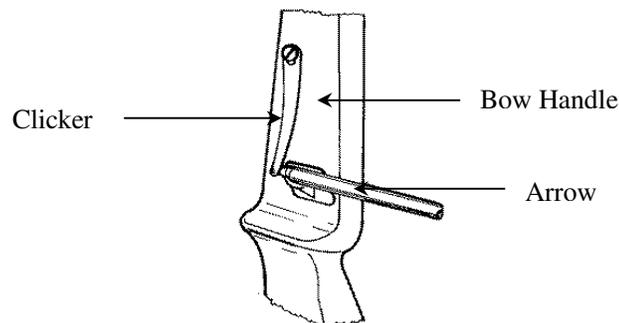
#### **2.1. Archery**

Although archery does not appear to be very fitness demanding, when closely examined, both training and competition do demand a certain extent of long hours lasting of concentration with some ability of strength, endurance, and postural fine control. During a national or an international competition, archer is forced to shoot over 75 shots in a day, where a female archer is to apply approximately 15-16 kg and male 18-20 kg of force each time the bow is pulled. This sums up to at least 1125-1200 kg for females and 1350-1500 kg of force applied in a single day competition in an intermittent manner against an opponent under very stressful situation. It, therefore, is very demanding event on certain musculature and abilities to perform well under every possible environmental conditions when performed for indoors or outdoors, providing that everything is equal (Açıkada et al., 2004).

The sport archery is described as a static sport requiring strength and endurance of the upper body, in particular the shoulder girdle (Mann, 1994; Mann & Littke, 1989; Ertan et al., 2005). Skill in archery is defined as the ability to shoot an arrow at a given target with accuracy (Leroyer, 1993; Ertan et al., 1996). Shooting an arrow to a given target can be considered as a motor skill that is directed to hitting the center of

the target. It needs a relatively permanent change in the performance of arrow shot resulting from practice or past experience. Arrow shot includes some specific movement patterns. These patterns occur in the same sequence all the time. Archer inserts the arrow into the bow, holds the bowstring and the grip, starts drawing, reaches a full draw position, aims to the target, releases the bowstring, and performs a follow-through phase. The important thing is that movements of the bow arm and the drawing arm should be performed simultaneously and the strength of the both arms should be equal to each other (Kamei et al., 1971).

The bowstring is released when an audible stimulus is received from a device called “clicker” that is used as a draw length check (Leroyer, 1993). Each arrow can be drawn to an exact distance and a standard release can be obtained using this device (Figure 1). The clicker is reputed to improve the archer’s score and is used by all target archers. The archer should react to the clicker as quickly as possible, and synchronize the muscle activity of the whole body to attain eventual optimal accuracy. In particular, there should be a repeated contraction and relaxation in the back, shoulder, arm, forearm, and pull finger muscles during archery training and competitions according to the high number of arrows. That is why the movements in archery are suitable for studying the motor control and skill acquiring processes. So, the purpose of this review is to make summary of the findings of the previous studies related with the muscular activation patterns in different muscular groups having specific participation in the whole shooting movement.



**Figure 1.** Clicker; a spring-loaded lever that produces an audible impetus to the archer that the arrow has been drawn to a fixed distance.

If someone wants to make permanent changes in archery shooting techniques, he/she should define stable stages of arrow shot. In the literature, some of the researchers describe the shot as a three-phase movement: the stance, the arming, and the sighting (Leroyer, 1993; Pekalski, 1990; Martin et al., 1990). Alternatively, Nishizono (1987) divides the stages of a shot into six: bow hold, drawing, full draw, aiming, release, and follow through. Each of these phases represents a stable sequence of the collective movement and are suitable for studying the motor control and skill-acquired.

The archer should coordinate the whole muscles involved in archery shooting movement in a short time span. As it is mentioned before, the clicker is used in target archery. The archer should release the bowstring as soon as possible after receiving the stimulus from the fall of the clicker to reach an optimal accuracy.

### **2.1.2. Classification of Archery Skill**

During the drawing phase, an archer pushes the bow with extended arm and pulls the bowstring with the other arm. He/she places the bowstring on his/her face (the tip of the nose, the lips, and the chin) by reaching the final position of drawing phase. In the full draw position; archer should accomplish many tasks at the same time (Landers et al., 1994; Leroyer, 1993; Ertan et al., 2003). He/she should both aim to the target and release the bowstring without disturbing the aiming position. So, the release phase must be well balanced and highly reproducible to achieve commendable results in an archery competition (Landers et al., 1985; Stuart and Atha, 1990; Keast & Alliot, 1990; Ertan et al., 1999).

In the classification of archery skill, one should look at first precision of archery shooting movement. Archery can be classified as a fine motor skill. Because archery shooting skill requires the use of small muscle groups in order to accomplish the shooting or releasing the bowstring. The archer is supposed to react to an auditory

stimulus from the fall of the clicker by coordinating the forearm muscles. He/she contracts the extensor and relaxes the flexor muscles in the forearm to accurately release the bowstring. Besides, there should be a balance between the shoulder and back muscles. So, the archer can produce a pull-push balance in between the drawing and bow arms to achieve commendable results in archery. Thus, Archery is placed in fine ability part of the precision continuum.

The second classification approach for any motor skill is the distinctiveness of the actual movement patterns. As mentioned before the distinctiveness is based on the beginning and end points of a particular skill. Archery shooting skill falls on the discrete part of the continuum, as having definite beginning and end points. There is an obvious sequence among the shooting stages. It starts with holding and ends with release or follow-through movements. So, having fixed sequence or starting and end points of the movements involved in archery shooting, the sport archery is classified as discrete.

The final classification of any motor skill is made according to its stability. Skills, in which the environment is stable, are categorized as closed or self-paced motor skills. In other words, the individual participating in any motor activity is in control of initiating the response or movement. In archery, archers are free to shoot the arrows in a given time span and there is no opponent or any environmental factor directly effecting the shooting movement. So, archery can also be classified as closed or self-paced motor skill. However, the weather conditions like strong wind, rain etc. can be considered as environmental factors effecting the archers' decision. But these are not totally affecting the archer's initiating the drawing or release movements (Açıkada et al., 2004).

It is concluded that archery shooting movement can be classified as fine, discrete, and closed or self-paced motor skill.

### **2.1.3. Heart Rate Changes during Archery Shooting**

One of the most consistent psycho physiological measures have been beat by beat in HR during the preparatory, shooting, and follow through phases in archery. HR has been considered an important psycho physiological indicant of attention. In relating HR to attention, Lacey (1967) suggested the intake-rejection hypothesis, which proposes that in situations where attention should be paid to the external environment (e.g., watching a flash light), HR should decrease. Although investigators generally agree that HR changes in a few moments prior to response execution index attentional processes, there is far less agreement concerning the mechanisms underlying this intake-rejection hypothesis (Landers et al., 1994).

Several studies have investigated the relationship between HR declaration and attention in archery. A case study of an elite female archer was conducted to gain insight into individual psychophysical reactions accompanying an athletic event, and to test predictions of pre-performance emotions effects upon performance. Good performance was expected when the actual pre-performance emotions resembled the recalled optimal emotion pattern. Conversely, poor performance was expected when the actual pre-performance emotions paralleled the recalled ineffective emotion pattern (Robazza et al., 1999). The research was accomplished during the 1996 European Archery Championships, one of the most important international archery competitions. An 18-year-old female athlete of the Italian archery national team volunteered to the study. Emotions, heart rate, and performance were monitored across the five days of practice and competition. They found that HR decreased by the initiation of the drawing of the bowstring.

Caterini et al. (1993) conducted a study involving 7 archers to search for HR changes during the archery shooting. They found that before the shooting or during the concentration phase HR was decreased and during the shooting phase the HR was increased compared to resting HR values. Besides, Salazar et al. (1990) have also reported an increase in HR values during the shooting. The increase was from 100

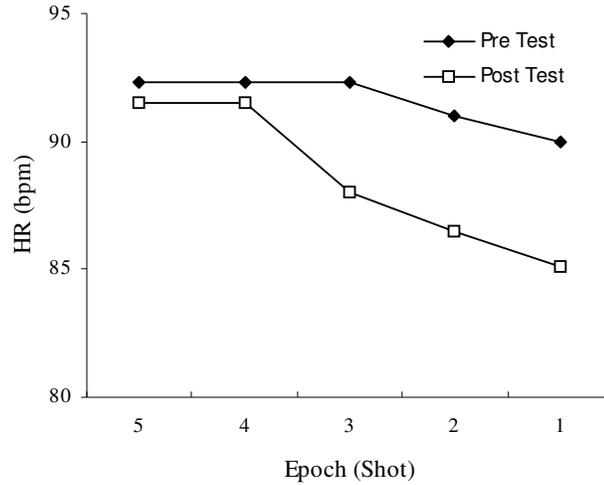
bpm to 104 bpm. Tinazci (2001) had almost similar results with Salazar et al. (1990). He divided subjects as male and female and reported increase in HR after the release in both subject groups. However, the increase in both subject groups was not statistically significant. (Female: HR during shooting =  $117,65 \pm 16,67$  and HR after release =  $119,13 \pm 13,63$ ; Male: HR during shooting =  $101,69 \pm 9,50$  and HR after release =  $102,63 \pm 9,26$ ) (Table 1).

**Table 1.** Heart rate changes during shooting and after release among elite archers.

Male		Female	
During Shooting	After Release	During Shooting	After Release
$117,65 \pm 16,67$	$119,13 \pm 13,63$	$101,69 \pm 9,50$	$102,63 \pm 9,26$

Source: Tinazci, 2000.

Landers et al. (1994) involved, who were right handed 5 male and 6 female students enrolled in a 15 week beginning archery class, in their study. Subjects were tested at week 2 and again at week 14 after they had received 27 sessions of archery training. The pre and post test findings revealed that HR deceleration was significant only during the post-test. Means were the post test were 91.5 bpm at Epoch 5 and 85.1 bpm at Epoch 1 (Figure 1). With practice, archers made significant improvements (62%) in performance from week 2 to week 14. The psycho physiological changes from pre to post-test were characteristic of what would theoretically be expected if archers were to develop their attentional skills over the course of training. According to the environmental “intake” part of Lacey’s hypothesis, HR should decelerate as one focuses more attention on the external environment.



Source: Landers et. al., 1994

**Figure 2.** Heart Rate (bpm) changes at pre- and post-test from epoch 5 to epoch 1 before arrow release.

Although somatic activity was considered to be constant from pre to post-test, HR deceleration was only found during the post-test. This 6-bpm change within 3 s prior to arrow release was within the range found in other sport studies where pre-elite and elite athletes have been studied (Landers et al., 1994).

## **2.2. Event-related Potentials**

Event-related potentials (ERPs) are voltage fluctuations that are associated in time with some physical or mental occurrence. These potentials can be recorded from the human scalp and extracted from the ongoing electroencephalogram (EEG) by means of filtering and signal averaging. Although ERPs can be evaluated in both frequency and time domains, these particular guidelines are concerned with ERPs recorded in the time domain, that is, as waveforms that plot the change in voltage as a function of time. These waveforms contain components that span a continuum between the exogenous potentials (obligatory responses determined by the physical characteristics of the eliciting event in the external world) and the endogenous potentials (manifestations of information processing in the brain that may or may not be invoked by the eliciting event). Because the temporal resolution of these measurements is on the order of milliseconds, ERPs can accurately measure when processing activities take place in the human brain. The spatial resolution of ERP measurements is limited both by theory and by our present technology, but multi-channel recordings can allow us to estimate the intra-cerebral locations of these cerebral processes. The temporal and spatial information provided by ERPs may be used in many different research programs, with goals that range from understanding how the brain implements the mind to making specific diagnoses in medicine or psychology (Picton et, al., 2000).

### **2.2.1. The generation of the ERP**

It is generally accepted that the ERP reflects activity originating within the brain. However, the relationship between what is going on in the brain and what we observe at the scalp is not completely understood. Nevertheless, the following points appear to be clear. First, ERPs recorded from the scalp represent net electrical fields associated with the activity of sizeable populations of neurons. Second, and relatedly, the individual neurons that comprise such a population must be synchronously active, and have a certain geometric configuration, if they are to produce fields that

can be measured at the scalp. In particular, the neurons must be configured in such a way that their individual electrical fields summate to yield a dipolar field (a field with positive and negative charges between which current flows). Such configurations are known as “open fields” and usually involve the alignment of neurons in a parallel orientation. Finally, biophysical and neurophysiological considerations strongly suggest that scalp-recorded ERP waveforms are principally a reflection of post-synaptic (dendritic) potentials, rather than of axonal action potentials (Allison et al. 1986).

Consideration of the neural processes that we probably detect in the ERP has important consequences for their interpretation. First, there is undoubtedly much neural activity that is never apparent at the scalp. In many neuronal populations, even those with an “open field” configuration, activity might be insufficiently synchronous to generate an electrical field that can be recorded at a distance. In some structures, such as the cerebral cortex, the geometric arrangement of neurons is conducive to the summation and propagation of their electrical activity because the neurons share the same orientation, perpendicular to the cortical surface. However, in other structures, such as the thalamus, the arrangement of neurons almost certainly guarantees their invisibility to distant recording electrodes. They are arranged in such a way as to produce no detectable field outside them.

The resultant selectivity of the ERP is both an advantage and a disadvantage. If we observed the totality of brain activity at the scalp, the resultant measures arguably would be so complex as to be difficult or impossible to analyse. However, we need always to be aware that there are almost certainly numerous functionally important neural processes that cannot be detected using the ERP.

### **2.2.2. Effects of stimulus repetition on ERPs**

The late ERPs are sensitive to stimulus repetition. Especially, the vertex negativity (N1) and the N1-P2 amplitude (difference between the N1 and P2 peak amplitudes)

as well as the P3 diminish with stimulus repetition (Picton *et al.*, 1976). The late ERPs to the first stimulus in a train are large in amplitude and diminish rapidly with repetition, reaching a low asymptotic level after a few stimulus presentations. The decrease of ERPs is faster and more pronounced with faster stimulus presentation rates (Angel *et al.*, 1985; Fruhstorfer *et al.*, 1970). The ERP components differ from each other in sensitivity to this rate effect. In general, the longer the latency of a component, the more sensitive it is to the rate effect. For example, in the work of Tomberg *et al.* (1989), the somatosensory N140 totally disappeared when the inter-stimulus interval (ISI) was shortened from 2500 ms to 1400 ms. Simultaneously, the early components also decreased in amplitude but were still clearly discernible when the ISI was 450 ms. Only the first somatosensory cortical component N200 did not change with these ISIs.

Decrement in ERPs with stimulus repetition is not a consequence of sensory adaptation or fatigue in the receptors afferent pathway (except with very fast stimulus rates, hundreds stimuli/s) for the first cortical response is fully recovered with ISIs longer than 200 ms (Huttunen and Homberg, 1991; McLaughlin and Kelly, 1993). This is concordant with the results of Ibáñez *et al.* (1995) according to which the regional cerebral blood flow (rCBF) increases in the primary somatosensory area (SI) linearly with the stimulus presentation frequency up to the 4 Hz but not with faster rates (>8 Hz). Obviously, the primary cortical areas, in spite of stimulus repetition, receive accurate stimulus information which is available there some time for further processing if needed. This is in a good agreement with the fact that the subjective intensities of the evoked sensations do not depend on changes in ERPs with stimulus repetition (Chapman *et al.*, 1981). The amplitude decrease of the ERPs begins with too long ISIs to be explainable by refractory periods in simple cellular mechanisms (Näätänen and Picton, 1987). In the somatosensory systems, the ERP amplitude decrement is probably caused by complex inhibitory mechanisms within the parietal cortex that reduce the excitatory postsynaptic potentials (see Whitsel *et al.*, 1989; 1991).

Late ERPs increase in amplitude with the prolongation of ISI. Auditory N1, P2, and P3 and somatosensory N140, P200, and P300 components linearly increase in amplitude as a function of the ISI (Miltner *et al.*, 1991). The full recovery of the N1 requires about 10 s (Davis *et al.*, 1966; Fruhstorfer *et al.*, 1970; Näätänen, 1988; Ritter *et al.*, 1968). Interestingly, the human auditory sensory memory trace also persists about 10 s (Cowan, 1984; 1988; Cowan *et al.*, 1993; Lu *et al.*, 1992; Sams *et al.*, 1993). The enhancement of the late ERPs, especially the N1 and P3 components, to the first stimulus is often associated to the initial orienting reaction (I-OR) (Kenemans *et al.*, 1989; Näätänen and Gaillard, 1983). The very first stimulus in any series after a long ‘silent’ period probably catches attention and it elicits a large N1 which is followed by the large P3 (P3a), indicating the occurrence of the attention switch (Alho *et al.*, 1998; Snyder and Hillyard, 1976; Squires *et al.*, 1975), and then the full-scale classical orienting reaction (OR) (see Sokolov, 1975) occurs with its autonomic-nervous system responses (Lyytinen *et al.*, 1992; Lyytinen and Näätänen, 1987).

### **2.2.3. Effects of stimulus change on ERPs**

In the auditory system, an occasional change in a continuous flow of stimuli elicits a negative shift in ERP beginning at about 100 ms and lasting 100-200 ms. This mismatch negativity (MMN) reflects the detection of stimulus change in the nervous system (Näätänen *et al.*, 1978). This “enhancement” of negativity resembles the changes in ERPs to the first stimulus in stimulus series or to deviant stimuli presented rarely alone without standards (Fruhstorfer *et al.*, 1970 and Näätänen *et al.*, 1989). Attention or change in the direction of attention is an essential part in the orienting reaction (OR). Any supraliminal change in auditory stimulus trains elicits an MMN and it can trigger the change-orienting response (C-OR) (Näätänen and Gaillard, 1983), but it does not necessarily do so (Lyytinen *et al.*, 1992). The MMN is independent of attention and is elicited irrespective of whether the subject is attending or ignoring the deviant stimuli (Alho *et al.*, 1992; Näätänen, 1986;

Näätänen *et al.*, 1978). Some studies have, however, shown that attention could have effect on the MMN (Alho *et al.*, 1992; Paavilainen *et al.*, 1993; Trejo *et al.*, 1995).

In auditory ERPs, the responses to either the first stimuli in stimulus trains or to deviants among standards are enhanced compared with the responses elicited by the other subsequent or standard stimuli in the train, respectively. The initial response is mainly unspecific and is elicited by any first stimulus after a long silent period. On the contrary, an MMN is elicited by any supraliminal change (deviation from the standard stimulus) in auditory stimulus trains (Näätänen, 1992). Both responses rapidly attenuate with stimulus/deviant stimulus repetition (Sams *et al.*, 1984). On the other hand, neither the first auditory stimulus in a sequence (Sams *et al.*, 1985b) nor infrequent stimuli presented without standard stimuli elicit an MMN (Näätänen *et al.*, 1989; Sams *et al.*, 1985a).

In auditory passive or ignore oddball conditions, in which the attention of subject is directed away from stimuli, rare deviant stimuli among frequently presented standard stimuli elicit an MMN. It is a second (N2 sometimes N2a) late negative deflection (after the N1) and overlapped by the N1. In active oddball situations, i.e. when subject have to discriminate rare deviants among frequently presented standards, deviant (target) stimuli elicit an MMN and, in addition, a large negative N2b and positive P3 waves. N2b is peaking later than the MMN at 200-250 ms and is overlapped by it. In contrast to the MMN, the N2b and P3 are attention dependent, usually not occurring in ignore conditions (Näätänen *et al.*, 1982; Ritter *et al.*, 1992). N2b is usually followed by P3a, this association being quite strong (Courchesne *et al.*, 1975; Näätänen and Gaillard, 1983). N2b can, however, occur without P3a (Ritter *et al.*, 1992) for instance when discrimination was not successful (Sams *et al.*, 1985b), and vice versa P3a can occur without N2b in ignore conditions when deviants suddenly catch attention (Sams *et al.*, 1985b), suggesting different generators for these two components. Novak *et al.*, (1992a) found a sequential relationship between MMN and N2-P3b; factors that increased the onset or peak

latencies of MMN proportionately increased the latencies of the N2, P3b, and the reaction time (RT).

In the somatosensory system, a comparable late negative-positive wave complex has been obtained as a response to electric (Ito *et al.*, 1992) and tactile stimuli delivered to fingers (Kujala *et al.*, 1995). In a multitude of studies, the somatosensory N250 (or N220 or N240) is clearly discernible but, unfortunately, neither reported nor analysed. The somatosensory N250-P300 seems to behave similarly to the auditory and visual N2b-P3, occurring in active oddball or discrimination situations. However, the determinants of the somatosensory N250-P300 are still rather deficiently known.

#### **2.2.4. Signal Extraction**

ERP is defined as the combination of (1) the brain electric activity that occurs in association with the eliciting event, and (2) “noise,” which is brain activity not related to the event together with interference from non-neural sources. The signal is, to some extent, defined by experimental conditions and thus can be controlled by the experimenter. Noise appears to be random in behaviour, cannot be as easily controlled by the experimenter and interferes with observation of the signal. Both signal and noise currents are volume conducted from their regions of origin to the vicinity of the recording electrode. This adaptive model also applies to biological artefacts, such as high frequency electromyographic activity, when the artefacts are not related to the event. The additive model accounts for the effects of non-biological noise not related to the event, which can be due to factors such as thermal noise of electronic amplifiers and radiating electromagnetic fields associated with power lines and electrical equipment.

The assumption of an invariant signal implies that the population of cells which generates the signal responds in the same way to each occurrence of the eliciting event. This assumption may be reasonable one for some but not all cases. For

example, changes in degree of fatigue, adaptation, habituation or level or direction of attention of the subject can affect ERPs. Thus the same event can elicit somewhat different signals, depending upon the mental state of the subject when the event occurs. The variation may be in the amplitude and/or latency of the signal. In some cases variations in the signal may be relatively small over a wide range of experimental conditions, and so the invariant-signal additive-noise model will not be significantly violated by changes in factors such as degree of habituation, level of arousal or direction of attention. In other cases, the mental state of the subject may have a significant influence upon ERP (Ruchkin, 1988).

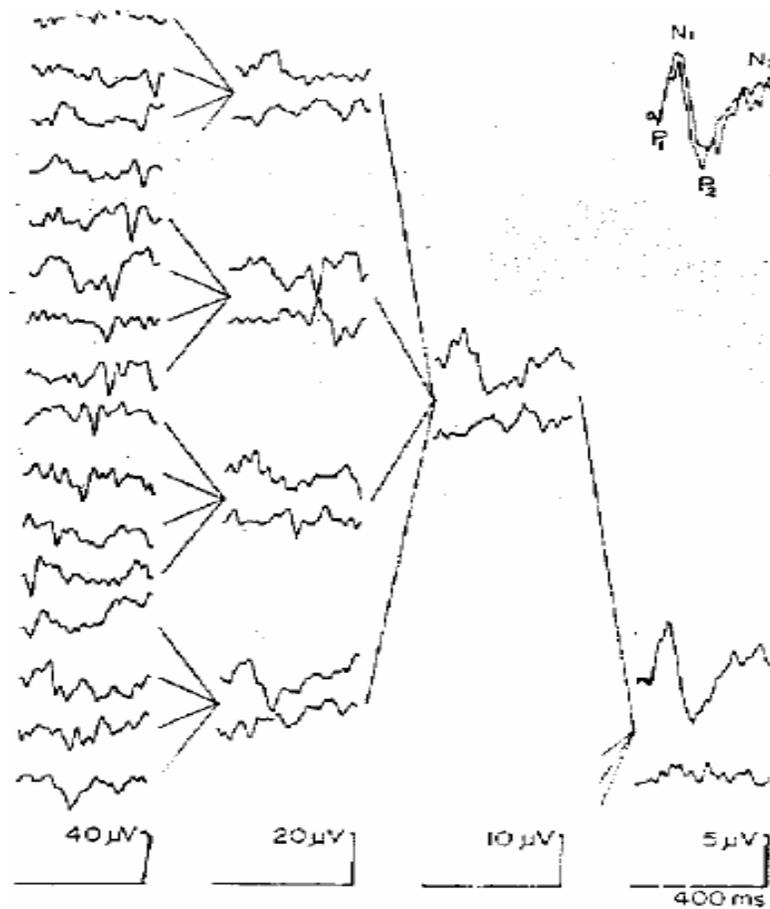
### **2.2.5. Averaging ERPs**

The purpose of signal extraction methods is attenuate interfering noise so that the signal can be clearly examined and rendered into a form suitable for further quantitative analyses. The validity of a method depends upon the model which actually applies to the data. The most convenient (simple, fast) method is averaging across a set of ERPs. Averaging is valid for the invariant-signal plus random-noise model, in the sense that it provides an unbiased estimate of the signal which improves as the number of ERPs contributing to the average increases. It may or may not be valid if there is random modulation of the signal. For such cases averaging could result in a biased, distorted estimate of the signal. The utility of averaging in such cases depends upon the objectives of the ERP analysis and the degree of signal modulation (Ruchkin, 1988).

#### **2.2.5.1. Invariant Signals**

Averaging across ERPs will attenuate the noise and produce an unbiased estimate of the signal provided that: (1) the signal is synchronized with the time of the eliciting event; (2) the signal waveform is invariant; (3) the signal and noise linearly sum together to produce the observed ERP. Averaging will attenuate the noise to a residuum that is directly proportional to the root-mean-square (rms) value of the

noise and inversely proportional to the square root of the number of ERPs in the average. This residuum is commonly referred to as the standard error of the average and is estimated, for each time point of the ERP, by dividing the standard deviation of the data at that time point by the square root of the number of trials used to compute the average (Ruchkin, 1988).



Source: Picton, 1980.

**Figure 3.** Leftmost column: 16 single trial ERPs.

Note: Second column from left: average ERPs computed across 4 trials (upper waveform of each pair) and an estimate of the noise residual (lower waveform of each pair). Second column from right: average ERP computed across 16 trials (upper waveform) and noise residual (lower waveform). Rightmost column: average ERP computed across 64 trials and noise residual.

Figure 3 illustrates the improvement of signal-to-noise ratio. Sixteen single trial ERPs are shown in the first (leftmost) column. The second, third and fourth columns show average ERPs (upper waveform of each pair) and estimates of the associated noise residuals (lower wave of each pair) for averages consisting of, respectively, 4, 16 and 64 trials. The vertical scale is adjusted from column to column so that the vertical range of the noise residuals is approximately the same in each column. Note that the amplitude of the noise residuals decreases and the average waveforms become larger in comparison with the noise residuals as the number of trials contributing to the average increases (Ruchkin, 1988).

#### **2.2.5.2. Estimation of Residual Noise**

It is often of interest to obtain a measure of the variability of the set of waveforms from which the average is computed. Such information allows a determination of the degree of reliance that can be placed upon the average wave shape. If the variability of the set of waveforms is low, then the fine detail of the average may be considered to correspond accurately to the underlying signal waveform. Conversely, if the variability is high, then, at best, only the broad outline of the underlying signal may be discerned in the average waveform. The fine detail of the average waveform, and in extreme cases the entire waveform, may be the residue of the random noise.

Schimmel (1967) has developed a method which provides a visual indication of the size and character of the residual noise. It is referred to as the “plus-minus reference method” and is based upon an invariant-signal model in which the noise samples are assumed to be statistically independent from trial to trial (Ruchkin, 1988).

#### **2.2.5.3. Further Reduction of Residual Noise – Artefacts**

An implicit assumption in computing an average waveform is that the noise samples associated with each ERP have similar statistical properties, and hence the summation process results in cancellation of the noise waves. However, occasionally

an atypically large noise wave may occur, perhaps due to an artefact such as eye movement or tensing of muscles. If such large noise waves occur more frequently the usual assumptions of Gaussian statistics indicate, then the noise reduction performance of averaging will be degraded.

One means of coping with the problem of atypically large noise waves is to set limits on the amplitude excursion that an ERP may have in order to be included in the average. A further step is to monitor sources of artefact, such as eye movements and blinking, and only include in the average those ERPs in which artefact activity is below a criterion level. For example, one way of dealing with eye movements is to record the electrooculogram (EOG) with a pair of electrodes placed above the inner canthus and below the outer canthus of one eye, and exclude from the average data from trials in which the EOG exceeds a criterion voltage (Ruchkin, 1988).

#### **2.2.6. Filtering**

Averaging is a means of reducing noise interference by selectively attenuating brain and non-neural electrical activity that is not synchronized with occurrences of eliciting events. Reduction of noise interference may also be achieved by operating upon the data with linear filters. Since linear filters selectively attenuate on the basis of frequency, linear filtering can improve the signal-to-noise ratio provided that there is sufficient separation over frequency between the signal and noise. In terms of the model of an ERP consisting of a signal and additive noise, linear filtering improves signal-to-noise ratio by selectively attenuating the additive noise (Ruchkin, 1988).

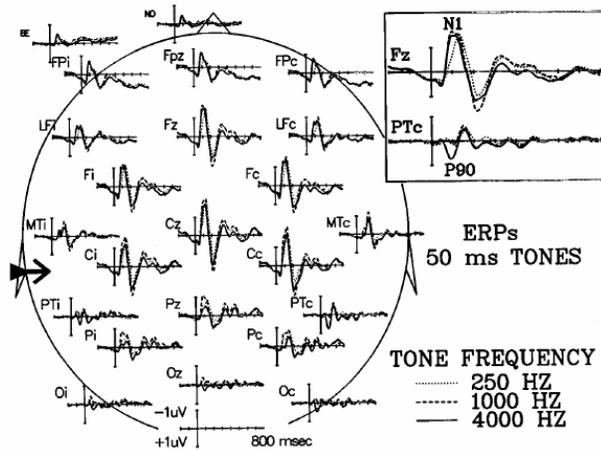
### **2.3. A Review of N1 Component**

Almost a half-century ago, P.A. Davis (1939) described the sound-evoked changes in the electroencephalogram of the waking human brain (Picton, 1988). The N1 wave, the most prominent deflection of the human auditory evoked potential, is a broad negativity over the fronto-central scalp that begins at 60-80 msec and can last until 160 msec after the onset of a sound. The N1 and the subsequent positive P2 waves constitute the ‘vertex potential’, originally thought to arise in polymodal association cortex (Davis et al. 1972; cited in Woods, 1995). As will become evident, many different processes generate negative waves during the latency of the N1 between 50 and 200 ms after the onset of an auditory stimulus. We shall distinguish the “true” or “obligatory” components that are mainly controlled by the physical and temporal features of the stimulus and by the general state of the subject. These components can be distinguished from each other by their different source locations within the brain and by their different sensitivity to stimulus features and state factors (Näätänen and Picton, 1987). The current review focuses on the generators of the ‘true N1’ wave, emphasizing the results obtained in studies performed since 1987.

#### **2.3.1. N1/N100: Classification of components**

Several different cerebral processes contribute to the N1 wave of the scalp-recorded auditory EP. These “component” processes occur in different cerebral locations and sub serve different psycho physiological functions. They are distinguished by their characteristic electrical and/or magnetic fields and by their specific relationship to various experimental manipulations (Näätänen and Picton, 1987). Figure 4 shows auditory ERPs elicited by tones of different frequencies presented at high rates in a dichotic selective attention task. The N1 wave reaches maximal amplitudes at fronto-central sites where it shows an early peak at 95-100 msec and often a second peak at 120-130 msec. It returns to baseline at 160-180 msec (Woods, 1995).

The following paragraphs describe six components that are hypothesized as contributing to the scalp-recorded N1 wave. The first three can be considered as true N1 components, whereas the other three are components that often exist in the latency region of the N1 wave but may occur independently.



Source: Woods, 1995.

**Figure 4.** Scalp distribution of ERPs.

Note: Grand mean ERPs to tone bursts of 250, 1000, and 4000 Hz averaged over attention conditions in a high-rate auditory selective attention task. Electrodes have been transposed so that those on the right of the figure were contralateral to the stimulated ear. Key: i = ipsilateral; c = contralateral. BE = below eye, FP = frontopolar, LF = lateral frontal, F = frontal, MT = mid-temporal, C = central, PT = posterior temporal, P = parietal, O = occipital. Insert: enlarged ERPs from Fz and P90 electrodes.

Wolpaw and Penry (1975) first proposed that the N1 consisted of midline and temporal components. A classification scheme based on this distinction is presented in Table 2. Midline components, including the N1'/P90 and N1b, can be modelled with tangential generators on the superior temporal plane (STP) and pointing toward the midline of the scalp. There appear to be at least two midline components distinguished by the tonotopy of their generators (Figure 5, left panel). The early frontocentral peak (N1', peak latency 90-110 msec) shows reliable tonotopic changes in distribution (Bertrand et al. 1991; Woods et al. 1991; Woods 1992; Woods et al. 1993b; cited in Woods, 1995). It is frontally distributed for high frequency tones,

centrally distributed for middle frequencies, and posteriorly distributed for low frequencies. The P90 component is seen over posterior temporal regions only for high frequency tones. The N1'/P90 subcomponent is modulated by selective attention (Woods et al. 1994). The later midline component, the N1b, peaks at 120-130 msec over fronto-central sites. It shows a similar distribution for tones of different frequency (Woods, 1995).

**Table 2.** Components of the N1 Wave

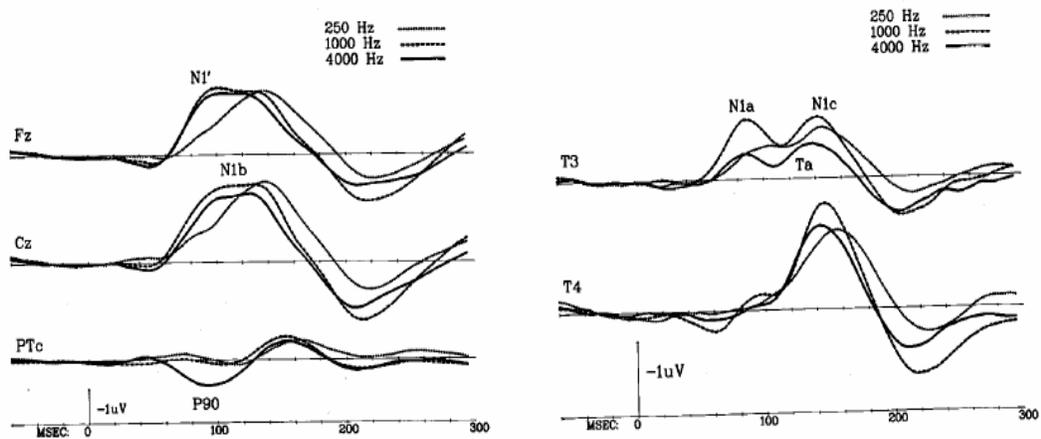
Component	Latency (msec)	Temporal integration (msec)	Refractory period (see)	Orientation	Characteristics	Possible generators
N1a	75-95	8-24	< 5	Radial	Larger in left hemisphere. Larger over hemisphere contra lateral to stimulation.	Lateral planum temporale
Ta	100-115	8-24	< 5	Radial	Notch between N1a and N1c. Larger over left hemisphere.	Lateral superior temporal plane
N1'/P90	85-110	>30; needs tone duration >8 msec	< 5	Tangential	Tonotopically organized. Larger contralateral to stimulated ear. Larger right hemisphere.	Tonotopic auditory field oriented obliquely to Heschl's gyrus
N1b	110-140	>30	> 20	Tangential	Not tonotopic. Intact in some cases of cortical deafness. Possibly polymodal.	Mesial planum temporale or posterior bank of the superior temporal sulcus
N1c(Tb)	130-170	8-24	< 5	Radial	Larger in hemisphere contralateral to stimulation. Larger in right hemisphere.	Lateral surface of posterior area 22
N1-delta	100-130	?	?	Tangential	No evidence of to no topic organization; spared in lesions of posterior aud. cortex	Mesial planum polare

Source: Woods, 1995

A list of components contributing to the N1 wave. Components are labelled after Wolpaw and Penry (1975) and McCallum and Curry (1980). Temporal integration refers to signal duration over which component amplitude changes.

Double-peaked waveforms are usually recorded at mid-temporal sites (Figure 5 right panel). These consist of an early negativity peaking at 80-85 msec, a positivity at 95-105 msec, and a later negativity peaking at 140-160 msec. These mid-temporal

negativities N1a and N1c, with the positivity between them labelled Ta, are labelled after Wolpaw and Penry (1975). The N1a is larger over the left hemisphere (McCallum and Curry 1980; Knight et al. 1988; Woods et al. 1993c; cited in Woods, 1995) while the N1c is larger over the right hemisphere (Cacace et al. 1988; cited in Woods, 1995). Both midline and lateral temporal components are enhanced in amplitude over the hemisphere contra lateral to the stimulated ear (Cacace et al. 1988; Knight et al. 1988; Woods et al. 1992; Connolly 1993; cited in Woods, 1995).



**Figure 5.** Midline components of the N1. Grand mean ERPs from Fz, Cz, and posterior temporal electrodes contralateral to the stimulated ear (PTc) showing N1'/P90 and N1b components.

Mid-temporal components of the N1. Grand mean ERPs from T3 and T4 electrodes showing N1a, Ta and N1b components tones of 250, 1000, and 4000 Hz. ERPs were averaged over ear of stimulation and attention conditions in an auditory selective attention task.

Source: Woods, 1995.

The lateral temporal components do not show tonotopic displacements, although frequency-related differences in peak latency can be noted (Figure 5). Both midline and lateral temporal components are reduced during drowsiness and sleep (Nielsen-Bohlman et al. 1991; cited in Woods, 1995).

### **2.3.1.1. Component Structure of N1 Wave**

*Component 1* is generated in the cortex of the supratemporal plane, as originally proposed by Vaughan and Ritter (1970). This component has a peak latency at 100 ms, is maximally recorded from the frontocentral scalp, and has a scalp field that is slightly greater over the hemisphere contralateral to stimulation. The justification for this component derives from the magnetic recordings (Elberling et al., 1980; Hari et al., 1980) and the scalp distribution analysis of Scherg and von Cramon (1985, 1986a).

The component is probably generated over a much wider region of the supratemporal plane than that occupied by the primary auditory cortex on Heschl's gyri. This is suggested by the finding that bilateral lesions of the temporal lobe must extend into the temporoparietal region before the N1 is abolished (Woods et al., 1987). Because of the variable orientation of the supratemporal plane among individuals, the degree of frontal spread and of left-right asymmetry will vary from one subject to another. It is possible that hemispheric asymmetries in the response may be related to asymmetries in the size and orientation of auditory cortex on the supratemporal plane.

The amplitude of this component probably changes with intensity according to the rules proposed by Bak et al. (1985) for the current dipole generating the magnetic fields that they recorded. If so, with increasing intensity the component increases in amplitude and the slope of this change decreases. Judging from the data of Hari et al. (1982), we suggest that the relative refractory period for this component is 4 s or slightly longer. It is possible that this component may be enhanced by attention through some thalamocortical gating mechanism.

*Component 2* is a biphasic component with a positive wave at about 100 ms and a negative wave at approximately 150 ms, as originally proposed by Wolpaw and Penry (1975). It is probably generated on the superior temporal gyrus and is recorded

from the scalp with maximum amplitude at the mid-temporal electrodes. The justification for this component derives from the cortical recordings of Celesia (1976) and of McCallum and Curry (1979), and from the scalp-distribution analysis of Scherg and von Cramon (1985, 1986a). This component has a radially oriented generator and therefore is not picked up magnetically.

This component would be generated in the auditory association areas, activated by connections from the primary auditory cortex and also possibly from the thalamus. The response may be larger if the association cortex is primed by some expectancy mechanism. This would explain the results of Arezzo et al. (1975) who found that the lateral surface of the temporal lobe was activated only in monkeys who had previously used auditory stimuli in behavioural tasks. Furthermore, Perrault and Picton (1984) found that the component was enhanced when subjects attended to a train of monaural stimuli compared to when the subjects ignored the stimuli.

The component is much larger, and slightly earlier, over the hemisphere contralateral to the ear of stimulation than over the ipsilateral hemisphere. There are at present no data concerning the effects of intensity or ISI on this component.

*Component 3* is a vertex negative wave with a peak latency of approximately 100 ms. The location of the generator of this component is not known. We suggest that this component is generated in the frontal motor and premotor cortex under the influence of the reticular formation and the VL nucleus of the thalamus, which projects to the precentral gyrus, to the adjacent regions of the superior, middle and inferior frontal gyri, and to the supplementary motor area on the medial surface of the frontal lobe. These areas may also receive auditory input from the auditory association cortices. Component 3 is recorded on the scalp with maximal amplitude at the vertex and the lateral central electrodes. The justification for this component derives from Hari et al. (1982), who found that at long ISIs the vertex potential increased independently of the magnetic response, and from Velasco et al. (1985; Velasco & Velasco, 1986) who found the reticular formation and the VL nucleus of the thalamus to be very

active during the auditory vertex potential. Arezzo et al. (1975) found that some areas of the monkey frontal cortex were active during the scalp-recorded N1. Hari (1983) speculated that the supplementary motor area (on the medial frontal cortex above the cingulate gyrus) may be involved in this component. Libet, Alberts, Wright, Lewis, and Feinstein (1975, their Figure 3) reported that the human auditory evoked potential recorded from the supplementary motor area in the latency region of 135-220 ms showed polarity reversal between the surface and the depth.

We cannot rule out the possibility of another generator location for this component. We doubt that the scalp recordings pick up fields with amplitudes on the order of 10  $\mu$ V that were generated in the reticular formation or thalamus, as suggested by Loveless (1983), because of the small area, the relative lack of dipole structure, and the large depth of these regions. The largest local fields recorded from sub-cortical areas by Velasco et al. (1985; Velasco & Velasco, 1986) were on the order of 50  $\mu$ V. If these fields were indeed contributing to the scalp recording one might expect local sub-cortical fields that were some hundred times larger (cf. Nunez, 1981, p. 168). It is possible that component 3 may be generated in the supratemporal plane with a dipole orientation that is tilted somewhat more posteriorly than the dipole underlying component 1 (personal communication, Michael Scherg, October, 1986). Such a tilt in the dipole may not have been recognizable in the single-channel magnetic recording of Hari et al. (1982). However, the possibility of both these components originating in the supratemporal plane is difficult to reconcile with the persistence of an N1 wave in bilateral lesions of the temporal lobe (Woods et al., 1987).

We therefore suggest that this response is the cortical projection of a reticular process that facilitates motor activity. The early work of Larsson (1956, 1960a, 1960b) related the vertex potential to components of the startle reflex. Rossignol and Jones (1976) measured the H-reflex following 110dB SPL tones presented at a rate of 1/15 s. They found an enhancement of the reflex that began at 80 ms, peaked between 110 and 130 ms, and lasted for about 200 ms. This period of enhancement may have been

mediated by descending influences from the reticular formation and the frontal cortex. Hazemann, Audin, and Lille (1975) found that the N1 wave of the auditory EP is reduced during voluntary self-paced movements. This finding further supports the relations between the N1 and the motor system. We suggest that component 3 may be largely generated in areas of the cortex mainly responsible for motor activity. This component is most easily recorded in response to auditory stimuli presented at intensities of greater than 60dB SPL and at ISIs of greater than 4-5 s. The relative refractory period for the response lasts for at least 30 s. A similar response would be elicited by intense and infrequent stimuli in other modalities and there would be definite intermodal refractory effects. Much like the startle reflex, this response would be attenuated by knowledge of the timing of the stimulus.

*Component 4* is the mismatch negativity. It is generated in the same regions of the brain that generate the first component, although probably by somewhat different neuronal processes. This MMN reflects the results of an automatic comparison between the present stimulus and those preceding it.

*Component 5* is the sensory-specific processing negativity. This begins at approximately 50- 100 ms and lasts during the processing of an attended auditory stimulus. This component is probably generated in the auditory sensory and association areas on the supratemporal plane and on the lateral aspects of the temporal lobe. Its scalp distribution may vary with the relative amounts of processing in the different areas.

*Component 6* is the “attentional supervisor” a second component of the processing negativity. This wave has a longer time span than the sensory-specific processing negativity. We propose that it is generated in the anterior frontal cortex since it receives information from the auditory association cortex and since it feeds back to these sensory areas in order to bias particular kinds of auditory processing. The justification for proposing this component derives from the scalp-distribution studies of Hansen and Hillyard (1980), which show an Nd wave, the later part of which is

more frontal than the early part, and from the results of Roland (1981, 1982) and of Roland et al. (1981), who found patterns of enhanced blood flow in the frontal lobes during attention to auditory stimuli.

### **2.3.2. Subject Factors**

#### **2.3.2.1. Temporal and Event Uncertainty**

In 1973 Schafer and Marcus reported that the EP to an auditory or visual stimulus that was triggered by the subject pressing a button was smaller than that evoked by a stimulus presented by a machine. They attributed this effect to temporal uncertainty (Klemmer, 1956), since “the subjects possessed complete foreknowledge of stimulus timing when they stimulated themselves” and “no foreknowledge when the machine delivered the stimuli randomly in time”. Furthermore, the size of this effect appeared to vary with the intelligence of the subject, a more intelligent subject showing a greater reduction in amplitude under the self-stimulation condition. The N1 reduction with time certainty probably stems from some dampening of the relatively non-specific component 3, since the N1 under time-uncertainty conditions, unlike the N1 under time-certainty conditions, was attenuated by moderate doses of ethanol and was vulnerable to other changes in state.

The self-stimulation results were corroborated and extended by McCarthy and Donchin (1976), Braff, Callaway, and Naylor (1977), and Schafer (1982). McCarthy and Donchin showed that the EPs recorded during self-stimulation contained a slow negative shift prior to the stimulus, but that the reduction in the EP with self-stimulation was not caused by any overlap with motor-related potentials. By using two different auditory stimuli, one of which occurred more frequently than the other, they were able to distinguish the effects of temporal uncertainty (when an event occurs) from event uncertainty (which event occurs). They found that the N1 was increased under conditions of either temporal or event uncertainty but that the P3

wave occurred only under conditions of event uncertainty and only in response to the more infrequent stimulus.

Schafer, Amochaev, and Russell (1981) evaluated the effect of a subject knowing when a stimulus would occur (temporal uncertainty) independently of any self-stimulation effect. Subjects were asked to press a button in response to a tone that occurred regularly every 10 s either coincident with a visually displayed counter reaching zero or without any temporal relation to the counter. The N1 amplitude was larger and the N1 latency longer in the time uncertainty condition. However, in the time-certainty condition, the task was less demanding and the subject did not need to attend to the auditory stimuli for response initiation. The results may therefore be confounded by effects of task difficulty and attention. Furthermore, considering the long intervals between the auditory stimuli, the N1 amplitudes in this study were very small, on the order of only 1-2  $\mu\text{V}$ . This suggests strong intermodal refractory effects of the visual stimuli upon the auditory N1 response (probably the non-specific component).

Wastell, Kleinman, and MacLean (1982; Wastell, 1980) have suggested that diminished temporal uncertainty may explain the reduction in the N1-P2 waves of the EP at short stimulus intervals. The idea is that it is much more difficult to predict the moment when a stimulus occurs if the ISI is long. Näätänen, Muranen, and Merisalo (1974) evaluated the ability of subjects to predict time intervals by having them press a button after an estimated duration of 0.5, 1, 2, or 4 s. For short durations, the timing of the button-presses was quite accurate, but for the 4-s duration the timing was very variable. With long ISIs the subject probably seldom experiences “peaks of expectancy” just prior to the stimulus. Time uncertainty is large when the first stimulus of a train occurs after a long inter-train interval and according to Wastell; this could explain the very large N1 to the first stimulus of a sequence—the “first stimulus effect.”

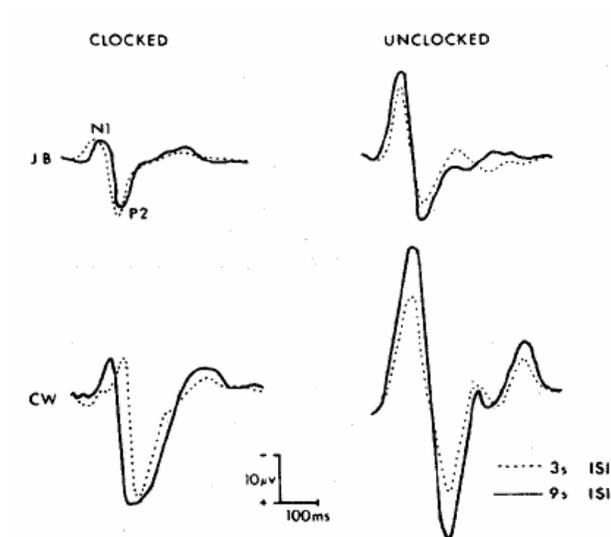
Similarly, Loveless (1983) concluded that “the temporal information content of the evoking stimuli is the critical variable underlying fast habituation as well as temporal recovery.” According to him, event uncertainty is relatively low when the subject is exposed to repeated stimuli of the same kind but it is their precise moment of occurrence that is imperfectly known. Therefore the occurrence of a stimulus resolves temporal rather than event uncertainty.

Öhman et al. (1972) reduced temporal uncertainty by switching on a red lamp about 3 s before the first stimulus of a train of clicks presented at ISIs of 3 s. Under these conditions the amplitude of the response to the first stimulus of the train was greatly reduced and was not much larger than that to the subsequent stimuli. This reduction, however, could also be explained, as mentioned earlier, by cross-modal refractory effects between the light and the first click.

In order to dissociate the effects of ISI from temporal uncertainty, Wastell (1980) provided subjects with visual cues about when an auditory stimulus could occur. A spot on an oscilloscope revolved around a circular course once every 3 s. One or three stationary spots above the rotating spot indicated whether a brief tone would occur, when the rotating spot reached the fixation point after one or three revolutions. The subjects pressed a button in response to the tone, the timing of which varied between  $\pm 150$  ms from the moment that the rotating spot reached the fixation point. In an un-clocked condition there were no visual stimuli. The N1 amplitude was significantly smaller in the clocked condition than in the un-clocked condition. Furthermore, the N1-P2 was not significantly larger when the preceding interval was 9 s rather than 3 s in the clocked condition but was so in the un-clocked condition. Sample results from 2 subjects are shown in Figure 6. The average vertex N1 amplitudes at 3- and 9-s intervals were 4.1 and 4.1  $\mu$ V, respectively, in the clocked condition, and 14.7 and 20.1  $\mu$ V in the un-clocked condition (Wastell, personal communication, December, 1985). In a “control” experiment a rotating spot was present but its cycle (6.7 s) was not related to the timing of the tone. In this condition the N1 amplitudes at the 3 and 9-s intervals were 5.6 and 9.5  $\mu$ V respectively

(Wastell, personal communication, September, 1986). These findings indicate two separate processes. First, there were significant cross-modal refractory effects causing the auditory N1 to be much smaller when there were concomitant visual stimuli. Second the results suggest that the larger N1 amplitudes at longer ISIs are actually due to increased temporal uncertainty, since when this was eliminated by the clock there was no ISI effect.

If the effects of ISI on the auditory N1 wave are due to the concomitant changes in the subject's uncertainty about the timing of a stimulus, the response to stimuli presented irregularly should be larger than the response to regularly presented stimuli. Unfortunately, the data reported in the literature are equivocal on this point. Nelson, Lassman, and Hoel (1969) found no differences in the N1-P2 amplitudes for tones presented regularly every 2 s and for tones presented at intervals between 1 and 4.5 s with a mean of 2 s. These findings were confirmed in later studies (Nelson & Lassman, 1977). The results of both studies suggest that temporal uncertainty does not necessarily increase the amplitude of the response. Rothman et al. (1970) found a small but apparently insignificant enhancement of the N1-P2 response to tones when they were presented at an irregular ISI. The amplitude of the response varied directly with the immediately preceding interval, and to a very small extent with the interval before that. They suggested that the amplitude was more affected by a prolonged recovery of excitability than by any unpredictability of timing. Öhman et al. (1972) found a small increase (about 10%) in the N1-P2 amplitude when the ISI was made irregular. Irregularity in stimulus interval may therefore increase the N1 response but not as much as might be expected if the interval effect were caused only by the subject's ability to predict stimulus timing.



Source: Näätänen and Picton, 1987.

**Figure 6.** Vertex evoked potentials for 2 typical subjects for the 3- and 9-s ISIs under “clocked” and “un-clocked” conditions. From Wastell, 1980, in Barber (Ed.), *Evoked potentials*, MTP Press.

It is, of course, essential not to change the overall stimulus rate when making the ISI irregular or random. The results of Tyberghein and Forrez (1969) have been quoted as showing that the auditory EP is larger when the stimuli are presented randomly than when presented periodically. However, their overall stimulus rate was quite different between conditions: the ISI varied between 1 and 3 s during their random condition and was constant at 1 s for their periodic condition. Furthermore, since the N1 amplitude is not linearly related to ISI, the perfectly designed protocol for irregular stimulation would use a non-uniform distribution of ISIs.

In conclusion, many studies clearly demonstrate that temporal refractoriness can affect the amplitude of the N1 component of the auditory EP independently of temporal uncertainty. The evidence supporting the effect of time uncertainty on the N1 wave derives mainly from studies of the response occurring after silent intervals of more than 3 s. It is therefore possible that refractoriness is the major determinant of the amplitude of components 1 and 2 but that both temporal uncertainty and refractoriness determine the amplitude of component 3.

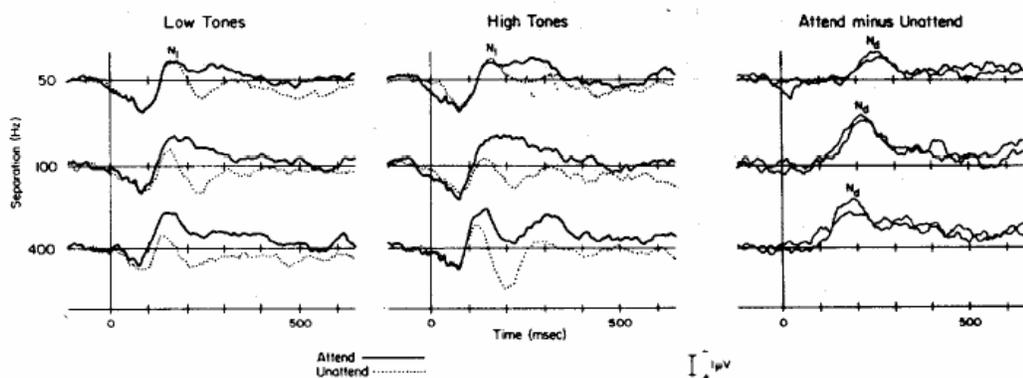
### **2.3.2.2. Selective Attention**

Several early experiments (reviewed by Näätänen, 1967, 1975) suggested that the N1 wave of the auditory EP was larger when the subject was attending to the stimuli than when ignoring them. There were two basic kinds of studies. Those belonging to the first category usually managed to demonstrate that the N1 amplitude was larger when the eliciting stimuli were attended to than when the identical stimuli were ignored in a separate experimental condition. In the second kind of study, the relevant and irrelevant stimuli were presented in the same block. Spong, Haider, and Lindsley (1965) alternated flashes and clicks at 1-s intervals. When the subject's task was to attend to the clicks and to ignore the flashes, the clicks elicited much larger EPs than those elicited by the same clicks when the subject was attending to the flashes. In turn, the flash-elicited potentials were larger when the task involved the visual stimuli. Such a paradigm, however, did not dissociate the effects of selective attention from those due to non-specific arousal. The subject could predict the timing of the stimuli to be attended and become physically more aroused before these stimuli than before the equally predictable irrelevant stimuli. Näätänen (1967, 1970b) provided evidence that arousal-related changes (a decrease in the EEG amplitude and the development of the CNV) preceded the relevant but not the irrelevant stimuli. Consistent results were obtained by Donchin and Smith (1970) and by Wilkinson and Ashby (1974). There are thus two probable effects that may increase the N1 amplitude: any prior uncertainty about stimulus timing and any prior preparation for performing a demanding task.

If, in the selective attention experiments, the timing of the attended and ignored stimuli was made unpredictable (thereby eliminating the possibility of selective prior preparation), there were no attention-related changes in the N1 (Hartley, 1970; Näätänen, 1967; Wilkinson & Ashby, 1974). However, these experiments used a rather slow rate of stimulus delivery, and it is possible that the subject was able to attend to both types of stimuli despite instructions to the contrary (Hartley, 1970). Hillyard, Hink, Schwent, and Picton (1973), following Wilkinson and Lee (1972),

increased the rate of stimulation to eliminate this possibility. Hillyard et al. found that the N1 wave of the auditory EP was increased by selective attention. Subsequent studies (reviewed by Hillyard & Picton, 1979) demonstrated that this N1 effect occurred only when the rate of stimulation was rapid (Schwent, Hillyard, & Galambos, 1976a) and when the intensity of the stimuli was not too loud (Schwent, Hillyard, & Galambos, 1976b). Schwent and Hillyard (1975) reported that the magnitude of the N1 effect varied with the amount of attentional resources allocated to different incoming stimuli.

Näätänen et al. (1978; Näätänen, Gaillard, & Mantysalo, 1980) and Näätänen and Michie (1979) pointed out that the attentional effect could be dissociated in time from the N1 wave and often extended for several hundred milliseconds beyond the N1 peak. The attentional effect could be best evaluated in the “difference waveform” obtained by subtracting the EP to stimuli being ignored from the EP to the same stimuli when they were being attended. Such a subtraction shows a broad negative wave that Näätänen and his colleagues called the “processing negativity” and Hansen and Hillyard (1980) called “Nd”. Figure 7 illustrates these difference waveforms. The processing negativity has been reviewed extensively elsewhere (Näätänen, 1982; Hillyard & Kutas, 1983). It will be considered here only as it relates to the N1 wave.



Source: Näätänen and Picton, 1987.

**Figure 7.** The processing negativity or difference wave during attention to channels characterized by different frequencies.

Note: The subject's task was to attend either to a sequence of 300 Hz (low frequency) tones or to a sequence of higher frequency tones in order to detect an occasional tone of longer duration. These waveforms represent the grand average ERPs to the shorter duration tones for 12 subjects at three different interchannel frequency separations. The tracings on the right show the difference waves between the attended and the unattended ERPs for the high tones and the low tones that are plotted on the left and centre of the figure. Recordings were taken from the vertex with negativity being represented by an upward deflection. In the difference recordings, there is an Nd wave that has a longer latency when the channels become closer together in frequency. From Hansen and Hillyard, 1980, *Electroencephalography & Clinical Neurophysiology*, 49, 277-290.

In sum, it appears that in most conditions auditory selective attention causes the superimposition on the N1 wave of a processing negativity, consisting of two components (5 and 6) that overlap the true N1 components. It is possible that under certain conditions attention may selectively enhance a true N1 component, as suggested by Hillyard et al. (1973). In that case, this enhanced component would probably be the supratemporal component (component 1).

### **2.3.2.3. States of Arousal and Levels of Performance**

Although the early attention experiments failed to establish a correlate of selective attention, they could be interpreted as suggesting that attention is accompanied by a general and non-specific increase in cerebral excitability which might increase the amplitude of the N1 wave. Näätänen (1967) studied the auditory EPs when attention was directed toward visual stimuli. An imperative flash (S2) requiring a rapid key press, was delivered randomly either 1, 2, or 3 s after a warning flash (S1). On one third of the trials an irrelevant click was presented during the S1-S2 interval so that its timing was irregular in relation to the occurrence of S1 and S2. This click elicited a larger vertex N1 than an identical control click delivered during the S2-S1 interval (11 s). Somewhat analogous results with relevant and irrelevant stimuli both in the auditory modality were obtained by Hermanutz, Cohen, and Sommer (1981). Näätänen (1967) concluded that "It might be that all kinds of stimuli, even of a completely irrelevant sense modality, elicit EPs with enhanced amplitudes, if presented during attention directed to one sense modality".

The responses to probe stimuli presented during tasks other than fore-period RT paradigms have also suggested that arousal enhances the N1 amplitude. When probe stimuli are presented during tasks such as mental arithmetic, EPs to the probe stimuli are larger than when delivered during relaxation (Eason, Aiken, White, & Lichtenstein, 1964; Eason & Dudley, 1971) or during less demanding performance (Nash & Williams, 1982). In the latter study, the N1 to auditory probe stimuli (requiring a switch press) presented under high-speed instructions in the visual primary task was larger than under moderate-speed instructions. Loveless (1977) found that the N1 response to a visual imperative stimulus of a go/no-go RT task was larger in blocks in which the auditory no-go stimulus was very loud (97dB) than in blocks in which it was soft (57dB). Arousal may not always enhance the N1, however. Schafer (1978) found no difference in the N1 amplitude to a probe stimulus (a momentary slight increase in the brightness of the TV picture) when the subjects watched a dull TV program (Dick and Jane Talk) and when they watched a program they regarded as considerably more interesting (Dick and Jane Make Love).

The N1 evoked by unattended auditory stimuli is larger at higher levels of alertness, as estimated on the basis of the pre stimulus EEG (Fruhstorfer & Bergstrom, 1969). Since the mean ISI in this experiment was 12 s and because the response was recorded between the vertex and the forehead, it is possible that the non-specific N1 component is mainly responsible for this relationship.

It is possible that the effects of arousal are mediated in the brain by the same processes that underlie the enhancement of the N1 with selective attention. Picton, Ouellette, Hamel, and Smith (1979) pointed out that it is “probably impossible to change levels of arousal in the waking state independently of any attentional change.” During heightened states of arousal subjects usually (although not always) increase their alertness or general attentiveness to the external world.

Several studies have shown that the auditory N1 amplitude is larger when the simple reaction time to the stimuli is shorter (Bostock & Jarvis, 1970; Dustman & Beck,

1965; Näätänen & Gaillard, 1974). Wilkinson and Morlock (1966), however, did not find such a correlation. Bostock and Jarvis (1970) demonstrated that the correlation between the N1 amplitude and the reaction time is largely (but not entirely) due to similar time-on-session effects on both measures.

Increasing motivation by making the amount of monetary reward dependent on performance (Wilkinson & Morlock, 1966) has resulted in enhanced N1 amplitudes and better performance, but again, it is not possible to conclude with certainty that increased arousal enhanced the N1 amplitude.

Considering all of the evidence reviewed in this section, there is some evidence for task- or attention-induced stimulus-non-specific increase in the excitability of some neuronal population contributing to the N1 deflection. This increase causes the N1 amplitude to any input, relevant or irrelevant, to be larger when the subject is engaged in some task rather than relaxing, and larger when performing a more rather than less involving task. Similarly, while performing a continuous task, if he or she can predict the moments of delivery of the relevant events above the chance level, these moments tend to be preceded and coincided by an excitability increase which causes the N1 amplitude to the relevant stimuli to be bigger than that elicited by the irrelevant stimuli.

It is possible; however, that this non-specific excitability increase should not be exclusively interpreted in terms of increased arousal but rather as also being due to a general increase in sensory sensitivity. This might be independent of arousal, since a subject who is highly aroused when attending to his or her internal thoughts may not notice events in the external world at all. It is therefore possible that the brain possesses a general gain control over its own sensory input. This is proposed, for instance, in the orienting-response theory (Sokolov, 1963) and there is strong evidence for that proposal (for a review, see Lynn, 1966). When an unpleasant stimulus is anticipated, the sensory sensitivity is decreased (“negative perception”; Lykken & Tellegen, 1974) whereas, when a pleasant, important or interesting

stimulus is expected, sensitivity is increased (“positive perception”). A similar basic assumption underlies the “directional-fractionation” hypothesis (Lacey, 1967) which proposes that the heart rate changes differentially depending on whether the subject prepares for stimulus intake (heart rate deceleration) or for stimulus rejection (acceleration).

The EP literature provides some support for interpreting the non-specific excitability increase in terms of modulation of sensory sensitivity. Young and Homer (1971) obtained smaller EPs (although there is some uncertainty as to whether the N1 was reduced) in response to affective than non-affective verbal stimuli in those subjects to whom the emotionality of the affective word was great (such as the word “rape” presented to an unmarried female by a male experimenter).

The N1 wave is susceptible to three different modulations of the general state of the individual: 1) arousal changes associated with the sleep-wakefulness dimension, with certain drugs and alcohol, with circadian rhythms, and with involvement in task performance; 2) a sensory acceptance-rejection factor which may enhance responses to all sensory inputs during expectancy for important, interesting or pleasant stimuli, and attenuate responses elicited during expectancy for irrelevant, uninteresting or unpleasant stimuli; and 3) the degree of time uncertainty with regard to the next significant stimulus. These non-specific influences probably have more effect on component 3 than on the first two components.

## **2.4. Stimulus Parameters**

### **2.4.1. Change**

The N1 potential is evoked by a relatively abrupt change in the level of energy impinging on the sensory receptors. Stimuli with very slow onsets do not elicit this response (Clynes, 1969). Sustained stimuli elicit the N1 potential only at their onset, with prolongation of the stimulus increasing the N1 amplitude only up to durations of

30-50 ms (Kodera, Hink, Yamada, & Suzuki, 1979; Onishi & Davis, 1968). There are two possible explanations for an onset response. One is that the response is generated by cerebral systems that respond specifically to the onset. The other is that the neuronal responses are sufficiently synchronized to generate a field potential only at stimulus onset, and that during the continuation of a stimulus the positive and negative potentials generated by unsynchronized neurons cancel. Such an argument is used to explain the compound nerve action potential of the auditory nerve (Elberling, 1976). This explanation could apply in part to the activity evoked in the cortex. However, the majority of cortical neurons, unlike the auditory nerve fibres, respond to the onset and not to the continuation of sensory stimuli (Goldstein, Hall, & Butterfield, 1968). We are therefore probably dealing with a true onset response rather than with some artefact of synchronization.

Gersuni (1971) suggested that the auditory system works through two different mechanisms, one with a short time constant for measuring change and time, and the other with a long time constant for evaluating pitch and intensity. The N1 appears to reflect the short time constant system. It probably indexes a cerebral system that monitors abrupt changes in sensory input and does not record the stable state-"if information about the steady or absolute level of all possible stimuli were transferred to the later stages of the system, these would soon be jammed with irrelevant and useless stores of obsolete data" (Walter, 1964, p. 338).

The response can be elicited by the offset as well as the onset of a stimulus (Davis, 1939; Davis & Zerlin, 1966). An offset response is recognizable only if the stimulus has been on for more than about 0.5 s, and the response increases in amplitude as the stimulus duration is increased (Davis & Zerlin, 1966; Hillyard & Picton, 1978; Onishi & Davis, 1968; Pfefferbaum, Buchsbaum, & Gips, 1971; Rose & Malone, 1965). For equal on-off cycles the offset response is smaller than the onset response. In this comparison, one has to consider that during the offset response there is a superimposed return to baseline of the negative "sustained potential" evoked during the continuation of a stimulus (Hillyard & Picton, 1978; Keidel, 1971; Picton,

Woods, & Proulx, 1978a, 1978b). In the auditory system the offset response has a latency that is 10-20 ms shorter than that of the onset response (Onishi & Davis, 1968). Unfortunately, it is not possible to determine from available data whether the onset and offset N1 waves have the same or different component structures, although Picton, Woods, and Proulx (1978a) showed that their midline scalp distributions are very similar.

Picton, Woods, and Proulx (1978a, 1978b) showed that the N1 response to the onset of a tone differs significantly from the sustained response to the continuation of the tone. The two parts of the response show different relations to the intensity and frequency of the tone. The sustained response is much less sensitive to decreasing ISI than the onset-evoked N1. Furthermore, the refractory effects of the N1 are more widely generalized than those of the sustained potential. Combining rapidly presented clicks with the tones significantly reduced the amplitude of the N1 evoked by tone onset but did not affect the sustained potential. These results show that the sustained potential is more stimulus specific than the N1 wave. As already suggested, the N1 wave appears to contain both stimulus-specific and stimulus-non-specific components.

The latency and amplitude of the N1 are determined by the slope of the energy change—the rise time or the fall-time. Several papers (Kodera et al., 1979; Milner, 1969; Onishi & Davis, 1968; Ruhm & Jansen, 1969) have reported that the N1-P2 amplitude of the response decreased as the rise-time or fall-time of the stimulus became longer than 3050 ms. Furthermore, as already mentioned, the amplitude of the response increased as the duration of a tone burst increased up to 30-50 ms. These times do not relate to psychophysical measurements of the temporal integration time for loudness (about 200 ms). In addition, the effects of ISI on the N1, to be reviewed later in this paper, clearly show that the N1 has little to do with loudness.

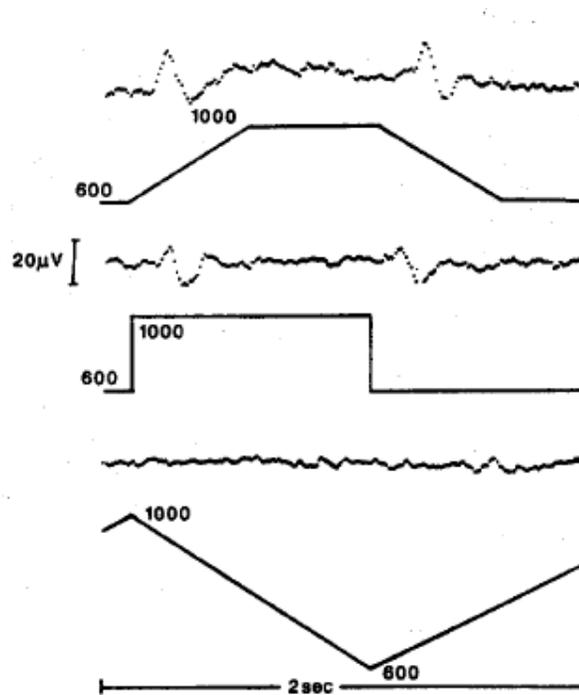
The N1 can be elicited by a change in the tonal frequency of a continuous auditory stimulus as well as by a change in intensity (Clynes, 1969; Spoor, Timmer, & Odenthal, 1969). Arlinger et al. (1982) recorded the magnetic fields evoked by brief changes in frequency of a continuous tone. Like the response to the onset of a tone, the equivalent dipole underlying the response to a frequency glide was localized to the supratemporal plane.

If a continuous stimulus is already changing, even quite slowly, a further change may not elicit an N1 response. Clynes (1969) recorded the N1-P2 response to changes in a continuous tone. The amplitude or frequency was changed from one value to another over a steady ramp. The onset of a ramp elicited an N1-P2 response but the offset of a ramp, where the tone became constant again, did not elicit the response (Figure 8, top panel). Furthermore, the response to a ramp onset could be markedly reduced or eliminated by preceding the ramp by another ramp in a similar or different direction (Figure 8, bottom panel). These findings were confirmed by Kohn, Lifshitz, and Litchfield (1978, 1980). Furthermore, Clynes reported that a ramp change in one attribute can attenuate the response to a subsequent change in another attribute. Clynes observed such interactions between the pitch and intensity of a continuous auditory stimulus. However, these interactions did not cross modality boundaries. An ongoing pitch modulation did not affect the visual response, although there were similar basic rest-motion findings in the visual (and also somatosensory) modality. Clynes (1969) concluded that the N1-P2 response is initiated by a change from sensory “rest” (a steady stimulus or silence) to sensory “motion” (from one level of a stimulus parameter to another).

The stimuli most commonly used to evoke auditory EPs are short in duration and separated from each other by silent intervals. An N1 wave is evoked by the onset of the sound-the change from silence to stimulus. Because of the short duration of the stimulus, there is little if any offset response.

In these simple paradigms, another kind of “change” can also occur: the stimulus

may change from the preceding stimulus in some parameter such as intensity or frequency. It is therefore important to distinguish between two kinds of change: “change 1” or “level change” a change from the immediately preceding stable level (usually from silence to sound); and “change-2” or “stimulus change”-a change from some previously presented stimulus. Change-2 cannot occur without a concomitant change-1 and only when successive presentations of change-1 are different (a duration change-2 is an exception). In contrast to the response to a level change, the response to a stimulus change requires that there is neuronal trace or memory of the previous stimulus (Näätänen, 1985).



Source: Näätänen and Picton, 1987.

**Figure 8.** Vertex evoked potentials to changes in the pitch of a sound having constant intensity.

Note: The pitch of the sound is shown below each of the three paired recordings. The upper recordings are in response to ramp changes in pitch. Note the absence of any response at the end of the ramp. The middle tracings show the responses to abrupt changes in pitch. The lower recordings show no clear response when one ramp-change of pitch leads immediately into another. Figure is derived from the data of Clynes, 1969, in Donchin and Lindsley (Eds.), *Average evoked potentials*, NASA.

The N1 wave is apparently generated by cerebral mechanisms which are primarily sensitive to change-1. This is probably true for all three of the components that we have so far considered. The N1 is triggered by the onset of a change in some physical characteristic from an immediately preceding stable level. The N1 mechanisms may, however, at times appear as if responding to the second kind of change also. When, for instance, an occasional tone of 1500 Hz occurs in a sequence of 1000 Hz tones, the N1 wave in response to the 1500 Hz tone is usually larger than that in response to the 1000 Hz tone (Butler, 1968; Picton, Campbell, Baribeau-Braun, & Proulx, 1978). However, this may be a case of selective refractoriness rather than a specific response to the change. The deviant stimulus apparently activates some "fresh" elements that were not activated by the preceding standard stimuli. As we shall discuss in the next section of this paper, the responsiveness of the N1 generators is decreased for a period of time after the presentation of a stimulus, and this decrease is partially specific to the stimulus. Thus the amplitude decrease is greater when the N1 is reactivated by a similar stimulus than when the N1 is reactivated by a different stimulus, i.e., the neuronal population responding to the deviant stimulus is less refractory than the neuronal population responding to the standard stimulus.

The amplitude of the N1 is therefore jointly determined by the immediate change in the stimulus level and by the refractory state of the generator mechanism. The refractory state will vary with the time from the preceding stimulus and the similarity between the present and the preceding stimulus (this similarity determining the magnitude of overlap between the neuronal populations responding to each stimulus). Therefore, when a stimulus signifies change-2 in addition to change-1, the N1 response may be bigger than when change-1 alone occurs. Another component of the response -the "mismatch negativity" (MMN) to be discussed in the following paragraphs- is much more closely related to change-2.

The cerebral response to deviant stimuli that have a lower intensity than standard stimuli provides crucial evidence differentiating the N1 and the MMN and their relations to change-2. Figure 9 illustrates such responses (Näätänen, Paavilainen,

Alho, Reinikainen, & Sams, 1987). In this experiment, the standard stimulus had an intensity of 80dB SPL and, in different blocks, the deviant stimulus ( $p=0.10$ ) had an intensity of 57, 70, 77, 83, 90, or 95 dB. The subject read a book and ignored the sequence of auditory stimuli presented at a constant ISI of 460 ms. The responses to the deviant stimuli that had higher intensities than the standard stimulus showed a larger N1 deflection than the responses to the standard stimuli. However, the response to the 77dB deviant stimulus had a smaller N1 deflection than the response to the 80dB standard stimulus. These results are similar to those obtained by Butler (1968). They indicate that the N1 deflection (at least when recorded from the mid-line) is more related to the physical characteristics of the stimulus than to the change in the stimulus from a preceding stimulus.

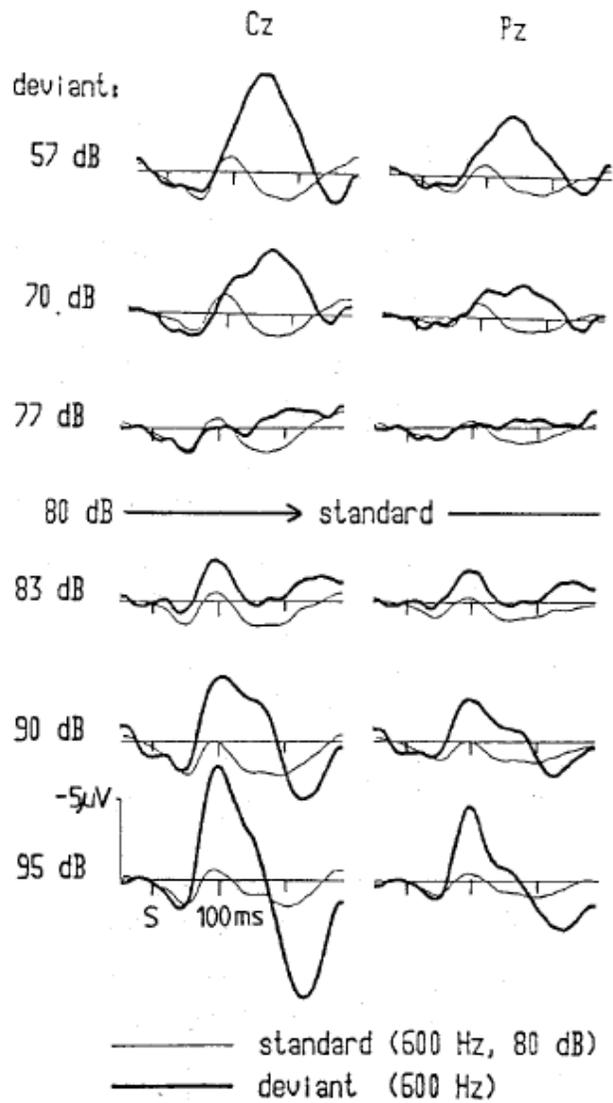
The response to the 77dB deviant stimulus revealed a separate negative deflection called the mismatch negativity (MMN). This MMN occurred in the responses to all of the deviant stimuli, both those with higher intensity and those with lower intensity than the standard stimulus. As the difference between the deviant and standard stimuli increased, the MMN became larger and earlier, overlapping the N1 deflection and making it impossible to measure the MMN and the N1 separately. These effects were similar for deviant stimuli that were higher or lower in intensity than the standard stimuli. The MMN therefore appears to vary specifically with change-2 (the difference between successive stimuli) and not with change-1 (the intensity of the stimulus). Since the latency of the MMN may overlap with that of the N1 wave, the MMN must be considered as a possible component of the scalp-recorded N1 wave.

This distinction between the N1 response to individual stimuli and the MMN response to the relations between stimuli is further supported by the changes in MMN latency with different degrees of deviance, the N1 latency showing no such variation. As seen in Figure 9, the latency of the MMN decreases with increasing difference in intensity. Figure 10 presents results from an experiment (Sams, Paavilainen, Alho, & Näätänen, 1985) in which the deviant stimuli ( $p=0.20$ ) differed in pitch from the standard stimuli. The N1 wave in response to the different

deviant stimuli (held constant within a block) is similar to that evoked by the standard stimuli and does not vary in amplitude or latency with the degree of deviance. As shown in the difference waveforms on the bottom of Figure 10 the MMN is clearly seen in response to deviant stimuli that could be discriminated from the standard stimuli, i.e. those with frequencies greater than 1008 Hz. There may be a very small MMN at 1008 Hz. The amplitude of the MMN remains relatively stable once the deviance has become clearly recognizable but the MMN peak is earlier for larger deviations in pitch. With very large deviations, the decreased latency of the MMN results in an increasing overlap between the MMN and the N1 wave (Näätänen & Gaillard, 1983). Some of the increase in the N1 wave previously attributed to specific refractoriness in one or more of the N1 generators may be explained on the basis of this overlap.

The MMN introduced by these two experiments has been reviewed more extensively elsewhere (Näätänen, 1985, 1986a). We shall discuss in the next few paragraphs some of the specific characteristics of this component and its relation to the N1 wave.

The MMN component was isolated from the N2-P3 and N2-P3a deflections of the EP by Näätänen, Gaillard, and Mantysalo (1978, 1980), by Näätänen, Simpson, and Loveless (1982), and by Näätänen and Gaillard (1983). Näätänen et al. (1978) suggested that the MMN reflects a pre-perceptual detection of stimulus change. Their subjects performed a selective dichotic-listening task, counting occasional intensity (or pitch) changes presented to one ear. The difference wave obtained by subtracting the EP to standard stimuli from that to deviant stimuli revealed a negative shift with an onset latency of about 100 ms and a duration of 200 ms. This MMN occurred in response to the deviant stimuli in either the attended or unattended inputs and its amplitude was unaffected by attention. The P300 positive wave was much larger to the (target) deviant stimuli in the attended input than to the (non-target) deviant stimuli in the unattended input.

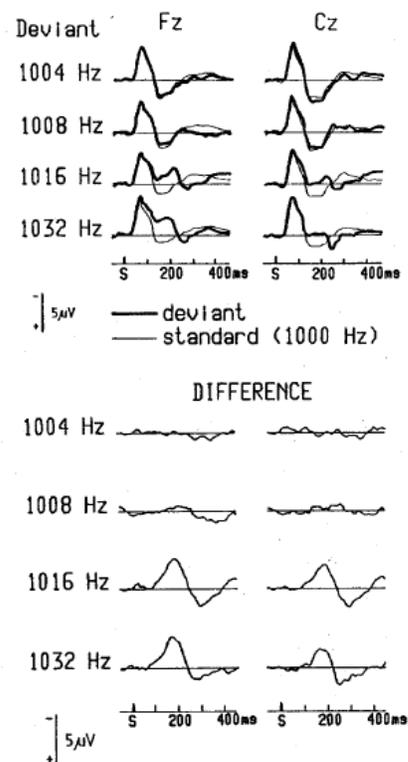


Source: Näätänen and Picton, 1987.

**Figure 9.** The mismatch negativity to intensity changes. Across-subject average vertex ( $C_z$ ) and parietal ( $P_z$ ) ERPs to standard stimuli of 80dB (thin-line) and to deviant stimuli of different intensity (thick line) as indicated on the left side of the figure.

Note: In each stimulus block, the probability of the standard stimulus was 90% and that of the deviant stimulus was 10%, stimuli being presented in random order. There was only one kind of deviant stimulus in each block. From Näätänen, Paavilainen, Alho, Reinikainen, and Sams, 1987.

Näätänen et al. (1982; see also Näätänen & Gaillard, 1983) proposed that the N2 deflection elicited by a deviant stimulus in the oddball paradigm consists of a MMN, generated in the specific auditory areas of the cortex, and an N2b wave, a later and sharper component probably of non-specific origin, that occurred when the subject attended to the stimulus sequence. When the stimuli were ignored, only the MMN occurred, unless the deviant stimuli were widely deviant, in which case some N2b waves were detected in the records of some subjects. In the dichotic listening paradigm, the N2b to targets is much smaller than in the oddball paradigm (Näätänen et al., 1978; Näätänen, Gaillard, & Mantysalo, 1980). Non-target deviant stimuli that elicit an N2b when a single attended train of stimuli is presented do not elicit this wave when they belong to the ignored input during a dichotic paradigm (Näätänen et al., 1982).



Source: Näätänen and Picton, 1987.

**Figure 10.** The mismatch negativity to small changes in frequency. The left column shows the frequency of an occasional deviant stimulus in a train of standard stimuli.

Note: The middle column shows the EPs recorded from Fz and the right column shows the EPs recorded from Cz. The top of this figure shows the across-subject average ERPs to standard stimuli of 1000 Hz (thin line) and deviant stimuli (thick line) of 1004 Hz, 1008 Hz, 1016 Hz, and 1032 Hz. There was only one kind of deviant stimulus in each block. In each stimulus block, the probability of the standard stimulus was 80% and that of the deviant stimulus was 20%, stimuli being presented in random order. The bottom of the figure shows the respective difference waveforms obtained by subtracting the EP to the standard stimuli from the EP to the deviant stimuli. Figure derived from data of Sams et al., 1985, *Electroencephalography & Clinical Neurophysiology*, 62, 437-448.

#### **2.4.2. Intensity, Frequency, and Threshold**

With decreasing the stimulus intensity, the N1 amplitude decreases and latency increases (Beagley & Knight, 1967; Picton, Woods, Baribeau-Braun, & Healey, 1977; Rapin, Schimmel, Tourk, Krasnegor, & Pollak, 1966). The change in amplitude is more variable than that of the change in latency. The latency change is more prominent with tonal stimuli than with clicks.

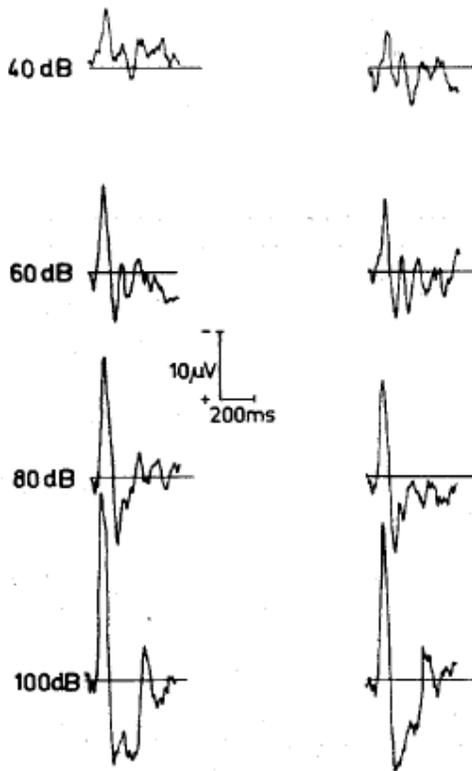
There have been many studies relating the amplitude of the N1 response to sensory magnitude. Davis and Zerlin (1966) found that the N1-P2 response to a tone pip (presented every 3.2 s) increased according to a power function with an exponent of 0.12 (compared to the psychophysical power function of 0.3). Keidel and Spreng (1965) reported a higher exponent and a closer correlation to the loudness power function at longer ISIs (30 s), but the N1 that they recorded was later (130170 ms) than usual. This may have been caused by their unusual electrode montage: their recordings were taken between the glabella (mid-forehead) and the mastoid. Keidel (1976) reviewed the field and found that the vertex N1 wave shows a power function exponent of between 0.07 and 0.28, the higher values occurring when longer ISIs (30 s) are used. He further reported that the N1 amplitude is more closely related to loudness than the N1-P2 amplitude. Pratt and Sohmer (1977) recorded auditory EPs to clicks at the same time as subjects assessed the loudness of the stimuli. A set of clicks occurred one second after the subject pressed a button to record his or her loudness estimate for the preceding clicks. Pratt and Sohmer found no clear cor-

relation between the amplitude of the late auditory response and the concurrently recorded subjective estimates of loudness.

At high intensities the amplitude of the N1 often levels off or even reduces (Buchsbbaum, 1976). This is particularly true when stimuli are presented at intervals of 2.5 s or less and the intensity is held constant within blocks (Picton, Goodman, & Bryce, 1970). This saturation of the N1 amplitude occurs well below any saturation of subjective loudness, and thus indicates a clear dissociation between sensory magnitude and the N1 amplitude. When the ISI is long and stimuli of different intensity are delivered in the same block, the N1 increases in amplitude with increasing intensity even at high intensities (Gille, Bottcher, & Ullsperger, 1986 (Figure 11)). These findings suggest that part of the N1 wave evoked by a stimulus of high intensity undergoes a more profound and longer lasting refractory period than the rest of the response. This part is probably that which we have tentatively identified as component 3.

The change in the amplitude of the N1 with increasing intensity varies greatly among subjects. Some subjects have an N1 that continues to increase with increasing intensity at all levels, whereas others have an N1 that saturates or becomes smaller at high intensities. This has led to dividing subjects into "augmenters" and "reducers," a classification supposedly reflecting an individual's characteristic response to stimulation (Buchsbbaum, 1976). Much research has therefore attempted to relate this EP phenomenon to aspects of personality and psychopathology. Augmentation and reduction have most often been assessed by measuring the vertex P1-N1 amplitudes in the response to visual or auditory stimuli presented at multiple stimulus intensities. Unfortunately, the type of intensity function can change with the sensory modality (Raine, Mitchell, & Venables, 1981); with the ISI (Picton et al., 1970), with the intensity range over which it is measured (Prescott, Connolly, & Gruzelier, 1984), and with the attentive strategy of the subject (Schechter & Buchsbbaum, 1973). Moreover, the phenomenon varies with the technique of measurement: individuals who augment at one electrode location for one peak measurement may reduce at

another location or for another peak (Prescott et al., 1984). Consequently, a recent review has concluded that the results of augmenting/reducing research have often been “inconclusive or equivocal” (Prescott et al., 1984, p. 32). “Certainly, before the concept is further utilised to distinguish normal from pathological groups there remains a need to demonstrate which EP measures and recording sites will yield reliable, unambiguous and, more importantly, useful measures of an individual's enduring mode of stimulus intensity control” (Prescott et al., 1984, p. 42). The findings of Raine et al. (1981) suggest that “cortical augmenting-reducing is modality-specific and mitigate against the notion of a general mechanism residing in the CNS regulating sensory input” (p. 705).



Source: Näätänen and Picton, 1987.

**Figure 11.** EPs of a typical subject to 40, 60, 80 and 100 dB stimuli, presented in a pseudorandom order at constant 4-s ISIs while the subject was reading a book.

Note: The baseline represents the mean of the 200-ms period before stimulus onset. The left side shows data from the 1st session, the right side from the 2nd session. Unpublished figure from the study of Gille, Bottcher, and Ullsperger, 1986, *Zeitschrift für gesamte Hygiene*, 32, 566-568.

Even at constant levels of stimulus intensity or perceived loudness, the amplitude of the N1 varies with the tonal frequency of the stimulus (Antinoro & Skinner, 1968; Antinoro, Skinner, & Jones, 1970; Picton, Woods, & Proulx, 1978b; Stelmack, Achom, & Michaud, 1977). The N1 decreases with increasing tonal frequency particularly at frequencies greater than 2000 Hz. Some of this effect may be related to the asymmetry of the travelling wave in the cochlea: low-frequency tones activate a much broader region of the basilar membrane than high frequency tones.

The N1 evoked by binaural tones is slightly larger (about 10%) than that evoked by monaural tones (Davis & Zerlin, 1966; Picton, Woods, & Proulx, 1978b). Nevertheless, the N1 evoked by binaural stimuli increases in amplitude in much the same way as that evoked by monaural stimuli (Butler, Keidel, & Spreng, 1969). The N1 evoked by binaural stimuli is much smaller than what would be expected from the addition of the two monaural responses (Berlin, Hood, & Allen, 1984; Picton, Rodriguez, Linden, & Maiste, 1985). This could result from either occlusion or mutual inhibition between the neuronal populations generating the N1 response. Occlusion occurs when the response elicited by two stimuli presented together is less than the sum of the responses to each stimulus presented separately. Mutual inhibition occurs when the two responses inhibit each other. Some recent evidence from magnetic recordings (Pantev et al., 1986) suggests that there may indeed be some inhibitory interactions between the left and right auditory cortices. The amplitude of the magnetic field response at 100 ms over the temporal cortex contralateral to a monaural stimulus became smaller when the stimulus was made binaural.

At near-threshold levels the N1 amplitude is remarkably larger when a subject counts the auditory stimuli than when reading (Mast & Watson, 1968). This task-related

difference is attenuated at higher intensities (Davis & Yoshie, 1963; Gross, Begleiter, Tobin, & Kissin, 1965). Some studies have indicated that the amplitude of the N1 evoked by near threshold stimuli presented to an attentive subject correlates strongly with sensory magnitude. The N1 amplitude varies directly with the detection of the stimulus and with the subject's confidence in the detection (Squires, Hillyard, & Lindsay, 1973; Squires, Squires, & Hillyard, 1975).

Parasuraman and his colleagues (Parasuraman & Beatty, 1980; Parasuraman, Richter, & Beatty, 1982) have recorded auditory EPs during the detection and recognition of a near-threshold tone presented in continuous wideband noise. On half the trials there was a tone and on the other half there was only noise. The subjects reported whether they had heard a tone, using a four-category rating scale, and then chose between two (Experiment 1) or four (Experiment 2) possible frequencies (this choice being made whether or not the subject was sure that the stimulus had occurred). The tones evoked a P300 wave that varied in amplitude with both the confidence in detecting the tone's occurrence and the accuracy in recognizing its frequency. In contrast, the N1 wave, although being larger for the more confident detections, did not vary significantly with the correctness of the recognition. There is some suggestion in the reported data that the N1 amplitude was slightly (but not significantly) larger at higher levels of recognition accuracy. Furthermore, a principal component analysis did not delineate an N1 component as separate from the P300. Nevertheless, the results show that the N1 is much more related to the detection than the recognition of a near-threshold tone.

The latency of the N1 wave increases with decreasing intensity and the N1 evoked near threshold is a small broad wave with a peak latency between 150 and 200 ms. It is difficult to be sure that the cerebral processes underlying this wave are the same as those underlying the N1 at higher intensities. The near-threshold N1 may be overlapped by negative components such as the N2b, which we have already discussed, and the processing negativity.

In subjects who are not particularly attending to the stimuli, the N1 becomes difficult to measure at low intensities. The average difference between the EP threshold and subjective threshold is often less than 20dB (Beagley & Kellogg, 1969; Davis & Zerlin, 1966; Keidel, 1976). This is sufficient for the objective assessment of hearing. However, there are occasional discrepancies between the EP thresholds and subjective thresholds of greater than 60dB (Rose, Keating, Hedgelock, Miller, & Schreurs, 1972). Using interpreters who were blind to the actual stimulus intensity being used, Mendel et al. (1975) measured a mean response threshold in waking adults of 27dB SL with a range of 10 to 45 dB SL. In sleeping subjects, they found the EP threshold to be even higher and more variable. Stapells (1984) reported a mean threshold of 16dB SL and a standard deviation of 17.2 dB in waking subjects. This variability may be partially caused by fluctuations in arousal or attention (Picton et al., 1977). Since it is difficult to control these parameters in young children, EP audiometry with the N1 wave is used only in waking adults. In sleeping subjects and in children the threshold for the N1 response is too high and too variable for clinical use.

## **2.5. A Review of P2 Component**

Event-related brain potentials (ERPs) are induced exogenously by environmental events (such as sensory stimuli) or endogenously by processes such as decision making. ERPs appear as transient changes in the ongoing electrical brain activity (recorded as EEG) within a short time frame following the eliciting event. ERPs typically consist of a series of voltage polarity changes, seen as peaks and troughs in the ERP waveform. Each ERP deflection is a composite of several components that are generated by parallel streams of neural activity, overlapping in time. A component can therefore be defined as a voltage contribution to the ERP which reflects a functionally discrete stage of neural processing, occurring in a restricted cerebral area (Näätänen and Picton, 1987). Peaks within an ERP can be classified according to their magnitude, timing relative to stimulus onset, polarity, anatomical site of generation, or function reflected by them. Depending on the stimulus modality, the ERP will have a series of early cortical peaks, reflective of initial processing in a sensory receiving area, for example, P100 in a visual evoked potential (VEP), P50 in an auditory evoked potential (AEP) or P1 or N1 in a respiratory related evoked potential (RREP). Following these are later peaks such as N1, P2, P3a, P3b and N400, which reflect later more integrative cognitive processing. Unlike the N1 or the other late ERPs, little work has been done on the P2 to elucidate the generation mechanisms, the cognitive/attentional correlates or its functional significance in general. The P2 is usually referred to in the context of the tail end of the N1-P2 complex or the 'vertex potential'. Linkage to the N1 has particularly been used to describe the changes that occur to the N1 during drowsiness, sleep onset and stable sleep. Thus, this part of the literature review attempts to provide a selective review of what is known about the P2 during wake and sleep and considers whether it is sensible to continue to view it as linked to N1 or whether it should be viewed as a functionally distinct entity (Crowley and Colrain, 2004).

### **2.5.1. Phenomenology of the P2**

Auditory stimuli elicit a series of long latency ERPs. In the 80–200 ms latency range ERPs mainly consist of two peaks, the first with negative polarity, called N1, occurring roughly 75–150 ms after stimulus onset and the second with positive polarity, P2, with a latency of approximately 150–250 ms. Although the N1 and P2 are most often elicited by auditory stimuli, a response of similar morphology also occurs following stimuli in the somatosensory modality. These two components are assumed to at least partially represent an exogenous response, that is, they are elicited by both attended and non-attended stimuli. Throughout the literature, the P2 is often treated for convenience as unitary, with the earlier N1 peak being referred to as the slow vertex potential, long latency response etc. Although P2 co-varies with N1 along many stimulus dimensions, P2 can be dissociated experimentally (Ford et al., 1976, 1999; Oades et al., 1997; Cited in Crowleya and Colrain, 2004), developmentally (Oades et al., 1997; Ponton et al., 2000b; Cited in Crowleya and Colrain, 2004), and topographically (Roth et al., 1976; Vaughan et al., 1980; Cited in Crowleya and Colrain, 2004). Studies on the P2 as an independent component, however, are relatively scarce and thus little is known regarding its endogenous or exogenous processing correlates.

#### **2.5.1.1. Basic parameters of the P2 – effects of stimulus properties**

Much of the published work on the impact of stimulus parameters on the P2 is from the 1960s and 1970s, with a few more recent studies. A disadvantage common to most of the earlier studies is that the N1 and P2 peaks were combined in a single peak to peak measurement, thus eliminating any elucidation of the differential effects of stimulus and subject-related variables on the individual components. Another problem is that the effects of these variables are non-linear and interactive and much of the earlier work tended to focus on one parameter at a time. This practice has obvious limitations given the number of other components that overlap the P2 (including P3a, MMN and N2). To these issues must be added differences in

terminology, response measures and measurement protocols and of over-inference from small and non-representative study subject samples (Crowleya and Colrain, 2004).

### **2.5.1.2. Intensity**

The most consistent finding in earlier studies of the influence of stimulus loudness on these components was an increase of the N1/P2 peak-to-peak amplitude when increasing stimulus intensity (Beagley and Knight, 1967; Gerin et al., 1972; Picton et al., 1970; Rapin et al., 1966; Cited in Crowleya and Colrain, 2004). Although the form of the input-output function has been strongly debated, the consensus is that the increase appears to be roughly linear in micro volts per decibel, with a tendency to saturate or even reverse at high levels (i.e. above 70 dB) (Geisler and Murphy, 2000; Picton et al., 1977; Pineda et al., 1991; Rothman, 1970; Spoor et al., 1969; Sugg and Polich, 1995; Cited in Crowleya and Colrain, 2004). The saturation level may decrease at short inter-stimulus intervals and with higher pitched stimuli but reports have been inconsistent (Beagley and Knight, 1967; Kaskey et al., 1980; Nelson and Lassman, 1968; Onishi and Davis, 1968; Picton et al., 1970; Rothman et al., 1970; Cited in Crowleya and Colrain, 2004).

There have been a few studies, however, applying baseline to peak measurements and more recent studies have clearly demonstrated that the intensity/amplitude functions differ for N1 and P2 when they are analysed separately. When measuring the N1 and P2 amplitudes at Fz and Cz baseline to peak, Adler and Adler (1989) found an increase of about 0.6 mV/10 dB between 30 and 70 dB for both peaks. However, the essential difference between these two peaks occurs at stimulus intensities above 70 dB, where with further increases in intensity level, the N1 amplitude decreased whereas the P2 amplitude increased. This could be used as an argument that the underlying negative component processes diminishing N1 are somehow overlapping P2. This is unlikely to be the case, however, due to the fact

that increases in intensity from baseline up to this point resulted in augmentation of both N1 and P2.

The latencies of N1 and P2 have been reported to decrease with increasing stimulus intensity from auditory threshold to 50 dB SL (Rapin et al., 1966) and 70 dB SL (Beagley and Knight, 1967; Cited in Crowleya and Colrain, 2004). Adler and Adler (1989) have reported a U-shaped relationship between stimulus intensity and the N1 and P2 latencies with a minimum latency value for both waves at 70 dB SL. Moreover, the latency changes of P2 were reported to be much greater than those of N1, particularly at low intensity levels (between 30 and 70 dB).

### **2.5.1.3. Pitch**

Despite contradictory reports, on balance, parametric studies of the effects of pitch on both the N1 and P2 peaks indicate that amplitudes from low pitch stimuli (250–400 Hz) produce larger and later components, compared to those from higher pitch stimuli (1000–3000 Hz) (Antinoro et al., 1969; Jacobson et al., 1992; O'Donnell et al., 1997; Sugg and Polich, 1995; Wunderlich and Cone-Wesson, 2001; Cited in Crowleya and Colrain, 2004).

Most studies also show that latency decreases as pitch increases and that this effect is most marked at higher stimulus levels (Alain et al., 1997; Jacobson et al., 1992; Cited in Crowleya and Colrain, 2004). Wunderlich and Cone-Wesson (2001), however, have reported that unlike the N1, there was no effect of pitch on the latency of the P2 peak.

### **2.5.1.4. Inter-stimulus interval (ISI)**

It is clear that ISI is a parameter that produces large and consistent changes in ERP components. Early studies, again analysing the N1/P2 peak to peak amplitude, reported that the magnitude of the response was a linear function of the logarithm of

the recovery period. That is, for every 10-fold increase in ISI, there was a 5.6 mV increase in the N1/P2 amplitude magnitude. This relation was found to hold true for ISIs from 0.5 s up to about 10 s (Davis et al., 1966; Davis and Zerlin, 1966; Keidel and Spreng, 1965; Cited in Crowleya and Colrain, 2004), with little change in latency. While this pattern shows large inter subject variation, it is consistent with an inter-stimulus adaptation recovery process that has a time constant in the region of 4–5 s (Davis et al., 1966; Nelson and Lassman, 1968; Picton et al., 1977).

In a study of the effects of random ISI, Roth et al. (1976) demonstrated different recovery periods for N1 and P2. More specifically, the effect of the first prior interval for P2 at Cz showed equal increments in amplitude with doubling of the interval, while N1 showed no significant amplitude increment between 0.75 and 1.5s.

#### **2.5.1.5. Habituation**

Two different processes have been described for habituation. In the typical paradigm, trains of stimuli are presented with short inter-stimulus intervals and considerable longer inter-train intervals. Changes in ERP component latencies within trains are referred to as short term habituation, whereas changes between trains are referred to as long-term habituation. In both cases, decrease of the N1 amplitude has been found as a hallmark of the ERP habituation (Fruhstorfer, 1971; Kenemans et al., 1989; Lutzenberger et al., 1979; Megela and Teyler, 1979; Rust, 1977; Cited in Crowleya and Colrain, 2004). With regard to P2, some authors suggested that this component is non-habituating (Kenemans et al., 1989; Megela and Teyler, 1979; Cited in Crowleya and Colrain, 2004).

#### **2.5.2. P2 and attention**

An increase in the level of attentiveness of a subject produces an increase in the amplitude of the N1 peak but a decrease in the amplitude of the P2. This effect can best be evaluated by subtracting the ERP from stimuli that have been ignored, from

the ERP to the same stimuli when they are attended. Such a subtraction shows a broad negative wave that has been labelled as 'Nd' by Hansen and Hillyard (1980) and 'Processing Negativity (PN)' by Näätänen et al. (1978). Thus, it has been hypothesized that PN overlaps and sums with both N1 and P2, causing N1 to appear larger in attend conditions, but P2 to be smaller (or less positive or more negative) (Michie et al., 1990, 1993; Näätänen and Michie, 1979; Näätänen and Picton, 1987; Cited in Crowleya and Colrain, 2004). The theoretical and functional differences between Nd and PN have been reviewed extensively elsewhere (for example see Näätänen, 1990). The modulation of the P2 wave has been thought to serve as an index of the ease with which relevant information can be distinguished from the irrelevant. The data do not however, rule out the existence of independent effects of attention on N1 and P2, such that N1 is enhanced and P2 diminished when stimuli are attended to.

## CHAPTER III

### METHOD

This chapter is devoted to the presentation of information on subjects, data collection instruments and methods, data reduction procedures and analysis and the details of control paradigms.

#### 3.1. Subjects

Before the commencement of the trial, all subjects were informed about the possible risks associated with the experimentation. The study protocol was approved by the Medical Ethical Committee of the Başkent University, Medical Faculty Ankara (Certificate No: 2004/85). Written informed consent was obtained from each subject before inclusion into this study. The study conformed to the ethical requirements of the 1975 Helsinki Declaration. The subjects of the present study were 10 non-archers (N=6 males; N=4 females) for control trials and 15 archers (N=9 males; N=6 females) for archery shooting experiments. The mean age of the non-archers group was 26.8 years (range 22 yrs – 32 yrs), that of the archers' group 22.8 years (range 16 yrs – 31 yrs). The mean years of archery experience and the highest FITA scores was 5.8 years (range 2 yrs –14 yrs) and 1120 (range 1050-1313 FITA score) for archers group respectively (Table 3).

The FITA score is a summation of four distances (for female: 70, 60, 50 and 30 m; for male: 90, 70, 50 and 30 m), which are set by the International Archery Federation (FITA). An archer shoots 36 arrows to each distance. So, he/she shoots totally 144 arrows in a FITA round where the highest score can be 1440 (FITA, 2004). Non-archers were recruited from the graduate students in the Biophysics laboratory. Archers were selected and volunteered to the current study from the competitors list in the Turkish Archery Federation with different performance levels. All subjects

reported normal hearing, had medical histories free of significant neurological problems, and were not taking medication known to affect brain activity.

**Table 3.** Subjects of the study.

<b>Subjects</b>	<b>N</b>	<b>Age</b>	<b>FITA Score</b>	<b>Archery Experience</b>
Non-archers	10	26.8 ± 2.35	-	-
Archers	15	22.8 ± 3.15	1120 ± 35.42	5.8 ± 2.61

### **3.2. Procedure**

#### **3.2.1. Control Conditions**

Control trials were conducted in the laboratory conditions without arrow shot. Six different paradigms were selected; (1) *Paradigm 1*: Subject presses to the enter button and sound is delivered with 1–3 s delay. When the subject hears the sound he/she responds to the stimulus by raising his/her forefinger without any wrist movement. The subject was asked to press the button with 8–10 sec intervals. (2) *Paradigm 2*: The stimulus is delivered automatically by the computer with an ISI of 8-10 s. When the subject hears the sound he/she responds to the stimulus by raising his/her forefinger without any wrist movement. (3) *Paradigm 3*: Subject presses the enter button and sound is delivered with 1–3 s delay. The subject does not respond to the sound with any type of movement patterns. The subject was asked to press the button with 8–10 sec intervals. (4) *Paradigm 4*: The stimulus is delivered automatically by the computer with an ISI of 8-12 sec. When the subject hears the sound he/she responds to the stimulus by raising his/her forefinger without any wrist movement. (5) *Paradigm 5*: The stimulus is delivered automatically by the computer with an ISI of 1 sec. The subject does not respond to the sound with any type of movement patterns. (6) *Paradigm 6*: The stimulus is delivered as soon as the subject presses the enter button. There was no task in paradigm 6 (Table 4).

**Table 4.** The details of control paradigms.

<b>Paradigm</b>	<b>A / M</b>	<b>Surprise</b>	<b>Task</b>	<b>ISI</b>	<b>Short Notes</b>
1	M	Yes	Yes	Long	Subject presses enter. 1-3 s stimulus delay.
2	A	Yes	Yes	Long	Automatic delivery of stimulus with 8-10 s ISI.
3	M	Yes	No	Long	Subject presses enter. 1-3 s stimulus delay
4	A	Yes	No	Long	Automatic delivery of stimulus with 8-10 s ISI.
5	A	No	No	Short	Automatic delivery of stimulus with 1 s ISI.
6	M	No	No	Long	Stimulus delivered as soon as subject press.

A: Automatic delivery of stimulus.

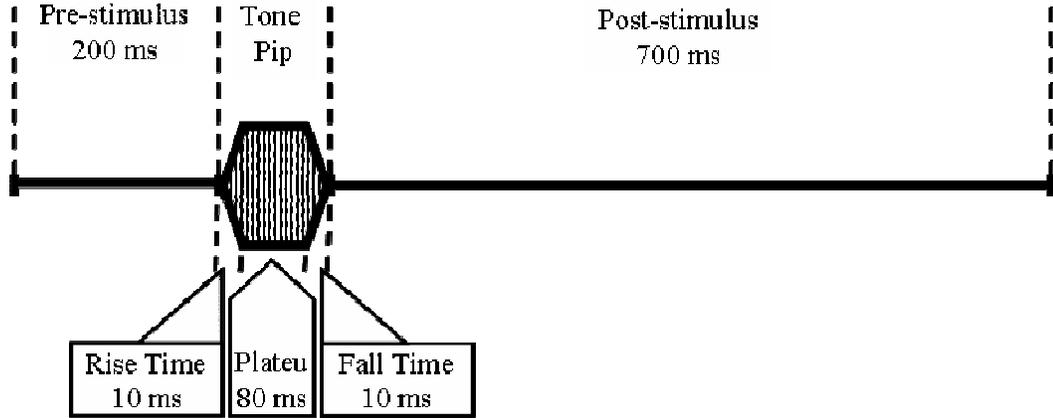
M: Manual delivery of stimulus by subject press.

Long: Long ISI.

Short: Short ISI.

ISI: Inter Stimulus Interval.

Sound pips are obtained by abruptly changing either the timing (inter-stimulus interval (ISI)) or the occurrence (automatic or manual) of the sound, which are produced by energizing a pair of matched earphones by electric pulses of 1 ms duration (Ungan et. al., 2001). Basically, 6 paradigms consisted of two different factors. The first factor made subjects listen to the sound pips in different inter-stimulus interval (ISI) between the paradigms, being 1, 3 or 12 sec offset-to-onset. The second one required the subjects to receive the sound pips either manually or automatically. Moreover, subject is asked to respond to the sound by raising his/her forefinger. All tones were 1000 Hz. Specifications of tone pip that is used for control conditions for both non-archers and archers is shown in Figure 12.



**Figure 12.** Specifications of tone pip that was used for control conditions for both non-archers and archers.

### 3.2.2. Archery Shooting

Shootings were performed from 18 m that is official competition distance with target face. Auditory Evoked Brain Potentials (AEBPs) were recorded 200 ms before and 800 ms after the trigger (fall of the clicker) over the vertex during the shots of each subject (Figure 13). Paradigm 1 and 5 was conducted just before and after the archery shooting to test the effect of archery shooting on AEBPs.

The arrow was initially positioned between the unattached end of the clicker and the bow-grip. As the arrow was pulled beyond the clicker, the clicker-lever fell on the bow-handle, which conveyed the signal to the archer that the arrow was appropriately positioned and is ready to be released (Ertan et. al., 2003; Ertan et. al., 2005). A mechanical switch was attached under the clicker to accurately measure the point of the auditory stimulus from clicker. This stimulus was superimposed with the EEG recordings in the same time frame.



**Figure 13.** The placement of the electro cap on the scalp of an archer.

Each archer shot 72 arrows from 18 m after completing twelve trial shots to acquaint with the measurement conditions. The order of shots was controlled by assigning colours and numbers (yellow: 1<sup>st</sup> arrow; red: 2<sup>nd</sup> arrow; blue: 3<sup>rd</sup> arrow; black: 4<sup>th</sup> arrow; white: 5<sup>th</sup> arrow; green: 6<sup>th</sup> arrow) on each arrow. Each end consisted of six arrows. Photos were taken on the target face after each end to determine the hits. The front view of the picture was used to decide the place of the arrow on the target (Picture 14a). Moreover two more photos were taken for each end from both sides to use for determining hit properly on the target face (Figure 14 a and b). Hits of an archer in each end were processed by placing the hits on a coordinate system for further analysis (Figure 15). All these hits were matched with the single sweeps of EEG recordings. Finally, hits were grouped as falling into the hit-area and miss-area with their corresponding EEG recordings.

The hit-area is defined as the rectangle between  $(x_1, y_1)$ ,  $(x_1, y_2)$ ,  $(x_2, y_1)$ ,  $(x_2, y_2)$  and the miss-area is the outer part of the hit-area on the target face. Hits on the target were divided into two areas: hit-area and miss-area according to the formula given below:

$$x_1 = m_x - k_x$$

$$x_2 = m_x + k_x$$

$$y_1 = m_y - k_y$$

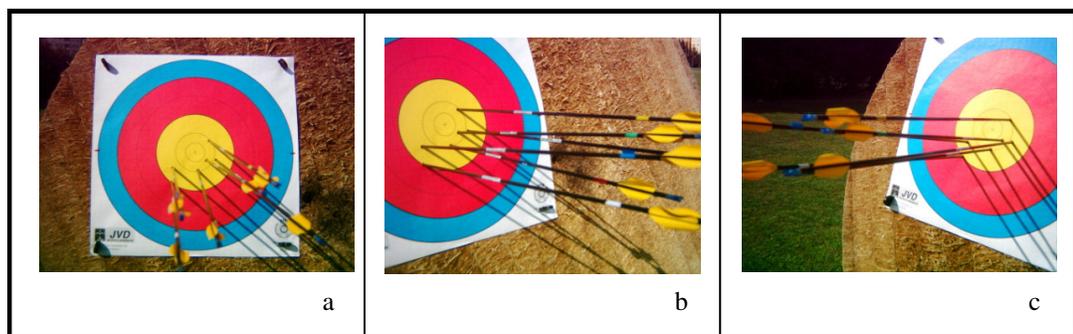
$$y_2 = m_y + k_y, \text{ where}$$

$m_x$ : mean of x-values of hits

$m_y$ : mean of y-values of hits

$k$ : a positive real number

The processing of the hits on the target was illustrated in Figure 14 and Figure 15. During the measurements, pictures of each end were taken first from front view than from both sides for later analysis. Right and left side pictures were used for discriminating the arrow's order from the colours on their shafts just below the feathers. The hit area was increased and/or decreased by changing the  $k_x$  and  $k_y$  values. The number of arrows was summarized corresponding to hit and miss areas for different values of  $k_x$  and  $k_y$ . Comparison of successful and unsuccessful hits was made according to the values of 1,2 cm and 0,8 cm for  $k_x$  and  $k_y$  respectively (Table 5).



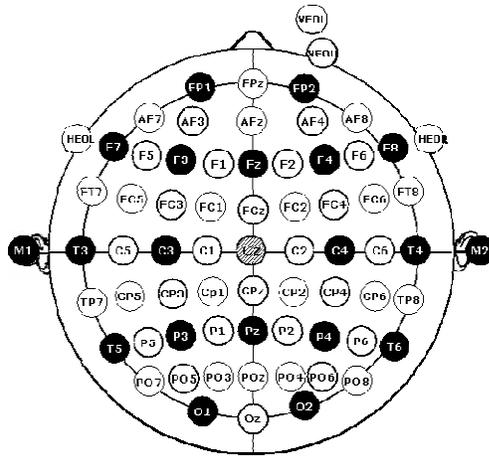
**Figure 14.** The processing of the hits on the target (a) front view of the hits that is used for analysis, (b) left view and (c) right views were used for determining the order of shot.



**Table 5.** The number of arrows corresponding to hit and/or miss area for each subject separately and whole group.

$k_x=$ $k_y=$	hit or miss area	Subject															Total
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
		Number of Shots for Each Subject															
		70	72	72	72	72	72	72	72	72	72	72	72	64	72	72	
0.3	hit	2	4	4	6	2	4	8	2	5	5	2	6	5	5	5	65
0.3	miss	68	68	68	66	70	68	64	70	67	67	70	58	67	67	67	1005
0.4	hit	4	5	5	12	4	9	10	4	11	5	7	9	6	11	6	108
0.4	miss	66	67	67	60	68	63	62	68	61	67	65	55	68	61	68	962
0.5	hit	7	9	6	16	8	14	14	6	19	8	17	10	10	15	11	169
0.5	miss	63	63	68	56	64	58	58	68	53	64	55	54	62	57	61	901
0.6	hit	12	13	10	19	13	18	19	9	20	17	19	16	13	18	16	232
0.6	miss	58	59	62	53	59	54	53	63	52	55	53	48	59	54	56	838
0.7	hit	17	19	14	24	19	21	25	14	24	27	21	18	18	28	24	313
0.7	miss	53	53	58	48	53	51	47	58	48	45	51	46	54	44	48	757
0.8	hit	23	20	19	32	20	23	30	19	27	28	22	20	25	35	26	369
0.8	miss	47	52	53	40	52	49	42	53	45	44	50	44	47	37	46	701
0.9	hit	28	26	25	35	22	27	33	30	32	32	25	26	33	38	32	444
0.9	miss	42	46	47	37	52	45	39	42	40	40	47	38	39	34	40	626
1.0	hit	34	34	30	37	27	34	37	34	37	36	31	34	35	42	36	518
1.0	miss	36	38	42	35	45	38	35	38	35	36	41	38	37	30	36	552
1.1	hit	38	40	38	39	34	40	39	39	44	42	34	36	40	48	39	590
1.1	miss	32	32	34	33	38	32	33	33	28	30	38	28	32	24	33	480
1.2	hit	41	44	42	41	42	45	44	43	45	48	40	41	44	51	41	652
1.2	miss	29	28	30	31	30	27	28	29	27	24	32	23	28	21	31	418
1.0	hit	32	30	26	36	24	31	35	31	36	34	27	30	34	42	35	483
0.9	miss	38	42	46	36	48	41	37	41	36	38	45	34	38	30	37	587
1.1	hit	30	27	25	35	25	32	32	26	37	36	26	30	29	43	32	465
0.8	miss	40	45	47	37	47	40	40	46	35	36	46	34	43	29	40	605
1.1	hit	34	33	32	36	25	33	35	33	38	37	28	31	36	45	37	513
0.9	miss	36	39	40	36	47	39	37	39	34	35	44	41	36	27	35	557
1.2	hit	30	29	26	36	26	34	35	29	37	38	30	30	32	44	33	489
0.8	miss	40	43	46	36	46	38	37	43	35	34	42	34	40	28	39	581
1.2	hit	34	35	33	37	26	35	38	36	38	39	32	31	39	46	38	537
0.9	miss	36	37	39	35	46	37	34	36	34	33	40	33	33	26	34	533
1.2	hit	35	35	33	37	27	35	38	36	38	39	32	31	39	46	39	540
0.91	miss	35	37	39	35	45	37	34	36	34	33	40	33	33	26	33	530
1.2	hit	35	35	34	37	28	35	38	36	38	39	32	32	39	46	39	543
0.92	miss	35	37	38	35	44	37	34	36	34	33	40	32	33	26	33	527

Each experimental session lasted no longer than 2 h in both control experiments and archery shooting. EEG was continuously recorded and stored for off-line analysis. Continuous records were epoched beginning 200 ms before and ending 800 ms after the auditory stimulus and averaged for both control experiments and archery shootings. EEG recording procedures were the same in both non-archer and archer groups. Potentials were averaged separately for each subject.



**Figure 16.** Auditory Event Related Potentials recording locations (20 sites) where Cz was set as reference electrode.

### 3.4. Data Processing

EEG was continuously recorded (digitized at 500 Hz) and stored for off-line analysis. The ERP data were averaged with the sweep beginning 200 ms before the stimuli and lasting until 800 ms after stimulus onset (sound pip and/or the fall of the clicker). Potentials were averaged separately for each combination of response type in control trials and for archery shooting. Transient ERPs (N100, P200) were band pass filtered (1–12 Hz, Butterworth 12 dB/oct slopes). The maximum amplitudes and peak latencies of the auditory N100 and the P200 ERP components were measured. Latencies were measured relative to the stimulus onset and amplitudes measured relative to the mean value in the 200 ms preceding stimulus onset. N100 amplitude and latency were defined at the maximum negativity between 50 and 150 ms, and

P200 amplitude and latency at the maximum positivity between 150 and 250 ms. Statistical analyses evaluated for each wave were limited to measurements at the electrode site where the peak amplitude was maximal. The M<sub>2</sub> electrode was chosen to be the site of measurement for both N100 and P200 referenced to Cz. Grand-averaged waveforms did not reveal consistent long latency components (e.g. N200, P300 etc.) (Golob et al., 2002).

### **3.5. Statistical Analysis**

Data are expressed as means  $\pm$  STDs. P values  $<0.05$  were considered significant. Electrophysiological (ERP peak amplitudes, latencies, and mean amplitudes of potentials) measures were compared using paired and unpaired samples *t* tests for normally distributed data sets. When the data set has no Gaussian distribution, the non-parametric tests were applied instead of *t* tests. Besides, pre and post interventions (archery shooting session) were compared by using paired samples *t* test. Analysis of variance (ANOVA) was used to compare the ERPs during archery shooting and control conditions. When the ANOVA was significant, Tukey *post hoc* test were used to locate the significant differences.

## **CHAPTER IV**

### **RESULTS**

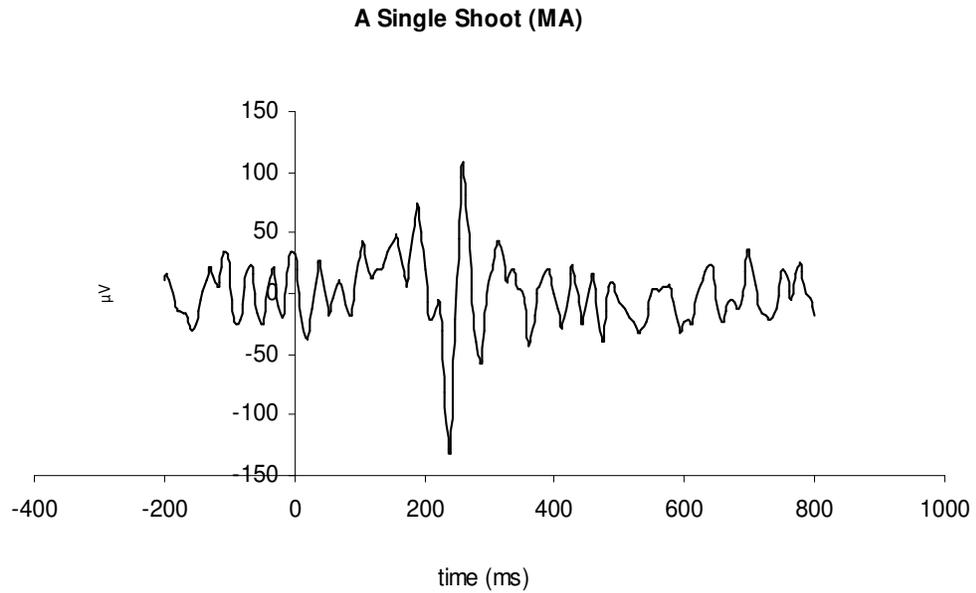
This chapter presents the results of the current study which examines the “Auditory Evoked Brain Potentials (AEBPs) in Archery” from different aspects. First, a brief summary of preliminary analysis of AEBPs during archery shooting and control conditions is provided. Second, after application of data reduction procedures, the EEG data set was epoched as 200 msec before and 800 msec after the stimulus (fall of clicker). Moreover, EEG data was classified according to the hits on the target. The epoched EEG data was matched with the hits on the target and divided accordingly as being in the hit and miss area. The EEG sweeps in hit and miss area was analyzed to test if there is any differences between EEG data in hit and miss area. The same procedure was applied for testing the effect of archery shooting on the given brain response. Below is the summary of the analysis applied during data reduction and analysis.

#### **4.1. Preliminary analysis of AEBPs during Archery Shooting**

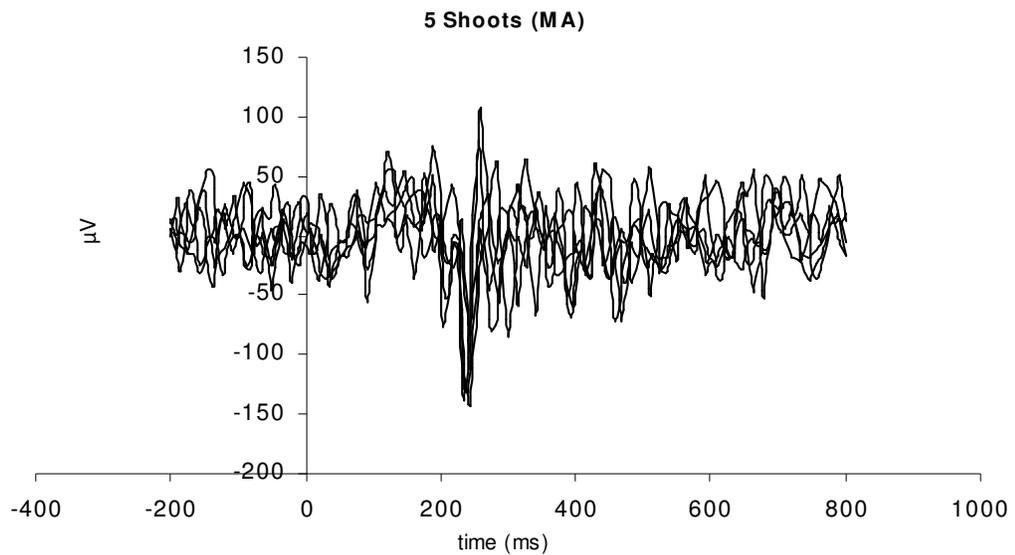
##### **4.1.1. Results of Archery Shootings**

Two high level archers have participated in shooting trials as being the preliminary measurements during the time their EEG recording, which was synchronized with the fall of the clicker, was made without having any paradigm. Shootings were performed from 18 m that is an official competition distance with target face. Auditory Evoked Potentials were recorded 200 ms before and 800 ms after the signal over the vertex. Stages of preliminary measurements in relation with the purpose of present study have been shown in following figures. The following figures illustrate that the brain response to an auditory stimulus involves a negative going wave

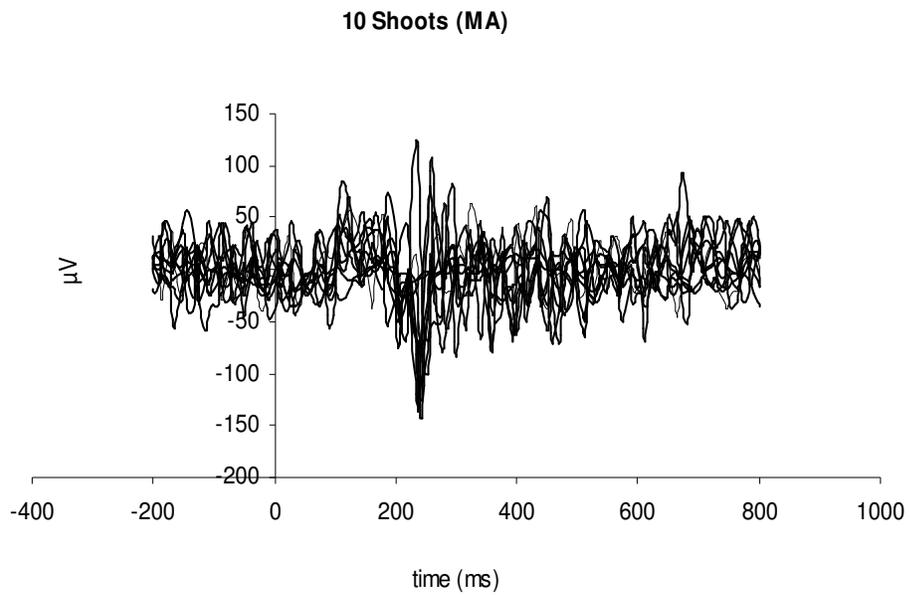
occurring about 100 msec and a positive going wave occurring about 200 msec. However, it is enough to reach any conclusion by analyzing the preliminary data set.



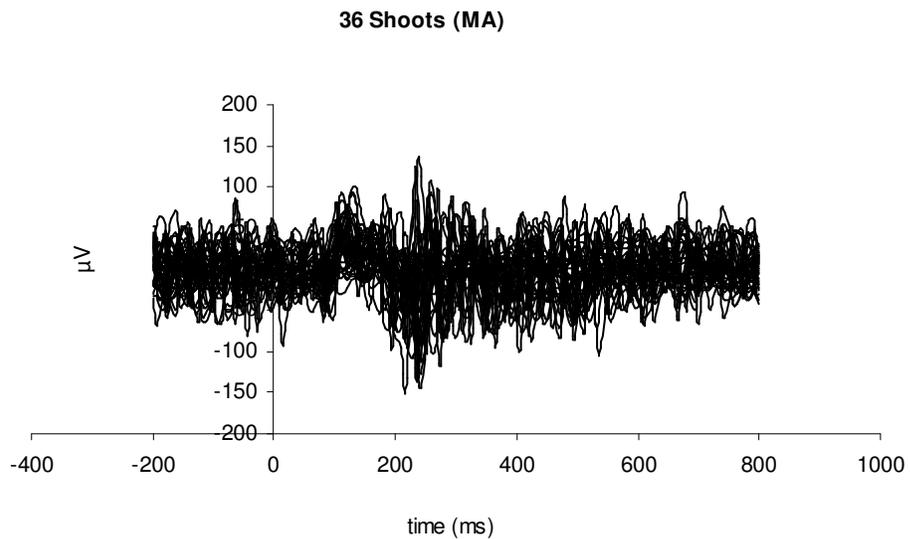
**Figure 17.** Brain electro potential response during a single shoot done by a high level archer.



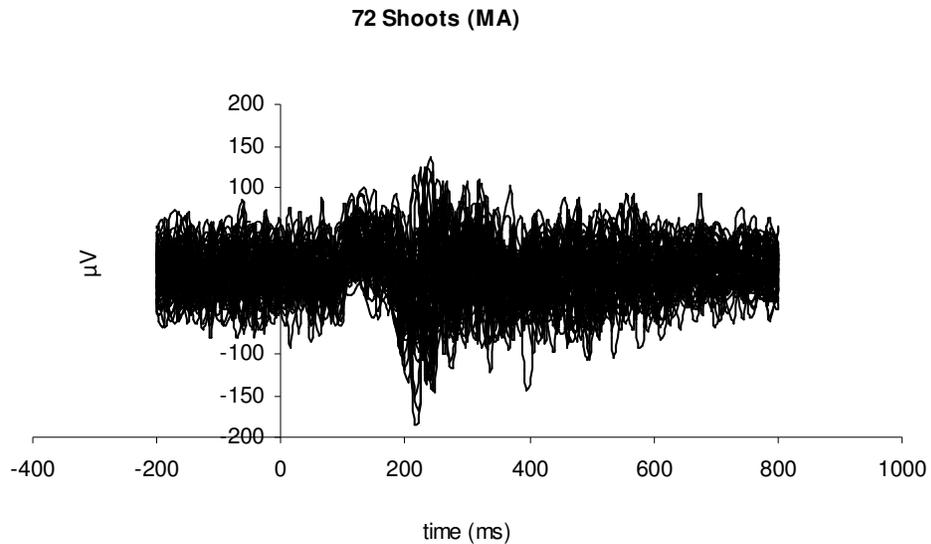
**Figure 18.** Brain electro potential responses measured during archery shooting done by a high level archer. Tabulation of 5 shoots together.



**Figure 19.** Brain electro potential responses measured during archery shooting done by a high level archer. Tabulation of 10 shots together.



**Figure 20.** Brain electro potential responses measured during archery shooting done by a high level archer. Tabulation of 36 shots together.



**Figure 21.** Brain electro potential responses measured during archery shooting done by a high level archer. Tabulation of 72 shots together.

The preliminary analysis has shown that fall of the clicker evokes long latency auditory brain potentials with the latency of 100 msec and 200 msec. These responses are called as N1-P2 components. As long as the earlier findings have shown that the response is typical N1-P2 component, these measurements were not enough to clarify the differences in terms of amplitude and latency between laboratory testing and archery shooting.

Different paradigms were tested to correlate the potentials measured in the laboratory conditions and during archery shooting.

Some more preliminary testing was also made to analyze if there was any subcomponents of auditory N1-P2 component. To convert the auditory evoked condition to somatosensory one, the subjects have worn on ear phones with high volumes from a radio.

#### **4.1.2. Results of Control Trials**

During the preliminary laboratory measurements, it has been triad to set control paradigms. The paradigms that have been tested are summarized in Table 6. The first trial was selected as the subject by himself has hold the bow handle in his hand and raise the clicker lever and release it as he could see the movement of clicker and could hear the sound from it. As it can be seen from Figure 22, there was no clear N1 response to the fall of the clicker. However, there is a positive going wave peaking about 200 msec after the release of the clicker lever.

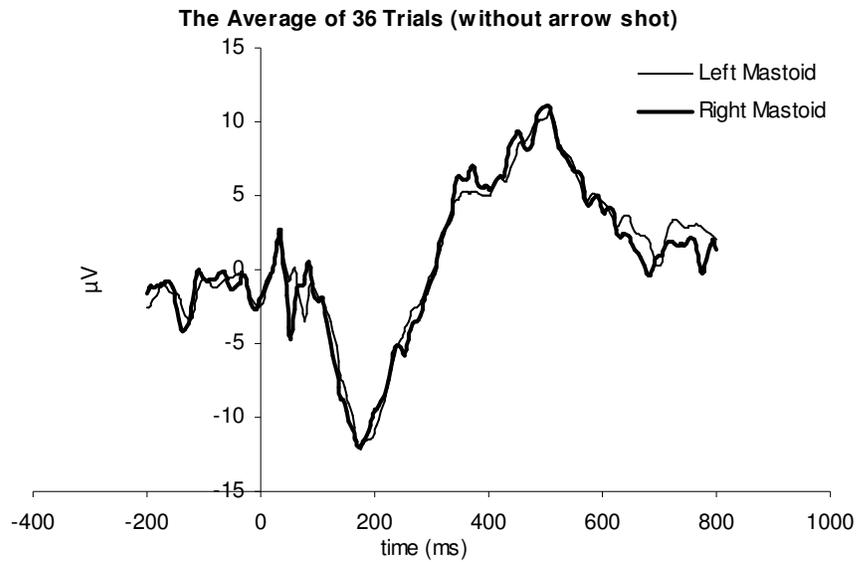
For the second trial for preliminary analysis of the evoking effect of clicker has been shown in Figure 23. Researcher has hold the bow on his hand, raise the clicker lever and release it as the subject could not see the movement of clicker, but could hear its sound. This trail has increased the surprise condition of the evoking sound. As it can be observed from Figure 23 a negative going wave occurring about 100 msec and a positive going wave about 200 msec after the trigger can be evoked by changing trial 1 to trial 2.

Researcher hold the bow-handle in his hand and raise the clicker lever and release it as the subject can see the movement of clicker, but cannot hear the sound from clicker as being the 3<sup>rd</sup> trail (Figure 24). Almost the same results have been measured in trial 3 with trial 1. There is no clear negative going wave was observed at about 100 msec. But a positive going wave has been measured at about 20 msec.

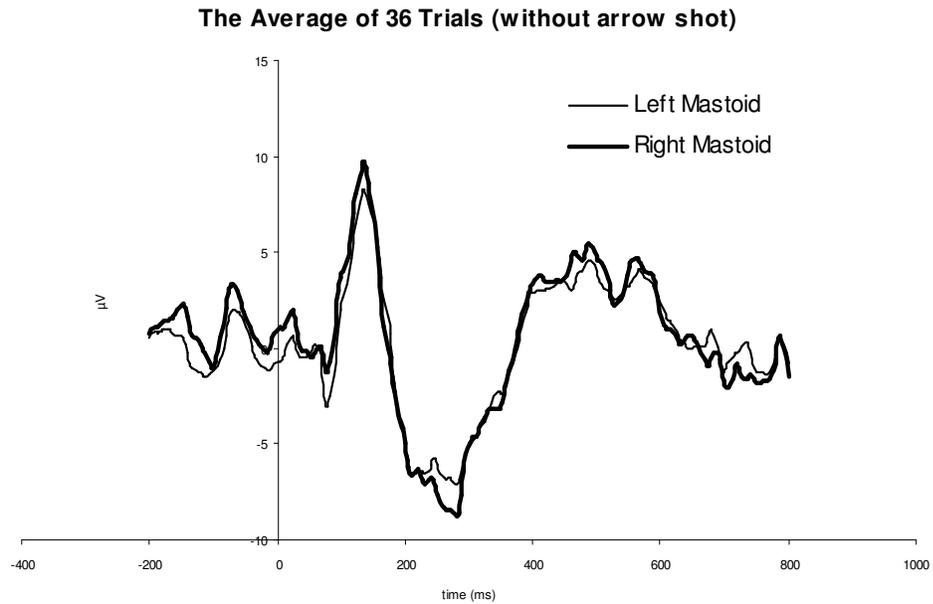
In the 4<sup>th</sup> trial, subject hold the bow handle in his hand and the researcher raise the clicker lever and release it as the subject cannot see the movement of clicker and cannot hear the sound from it. The subject was asked to raise his forefinger when he felt the clicker's fall. Figure 25 illustrates that there has been a negative and a positive going waves as in the trial 2. However, the latency of the evoked potentials has shifted to about 200 msec for N100 and 400 msec for P200 components (Figure 25).

**Table 6.** The control trials during preliminary measurements

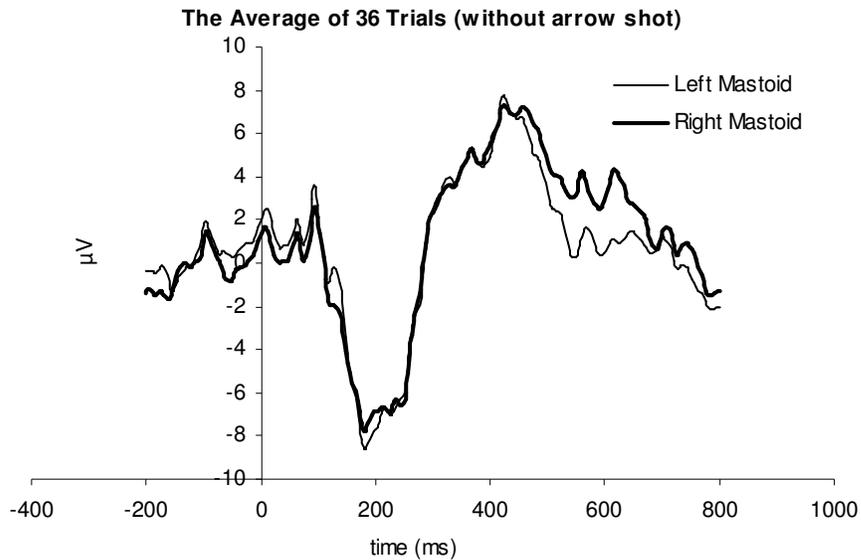
Trial	Who Does	Eyes	Ear	ISI	Task
1	Subject	Sees	Hears	Long	No
2	Researcher	Cannot see	Hears	Long	No
3	Researcher	Sees	Cannot hear	Long	No
4	Researcher	Cannot see	Cannot hear	Long	No
5	Researcher	Cannot see	Cannot hear	Long	Yes



**Figure 22.** Subject hold the bow-handle in his hand and raise the clicker lever and release it as he can see the movement of clicker and can hear the sound from clicker.

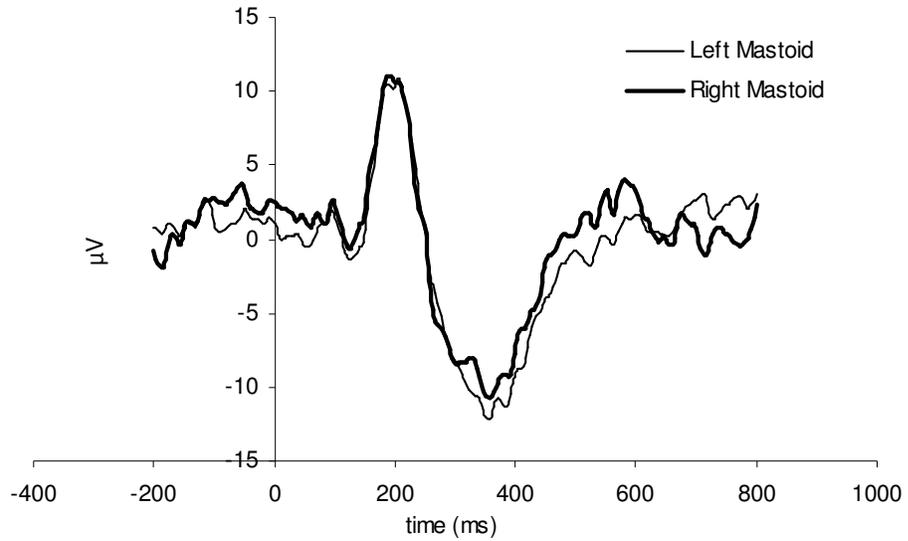


**Figure 23.** Researcher hold the bow-handle in his hand and raise the clicker lever and release it as the subject cannot see the movement of clicker, but can hear the sound from clicker.



**Figure 24.** Researcher hold the bow handle in his hand and raise the clicker lever and release it as the subject can see the movement of clicker, but cannot hear the sound from clicker.

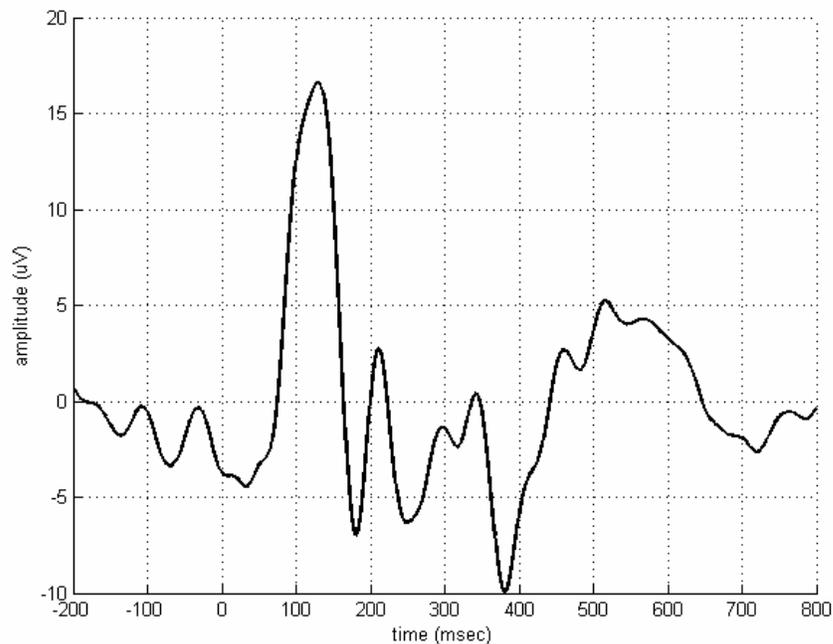
The Average of 36 Trials (without arrow shot with finger rise)



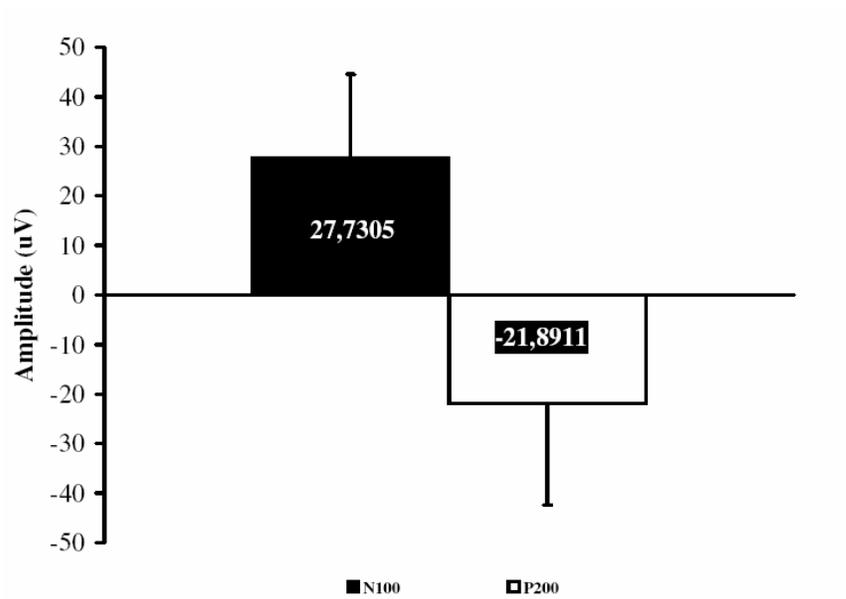
**Figure 25.** Subject hold the bow handle in his hand and the researcher raise the clicker lever and release it as the subject cannot see the movement of clicker and cannot hear the sound from it. The subject was asked to raise his forefinger when he felt the clicker's fall.

#### 4.2. Results concerning the kind of brain potentials that are evoked by the event (fall of clicker) during archery shooting.

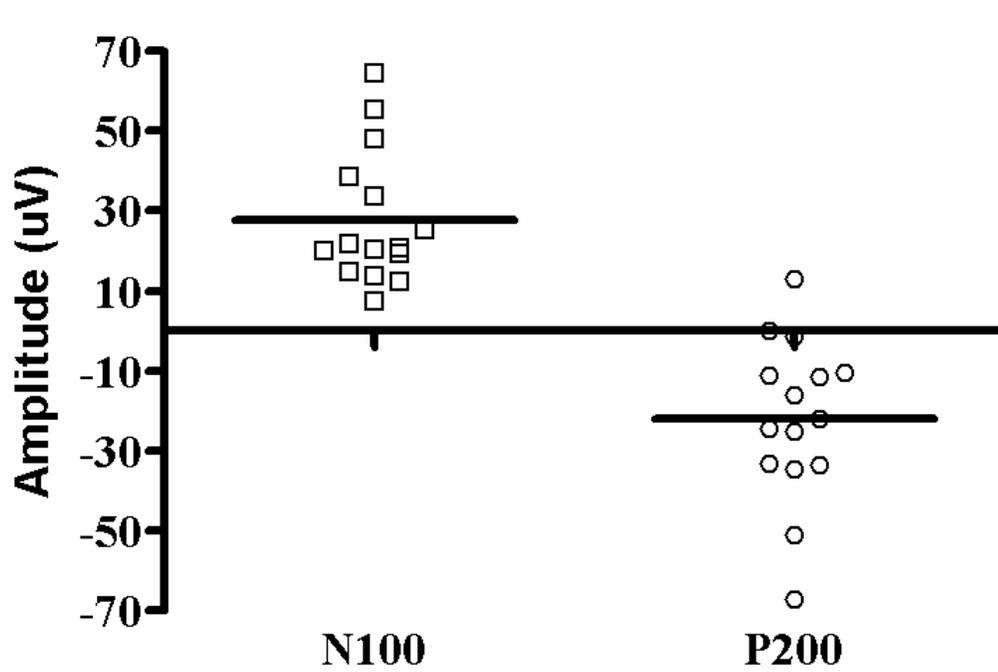
As it can be seen from Figure 26, fall of the clicker during archery shooting evokes a negative going wave (N100) with the amplitude of  $16,5959 \mu\text{V}$  and latency of 129 msec and a positive going wave (P200) with the amplitude of  $-6,9845 \mu\text{V}$  and latency of 180 msec achieved by grand averaging the data set. Figure 26 illustrates the means and standard deviations of both N100 and P200 amplitudes ( $\text{N100} = 27,7305 \pm 16,8281$ ,  $\text{P200} = -21,8911 \pm 20,46$ ) gathered by averaging each subject separately and then averaging for the whole group. The means of both N100 and P200 amplitudes and the values of each subject are illustrated in Figure 27. The latencies of given brain responses are also summarized in terms of means and standard deviations in Figure 28 ( $\text{N100} = 141,933 \pm 41,465$ ;  $\text{P200} = 211,8 \pm 43,974$ ) and the means and the values of each subject in Figure 29.



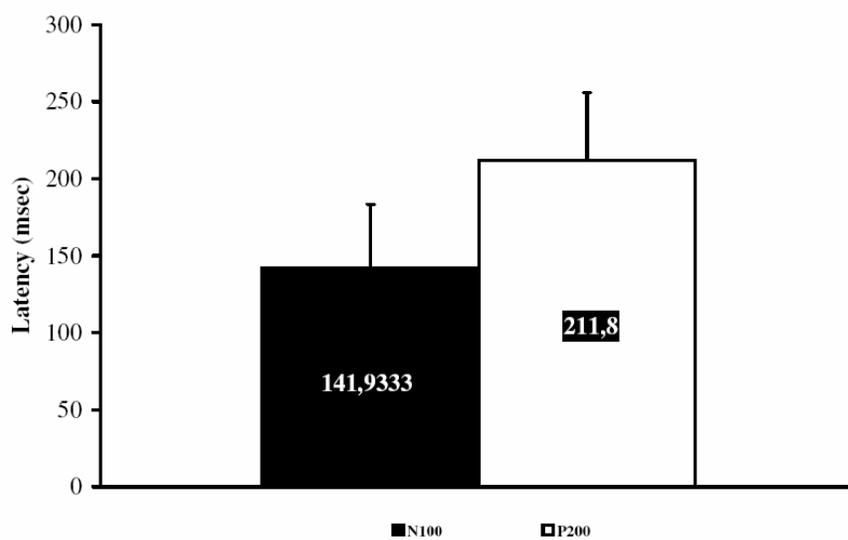
**Figure 26.** Brain electro-potential response to auditory stimulus during archery shooting (grand average (n=15)).



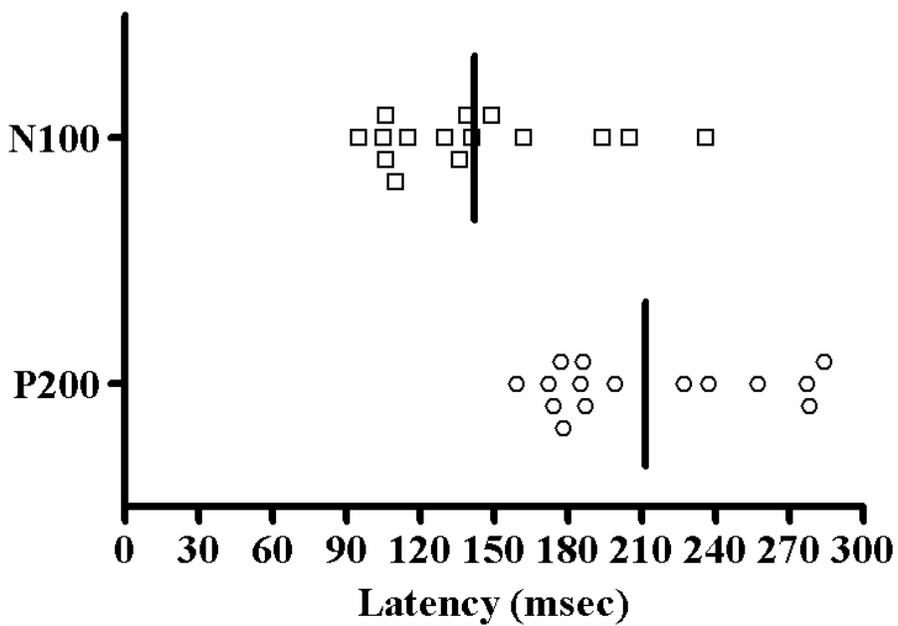
**Figure 26.** Means and standard deviations of N100 and P200 amplitudes as response to the fall of the clicker.



**Figure 27.** Means of N100 and P200 amplitudes as response to the fall of the clicker.



**Figure 28.** Means and standard deviations of N100 and P200 latencies as response to the fall of the clicker.



**Figure 29.** Means of N100 and P200 latencies as response to the fall of the clicker.

**4.3. Interpretation of the results analysing difference between the characteristics of the potentials measured in laboratory conditions and during archery shooting.**

The amplitude and latency values of N100 and P200 components during archery shooting and 6 different trials are summarized in Table 7. Grand mean of AEBP waveforms are presented from each of the experimental conditions and archery shooting (Figure 30, Figure 31, Figure 32). More detailed comparisons are made for amplitude and latency values for both N100 and P200 components in Figure 33, Figure 34, Figure 35 and Figure 36. The comparison between the components of AEBPs during archery shooting and control trials in terms of amplitude and latency are made in following subsections.

**Table 7.** Amplitude and latency values of N100 and P200 components during archery shooting and 6 different trials ( $\mu\text{V}$ ). Values are means ( $\pm$  standard deviations).

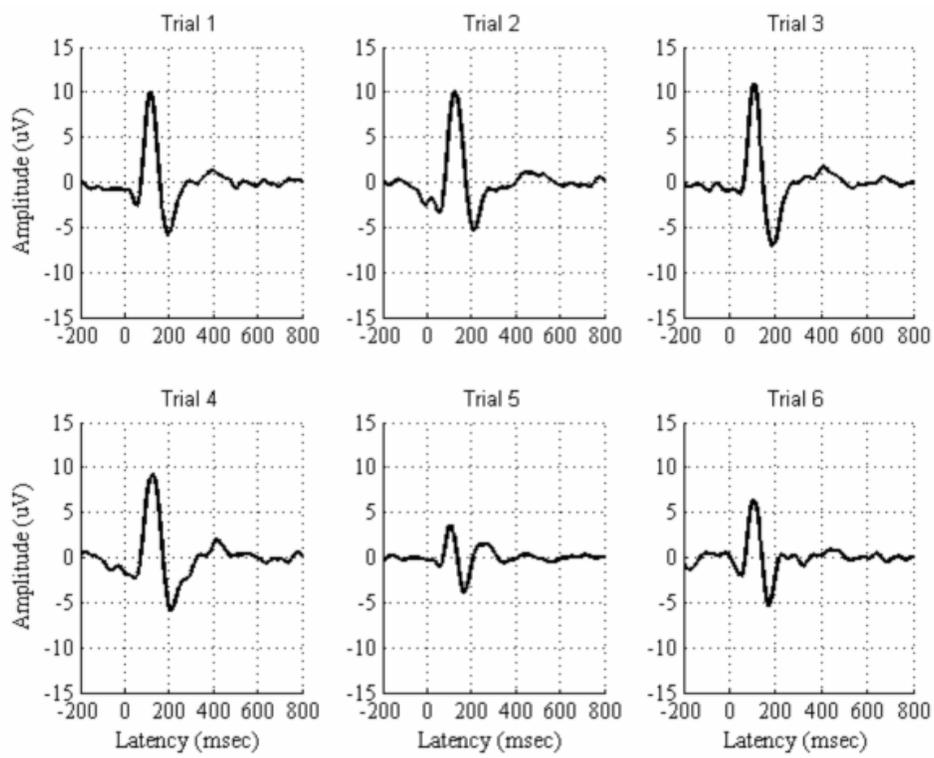
	N100 Amplitude	N100 Latency	P200 Amplitude	P200 Latency
Trial 1	12,14 $\pm$ 5,68*	123,4 $\pm$ 25,44	-10,18 $\pm$ 7,27	187,2 $\pm$ 19,53
Trial 2	11,01 $\pm$ 5,84*	112 $\pm$ 17,33*	-10,69 $\pm$ 3,89	185,7 $\pm$ 38,11
Trial 3	14,53 $\pm$ 7,67	118,7 $\pm$ 20,33	-9,56 $\pm$ 7,08	189,1 $\pm$ 18,34
Trial 4	14,09 $\pm$ 7,01*	132,2 $\pm$ 29,33	-10,36 $\pm$ 7,43	212,3 $\pm$ 40,20
Trial 5	8,80 $\pm$ 5,29*	107,5 $\pm$ 27,86*	-9,68 $\pm$ 7,89	180,9 $\pm$ 30,06
Trial 6	11,19 $\pm$ 4,87*	122 $\pm$ 32,95	-5,645 $\pm$ 6,41*	194,9 $\pm$ 30,45
Arc Shot	27,73 $\pm$ 16,82*	141,93 $\pm$ 41,46*	-21,89 $\pm$ 20,46*	211,8 $\pm$ 43,97

\* Significant difference ( $p < 0.05$ ) between AEBPs during archery shooting and control trials determined by unpaired t test.

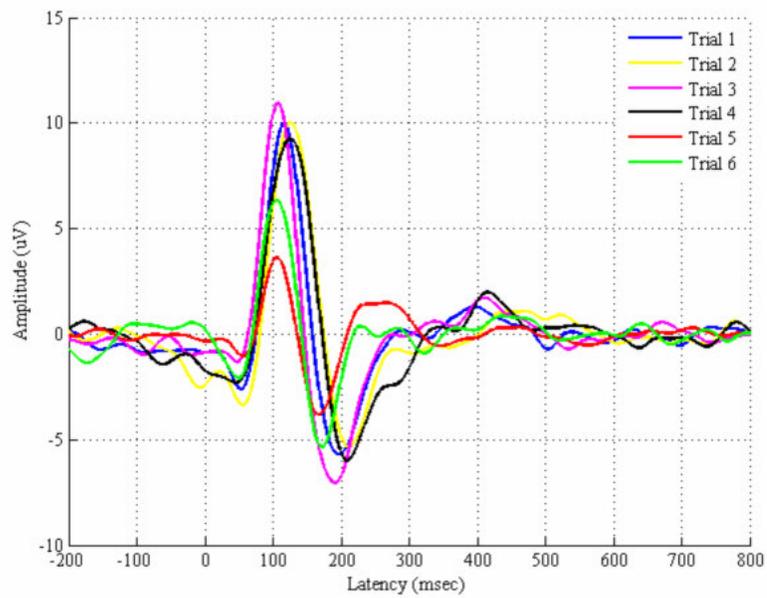
*Question 1: Do the means of N100 amplitudes during Archery shooting and the Trials that have been measured in the laboratory conditions differ significantly?*

**4.3.1. Assumption Test 1: Are the standard deviations equal?**

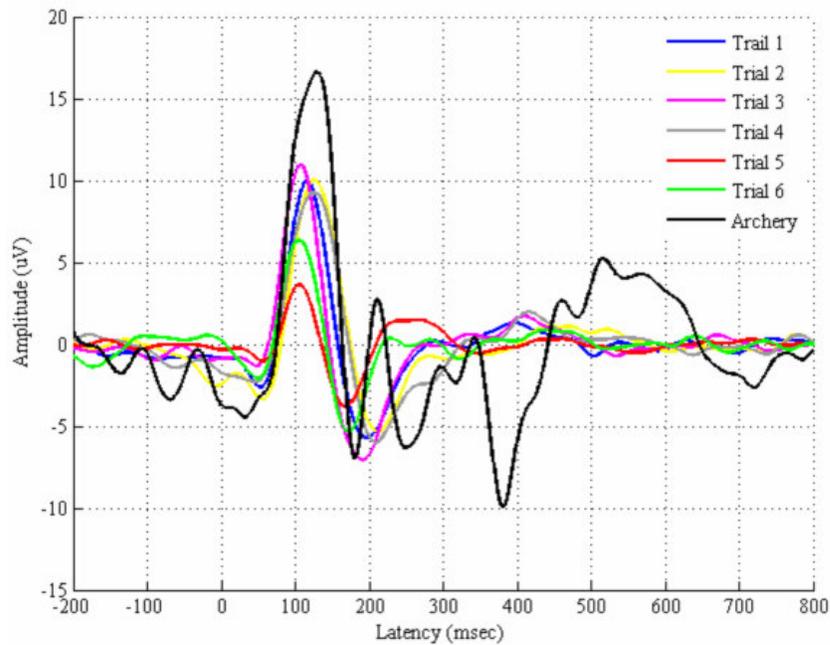
As the t-test assumes that the data set comes from population with equal SDs. During comparing the N100 amplitudes of archery shooting with Trials that are measured in laboratory conditions, the differences between the SDs is not significant ( $p > 0.05$ ).



**Figure 30.** AEBPs during control conditions.



**Figure 31.** AEBPs during control conditions.



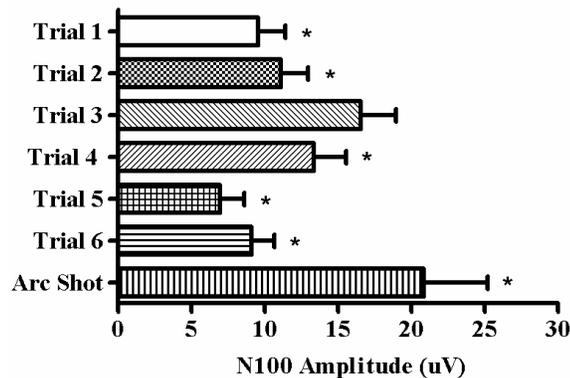
**Figure 32.** AEBPs during archery shooting and control conditions.

#### 4.3.2. Assumption Test 2: Are the data sampled from Gaussian distribution?

The t-test assumes that the data are sampled from populations that follow Gaussian distribution. This assumption is tested using the method Kolmogorov and Smirnov. Trial 1 and Archery Shooting Potentials do not pass the normality testing ( $p < 0.05$ ). Thus, Mann Whitney U test is applied to test median differences between N100 amplitudes during Archery Shooting and the 6 different trials that were measured in the laboratory conditions (Trial 1 to 6).

#### 4.3.3. Result

Two-tailed unpaired t-test has shown that the medians of amplitudes of N100 during Archery Shooting and Trial 1 ( $p = 0.041$ ), Trial 2 ( $p = 0.002$ ), Trial 4 ( $p = 0.019$ ), Trial 5 ( $p = 0.0001$ ) and Trial 6 ( $p = 0.0015$ ) differ significantly. However, the difference between the N100 amplitudes during archery shooting and Trial 3 ( $p = 0.062$ ) is not significant (Figure 33).



**Figure 33.** Medians and ranges of N100 amplitudes during Archery shooting and the Trials that have been measured in the laboratory condition (\*  $p < 0.05$ ).

*Question 2: Do the means of N100 latencies during Archery shooting and the Trials that have been measured in the laboratory conditions differ significantly?*

#### 4.3.4. Assumption Test 1: Are the standard deviations equal?

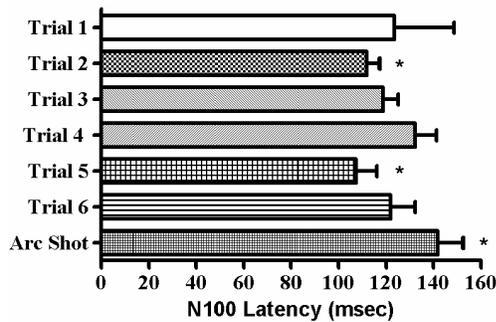
As the t-test assumes that the data set comes from population with equal SDs. During comparing the N100 latencies of archery shooting with Trials that are measured in laboratory conditions, the differences between the SDs is not significant ( $p > 0.05$ ).

#### 4.3.5. Assumption Test 2: Are the data sampled from Gaussian distribution?

The t-test assumes that the data are sampled from populations that follow Gaussian distribution. This assumption is tested using the method Kolmogorov and Smirnov. All of the data sets pass the normality testing.

#### 4.3.6. Result

Two-tailed unpaired t-test has shown that the means of latencies of N100 during Archery Shooting and Trial 1 ( $p = 0.220$ ), Trial 3 ( $p = 0.115$ ), Trial 4 ( $p = 0.527$ ), and Trial 6 ( $p = 0.215$ ) not differ significantly. However, the difference between the N100 latencies during archery shooting and Trial 2 ( $p = 0.042$ ) and Trial 5 ( $p = 0.031$ ) is significant (Figure 34).



**Figure 34.** Means and standard deviations of N100 latencies during Archery shooting and Trials that have been measured in the laboratory condition (\*  $p < 0.05$ ).

*Question 3: Do the means of P200 amplitudes during Archery shooting and the Trials that have been measured in the laboratory conditions differ significantly?*

#### 4.3.7. Assumption Test 1: Are the standard deviations equal?

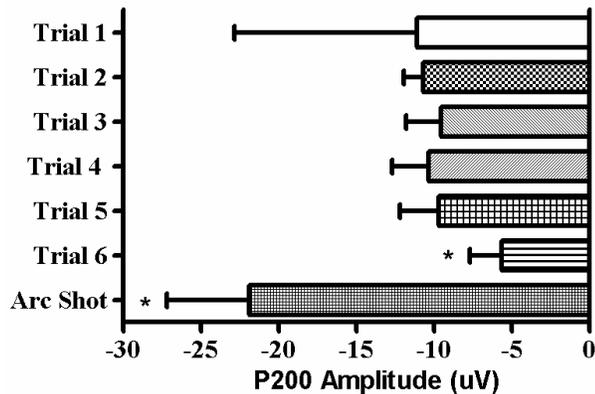
As the t-test assumes that the data set comes from population with equal SDs. During comparing the P200 amplitudes of archery shooting with Trials that are measured in laboratory conditions, the differences between the SDs is not significant ( $p > 0.05$ ).

#### 4.3.8. Assumption Test 2: Are the data sampled from Gaussian distribution?

The t-test assumes that the data are sampled from populations that follow Gaussian distribution. This assumption is tested using the method Kolmogorov and Smirnov. At least one of the Trials and/or Archery Shooting P200 amplitudes do not pass the normality testing ( $p < 0.05$ ). Thus, Mann Whitney U test is applied to test median differences between P200 amplitudes during Archery Shooting and the 6 different trials that were measured in the laboratory conditions (Trial 1 to 6).

#### 4.3.9. Result

Two-tailed unpaired t-test has shown that the medians of amplitudes of N100 during Archery Shooting and Trial 1 ( $p = 0.091$ ), Trial 2 ( $p = 0.115$ ), Trial 3 ( $p = 0.102$ ), Trial 4 ( $p = 0.062$ ) and Trial 5 ( $p = 0.070$ ) not differ significantly. However, the difference between the P200 amplitudes during archery shooting and Trial 6 ( $p = 0.011$ ) is significant (Figure 35).



**Figure 35.** Medians and ranges of P200 amplitudes during Archery shooting and the Trials that have been measured in the laboratory condition (\*  $p < 0.05$ ).

*Question 4: Do the means of P200 latencies during Archery shooting and the Trials that have been measured in the laboratory conditions differ significantly?*

#### 4.3.10. Assumption Test 1: Are the standard deviations equal?

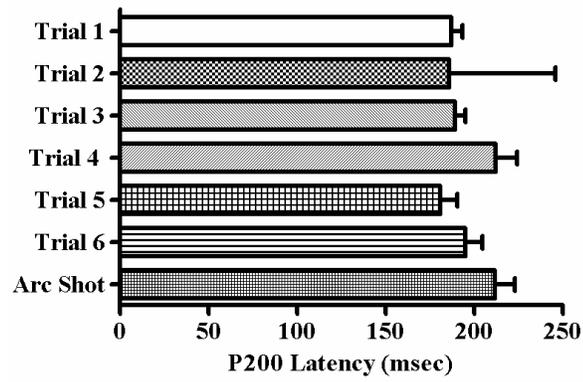
As the t-test assumes that the data set comes from population with equal SDs. During comparing the P200 latencies of archery shooting with Trials that are measured in laboratory conditions, the differences between the SDs is not significant ( $p > 0.05$ ).

#### 4.3.11. Assumption Test 2: Are the data sampled from Gaussian distribution?

The t-test assumes that the data are sampled from populations that follow Gaussian distribution. This assumption is tested using the method Kolmogorov and Smirnov. At least one of the Trials and/or Archery Shooting P200 latencies do not pass the normality testing ( $p < 0.05$ ). Thus, Mann Whitney U test is applied to test median differences between P200 latencies during Archery Shooting and the 6 different trials that were measured in the laboratory conditions (Trial 1 to 6).

#### 4.3.12. Result

Two-tailed unpaired t-test has shown that the medians of amplitudes of N100 during Archery Shooting and Trial 1 ( $p = 0.233$ ), Trial 2 ( $p = 0.260$ ), Trial 3 ( $p = 0.488$ ), Trial 4 ( $p = 0.955$ ), Trial 5 ( $p = 0.085$ ) and Trial 6 ( $p = 0.683$ ) not differ significantly (Figure 36).



**Figure 36.** Medians and ranges of P200 latencies during Archery shooting and the Trials that have been measured in the laboratory condition.

**4.4. Results analysing the difference between the successful and unsuccessful shots in terms of Auditory Evoked Brain Potentials.**

Mean amplitude ( $\mu\text{V}$ ) and latency (msec) values of N100 and P200 components corresponding to hit and miss areas during archery shooting are summarized in Table 8. There is no significant difference between the AEBPs corresponding to hit and miss areas in terms of amplitude and latency for both N100 and P200. More detailed comparisons are made for amplitude and latency values for both N100 and P200 components corresponding to hit and miss areas in Figure 38, Figure 38.

**Table 8.** Amplitude ( $\mu\text{V}$ ) and latency (msec) values of N100 and P200 components corresponding to hit and miss areas during archery shooting. Values are means ( $\pm$  standard deviation).

N100 Amplitude		N100 Latency		P200 Amplitude		P200 Latency	
Hit	Miss	Hit	Miss	Hit	Miss	Hit	Miss
27,85	26,60	116,98	116,90	-18,68	-16,63	198,53	201,53
$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$
15,41	20,04	15,60	16,84	20,68	20,27	30,35	29,57

*Question 1: Do the means of N100 and P200 amplitudes that corresponding to successful and unsuccessful shots differ significantly?*

**4.4.1. Assumption Test 1: Are the standard deviations equal?**

As the t-test assumes that the data set comes from population with equal SDs. The following calculations test that assumption (Table 9).

**Table 9.** Assumption testing for N100 and P200 amplitudes.

	<b>N100</b>	<b>P200</b>
<b>Assumption Test 1</b>	F = 1.686	F=1,041
	P=0.3398	P=0.9415

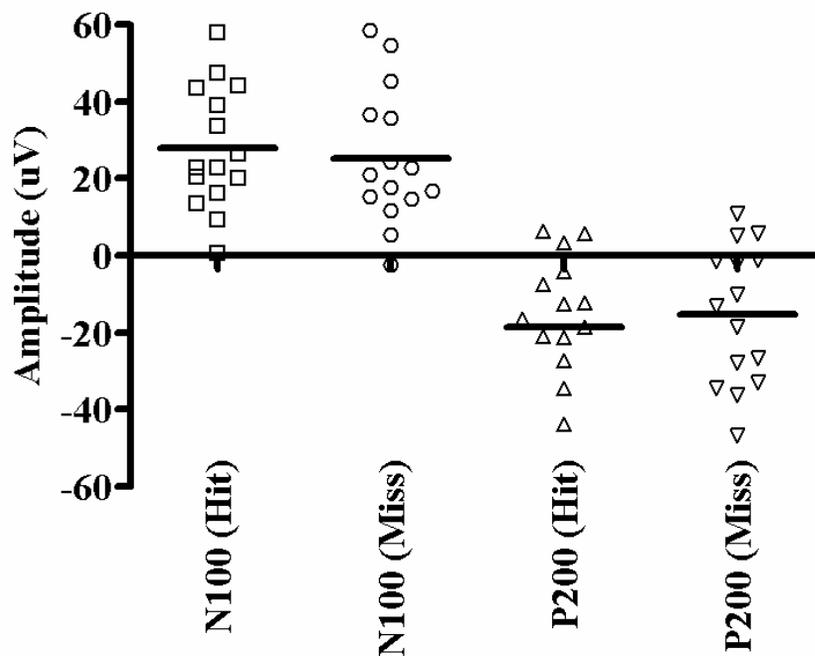
These tests suggest that the difference between the two SDs is not significant for both N100 and P200 amplitudes corresponding to successful and unsuccessful shots.

#### 4.4.2. Assumption Test 2: Are the data sampled from Gaussian distribution?

The t-test assumes that the data are sampled from populations that follow Gaussian distribution. This assumption is tested using the method Kolmogorov and Smirnov. Both data sets pass the normality testing.

#### 4.4.3. Result

Two-tailed paired samples t-test has shown that the means of N 100 ( $P=0.8550$ ) and P 200 ( $P=0.7929$ ) amplitudes corresponding to successful and unsuccessful shots do not differ significantly (Figure 37).



**Figure 37.** Comparison of the N 100 and P200 amplitudes corresponding to successful and unsuccessful shots separately.

*Question 2: Do the means of N100 and P200 latencies that corresponding to successful and unsuccessful shots differ significantly?*

#### 4.4.4. Assumption Test 1: Are the standard deviations equal?

As the t-test assumes that the data set comes from population with equal SDs. The following calculations test that assumption (Table 10).

**Table 10.** Assumption testing for N100 and P200 latencies.

	<b>N100</b>	<b>P200</b>
<b>Assumption Test 1</b>	F = 1.175	F=1,053
	P=0.7671	P=0.9246

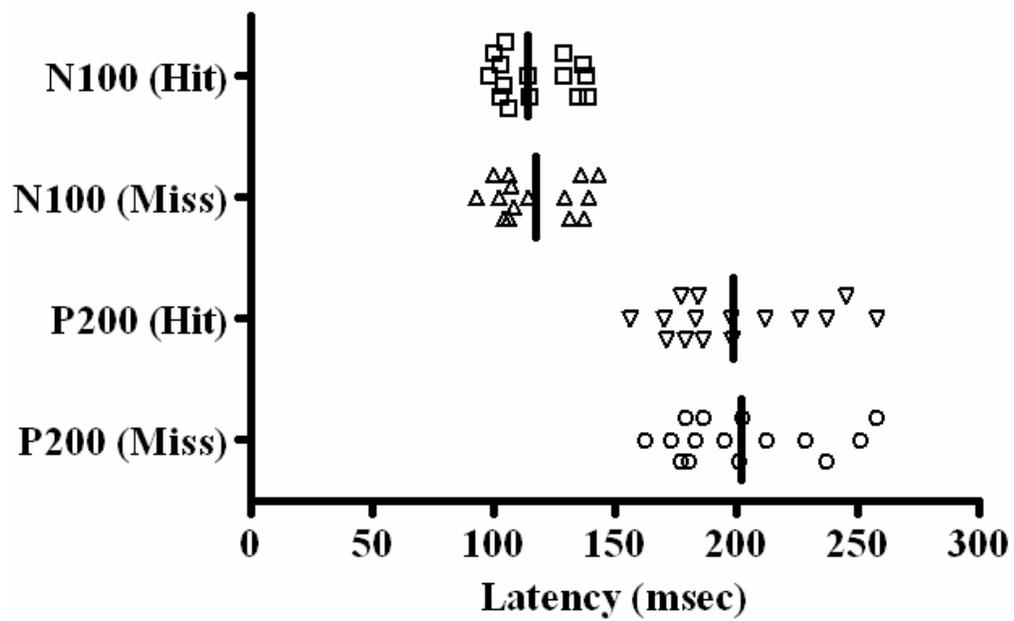
These tests suggest that the difference between the two SDs is not significant for both N100 and P200 latencies corresponding to successful and unsuccessful shots.

#### 4.4.5. Assumption Test 2: Are the data sampled from Gaussian distribution?

The t-test assumes that the data are sampled from populations that follow Gaussian distribution. This assumption is tested using the method Kolmogorov and Smirnov. P200 latencies pass the normality testing. However, N100 latencies failed the normality test with  $p < 0.05$ . Thus, Mann Whitney U test is applied to test median differences between N100 latencies for successful and unsuccessful shots.

#### 4.4.6. Result

Two-tailed paired samples t-test has shown that the means of P200 ( $P=0.7860$ ) and medians of N100 ( $P=0.8682$ ) latencies corresponding to successful and unsuccessful shots do not differ significantly (Figure 38).



**Figure 38.** Comparison of Medians of N 100 and P200 latencies corresponding to successful and unsuccessful shots separately.

#### 4.5. Results analysing the effect archery shooting session on Auditory Evoked Brain Potentials.

*Question 1: Do the means of N100 and P200 amplitudes for “trial 1” differ significantly before and after archery shooting?*

##### 4.5.1. Assumption Test 1: Are the standard deviations equal?

As the t-test assumes that the data set comes from population with equal SDs. The following calculations test that assumption.

**Table 11.** Assumption testing for N100 and P200 amplitudes before and after archery shooting for trial 1.

	<b>N100</b>	<b>P200</b>
<b>Assumption Test 1</b>	F = 1.145	F=1,113
	P=0.8063	P=0.8438

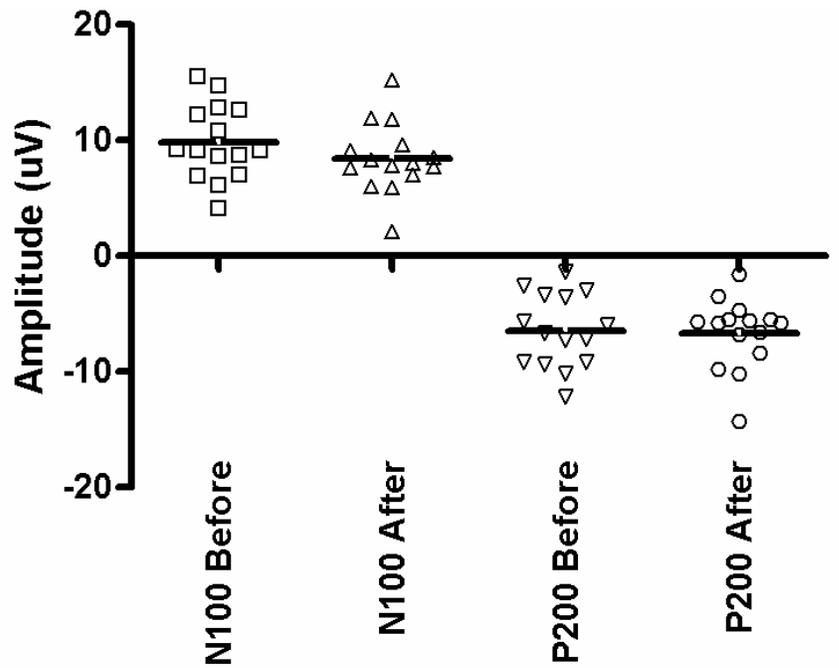
These tests suggest that the difference between the two SDs before and after the archery shooting is not significant for both N100 and P200 amplitudes (Table 11).

##### 4.5.2. Assumption Test 2: Are the data sampled from Gaussian distribution?

The t-test assumes that the data are sampled from populations that follow Gaussian distribution. This assumption is tested using the method Kolmogorov and Smirnov. Both data sets pass the normality testing.

##### 4.5.3. Result

Two-tailed t-test has shown that the means of N100 (P=0.2371, t=1.208) and P200 (P=0.8846, t=0.1465) amplitudes for successful and unsuccessful shots do not differ significantly (Figure 39).



**Figure 39.** Means and standard deviations of N100 and P200 amplitudes for “trial 1” before and after the archery shooting.

*Question 2:* Do the means of N100 and P200 latencies for “trial 1” differ significantly before and after the archery shooting?

**4.5.4. Assumption Test 1: Are the standard deviations equal?**

As the t-test assumes that the data set comes from population with equal SDs. The following calculations test that assumption.

**Table 12.** Assumption testing for N100 and P200 latencies before and after archery shooting for trial 1.

	<b>N100</b>	<b>P200</b>
<b>Assumption Test 1</b>	F = 1.839	F=1,665
	P=0.2663	P=0.3513

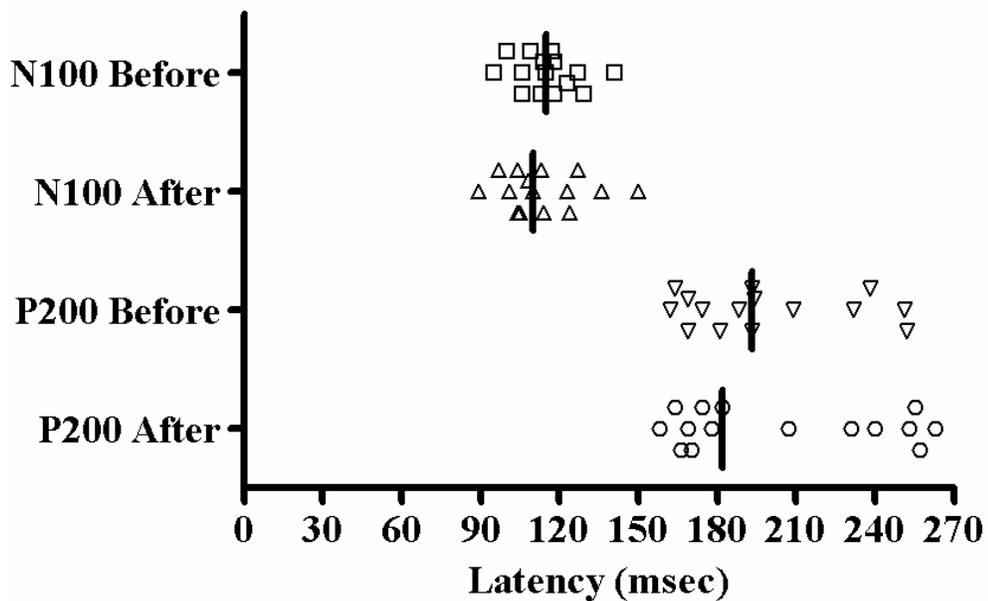
These tests suggest that the difference between the two SDs before and after the archery shooting is not significant for both N100 and P200 amplitudes (Table 12).

#### 4.5.5. Assumption Test 2: Are the data sampled from Gaussian distribution?

The t-test assumes that the data are sampled from populations that follow Gaussian distribution. This assumption is tested using the method Kolmogorov and Smirnov. N100 latencies pass the normality testing. However, P200 latencies failed the normality test with  $p < 0.05$ . Thus, Mann Whitney U test is applied to test median differences between P200 latencies for successful and unsuccessful shots.

#### 4.5.6. Result

Two-tailed t-test has shown that the means of N100 ( $P=0.7367$ ,  $t=0.3395$ ) and medians of P200 ( $P=0.7715$ ) latencies for “trial 1” before and after archery shooting (Figure 40).



**Figure 40.** Medians and ranges of N100 and P200 latencies for “trial 1” before and after the archery shooting.

*Question 3: Do the means of N100 and P200 amplitudes for “trial 5” differ significantly before and after the archery shooting?*

#### 4.5.7. Assumption Test 1: Are the standard deviations equal?

As the t-test assumes that the data set comes from population with equal SDs. The following calculations test that assumption.

**Table 13.** Assumption testing for N100 and P200 amplitudes before and after archery shooting for trial 5.

	<b>N100</b>	<b>P200</b>
<b>Assumption Test 1</b>	F = 2.680	F=1,016
	P=0.075	P=0.9761

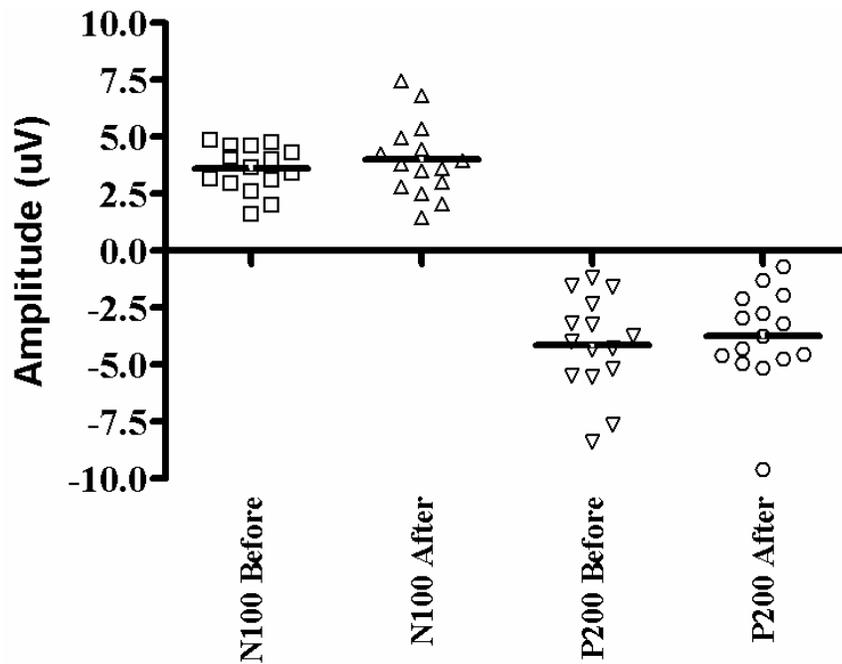
These tests suggest that the difference between the two SDs before and after the archery shooting is not significant for both N100 and P200 amplitudes (Table 13).

#### 4.5.8. Assumption Test 2: Are the data sampled from Gaussian distribution?

The t-test assumes that the data are sampled from populations that follow Gaussian distribution. This assumption is tested using the method Kolmogorov and Smirnov. Both data sets pass the normality testing.

#### 4.5.9. Result

Two-tailed t-test has shown that the means of N100 (P=0.4116, t=0.8336) and P200 (P=0.6535, t=0.4537) amplitudes for “trail 5” before and after archery shooting (Figure 41).



**Figure 41.** Means and standard deviations of N100 and P200 amplitudes for “trial 5” before and after the archery shooting.

*Question 4: Do the means of N100 and P200 latencies for “trial 5” differ significantly before and after the archery shooting?*

**4.5.10. Assumption Test 1: Are the standard deviations equal?**

As the t-test assumes that the data set comes from population with equal SDs. The following calculations test that assumption.

**Table 14.** Assumption testing for N100 and P200 latencies before and after archery shooting for trial 5.

	<b>N100</b>	<b>P200</b>
<b>Assumption Test 1</b>	F = 3.755	F=1,653
	P=0.018	P=0.3583

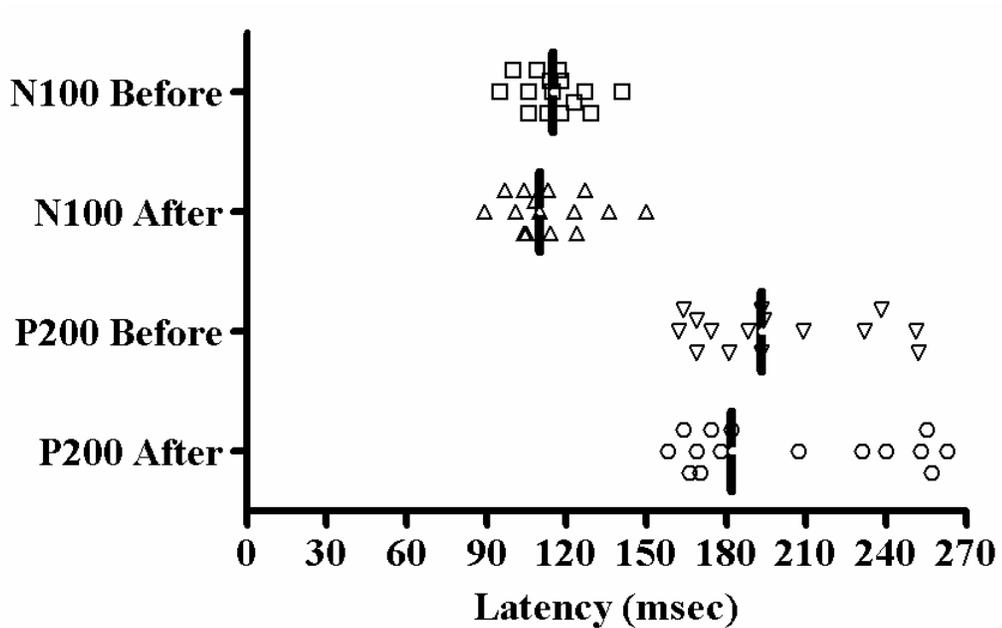
These tests suggest that the difference between the two SDs before and after the archery shooting is not significant for both N100 and P200 amplitudes (Table 14).

#### 4.5.11. Assumption Test 2: Are the data sampled from Gaussian distribution?

The t-test assumes that the data are sampled from populations that follow Gaussian distribution. This assumption is tested using the method Kolmogorov and Smirnov. N100 latencies pass the normality testing. However, P200 latencies failed the normality test with  $p < 0.05$ . Thus, Mann Whitney U test is applied to test median differences between P200 latencies for successful and unsuccessful shots.

#### 4.5.12. Result

Two-tailed t-test has shown that the means of N100 ( $P=0.5272$ ,  $t=0.6402$ ) and medians of P200 ( $P=0.74$ ) latencies for “trail 5” before and after archery shooting (Figure 41).



**Figure 42.** Medians and range of N100 and P200 latencies for “trial 5” before and after the archery shooting.

## CHAPTER V

### DISCUSSION, IMPLICATIONS, AND RECOMMENDATIONS

In this chapter, discussion regarding the results of the current study is presented by following the four major questions of the study. Each section introduces the discussion related with the results presented in the previous chapter.

#### **5.1. Discussion regarding the kind of brain potentials that are evoked by the event (fall of clicker) during archery shooting.**

Both the preliminary measurements and the results of the current study has shown that the sound from the fall of the clicker during archery shooting evokes a negative (N100) and a positive (P200) going waves. The findings have been summarized in Results' chapter (Amplitudes: N100 =  $27,7305 \pm 16,8281$ , P200 =  $-21,8911 \pm 20,46$ ; Latencies: N100 =  $141,933 \pm 41,465$ ; P200 =  $211,8 \pm 43,974$ ).

Any auditory stimuli elicit a series of long latency ERPs. In the 80–200 msec latency range ERPs mainly consist of two peaks, the first with negative polarity, called N1/N100, occurring roughly 75–150 msec after stimulus onset and the second with positive polarity, P2/P200, with a latency of approximately 150–250 msec. These two components are assumed to at least partially represent an exogenous response, that is, they are elicited by both attended and non-attended stimuli (Ford et al., 1976, 1999; Oades et al., 1997; Cited in Crowleya and Colrain, 2004).

Almost a half-century ago, P.A. Davis (1939) described the sound-evoked changes in the electroencephalogram of the waking human brain (Picton, 1988). However, there was no literature on the specific sound coming from the fall of the clicker in archery shooting. The findings of the current study have shown that the sound from the clicker is anticipated as a regular sound used in biophysical applications in terms of the waveforms after receiving the auditory stimulus. Fall of the clicker, first evokes a

negative (N1/N100) and than a positive going wave (P2/P200). The N1 wave, the most prominent deflection of the human auditory evoked potential, is a broad negativity over the fronto-central scalp that begins at 60-80 msec and can last until 160 msec after the onset of a sound (Woods, 1995).

Several different cerebral processes contribute to the N1 wave of the scalp-recorded auditory EP. These “component” processes occur in different cerebral locations and sub serve different psycho-physiological functions. They are distinguished by their characteristic electrical and/or magnetic fields and by their specific relationship to various experimental manipulations (Näätänen and Picton, 1987). Figure 4 shows auditory ERPs elicited by tones of different frequencies presented at high rates in a dichotic selective attention task.

The P2 is usually referred to in the context of the tail end of the N1-P2 complex or the ‘vertex potential’ (Crowley and Colrain, 2004) and occurs with a latency of approximately 150-250 msec. Studies on the P2 as an independent component, however, are relatively scarce and thus little is known regarding its endogenous or exogenous processing correlates.

## **5.2. Discussion regarding the results analysing difference between the characteristics of the potentials measured in laboratory conditions and during archery shooting.**

During the piloting of the current research, some preliminary trials were made to decide the control trails using real “recurve bow” and “clicker”. The results of the preliminary analysis have been summarized in chapter of results. The idea of using bow and clicker for control trials without arrow shot could help the researchers to imitate the real archery conditions. But the research group has confronted some standardization problems with the sound of clicker and the onset of sound from clicker. Because of that limitation, it has been decided to create some control conditions which does not cause any biased measurement. The following paragraph

summarizes the control experiments which enables the researchers to make comparisons between ERPs during archery shooting and control conditions. It should be noted that all tones were 1000 Hz for each control condition.

(1) *Paradigm 1*: Subject presses to the enter button and sound is delivered with 1–3 s delay. When the subject hears the sound he/she responds to the stimulus by raising his/her forefinger without any wrist movement. The subject was asked to press the button with 8–10 sec intervals. As long as subject initiated the sound in between 1-3 s delay increases the temporal uncertainty of the stimuli and attend condition has been added by responding to the stimuli in the first paradigm. (2) *Paradigm 2*: The stimulus is delivered automatically by the computer with an ISI of 8-10 s. When the subject hears the sound he/she responds to the stimulus by raising his/her forefinger without any wrist movement. Like in the 1<sup>st</sup> paradigm temporal uncertainty and attend condition has been observed in the 2<sup>nd</sup> paradigm. The only difference between the 1<sup>st</sup> and 2<sup>nd</sup> paradigms is the machine delivery of the stimulus in the 2<sup>nd</sup> paradigm. (3) *Paradigm 3*: Subject presses the enter button and sound is delivered with 1–3 s delay. The subject does not respond to the sound with any type of movement patterns. The subject was asked to press the button with 8–10 sec intervals. 3<sup>rd</sup> paradigm resembles the 1<sup>st</sup> one. But the 3<sup>rd</sup> paradigm does not have any task. So, 3<sup>rd</sup> paradigm has temporal uncertainty but does not have “attend” condition. (4) *Paradigm 4*: The stimulus is delivered automatically by the computer with an ISI of 8-12 sec. When the subject hears the sound, he/she responds to the stimulus by raising his/her forefinger without any wrist movement. 4<sup>th</sup> paradigm resembles the 2<sup>nd</sup> one. The only difference is not having any task in 4<sup>th</sup> paradigm. Like in the 3<sup>rd</sup> paradigm, temporal uncertainty is increased but there is no task in the 4<sup>th</sup> control paradigm. (5) *Paradigm 5*: The stimulus is delivered automatically by the computer with an ISI of 1 sec. The subject does not respond to the sound with any type of movement patterns. In the 5<sup>th</sup> paradigm the computer delivers the sound pip. This paradigm has no timing uncertainty and task. (6) *Paradigm 6*: The stimulus is delivered as soon as the subject presses the enter button. There was no task in paradigm 6. The difference between 5<sup>th</sup> and 6<sup>th</sup> paradigms is the self delivery of the

sound pip in the 6<sup>th</sup> one. There is no timing uncertainty and task in 6<sup>th</sup> paradigm like in the 5<sup>th</sup> one (Table 4).

Archery shooting has a stable sequence and it includes the stance, holding, drawing, full drawing, aiming, releasing and follow-through phases. Each of these phases represents a stable sequence of the collective movements. An archer is supposed to hold the string by three-finger hook in the drawing hand (Ertan et al., 2005). Related with responding to the fall of the clicker or the click sound, it was established that archers develop a specific forearm flexor and extensor muscular strategy to accurately shoot an arrow to a given target after the fall of the clicker. Active contraction of the M. extensor digitorum and gradual relaxation of the M. flexor digitorum superficialis is an integral part of this strategy (Ertan et al., 2003). So, responding to the fall of the clicker or the click sound is not a simple activity. Archery shooting involves responding to the click sound like in the control condition that has been conducted in laboratory condition. However, archery shooting paradigm involves both temporal uncertainty and attend condition. It should be mentioned that attend condition does not have a simple movement pattern. The task that archer should accomplish to respond to the sound from clicker is a demanding movement pattern.

The following paragraph includes the possible reasons why ERPs during archery shooting has higher amplitudes than that of control conditions. The sub-components of N1 and P2 waves should be understood to explain this significant difference between archery shooting and control conditions.

Woods (1995) has published a review article on the classification of the components of N1 wave and he has focused on the generators of the “true N1” wave. He has impressed on the Wolpaw and Penry’s (1975) proposal that the N1 consisted of midline and temporal components (Table 2). Midline components, including the N1’/P90 and N1b, can be modelled with tangential generators on the superior temporal plane (STP) and pointing toward the midline of the scalp. There appear to

be at least two midline components distinguished by the tonotopy of their generators (Figure 5, left panel). The early frontocentral peak (N1' peak latency 90-110 msec) shows reliable tonotopic changes in distribution (Bertrand et al. 1991; Woods et al. 1991; Woods 1992; Woods et al. 1993b, Cited in Woods, 1995). It is frontally distributed for high frequency tones, centrally distributed for middle frequencies, and posteriorly distributed for low frequencies. The P90 component is seen over posterior temporal regions only for high frequency tones. The N1'/P90 subcomponent is modulated by selective attention (Woods et al. 1994, Cited in Woods, 1995). The later midline component, the N1b, peaks at 120-130 msec over fronto-central sites. It shows a similar distribution for tones of different frequency.

Double-peaked waveforms are usually recorded at mid-temporal sites (Figure 5, right panel). These consist of an early negativity peaking at 80-85 msec, positivity at 95-105 msec, and a later negativity peaking at 140-160 msec. These mid-temporal negativities N1a and N1c, with the positivity between them labelled Ta, after Wolpaw and Penry (1975) (Cited in Woods, 1995). The N1a is larger over the left hemisphere (McCallum and Curry 1980; Knight et al. 1988; Woods et al. 1993c, Cited in Woods, 1995) while the N1c is larger over the right hemisphere (Cacace et al. 1988, Cited in Woods, 1995).

Both midline and lateral temporal components are enhanced in amplitude over the hemisphere contralateral to the stimulated ear (Cacace et al. 1988; Knight et al. 1988; Woods et al. 1992; Connolly 1993, Cited in Woods, 1995). The lateral temporal components do not show tonotopic displacements, although frequency-related differences in peak latency can be noted (Figure 5, right panel). Both midline and lateral temporal components are reduced during drowsiness and sleep (Nielsen-Bohlman et al. 1991, Cited in Woods, 1995).

All of the archers volunteered in the current study were right-handed. Thus, they were all drawing the bow string with their right hand and pushing the bow handle with their left hand. As clicker being placed on the sight window of bow handle, the

sound from the clicker has been delivered during all of the shots. Measuring the highest amplitude on the right mastoid electrode referenced to C<sub>z</sub> electrode can be explained by clicker evoking the contralateral side of the hemisphere. The click sound first reaches to left ear and then right one.

P.A. Davis (1939) described the sound-evoked changes in the electroencephalogram of the waking human brain. The onset of a tone elicited a negative-positive wave that was larger at the vertex than at the occipital, frontal, or temporal regions of the scalp. A similar evoked potential (EP) occurred following the offset of the tone which lasted “a few seconds”. In the published figures, the peak latency of the negative wave varies from 100 to 150 ms (Näätänen and Picton, 1987).

The vertex response may not represent a single cerebral event since the different waves of the response respond differently to experimental manipulation and have different scalp distributions. In the auditory modality, the latencies of the N1 and P2 peaks are about 100 and 175 ms, respectively; in the visual and somatosensory modalities the latencies are 30-40 ms longer. Another nomenclature identifies the waves by their polarity and their usual latency - N100 and P175 would be equivalent to N1 and P2, respectively, in the auditory response (Näätänen and Picton, 1987).

The EP consists of a sequence of positive and negative waves or peaks. Although these deflections in the waveform provide a convenient point for measurement, they are not necessarily generated by individual cerebral events. At any point in time, multiple cerebral processes may contribute to the EP waveform (Näätänen and Picton, 1987).

As will become evident, many different processes generate negative waves during the latency of the N1-between 50 and 200 ms after the onset of an auditory stimulus. We shall distinguish three “true” or “obligatory” components that are mainly controlled by the physical and temporal features of the stimulus and by the general state of the subject. These components can be distinguished from each other by their

different source locations within the brain, and by their different sensitivity to stimulus features and state factors (Näätänen and Picton, 1987).

In addition we shall distinguish three other negative components which are not obligatorily evoked by a stimulus but which depend upon the conditions under which the stimulus occurs. These components overlap the true N1 components and often have a considerably longer duration. One of these components, the “mismatch negativity,” is not elicited by any particular stimulus but occurs only when a stimulus differs from those preceding it in a homogeneous sequence. The remaining two components are elicited during selective attention as components of the “processing negativity” (Näätänen and Picton, 1987).

Some of the control conditions can be considered as “true or obligatory N1”. Especially 5<sup>th</sup> and 6<sup>th</sup> paradigms do not have temporal uncertainty and attend condition. However, the other paradigms and archery shooting can be classified as “true or obligatory N1” because of having no temporal uncertainty and attend condition. This finding illustrates that 1 to 4 control conditions and archery shooting do not evoke “true N1” component. What may create the difference between archery shooting and control conditions is the meaning of the sound from the clicker to an archer and the complexity of the task that archer should accomplish. Moreover, some of the overlapping N1 components to “true N1” can also explain why archery “click” sound evokes higher amplitudes.

On the basis of extensive intracranial recordings, Walter (1964) suggested that the N1-P2 represented the widespread activation of the prefrontal regions of the cortex. Vaughan and Ritter (1970), however, proposed that the auditory vertex response was generated in the primary auditory cortex on the superior aspect of the temporal lobe. The results of Kooi et al. (1971) were confirmed by Picton et al. (1974), who suggested that the N1-P2 response was mainly generated in the association areas of the frontal cortex. These areas could be activated by projections from the medial thalamus or by corticocortical connections (Näätänen and Picton, 1987).

Intracranial recordings have also indicated that several regions of the brain are active at the time of the scalp-recorded N1. The most important are the superior surface of the temporal lobe, some areas of the frontal lobe, the midbrain reticular formation, and the VL nucleus of the thalamus. Activity in anyone of these areas may contribute to the electrical field recorded at the scalp. The amount contributed depends upon the synchronization of the activity, the geometry of the activated area, and the impedance of the brain, skull, and scalp (Näätänen and Picton, 1987).

Having higher amplitudes during archery shooting can also be explained by involving several regions of the brain in responding to the fall of the clicker. As being explained by Näätänen and Picton (1987), different lobes and regions of the brain can be active during at the time of the scalp-recorded N1 and any of these areas may contribute to the electrical field recorded at scalp like in the archery shooting paradigm.

The idea that multiple generators contribute to the scalp-recorded auditory N1 was first clearly stated by Wolpaw and Penry (1975). The waveform recorded from the temporal regions of the scalp using a noncephalic reference contains a negative peak that occurs about 30 ms later than the vertex N1 (Kooi et al., 1971). Wolpaw and Penry recorded auditory EPs from  $C_z$ ,  $T_3$ , and  $T_4$  in response to clicks presented at 1/4.7 s. They proposed that a temporal “T-complex,” with a positive peak at 105 ms ( $T_a$ ) and a negative peak at 155 ms ( $T_b$ ), overlapped a separately generated vertex N1-P2 response. They suggested that the T-complex was generated in the secondary auditory cortex on the lateral aspect of the temporal lobe and that the N1-P2 was generated in widespread areas of cortex, particularly frontal (Näätänen and Picton, 1987).

Click sound during archery shooting most probably evokes “T-complex” because of having a mean latency value of 141,93 ( $T_b$  or  $N1c$ ).  $T_b$  or  $N1c$  component is larger over right hemisphere. Besides, these components are enhanced in amplitude over the hemisphere contra lateral to the stimulated ear (Woods, 1995).

Wolpaw and Penry (1977) reported that the peak latency of wave Ta was significantly longer and the Ta-Tb amplitude significantly smaller in the temporal electrodes ipsilateral to stimulation than in contralateral electrodes. In addition, they found that the response was significantly larger over the right hemisphere than over the left (Näätänen and Picton, 1987).

The N1b wave of McCallum and Curry was maximally recorded from the central electrodes at a mean peak latency of 106 ms and appeared to correspond to the N1 wave of the vertex response. It was slightly larger contralateral to the stimulated ear, and was larger in the more demanding experimental conditions involving cross-over between the side of stimulation and the side of response (Näätänen and Picton, 1987).

The N1b (and N1a) may have been generated by a source in the supratemporal cortex similar to that postulated by Vaughan and Ritter. However, the N1b wave may also have reflected the non-specific component suggested by the recordings of Velasco and his colleagues. If so, we have to assume that the contralateral predominance resulted from an overlapping with the field of a component that was larger contralaterally, such as the N1c which was recorded maximally from temporal electrodes at a peak latency of 129 ms. This wave was considerably larger over the hemisphere contralateral to stimulation (Näätänen and Picton, 1987).

Perrault and Picton (1984) demonstrated that the N1c was much larger for contralateral stimulation and found that it was larger when the stimuli were attended to than when they were ignored (and the subject read a book) (Näätänen and Picton, 1987).

The auditory N1 wave does not reflect a single underlying cerebral process and should not therefore be considered as a unitary event. The scalp distribution of the auditory N1, the magnetic fields recorded at the same latency as the N1, and the effects of cerebral lesions on the N1 suggest three different components contributing

to this scalp-recorded wave. The *first* component is a frontocentral negativity generated by bilateral vertically oriented dipole planes in the auditory cortices on the superior aspect of the temporal lobe. The *second* component is the T-complex with a positive wave at 100 ms and a negative wave at 150 ms, as originally described by Wolpaw and Penry (1975). This complex probably originates in the auditory association cortex in the superior temporal gyrus. The *third* component is one that generates a negative wave at the vertex with a latency of 100 ms. The location of its generator is not known. The justification for this component comes from the intracerebral recordings (Näätänen and Picton, 1987).

We shall find more evidence supporting these three components, and we shall describe more fully these “true” components of the auditory N1 wave. We shall find that they are largely determined by the physical characteristics of the stimulus and by the general state of the subject. We shall also consider other components in the latency region of the N1 which are related more to memory and cognition than to stimulus and state, and which we shall not classify among the “true” N1 components (Näätänen and Picton, 1987).

In 1973 Schafer and Marcus reported that the EP to an auditory or visual stimulus that was triggered by the subject pressing a button was smaller than that evoked by a stimulus presented by a machine. They attributed this effect to temporal uncertainty (Klemmer, 1956), since “the subjects possessed complete foreknowledge of stimulus timing when they stimulated themselves” and “no foreknowledge when the machine delivered the stimuli randomly in time” (Cited in Näätänen and Picton, 1987).

Archer pushes the bow handle with the extended arm and pulls the string by three finger hook on the drawing arm. When he/she reaches the final position, archer should accomplish and/or synchronize some tasks at the same time. As long as the archer pulls the point of arrow beyond the clicker, the onset of the click sound can not be considered like pressing a button and/or delivering the stimuli by a machine. Archer receives foreknowledge of the timing of the stimulus from the vibrations on

the tip of arrow. However, the timing of the onset of the sound is not totally under control of the archer. The mentioned temporal and/or time uncertainty of the timing of the stimulus may cause an increase in the N1-P2 amplitude. The findings of the current study support the earlier studies stating that the N1 amplitude was larger and the N1 latency longer in the time uncertainty condition (Schafer, Amochaev, and Russell, 1981, cited in Näätänen and Picton, 1987).

Wastell, Kleinman, and MacLean (1982; Wastell, 1980) have suggested that diminished temporal uncertainty may explain the reduction in the N1-P2 waves of the EP at short stimulus intervals. The idea is that it is much more difficult to predict the moment when a stimulus occurs if the ISI is long (cited in Näätänen and Picton, 1987). Those findings may explain the differences between ERPs during 6 different trials and the archery shooting. The results of the present study show that increasing the ISI increases the amplitude of N1-P2 component.

Several early experiments (reviewed by Näätänen, 1967, 1975) suggested that the N1 wave of the auditory EP was larger when the subject was attending to the stimuli than when ignoring them. There are two probable effects that may increase the N1 amplitude: any prior uncertainty about stimulus timing and any prior preparation for performing a demanding task (cited in Näätänen and Picton, 1987).

The effect of uncertainty about stimulus timing is explained in the previous paragraphs. As for the effect of prior preparation for performing a demanding task, one should understand the details of archery shooting to explain the effect of the type of task on ERPs. An archer pushes the bow with an extended arm, which is statically held in the direction of the target, while the other arm exerts a dynamic pulling of the bowstring from the beginning of the drawing phase, until the release is dynamically executed (Leroyer et al., 1993). The release phase must be well balanced and highly reproducible to achieve commendable results in a competition (Nishizono et al., 1987). The archer should react to the clicker as quickly as possible. In particular, a repeated contraction and relaxation strategy in the forearm and pull finger muscles

should be developed for this reason (Ertan et al., 2003, 2005, Soylu et al., 2006). That is why archery shooting can be considered as a highly demanding task.

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### **5.3. Discussion regarding the difference between the successful and unsuccessful shots in terms of Auditory Evoked Brain Potentials.**

The 15 archers have made totally 1070 shots during the measurements. Hits, which were made by 15 archers, were processed by placing the all hits on a coordinate system for further analysis. All these hits were matched with the single sweeps of EEG recordings. Finally, hits were grouped as falling into the hit-area and miss-area with their corresponding EEG recordings. Thus, ERPs were achieved corresponding for hit and miss areas separately.

The analysis has shown that there was no significant difference between EEG recordings in terms of amplitudes and latencies of both N1 and P2 components.

Many studies suggest that the N1 amplitude recorded during wakefulness correlates with task performance, being larger on those trials which are associated with a higher level of performance (other factors being constant). Näätänen and Picton (1987) have reviewed studies showing that the N1 is larger during better detection of threshold auditory stimuli. Moreover, several studies have shown that the auditory N1 amplitude is larger when the simple reaction time to the stimuli is shorter (Bostock & Jarvis, 1970; Dustman & Beck, 1965; Näätänen & Gaillard, 1974, cited in Näätänen and Picton, 1987).

Increasing motivation by making the amount of monetary reward dependent on performance (Wilkinson & Morlock, 1966) has resulted in enhanced N1 amplitudes and better performance, but again, it is not possible to conclude with certainty that increased arousal enhanced the N1 amplitude.

Considering all of the evidence, there is some evidence for task or attention-induced stimulus-nonspecific increase in the excitability of some neuronal population contributing to the N1 deflection. This increase causes the N1 amplitude to any input, relevant or irrelevant, to be larger when the subject is engaged in some task rather than relaxing, and larger when performing a more rather than less involving task. However, all these findings are not exemplifying the performance like in archery shooting because, the performance means the speed of the response (Reaction Time paradigm) in earlier studies not the success of the performance. Ertan et al. (1996) has made a research on archers to measure the effect of reaction time on the scores on the target. They have concluded that there was no correlation between the hits on the target and the reaction times of the subject.

It can be concluded that the fall of the clicker in archery shooting increases the amplitude of N1 component. However, there is no difference between successful and unsuccessful shots in terms of N1-P2 amplitude and latency as a response to the sound from the clicker. Thus, both successful and unsuccessful shots have the same effect on the brain evoked potentials in archery shooting.

#### **5.4. Discussion regarding the effect archery shooting on Auditory Evoked Brain Potentials.**

Proponents of exercise report that brief bouts of exercise help them think more clearly and improve their mood and psychological well-being. There is considerable support for the view that acute bouts of exercise has a positive impact on mood states and affect (Morgan & O'Connor, 1988; Raglin, 1997, cited in Tomporowski, 2003). A panel of experts conducting a review of research for the National Institute of

Mental Health concluded that exercise is positively related to several indices of mental health (Morgan, 1984, cited in Tomporowski, 2003). Exercise is associated with a reduction in physiological measures of stress and psychological measures such as anxiety and depression. Furthermore, exercise is associated with elevations in mood states and heightened psychological well-being (Berger, 1996; Shephard, 1996, cited in Tomporowski, 2003).

Attention plays a crucial role in the control of behaviour. In particular, sporting activities are influenced by attentional states, and various attentional styles are developed by athletes engaged in different sports. The covert orienting of attention is achieved by central mechanisms (Posner 1980, cited in Fontani, et al., 1999), and rapid shifting from broad to selective attention is an important ability for the successful performance of open-skill activities (Fontani 1994, cited in Fontani, et al., 1999). Testing and analysis of the central processes involved in the generation of an attentional style can be useful for an improvement in performance. The repeated occurrence of stimuli with a high probability of association elicits distinct brain responses (Fontani, et al., 1999).

Although archery does not appear to be very fitness demanding sport, when closely examined, both training and competition take long time. During a national or an international competition, archer is forced to make over 72 shots in a single day, where a female archer is to apply approximately 15-16 kg and male 18-20 kg of force each time the bow is pulled. This sums up to at least 1125-1200 kg for females and 1350-1500 kg of force applied in a single day competition in an intermittent manner under very stressful situation. It, therefore, is very demanding event on certain musculature and abilities to perform well under every possible environmental conditions (Açıkada et al., 2004).

It was concluded that if an archery session has any effect on event-related brain potentials or not. The findings of the current study have shown that archery session composed of 72 shots like in international organization does not create any

difference on brain potentials in training. However, it should be noted that archery shooting session has been conducted in training without having any opponent and stress condition.

### **5.5. Implications and Recommendations**

The present study has some implications for AEBPs in Recurve archery shooting. These implications with some recommendations are presented below.

The results of the current study have shown that the sound from the fall of the clicker during archery shooting evokes a negative (N100) and a positive (P200) going waves. The amplitude of N1 component is higher than that of all other paradigms created in laboratory condition. However, there is no significant difference between successful and unsuccessful shots in terms of the amplitude and latency of N1-P2 components. Besides, archery shooting session does not have any effect on the amplitudes and latencies of N1-P2 components.

Based on the results of the present study, the recommendations might also be given for further studies:

1. The current study involves high level and some middle class archers (N=15). But it does not concentrate on the differences between world and middle class archers in order to first define the archery shooting from more general point of view. Creating two or three subject groups with different performance levels may help further explanation of archery shooting paradigm. Besides, successful and unsuccessful shots may display different patterns in different groups of archers. That may also help to clarify the effect of “click” sound on event-related brain potentials.
2. What has been clarified with the current study is “click” sound evokes N1-P2 component and this component has higher amplitudes than that of any control

conditions. The reasons of that finding have been speculated in relation with the current literature on especially N1-P2 component. A more detailed study can be conducted to further discuss why fall of the clicker evokes higher amplitudes of N1-P2 component. That may correlated with source location studies of mentioned component.

3. As long as archery shooting is not highly physiological demanding event, an archery session may have some effect on brain potentials. This effect may not only be related with the physiological aspects of archery shooting sessions. There may be some psychological effects of archery shooting on brain potentials. In the current research, physiological aspect of archery shooting has been imitated by having 72 arrows shot in a session like in an international competition, but psychological aspects of archery shooting could not be imitated with the current research design. It is recommended that the effect of archery shooting session on brain potentials should be analysed during the real archery competitions.

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## TÜRKÇE ÖZET

### OKÇULUKTA İŞİTSEL UYARILMIŞ BEYİN POTANSİYELLERİNİN İNCELENMESİ

#### 1. GİRİŞ

Okçuluk, üst vücut bölgesinde ve özellikle omuz çevresinde kuvvet ve dayanıklılığa ihtiyaç duyulan statik bir spordur (Mann, 1984; Mann & Littke, 1989). Bir okçuluk yarışmasında yüksek puan atabilmek için dengeli ve tekrar edilebilir hareketler ortaya konulmalıdır (Nishizono, 1987). Üst ve orta düzey sporcular aynı özelliklere sahip okçuluk malzemelerini kullanırlar. Bu iki grup arasındaki farklı aynı özelliklerle tekrar edilebilen hareketler belirler (Stuart, 1990). Bir okçu yayı itiş kolu ile iterken, çekiş kolu ile de kirişi çekmektedir. Bu hareket çekiş fazının başlarından başlayıp, tam çekiş aşamasında sonlanır (Leroyer et al., 1993). Tam çekiş aşamasına ulaşan okçu uygun koşullar sağlandığında bırakış hareketini gerçekleştirir. Bırakışın yarışmalarda yüksek puan atabilmek için dengeli ve tekrar edilebilir olması gereklidir (Nishizono, 1987).

Kirişin bırakılma kararı “klikır” adı verilen bir cihazdan işitsel bir uyarın geldiđi anda verilmiş olur. Bu sayede atılan her ok aynı çekiş uzunluđuna kadar çekilerek atılmış olur. Başka bir deyişle yayın itiş gücü bu sayede sabitlenmiş olur. Klikır’ın atılan puanı arttırdığına inanılır ve dünyada olimpik yayla atan tüm sporcular tarafından kullanılır (Leroyer et al., 1993). Okçu klikırdan gelen sese optimum hızla tepki verir ve tüm vücut bölgelerinde kassal aktivasyonu senkronize eder. Özellikle, okçuların, yapılan fazla sayıda ok atışı sırasında kassal kasılma ve gevşeme stratejileri geliştirmeleri gereklidir. “Çıt” sesini duyduğunda okçu, çekiş kolu ön kolunda fleksör kasları gevşetip, ekstensör kasları kasmaktadır. Böylece kirişin üç

parmak kancası olarak bilinen parmaklardan kurtulması mümkün olabilmektedir (Ertan et al., 2003; Hennesy et al., 1990; Clarys et al., 1990; Nishizono et al., 1987).

Klikır'ın düşmesi işitsel bir uyarana neden olması sebebiyle bir takım beyin potansiyeli uyarması mümkündür. İşitsel Uyarılmış Beyin Potansiyelleri (İUBP) belli latanslarla ve işitsel uyarana bir takım ilişkiler sergileyerek oluşurlar. İUBP'nin en yaygın sınıflaması onların latanslarına göre yapılan sınıflamadır. Latanslarına göre ise İUBP ilk, hızlı, orta, yavaş ve geç latanslı olacak şekilde sınıflanırlar. Ayrıca İUBP'i "egzojen" ve "endojen" olarakta sınıflandırılırlar. Egzojen potansiyeller uyarının fiziksel özelliklerine göre farklılık gösterirken, endojen potansiyeller uyarının deneğe olan fizyolojik anlamlılığına göre değişiklik ortaya koyarlar (Picton, 1982).

Bu nedenle mevcut araştırma da okçulukta uzun latanslı İşitsel Uyarılmış Beyin Potansiyellerinin incelenmesi amaçlanmıştır. Araştırma soruları şu şekilde sıralanmıştır:

1. Klikır uyarını ile ne tür beyin potansiyelleri uyarılmaktadır?
2. Laboratuvar ortamı ve ok atışı sırasında ölçülen beyin potansiyelleri arasında fark var mıdır?
3. Başarılı ve başarısız atışlar karşılaştırıldığında İşitsel Uyarılmış Beyin Potansiyellerinde bir farklılık oluşmaktadır mı?
4. Bir antrenman birimi İşitsel Uyarılmış Beyin Potansiyelleri üzerinde bir etkiye sahip midir?

## **2. YÖNTEM**

Araştırmaya başlamadan önce her deneğe bilgilendirilmiş onam formu okutulmuş ve imzalatılmış. Bu formda araştırmanın muhtemel risklerinden bahsedilmiştir. Ayrıca araştırma protokolü Başkent Üniversitesi, Tıp Fakültesi Etik Komitesi tarafından onaylanmıştır (Sertifika No: 2004/85). Bununla birlikte bu araştırma 1975 Helsinki bildirgesinin etik gereklerini yerine getirmiştir. Bu araştırmaya kontrol denemeleri

için okçu olmayan 10 kişi (N=6 erkek; N=4 bayan) ve ok atışı denemeleri için aktif okçuluk yapan 15 kişi (N=9 erkek; N=6 bayan) katılmıştır. Denekler, normal işitme özelliklerine sahip olan, nörolojik sebeplerle ve beyin aktiviteleri üzerinde etkisi olan herhangi bir ilaç kullanmayan kişilerden oluşmuştur. Altı farklı control paradigması oluşturulmuştur. Ok atışları ise resmi yarışma mesafesi olan 18 m'den hedef kağıtlı olarak yapılmıştır. İUBP 200 ms uyarın öncesi ve 800 ms uyarın sonrası olmak üzere toplam 1000 ms olarak atış sırasında kafatası üzerinden kaydedilmiştir. 1 ve 5. paradigmalarda atış antrenmanının etkisini araştırmak amacıyla atış öncesi ve sonrası kaydedilmiştir.

## **2.1. Ölçüm Yöntemi**

### **2.1.1. Kontrol Denemeleri**

Kontrol denemeleri laboratuvar ortamında ok atmaksızın yapılmıştır. 6 farklı kontrol denemesi seçilmiştir: (1) *Kontrol denemesi 1*: Denek kendisi bilgisayarın enter düğmesine basar ve ses uyarını basıldıktan sonra 1-3 saniye gecikme ile gelir. Denek sesi duyduğunda ise işaret parmağını kaldırarak bir motorik tepki ortaya koyar. (2) *Kontrol denemesi 2*: Uyarın bilgisayar tarafından 8-12 saniye aralıklarla otomatik olarak verilmektedir. Denek sesi duyduğunda işaret parmağını kaldırarak bir motorik tepki ortaya koyar. (3) *Kontrol denemesi 3*: Denek kendisi bilgisayarın enter düğmesine basar ve ses uyarını basıldıktan sonra 1-3 saniye gecikme ile gelir. Denek sesi duyduğunda bir tepki ortaya koymaz. (4) *Kontrol denemesi 4*: Uyarın bilgisayar tarafından 8-12 saniye aralıklarla otomatik olarak verilmektedir. Denek sesi duyduğunda bir tepki ortaya koymaz. (5) *Kontrol denemesi 5*: Uyarın bilgisayar tarafından 1 saniye aralıklarla otomatik olarak verilmektedir. Denek sesi duyduğunda bir tepki ortaya koymaz. (6) *Kontrol denemesi 6*: Uyarın denek enter düğmesine basar basmaz oluşmaktadır. Bu denemede de denekten bir tepki ortaya koyması beklenmez.

### 2.1.2. Ok Atışı Denemeleri

Ok atışı denemeleri resmi yarışma mesafesi olan 18 m'de yapılmıştır. Sporcular ok atarken kafa tası üzerine yerleştirilmiş olan elektro cap aracılığıyla işitsel uyarılmış beyin potansiyelleri (İUBP) kaydedilmiştir. Yapılan ölçümler bilgisayar ortamına aktarılmış ve ölçümler sonrası klikir'in düşüşünden 200 ms öncesi ve 800 ms sonrası olacak şekilde herbir atış için hesaplanmıştır. Ok atışı sırasında yapılan kayıtlara ek olarak, ok atışı öncesi ve sonrası olmak üzere 1. ve 5. kontrol denemeleri uygulanmıştır.

Klikir'in düşüşü ile EEG kayıtlarının senkron hale getirilebilmesi için klikir'in altına mekanik bir sistem yerleştirilmiştir. Klikir okun ucundan düştüğü zaman klikir'in ucu ile yay gövdesi bir araya gelmekte ve EEG ölçüm penceresine bir sinyal göndermektedir. Böylece klikir'in düştüğü an tam olarak tespit edilebilmektedir (Ertan et. al., 2003; Ertan et. al., 2005).

Her okçu 18 m mesafeden önce 12 okluk ısınma serisini tamamlamışlar ve ardından 72 oktan oluşan deney oklarını atmışlardır. Hedefte hangi okun hangi sıra ile atıldığı ve nereye isabet ettiği okların üzerine yapıştırılan farklı renklerdeki bantlar aracılığı ile tesbit edilmiştir. Buna ek olarak ise atılan okların hedefte resimleri çekilmiş ve bu resimler bilgisayar ortamına aktarılmıştır. İsabetli atışlar  $(x_1, y_1)$ ,  $(x_1, y_2)$ ,  $(x_2, y_1)$ ,  $(x_2, y_2)$  dikdörtgeni içinde kalan atışlardan oluşurken bu dikdörtgenin dışında kalan atışlar isabetsiz atışlar olarak sınıflandırılmıştır.

$$x_1 = m_x - k_x$$

$$x_2 = m_x + k_x$$

$$y_1 = m_y - k_y$$

$$y_2 = m_y + k_y, \text{ burada}$$

$m_x$ : x değerlerinin ortalamasını

$m_y$ : y değerlerinin ortalamasını

k: bir pozitif sayıyı ifade etmektedir.

### **2.1.3. EEG Kayıtları**

EEG kayıtları Ag/AgCl özelliğine sahip elastik bere aracılığı ile yapılmıştır. Medium (54-58 cm) ve large (58-62 cm) olmak üzere iki elektro bere kullanılmıştır. Her EEG kanalına elektro jel enjekte edilmiştir. Kanalların empedansları  $5K\Omega$ 'un altına düşünceye kadar jelleme işlemine devam edilmiş ve belirli aralıklarla kontrol edilmiştir. Uluslararası 10-20 sistemine (Jasper, 1958) uygun olacak şekilde 21 kanaldan kayıt alınmıştır (Fp1, Fp2, F3, F4, F7, F8, Fz, C3, C4, P3, P4, Pz, T3, T4, T5, T6, O1, O2, Right Mastoid, Left Mastoid).

### **2.1.4. Verilerin Analizi**

EEG kayıtları sürekli olacak şekilde yapılmış ve digitize edilmiştir (500 Hz). Sürekli EEG verileri klikir'in düşüşünden 200 ms öncesi ve 800 ms sonrası olacak şekilde epoklanmıştır. Elde edilen potansiyeller önce her denek için ayrı ayrı sonrada grup için ortalanmıştır. Daha sonra ise bant geçiren filtre aracılığıyla filrelenmiştir (1–12 Hz, Butterworth 12 dB/oct slopes).

## **3. BULGULAR**

### **3.1. Ön Ölçüm Sonuçları**

Ön ölçümlere iki üst düzey okçu katılmıştır. Bu ölçümler sırasında literatürde bulgu yer almayan ok atışı sırasında ortaya çıkan İUBP'nin tanımlanması amaçlanmıştır. Ön ölçümler, klikir'in düşüşünün 100 ve 200 ms gecikmelerle ortaya çıkan uzun latanslı beyin potansiyellerini uyardığını göstermiştir. Böylece gerçek ölçümlerin nereye odaklanacağı tesbit edilebilmiştir.

### **3.2. Klikir'in düşüşü ile uyarılan beyin potansiyellerinin incelenmesi**

Ok atışı sırasında klikir'in düşüşü  $16,59 \mu V$  genlikli ve 129 msec latanslı bir negatif yönlü (N1) ve  $-6,98 \mu V$  genlikli ve 180 msec latanslı bir pozitif yönlü (P2) dalga

formu uyarmaktadır. Bu tepkiler N1-P2 bileşeni olarak tanımlanmaktadır. Bu bileşenlerin genlikleri; N100= 27,73 ± 16,82, P200= -21,89 ± 20,46 ve latansları ise; N100= 141,93 ± 41,46; P200= 211,8 ± 43,97 şeklinde ortaya çıkmıştır.

### 3.3. Ok atışı sırasında kaydedilen potansiyeller ile laboratuvar ortamında kaydedilen potansiyellerin karşılaştırılması

Ok atışı sırasında ölçülen genlikler ile 3. deneme hariç tüm denemeler arasında N1 genliği bakımından anlamlı farklılık gözlenmiştir ( $p<0.05$ ). N1 latansı ile de sadece 2 ve 5. denemeler arasında farklılık gözlenmiştir ( $p<0.05$ ). Ok atışı sırasında ki P2 genliği ile sadece 6. deneme arasında anlamlı farklılık gözlenirken ( $p<0.05$ ), P2 latansı konusunda ok atışı ile hiçbir deneme arasında anlamlı fark gözlenmemiştir ( $p>0.05$ ) (Tablo 7).

**Tablo 7.** Ok atışı ve 6 farklı laboratuvar ölçümünde elde edilen N1 ve P2 bileşenlerinin latans ve genlik değerlerinin karşılaştırılması ( $\mu V$ ). Ortalama değerler verilmiştir ( $\pm$  standart sapmalar).

	N100 Genliği	N100 Latansı	P200 Genliği	P200 Latansı
Deneme 1	12,14±5,68*	123,4±25,44	-10,18±7,27	187,2±19,53
Deneme 2	11,01±5,84*	112±17,33*	-10,69±3,89	185,7±38,11
Deneme 3	14,53±7,67	118,7±20,33	-9,56±7,08	189,1±18,34
Deneme 4	14,09±7,01*	132,2±29,33	-10,36±7,43	212,3±40,20
Deneme 5	8,80±5,29*	107,5±27,86*	-9,68±7,89	180,9±30,06
Deneme 6	11,19±4,87*	122±32,95	-5,645±6,41*	194,9±30,45
Ok Atışı	27,73±16,82*	141,93±41,46*	-21,89±20,46*	211,8±43,97

\* Ok atışı sırasında ölçülen İUBP'leri ile control denemeleri arasında anlamlı farkı ifade etmektedir ( $p<0.05$ ).

### 3.4. Ok atışı sırasında başarılı ve başarısız atışlara karşılık gelen işitsel uyarılmış beyin potansiyellerinin karşılaştırılması

Başarılı ve başarısız atışlara karşılık gelen beyin potansiyellerinin ortalama genlik ( $\mu\text{V}$ ) ve latans değerleri (msec) Tablo 8’de gösterilmiştir.

**Tablo 8.** Başarılı ve başarısız atışlara karşılık gelen N1 ve P2 komponentlerinin genlik ( $\mu\text{V}$ ) ve latans (msec) değerleri. Değerler ortalamalar olarak verilmiştir ( $\pm$  standart sapma).

N100 Genliği		N100 Latansı		P200 Genliği		P200 Latansı	
Başarılı	Başarısız	Başarılı	Başarısız	Başarılı	Başarısız	Başarılı	Başarısız
27,85	26,60	116,98	116,90	-18,68	-16,63	198,53	201,53
$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$
15,41	20,04	15,60	16,84	20,68	20,27	30,35	29,57

N1-P2 bileşenlerinin genlik ve latansları konusunda isabetli ve isabetsiz atışlar arasında anlamlı farka rastlanmamıştır ( $p>0.05$ ).

### 3.5. Bir seri ok atışının işitsel uyarılmış beyin potansiyelleri üzerine etkisinin incelenmesi

Alınan ön ve son ölçümlerin gerek genlik ve gerekse latansları arasında anlamlı bir farka rastlanmamıştır. Buradan atış antrenmanının beyin potansiyelleri üzerinde bir etki yaratmadığı sonucuna varılmıştır ( $p>0.05$ ).

## 4. SONUÇ VE ÖNERİLER

Gerek ön ölçümler ve gerekse gerçek denemeler ok atışı yada klikir sesinin negatif (N1) ve pozitif (P2) yönlü potansiyeller uyardığını ortaya koymuştur. N1-P2 komponenti olarak adlandırılmakta olan bu potansiyeller dikkat ve sürpriz durumlarına bağlı olarak genlikler artan veya azalan potansiyeller olarak bilinmektedirler. Denek

verilen uyarana bir tepki ile karşılık verildiği durumda bu potansiyellerin genlikleri verilmediği duruma göre daha yüksek ölçülmektedir. Benzer şekilde uyarının gelme zamanı konusunda bir sürpriz durumu söz konusu ise gene genlikte büyüme gözlenmektedir.

Ok atışı sırasında daha yüksek genlikli potansiyellerin ölçülmesi atış sırasında bir çok farklı beyin bölgesinin aktive edilmesi ile açıklanabilir. Farklı beyin lobları ve bölgelerinden birkaç tanesi aynı anda aktif hale getirilmiş olabilir ve bu nedenle de ok atışı sırasında N1 genliğinde laboratuvar ortamındakinden daha yüksek bir değere ulaşılmış olabilir.

Mevcut araştırmada ortaya konulan sonuçlara dayalı olarak aşağıda ki tavsiyelerde bulunulabilir;

1. Bu araştırmada orta ve üst düzey okçular üzerinde yapılan ölçümlerle ok atışının işitsel uyarılmış beyin potansiyelleri üzerine yarattığı incelenmeye ve tanımlanmaya çalışılmıştır. Daha sonra yapılacak araştırmalarda farklı düzeyde ki okçulardan oluşan üç farklı grup üzerinde böyle bir araştırma yapılarak performans düzeyleri farklı deneklerde farklılık gözlenip gözlenmediğinin incelenmesi uygun bir yöntem olabilir.
2. Yapılan bu araştırmayla “Çıt” sesi ile N1-P2 komponentinin uyarıldığı ve bu komponentin tüm laboratuvar denemelerinden daha büyük bir genliğe sahip olduğu ortaya konulmuştur. Bu doğrultuda genlikteki bu büyüme sadece speküle edilerek yorumlanmaya çalışılmıştır. Bu konuların daha derinlemesine yapılacak bir araştırma ile incelenmesi ve açıklanmaya çalışılması uygun bir yöntem olabilir.
3. Ok atışı her ne kadar fizyolojik olarak sporcu üzerine yük bindirmesede yapılan toplam işe bakıldığında küçümsenmeyecek oranda ağırlık taşındığı gözlenebilir. Bu durum mevcut araştırma da oluşturulmaya çalışılmıştır. Ancak okçuluğun psikolojik yönü test edilememiştir. Herhangi bir stres yada kaygı ortamı yaratılmamıştır. Böylece ok atışın işitsel uyarılmış beyin potansiyelleri üzerine etkisi tek boyutlu incelenmiştir. Bu nedenle psikolojik

baskı altında yapılan atışlar öncesi ve sonrası ölçümlerinde alınması uygun olabilecektir.

## VITA

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