THE EFFECT OF SMOKELESS TOBACCO INGESTION ON THE MOTOR DISTAL LATENCY OF THE MEDIAN NERVE

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Tobacco in all of its various forms has an elaborate history dating back to its early beginnings nearly five centuries ago. Most of the tobacco used worldwide is smoked. Recently, the practice of chewing tobacco has emerged as a popular habit in the United States. "Snuff dipping" is currently a popular manner of tobacco use in this country (Rohrer and Young, 1982).

Smokeless tobacco refers to tobacco which is designed to be chewed or sniffed rather than smoked. Dipping is the process of placing a pinch of tobacco (snuff) between the lower lip and teeth, where it stimulates the flow of saliva and mixes with it (American Cancer Society, 1981).

Smokeless tobacco is currently being heavily promoted through mass media advertising using well-known sports figures and entertainers to act as spokespersons. The sales of smokeless tobacco have increased about 11% each year since 1974, with an estimated 22 million users in the United States (Christen, 1980). Recent surveys have shown that many males in elementary schools, high schools, and colleges are adopting these habits. In one study of Texan collegiate athletes, up to one third of the varsity football and baseball team members were using some form of smokeless tobacco (Christen et al., 1979). Marty and associates (1985) reported that 15 to 20% of high school pupils in some

parts of the United States regularly use smokeless tobacco. In a survey conducted in Arkansas, it was reported that 18.9% of the total population used smokeless tobacco, with the majority of users being males (Marty et al., 1985). Winn and associates (1981) reported that smokeless tobacco use among women in the southeastern United States represented a substantial risk for the development of oral and pharyngeal cancer. In a recent study in Oklahoma it was reported that 13% of third grade males regularly use smokeless tobacco (Glover and Edwards, 1985).

Researchers disagree on the possible harmful effects of smokeless tobacco use. Russell and co-workers (1980) have suggested that smokeless tobacco may be a less harmful substitute for cigarette smoking. Other researchers elucidate the incidence of oral cancers with smokeless tobacco use (Christen et al., 1979; Waldron and Shafer, 1974; World Health Organization, 1978).

Reasons for smokeless tobacco use vary considerably, ranging from sociability to psychological and physiological effects obtained. Many athletes who use smokeless tobacco while performing claim that it improves their reaction time, heightens their ability to concentrate for extended periods of time, and generally improves their overall performance (Glover et al., 1984). Additionally, some football and baseball players claim that smokeless tobacco use keeps their mouths from drying out excessively while they are engaged in their sport (Christen, 1980).

Regardless of the varied reasons attributed to why smokeless tobacco is used, there appears to be a void in the literature related to the electrophysiological effects of smokeless tobacco.

Electrophysiological studies involve the study of the chemical and electrical mechanisms that occur in biological organisms. Studies measuring reaction time would be an example of this type of research.

Reaction time is defined as the period of time that elapses between the presentation of a stimulus to the initiation of a response (Sage, 1971). Reaction time can be further broken down into four components: sense organ time, brain time, nerve time, and muscle time (Drowatzky, 1975). Nerve and muscle time can be measured through the use of electromyography and nerve conduction instrumentation. Nerve time involves the time period that is necessary for the electrical impulse to transverse the length of a nerve. Motor nerve conduction velocity measurements involve stimulating a nerve at one point and recording the response either at the muscle or at some distance along the nerve (Goodgold and Eberstein, 1972). Motor nerve distal latency measurements consist of stimulating a nerve near its terminal end and are usually expressed in time values indicating when a response is observed. The units of this measurement are usually expressed in milliseconds. These units are used to indicate the importance of the residual latency values that are obtained in this region of the nerve. The nerve conduction velocity is considerably slower in this region of the nerve and is attributed to two major factors: 1) the decreasing size of the nerve fiber, and 2) a delay in conduction at the neuromuscular junction (Johnson, 1980).

A limited number of research studies on smokeless tobacco exist in the professional literature, due probably to the relative obscurity of smokeless tobacco in the past century. Most of the research that has measured the electrophysiological effects of tobacco in humans has

been obtained in cigarette smoking studies. However, with the recent mass marketing techniques of the manufacturers, the increasing use of smokeless tobacco products, and the controversies involving smokeless tobacco use, further research is needed to determine the full effects of smokeless tobacco use.

Statement of the Problem

The purpose of this study was to compare the median nerve motor distal latency differences in a group of subjects who regular used smokeless tobacco, before and after ingesting smokeless tobacco in two different dosages and a placebo chewing gum.

Significance of the Study

This study is significant in that it will contribute to the body of knowledge regarding the electrophysiological effects of smokeless tobacco. This study could also make a contribution to the health education community, both in the private and public sectors. It is further hoped that the results of this investigation will provide students, athletes, coaches, and educators with additional knowledge concerning smokeless tobacco use so that they may make more informed decisions concerning its use.

Hypotheses

The hypotheses that were tested in this research were for the purpose of investigating the differences between the median nerve pretreatment motor distal latency values and the 15 posttreatment

motor distal latency values of smokeless tobacco users during three different treatment conditions. The following null hypothesis were tested at the .05 level of significance:

1. There is no significant difference between the pretreatment median nerve motor distal latentices and the 15 posttreatment median nerve motor distal latencies of those subjects who ingest the placebo chewing gum.

2. There is no significant difference between the pretreatment median nerve motor distal latencies and the 15 posttreatment median nerve motor distal latencies of those subjects who ingest one pouch of "Skoal Bandits" smokeless tobacco.

3. There is no significant difference between the pretreatment median nerve motor distal latencies and the 15 posttreatment median nerve motor distal latencies of those subjects who ingest their regular smokeless tobacco dip.

4. There are no significant differences in the 15 posttreatment median nerve motor distal latencies of the subjects after receiving each of the three treatment conditions.

Limitations of the Study

This investigation was limited in the following ways:

1. The results and conclusions are limited to the population of college-aged students having similar characteristics as the sample that was utilized in the study.

2. No attempt was made to control interest or motivation of the subjects. Participation in the study was on a voluntary basis.

Delimitations of the Study

The study was restricted in the following ways:

1. Only male subjects participated in the study.

2. Subjects who exhibited an abnormally long (greater than 4.5 milliseconds) median nerve motor distal latency value in their pre-treatment test were eliminated from the study. This could indicate some type of median nerve pathological condition.

Assumptions of the Study

In the investigation, the following assumptions were made:

1. It was assumed that there was no significant differences in the diurnal variations of the distal latencies of the subjects.

2. The order of treatments received by the subjects was counterbalanced, so it was assumed that no treatment order effects existed.

3. It was assumed that the standardized smokeless tobacco dose ("Skoal Bandits") represented a smaller dose of nicotine than the subject's regular tobacco dip.

4. It was assumed that the subjects refrained from any tobacco use and strenuous physical activity during the two hour time period prior to each of the testing periods.

Definitions of Terms

Acetylcholine. "A neurotransmitter at the myoneural junction" (Kruk and Pycock, 1979, p. 22).

Agonist. "A drug that interacts with a receptor to produce

the normal physiological effect" (Leavitt, 1982, p. 25).

Anastomosis. "An end-to-end union, joining together, or intercommunication of parts of any network or set of fibers such as nerves" (Thomas, 1973, p. A-73).

Antagonist/Blocker. "A drug which acts upon receptors and blocks the normal physiological process" (Leavitt, 1982, p. 26).

Axon. A process of a neuron that typically conducts nerve impulses away from the cell body; however, axons can transmit impulses in either direction (Sage, 1971, p. 32).

Buccal Mucosa. The surface (innermost) layer of tissue of the mouth in the region of the cheek and gum (Wilson and Wilson, 1983, p. 311).

Cholinergic Nerve. "Nerves that release acetylcholine at their terminal endings" (Levitt, 1981, p. 65).

"Copenhagen". Snuff produced by the United States Tobacco Company (Nashville, TN 37203).

Drug Affinity. "A measure of the attraction of a drug for it's target site" (Kruk and Pycock, 1979, p. 17).

Electromyography. The process of recording the electrical activity that is occurring in a muscle by placing electrodes over the muscle (deVries, 1980, p. 61).

Hertz. Cycles per second.

Ion. "An atom or group of atoms bearing a negative or positive electric charge" (Scott, 1975, p. 34).

Median Nerve. "A nerve in the arm that sends a motor branch to the superficial head of the flexor pollicis brevis, the abductor

pollicis brevis,and opponens pollicis muscles of the thumb" (Johnson, 1980, p. 31).

Motor Nerve Distal Latency. The period of time required for an electrical signal to travel down the terminal branch of a nerve (Johnson, 1980, p. 20). In this study, it involved the median nerve and a measured distance of 7 centimeters.

Myelin. "A fatlike substance forming the principal component of the myelin sheath of nerve fibers" (Thomas, 1973, p. M-77).

Neuromuscular (Myoneural) Junction. The point where a nerve joins a muscle whose function is to ". . . transfer impulses from the very small motor nerve endings to the much larger muscle fibers" (MacLean, 1980, p. 73).

Neurotransmitter. "A chemical that transmits neural information across a synapse" (Levitt, 1981, p. 58).

Nictoine. "The major chemical compound present in tobacco products that is generally agreed to be responsible for the behavioral use of tobacco products" (Dunn, 1978, p. 19).

Nicotinic Receptor. "A receptor in which nicotine can mimic the effects of acetylcholine" (Levitt, 1981, p. 68).

Receptor. "A highly specific glycoprotein cell component on a postsynaptic membrane" (Leavitt, 1982, p. 25).

"Regular Skoal". Wintergreen flavored powdered tobacco produced by the United States Tobacco Company (Nashville, TN 37203).

"Skoal Bandits". Small, teabag-like pouches of wintergreen flavored tobacco produced by the United States Tobacco Company (Nashville, TN 37203).

Smokeless Tobacco. "Tobacco that is designed to be chewed or

sniffed rather than smoked" (Glover et al., 1984, p. 22).

Snuff. Finely ground (powdered) tobacco.

Snuff Dipping. "The process of placing a pinch of powdered tobacco between the cheek and gum" (Glover, Christen, and Henderson, 1982, p. 2).

Sweep. The horizontal (x-axis) linear time axis of a cathode ray tube display generated by the left-to-right movement of the trace spot at constant preselected speeds across the face of the cathode ray tube display (Johnson, 1980, p. 433).

Synapse. "The gap between a nerve terminal and another tissue" (Kruk and Pycock, 1979, p. 10).

TECA TE42. The electromyography and nerve conduction instrumentation used in this study (Teca Corporation, Three Campus Drive, Pleasantville, New York 10570).

CHAPTER II

REVIEW OF THE LITERATURE

Literature Related to the Problem

Investigators planning to conduct research studies that involve the electrophysiological effects of tobacco and nicotine should be knowledgeable about the various actions of nicotine on the particular biological system concerned. It is essential that certain understandings about the nervous system be acquired. Similarly, it is important to recognize and understand the characteristic actions of nicotine on the nervous system. In addition, it is essential that the investigator develops a thorough background in the instrumentation that is required for the collection of the information needed in these types of studies. The researcher must become competent in the procedures and techniques required in order that accurate measurements can be obtained.

It is, therefore, requisite that the literature review should address all of these issues which will be associated with the findings so that accurate interpretation of the results is possible. The first section of this chapter addresses the electrophysiological characteristics of the nervous system, particularly the neuromuscular junction. A second portion deals with the characteristics of tobacco and nicotine, and reviews the existing literature in smoking and

smokeless tobacco studies. A third section concerns the instrumentation, data collection, and data handling procedures and methodologies.

Electrophysiological Characteristics of Neural Transmission

The human nervous system is often anatomically divided into two major systems: the central nervous system, which consists of the brain and spinal cord, and the peripheral nervous system, which includes 12 pairs of cranial and 31 pairs of spinal nerves, in addition to the peripheral parts of the autonomic nervous system (Sage, 1971). It is also commonly divided into two divisions according to the functions performed: the voluntary and involuntary nervous systems. The portion that supplies the skeletal muscles used in movement is called the voluntary, or somatic, nervous system. The portion that supplies the internal organs and associated structures is known as the involuntary, or visceral, nervous system (Levitt, 1981). The reader should also realize that there are overlaps within these functional classifications.

The structural and functional unit of the nervous system is the neuron. In general, nerve fibers are classified into afferent or efferent fibers. Afferent fibers conduct impulses toward the central nervous system and are referred to as sensory neurons. Efferent fibers conduct impulses away from the central nervous system and are called motor neurons (Sage, 1971). Normally a nerve fiber transmits impulses in only one direction. For motor nerves, this is towards the muscles. However, nerve fibers are also able to conduct impulses in the

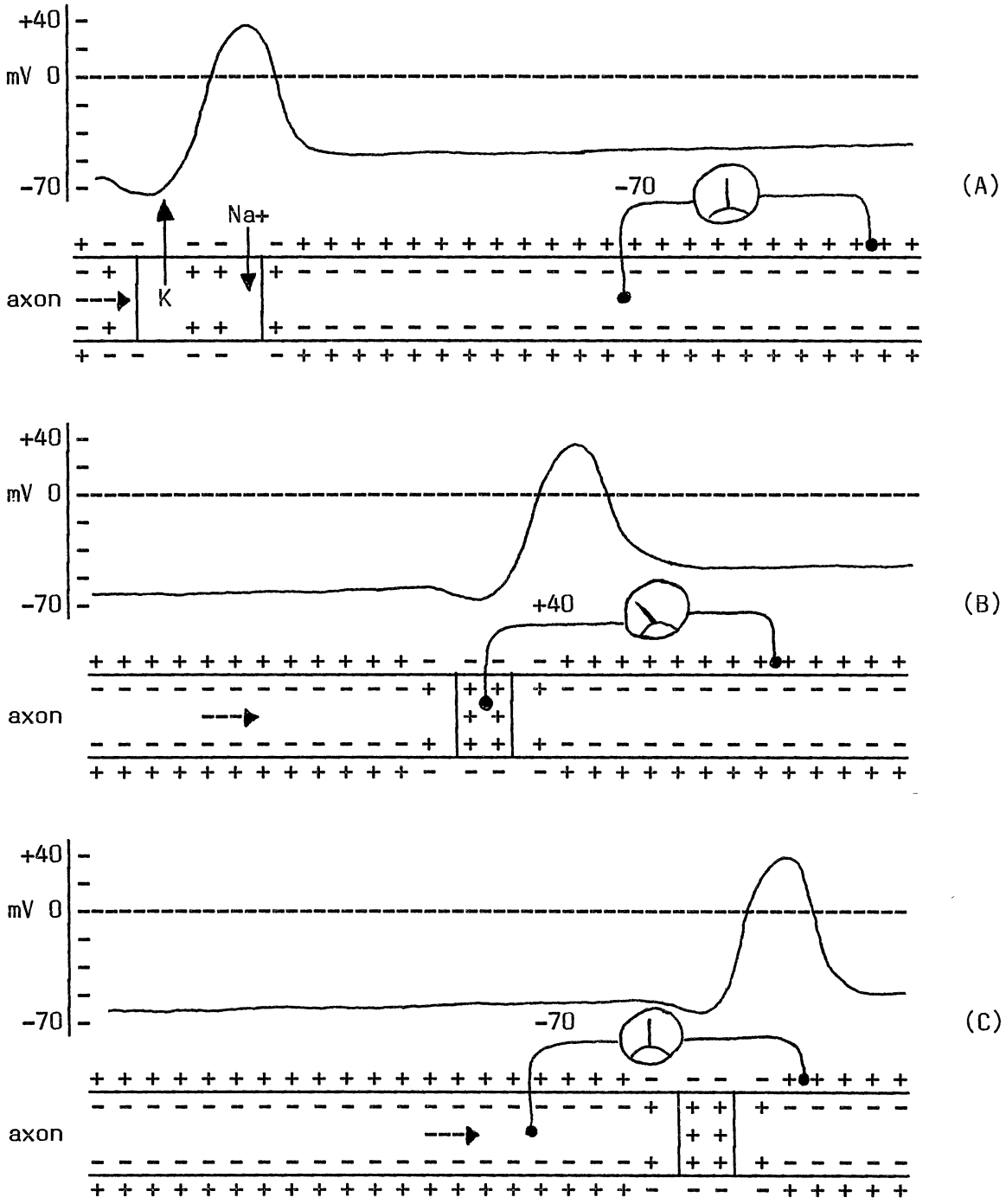
direction opposite to what occurs normally, but the functional characteristics at the synapses generally allow signal transmission to occur in only one direction (Ottoson, 1983).

A nerve is a collection, or bundle, of nerve fibers bound together by a connective tissue sheath. Nearly all nerves are mixed nerves, which indicates that they contain both afferent and efferent fibers (Sage, 1971).

The function of the neuron is to carry nerve impulses. The nerve impulse can be thought of as an electric current being conducted (Sage, 1971). This current is the result of changing concentrations of ions inside and outside of the nerve cell membrane. The polarity of the nerve axon is maintained by a differential balance of ions on the inside and outside of the membrane. When a neuron is at rest, the cytoplasm inside the axon contains a large number of protein molecules along with some chloride ions, and a low concentration of sodium ions. The combined charge on the inside of the membrane is negative. The tissue fluid bathing the outside of the membrane is rich in sodium, having a sodium concentration size of approximately ten times greater than that existing on the inside of the axon. The resulting charge outside the membrane is positive. The membrane voltage has been measured between -70 and -90 mV, and produces a strong pressure for an inflow of the sodium ions. However, the resting membrane is impermeable to sodium ions, effectively blocking the flow (Sage, 1971). When the neuron is stimulated to a sufficient degree, the membrane suddenly becomes permeable to sodium, which enters the axon at a rapid rate. This influx of sodium ions reverses the internal potential of the membrane from about -70 mV to +40 mV. The outside of the

membrane becomes negative in relation to the inside. This process ignites the nerve impulse (Sage, 1971). The membrane potential then immediately returns to its resting value. This sequence of potential changes is called the action potential (Schriber, 1975). This action potential propagates, section by section, along the whole length of the axon until it reaches its terminal ends (Figure 1). The nerve endings of the axon interface with other neurons or other cells, such as a muscle fiber. The junction of one neuron to another is called a synapse (Bickerton and Small, 1982). A synapse represents a structural discontinuity in the system and is characterized by special properties. The connection is not a physical one as there exists a physical gap, called the synaptic gap or cleft, that separates the two connections. It is at the synapses that the action potential is transmitted chemically to the next element in the neural sequence (Levitt, 1981). The chemical synapse has been described as a "one-way valve," since it allows transmission in only one direction (Ottoson, 1983). There is also a delay in the transmission at the synapse that is attributed to the chemical processes involved (Ottoson, 1983).

The action potential results in changes in the presynaptic membrane of the axon. These changes initiate the release of a chemical from the neuron into the synaptic cleft. Release of chemicals in response to activation of a nerve is termed neurochemical transmission. The chemical that is released is called a neuro-transmitter, since the chemical transmits neural information (Kruk and Pycock, 1979). This neurotransmitter then diffuses across the synaptic cleft and binds with receptor molecules in the postsynaptic membrane. Receptors are discrete regions of the postsynaptic membranes



Source: George H. Sage, Introduction to Motor Behavior: A Neuropsychological Approach (1971).

Figure 1. Propagation of an Action Potential in a Neuron. The arrows show the direction of transmission

which selectively bind molecules of a specific structure (Kruk and Pycock, 1979). The interaction of a neurotransmitter with its receptor initiates a response within the postsynaptic structure (Levitt, 1981). There is some question as to the exact nature of the receptor activation process. The occupation theory suggests that the response is initiated by the process of occupation of a receptor, whereas the rate theory hypothesizes that the response is initiated by the whole process of linking onto and the detachment from the receptor (Kruk and Pycock, 1979). Regardless of which theory is valid, the combination of neurotransmitter with its receptor must be quickly reversed between each impulse, leaving the receptor free to combine with the next packet of neurotransmitter produced (Ashton and Stepney, 1982). Following release, a neurotransmitter would continue to stimulate receptors if mechanisms for terminating its action did not exist. Enzymatic inactivation and neuronal re-uptake are the two major mechanisms that have been identified (Kruk and Pycock, 1979).

Nerves are generally classified according to the neurotransmitter that they release. Those that release acetylcholine are called cholinergic fibers (Levitt, 1981). Acetylcholine was the first chemical to be identified and proven as a neurotransmitter. Acetylcholine is synthesized and then stored in the end terminals of the neuron in subcellular structures called vesicles. It is a widely held view that the acetylcholine stored within the vesicles is released in a quantal manner in response to an action potential arriving at the nerve terminal (Kruk and Pycock, 1979). Kruk and Pycock (1979) also suggest that this hypothesis is tentative. It has also been discovered that approximately half of the acetylcholine

content of the nerve terminal is floating freely in the cytoplasm of the terminal (Dunant and Israel, 1985). Recently, Dunant and Israel (1985) found, in their studies of the electric fish Torpedo, that the source of acetylcholine emitted by a neuron is the cytoplasm rather than the synaptic vesicles.

Motor nerve fibers and their endings are known to contain acetylcholine and the enzymes needed to synthesize it. The postsynaptic membrane area also contains the enzyme, acetylcholinesterase, which breaks down acetylcholine through hydrolysis (Katz, 1966).

Two major categories of cholinergic receptors have been formed based on the actions of two drugs of plant origin which were found to mimic the effects of stimulating specific nerves: muscarine and nicotinic receptors. Most cholinergic receptors in the nervous system and at the neuromuscular junction are nicotinic receptors. Nicotinic receptors show different sensitivities to agonists and receptor blockers, which seems to indicate that more than one type of nicotinic receptor may exist (Kruk and Pycock, 1979).

Neuromuscular Junction

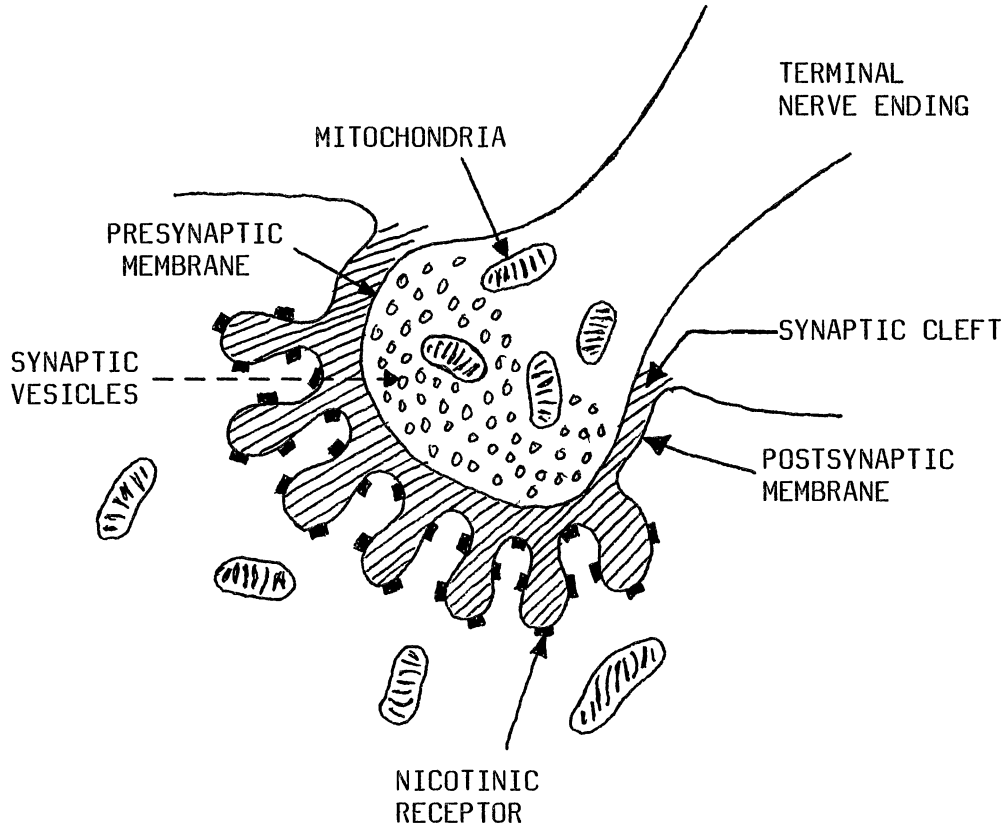
The point at which motor neurons interface with muscle fibers is called the neuromuscular, or myoneural, junction. This is the site at which information is transformed from an electrochemical impulse along the neuron into the mechanical contraction of a muscle fiber (Levitt, 1981). A motor unit is composed of a motor neuron and all the muscle fibers that the neuron innervates (MacLean, 1980).

The structure of the junctional apparatus includes a nerve

terminal (presynaptic membrane), a motor endplate (postsynaptic membrane), and a synaptic cleft that separates the two (Figure 2). The motor endplate is a structural enlargement of the muscle fiber that forms a trough, into which fits the terminal nerve ending. The endplate has many infoldings resembling little mounds and gutters that are continuous with the muscle sarcolemma. Because of these many infoldings, the motor endplate has a much larger surface area than the nerve terminal it envelops (MacLean, 1980).

The nerve terminal is an unmyelinated extension of the axon and is rich in mitochondria and numerous spherical synaptic vesicles which contain acetylcholine. The postsynaptic membrane contains highly specialized protein receptor molecules that recognize and bind the acetylcholine (Ottoson, 1983). The synaptic cleft between the two membranes is approximately 20 nanometers (Korenyi-Both, 1983). This synaptic cleft is too wide to allow the currents underlying the nerve impulse to generate impulses in the muscle fibers electrically. Since the diameters of the nerve terminals are much smaller than the connecting muscle fibers, the current produced by the nerve terminal would be insufficient to initiate impulses in the underlying muscle membrane. A delay of 0.5 to 0.8 milliseconds occurs between the arrival of a nerve impulse and the onset of a muscle action potential and is inconsistent with the assumption of electrical transmission (Ottoson, 1983).

The neurotransmitter role of acetylcholine has been known since the mid 1920s and has been well established as the neurotransmitter at the neuromuscular junction (Levitt, 1981). Acetylcholine is synthesized chiefly in the motor nerve terminals and floats



Source: Ian C. MacLean, "Neuromuscular Junction", Ernest W. Johnson (ed.), Practical Electromyography (1980).

Figure 2. The Neuromuscular Junction

freely within the cell cytoplasm as well as being stored within the synaptic vesicles. Spontaneous release of acetylcholine occurs in both quantal and nonquantal forms. The release of one or a few quanta gives rise to depolarizations of small amplitudes called miniature endplate potentials, or mepps, that remain localized to the region of the endplate. Conducted nerve impulses result in the release of large numbers of acetylcholine quanta which generate large endplate potentials, or epps, which trigger a complex sequence of events which initiates muscle contraction (Drachman, Pestronk, and Stanley, 1982).

A description of the major events that summarizes how the neural impulse is transmitted from the motor nerve to the muscle fiber is given by Ottoson (1983) below:

> When the nerve terminals are invaded by an impulse, calcium ions enter the endings and become attached, probably to the membranes of the vesicles. This appears to induce the vesicular membrane to fuse with the plasma membrane of the terminal. Finally, the fused membranes rupture and the acetylcholine content of the vesicles is emptied into the synaptic cleft. The acetylcholine so released diffuses through the cleft and reacts with specific glycoprotein receptors in the endplate membrane. Under the influence of acetylcholine, the receptor molecules change their conformation, and the channels which admit both sodium and potassium open. This results in a depolarization of the endplate and the generation of the endplate potential, which has all the characteristics of a local signal; by depolarizing adjacent regions of the muscle membrane, the endplate potential initiates the action potential of the muscle fiber, which travels from the endplate and induces the contractile response of the muscle fiber (p. 116).

Acetylcholine released at the neuromuscular junction acts postsynaptically on nicotinic receptors (Kruk and Pycock, 1979). The acetylcholine-receptor combination is broken down rapidly, within an interval of approximately one millisecond. This process must occur

rapidly, otherwise the endplate would not be able to respond to motor impulses arriving in close succession. A characteristic feature of peripheral nerves is that they are able to conduct impulses for a considerable time without becoming fatigued (Ottoson, 1983). Failure of a muscle fiber to generate an action potential could be attributed to either an inadequate release of acetylcholine or a decreased receptivity of the muscle fiber membrane (Levitt, 1981).

Anything that interferes with the interaction between acetylcholine and its receptors will block neuromuscular transmission. A number of different substances have been identified that will block neural transmission in the neuromuscular junction by binding with the acetylcholine-nicotinic receptor molecules, thus preventing the normal acetylcholine-receptor combination. Some known blockers include curare, tubocurarine, polymyoxin, neomycin, streptomycin, and kanamycin (Gilroy and Meyer, 1975; Katz, 1966; Kruk and Pycock, 1979; and Ottoson, 1983). Other drugs can have various effects at the neuromuscular junction using different mechanisms of action. Botulinum toxin prevents acetylcholine from being released from all cholinergic nerves. Black Widow spider venom is thought to cause an explosive release of acetylcholine from the cholinergic nerve fibers (Kruk and Pycock, 1979).

The synapse and the neuromuscular junction are very sensitive to asphyxia and ischemia, along with being very susceptible to a variety of drugs, such as those mentioned above. In general, the more synapses involved, the more susceptible is the transmission process to the influence of drug factors (Ottoson, 1983).

Literature Related to Tobacco

A vast amount of literature exists on tobacco use. Kozlowski (1984) reports an amusing Arabian story written by Bain in 1896 relating the mythical origin of tobacco and its continued use:

> ... the Prophet, Mahomet, rescued a snake from freezing by warming the snake against his body. The thankless snake bit him, but Mahomet sucked the venom from his wound and spat it upon the ground. On that spot, it was said, grew the first tobacco plant, combining the compassion of the prophet with the venom of the serpent (pages 311-312).

Kozlowski (1984) further states that

> The quest for less hazardous tobacco products has been directed toward reducing or eliminating the 'venom' of tobacco, while at the same time keeping its 'compassion' (p. 312).

Tobacco has been used for nearly 500 years in various forms. American Indians were apparently the first to use tobacco in all its various forms, which included smoking, chewing, and sniffing it through their nostrils (Christen et al., 1982). Tobacco apparently provides the user with both beneficial and adverse effects. Tobacco has been found to be physiologically and psychologically arousing, may enhance certain aspects of learning and memory, and may also serve as a relaxant. Some common acute adverse effects of tobacco include nausea, vomiting, and dizziness. The chronic use of tobacco is probably the more serious problem. The user can develop a tolerance to tobacco. There are also withdrawal symptoms associated with tobacco discontinuance, however, these are mild and never life-threatening.

Some of these symptoms may include drowsiness, nervousness, anxiety, headaches, and energy loss (Leavitt, 1982). Leavitt (1982) also notes that the tobacco habit may be the hardest of all drug habits to kick. Russell and co-workers (1980) observed that tobacco has been used in its various forms for four centuries, but noted that no population has been able to give up one form of tobacco without replacing it with another. They further indicated that the common factor is nicotine (Russell et al., 1980).

Nicotine

There have been many chemicals identified in cigarette smoke, but there is general agreement among researchers that nicotine is the pharmacological agent of prime importance accounting for tobacco use (Ashton and Stepney, 1982). Tobacco is the only natural source of nicotine (Russell et al., 1980). Nelson (1978) observes that nicotine has been described as a psychoactive agent possessing many different properties including tranquilizing, antianxiety, stimulating, depressing, anti-aggressive, mood-stabilizing, and stress-attenuating effects.

Nicotine is used primarily in individual and social settings and is placed in the classification of social drugs by Leavitt (1982). Nicotine is colorless, volatile, strongly alkaline, and considered a very powerful drug. Two or three drops of nicotine placed on the tongue would rapidly kill an adult, and the nicotine content of about 60 milligrams, that contained in one cigar, would be fatal to a human if injected intravenously (Russell, 1976). In fact, nicotine is found in some insecticides (Kruk and Pycock, 1979).

At the 7.4 pH level of the blood and at normal body temperature of 37° C., over 30% of the nicotine molecule exists in an un-ionized free form, which is extremely lipid soluble (Ashton and Stepney, 1982). The rate of absorption of free form nicotine is dependent on pH. At an acid pH very little nicotine is absorbed; however, as the pH becomes more basic, the amount of nicotine that is absorbed increases (Russell, 1976). Armitage and colleagues (1970) have shown that buccal absorption of nicotine is also pH-dependent. It appears that nicotine is actively secreted by the salivary glands. It is interesting to note that the pH of saliva shows a diurnal variation and ranges from 5.6 to 7.6. It increases very slightly after smoking and also changes before, during, and just after a meal. Depending upon the buccal fluctuations, some of the salivary nicotine would be reabsorbed through the buccal mucosa (Russell, 1976).

One of the important factors that influences the absorption rate of nicotine is the blood flow through the tissues at the site of administration (Leavitt, 1982). Once taken up into the blood flowing through the administration site, the nicotine is pumped rapidly to all parts of the body. The nicotine may reach the brain in about 7 seconds and the big toe in 15 to 20 seconds (Ashton and Stepney, 1982). Stalhandske (1970) showed that intravenously injected nicotine is immediately taken up in the brain of mice, reaching a maximum concentration within one minute after injection; however, by 60 minutes the concentration had fallen to within 1% of the maximum level attained. Studies with many species of animals including mice, cats, dogs, and monkeys have shown that nicotine is taken up quickly by the brain and other nervous tissues (Ashton and Stepney, 1982). Ashton,

Thompson, and Marsh (1981) note that most of the actions of nicotine are exerted on the nervous tissues. The major reasons for this preferential uptake of nicotine by the nervous system are probably the result of its high lipid content, along with its abundant blood flow. However, the high lipid solubility of free nicotine and constant stream of blood through these tissues means that the nicotine can diffuse out of the tissue readily and be carried away. The speed of exit of nicotine from the brain is almost as dramatic as its speed of entry (Ashton and Stepney, 1982).

Nicotine is one of the shortest acting of the common psychoactive drugs. It is destroyed rapidly in the liver. After repeated does, about half of the nicotine in the blood is metabolized with 80 to 100 minutes (Gerstein and Levison, 1982). Russell (1976) reported that the plasma half-life of nicotine is less than 30 minutes. As indicated above, the metabolism of nicotine is relatively rapid. The chief organ of metabolism is the liver; however, some metabolism does occur in other organs, including the lung, but not the brain. When nicotine is absorbed via the stomach into the portal system, most of the nicotine, in its first passage through the liver, is metabolized into cotinine and nicotine-N-oxide, which are pharmacologically inert substances. When absorption occurs through the lungs or nasal and buccal mucosae, the liver is bypassed, allowing the nicotine to get to the brain and other parts of the body in the active form (Russell, 1976).

Nicotine is also present in sweat and saliva, but the urine is by far the major avenue of excretion from the body of nicotine and its metabolites (Russell, 1976). Schachter and coworkers (1977) observed that nicotine is excreted more slowly when the urine is alkaline. The

rate at which nicotine is excreted is thus pH-dependent. Drugs that are basic are excreted fastest when the urine is acidic, whereas acidic drugs are excreted fastest when the urine is alkaline. Eating is another factor that affects urinary pH. The urine becomes more acidic about 30 minutes after eating, and then grows more alkaline and remains so for several hours (Leavitt, 1982). It is interesting to note that many users tend to use tobacco just after a meal.

Nicotine produces physiological effects similar to those of sympathetic stimulation including increased heart rate, blood pressure, metabolic rate, and the secretion of epinephrine (Leavitt, 1982). Nicotine has been found to help rats learn more rapidly and affects memory. This facilitation of learning and conditioning illustrates the dual actions of nicotine, which stimulates and arouses, or sedates and relaxes. The response appears to be dependent on dose and individual constitution as well as on mood and situation. In general, smaller doses improve performance, while larger doses impair performance. These responses may be attributable to two tranquilizing effects of nicotine, namely, 1) a relaxing effect on skeletal muscle, and 2) an increase in the rate of habituation to unimportant stimuli (Russell, 1976).

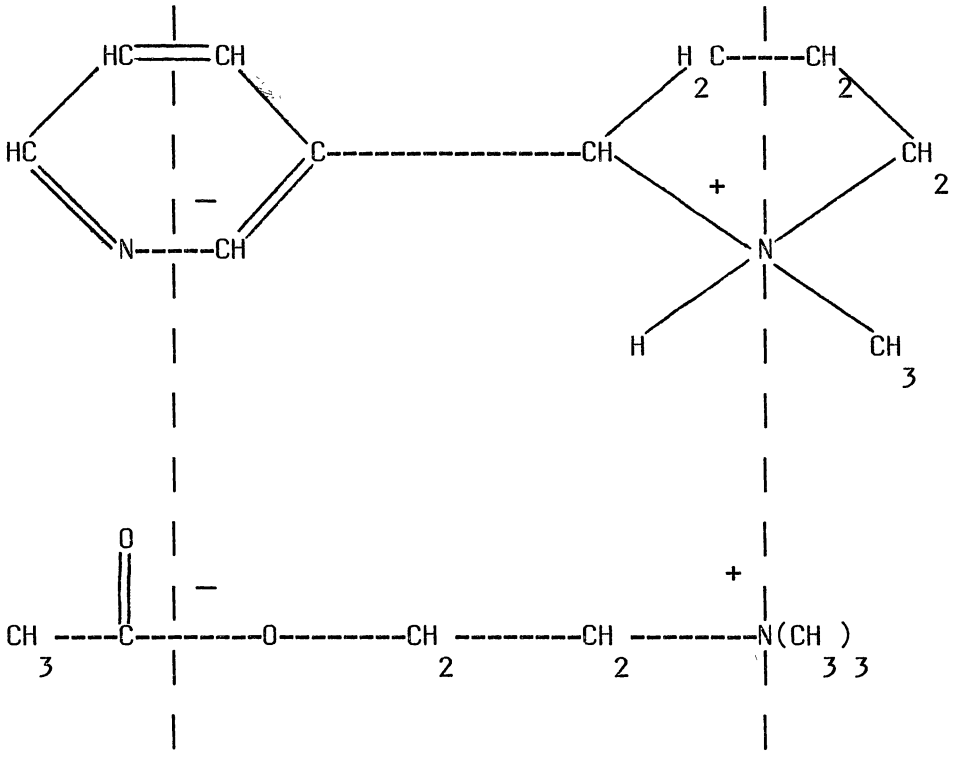
It is in the nervous system, specifically at the synapses and the neuromuscular junction, where the effects of nicotine are unique. The nicotine molecule is structurally very similar to the

acetylcholine molecule (Figure 3). Ashton and Stepney (1982) note that

> One way in which the aceytylcholine receptor recognizes its key, the acetylcholine molecule, is by the position of two electrical charges, one positive and one negative, located at certain sites on the molecule. The distance between these two charges is always the same and corresponds with two equally spaced and oppositely charged sites on the receptor. In the nicotine molecule, the positive charge on the ammonium head and the negative charge on the pyridine ring are just the same distance apart as they are on the acetylcholine molecule. This structural similarity makes nicotine a perfect skeleton key to interact with the acetylcholine receptors (p. 37).

Several studies have shown that nicotine mimicks the effects of acetylcholine at the myoneural junction (Horrobin, 1968; Kruk and Pycock, 1979; Remond, 1979). In effect, nicotine can also initiate muscular contraction. However, nicotine has been found to have a biphasic effect at the myoneural junction; that is, the initial combination of nicotine with the muscle receptor causes a predominantly excitatory effect followed by a predominantly inhibitory effect. However, the receptor molecule appears to possess an equal affinity for the nicotine and acetylcholine molecules. The nicotine combination with the muscle receptor is more enduring than the acetylcholine-muscle receptor combination, thereby effectively blocking any further muscular response. These biphasic effects depend both upon the amount of nicotine present and the time interval between nerve impulses generated at the synapse (Ashton and Stepney, 1982). In summary, much of the complexity and paradox of the effects of nicotine appear to be a result of the biphasic action at the cholinoceptive sites where nicotine is an agonist, then a blocker of acetylcholine

Nicotine



Acetylcholine

Source: Heather Ashton and Rob Stepney, Smoking--Psychology and Pharmacology (1982).

Figure 3. The structure of nicotine and acetylcholine

(Russell, 1976).

From the previous studies, it is obvious that the actions of nicotine are complex. It is clear that there is a mixture of stimulant and depressant properties of nicotine at a variety of levels that involve the central and peripheral nervous systems. Domino (1979) points out that much more research is needed in these areas, and further notes that an important area that needs to be observed in explaining tobacco use behavior is in the subsequent, secondary relaxant effect of nicotine following the initial excitatory effect.

Smoking Studies. Most of the research which exists on tobacco has been obtained by studies involving smoking tobacco. Much of the information has been obtained in both animal and human studies. After reviewing many studies, Ashton and Stepney (1982) feel that nicotine and smoking in humans can, in certain circumstances

> ... facilitate performance, increase or decrease arousal, and combat the disruptive effects of aggression and stress on behavior (p. 116).

Russell (1976) notes that the experimental data seems to support the subjective experience of smokers that it helps them to think, concentrate, and cope with stresses. He further indicates that these appear to be acquired symptoms of dependence, rather than primarily rewarding effects. Nicotine has been found to reduce aggressiveness in rats, and smoking may also improve the memorization of new material (Russell, 1976). The effects of nicotine are not the same in all individuals, but seem to be influenced by the mental and physical

makeup of the smoker (Stepney and Pycock, 1982). Stepney (1979) suggests that smoking tends to normalize the smoker's state of arousal by decreasing it under stress and increasing it during boredom. He goes on to say that smoking enhances performance on tasks that require vigilance and attention by reducing the disruptive effects of anxiety and aggression on performance and behavior.

Dunn and associates (in Battig, 1978) reported that in a contrived situation to produce anger, the incited anger was less disruptive of task performance in the smoker than in the nonsmoker and the deprived smoker. They go on to suggest that perhaps smoking provides a coping ability from the disruptive effects of excessive arousal. Schachter (1973) reported similar results using an arousal state produced by increasing levels of pain (electric shock) and found the ongoing behavior less disrupted in smokers than in nonsmokers. Another study in which smoking made the disruptions less intrusive was reported by Friedman and coworkers (1974) in which habituation to a stimulus (loud, brief bursts of sound presented at random intervals) was more rapid when subjects were smoking than when not smoking.

Nicotine is primarily absorbed through the lungs in cigarette smoking. Russell and coworkers (1980) observed that it takes the smoker about 10 minutes to reach peak levels of nicotine in the circulating blood. The cardiovascular effects of smoking are predominantly stimulant, causing an increase in heart rate, blood pressure, and the basal metabolic rate. The effect of smoking on the heart rate and blood pressure can last for as long as an hour. Smoking tobacco cigarettes has also been found to cause a drop in skin temperature (Russell, 1976). Stepney and Ashton (1982) report

an increase in the efficiency of nicotine metabolism in smokers; that is, smokers metabolize nicotine more quickly than nonsmokers. This increased nicotine tolerance develops in a relatively short period of time after nicotine use (Ashton and Stephney, 1982). The nicotine levels attained in smoking are degraded in the manner previously discussed on nicotine metabolism. It may be important to note that the conversion of nicotine to one of its chief metabolites, cotinine, is inhibited by the presence of one of the products of cigarette smoke, carbon monoxide (Russell, 1976). This may have some significance in the process of cigarette smoking.

Most of the research measuring the electrophysiological effects of nicotine in humans and animals has been conducted in studies using cigarette smoking. Knott (in Remond, 1979) reported longer reaction times in subjects after smoking. There have been some studies that reported the effects of nicotine and tobacco smoking on skeletal muscle tone (Clark and Rand, 1968; Domino and Von Baugarten, 1969; Fagerstrom and Gotestam, 1977; and Webster, 1964). These studies report varying effects of smoking. In two of these studies (Clark and Rand, 1968; and Domino and Von Baumgarten, 1969) the patellar reflex was depressed by nicotine. Fagerstrom and Gotestam (1977) however, reported that the electromyographical activity of the trapezius muscle in the neck increased during tobacco smoking. Summarizing these studies, Domino (1979) states that

> ... tobacco smoking of varying nicotine content has biphasic effects as far as electromyographic activity in concerned. Some skeletal muscles such as the trapezius show enhanced electromyographic activity while other muscles such as the quadriceps femoris show reduced electromyographic activity (p. 142).

These studies further seem to emphasize the recurring dual effects of nicotine at various sites in the body found by several investigators.

Smokeless Tobacco Studies. In contrast to the large amount of literature existing on tobacco smoking studies, there exists very little research dealing with smokeless tobacco. This is probably a result of the obscurity of smokeless tobacco use within the past century. Most of the literature that does exist concerning smokeless tobacco involves surveys of smokeless tobacco use and the health-related risks associated with its use. An extensive amount of health-related literature seems to suggest a link between smokeless tobacco use and the increased incidence of oral cancers (Christen et al., 1979; McGuirt, 1983; Squires et al., 1984; Winn et al., 1981). A substantial amount of literature also exists that would not appear to confirm this relationship (Shklar et al., 1985; Smith et al., 1970; Smith, 1975), or suggest that smokeless tobacco may be a viable alternative to cigarette smoking (Kirkland, 1980; Russell et al., 1980).

Kozlowski (1984) reports that there is really no dispute that smokeless tobacco use presents fewer hazards to the user than the risks that are involved in smoking. Smokeless tobaccos expose the lungs to virtually no tobacco toxins, since no carbon monoxide or tar is produced. In addition, the oral cancers associated with smokeless tobacco are substantially less lethal and are more easily diagnosed than lung cancers (Kozlowski, 1984). This is a very controversial topic at this time and it is obvious that additional research is needed before the issue can firmly be resolved.

Historical evidence seems to suggest that tobacco provides some satisfaction when it is taken as snuff or when held in the mouth and chewed (Russell, 1978). Nicotine is absorbed through the buccal mucosa during tobacco chewing. Russell and coworkers (1980) observed that a peak nicotine level is attained in the circulating blood in 5 minutes or less with the use of snuff. It has also been reported that nicotine transfer from lip to brain can require less than 10 seconds (Jaffe, 1980). Any nicotine absorbed through the nasal or buccal mucosae gains access to the general circulation in the active form before being metabolized in the liver, thereby delivering its effects quickly. Whereas inhaled powdered snuff and cigarette smoke produce a very rapid rise and subsequent fall in the plasma nicotine level (Russell, Jarvis, and Feyerabend, 1980), smokeless tobacco use produces a more gradual increase in the blood nicotine level as it attains similar peak concentrations (Gritz et al., 1981). Squires and associates (1984) suggest that the absorption of nicotine in its aqueous solution, when smokeless tobacco is used, should be similar to the absorption process involved in cigar smoking when the pH level is alkaline.

Smokeless tobacco appears to produce the same cardiovascular effects of smoked tobacco and intravenously administered nicotine. Squires and associates (1984) found increases in heart rate and blood pressure in human subjects after using smokeless tobacco. A recent study corroborated these acute increased heart rate effects after smokeless tobacco use in a college-aged group of athletes and nonathletes (Edwards et al., 1985).

A minimal amount of research exists that involves the

electrophysiological effects of smokeless tobacco. The studies that do exist attempt to identify the effects of smokeless tobacco use on various simple motor tasks, including reaction and movement times, as well as total performance times. A recent study concluded that there was no improvement in neuromuscular performance when reaction, movement, and performance times were measured after ingesting smokeless tobacco (Edwards et al., 1985). It was also suggested that the reason athletes may feel that their performances are improved while under the influence of smokeless tobacco may be attributable to the increased hemodynamic responses attained, which may lead to a psychological feeling of readiness (Edwards et al., 1985). In another study, it was concluded that reaction and performance times did not improve after ingestion of smokeless tobacco when using either a simple visual stimulus reaction time test or an ocular rotary pursuit test (Glover et al., 1984). It was also hypothesized that perhaps nicotine present at the neuromuscular junction assists acetylcholine in the neural transmission, thereby decreasing the reaction time component at this stage (Glover et al., 1984).

Literature Related to Methodology

Electromyography

It has been known, at least since the middle of the nineteenth century, that the contraction of muscle tissue is accompanied by an electrical change. This electrical change has been called the muscle action potential, or map (deVries, 1980). Electromyography is the study of this electrical activity that is occurring in the muscle by

recording the muscle action potentials generated.

The practical use of this discovery had to await the invention of more sensitive instrumentation, as the early equipment was somewhat crude. The physiological aspects of electromyography were first investigated in Germany, in the early 1910's. English-speaking countries did not become involved in electromyography until after a publication by E.D. Adrian in 1925 (Rasch and Burke, 1978). This paper by Adrian (1925) reported that it was possible to determine the amount of activity occurring in human muscles during movement by using electromyography.

The electromyograph probably represents one of the most significant developments in kinesiology as it has provided the means to study the neuromuscular mechanisms in great detail. Electromyography is used in many kinesiological research studies as well as in several clinical settings. In clinical settings, physicians, physical therapists, and other professionals may use electromyography to assist them in making diagnoses, as well as for making accurate prognosis and determination of appropriate treatment (Kraft, 1980). Much electromyographic instrumentation and procedures have been developed for the use of physicians in diagnosing abnormal neuromuscular function (deVries, 1980).

In practice, the electromyographic instrumentation is a unique system designed as a whole, with considerable interrelation between circuit functions and with a number of common power supply and control circuits. In general, it contains some type of transducer (electrodes) connected to an amplifier system that amplifies the action potential so that it can be displayed on various types of display mediums,

including cathode ray tubes, loudspeakers, and paper and fiber optic recorders. The electrodes represent the critical link in the electromyographic system. The choice of recording electrodes, their location relative to the anatomical structures being studied, and their state of cleanliness and repair have a large effect on the observed potentials. Recorded electromyographic potentials and applied electrical stimulations are obtained using metal needle and skin surface electrodes. These techniques impose different performance requirements on the electromyography system (Reiner and Rogoff, 1980).

Needle electrodes are used when the activity of individual motor units or muscle fibers are studied. Most of the 434 skeletal muscles in the human body are accessible using needle electrodes (Johnson and Parker, 1980).

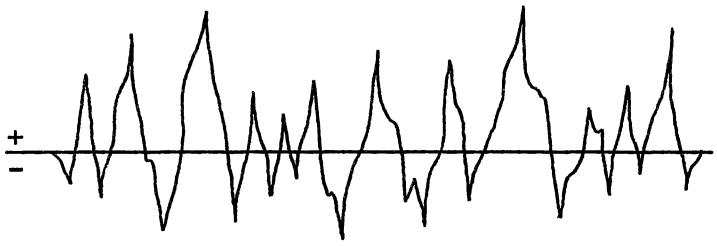
Surface electrodes are used mainly when recording gross electromyographic activity and compound nerve and muscle potentials resulting from nerve stimulation, and are not useful when recording the details of individual motor unit activity. A wide range of skin surface electrodes are available. Surface electrodes may be discs, or rectangular or strip forms, of various size, with the larger sizes used as ground electrodes. These electrodes are held in place with adhesive tape, straps, or double-sided adhesive discs. Bare metal electrode contact is made to the skin via electrically conductive electrode paste which reduces contact resistance, improves recording, and permits stimulation with less intensity, thus reducing the subject's discomfort, and reducing artifact. The skin surface should be cleaned to remove perspiration, which is electrically conductive,

from the areas of recording and stimulation (Reiner and Rogoff, 1980).

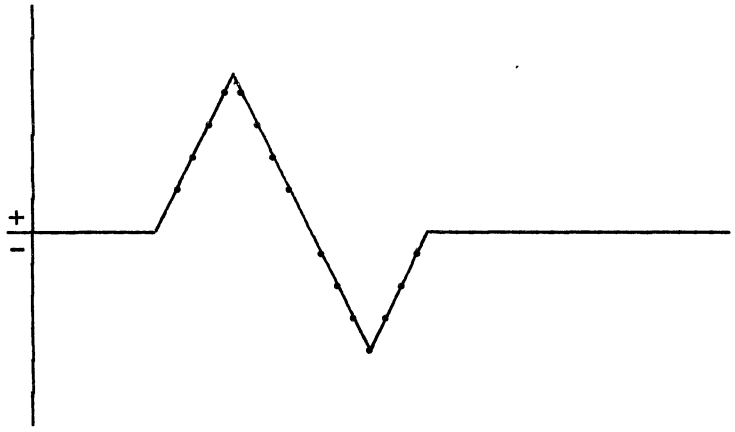
The muscle action potentials, when passing through the muscle tissue, fascia, subcutaneous fat, and skin, are severely decreased in magnitude (deVries, 1980). The ion movement which constitutes the action potential is an electric current in the extracellular fluid that is volume conducted in three dimensions (Reiner and Rogoff, 1980).

The equipment that is used in modern electromyography must be adjustable over a wide range of sensitivities and time scales and be capable of a number of modes of operation. The electrical potentials that may be of interest can range from millionths of a volt (microvolts) that are involved in sensory studies, up to thousandths (millivolts) of a volt encountered in some muscle action potential recording. In addition, the time resolution required can vary from seconds in gross electromyography studies, down to millionths of a second (microseconds) in single fiber electromyography (Reiner and Rogoff, 1980). The strength of contraction, and the appropriate instrumentation required, depends upon the nature of the investigation and is related to the number of motor units activated in motor studies (Scott, 1975).

No activity is observed in normal muscle when at rest; however, action potentials are produced on contraction. These action potentials are summated from individual motor units asynchronously and result in a characteristic recording displayed on the cathode ray tube known as an interference pattern (Figure 4). When an individual fiber contracts, a wave of reversal of polarity passes along it and current flows first in one direction, then in the other (Figure 4). This produces the typical diphasic action potential on



(A) INTERFERENCE PATTERN



(B) DIPHASIC ACTION POTENTIAL

Source: Herbert A. deVries, Physiology of Exercise for Physical Education and Athletics (1980).

Figure 4. Electromyograph Recordings

the recorder (Scott, 1975). When electromygraphical investigations are conducted, the recordings are usually standardized so that downward deflections indicate positivity (Johnson and Parker, 1980).

Finally, Johnson and Parker (1980) note that when electromyography studies are performed, it is essential that the investigator have a thorough knowledge of functional anatomy, kinesiology, and surface anatomy, and suggest that an illustrated anatomic text be near the electromyograph.

Nerve Conduction

Reaction time has been recognized since the mid-nineteenth century as a potentialy powerful means of relating mental events to physical measures (Brebner and Welford, 1980). The time relationships involving the nerves and muscles can be measured using nerve conduction instrumentation. Stimulation of a motor nerve trunk elicits a contraction of the muscle which it supplies. The speed with which the nerve impulse travels can be assessed by measuring the interval of time that elapses between the application of the stimulus and the resulting action potential in the muscle (Scott, 1975).

The use of motor nerve conduction studies is a relatively recent phenomenon, even though these studies are now routinely performed in clinical practice. The procedure is limited to those nerves which are accessible to stimulation, such as the median nerve in the arm (Goodgold and Eberstein, 1972). Procedures have been formulated for stimulating various other nerves within the body, including the ulnar and radial nerves in the upper extremity, and the peroneal, tibial, femoral, and sciatic nerves in the lower

extremity (Braddom and Schuchmann, 1980).

In clinical nerve conduction studies many nerve fibers are depolarized simultaneously, which results in a compound action potential; that is, the combined electrical effects of many action potentials from different nerve fibers is obtained (Melvin, 1980). The stimulation of large nerve trunks evokes nerve action potentials that are recordable mainly from the larger fibers, and the conduction velocity obtained represents the fastest conducting fibers of the mixed nerve (Braddom and Schuchmann, 1980).

Nerve conduction velocity has also been demonstrated to be related to the intraneural temperature, with the velocity decreasing with decreasing temperature until approximately 8 centrigrade, when nerve function ceases (Abramson et al., 1966; Braddom and Schuchmann, 1980).

Nerve conduction velocity has not been found to vary significantly with increasing age. Normal adult values of nerve conduction velocity are usually reached by age 4. Braddom and Schuchmann (1980) report that these values remain relatively stable through age 60 before any decrease is observed.

In motor nerve conduction studies there is a difference in the distal latency that is observed and that which would be predicted on the basis of the proximal nerve conduction velocity. This occurrence was discussed earlier in the introduction and is related to the fiber size and the time required to cross the neuromuscular junction. Normal distal latencies have been fairly well estabished for the median nerve (Table I).

TABLE I

NORMAL VALUES OF MEDIAN NERVE DISTAL LATENCIES

Recording Distance	Mean Value	Std. Dev.	Range	Reference
7 cm.	3.3 msec.	---	2.6-4.5 msec.	(1)
8 cm.	3.7 msec.	0.3 msec.	---	(2)

Source (1): William J. Shriber, A Manual of Electrotherapy (1975).

Source (2): Ernest W. Johnson, Practical Electromyography (1980).

Nerve conduction studies are usually performed using commerical electromyographical apparatus that incorporates built-in conduction equipment. This equipment requires the addition of a nerve stimulator to the standard electromyograph apparatus that is synchronized with the cathode ray tube display so that the sweep is triggered slightly before the stimulus is delivered to the subject. There are many types of stimulators available for use in conduction studies. Generally, there are two basic types: a surface bipolar stimulator that stimulates on top of the skin, and needle electrode stimulators (Braddom and Schuchmann, 1980). A current that changes suddenly in intensity has been found to be the most effective in producing a stimulus, rather than one which changes gradually; as a result, most stimulators provide a square wave stimulus (Scott, 1975). Braddom and Schuchmann (1980) state that the nerve stimulator should be able to deliver a stimulus for a duration that can be varied between 0.1 millisecond to at least 1.0 millisecond, and also have variable frequency selection ranging from 0.5 to 50 hertz. There is some disagreement regarding where the actual site of stimulation is occurring--the cathode, anode, or someplace between these two poles--when stimulating at the skin's surface. It is common practice to orient the bipolar stimulator so that the cathode is closest to the active recording electrode (Braddom and Schuchmann, 1980).

Although most of the techniques for performing nerve conduction studies are relatively simple, the researcher must be aware of some potential sources of error that can lead to invalid results. There are specific sites in different regions of the body where normal

anatomical variations can and frequently do occur. An example of this occurrence in the forearm is a condition referred to as the Martin-Gruber anastomosis, where the median nerve sends fibers across to the ulnar nerve. This reportedly occurs 15 to 25% of the time in normal subjects (Braddom and Schuchmann, 1980).

Cress and associates (1963) reported a relationship between hand dominance and the conduction velocity in the median and ulnar nerves, with the velocity being slightly faster in the preferred arm. However, several other studies failed to find any relationship in hand dominance (Bhala and Goodgold, 1968; Currier and Nelson, 1974; LaFratta and Smith, 1964).

Basmajian (1967) cautioned against making single clinical estimations of nerve conduction velocity in a brief period of time because of the normal diurnal rhythms observed in any individual. However, Wyrick and Duncan (1970) concluded that nerve conduction velocities obtained throughout the day within subjects will be consistent as long as the measures are obtained in a consistent manner and do not require more than 15 or 20 minutes.

Electromyographic and nerve conduction equipment should be properly maintained and calibrated to minimize nerve conduction error due to equipment malfunctions. Probably the most common error is having the time scale miscalibrated (Braddom and Schuchmann, 1980).

It is imperative that the technique used is standardized so that previous results can be compared with the current results. A standardized distance between the active recording electrode and the stimulator cathode should be used. Braddom and Schuchmann (1980) noted that the lengths of nerve segments in the median nerve below the elbow

can be measured accurately with a calibrated tape.

There is also some variation in reading the exact latency value from the recording obtained. Honet and associates (1968) reported an error of 4 to 5% due just to the variabiity of reading the latency values in these studies. Melvin and coworkers (1973) reported standard deviations of 0.3 milliseconds for the distal latencies in motor conduction using these techniques.

Reversal of the stimulating cathode and anode can result in a prolonged distal latency value. The stimulus that is applied should always be supramaximal, otherwise some of the fastest conducting fibers may not be stimulated. This would result in a falsely prolongated latency value. However, if an extremely high supramaximal stimulus is used, the stimulus could be volume conducted to adjacent nerves and could also result in an erroneously short distal latency. These techniques must be considered when performing these studies (Braddom and Schuchmann, 1980).

Finally, it should be obvious that for the safety of the subject and to facilitate interference-free studies, it is important to make sure that the equipment is plugged into a receptacle having a functioning ground.

The investigator should also have a good knowledge of basic neurophysiology and neuropathology in order to accurately interpret the results of these studies.

CHAPTER III

METHODOLOGY AND DESIGN

Introduction

The purpose of this chapter is to describe the research methodology that was employed in this study. Descriptions of the subjects, the research design, the variables, the instruments that were used for the collection of data, the procedures followed, and the method that was used for the statistical analysis of the data are presented.

Subjects

The sample for this study was selected from volunteer students that were enrolled in courses that were taught in the School of Health, Physical Education, and Leisure Studies at Oklahoma State University during the 1985 spring semester. Information and signup materials were distributed to all of the teachers in the department. Sample forms are included in Appendix A. A total of thirty-five students indicated an interest in participating in the study. Twenty subjects actually participated in the study. Two subjects were eliminated from the study since they did not complete all three of the treatment conditions used in the study. One additional subjects data was not used in the analysis because one of the pretreatment distal latency values in one of the treatment conditions was

abnormally long. Complete data was obtained for seventeen subjects, which comprised the final sample size.

Subjects were allowed to schedule their testing periods throughout the day between the hours of 8:00 a.m. and 9:00 p.m. during the 1985 spring semester. The actual testing period lasted approximately 30 to 45 minutes. The subjects were required to abstain from any tobacco use during the two hours prior to each testing period. In addition, subjects were required to abstain from any strenuous physical activity involving the right arm during the two hours preceding each testing period.

Each of the subjects received each treatment (placebo chewing gum, one pouch of Skoal Bandits, and the subject's regular "dip") during the study. Only two different brands of smokeless tobacco were used by the subjects in this study: Copenhagen and Skoal. Each subject received only one treatment in each testing session; therefore, each subject was required to complete three testing periods. The testing sessions were scheduled at least 24 hours apart.

Research Design and Variables

The research design that was utilized in this study to test the statistical hypotheses was the control-group time-series (repeated measures) design (Campbell and Stanley, 1963). This design was chosen because it was considered essential to obtain a pretreatment measure of distal latency in order to reliably evaluate the effects of the treatment.

The fixed independent variables that were used in this study consisted of time and the separate treatments that were administered

to each subject. The three treatments administered to the subjects consisted of the following:

1. Experimental #1: One pouch of "Skoal Bandits" (United States Tobacco Company) smokeless tobacco.

2. Experimental #2: The subject's regular brand and "dip" of smokeless tobacco, Copenhagen or Skoal (United States Tobacco Company).

3. Control: One rolled-up stick of "Extra" (Wm. Wrigley Jr. Co.) chewing gum, without nicotine.

The continuous dependent variable in this study formed a time construct of median nerve motor distal latencies. The sixteen levels of time forming the construct included one pretreatment distal latency and the distal latencies obtained after the treatment had been administered and was obtained for every minute from one minute through fifteen minutes.

Instrumentation

The NS6 nerve stimulator module, two AA6T electromyography/ biological amplifiers, and two PA63 preamplifiers of the TE42 electromyography system (TECA Corporation) was used to measure the median nerve motor distal latencies. TECA paired surface electrodes were used to record the electromyographic potentials over the abductor pollicis brevis and flexor pollicis muscles of the right hand. A TECA bipolar stimulator electrode was used to stimulate the median nerve at the wrist of the right hand. The stimulator was synchronized with the cathode ray tube display so that the sweep was triggered slightly before the stimulus was delivered to the subject.

The distance between the active recording electrode and the wrist stimulation site was measured with a calibrated cloth tape to the nearest millimeter.

The nerve stimulator provided a single, director pulse output of one pulse per second (±5%) for a duration of 0.1 millisecond (±5%). The stimulating voltages available were variable from 0 to 300 volts.

The AA6T electromyography/biological amplifier utilized the PA63 remote preamplifier at the end of an input cable, which provided minimal interference. The amplifier gain settings were variable from 5 microvolts/centimeter to 10,000 microvolts/centimeters, and also provided three different pairs of frequency filter settings. The amplifier provided four internal calibration voltages.

The TECA TE42 electromyography system utilized a cathode ray tube to display the potentials and a high speed direct recorder that copied the cathode ray tube display onto a fiber optic film recording. Sweep speeds that were available on the cathode ray tube display ranged from 0.5 to 500 milliseconds per division. The high speed recorder allowed recording speeds from 0.5 to 100 centimeters per second.

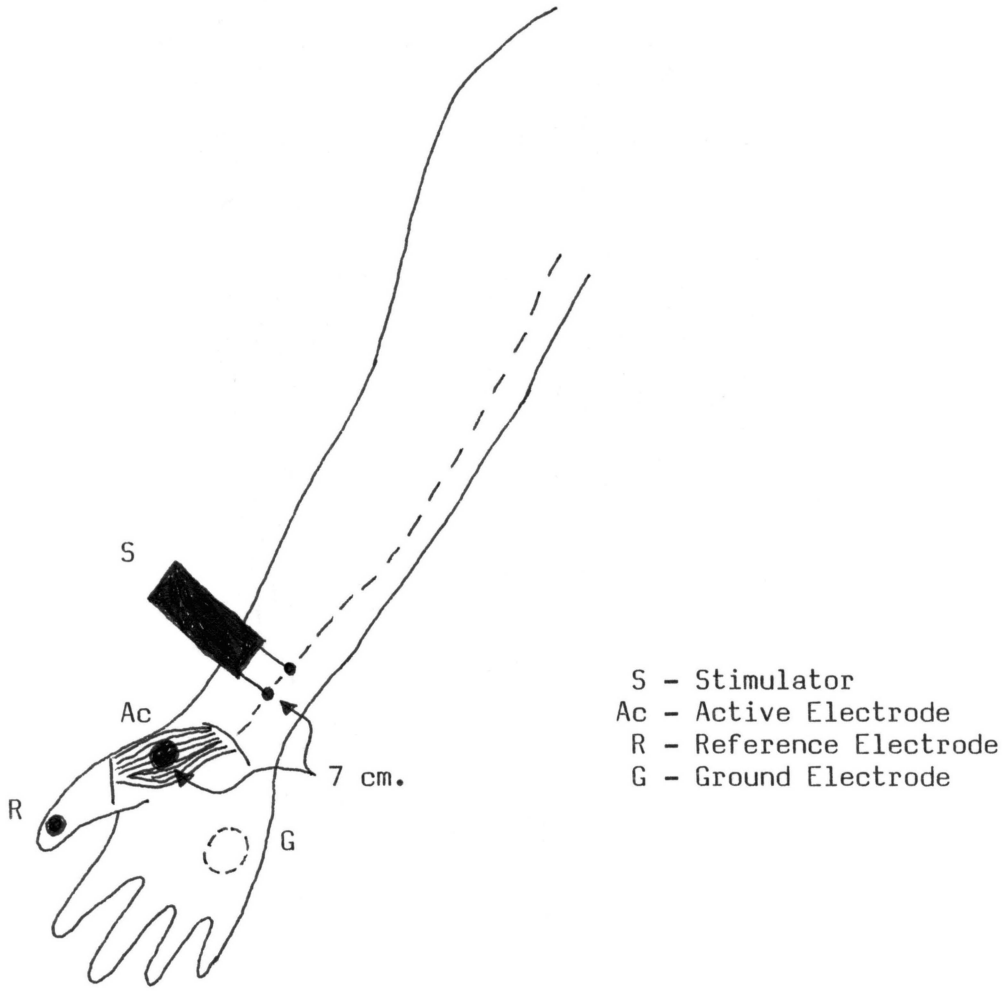
Procedure

All of the motor nerve distal latency testing was performed in the Human Performance Laboratory (room 126) of the Colvin Center at Oklahoma State University. The testing was conducted in a room that provided a constant environmental temperature.

Preceding the first test session, each subject completed a human subjects consent form (Appendix B) and a questionnaire describing their

tobacco habits (Appendix C). After completing the forms in the first test session, the test period was initiated. After a pretreatment distal latency was obtained, the subject was administered a treatment. The subjects were allowed to expectorate the accumulated saliva into a container freely. The subjects were asked not to swallow the tobacco juice. Median nerve motor distal latencies were then recorded at each of the following posttreatment times: one minute through fifteen minutes, one per minute. Two additional test periods were then scheduled, during which the subject received the remaining two treatments. All of the subjects completed the three testing sessions at least 24 hours apart, and all subjects reported that they had adhered to the requirement of abstinence from tobacco use and strenuous activity prior to the testing sessions. All procedures were identical except for the treatments received.

Since the literature failed to show any significant differences between distal latency values obtained involving handedness, all subjects used the right hand in all of the tests, which facilitated the experimental apparatus setup. The subject was in a seated position with the right arm extended and resting on a flat surface at shoulder level. The active recording electrode was placed one half the distance between the metacarpophalangeal joint of the thumb and the midpoint of the distal wrist crease (Figure 5). The reference electrode was placed distally on the thumb, and the ground electrode was placed over the dorsal side of the ulnar border of the hand. The nerve stimulation site was a distance of seven centimeters proximal to the active electrode in a location between the palmaris longus and flexor carpi



Source: Ernest W. Johnson, Practical Electromyography (1980).

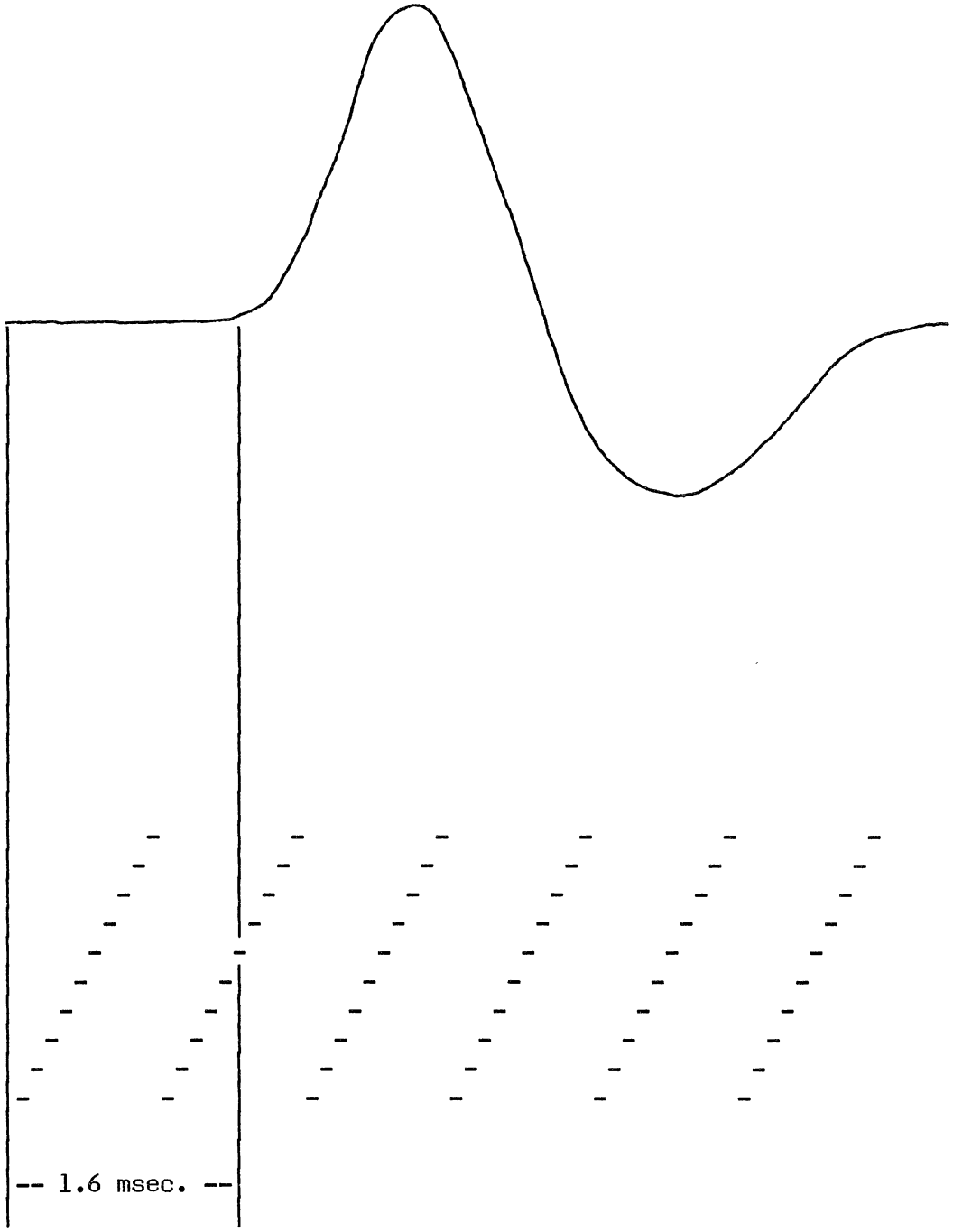
Figure 5. Electrode Placement and Stimulation Site

radialis tendons. This site was marked with a felt-tipped pen to facilitate accurate stimulating placements.

The gain setting that was used on the amplifier was 5000 microvolts/centimeter, with a low frequency filter setting of 2 hertz and a high filter setting of 10,000 hertz. The sweep speed that was used on the cathode ray tube was 2 milliseconds/centimeter. The optic recorder was set to the single-sweep superimposed mode, which recorded the trace on the cathode ray tube onto stationary paper. The paper was then advanced manually after each record was made.

A calibration voltage of 1000 microvolts/centimeter was used at the beginning of each testing session. All distal latency measurements were obtained by slowly increasing the stimulator output voltage until the maximum amplitude response was obtained. The voltage was then increased an additional 25 to 50% to ensure a supramaximal stimulus. The stimulus voltages used in this study ranged from about 160 to 280 volts.

All responses were recorded on the fiber optic film, which was then used to calculate the distal latencies. The distal latencies were calculated by counting the time marks from the beginning of the sweep on the recording to the initial rise of the action potential recorded on the paper. Each small time mark on the recording represented a time interval of 0.1 milliseconds. A vertical line was drawn on the recording that coincided with the initial deflection of the action potential, and extended down into the time recording (Figure 6). A sample distal latency recording form that was used in the study is included in Appendix D.



Source: Ernest W. Johnson, Practical Electromyography (1980).

Figure 6. Distal Latency Calculation.
Slanted dotted lines represent 1 msec.

After all the data had been obtained and analyzed, all participants in the study were contacted and provided a summary of the overall findings along with each subjects individual results by a letter (Appendix E). This letter also informed each subject that the chewing gum treatment that they had received in the study had been a placebo and did not contain any nicotine.

Statistical Analysis

The P2V package of the BMDP computer program on an IBM 3081D computer system was used to analyze the data. This program allowed the median nerve motor distal latencies to be analyzed using an analysis of variance (ANOVA) with repeated measures. The .05 level of significance was used in testing the hypotheses.

CHAPTER IV

RESULTS AND DISCUSSION

Characteristics of Subjects

Seventeen volunteer subjects participated in this study, ranging in age from 18 to 32 years. Background information concerning each subject's tobacco habits was collected from the prestudy questionnaire (Appendix C) and is presented in Table II. The average period of time that the subjects reported using smokeless tobacco products was 6.3 years, with a range from 2.5 years up to 12 years. Seven of the smokeless tobacco users in this study regularly used Copenhagen brand snuff, while the remaining ten subjects used regular Skoal. The subjects estimated the number of dips that they used each day, as well as estimated the size of the pinch they normally ingested. The general concensus showed that a medium-sized pinch was administered about six times per day by the subjects used in this study. An interesting observation surfaced from the prestudy questionnaire concerning when smokeless tobacco was used by the subjects. The majority of subjects reported using smokeless tobacco routinely after eating and when studying. Although this finding was incidental to this study, these behaviors were supported by similar findings in the review of the literature.

All subjects reported that they had adhered to the requirement of

TABLE II

CHARACTERISTICS OF SUBJECTS

	Subject Number																	
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	Mean
Age (Years)	22	23	21	23	22	22	19	18	22	22	18	20	23	23	27	30	32	22.8
Regular Tobacco Habit (C=Copenhagen S=Skoal)	C	C	C	C	C	C	S	S	S	S	S	S	S	S	S	S	C	C-7 S-10
Length of Habit (Years)	12	5	3.5	6	6	8	2.5	7	8	10	5	6	5	9	4	6	4	6.3
Avg. Dips per Day	9	11	4	5	8	7	6	5	4	5	11	5	7	3	2	4	5	6
Size of Pinch (S=Small M=Medium L=Large)	M	L	M	M	L	L	M	M	S	M	L	M	S	L	M	M	M	S-2 M-10 L-5

abstinence from tobacco use and strenuous activity prior to the testing sessions. No subject reported any physical problems as a result of their participation in the study. All subjects seemed to adapt to any minor discomfort that may have occurred in the initial testing session.

Data Analysis Results

The raw data results area included in Appendix F. A repeated measures analysis of variance (ANOVA) was used to evaluate changes in the median nerve motor distal latency values over the duration of the investigation. The 3 X 16 repeated measures ANOVA included one within factor, condition, with three levels (chewing gum, Skoal Bandits, and regular dip), and another within factor, time, with sixteen levels (one pretreatment distal latency and fifteen posttreatment distal latencies, spaced one minute apart). The chewing gum level of the first factor provided a non-nicotine control situation.

Table III presents the means and standard deviations of the distal latencies measured at each time for the tobacco usage groups and the treatments that they received. Table IV presents a summary of the repeated measures ANOVA analysis.

A significant F ($F = 1.72$, $p < 0.05$) was found with the interaction between condition and time. A graph of the condition X time interaction (Figure 7) indicated that both nicotine conditions resulted in a slowing of the median nerve distal latencies. The graph of the non-nicotine chewing gum does not appear to follow this same pattern.

A significant F ($F = 8.59$, $p < 0.05$) was also found for the main effect of time. Pair wise post hoc contrasts, using Tukey's HSD test, were performed comparing the mean of the pretreatment distal latency

TABLE III

MEANS AND STANDARD DEVIATIONS OF THE MEDIAN NERVE MOTOR DISTAL LATENCIES (MSEC)

		Treatment Condition					
		Gum		"Bandits"		Regular Habit	
Time	N	Mean	SD	Mean	SD	Mean	SD
PreTest	17	3.56	.433	3.66	.352	3.47	.291
1 Min Post	17	3.56	.421	3.68	.366	3.49	.333
2 Min Post	17	3.58	.421	3.68	.373	3.48	.319
3 Min Post	17	3.56	.436	3.66	.367	3.51	.309
4 Min Post	17	3.59	.403	3.69	.409	3.49	.324
5 Min Post	17	3.59	.423	3.70	.384	3.49	.331
6 Min Post	17	3.61	.426	3.70	.400	3.51	.315
7 Min Post	17	3.58	.425	3.70	.391	3.53	.324
8 Min Post	17	3.59	.427	3.71	.397	3.54	.343
9 Min Post	17	3.59	.425	3.73	.410	3.54	.324
10 Min Post	17	3.59	.407	3.75	.411	3.55	.312
11 Min Post	17	3.58	.422	3.75	.403	3.55	.354
12 Min Post	17	3.60	.409	3.75	.429	3.56	.308
13 Min Post	17	3.57	.418	3.75	.421	3.56	.312
14 Min Post	17	3.57	.403	3.75	.417	3.57	.326
15 Min Post	17	3.59	.412	3.79	.412	3.58	.354

TABLE IV

REPEATED MEASURES ANOVA FOR THE MEDIAN NERVE
MOTOR DISTAL LATENCY VALUES

Source	Sum of Squares	Degrees of Freedom	Mean Square	F Ratio
Condition (C)	5.1914	2	2.59570	4.40*
Error	18.8928	32	0.59040	
Time (T)	0.4935	15	0.03290	8.59*
Error	0.9190	240	0.00380	
C X T	0.2157	30	0.00719	1.72*
Error	2.0068	480	0.00418	

*Significant at the 0.05 level.

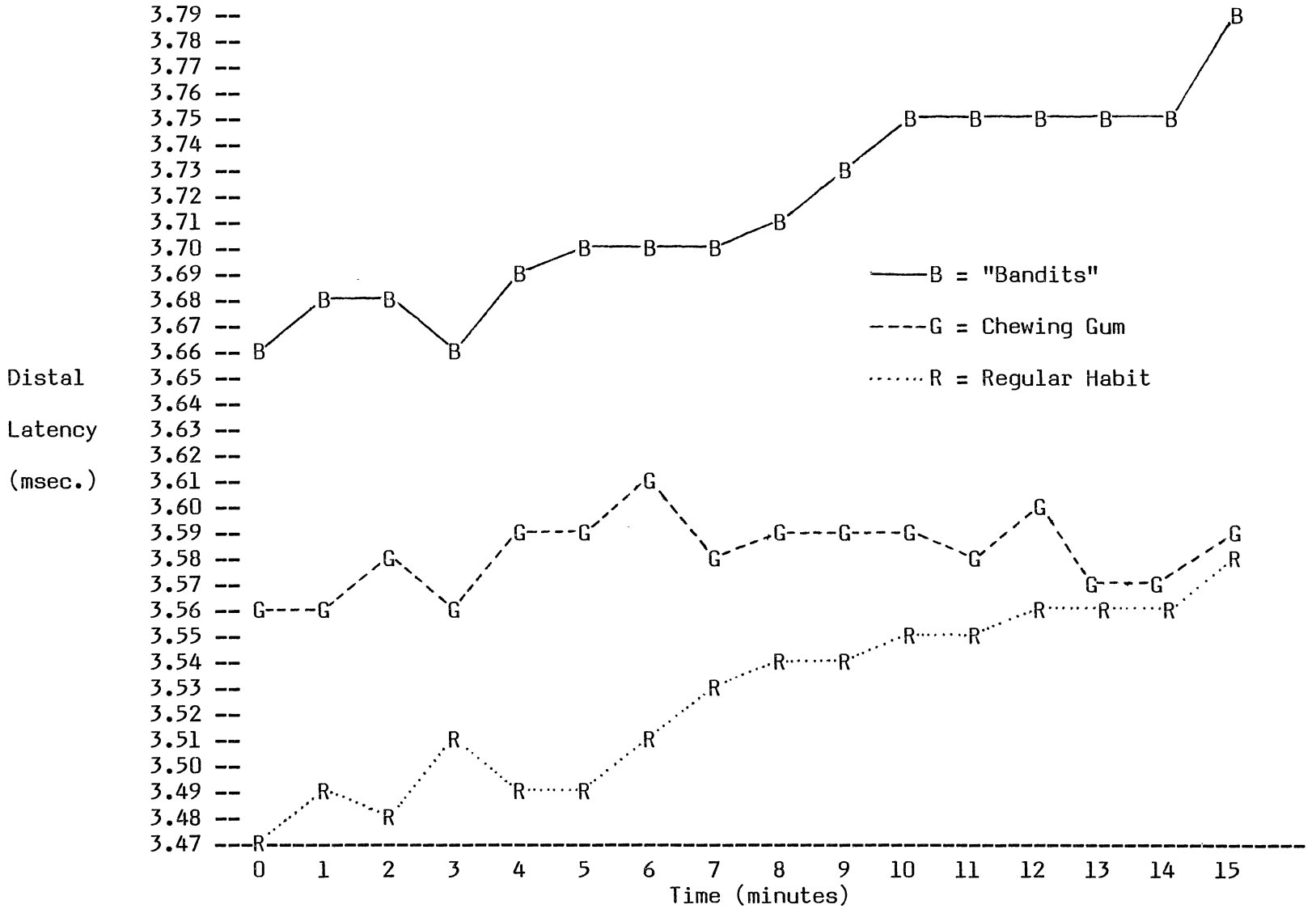


Figure 7. Condition X Time Interaction

with the mean of each of the 15 posttreatment distal latencies. Each treatment condition was tested using these contrasts. No significant differences were found for the time main effect in the chewing gum condition. In both the Skoal Bandits and the regular dip treatment conditions significant differences were found between the pretreatment distal latency and the 10, 11, 12, 13, 14 and 15 minute posttreatment distal latencies.

A significant F ($F = 4.40$, $p < 0.05$) was also found for the main effect of condition. Post hoc contrasts were also performed on this effect; however, no meaningful differences were obtained.

Testing the Hypotheses

A summary of the hypotheses tested, and the results for each is stated below:

Hypothesis 1.

There is no significant difference between the pretreatment median nerve motor distal latencies and the 15 posttreatment median nerve motor distal latencies of those subjects who ingest the placebo chewing gum.

Post hoc comparisons, using the Tukey test, yielded no significant differences in any of the distal latencies after ingesting the chewing gum. The study failed to reject this null hypothesis.

Hypothesis 2.

There is no significant difference between the pretreatment median

nerve motor distal latencies and the 15 posttreatment median nerve motor distal latencies of those subjects who ingest one pouch of "Skoal Bandits" smokeless tobacco.

Post hoc comparisons, using the Tukey test, found significant differences in the 10 through 15 minute posttreatment distal latencies when compared to the pretreatment distal latencies. The study rejected this hypothesis at the 0.05 level of significance.

Hypothesis 3.

There is no significant difference between the pretreatment median nerve motor distal latencies and the 15 posttreatment median nerve motor distal latencies of those subjects who ingest their regular smokeless tobacco dip.

Post hoc comparisons, using the Tukey test, found significant differences in the 10 through 15 minute posttreatment distal latencies when compared to the pretreatment distal latencies. Therefore, this study also rejected this null hypothesis at the 0.05 level of significance.

Hypothesis 4.

There are no significant differences in the 15 posttreatment median nerve distal latencies of the subjects after receiving each of the three treatment conditions.

The finding of a significant interaction between the treatment conditions and time resulted in this hypothesis also being rejected at the 0.05 level of significance.

In summary, this study attempted to identify the differences that resulted in a group of college-aged male smokeless tobacco users under two conditions of nicotine, and one placebo non-nicotine chewing gum control situation. Hypotheses 2, 3, and 4 were rejected at the 0.05 level of significance, whereas this study failed to reject hypothesis 1 at the 0.05 level of significance.

Discussion

The findings of this investigation indicated that the action potential transmission is delayed as a result of smokeless tobacco ingestion, presumably as a result of the presence of nicotine at the neuromuscular junction. This finding does not support the hypothesis proposed by Glover and Associates (1984), and is, in fact, in opposition. No enhancement of the conducted impulse across the neuromuscular junction seemed to occur due to the presence of nicotine. This study supports the antagonistic role of nicotine (Kruk and Pycock, 1979) by suggesting that nicotine present at the neuromuscular junction slows down the conducted impulses. Since the nicotine-receptor combination has been found to be longer lasting than the acetylcholine-receptor combination (Ashton and Stepney, 1982), perhaps this slowing is the result of the nicotine-receptor combinations at the neuromuscular junction that selectively block some of the faster conducting nerve fibers.

This study also supported the expected research hypotheses of the investigator. It was anticipated that there would be a slowing of the distal latency values once nicotine reached the neuromuscular junction, due to nicotine's blocking action. No effect was expected following

ingestion of the nicotine-free chewing gum. Nervous transmission at the neuromuscular junction was not expected to improve when nicotine was initially administered. If any facilitation did occur with the initial administration of nicotine at the neuromuscular junction, it probably would not have been observed using the techniques in this study, since the stimulation impulse was applied by an external stimulus.

The apparent absorption rate of nicotine in this study agreed with earlier studies (Gritz et al., 1981; Russell et al., 1980) that found peak nicotine levels in the blood after about 5 minutes with the use of snuff. Gritz and associates (1981) reported a gradual increase in the blood nicotine level with the use of smokeless tobacco. Examination of the graphs of the means during the two nicotine treatment conditions (Figure 7) indicated that the distal latencies were gradually beginning to slow after about 5 minutes. Statistical significance between the pretreatment distal latency and the posttreatment distal latencies did not occur until the 10 minute posttreatment time period; however, both graphs show the gradual slowing, which suggests that there was a gradual increase in the amount of nicotine present at the neuromuscular junction.

Another interesting finding was the variation in the pretreatment distal latencies among the three treatment conditions. Since these differences were not statistically significant, however, they probably represent normal variabilities in nerve conduction and distal latency studies. It was noted earlier (Table I) that normal variations in the median nerve distal latencies can range from 2.6 to 4.5 milliseconds.

Another possible explanation of the variations that occurred in these measures might be attributed to the subjects tobacco use before each testing period. Although the order of treatments received was counterbalanced, the subjects were aware of what treatment condition they were going to receive in each session. This may have influenced their tobacco habits in the time period immediately before the two hour abstinence period prior to the scheduled test. Subjects may have abstained from their regular tobacco habit for a longer period of time before the two hour abstinence period on that test day, since they knew that they would be receiving a dip of their regular habit on that day. In followup studies, a longer abstinence period than the two hours used in this study should be considered.

Although significance was found in the interaction and both main effects, caution must be observed in interpretation. Strength of association measures were calculated (η^2) for each of these effects. Roughly 19% of the variance in the distal latency measures in this sample was accounted for by the treatment condition, which is relatively strong. However, the strength of association for the time and interaction effects, 1.8% and 0.8% respectively, are very weak. Even though the main effects and interaction were statistically significant, they may not represent practical significance. A slowing of the impulse by one-tenth of a millisecond may not be of too much concern to tobacco users to alter their habit. However, when one considers the complexity of the nervous system and the importance of acetylcholine as a neurotranmitter in many synapses, the results could take on added importance. This study evaluated the effects only at the

neuromuscular junction in the right thumb. This mechanism is likely occurring and influencing several other links in the conduction system of the human body.

CHAPTER V

CONCLUSIONS AND RECOMMENDATIONS

The major conclusion that can be made from this study is that the distal latency recorded in the median nerve at the wrist is slowed as the result of smokeless tobacco ingestion. This slowing did not seem to be related to the dosages administered in this study, as similar changes were observed in both tobacco conditions. Perhaps some minimal required dose was obtained in the treatments used in this study that sufficed to produce the observed effects.

In addition, the non-nicotine chewing gum did not appear to follow the same trend as the two tobacco treatment conditions, even though this was not able to be statistically demonstrated.

From the research studies that were reviewed for this study, it seems obvious that the actions of nicotine are complex. It is further concluded that not enough research has been completed to help answer many of the perplexing questions that exist.

The relationship that seems to exist between tobacco use and eating was identified in the literature review and also surfaced in the responses obtained on the prestudy questionnaire. This would be an interesting area for additional research. There also appears to be some type of relationship that is behaviorally related to why people use smokeless tobacco. This is also an area that deserves additional research.

It is recommended that this study be replicated with a larger sample size. Although the minimal sample size was obtained for this study, additional subjects may provide a more powerful test of these hypotheses. A mixed model design could be employed with a larger sample size, thereby allowing the investigator to randomly assign subjects to different treatment conditions. This could facilitate the recruitment of subjects since the subject would only have to complete one testing session. Perhaps the variance between the pretreatment distal latencies observed in this study would be decreased. In addition, the distal latency values could be observed beyond the 15 minute posttreatment times that were used in this study. Other nerve-muscle junctions in the body could also be readily observed using these techniques.

It is recommended that this study be replicated with a more quantified dosage of nicotine administered to the subjects. Although the "Skoal Bandits" condition used in this study was somewhat standardized, the regular habits of the subjects may have varied considerably since two brands of smokeless tobacco (Copenhagen and Regular Skoal) were used by the subjects. A study using nicotine-free and nicotine gum could be used to obtain this quantification.

Another recommendation for follow up studies of this nature involves the element of control. It is recommended that the subjects that participate in any future studies be carefully screened in regards to their usage of other drugs such as alcohol or marijuana. These factors were not involved in this study and should probably be considered. Two additional factors that should also be considered include the time of day the subjects are tested and the subjects

previous activity and sleep levels. More control might be obtained by addressing these factors in future studies.

Finally, these smokeless tobacco and nicotine studies could and should be extended to sensory and motor nerve conduction velocity investigations in order to observe the effects of nicotine in other areas of the human nervous system. It would be interesting to observe if the results of these future studies would parallel the results that were obtained in this investigation.

It is somewhat disconcerting to read about the growing number of smokeless tobacco users in this country, especially among our young children, who are using these products without a great deal of knowledge concerning the acute and chronic effects of smokeless tobacco products on the human body. The scientific community must rapidly rise to the challenge of discovering the full effects of smokeless tobacco use on the human body.

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APPENDIXES

APPENDIX A

SAMPLE SELECTION FORMS

NERVE CONDUCTION-SMOKELESS TOBACCO STUDY

Dear Instructor:

I am currently involved in the process of identifying and selecting subjects for my doctoral research. My research seeks to measure the electrophysiological effects of smokeless tobacco on the nerve-muscle junction in the median nerve of the arm.

I would appreciate your assistance in identifying possible subjects for my study. I would ask that you make a brief announcement to your class concerning the following information and pass around the signup sheet included, and then return it to me as soon as possible.

I am especially interested in identifying subjects currently using smokeless tobacco and who would be interested in participating in this study. I plan to randomly select from those subjects indicating an interest to participate, so there is a possibility of a subject not being selected to participate in the study.

Those subjects that are selected for the study will be required to complete three (3) testing sessions, each of which will last approximately 30 minutes. In each session the subjects finger muscles will be electrically stimulated and an electromyograph recording made. The entire procedure will be more fully explained to those subjects wishing to participate in the study.

Please instruct those students who are interested in participating to sign the enclosed form and indicate a phone number where they may be contacted. If any student wishes additional information before committing to the study, please have them indicate on the form or have them stop by my office (room 126) for additional information.

Thank you for your time and assistance in this matter. I greatly appreciate your help!

Sincerely,

Mike Lester

NERVE CONDUCTION - SMOKELESS TOBACCO STUDY
SIGN-UP SHEET

Please list name, phone number, and tobacco use below if you are interested in participating in the study.

	NAME	PHONE NO.	DO YOU USE SMOKELESS TOBACCO
1.			
2.			
3.			
4.			
5.			
6.			
7.			
8.			
9.			
10.			
11.			
12.			
13.			
14.			
15.			
16.			
17.			
18.			
19.			
20.			

APPENDIX B

HUMAN SUBJECTS CONSENT FORM

Protection of Human Subjects Consent Form

Subject's Name: _____ Date: _____

Project Title: The Effects of Smokeless Tobacco Ingestion on the Motor Distal Latency of the Median Nerve

Explanation of the procedure to be undertaken:

Each subject will be asked to complete a written prestudy questionnaire. Each subject will be required to complete three (3) separate testing sessions. Each subject will receive a different treatment in each session. The subject will be required to abstain from any tobacco or strenuous physical activity during the two (2) hours preceding each testing session. After reporting to the Human Performance Lab (Colvin Center, room 126) the subject will be prepared for the EMG/nerve conduction procedures by having three (3) surface electrodes placed on the right hand. The median nerve will then be stimulated at the wrist with the nerve stimulator so that a pretreatment recording can be obtained. The subject will then receive one (1) of the following treatments: 1) one pouch of "Skoal Bandits" smokeless tobacco, 2) the subjects regular dip, or 3) one piece of nicotine substitute chewing gum. One minute after the treatment is received the median nerve will again be stimulated at the wrist and a recording made. This procedure will continue each succeeding minute for a period of fifteen (15) minutes. The subject will be allowed to expectorate at will into a cup that will be provided. The first testing period will last approximately forty-five (45) minutes. The second and third testing periods will last approximately thirty (30) minutes each. The total time required for each subject in the study will be approximately two (2) hours. Each testing session will be scheduled at least twenty-four (24) hours apart.

Explanation of attendant discomforts and risks to be expected:

In this experiment each subject will have the muscles of the right thumb electrically stimulated with the stimulator electrode by the investigator, Mike Lester. Mike Lester has performed these procedures in earlier investigations and is competent in the nerve conduction technique. The subject may experience some discomfort in the initial stimulation process. Although the procedure may result in some minor discomfort, it will not result in any physical damage to the nerve-muscle mechanism. These procedures are routinely performed in clinical settings.

Explanation of benefits to be expected:

The benefits of this project include the opportunity for the subject to become acquainted with experimental procedure as well as the possibility of obtaining additional information on smokeless tobacco effects on the body. In addition, some subjects may receive "bonus points" in a course being taught in the HPELS department by participating in this investigation.

Explanation of confidentiality:

The subject is free to deny answering any question on the prestudy questionnaire. No subject's name or responses to any of the questions on the prestudy questionnaire will be accessible to anyone except the investigator, Mike Lester. Each subjects results will be coded with a number and no names will be used when the results are reported.

I have fully explained to the Subject _____ the nature and purposes of the procedures described above and such risks as are involved in its performance. I have asked the Subject if any questions have arisen regarding the procedures and have answered these questions to the best of my ability.

_____
Investigator's Signature

I have been fully informed of the above noted procedure with its possible benefits, risks, and consequences. I hereby agree to become a subject in this investigation. I understand that if physical, psychological, or other injury should occur as a direct result of this activity, neither compensation nor long-term treatment will be provided. Furthermore, I recognize that I am free to withdraw this consent and to discontinue participation in this project and activity at any time without prejudice to me.

_____
Subject's Signature

APPENDIX C

PRE-STUDY QUESTIONNAIRE

PRE-STUDY QUESTIONNAIRE
(SMOKELESS TOBACCO USERS)

Name: _____ Date: _____

Sex: M F Birthdate: _____ Age: _____

---

1. Are you involved in any organized sport? Y N

   If yes, what sport: _____

---

2. What brand of smokeless tobacco do you regularly use?

   _____

---

3. How often do you regularly use smokeless tobacco?

   a. Number of "dips" per day: _____

   b. How Long (number of days) does one can of smokeless tobacco usually last you?

      _____

   c. What size of "pinch" or "dip" would you classify as your usual dose?

      ___ small pinch ___ medium pinch ___ large pinch

---

4. What do you usually do with the tobacco juice produced?

   _____ always expectorate (spit out)

   _____ usually expectorate; occasionally will swallow juice

   _____ usually swallow juice

---

5. Are there specific times during the day when you normally use smokeless tobacco? Y N

   If yes, when? (describe circumstances): _____

   _____

6. How long have you used smokeless tobacco?

____ less than 6 months

____ between 6 months and 1 year

____ longer than 1 year

If you have used smokeless tobacco for more than 1 year, estimate how long you have used it: _____

---

7. Do you currently use any other forms of tobacco? Y N

If yes, what type and how often?

___ chewing tobacco (leaf or plug) How often used: _____

___ cigar How often used: _____

___ cigarette How often used: _____

___ pipe How often used: _____

___ other: _____ How often used: _____

If no, have you used any other form of tobacco products regularly in the past, and for what length of time?

___ chewing tobacco (leaf or plug) How often used: _____

___ cigar How often used: _____

___ cigartte How often used: _____

___ pipe How often used: _____

___ other: _____ How often used: _____

---

8. Have you ever used Skoal Bandits smokeless tobacco regularly in the past? Y N

If yes, do you still use it regularly? Y N

---

9. Briefly describe why you use smokeless tobacco: _____

_____

_____

APPENDIX D

DISTAL LATENCY RECORDING FORM

DISTAL LATENCY RECORDING FORM

Name: _____ Date: _____

Treatment Received: _____

_____ Subject has abstained from tobacco use for previous 2 hours.

_____ Subject has abstained from strenuous physical activity for previous 2 hours.

Completed	Measure	Calculated Value (msec)
_____	PreTreatment Distal Latency	_____
_____	Treatment Administered	_____
_____	1 minute posttreatment DL	_____
_____	2 minute posttreatment DL	_____
_____	3 minute posttreatment DL	_____
_____	4 minute posttreatment DL	_____
_____	5 minute posttreatment DL	_____
_____	6 minute posttreatment DL	_____
_____	7 minute posttreatment DL	_____
_____	8 minute posttreatment DL	_____
_____	9 minute posttreatment DL	_____
_____	10 minute posttreatment DL	_____
_____	11 minute posttreatment DL	_____
_____	12 minute posttreatment DL	_____
_____	13 minute posttreatment DL	_____
_____	14 minute posttreatment DL	_____
_____	15 minute posttreatment DL	_____

APPENDIX E

FOLLOW UP MATERIALS SENT TO SUBJECTS

Mike Lester
School of HPELS
Oklahoma State Univ.
Stillwater, OK 74078

June 16, 1985

(Study Participant's Name)
(Street Address)
(City, State Zip)

Dear (Participant's Name),

Well I finally finished analyzing all of the data for the smokeless tobacco nerve conduction velocity experiment that you participated in! Your individual results appear on a separate sheet along with the overall results of all of the other participants. Good thing that the computer was invented! I would not have wanted to do all of the statistical calculations by hand...I'd still be doing them!

First off though, I need to inform you that the chewing gum treatment that you received in the study did NOT contain any nicotine! It was just plain old Wrigley's "Extra" spearmint flavored chewing gum. You were told that it contained nicotine so that you "thought" you were still receiving some nicotine. Thus, this treatment condition served as my "control" situation. Sorry about that!

I have enclosed a copy of the abstract from my study, which outlines the major results. Your results are listed on the sheet listing the group scores in each of the treatment conditions. In general, the overall effect of the two nicotine conditions seemed to slow the conduction speed by about 0.1 millisecond!! You can compare your own results to the averages, as a couple subjects actually increased slightly!

I would like to thank you for participating in this study and if you should ever have any other questions concerning it you can contact me around the Colvin Center. Thanks again and have a good summer!

Sincerely,

Mike Lester

Results of all participants:

Treatment Condition	Pre-Treat.	Post-Treatment 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Chewing Gum	3.56	3.56	3.58	3.56	3.59	3.59	3.61	3.58	3.59	3.59	3.59	3.58	3.60	3.57	3.57	3.59
"Bandits"	3.66	3.68	3.68	3.66	3.69	3.70	3.70	3.70	3.71	3.73	3.75	3.75	3.75	3.75	3.75	3.79
Reg. Habit	3.47	3.49	3.48	3.51	3.49	3.49	3.51	3.53	3.54	3.54	3.55	3.55	3.56	3.56	3.57	3.58
Your Results:																
Gum	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----
Bandits	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----
Reg. Habit	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----

Note: The values in the table are the median nerve motor distal latencies obtained.
Units are milliseconds (msec.).

Name: Michael Joseph Lester Date of Degree: July, 1985

Institution: Oklahoma State University Location: Stillwater, Oklahoma

Title of Study: THE EFFECT OF SMOKELESS TOBACCO INGESTION ON THE MOTOR DISTAL LATENCY OF THE MEDIAN NERVE

Pages in Study: 95 Candidate for Degree of Doctor of Education

Major Field: Higher Education

Scope of Study: The purpose of the study was to determine the effects of smokeless tobacco ingestion on the median nerve motor distal latencies in a group of subjects who regularly used smokeless tobacco products. Specifically, the nervous transmission at the neuromuscular junction in the right thumb was compared before and after the subjects were administered three treatment conditions. Two of the treatments contained nicotine (one pouch of "Skoal Bandits" and a pinch of the subjects regular tobacco habit --- Copenhagen or Regular Skoal), while the third treatment consisted of a non-nicotine chewing gum.

Median nerve distal latencies were obtained, using electromyographic and nerve conduction instrumentation, before the treatment was administered to the subject, and once every minute for 15 minutes after the treatment was administered.

The population of the study consisted of seventeen volunteer subjects between the ages of 18 and 32 years. All subjects were males. All subjects received all three of the treatment conditions, spaced at least 24 hours apart.

Findings and Conclusions: Significant differences were obtained among the three treatment conditions over time. The distal latencies observed during the two nicotine treatment conditions gradually increased during the posttreatment time periods. The distal latencies in the non-nicotine treatment condition did not follow this same trend.

It was concluded that smokeless tobacco ingestion resulted in a delay in the nervous transmission across the neuromuscular junction. This delay was hypothesized to be caused by the enduring binding action of nicotine to the acetylcholine receptor sites, thereby effectively blocking the impulse in some of the faster conducting nerve fibers in the neuromuscular junction.

ADVISOR'S APPROVAL _____

APPENDIX F

ACTUAL DISTAL LATENCY DATA

ACTUAL TEST SCORES (Times in Table are in milliseconds)

Subject	Treatment Condition	Pre-Treat.	Post-Treatment 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1	Chewing Gum	2.9	2.9	2.9	2.9	3.0	3.0	3.0	3.0	2.9	3.0	3.0	2.9	3.0	2.9	2.9	2.9
	"Bandits"	3.5	3.5	3.5	3.5	3.5	3.6	3.5	3.5	3.6	3.5	3.6	3.5	3.5	3.6	3.7	3.7
	Reg. Habit	3.2	3.1	3.1	3.1	3.0	3.0	3.0	3.0	3.0	3.1	3.1	3.0	3.1	3.1	3.1	3.1
2	Chewing Gum	3.3	3.3	3.3	3.3	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4
	"Bandits"	3.6	3.7	3.6	3.6	3.6	3.5	3.7	3.6	3.6	3.7	3.6	3.6	3.6	3.7	3.6	3.7
	Reg. Habit	3.6	3.5	3.5	3.6	3.6	3.6	3.6	3.7	3.7	3.6	3.7	3.7	3.7	3.7	3.7	3.7
3	Chewing Gum	3.4	3.4	3.3	3.3	3.3	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4
	"Bandits"	3.4	3.4	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5
	Reg. Habit	3.5	3.5	3.5	3.5	3.4	3.5	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4
4	Chewing Gum	4.3	4.4	4.3	4.3	4.3	4.3	4.3	4.3	4.3	4.3	4.2	4.3	4.3	4.3	4.2	4.2
	"Bandits"	4.0	4.2	4.2	4.2	4.2	4.2	4.2	4.0	4.0	4.2	4.2	4.1	4.3	4.2	4.2	4.3
	Reg. Habit	3.9	4.0	3.9	3.9	3.9	3.8	3.8	3.8	3.9	3.9	3.9	3.9	3.9	3.9	4.0	4.0

ACTUAL TEST SCORES (Times in Table are in milliseconds)

Subject	Treatment Condition	Pre-Treat.	Post-Treatment 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
5	Chewing Gum	3.4	3.4	3.5	3.4	3.4	3.5	3.5	3.4	3.4	3.5	3.5	3.5	3.5	3.4	3.4	3.4
	"Bandits"	3.5	3.5	3.5	3.4	3.5	3.4	3.4	3.4	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5
	Reg. Habit	3.6	3.7	3.7	3.7	3.7	3.8	3.8	3.8	3.8	3.8	3.8	3.9	3.9	3.9	3.9	4.0
6	Chewing Gum	3.2	3.2	3.2	3.2	3.3	3.3	3.3	3.2	3.2	3.2	3.2	3.3	3.3	3.3	3.3	3.3
	"Bandits"	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.3	3.3	3.4	3.4	3.4	3.4	3.4	3.4
	Reg. Habit	3.3	3.3	3.2	3.3	3.3	3.3	3.4	3.4	3.3	3.3	3.4	3.3	3.4	3.3	3.4	3.3
7	Chewing Gum	3.4	3.4	3.5	3.4	3.4	3.4	3.5	3.4	3.4	3.5	3.5	3.4	3.4	3.4	3.4	3.5
	"Bandits"	3.7	3.7	3.6	3.7	3.7	3.8	3.8	3.9	3.9	3.8	3.8	4.0	3.8	3.8	3.8	3.9
	Reg. Habit	3.1	3.0	3.0	3.1	3.0	3.0	3.0	3.0	3.0	3.0	3.0	3.1	3.0	3.1	3.0	3.0
8	Chewing Gum	4.1	4.0	3.9	3.9	3.9	3.9	3.9	3.7	3.9	3.8	3.8	3.9	3.9	3.8	3.8	3.8
	"Bandits"	4.0	4.0	3.9	3.9	3.9	3.9	3.9	3.9	3.9	3.9	3.9	4.0	4.0	4.0	4.0	4.1
	Reg. Habit	3.8	4.0	4.0	4.0	4.0	4.0	3.9	3.9	3.9	4.0	4.0	4.0	3.9	3.9	3.9	4.0

ACTUAL TEST SCORES (Times in Table are in milliseconds)

Subject	Treatment Condition	Pre-Treat.	Post-Treatment 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
9	Chewing Gum	3.3	3.4	3.4	3.4	3.4	3.3	3.4	3.4	3.4	3.3	3.3	3.4	3.4	3.3	3.4	3.4
	"Bandits"	3.4	3.4	3.5	3.4	3.4	3.5	3.4	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5
	Reg. Habit	3.5	3.6	3.6	3.5	3.5	3.6	3.6	3.7	3.8	3.7	3.7	3.7	3.7	3.7	3.8	3.8
10	Chewing Gum	4.0	4.0	4.1	4.1	4.1	4.1	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.2
	"Bandits"	3.9	3.9	3.9	3.9	4.1	4.1	4.1	4.1	4.3	4.3	4.2	4.3	4.3	4.3	4.3	4.3
	Reg. Habit	3.9	3.7	3.7	3.7	3.8	3.8	3.8	3.9	3.8	3.8	3.8	3.7	3.8	3.8	3.8	3.9
11	Chewing Gum	3.7	3.8	3.8	3.8	3.8	3.9	3.8	3.8	3.9	3.9	3.9	3.9	4.0	3.8	3.8	3.9
	"Bandits"	4.3	4.4	4.5	4.4	4.6	4.5	4.5	4.5	4.4	4.5	4.6	4.5	4.5	4.5	4.5	4.5
	Reg. Habit	3.6	3.8	3.7	3.8	3.7	3.7	3.8	3.8	3.8	3.8	3.8	4.0	3.7	3.8	3.8	3.8
12	Chewing Gum	3.3	3.3	3.3	3.3	3.4	3.3	3.3	3.3	3.4	3.3	3.3	3.3	3.3	3.3	3.4	3.4
	"Bandits"	3.3	3.4	3.3	3.3	3.3	3.3	3.4	3.3	3.3	3.3	3.3	3.3	3.3	3.4	3.3	3.4
	Reg. Habit	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.4	3.4	3.3	3.4	3.4	3.4	3.4

ACTUAL TEST SCORES (Times in Table are in milliseconds)

Subject	Treatment Condition	Pre-Treat.	Post-Treatment 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
13	Chewing Gum	4.3	4.3	4.3	4.3	4.3	4.3	4.4	4.3	4.3	4.3	4.3	4.2	4.2	4.2	4.2	4.2
	"Bandits"	4.1	4.0	4.0	4.0	4.0	4.0	4.1	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.2
	Reg. Habit	3.8	3.8	3.8	3.8	3.9	3.9	3.9	3.9	3.9	3.9	3.9	3.9	3.9	3.9	3.9	3.9
14	Chewing Gum	4.0	4.0	4.0	4.1	4.0	4.1	4.0	4.1	4.0	4.0	4.0	4.0	4.0	4.1	4.1	4.2
	"Bandits"	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.3	4.2	4.3	4.3	4.3	4.3
	Reg. Habit	3.5	3.6	3.7	3.7	3.6	3.6	3.6	3.7	3.8	3.8	3.7	3.8	3.8	3.8	3.8	3.8
15	Chewing Gum	3.7	3.4	3.6	3.6	3.6	3.6	3.6	3.6	3.6	3.5	3.5	3.5	3.5	3.5	3.5	3.5
	"Bandits"	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.4	3.4	3.5	3.5	3.5	3.5	3.5	3.5
	Reg. Habit	3.0	3.0	3.1	3.0	3.1	3.0	3.1	3.1	3.0	3.1	3.1	3.0	3.1	3.1	3.1	3.1
16	Chewing Gum	3.2	3.3	3.3	3.2	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.3	3.3
	"Bandits"	3.4	3.4	3.4	3.4	3.4	3.5	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4
	Reg. Habit	3.4	3.4	3.4	3.5	3.4	3.4	3.5	3.4	3.5	3.4	3.4	3.4	3.5	3.5	3.4	3.4

ACTUAL TEST SCORES (Times in Table are in milliseconds)

Subject	Treatment Condition	Pre-Treat.	Post-Treatment 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
17	Chewing Gum	3.1	3.1	3.1	3.1	3.1	3.0	3.0	3.0	3.1	3.0	3.1	3.0	3.1	3.1	3.1	3.1
	"Bandits"	3.2	3.2	3.2	3.2	3.1	3.2	3.1	3.2	3.1	3.2	3.2	3.2	3.1	3.0	3.1	3.2
	Reg. Habit	3.0	3.0	3.0	3.1	3.1	3.1	3.1	3.2	3.2	3.2	3.3	3.2	3.3	3.2	3.3	3.2
18	Chewing Gum	5.4	5.3	5.3	5.4	5.4	5.3	5.4	5.4	5.4	5.4	5.4	5.4	5.5	5.6	5.5	5.5
	"Bandits"	4.4	4.4	4.3	4.4	4.4	4.4	4.4	4.5	4.5	4.5	4.5	4.6	4.5	4.5	4.6	4.6
	Reg. Habit	4.3	4.3	4.3	4.4	4.3	4.4	4.7	4.7	4.7	4.7	4.8	4.8	4.8	4.8	4.8	4.8

NOTE: Data for subject # 18 was not used in the analysis.

VITA 2

Michael Joseph Lester

Candidate for the Degree of

Doctor of Education

Thesis: THE EFFECT OF SMOKELESS TOBACCO INGESTION ON THE MOTOR DISTAL LATENCY OF THE MEDIAN NERVE

Major Field: Higher Education

Minor Field: Health, Physical Education and Recreation

Biographical:

Personal Data: Born in Racine, Wisconsin, September 7, 1952, the son of Michael S. and Beverly Ann Lester. Married to Marietta P. Rasmussen on August 16, 1980 and to this union Justin and Kyle were born.

Education: Graduated from Sacred Heart High School, Miles City, Montana, 1970; received the Bachelor of Science Degree in Physical Education from Lewis-Clark State College, 1976; received the Master of Science Degree in Physical Education from the University of Arizona, 1979; enrolled in the doctoral program at Oklahoma State University, 1981 and completed the requirements for the Doctor of Education degree, July, 1985.

Professional Experience: Teaching Assistant, Department of Physical Education, University of Arizona, August, 1976, to June, 1977; employed as an elementary Physical Education teacher 1977 to 1978; employed as an assistant baseball coach and men's dorm director 1980 to 1981; Teaching Assistant, Department of Health, Physical Education and Leisure Studies, Oklahoma State University, August, 1981 to June, 1985.

Professional Organizations: American Alliance for Health, Physical Education, Recreation and Dance; International Society of Biomechanics in Sports; American Baseball Coaches Association.