1988

Effects of contralateral masking on the summatting and action potentials in humans during electrocochleography

Peter K. Wightman

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THE EFFECTS OF CONTRALATERAL MASKING ON THE SUMMATING AND ACTION POTENTIALS IN HUMANS DURING ELECTROCOCHLEOGRAPHY

by

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Presented in partial fulfillment of the requirements for the degree of
Master of Arts
University of Montana
1988

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The purpose of the present study was to examine the effects of contralateral stimulation on the fast evoked potentials measured by electrocochleography. This study attempted to replicate a previous investigation which indicated that contralateral stimulation suppressed the amplitude of the action potential. In addition, several different techniques were employed to determine the effects of contralateral stimulation on the summating potential, the action potential, and the SP/AP ratio. Responses were recorded from 10 young-adult subjects with normal hearing sensitivity. One-third octave filtered clicks centered around 4000 Hz were presented at 45 dB SL to the ipsilateral ear. In the control condition, the ipsilateral stimulus was presented without contralateral masking. In the experimental condition, a 4000 Hz pure-tone was presented at 45 dB SL as the contralateral masker. The waveforms were recorded separately in response to rarefaction and condensation clicks, and then summed in order to produce composite waveforms for each condition. These composite waveforms were replicated, providing two trials in each condition for each subject. The amplitude and latency of the action potential, the amplitude of the summating potential, and the SP/AP ratio were calculated for each trial under each condition.

The results indicated that the amplitudes of the action potential and summating potential responses, the latencies of the action potential, as well as the SP/AP ratios, did not change in any systematic manner during the application of contralateral masking. The responses obtained in the present study were reliable and free from any systematic variation. In addition, the values for the latencies of the action potential were consistent with normative data obtained from earlier studies. This evidence failed to support the findings of Folsom and Owsley (1987) which had indicated an inhibitory effect of contralateral masking on the action potential. Further research is suggested to investigate the effects of contralateral masking on the fast evoked potentials in order to resolve the discrepancies between the two studies.
ACKNOWLEDGEMENTS

I wish to thank my thesis committee members, Dr. Richard L. Gajdosik, Ms. Susan A. W. Toth and Dr. Michael K. Wynne, for their support and guidance throughout this project. I thank Rich for his invaluable assistance in the areas of experimental design and statistical analysis. I am grateful to Sue for her insightful suggestions regarding experimental methodology and the interpretation of the results of this study. I wish especially to thank Mike for graciously accepting the role of mentor, for inspiring me to enter into this work with joy and enthusiasm, for affirming my scientific potential, and for challenging me to transcend the limits of my knowledge. I also thank him for his insights into clinical audiology, hearing science and computer science, and for his friendship.

I wish also to express my appreciation to Dr. David Hayes and Dr. Thomas Hoshaw for their generous assistance in this investigation. I am especially grateful of their contribution in preparing the subjects for electrocochleography in spite of their busy patient schedule.

This study would not have been possible without the courageous support of my fellow students who acted as subjects. I would like to thank them for donating their time and for enduring the rigors of electrocochleography.

I would like to take this opportunity to thank my mother...
Lois, for her encouragement and support throughout this program. Without her help, it would not have been possible for me to pursue this graduate degree and to begin a new and exciting career in audiology. I would also like to thank my friends, both in Missoula and in Canada, for the support they have extended to my wife and myself during the completion of this thesis and throughout my graduate program.

Most especially, I would like to thank my wife, Anne, for her unconditional love, trust and support. I am grateful to her for assuming my responsibilities while I returned to college, for allowing me the freedom to explore my personal and intellectual potential, and for her unfailing confidence in me.

This thesis is dedicated to the loving memory of my father, Kager, whose life and work in the field of medicine are for me a perpetual inspiration.
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CHAPTER I: INTRODUCTION

Stimulation of the olivocochlear bundle of the efferent auditory nervous system has been demonstrated to have inhibitory effects on the response of the afferent auditory nerve fibers to ipsilateral acoustical stimulation. This phenomenon has been observed in direct measurements of single auditory nerve fibers and in recordings of far field evoked potentials (see Appendix A for definitions of terms). The inhibitory effects have been shown to occur in response to direct electrical stimulation of the olivocochlear bundle in cats (Galambos, 1956; Gifford & Guinan, 1983) and in response to acoustical stimulation of the contralateral ear in cats (Buno, 1978) and in human subjects (Folsom & Owsley, 1987).

Folsom and Owsley (1987) observed the inhibitory effects of contralateral acoustical masking in humans. They demonstrated that acoustical stimulation of the efferent system (by a contralateral masker) resulted in a reduction in the amplitude of the whole auditory nerve action potential (AP or N1) with no accompanying change in latency. These effects were demonstrated in response to narrow band filtered click ipsilateral stimuli with stimulation of the efferent system by a pure tone contralateral masker. In order to provide the most effective masking, the masker frequency was selected to coincide with the center frequency of the ipsilateral stimulus (Buno, 1978). The effects were observed at two differing ipsilateral stimulus intensities. The intensity of the
contralateral masking tone was maintained at levels that were below interaural attenuation and were also well below the acoustic reflex threshold. Folsom and Owsley concluded that a neural mechanism was responsible for these effects because they were able to rule out the possibility of crossover of the contralateral masker as well as possible contributions of the middle ear reflex.

The techniques employed by Folsom and Owsley (1987) did not allow for the recording of the summating potential (SP) in the response because they employed filter settings appropriate for recording the auditory brainstem response (ABR). Electrocochleography (ECochG) filter settings are more appropriate for far field recordings of the summating potential. Furthermore, Folsom and Owsley presented single-polarity filtered clicks as the ipsilateral stimulus, and thus could not eliminate the cochlear microphonic (CM) from the response to allow for measurement of the SP. The cochlear microphonic may be eliminated by employing either of the following techniques:

1. Providing click stimuli of alternating polarity, or
2. Summating responses recorded during presentation of click stimuli of opposite polarity.

Accordingly, the possible effects of contralateral masking on the SP and on the SP/AP ratio were not explored by these authors. It was therefore not possible to demonstrate whether
the effects of efferent system stimulation in human subjects extended to the level of the receptor hair cells or whether the effects were first manifested at the level of the spiral ganglion of the auditory nerve.

Electrical stimulation of the crossed olivocochlear bundle has been shown to cause a decrease in the resting endolymphatic potential and a slight increase in the amplitude of the cochlear microphonic receptor potentials in animal subjects (Klinke and Galley, 1974; Desmedt, 1975). These observations imply that the effects of efferent stimulation do extend to the hair cells and beyond to alter the concentration of ions within the scala media. To account for the changes in the resting potential and the CM, Desmedt (1975) proposed a mechanism whereby the efferent system increases the anion conductance of the outer hair cell membrane.

Folsom and Owsley speculated that a possible cause of the action potential inhibition phenomenon might be an alteration of the basilar membrane mechanics presumably mediated by the outer hair cells (OHC). This hypothesis reflects the current thinking regarding cochlear micro-mechanics (Durrant and Lovrinic, 1984; Dallos, 1988). This hypothesis would imply that the effects of the contralateral masker extended to the OHC and could possibly be demonstrated in evoked responses that occur earlier than the AP. This theory is consistent with the anatomical connections of the crossed and uncrossed
olivocochlear bundles (COCB and UOCB) to the outer and inner hair cells (OHC and IHC). The majority of crossed OCB fibers innervate outer hair cells and synapse both directly onto the OHC and onto the afferent nerve fibers. In contrast, most of the uncrossed OCB fibers innervate inner hair cells, but synapse onto the afferent fibers rather than onto the IHC directly (Pickles, 1982; Spoendlin, 1972; Warr, 1975). This hypothesis is also supported by studies of electrical stimulation to the olivocochlear bundles in cats, wherein the crossed OCB fibers, which more directly innervate the OHC, have been shown to sustain more effective action potential inhibition than the uncrossed OCB fibers (Sohmer, 1966).

The purpose of this study is to further explore the physiological effects of acoustical stimulation of the efferent auditory pathways in normal human subjects. Extending the work of Folsom and Owsley (1987), the evoked responses of the earlier-occurring summating potential will be recorded under similar experimental conditions. This will be undertaken by recording the electrocochleography (ECochG) response from electrodes placed in the external auditory meatus in the presence and absence of contralateral masking. The ECochG response will be recorded for ipsilateral filtered click stimuli of both positive and negative polarity. This will allow for reliable quantification of both the SP, a hair cell receptor potential which reflects the mechanical events in the cochlea, and the AP, a whole-nerve action potential
which originates from the spiral ganglion in the habenula perforata. This study will attempt to determine whether the effects of stimulating the efferent auditory system extend to the level of the receptor hair cells within the human cochlea. More specifically, the following questions will be addressed:

1) Is the SP/AP amplitude ratio calculated from the response to a frequency-specific filtered click stimulus altered by the introduction of a contralateral pure tone masking signal at its center frequency?

2) Are the absolute amplitudes of the SP and/or AP responses to a frequency-specific filtered click stimulus altered by the introduction of a contralateral pure tone masking signal at its center frequency?

It is hypothesized that if the stimulation of the efferent auditory system by a pure-tone presented to the contralateral ear produces changes in the afferent auditory system, then these changes will be demonstrated in the amplitude of the summating potential, the amplitude of the action potential and the SP/AP amplitude ratio in response to filtered click stimuli as measured by ECochG techniques.
CHAPTER II: REVIEW OF THE LITERATURE

A review of the anatomy of the auditory nervous system is provided, with particular emphasis on the olivocochlear bundle (OCB) of the efferent system to orient the reader to the questions explored in this study. This review is followed by a discussion of the physiology of the auditory system, and then by a third section in which the auditory potentials are discussed, including the cochlear resting potentials, the receptor potentials (the cochlear microphonic and summating potential), the generator potential and the action potential (AP or N1). The measurement of these potentials is also examined in sections focusing on single-unit recordings and evoked potentials. The clinical measurement of the cochlear potentials through the application of electrocochleography will be discussed in detail. Finally, the techniques commonly employed to stimulate the olivocochlear bundle are explained, and relevant research findings are cited.

Anatomy of the Auditory System

Cochlea

The cochlea is found coiled within the petrous portion of the temporal bone. The bony cochlea is filled with perilymph and communicates with the middle ear system by way of the footplate of the stapes. The membranous cochlea, filled with endolymph, lies within the bony cochlea and is enclosed by enclosed by the basilar membrane, stria vascularis, osseous spiral lamina and Reissner's membrane.
This membranous cochlear partition separates the scala vestibuli from the scala tympani throughout the length of the cochlea, with the exception of an opening, the helicotrema, located at the apex. The sensory end organ of the auditory system, the organ of Corti, lies within the cochlear partition and is supported by the basilar membrane.

Within the organ of Corti lie the receptor hair cells arranged in rows from base to apex, with one inner and three outer hair cell rows. Hair-like stereocilia project upward from the hair cells. The stereocilia of the outer hair cells are imbedded in the under side of the overlying tectorial membrane, whereas those of the inner hair cells are not embedded in this gelatinous membrane. Both types of hair cells stimulate the afferent auditory nerve fibers of the auditory branch of the vestibulocochlear (VIII) cranial nerve. However, the inner and outer hair cells are differentially connected to the VIII cranial nerve and exhibit anatomical differences.

The great majority of the afferent nerve fibers arise from inner hair cells (95%), forming the inner radial bundle. Each inner hair cell may synapse with up to twenty nerve fibers (Durrant and Lovrinic, 1984). The remaining 5% of the afferent nerve fibers arise from outer hair cells to form the outer spiral fibers. These outer hair cell fibers are extensively branched, with many receptor cells stimulating one nerve fiber.
The Afferent Auditory System

The afferent, centripetal or ascending auditory system conveys information encoded as neural impulses from the periphery (cochlea) to the auditory cortex through several intermediate nuclei in the brain stem. The afferent fibers project from the inner and outer hair cells through the habenulae perforata of the osseous spiral lamina, through the modiolus and internal auditory meatus to the ipsilateral cochlear nuclei in the upper medulla. The cell bodies of these nerve fibers lie in the spiral ganglion within the modiolus of the cochlea. These first order nerve fibers bifurcate and project to the anteroventral cochlear nucleus and also to either the posteroventral or dorsal cochlear nucleus.

Second order neurons with cell bodies in the cochlear nuclei project primarily to the contralateral superior olivary complex in the lower pons, with 90% of these fibers decussating through the trapeziod body. Some of the ascending fibers project ipsilaterally, and some of the crossed and uncrossed second order fibers project directly to nuclei located above the superior olive. Third order neurons ascend from the superior olive through the lateral lemniscus to the inferior colliculus in the mid-brain. Fourth order neurons project upward to the medial geniculate body of the thalamus, and fifth order neurons ascend from the thalamus to the transverse gyrus of Heschl in the temporal lobe of the
cerebral cortex (the auditory cortex). This is the primary pathway from the cochlear nucleus to the cortex, but various other routes and interconnections also exist and contribute to the processing of auditory information in a complex manner.

The Efferent Auditory System

The efferent, centrifugal or descending auditory pathways extend from the cortex through intermediate nuclei to the cochlea. The efferent nerve fibers and cell bodies lie close to, but not within, the afferent (ascending) fiber tracts and nuclei (Pickles, 1982; Desmedt, 1975). According to Pickles (1982), there are two efferent systems descending from the auditory cortex: one to the medial geniculate body and another system to the inferior colliculus and several other nuclei. The efferent fibers that lead to the medial geniculate body terminate on the cell bodies of afferent neurons which project back to the auditory cortex. The inferior colliculus also receives efferent innervation from the medial geniculate body. Fibers descend from the inferior colliculus to the lateral lemniscus, the superior olivary complex and the cochlear nuclei. Efferent fibers from the lateral lemniscus descend to the cochlear nuclei, however the largest efferent innervation of the cochlear nuclei arises from the superior olivary complex. The final link in the efferent system is the olivocochlear bundle (OCB) which descends from the level of the superior olivary complex (SOC) to the hair cells.

The olivocochlear bundle (or Rasmussen's bundle) first
identified by Rasmussen (1946), projects from each superior olivary complex in two main descending tracts, the uncrossed OCB (UOCB) and the crossed OCB (COCB). The COCB fibers arise mainly from the medial accessory superior olive, course dorsally to decussate between the facial nerve genua near the floor of the fourth ventricle and cross the facial nerve root before joining the (afferent) vestibular nerve (Desmedt, 1975). These crossed fibers constitute approximately 3/4 of the OCB neurons. The UOCB fibers arise from the lateral superior olivary nucleus and project dorsally to join the COCB fibers within the vestibular nerve near the ventral aspect of the vestibular nucleus (Desmedt, 1975). Some fibers branch off to the cochlear nucleus lateral to the junction of the COCB and UOCB. Before entering the internal auditory meatus, the OCB crosses to the cochlear branch of the VIII cranial nerve by way of the vestibulocochlear anastomosis of Oort.

According to Zemlin (1987), the UOCB enters the cochlea between the basal and second turn, arborizes extensively and innervates primarily the IHCs. The approximately 50-200 UOCB fibers form the inner spiral bundle, then diverge to innervate approximately 3500 IHCs. The IHCs toward the basal end of the cochlea are less extensively innervated by efferent fibers than those near the apical end (Zemlin, 1987). This is in conflict with observations reported by Warr (1978), which indicated an even distribution of efferent innervation to the inner hair cells. The crossed OCB fibers, which mostly
innervate OHCs, undergo the greatest arborization as they
diverge from approximately 400 COCB fibers to approximately
8000 tunnel radial fibers. As these tunnel radial fibers
approach the OHCs, further arborization occurs resulting in
approximately 40,000 terminal collateral fibers. The OHCs at
the basal turn of the cochlea receive the most extensive
efferent innervation, with progressively fewer efferent fibers
as the apical end is approached. All three rows of OHCs are
equally well innervated in the basal turn, but at the apical
end of the cochlea, only the innermost row of OHCs are
innervated, and these are far less extensively supplied.

The outer hair cells are innervated by efferent fibers
in a very different manner than the inner hair cells (Zemlin,
1987). In the outer hair cells, the efferent neuron synapses
directly onto the cell body and can be expected to affect the
activity of the cell by a presynaptic process (Spoendlin,
1974). OHCs which are served by both efferent and afferent
neurons are type A cells, whereas those served by afferent
fibers only are type B cells. As explained previously, type
A OHCs predominate in the basal turn of the cochlea. The
efferent neurons serving IHCs do not synapse onto the hair
cell body, but onto the cell's afferent dendrite. Spoendlin
(1974) inferred that a postsynaptic affect occurs from
efferent innervation of IHCs.

Warr (1975) employed radioactive tracers to study the
OCB. He found that the total number of fibers in this bundle
was approximately 1800, of which 60% were uncrossed. Furthermore, he discovered that specific regions of the SOC innervated a particular type of hair cell bilaterally, suggesting that "efferent organization is organized according to the cells of origin in the brain stem rather than as crossed or uncrossed fiber bundles" (Zemlin, 1987).
Physiology of the Auditory System

Cochlea

Sounds in the environment are transformed and transmitted from air pressure disturbances in the external ear to vibrations within the perilymph in the scala vestibuli via the tympanic membrane and ossicular chain. The vibrations create a traveling wave along the cochlear partition as energy is transferred to the perilymph in the scala tympani. The flexible membrane of the round window at the basal end of the scala tympani permits the necessary relief of pressure for this traveling wave to occur along the cochlear partition. The traveling wave creates a frequency-dependent area of maximal displacement on the cochlear partition. The displacement of the cochlear partition causes a relative motion between the basilar and tectorial membranes. This, in turn, results in a deflection of the outer hair cell stereocilia that are embedded in the tectorial membrane. It also results in a flow of endolymph past the inner hair cells as the volume between the two membranes is altered. This fluid streaming is believed to cause deflections in the stereocilia of the inner hair cells and to be the primary receptive mechanism of the auditory system (Dallos, 1988).

The traveling wave has been found to be a nonlinear phenomenon with exceptionally sharp tuning. Dallos (1988) postulated that the outer hair cells actively contribute energy to the traveling wave in the region of maximal
vibration. This implies that the outer hair cells serve a motor function to enhance the externally driven traveling wave. The active enhancement of basilar membrane displacement is thought to occur at frequencies at which a particular nerve fiber is optimally sensitive, but not at other frequencies. The enhanced traveling wave in the area of maximal displacement presumably increases the fluid streaming past the inner hair cells in this region, thus invoking a stronger response to the acoustic signal of the corresponding frequency. A feedback loop from the afferent fibers of the outer hair cells to the level of the contralateral superior olivary complex (or higher) and back to the outer hair cells via the crossed olivocochlear bundle is implicated in this process.

**Central auditory system**

The auditory system is classified as a secondary sensory system, with secondary receptor cells (hair cells) absorbing the stimulus energy and exciting primary sensory neurons of the auditory nerve (Durrant and Lovrinic, 1984). The receptor hair cells are highly polarized with respect to the adjacent endolymph in the scala media. Stimulation of the stereocilia results in electrochemical changes within the hair cells termed presynaptic receptor potentials. The receptor potentials are thought to moderate the flow of neurotransmitters across the synapses between these receptor cells and the dendrites of the primary sensory neurons (Neely,
1985). Postsynaptic receptor potentials thus induced in the
dendrites of the primary neurons are summated to produce
generator potentials, or potentials which trigger or inhibit
spike discharges at the axon hillock. The spontaneous spike
discharge rate of an auditory nerve fiber (or single unit),
may thus be increased or decreased. Auditory information is
thought to be encoded by this spike rate and/or the temporal
pattern of the spike discharges in the nerve fibers.

The physiology of the higher efferent fibers is not well
understood. These fibers are believed to produce changes in
neuronal processing in the intermediate nuclei of the
afferent, or ascending auditory system (Pickles, 1982).
Auditory Potentials

Resting Potentials

There are two resting potentials within the Organ of Corti: the membrane potential of the hair cells and the endolymphatic potential. These are static direct current (DC) potentials which are not related to acoustic stimulation. The intracellular membrane potential within the hair cells is maintained by active ionic transport mechanisms in the cell membranes. It is a negative charge, typically -60 to -80 millivolts (mV) with respect to the perilymph in either the scala vestibuli or the scala tympani. The endolymphatic potential is an extracellular potential maintained in the scala media by the stria vascularis. It is a positive potential of approximately +80 to +100 mV. The total potential difference across the reticular lamina separating the hair cells from the scala media is therefore approximately 160 mV. This membrane supports the stereocilia. Deflection of the stereocilia during acoustic stimulation causes a change in the permeability of hair cell membranes. The resting potential within the hair cells are altered due to the flow of ions across this membrane, thereby creating the receptor potential. The high potential difference across the reticular lamina is thought to enhance the sensitivity of the receptor mechanism.
Receptor Potentials

Two receptor potentials are generated within the receptor hair cells: the cochlear microphonic (CM) and the summating potential (SP). These stimulus-related potentials are generated in response to displacement of the stereocilia. Although they are produced in individual hair cells, the CM and SP measured at an extracellular site represent the summation of the stimulus-related electrical responses from many hair cells.

The cochlear microphonic is an alternating current potential produced in response to the alternating pressure gradient across the cochlear partition (Durrant and Lovrinic, 1984). The resultant motion of the organ of Corti causes alternating displacements of the stereocilia. This displacement produces changes in the permeability of the hair cell membranes, resulting in the generation of a dynamic electrical potential within the hair cell that closely follows the stimulus waveform. The cochlear microphonic is an exact linear transduction of the stimulus with respect to phase, frequency and intensity at intensities below 105 dB SPL (Neely, 1985). It is present at intensities near threshold, and occurs with a 130 microsecond latency (Glatke, 1983). This minimal delay can be accounted for by signal propagation through the external and middle ear systems. At a particular recording site (e.g. within the scala tympani, at the round window or within the ear canal), the measured CM represents
the summation of electrical activity from neighboring hair cells primarily. The amplitude of the CM is greatest when measured at the area of maximal basilar membrane displacement (Gibson, 1978).

The summating potential is a direct current, stimulus-related potential which is also generated within the hair cells at the reticular lamina. It has a higher threshold than the cochlear microphonic, often requiring 20-30 dB more intensity before the signal will elicit the SP (Davis, Deatherage, Rosenblut, Fernandez, Kimura and Smith, 1958). The SP is a nonlinear potential as it does not follow the polarity alterations of the stimulus. However, it has a linear grade with respect to the intensity of the stimulus. The sequence of mechanical events responsible for its production are unknown, but appear to involve some form of rectification of the stimulus waveform. This may be attributed to a differential flexibility of the basilar membrane to condensation or rarefaction stimuli, or to a differential sensitivity of the reticular lamina to stereocilia movement in opposite directions (Durrant and Lovrinic, 1984).

Like the CM, the extracellular SP represents the summation of potentials produced by numerous hair cells. Dallos (1976) employed intracochlear recording techniques to measure the summating potential at various sites within the scala vestibuli and scala tympani. Dallos' results indicated
that the polarity and amplitude of the summating potential are dependent on the recording site within the cochlea, and reflect the mechanical events occurring on the basilar membrane. A negative SP was measured at the apical slope of the traveling wave envelope and a positive SP at the basal slope. Dallos concluded that the SP was comprised of several distinct components. Intracellular recordings of receptor potentials from inner and outer hair cells have demonstrated a large direct current component within these cells which corresponds to the summed SP (Durrant and Lovrinic, 1984).

Generator Potential

The receptor potentials that occur within the hair cells in response to acoustical stimulation cause these cells to release a chemical transmitter (Gibson, 1978). This diffuses across the synaptic cleft and is received by the afferent dendrites of the auditory nerve. The transmitter alters the permeability of the dendritic membrane, resulting in a transient depolarization known as the postsynaptic generator potential. This impulse is propagated along the unmyelinated dendrite to the habenula perforata. It then triggers the excitable portion of the neuron to discharge an action potential along the myelinated portion of the auditory nerve towards the cochlear nucleus.

Action Potential

The action potential is a transient all-or-none depolarization or spike discharge that occurs in the auditory
nerve fibers in responses to an acoustic stimulus. This spike discharge is identical to the ongoing spontaneous depolarizations that occur naturally in the nerve fibers without acoustic stimulation. However, the spontaneous discharge rate and temporal pattern are altered by stimulus-related action potentials. Single action potentials occur in individual nerve fibers. When the action potential is measured at a remote site (e.g. promontory, ear canal or mastoid), it represents the gross summation of many individual fiber action potentials (Teas, Eldredge and Davis, 1962). The synchronous discharge of a group of nerve fibers caused by stimulation of a discrete area of the organ of Corti is known as the compound action potential (CAP). This is typically elicited by a frequency-specific tone pip or tone burst. The whole-nerve action potential (WN AP) represents the discharge of fibers comprising the entire auditory nerve in response to a broad band click stimulus. The term action potential (AP) usually refers to either of the latter two summated responses (CAP or WN AP) (Gibson, 1978). When the term refers to a single unit action potential, this use of the term is explicitly specified.

The amplitude, latency and morphology of the action potential are dependent upon the intensity, frequency, rise time and duration of the stimulus. The AP is primarily an onset phenomenon and requires close synchronicity of single unit action potentials for a robust response (Goldstein and
Kiang, 1958). This is achieved by providing a stimulus with a short rise time (e.g. click, tone pip or tone burst). Intense high-frequency click stimuli produce a strongly synchronized response in the basal portion of the cochlea, yielding a robust AP (Elberling, 1974; Eggermont, 1976).

The action potential is composed of two dominant waves formed by the summated neural discharges (Pickles, 1982). These are known as N1 and N2, with N1 referring to the peak with shorter latency. The amplitudes and latencies of N1 and N2 vary with the intensity of the stimulus in a complex relationship. They are also influenced by stimulus frequency, recording site and status of the auditory system. The relative contributions of these two components of the AP determine the morphology of the AP response. The amplitude of the N1 response to click stimuli is a nonmonotonic function of the stimulus intensity, with a steep slope at intensities below 60 dB SPL (peak equivalent), a leveling off of the function from 60-80 dB SPL and a moderate slope above 80 dB SPL (Durrant and Lovrinic, 1984).
Single-Unit Recordings

Recordings of the electrical activity of single auditory nerve fibers are known as single-unit recordings. These are obtained using microelectrodes placed next to an individual neuron, and thus are extracellular recordings. Nonetheless, the extremely small size of the electrode tip allows them to represent the activity of individual nerve fibers. Single-unit recordings are employed to monitor the ongoing spontaneous spike discharges within a nerve fiber as well as the response of the fiber to acoustic stimulation. They are analyzed in terms of the total number of discharges per unit time (spike rates) or in terms of the temporal aspects of the discharge patterns (histograms).

The spike rate of a single unit varies as a function of stimulus frequency and intensity. Individual fibers have a limited dynamic range (20-30 dB) and usually exhibit either a high spontaneous spike rate (e.g. 100 spikes/msec.) or a low rate (less than 1 spike/msec). The tuning curve (or isorate function) is an informative measurement based on the discharge spike rate. A tuning curve is a graph of the stimulus intensity required to maintain a constant spike rate (above the spontaneous rate) as a function of the stimulus frequency. Tuning curves describe the frequency selectivity of the auditory nerve fibers. An illustration of an idealized tuning curve is provided in Figure 1. By virtue of its specific location along the basilar membrane, a nerve fiber
Figure 1.

Illustration of an idealized tuning curve.
is optimally tuned to a characteristic frequency (CF) at which it exhibits optimal sensitivity. The fibers respond to stimuli of frequencies below the CF (on the low-frequency "skirt" of the curve) if these are presented at higher intensities (Kiang and Moxton, 1974). The high frequency "skirt" (representing frequencies above the CF) has a much steeper slope, and the fiber does not respond to frequencies significantly above its characteristic frequency. Tuning curves reflect the mechanical events occurring on the basilar membrane.

Tuning curves provide insights into the mechanisms whereby frequency and intensity information are encoded in the auditory nerve fibers. The restricted dynamic range of single fibers is inadequate to represent the wide range of human hearing sensitivity. Numerous fibers are involved in the complex encoding process in which frequency and intensity encoding are interrelated.
Evoked Potentials

General Principles

Evoked potentials (EP) are recordings of the summated far field electrical activity of many components of the auditory system (hair cells, neural dendrites and axons in the auditory nerve and brainstem nuclei) in response to acoustic stimulation (Jewett and Williston, 1971). Clinically, these potentials are recorded from surface electrodes located at various sites on the head, ear lobe or ear canal. Specialized techniques must be employed in order to isolate the auditory evoked potentials of interest from the interfering background bioelectrical activity of the brain and muscles and other ambient electrical noise. Furthermore, because the EP response is primarily an onset response, an acoustic stimulus with an abrupt rise time must be used to elicit the synchronous discharge of many auditory nerve fibers.

The recording of auditory evoked potentials is made possible by the use of differential preamplifiers, filters and computer-based signal averaging techniques. Differential preamplifiers allow for the cancellation of electrical signals that are identical at the active and reference electrodes, while the potential difference between the different electrode site is preserved and amplified. This is accomplished by common-mode rejection. Evoked potentials are typically 1/10 to 1/1000 the magnitude of the ambient noise, and a minimum common-mode rejection ratio of 60 dB (1:1000 voltage ratio)
is required (Glatke, 1983). Noise levels are also reduced by ensuring that the electrode impedance is minimized. Filters within the amplification system are employed to isolate the desired frequency band and to eliminate the noise present in unwanted frequencies. The signal-to-noise ratio is further improved by time-locked sampling of the response immediately following the stimulus presentation and averaging of a large number of samples. Evoked potentials are typically 1/10 to 1/30 the amplitude of the internal bioelectric activity (Glatke, 1983). In order to record a reproducible response from such poor signal-to-noise conditions, an average of the responses to at least 1000 individual click stimuli is necessary.

Auditory evoked potentials are classified according to their latency, anatomic origin, stimulus-response relationship or electrode placement (Jacobson and Hyde, 1985). "Fast" evoked potentials occurring within 10 milliseconds of the stimulus are commonly known as the auditory brainstem response (ABR). ABR represents the neural activity of the auditory nerve and nuclei within the brainstem. Middle latency responses (MLR) occur 10-50 msec poststimulus, and "slow" EPs occur 50-300 msec after the stimulus. The three types of responses described above are all recorded with scalp electrodes, but with different amplifier filter settings. Their component wave peaks and troughs may be readily identified by experienced clinicians. Responses occurring
before 2 msec are measured by electrocochleography (ECochG).

**Electrocochleography**

Electrocochleography (ECochG) represents the cochlear evoked potentials (CM and SP) and the AP of the auditory nerve. The active electrode must be placed closer to the cochlea to achieve the required sensitivity to record the ECochG response. Several types of active electrodes are used in ECochG, including: transtympanic membrane, tympanic membrane, and ear canal electrodes. Transtympanic membrane electrodes are thin needle electrodes that are passed through the tympanic membrane and placed on the promontory. This procedure requires a local anesthetic in adult subjects. Due to their proximity to the cochlea, transtympanic membrane electrodes allow for increased sensitivity in the measurement of N1, with thresholds 40 dB better than when measured by ear canal electrodes (Simmons, 1974). There are three types of extratympanic ear canal electrodes:

1. The Life-Tech electrode, which consists of a silver ball attached to a butterfly spring. This is placed in close proximity to the tympanic membrane after the ear canals have been thoroughly cleaned.

2. The Axonics-3M system, in which a foam ear plug serves as the electrode. The plug is saturated with electrolyte and is positioned peripherally in the ear canal. An electrode of similar design, the Nicolet Tiptrode is covered with gold foil to act
as the electrical conductor. Both of these electrodes are incorporated into insert receiver earphones.

3. The prototype tympanic membrane (TM) electrode, which consists of a small foam sponge at the end of a flexible tube. The sponge is placed in direct contact with the tympanic membrane (Stypulkowski and Staller, 1987).

The reference electrode may be placed on the contralateral or ipsilaterial earlobe or mastoid. Coates (1986) recommended that the ipsilaterial earlobe be used for the reference electrode site to minimize contamination of the response by contributions from higher order auditory neurons and muscle fibers. The ground electrode is placed at the vertex of the scalp.

The recording electrodes must be isolated from the stimulus transducer to avoid contamination of the response by electromagnetic radiation. If traditional headphones (e.g. TDH-49P) are used, they must be shielded in order to eliminate this electromagnetic stimulus artifact. Insert receiver earphones (e.g. Etymotic Research ER-3A Tubephone) often resolve the problem of stimulus artifact by locating the transducer remote from the ear, and delivering the acoustic stimulus via plastic tubing. Insert earphones have been shown to be viable transducers for evoked potential audiometry (Beauchaine, Kaminski and Gorga, 1987).
For optimal measurement of N1, a low stimulus repetition rate (e.g. 8 clicks/sec.) is recommended (Coates, 1986). At higher rates the action potential component of the response is reduced by adaptation of the auditory nerve fibers. The amplifier filter settings for the recording of the ECochG response must include low frequencies (below 150 Hz) to record the summing potential component. The SP is a direct current shift in the potential of the hair cells, and is filtered out by a high-pass filter cutoff set at 150 Hz. Still, there are no consistent standard filter settings. Coates (1981) employed filter settings of 20-3000 Hz. This contrasts with the typical filter settings of 150-3000 Hz employed during auditory brainstem evoked response (ABR) testing. It is important to note that the action potential of the auditory nerve can be measured by either ABR or ECochG techniques, although the two techniques employ different filter settings and electrode sites. The AP is termed "Wave I" in ABR recordings and "AP" or "N1" in ECochG recordings. ECochG recordings offer a more sensitive measure of the compound action potential.

The summing potential and cochlear microphonic components of the waveform are superimposed on one another in recordings of the response evoked by single-polarity click stimuli. However, the CM, which is an exact replication of the stimulus, may be eliminated if half of the stimuli are presented in the opposite phase. In this condition, the CM
component will sum to zero (Glatke, 1983; Coates, 1986). The summating potential and action potential components of the response do not change in polarity when the phase of the stimuli are reversed, and thus remain intact. The elimination of the CM may be accomplished by employing either of the following techniques:

1. Providing click stimuli of alternating polarity so that the total number of rarefaction and condensation clicks are equal, or
2. Summating the two separate waveforms recorded in response to presentation of click stimuli of opposite polarity to form a composite waveform.

The second procedure was recommended by Coates (1986) as superior as it avoids the possibility of the artifact rejection system introducing bias by blocking a disproportionately high number of responses to clicks of one polarity.

Eggermont (1976) observed a great intersubject variability in measurements of the SP and AP. He assumed that this variation was due to influences affecting both responses, and proposed that an expression of the SP/AP ratio be calculated in order to reduce the variability across subjects. This SP/AP ratio has gained clinical acceptance as an indicator of pathologic condition (Meniere's disease) (Coates, 1986). However, a large degree of variability has been demonstrated in measurements of the SP/AP ratio (Chatrian,
Wirch, Edwards, Lettich and Snyder, 1984; Coates, 1981, 1986). As a result, Coates (1986) recommended that the AP-normalized SP amplitude be employed as it represents a more consistent relationship between the SP and the AP.
Physiological Studies of the Efferent Auditory System

The efferent auditory system has been studied by stimulating the olivocochlear bundle electrically and acoustically. The effects of OCB stimulation have been observed in single-unit recordings and in far field auditory evoked potentials. Electrical stimulation and single-unit recordings are laboratory techniques that are performed on animal subjects. Clinical investigations using human subjects are ethically restricted to acoustical stimulation and evoked potential recording techniques. Suppression of the action potential by electrical stimulation of the crossed OCB was first demonstrated by Galambos (1956) using cats as experimental subjects. He observed that electrical stimulation resulted in the complete suppression of N1 in response to click stimuli of low intensity (e.g. 15 dB SL), whereas more intense stimuli (35 dB SL) were only partial suppressed (40%). A similar relationship between ipsilateral click stimuli intensity and the degree of suppression was observed by Sohmer (1966) and Klinke and Galley (1974). Sohmer (1966) also demonstrated that the suppression of the action potential caused by electrical stimulation of the crossed OCB was about six times greater than that caused by stimulation of the uncrossed OCB. He reported that the cochlear microphonic did not change in response to stimulation of the OCB. In contrast, Klinke and Galley (1974) noted an increase in the cochlear microphonic in response to electrical
stimulation of the crossed OCB. The effects of electrical stimulation of the crossed OCB on the potentials of the peripheral system are discussed more thoroughly in reviews by Desmedt (1975) as well as by Klinke and Galley (1974). A succinct summary describing the effects of efferent stimulation has been provided by Dolan and Nuttall (1988):

"In general, the electrical stimulation of the COCB causes a decrease in the endocochlear potential (EP), a small increase in the cochlear microphonic (CM), and a suppression of the cochlear whole-nerve action potential (CAP) with low-to moderate-intensity acoustic stimulation." (p. 1081)

Finally, Klinke and Galley (1974) concluded that the CM is affected by a presynaptic process which produces a change in the receptor (hair) cell membrane resistance. The AP is suppressed by a postsynaptic process which affects the dendrites of the auditory nerve fibers (Klinke and Galley, 1974; Desmedt, 1975). Furthermore, the AP may suppressed by an active cochlear process involving the outer hair cells (Dallos, 1988).

Single-unit recordings of auditory nerve fiber responses have also been found to be suppressed by tonal stimulation of the contralateral ear (Buno, 1978). Buno demonstrated that contralateral tones at or near the center frequency of the ipsilateral stimulus produced the greatest suppression of the action potential.
As indicated in the introductory chapter, a recent study using human subjects performed by Folsom and Owsley (1987) demonstrated that acoustical stimulation of the crossed OCB produced an inhibitory effect on the action potential of the auditory nerve. In their investigation to demonstrate the suppressive effects of contralateral masking on N1, Folsom and Owsley (1987) employed a combination of ABR and ECochG techniques when recording the evoked potential responses. An ear canal electrode was used in their study (as in ECochG) however, their filter settings were selected for recording the ABR (150-3000 Hz) rather than ECochG (20-3000 Hz). This filter setting may have excluded the summating potential from the response.

The evidence cited above suggests that the efferent auditory system has a direct effect on cochlear micromechanics. The mechanism involved is still unknown, but current theory suggests that this mechanism resides in the hair cells and their activity. The effects of the efferent system that have been observed in the action potential in human subjects have yet to be demonstrated in the cochlear potentials. The summating potential can be readily measured, and is thus the response of interest in this investigation. The purpose of this investigation is to determine whether the effects of the efferent auditory system extend to a level within the cochlea or whether they are a retrocochlear phenomenon. More specifically, the following questions will
be addressed:

1) Is the SP/AP amplitude ratio calculated from the response to a frequency-specific filtered click stimulus altered by the introduction of a contralateral pure tone masking signal at its center frequency?

2) Are the absolute amplitudes of the SP and/or AP responses to a frequency-specific filtered click stimulus altered by the introduction of a contralateral pure tone masking signal at its center frequency?

If the stimulation of the efferent auditory system by a pure-tone presented to the contralateral ear produces a systematic change in the afferent auditory system, then this change will be demonstrated in the amplitude of the summating potential, the amplitude of the action potential and the SP/AP amplitude ratio in response to filtered click stimuli as measured by ECochG techniques.
CHAPTER III: METHODS

Subjects

Ten young-adult subjects with normal hearing volunteered for participation in this study. These subjects were recruited from graduate students in the Department of Communication Sciences and Disorders at the University of Montana, and included 4 male and 6 female students between the ages of 23 and 29 years. The mean subject age was 25.45 years with a standard deviation of 2.21 years. The subjects reported no history of significant ear disease as determined by a case history (see Appendix B). All subjects had hearing sensitivity within normal limits as measured by pure-tone air conduction audiometry (thresholds no poorer than 10 dB HL for the test frequencies at octave intervals between 500 and 8000 Hz). All had normal tympanograms (Jerger type A). The subjects were required not to demonstrate any measurable acoustic reflex responses to a 45 dB SL pure-tone stimulus at 4000 Hz.

Instrumentation

Stimuli: Filtered clicks were used for the ipsilateral stimulus. They were produced by generating direct current pulses of 0.15 milliseconds (msec) duration at a rate of 8.0 clicks per second from a Tracor Northern Nomad evoked potential system. The clicks were filtered through an Allison Laboratories AL2B variable filter set to pass a third-octave band centered at 4000 Hz. The filtered clicks were then
amplified (McIntosh model MC 40 amplifier) and delivered to a TDH-49P headphone. The contralateral pure-tone masking signal was generated by a portable audiometer (Qualitone Auditory Screener model) and delivered via a TDH-49P headphone. Both headphones were mounted in circumaural cushions. The portable audiometer was calibrated for frequency and intensity immediately before and after the experimental phase of the investigation.

**Recording:** The evoked potential responses were received by two Grass gold-plated surface EEG electrodes and one Life-Tech ear canal electrode. These were connected to a Tracor Northern recording system (Nomad model) with the preamplifier set to a bandpass of 20 to 3,000 Hz and the signal averager set to a 5 ms period. The 60 Hz notch filter of the preamplifier was enabled in order to reduce the interference from ambient electromagnetic radiation. The resultant waveforms were stored on diskettes by the system computer for later analysis.

**Procedure**

This study was approved by the Internal Review Board of the University of Montana. Prior to the collection of data, informed consent was obtained from the adults participating in this investigation. A copy of the consent form is included in Appendix C. All subjects were then screened following the guidelines outlined above (see Subjects).

In preparation for recording the evoked potentials, the
subject’s skin was cleaned with alcohol and omniprep cleanser at the surface electrode sites. Gold-plated surface EEG electrodes were attached to their forehead (non-inverting) and ipsilateral earlobe (ground) using electrode paste and skin tape. The subject’s ear canals were then cleaned with alcohol and hydrogen peroxide by an otolaryngologist and the ear canal electrode was placed inside the ipsilateral ear canal (inverting). The electrode montage was established to provide positive up electrocochleograms. The impedance between the surface electrodes was held below 1,200 ohms, and below 15,000 ohms at the ear canal electrode.

The subjects were seated comfortably in a reclining chair. Behavioral thresholds were obtained for the 4000 Hz filtered click and for the contralateral 4000 Hz pure tone. The subjects were then told to relax or to sleep if possible.

The ipsilateral stimulus (filtered clicks) was presented at 45 dB SL. This level was chosen because it was of sufficient intensity to elicit a clear AP and SP response, yet low enough to demonstrate the inhibitory effects of the efferent system on the action potential. The final averaged response was the consisted of 1024 stimulus presentations. An initial recording was taken to ensure that both SP and AP were sufficiently robust for scoring and analysis. If necessary, the ear canal electrode or the electrode leads were repositioned to provide clear responses before the testing commenced.
This study employed single-subject, pre-test post-test design with two trials in each condition. In the control condition, no contralateral masking tone was presented while the responses to ipsilateral filtered clicks were recorded. The experimental condition consisted of trials during which the ipsilateral evoked responses were recorded with the simultaneous presentation of a contralateral masking tone. The contralateral pure-tone masker was presented at 45 dB SL. This level was selected so as to provide adequate stimulation of the crossed OCB (Folsom and Owsley, 1987) without exceeding interaural attenuation (Goldstein and Neuman, 1985) or eliciting an acoustic reflex. This provided the maximum acoustical stimulation of the efferent system without interfering with the experiment.

Evoked potential waveforms were recorded separately in response to rarefaction and condensation clicks for both experimental conditions. The responses were recorded alternatively for each experimental condition to reduce the possible contaminating influence of subject state. A total of eight waveforms were recorded from each subject. These were stored independently in eight computer memory locations, and then saved on a floppy diskette for later analysis. Waveform pairs (in response to rarefaction and condensation clicks) for each experimental condition were then summed to produce composite waveforms from which the CM had been eliminated. This resulted in 4 final composite waveforms.
The composite waveform in each condition was thus replicated in order to demonstrate the reliability of AP and SP identification.

**Scoring and Analysis**

All waveforms were numerically coded prior to storage on the computer diskette to avoid examiner bias in the scoring of these responses. A separate subject-information sheet specifying the subject, stimulus and masking conditions for each waveform was maintained separate from the data. An example of this form is provided in Appendix D.

Each composite waveform was scored independently for AP and SP amplitude and for AP latency by two judges experienced in the analysis of evoked potential waveforms. Amplitudes of the AP were scored on hard copy by the triangulation method employed by Folsom and Owsley (1987). The amplitude of the action potential was defined as the vertical distance from the AP peak to the line joining the two deflection points of the AP peak. The method employed to measure the amplitude of the AP is illustrated in Figure 2. Amplitude measures were recorded in microvolts (uV). The AP latencies was defined as the time in milliseconds from the stimulus onset to the AP peak. The latencies of the APs were scored directly on the computer. The amplitude of the SP was defined as the vertical distance from the SP peak to the same triangulated baseline that was used to score the AP. The method used to measure the SP amplitude is also illustrated in Figure 2. Discrepancies
Figure 2.

Measurement of the action potential and the summating potential.
in scoring were resolved by rescoring the disputed waveforms with both judges present.

The differences between the experimental and control conditions were analyzed separately for the amplitudes of APs and SPs, the AP latencies and for the SP/AP amplitude ratios. This was accomplished using two-way analysis of variance (ANOVA) (trial by masking condition) with one repeated measure. Confidence levels were set to 0.05 prior to all analyses. The dependent variables included the amplitudes of AP and of SP, the latency of the AP, as well as the SP/AP amplitude ratio. In addition, data points for each experimental condition were analyzed to yield the mean and standard deviation for the two trials under both conditions.

Reliability

The amplitudes of the action potential and summating potential for each waveform were scored separately by two judges. Values differing by more than 0.05 microvolts were rescoring with both judges present. Rescoring was required on three waveforms. Pearson product-moment correlation coefficients for all waveforms were calculated separately for interjudge reliability for the summating potential \((r = 0.999)\) and for the action potential \((r = 0.997)\). These values indicated high inter-judge reliability. The amount of systematic variation present in the measurements of the AP and SP amplitudes and of the AP latencies for each subject in each condition were analyzed by calculating interclass reliability coefficients (Bartko, 1966).
CHAPTER IV: RESULTS

The present study extended the Folsom and Owsley (1987) investigation into the effects of contralateral masking on the ipsilateral electrophysiological response. Specifically, this study attempted to replicate the reported inhibitory effects of the masker on the action potential (AP). The current study also examined the effects of masking on the summating potential (SP), the SP/AP ratio, and on the latency of the AP. Two waveforms were obtained for each subject under each condition and the amplitude of the APs, SPs, as well as the latency of the APs were scored for each waveform. A two way analysis of variance (ANOVA) (trial by masking condition) with one repeated measure was applied to the data to address each of the research questions. In addition, the measurement variability of each repeated measure (AP and SP amplitudes, and AP latency) was analyzed by calculating Pearson’s product-moment correlation coefficients (Pearson’s r) for all trials in both conditions. Intraclass correlation coefficients (ICC) were also calculated to analyze the systematic variation within the repeated measures (Bartko, 1966).

Waveforms

The unmasked and masked waveform pairs constituting one trial are presented for two subjects in Figures 3 and 4. The waveforms in Figure 3 illustrate a case in which the SP/AP ratio increased with the introduction of the contralateral masker, whereas the waveforms in Figure 4 illustrate a de-
Figure 3.

Masked and unmasked waveform pair recorded from subject number 10 illustrating an increase in the SP/AP ratio from the control condition (contralateral masking tone not present) to the experimental condition (contralateral masking tone present).

a. Unmasked

b. Masked
Figure 4.

Masked and unmasked waveform pair recorded from subject number 8 illustrating a decrease in the SP/AP ratio from the control condition (contralateral masking tone not present) to the experimental condition (contralateral masking tone present).

a. Unmasked

b. Masked
crease in the SP/AP ratio from the unmasked to the masked condition.

The amplitude and latency values of the action potential and the amplitudes of the summating potential for all waveforms are presented in Appendix E. The calculated SP/AP ratios for the waveforms are also presented in Appendix D. The means and standard deviations for the AP amplitudes, SP amplitudes and SP/AP ratio for each of the two trials in both the control and experimental conditions are presented in Table 1. The means and standard deviations for the latencies of the AP for each of the trials in both conditions are presented in Table 2. The Pearson product-moment correlation coefficients and intraclass correlation coefficients for the amplitudes of the action potentials and summating potentials as well as for the latencies of the action potentials in both the unmasked and masked conditions are presented in Table 3.

**Action Potential Amplitudes**

The mean action potential amplitudes ranged from 0.453 to 0.463 microvolts across all trials and conditions, with standard deviations ranging from 0.282 to 0.342 microvolts. A two way ANOVA with one repeated measure indicated that there were no significant differences between the experimental and control conditions (F=0.024; df=1, 20; p>0.05) or between the two trials in either condition (F=0.002; df=1, 20; p>0.05) for the AP amplitude. In addition, there was no significant interaction between the trials and conditions for the action
Table 1.

Means and standard deviations of the action potential and summating potential (in microvolts) and of the AP/SP ratios for repeated trials in the control (unmasked) and experimental (masked) conditions.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Unmasked</th>
<th></th>
<th>Masked</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Trail 1</td>
<td>Trial 2</td>
<td>Trial 1</td>
<td>Trial 2</td>
</tr>
<tr>
<td>Action Potential</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.453</td>
<td>0.463</td>
<td>0.453</td>
<td>0.456</td>
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<tr>
<td>S.D.</td>
<td>0.342</td>
<td>0.282</td>
<td>0.318</td>
<td>0.322</td>
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<tr>
<td>Summating Potential</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.125</td>
<td>0.143</td>
<td>0.146</td>
<td>0.155</td>
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<tr>
<td>S.D.</td>
<td>0.083</td>
<td>0.083</td>
<td>0.109</td>
<td>0.107</td>
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<tr>
<td>SP/AP Ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.350</td>
<td>0.343</td>
<td>0.345</td>
<td>0.386</td>
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<tr>
<td>S.D.</td>
<td>0.202</td>
<td>0.121</td>
<td>0.170</td>
<td>0.166</td>
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</tbody>
</table>
Table 2.

Means and standard deviations of the action potential latency (in milliseconds) for repeated trials in the control (unmasked) and experimental (masked) conditions.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Unmasked</th>
<th>Masked</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Trail 1</td>
<td>Trail 2</td>
</tr>
<tr>
<td>Action Latency</td>
<td></td>
<td></td>
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<tr>
<td>Mean</td>
<td>2.353</td>
<td>2.332</td>
</tr>
<tr>
<td>S.D.</td>
<td>0.233</td>
<td>0.229</td>
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</table>
Table 3.

Pearson product-moment correlation coefficients and intraclass correlation coefficients for the amplitudes of the action potentials and summating potentials as well as for the latencies of the action potentials in both the unmasked and masked conditions.

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Pearson’s r</th>
<th>ICC</th>
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</thead>
<tbody>
<tr>
<td>Action Potential</td>
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<td></td>
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<tr>
<td>Unmasked</td>
<td>0.95</td>
<td>0.94</td>
</tr>
<tr>
<td>Masked</td>
<td>0.99</td>
<td>0.99</td>
</tr>
<tr>
<td>Summating Potential</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unmasked</td>
<td>0.70</td>
<td>0.71</td>
</tr>
<tr>
<td>Masked</td>
<td>0.84</td>
<td>0.85</td>
</tr>
<tr>
<td>Latency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unmasked</td>
<td>0.97</td>
<td>0.97</td>
</tr>
<tr>
<td>Masked</td>
<td>0.98</td>
<td>0.98</td>
</tr>
</tbody>
</table>
potential \((F=0.024; \text{df}=1,20; \ p>0.05)\). The results of the two way analysis of variance with repeated measure describing the effects of trials and condition on the action potential are presented in Table 4.

There was very little variability in the repeated measures of the AP as indicated by the Pearson product-moment correlation coefficients \((0.95 \text{ to } 0.99)\). The intraclass correlation coefficients \((0.94 \text{ to } 0.99)\) indicated that the reliability of the repeated measurements of the AP in both masked and unmasked conditions was excellent.

For each subject, a measure of the changes in the amplitudes of the AP for each trial \((\triangle \text{ AP})\) were calculated by subtracting the AP amplitude in each unmasked trial from the AP amplitudes measured in the masked trials. Figure 5 illustrates these changes in the AP amplitudes for each subject in the study. The changes in the masked AP amplitudes from the two unmasked trials are presented separately in this figure. A value of zero for the change in the AP in Figure 5 indicated that there was no difference between the AP measured in the masked and unmasked conditions. A positive value for the change in the AP indicated that the AP measured in the masked trial was greater than the unmasked AP, and a negative value indicated that the masked AP was less than AP obtained in the unmasked trial. The lack of any particular trend in the masked condition is readily apparent, as was expected from the statistical analysis.
Table 4.

Two way analysis of variance with repeated measure describing the effects of trials and condition on the action potential.

<table>
<thead>
<tr>
<th>Source</th>
<th>S.S.</th>
<th>D.F.</th>
<th>M.S.</th>
<th>F-Ratio</th>
<th>p</th>
</tr>
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<tr>
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<td>3.922</td>
<td>19</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial</td>
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<td>0.000</td>
<td>0.002</td>
<td>0.965</td>
</tr>
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<tr>
<td>Within Group</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
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<td>1</td>
<td>0.000</td>
<td>0.024</td>
<td>0.879</td>
</tr>
<tr>
<td>Trial by Condition</td>
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<td>0.000</td>
<td>0.024</td>
<td>0.879</td>
</tr>
<tr>
<td>Condition by Subjects</td>
<td>0.093</td>
<td>18</td>
<td>0.005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>4.015</td>
<td>39</td>
<td></td>
<td></td>
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</table>
Figure 5.

Changes in the action potential amplitudes from the unmasked to the masked trials (AP1 = action potential in trial 1; AP2 = action potential in trial 2).
Summating Potential Amplitudes

The mean values for the SP ranged from 0.125 to 0.155 microvolts across all trials and conditions, with standard deviations ranging from 0.083 to 0.109 microvolts. A two way ANOVA with one repeated measure indicated that there were no significant differences between the two experimental conditions ($F=0.676; \text{df}=1,20; p>0.05$) or between the two trials ($F=0.111; \text{df}=1,20; p>0.05$) for the SP amplitude. The interaction between the trials and conditions for the SP amplitude was not significant ($F=0.050; \text{df}=1,20; p>0.05$). The results of the two way analysis of variance with repeated measure describing the effects of trials and condition on the summating potential are presented in Table 5.

There was fair to good correlation between the repeated measures of the SP as indicated by the Pearson product-moment correlation coefficients (0.70 to 0.86). The intraclass correlation coefficients (0.71 to 0.85) indicated that the reliability of the repeated measurements of the SP in both masked and unmasked conditions was fair to good.

For each subject, the changes in the amplitudes of the SP ($\Delta$ SP) were calculated by subtracting the SP amplitude in each unmasked trial from the SP amplitudes measured in the masked trials. Figure 6 illustrates these changes in the SP amplitudes for each subject. The changes in the masked SPs from the two unmasked trials are presented separately in this figure. Positive and negative values for the changes in the
Table 5.

Two way analysis of variance with repeated measure describing the effects of trials and condition on the summatting potential.

<table>
<thead>
<tr>
<th>Source</th>
<th>S.S.</th>
<th>D.F.</th>
<th>M.S.</th>
<th>F-Ratio</th>
<th>p</th>
</tr>
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<tr>
<td>Between Groups</td>
<td>0.298</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Trial</td>
<td>0.002</td>
<td>1</td>
<td>0.002</td>
<td>0.111</td>
<td>0.743</td>
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<tr>
<td>Subjects Within Group</td>
<td>0.296</td>
<td>18</td>
<td>0.016</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Within Group</td>
<td>0.075</td>
<td>20</td>
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<td></td>
</tr>
<tr>
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<td>0.003</td>
<td>0.676</td>
<td>0.422</td>
</tr>
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<td>Trial by Condition</td>
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<td>0.000</td>
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<td>0.825</td>
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<td>Condition by Subjects</td>
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<td>18</td>
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<td></td>
</tr>
<tr>
<td>Total</td>
<td>0.374</td>
<td>39</td>
<td></td>
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</tr>
</tbody>
</table>
Figure 6.

Changes in the summating potential amplitudes from the unmasked to the masked trials (SP1 = summating potential in trial 1; SP2 = summating potential in trial 2).
SP represent trials in which the SPs measured in the masked trials were greater or less than, respectively, the unmasked SPs. No trend towards any systematic variation in the SP amplitude from unmasked to masked conditions was apparent.

**SP/AP Ratios**

The mean SP/AP ratios ranged from 0.343 to 0.386 across all trials and conditions, with standard deviations ranging from 0.121 to 0.202. A two way ANOVA indicated that there were no significant differences between the two conditions ($F=0.223; \text{df}=1,20; p>0.05$) or between the two trials ($F=0.062; \text{df}=1,20; p>.05$) for the SP/AP ratios. The interaction between the trials and conditions for the SP/AP ratios was not significant ($F=0.337; \text{df}=1,20; p>.05$). The results of the two way ANOVA with repeated measure describing the effects of trials and condition on the SP/AP ratios are presented in Table 6.

For each subject, the changes in the SP/AP ratios ($\triangle$ SP/AP ratio) were calculated by subtracting the value of the SP/AP ratios for each unmasked trial from the SP/AP ratios for the masked trials. Figure 7 illustrates these changes in the SP/AP ratios for each subject. The changes in the masked SP/AP ratios from the two unmasked trials are presented separately in this figure. Positive and negative values for the changes in the SP/AP ratios represent trials in which the ratios calculated in the masked trials were greater or less than, respectively, the unmasked SP/AP ratios. No trend
Table 6.
Two way analysis of variance with repeated measure describing the effects of trials and condition on the SP/AP Ratios

<table>
<thead>
<tr>
<th>Source</th>
<th>S.S.</th>
<th>D.F.</th>
<th>M.S.</th>
<th>F-Ratio</th>
<th>p</th>
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<tr>
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<td>0.807</td>
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<td>0.003</td>
<td>0.062</td>
<td>0.807</td>
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<td>Subjects Within Group</td>
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<tr>
<td>Within Group</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
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<td>1</td>
<td>0.004</td>
<td>0.223</td>
<td>0.643</td>
</tr>
<tr>
<td>Trial by Condition</td>
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<td>1</td>
<td>0.006</td>
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</tr>
<tr>
<td>Condition by Subjects</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>1.135</td>
<td>39</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 7.

Changes in the SP/AP ratios from the unmasked to the masked trials (SP/AP Ratio 1 = SP/AP ratio in trial 1; SP/AP Ratio 2 = SP/AP ratio in trial 2).
towards any systematic variation in the SP/AP ratio from unmasked to masked conditions was apparent.

**Action Potential Latencies**

The mean AP latency values for the two trials in each experimental condition are presented in Table 2. The mean AP latency ranged from 2.331 to 2.353 milliseconds, with standard deviations from 0.197 to 0.233 milliseconds. A two way ANOVA indicated that there were no significant differences between the control and the experimental conditions \( (F=0.011; \ df=1,20; \ p>0.05) \) or between the two trials \( (F=0.000; \ df=1,20; \ p>0.05) \) for the AP latency. There was no significant interaction between trials and masking conditions for the AP latency \( (F=0.1.140; \ df=1,20; \ p>0.05) \). The results of the two way ANOVA with repeated measure describing the effects of trials and condition on the AP latencies are presented in Table 7.

The repeated measures of the AP latencies were highly correlated as indicated by Pearson product-moment correlation coefficients (0.97 to 0.98). The intraclass correlation coefficients (0.97 to 0.98) indicated that the repeated measurements obtained for the latency of the action potential were highly reliable.

**Summary**

In summary, there were no significant changes in the AP or SP amplitudes, the SP/AP ratios or the AP latencies between the unmasked and masked conditions or between the two repeated trials. In addition, there was no significant interaction
Table 7.

Two way analysis of variance with repeated measure describing the effects of trials and condition on the AP Latencies.

<table>
<thead>
<tr>
<th>Source</th>
<th>S.S.</th>
<th>D.F.</th>
<th>M.S.</th>
<th>F-Ratio</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
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<td>1.823</td>
<td>19</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial</td>
<td>0.000</td>
<td>1</td>
<td>0.000</td>
<td>0.000</td>
<td>0.992</td>
</tr>
<tr>
<td>Subjects Within Group</td>
<td>1.823</td>
<td>18</td>
<td>0.101</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Within Group</td>
<td>0.672</td>
<td>20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
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<td>0.000</td>
<td>0.011</td>
<td>0.916</td>
</tr>
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<td>0.004</td>
<td>1.140</td>
<td>0.300</td>
</tr>
<tr>
<td>Condition by Subjects</td>
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<td>18</td>
<td>0.004</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>1.890</td>
<td>39</td>
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<td></td>
</tr>
</tbody>
</table>
between the trials and conditions for any of these parameters. The repeated measurements of the AP amplitude and latency were highly correlated and were demonstrated to possess a high reliability. Finally, the correlations of the repeated measurements of the SP amplitude were fair to good, and the reliability of the repeated SP amplitude measures was fair to good.
CHAPTER V: DISCUSSION

The purpose of the current study was to examine the effects of the efferent auditory system on the evoked response as measured by electrocochleography. An attempt was made to replicate a previous study which had indicated that the action potential was suppressed by acoustical stimulation of the efferent system. In addition, several different techniques were employed to record the summating potential in order to determine whether acoustical stimulation of the efferent system by a contralateral masking tone affected this cochlear potential, the action potential or the SP/AP ratio.

Action Potential

When a masking tone was introduced to the contralateral ear at the central frequency of the 4000 Hz filtered click, there were no systematic changes observed in the amplitude or latency of the action potential evoked by a one-third octave filtered click stimulus. Indeed, minimal change in the AP was observed between the control and experimental conditions. This evidence failed to support the findings of Folsom and Owsley (1987) which demonstrated an inhibitory effect of contralateral masking on the AP. Three possible explanations for the discrepant experimental findings between the present study and Folsom and Owsley's investigation are proposed. The differences in the data between these studies may be due to:

1. differences in the identification of the waveform peaks which were selected to represent the AP,
2. differences in the intensities at which the ipsilateral stimuli were delivered, or
3. differences in the measurement techniques between the two investigations.

Identification of the Action Potential

The correct identification of the AP is essential to the validity of either study. While the amplitudes of the various components of evoked responses are expected to vary greatly between subjects, the latencies of these responses remain stable. Normative data for the latency of any particular component (e.g. the action potential or Wave I) are routinely used to aid in its identification. The latencies of the components systematically vary as a function of the intensity of the ipsilateral stimulus in evoked response audiometry (Schwartz and Berry, 1985). Specifically, latencies consistently decrease as the intensity of the stimulus is increased. Thus, the identification of the AP may be validated by comparing its latency to established norms.

The measurements of the AP obtained in the present study were validated by referencing the latency measures to established normative data. Cox (1985) reported that mean values for the AP latency in adult subjects were 2.23 milliseconds (S.D. of 0.34 milliseconds) in response to a broad band click at 50 dB HL, and 2.46 milliseconds (S.D. of 0.31) at 40 dB HL. Singh and Mason (1981) reported mean AP latency values of 2.4 milliseconds (S.D. of 0.3) in response
to a 50 dB HL broad band click stimulus. However, the different types of stimuli employed (filtered versus broad band clicks) were also considered when comparing data across studies. Wynne (1988) demonstrated that the AP latency was decreased by an average of 0.07 milliseconds at 60 dBnHL by restricting the frequency of the stimulus from a broad band click to a one-half octave band region along the cochlear partition centered around 4000 Hz in a notched noise paradigm. A similar effect (a decrease in AP latency in the order of 0.1 millisecond) was expected to occur as a result of the filtering of the click stimuli in the current study. Therefore, 0.1 millisecond was added to the filtered click latencies to account for the effects of filtering before they were compared to the normative data from other studies. The intensity of the ipsilateral stimulus employed in the present study was 45 dB SL, with subject thresholds no poorer than 10 dB HL. Thus, the intensity of the filtered clicks was no greater than 55 dB HL, and was in the range from 45 to 55 dB HL. The mean AP latencies obtained in the current study ranged from 2.33 to 2.35 milliseconds. These mean AP latencies for the filtered click stimulus corresponded to approximately 2.45 milliseconds when corrected for comparison with responses evoked by broad band clicks. Thus, the AP latency values obtained in the current study were consistent with stimuli of the same intensity as reported in other normative studies.
In contrast, the AP latencies reported by Folsom and Owsley were prolonged by approximately 1 millisecond as compared with the normative data described in the previous paragraph. They reported mean AP latencies of 3.39 to 3.42 milliseconds at 40 dB SL, and 3.75 to 3.78 milliseconds at 30 dB SL. The values reported by Folsom and Owsley were not consistent with this normative data and, as a result, their identification of the AP and the experimental effects they reported may be invalid. They may have identified Wave II, which occurs approximately 1 millisecond later, as the AP. Wave II is thought to originate mainly from the proximal portion of the auditory nerve (Moller and Janetta, 1985). As such, the inhibitory effects that Folsom and Owsley observed may have been caused by changes in the synaptic potentials between the auditory nerve and the cochlear nucleus rather than suppression of the action potential of the auditory nerve itself.

**Intensity of the Ipsilateral Stimulus**

An audible equivalent input noise was present at the test ear in the absence of any filtered click stimuli. This electrical noise was generated by both the Nomad evoked response unit and the Macintosh amplifier. This noise may have confounded the data obtained in the present investigation by one or both of the following mechanisms:

1. the noise may have masked the behavioral threshold to the filtered click and, as a consequence, shifted
the presentation level of the ipsilateral stimulus, or

2. the noise may have somehow altered the AP response during the control and/or the experimental conditions.

The background electrical noise was present during the determination of the behavioral thresholds for the filtered clicks, and may have acted as a masking signal, falsely elevating these behavioral thresholds. The intensity of the ipsilateral click stimulus was adjusted to a level 45 dB greater than this filtered click threshold (a 45 decibel sensation level or 45 dB SL). In addition, each subject was required to have pure-tone thresholds no poorer than 10 dB HL at 4000 Hz. Therefore, the intended maximum presentation level was less than 55 dB HL. However, if the behavioral thresholds for the filtered clicks had been elevated by the background electrical noise and the presentation level for the ipsilateral stimulus was 45 dB SL in relation to this threshold, then the actual intensity level at which the clicks were presented in the present study could have been higher than the intended intensity level. As a result, the intensity of the ipsilateral stimulus might have exceeded the level at which the inhibitory effects of contralateral masking could be demonstrated in the amplitude of the AP. However, a significant elevation of the stimulus intensity would have caused a decrease in the latency of the AP. As the AP
latencies obtained in this study were in agreement with the normative data for moderate intensity levels, the equivalent input noise of the amplification system did not appear to significantly alter the hearing level at which the ipsilateral stimulus was presented.

As the electrical noise was present as a background acoustical stimulus upon which the filtered clicks were overlaid, it is also possible that this ongoing low-level stimulation may have confounded the elicitation of the AP and hidden any possible experimental effects. For example, if the noise present in the signal was sufficient to augment the amplitude of the AP, then any inhibition of the AP caused by the introduction of the contralateral masking tone might have been obscured.

**Measurement Techniques**

The measurement techniques employed by Folsom and Owsley may have interfered with their ability to successfully record and accurately identify the AP. They used single-polarity clicks as the ipsilateral stimulus. Accordingly, the cochlear microphonic and stimulus artifacts would have been present in their recordings. These components in the waveform can obscure the identification of the AP by reducing the morphology of the AP response. In the present study, the recordings obtained in response to rarefaction and condensation clicks were summed in order to produce a composite waveform from which the cochlear microphonic and
most of the stimulus artifact had been eliminated. This measurement technique thus enhanced the accurate identification of the AP by improving the morphology of the waveforms.

The differences in measurement technique between the two studies might have resulted in differences in the amplitudes of the AP responses that were recorded. The values obtained for the amplitude of the AP in the two studies were not consistent. Folsom and Owsley's mean values for AP amplitude ranged from 0.097 to 0.124 microvolts at 30 dB SL, and from 0.114 to 0.132 microvolts at 40 dB SL. The mean AP amplitudes in the present study were 3 to 4 times as large, ranging from 0.451 to 0.458 microvolts. The AP amplitudes should have been more directly comparable. However, the reduced AP amplitudes in Folsom and Owsley's study could have been caused by placement of the ear canal electrodes at a more distal site.
Summating Potential

The introduction of contralateral pure-tone masking did not produce a significant change in the amplitude of the SP. The reliability of the measurement of the SP was fair to good. The decreased reliability when compared to the AP was not surprising considering that the intensity of the ipsilateral stimuli employed approximated the threshold for measurement of this potential. Davis, Deatherage, Rosenblut, Fernandez, Kimura and Smith (1958) reported that the SP threshold was approximately 20-30 dB above that of the cochlear microphonic. Durrant (1986) reported that the SP was indistinguishable from the noise floor at intensities of 70-80 dB SL when measured in response to filtered click centered around 2000 Hz. The successful recording of the SP responses in the present study was attributed to the low impedance values attained for the ear canal electrode and the close proximity of the electrode to the tympanic membrane, enhancing the measurement of this cochlear potential. Still, the low levels at which this response was obtained may have obscured excitation of the SP due to the ceiling effects at this measurement level.
The SP/AP ratio did not change significantly with the introduction of contralateral masking. This finding was contrary to the hypothesis that the SP/AP ratio would increase with the introduction of contralateral masking. This ratio is reported to be a sensitive indicator of the interaction between the AP and the SP (Eggermont, 1976b). The SP/AP ratio would have increased if either:

1. the AP had decreased in the experimental condition, or

2. the SP had increased in the experimental condition.

Both of these events were expected to occur. A decrease in the AP amplitude in response to acoustical stimulation of the efferent auditory system has been reported by Buno (1978) in an investigation involving animal subjects, and by Folsom and Owsley (1987) in human subjects. Electrical stimulation of the olivocochlear bundle has been shown to cause an increase in the cochlear microphonic (Dolan and Nuttall, 1988). The SP was expected to increase with acoustical stimulation of the efferent system because it is hypothesized to be a rectified form of the CM. The SP/AP ratio might have increased significantly between the two conditions, even though neither the AP nor the SP had been altered sufficiently to demonstrate statistical significance. The SP/AP ratio reflects the changes in both the AP and the SP.
Experimental Methodology

Although no differences were observed between the trials and conditions, the possibility of a Type II error could not be discounted due to the small number of subjects used in this study. A Type II error has occurred if the null hypothesis has been accepted when it was truly false and would have been rejected if the sample size had been increased. A visual inspection of the data suggested that there were no apparent systematic variation across measures.

Two aspects of experimental methodology were crucial to the recording of the ECochG response. The first was the proximity of the Life-Tech electrode to the tympanic membrane. The second was the measurement of the impedance of the ear canal electrode.

The proximity of the electrode to the tympanic membrane was observed to have a direct effect on the morphology of the waveforms recorded and on the amplitudes of the responses obtained. For example, Subject 3 was initially tested with the ear canal electrode placed approximately 1 centimeter from the tympanic membrane. The amplitudes of the AP obtained with this electrode placement ranged from 0.08 to 0.18 microvolts, and the amplitude of the SP was too small to allow for the reliable identification of this above the noise present in these recordings. The same subject was retested on a second occasion with the ear canal electrode placed within 2-3 millimeters of the tympanic membrane. The waveforms recorded
with this electrode placement were much more robust, with AP amplitudes ranging from 0.12 to 0.23 microvolts. In addition, the SPs obtained when the ear canal electrode was placed closer to the tympanic membrane were sufficiently robust to be readily identified and quantified by both judges. Gibson (1978) demonstrated that the amplitude of the AP response varies inversely with the distance from the ear canal electrode to the tympanic membrane. The observations of this relationship between the distance from the electrode to the tympanic membrane and the amplitude of the responses obtained from Subject 3 in the present study were consistent with Gibson's findings.

The second methodological concern was related to the measurement of the impedance of the ear canal electrode. The impedance of the ear canal electrode was measured by the Nomad evoked potential unit by applying a small alternating voltage across the electrodes and measuring the resultant current. The impedance measurement was problematic for Subject 11 in this investigation as she experienced a painful electrical shock during the measurement of the ear canal electrode impedance. The subject ordered the otolaryngologist to remove the electrode immediately, and was emotionally upset by this experience. The testing was discontinued and this subject was excluded from the subject pool for the study. The otolaryngologist reported that a small reddened area typical of the mark left by a burn had appeared at the electrode site.
During the exit interview, this subject reported a "painful sensation" in the ipsilateral ear and side of the face which persisted for several hours after the shock had occurred.

Four possible factors may have contributed to produce this electrical shock during the testing of electrical impedances:

1. The subject's ear canal had been irrigated with alcohol and peroxide immediately prior to the impedance test, and had not completely dried,

2. The electrode leads connecting the electrodes to the preamplifier had been closely braided, possibly increasing the voltage in the lead to the ear canal via induction,

3. The impedance testing hardware and software in the evoked potential unit may have temporarily caused an excessively high voltage to be applied across the electrodes.

Before testing the next subject, the examiner applied the electrodes to his forearm and could not detect any sensation during the testing of the electrode impedances. For all subjects tested after this incident, the electrode leads were left unbraided until the impedance testing had been completed and their ear canals were thoroughly dried prior to the testing of electrode impedances. There were no other unpleasant experiences reported by any subjects.

The author is aware of one other incident in which an
ECochG subject received an electrical shock during the impedance testing procedure. In this case, a direct current voltage was applied in order to measure the resistance of the electrode in the ear canal. The subject reportedly lost consciousness for a very short period as a result of the electrical shock that was applied to the ear canal during the impedance measurement.

These two incidents raise obvious concerns about the safety and clinical utility of electrode impedance measurement and about the necessity of the procedure. The measurement of the impedance of the electrodes prior to testing is considered a necessary and routine protocol for auditory evoked response testing. These impedance measurements are taken in order to ensure that the impedances fall below critical values and are matched across electrode sites, thus increasing the likelihood of obtaining robust recordings. During the testing of an individual subject, if the impedance for the ear canal electrode is found to be unacceptably high, the electrode is then relocated to find a site which would provide a lower impedance. If this is not possible, the ear canal may be cleansed a second time and the electrode would then be reinserted. During these procedures, the impedance of the ear canal electrode is repeatedly measured and the subject is exposed to some risk of receiving an electrical shock.

An alternative approach for this measurement is to begin testing immediately after the insertion of the ear canal
electrode, without measuring the impedance at this site. If acceptable recordings could be initially obtained, that is, the potentials of interest (AP and/or SP) are readily identifiable, then the testing could proceed without knowing the specific impedance values for the ear canal electrode. If the initial recordings were unsatisfactory, then the electrode could be relocated and the electrode impedance could be measured at the new site. This modified procedure would reduce the risk of unintentionally applying an electrical shock to the subject participating in electrocochleography studies. The time required to perform the testing would be increased in cases where the electrode impedance was questionable, but decreased in cases where the initial recordings were robust.
Implications of the Research Findings

The results of this investigation indicate the need for alternative experimental designs employing ECochG with human subjects in order to further our understanding of the cochlear transduction mechanism. In addition, as these results demonstrate no change in the AP or SP amplitudes or in the SP/AP ratio in response to stimulation of the contralateral ear, they support the validity of clinical applications of ECochG in the differential diagnosis of Meniere's disease.

Cochlear transduction

The lack of interaction observed between the masked and unmasked trials does not imply that the efferent system has no effect on the processing of sound by the afferent auditory system. Rather, the hypothesized effects were not demonstrable within the experimental design of this investigation. The intensity, center frequency, bandwidth and temporal characteristics of the contralateral masker or ipsilateral stimulus may have been inappropriately selected. It is also possible that such effects cannot be measured by far field recording techniques. The suppression of the AP response to a one-third octave click stimulus may indeed occur in some individual auditory nerve fibers in response to an appropriate contralateral masker, and, as a result of this suppression, may be demonstrable in single unit recordings. However, as this effect may not occur in the majority of the fibers that are stimulated by the filtered clicks, they may
not be observed in the summated far field recordings. The same argument can be applied to the SP response. The SP may be enhanced in single unit activity, but it may not be possible to observe any of the enhancement in summated far field recordings. Thus, the inability to demonstrate these effects in the present investigation indicates the need to devise alternative experimental designs to explore the interaction of the efferent and afferent auditory systems in humans.

Clinical Implications

The findings of this study may have implications regarding clinical measurements of the SP and AP, and subsequent calculation of the SP/AP ratio when they are used in the differential diagnosis of Meniere's disease. High stimulus intensities (100 dB HL) are commonly used during such testing in order to evoke a robust waveform in which the SP may be easily identified. Crossover of the acoustic stimulus can be expected to occur at these high presentation levels (Goldstein and Newman, 1985). Crossover is defined as "a situation in which sound presented to one ear is transmitted to the opposite ear, usually by vibration of the skull" (Katz, 1985, p. 1067). At a presentation level of 100 dB HL, the crossover of the click stimulus will likely stimulate the contralateral cochlea at an intensity comparable to the stimuli employed in the present study. In the present study, the presence of contralateral masking tone had no effect on
the ipsilateral AP and SP amplitudes or on the SP/AP ratios. Thus, the validity of the differential diagnostic tests based on the measurement of the AP and SP may be supported by findings of the present study. That is, the crossover of the ipsilateral stimulus and possible stimulation of the efferent auditory system would not be expected to have any significant effect on the responses measured in electrocochleography. However, the effects of the efferent system on the ECochG responses may be more pronounced at the higher ipsilateral presentation levels, particularly at the levels which are employed clinically. Therefore, the effects of the interaction between the efferent system and the afferent system during clinical electrophysiological testing remains unknown.

Directions for Future Research

The immediate direction for future research projects is to resolve the different findings reported by Folsom and Owsley (1987) and the results obtained from the present investigation. In addition, future research questions should address the inhibitory effects of the contralateral masking on the summating and action potentials by systematically varying the stimulus and measurement variables.

Several improvements could be made in the measurement techniques employed in the present study in order to improve the quality of the recordings. The amplitude of the ECochG recordings could be greatly increased by employing tympanic
membrane electrodes (TM electrodes). These electrodes are known to double the amplitude of the recorded AP (Stypulkowski and Staller, 1987). The amount of stimulus artifact present in the recordings could be reduced by delivering the acoustic signals via shielded earphones which are designed to limit the amount of radiated magnetic flux. Alternatively, insert earphones could be used to isolate the recording electrodes from the magnetic flux of the transducer. Finally, the ipsilateral stimulus should be generated and amplified by equipment that does not introduce any audible equivalent input noise. The behavioral thresholds for the filtered click stimulus could then be measured more accurately and the presentation levels for the ipsilateral stimulus could be more precisely controlled.

In order to define the optimal masker, future investigations could systematically address the intensity, frequency, bandwidth and temporal characteristics of the contralateral masking signal. There may be differences in the optimal maskers for the AP and SP. Therefore, the effect of contralateral masking on these two potentials should be addressed separately and in relation to one another.

Finally, the effects of the efferent system on the cochlear microphonic (CM) component of the response should be explored. Electrical stimulation of the crossed olivocochlear bundle has been reported to cause an increase in the CM in animal subjects (Dolan and Nuttall, 1988). This cochlear
response can be recorded at low presentation levels, and therefore would be suitable for a study of the effects of acoustical stimulation of the crossed OCB in humans. The CM can be isolated by subtracting one waveform from another waveform which has been recorded with reversed stimulus and electrode polarities (Glatke, 1983). Still, the elimination of any stimulus artifact would be crucial during the recordings of the CM, because the two responses are in the same phase and this artifact cannot be eliminated by the techniques employed to isolate the CM in a composite waveform.
Conclusion

This study examined the effects of the efferent auditory system on the evoked response as measured by ECochG techniques. The amplitudes of the action potential and the summating potential as well as the SP/AP ratio were compared across two conditions:

1. the control condition, ipsilateral stimulation without contralateral masking, and
2. the experimental condition, ipsilateral stimulation with contralateral masking.

The amplitudes of the AP and SP responses, as well as the SP/AP ratios, did not change in any systematic manner during the application of the contralateral masking. This evidence failed to support the findings of Folsom and Owsley (1987) which demonstrated an inhibitory effect of contralateral masking on the action potential. The present results indicate the need for further research into the effects of contralateral masking on the AP in order to resolve the discrepancies between the two studies. In addition, the results suggest that the interaction of the efferent and afferent auditory systems in human subjects should be explored using different experimental designs and methodologies. Finally, the results of the current study lend support to the validity of the clinical use of ECochG in the differential diagnosis of Meniere’s disease by demonstrating that stimulation of the contralateral ear at moderate intensities
may not affect the amplitudes of the action potential or the summating potential or the magnitude of the SP/AP ratio.
REFERENCES


Warr, B. W. (1975). Efferent olivocochlear and vestibular efferent neurons of the feline brainstem: Their location, morphology and number, determined by retrograde axonal transport and acetyl cholinesterase histochemistry. *Journal of Comparative Neurology*, 161, 159-181.


APPENDIX A

Definitions of Terms

**Action Potential (AP):** A transient whole-nerve response to acoustical stimulation which originates from the spiral ganglion of the auditory branch of the VIII cranial nerve. The AP is one of the responses measured by ECochG. It is synonymous with wave I as measured by ABR.

**Afferent Auditory System:** The ascending pathways which transfer neural impulses from the cochlea via nuclei in the brainstem to the auditory cortex in the temporal lobe of the cerebrum.

**Auditory Brainstem Response (ABR):** "Fast" evoked potentials (including waves I-V) occurring within 10 msec of the stimulus which originate from the auditory nerve and brainstem nuclei in response to acoustic stimulation.

**Cochlear Microphonic (CM):** An alternating current stimulus-related electrical potential generated by the hair cells that closely follows the stimulus waveform. The CM is one of the responses measured by ECochG.

**Efferent Auditory System:** The descending neural pathways which transfer neural impulses from the auditory cortex and brainstem nuclei to the cochlea.

**Electrocochleography (ECochG):** "First" evoked potentials (including the SP, CM and AP) occurring within 3 msec of the stimulus which originate from the hair cells in the organ of Corti and from the auditory nerve in response to acoustic stimulation.

**Evoked Potentials/Responses (EP):** Recordings of the summated far field electrical activity of the various components of the auditory system (hair cells, neural dendrites and axons in the auditory nerve and brainstem nuclei) in response to acoustic stimulation.

**Latency:** The elapsed time (in milliseconds) from the stimulus onset to the measured response.

**Summating Potential (SP):** A direct current stimulus-related electrical potential generated by the hair cells that has a linear grade with respect to the stimulus waveform, but does not follow the phase changes of the stimulus. The SP is one of the responses measured by ECochG.

**SP/AP Ratio:** The ratio of the summating potential to the action potential. The value of this ratio is critical in the clinical differential diagnosis of Meniere's disease.
APPENDIX B

Screening Questionnaire

University of Montana
Department of Communication Sciences and Disorders

Screening Questionnaire
(To be given orally)

Subject Number: ___________________________ Date: ____________

PHYSICAL HEALTH

1. Do you now have a cold? Yes ______: No ______.

2. Have you been physically ill in the past 6 months? Yes ______: No ______. If yes, please explain:

3. How would you describe your recent health?
   Excellent ______: Good ______: Fair ______: Poor ______.

4. Are you now taking any medication? Yes ______: No ______. If yes, please indicate the type(s) of medication, and the length of time on the medication:

5. Have you ever had any disease or accident that you feel might affect your neurologic function? Yes ______: No ______ If yes, please describe:

HEARING STATUS

1. Do you believe you have previously had or do you now have a hearing problem? Yes ______: No ______.
   If yes, please explain:

2. Have you ever been medically treated for an ear problem? Yes ______: No ______
   If yes, please provide dates and explanations:

3. Is there any history of hearing loss in your family? Yes ______: No ______. If yes, please give the relationship of the person to you and describe the hearing problem:
APPENDIX C

UNIVERSITY OF MONTANA
DEPARTMENT OF COMMUNICATION SCIENCES AND DISORDERS

Title of Project: Effects of Contralateral Masking on the Cochlear Potentials in Humans

Principal Investigator: Michael K. Wynne, Ph.D., Assistant Professor
Department of Communication Sciences and Disorders
(243-4131)

STATEMENT OF CONSENT

The purpose of this investigation is to study the effects of the central nervous system on hearing in adults. The results of the study should further our understanding of the hearing mechanism. Subjects who participate in this study will benefit by receiving information about their hearing sensitivity.

I hereby agree to participate in tests of hearing as measured by the methods described below (see check marks). I understand that prior to hearing testing I will be asked questions regarding my health and hearing. I may review the questions in advance if I wish. This portion of the session will take approximately 2 minutes.

1. Evoked Potential Test. This method measures the brain's responses to sound. Recording discs will be attached to my scalp and earlobe with adhesive tape. My ear canals will be cleaned by an experienced otolaryngologist (ear, nose and throat specialist). Surface recording electrodes will then be placed within my ear canals. I will be positioned comfortably in a chair or on a cot and sounds will be presented to me through earphones. Though the level of the sounds will vary, the sounds will never be painful or harmful to my hearing. I will be asked to remain as still as possible, to relax, and even to sleep, if I am able. The test will take about 40 minutes.

2. Audicmetric Test. This method is a traditional hearing test to see how well I can hear very soft sounds. I will be tested in a sound-proof room. Sounds of varying loudness will be presented through earphones. Each time I hear a sound I will respond by raising my hand or pushing a button. When words are presented, I will repeat them back. This test will take about 20 minutes.

3. Tympanometry Test. This method measures the change in air pressure in the ear canals as sounds of varying loudness are presented to each ear. The pressure changes reflect the condition of the ear structures. No active response will be required of me. An earphone will be placed over one ear and a small, sterile rubber-covered tip will be painlessly placed in the opening of the canal of the other ear. This test will last approximately 5 to 10 minutes.

I understand that I may refuse to participate or to withdraw from the study at any time without penalty or loss of the benefits to which I would otherwise be entitled. No information which identifies me by name will be released without my permission. The Principal Investigator will have access to the study data which will be retained for three years. I have had the opportunity to ask questions and understand that any future questions that I have about the research or about subjects' rights will be answered by the investigator listed above.

I understand that these tests are routine, clinical procedures which are safe, but nonetheless in the event that I am physically injured as a result of this research I will individually seek appropriate medical treatment. If the injury is caused by the negligence of the University or any of its employees you may be entitled to reimbursement or compensation pursuant to the Comprehensive State Insurance Plan established by the Department of Administration under the authority of M.C.A., Title 2, Chapter 9. In the event of a claim for such physical injury, further information may be obtained from University Legal Council.

Signature of Subject ________________________________

Signature of Investigator ____________________________

Date ____________________

Copies to: Subject
Investigator
APPENDIX D

Subject Information Form

SUBJECT INFORMATION SHEET (WIGHTMAN--EFFERENT SYSTEM)

Subject Code: Examiner: Wightman Date: ____________

File Name: Amplification: Filter: 20 - 3000 Hz

Electrode Impedence Pre: ____________ Post: ____________

EXPERIMENTAL CONDITIONS

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Appendix E

Table E-1.

Action potential amplitudes (in microvolts) for repeated trials in the control (unmasked) and experimental (masked) conditions.

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Table E-2.

Summating potential amplitudes (in microvolts) for repeated trials in the control (unmasked) and experimental (masked) conditions.

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Table E-3.

SP/AP ratios for repeated trials in the control (unmasked) and experimental (masked) conditions.

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Table E-4.

Action potential latencies (in milliseconds) for repeated trials in the control (unmasked) and experimental (masked) conditions.

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