UNIVERSITY OF ALBERTA

AN INVESTIGATION OF NEUROFEEDBACK TRAINING WITH ALCOHOLICS OF CANADIAN ABORIGINAL ANCESTRY

BY

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ABSTRACT

This explorative study investigated the use of neurofeedback training (NT) with alcoholics of Canadian Aboriginal ancestry. Eleven alcoholics admitted to Poundmaker's Lodge alcohol treatment centre volunteered as informed participants. One participated in a single-case pilot study held at the Cognitive Re-regulation Clinic, Faculty of Education, University of Alberta. Methods were altered from the pilot study findings to reduce confounding factors and to minimize assessment procedures. Assessments used: Minneapolis Multiphasic Personality Inventory-revision two (MMPI-2), Beck Depression Inventory (BDI), Shipley Institute of Living Scale (SILS), pre and post-qualitative EEG recordings (QEEG), and NT recordings. Ten participants were classified *Secondary Alcoholics*, where alpha dominates with other factors such as psychological problems. One participant was classified *Primary Alcoholic*, where beta dominates and alcoholism is the primary problem. Pre-assessment recordings show a sample QEEG profile suggesting multiple scalp site markers (high F3, low P3, low T5, and alpha dominates) are better indicators of alcoholism than is a single site marker. Participants who produced a crossover effect with theta and alpha rhythms experienced abreaction of childhood traumas, suggesting NT is a viable adjunct to psychotherapy and may be necessary for sustained sobriety. A three year follow-up indicated lifestyle factors can override the efficacy of NT. Conclusions suggest NT should be researched and applied during grade school years, the common age of onset for alcoholism. Further suggestions are given for future research with this form of biofeedback for helping individuals to gain self-control over their addiction problems.

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INTRODUCTION

Context of the Problem

Alcoholism is a pervasive disease that causes personal and familial hardship and has placed a great financial burden on the economic resources of medical and social service systems. Studies of alcoholism were conducted between 1987 and 1988 from four international sites: St. Louis, Missouri, U.S.A.; Puerto Rico; Taipei, Taiwan; and Edmonton, Alberta, Canada (Helzer, Canino, Hwu, Bland, Newman, & Yeh, 1988). National studies were also conducted in Canada (Harinath, 1989), and the United States (Sullivan, 1991). These studies concluded that alcoholism is a serious problem and is strongly associated with a wide range of physical and mental illnesses. The cross-national study further indicated that, per capita, alcoholism and its associated health problems were found to be more prevalent in the Edmonton site (Helzer et al., 1988).

Canadian Aboriginal Peoples and Alcoholism

During the past three decades, a number of research studies have investigated the problem of alcoholism specifically among Canadian Aboriginal peoples (Grand Council Treaty 3, 1978; Jarvis and Boldt, 1982; Federation of Saskatchewan Indian Nations, 1984; Aboriginal Women's Council of Saskatchewan, 1988; Ontario Native Women's Association, 1989; Frank, 1992). These researchers stressed that alcoholism is a serious problem for many Canadian Aboriginal peoples and plays a major role in family violence in many First Nations communities.

Canadian Aboriginal Demographics

The nearly 1,000,000 Canadian Aboriginal peoples (Status and non-Status Indian, Metis, and Inuit), representing approximately 3.5% of the Canadian population (Health and Welfare Canada, 1992), live in six recognized cultural areas (Brizinsky, 1989), with diverse patterns in social customs as well as in alcohol consumption (Bray and Anderson, 1989). The results from the Health Status and Social Concerns section of the 1991 Aboriginal Peoples Survey concluded that that 61% of the people survey reported alcohol abuse was a problem in their communities, and 48% reported drug abuse was a problem. Prohibiting alcohol, in order to overcome this problem in First Nations communities, was believed to be the answer by less than 1% of those surveyed, and only 1.2% believed that returning to traditional values would overcome this problem (Statistics Canada, 1991a).

Effective Programs Needed

More specific to the Alberta area, a 1982 study on Indian and Metis mortality in Alberta concluded that the "special life circumstances" of Aboriginal peoples who lived under low socio-economic status, made them more vulnerable to alcoholism and accidental/violent deaths (Jarvis and Boldt, 1982). A recent mental health needs assessment conducted in 1995 on mental health issues in the North-Western region of Alberta indicated that the Aboriginal people living in this region perceive alcoholism as continuing to be a very serious problem in their communities. The assessment further indicated that essential to resolving the problem of alcoholism is the need for effective treatment programs for Aboriginal peoples (Layman and Holden, 1995).

Biofeedback Treatment Suggested for Alcoholism

In the early 1970s, research indicated that alcoholism treatment programs such as the Alcoholics Anonymous (AA) support system were often found to be unsuccessful and not serving the needs of all alcoholics (Wells, 1991). Recent studies have further concluded that self-help programs are largely successful during the early stages of alcoholism (Lewis, 1992). Also in the 1970s, biofeedback was suggested as a possible treatment for alcoholism. Biofeedback researchers, such as Lawrence (1972), suggested that altering the brain wave patterns of alcoholics through biofeedback techniques might be effective in changing alcohol habituation.

Prior to the development of biofeedback, the practice of medicine over the past centuries has relied on the four major curative mechanisms or treatment paradigms of: aiding the body's or the mind's natural recuperative power; pharmacological interventions; surgical interventions; and the effect of the practitioner. The development of biofeedback methods has provided a fifth mechanism in the form of behavioural control in which the individual can take a fully active role in learning how to be well (Birk, 1973).

Biofeedback: An Educational Model

Biofeedback training is based upon an educational model where learning is influenced by cognitive variables. Subjects engaged in biofeedback training learn to develop greater awareness and personal control of their physiology and behaviour. What would normally take years of conscious effort through meditation techniques, such as taught in Hatha Yoga, can now be learned in a few months with the aid of biofeedback equipment (Thomas, DasGupta, and Reyer, 1991).

Proof of Biofeedback Efficacy

In 1967, at the annual meeting of the Pavlovian Society of North America, Neil Miller presented evidence of the effects of biofeedback using techniques that were first developed during the 1960s by Jay Trowill. Miller placed electrodes in the pleasure centres of the brains of animals which were immobilized with d-tubo curarine and were artificially respirated. The animals were able to learn through operant conditioning to either increase or decrease heart rate, change blood pressure, increase or decrease kidney flow, change the vasodilation in their ears, and increase or decrease gastric motility (Lynch, 1973).

By the early 1970s, it appeared that nearly all functions of the body could be influenced to some extent by biofeedback training (Lynch, 1973). Changes to a specific part of the body were found to be due to simply concentrating on that one function. For example, changes in heart rate were found to be made by the act of concentrating only on the heart. Changes to the body from such awakened or altered states of consciousness were also found to be enhanced with the help of biofeedback instruments, such as the EMG (electromyography, for monitoring the electrical activity of muscles) or the EEG (electroencephalograph, for monitoring the electrical activity of the brain) (Orne and Paskewitz, 1974).

EEG Activity

The bioelectrical activity of the human brain is recorded in the form of EEG recordings by placing electrodes on the human scalp according to the International 10/20 electrode placement system (Appendix I). The electrical activity of the brain fluctuates in frequencies or cycles per second, referred to as Hertz (Hz). For

identification purposes, ranges of frequencies were assigned names using the names for the letters of the Greek alphabet as follows: delta, 0.5 Hz - 4 Hz; theta, 4 Hz - 8 Hz; alpha, 8 Hz - 12 Hz; beta, 13 Hz - 50 Hz (Berger, 1929/1969). The amplitude of specific frequencies such as alpha rhythms are found to increase by simply focusing all of one's attention on a single body function, such as breathing (Kamiya, 1969). When a feedback signal is linked to a specific aspect of the electrical activity of the brain, such as alpha frequency or amplitude, the process is then referred to as neurofeedback (Lubar, Swartwood, Swartwood, and O'Donnell, 1995).

Neurofeedback and Alcoholism

In 1988, Peniston and Kulkosky, at the Veterans Association Medical Centre in Fort Lyon, Colorado, found that 15 chronic alcoholic American Vietnam war veterans had low alpha activity and abnormally high beta amplitudes at their left occipital lobe scalp site, or electrode site O1. Using neurofeedback training (NT) methods the subjects were taught to increase the amplitude of their alpha/theta rhythms and at the same time lower their beta amplitudes at the O1 site. This method was reported from three year follow-up studies to be effective in reducing their alcohol habituation and in bringing about positive personality changes (Peniston and Kulkosky, 1991). Following this historic study, further research on the use of NT methods indicates that NT is proving to be an important treatment technique for alcohol addiction and should now be studied with specific groups, such as alcoholics of Aboriginal ancestry (Lowe and MacDowall, 1992).

Overview of Present Study

In order to explore NT for the reduction of alcoholism with alcoholics of Canadian Aboriginal ancestry, the present researcher; in co-operation with the Cognitive Re-regulation Clinic, Department of Educational Psychology, Faculty of Education, University of Alberta; conducted an exploratory study on this method during a period between August 1, 1993 and March 15, 1994. The study investigated NT with Canadian Aboriginal alcoholics who were admitted for treatment in Poundmaker's Lodge, a local Aboriginal operated drug and alcohol rehabilitation centre located in St. Alberta, Alberta, Canada. The centre's main treatment method is based on an integration of the traditional AA model with regional Aboriginal culture, particularly northern Cree.

Conceptual Framework

The emphasis of this research was to gain an understanding of the effects of NT as a treatment for alcoholism, specifically with alcoholics of Canadian Aboriginal ancestry, by examining participants' experiences with this method. Because this study investigated human perceptions of a treatment effect, qualitative methods of inquiry formed the main emphasis of the investigation. However, quantitative measures were also collected; from individual performance in the form of obtained scores on assessment instruments and from quantitative measures of participants' brain electrical activity. Due to the nature of the on site field research setting which was not in the control of the researcher, this study used an emergent research design which integrated both qualitative and quantitative methods.

Research Design

The research design of this study used an integrative approach applying both qualitative and quantitative methods in order to more accurately report the results obtained from the participants experiences with this method of treatment. The gathering of the qualitative data was based on procedures for reporting on a small number of participants in a field research setting as described by Patton (1990). Patton described three types of data collection procedures for reporting qualitative data: *interviews* to gather information from each participant; *gathering of documents* in the form of minutes from meetings, newspaper accounts, autobiographies and dispositions; and the researcher's *observations* of the phenomena being studied (Patton, 1990). Patton further described three types of questioning used in qualitative research: informal conversational, guided, and standardized or fixed response.

The qualitative data about the participant's individual experiences were gathered using conversational techniques during formal pre and post-interviews, using informal conversational techniques and guided questions during training sessions, and from standardized questionnaires with fixed responses.

The quantitative data were gathered in the form of measures obtained on standardized psychological assessment instruments administered to each participant and from participants' quantitative EEG measures from their pre and post-assessment EEG recordings. Measures of participants' changes to the microvolt levels of their brain rhythms were also collected during each training session.

Importance of the Study

This research represents the first study on the application of NT for the reduction of alcohol abuse with alcoholics of Canadian Aboriginal ancestry. An examination of the data has the potential of generating initial information about neurofeedback training with alcoholics of Aboriginal heritage. This study also includes a three year follow-up of the participants' well-being and sobriety after completing NT and the 28 day rehabilitation program. This research has provided important information on NT and has generated suggestions for future research on how NT training might be successfully applied in the treatment of the disease of alcoholism with other Aboriginal peoples in North American and indigenous peoples around the world.

CHAPTER TWO

LITERATURE REVIEW

The following literature review focuses on the historical background of problems associated with alcoholism and especially with Aboriginal peoples in Canada from the first introduction of distilled alcohol up to the time of this study. A brief review of ten major theories is presented to help understand possible underlying factors related to alcohol addiction with this population. Also a brief review is presented of contemporary treatment paradigms and models that have been used across North America and of the Canadian Aboriginal operated treatment centre where this study took place. The final sections of this review focus on the early discoveries of the biofeedback model that led to the development and use of neurofeedback training methods and also focuses on recent research findings showing both supportive and contradictory conclusions of neurofeedback training as a therapeutic intervention for alcoholism.

Early Problems Associated With Alcoholism Amongst North American Indians

Although natural occurring alcohol was used in traditional spiritual ceremonies by some American Indians, such as corn mash by the Hopi (Waters, 1963), it was virtually unknown to most North American Indians. Distilled alcohol as first introduced by the early explorers and fur traders is often cited as the principle cause in the destruction of the North American Indian peoples (Dailey, 1968). The associated problems from alcohol consumption by North American Indian peoples in the early post-contact period was well documented by the French Jesuits. The French monarchy

gave the Jesuits a monopoly on Canadian missions from 1632 to 1690 (Vecsey, 1983). The Jesuits kept meticulous notes of their observations of the Indians in the eastern regions of North America, such as the Oneida, the Ojibwa, the Huron, and the Iroquois. Their observations, referred to as *The Jesuit Relations*, were sent to France for purposes of raising funds. The Jesuit Relations were gathered into 73 volumes by Twaites between 1896 and 1901. Dailey (1968) selected from these 73 volumes the Jesuit references about the destructiveness caused by alcohol consumption among the eastern North American Indian tribes. The following six references cited from Dailey portray the extent of this destructiveness:

> "Every night is filled with clamours, brawls, and fatal accidents, which the intoxicated cause in the cabins" (Vol. 46, p. 105).
>
> "It (drunkenness) is so common here, and causes such disorders, that it sometimes seems as if all the people of the village had become insane, so great is the license they allow themselves when they are under the influence of liquor" (Vol. 51, p. 217).
>
> "When these people are intoxicated, they become so furious that they break and smash everything in their houses; they utter horrible yells and shouts, and like madmen, seek their enemies to stab them. At such times, even their relatives and friends are not safe from their fury, and they bite off one another's noses and ears" (Vol. 67, p. 39).
>
> "Disunion and the dissolution of their marriage invariably result from their drunkenness, owing to the sorrow and despair of their wives when they see themselves despoiled by their drunken husbands who take everything from them to obtain liquor; and are deprived of the proceeds of the hunting, which belong to them, but are taken from their husbands before they reach the village by their creditors" (Vol. 67, p. 39,41).
>
> "... after returning from the chase richly laden with beaver skins, instead of furnishing their families with provisions, clothing, and other necessary supplies, they drink away the entire proceeds in one day and are forced to pass the winter in nakedness, famine, and all sorts of deprivation" (Vol. 46, p 103).

> "One case was reported where a whole village was destroyed by a warring Iroquois band, because all its members were drunk and had neglected to leave even one sentinel" (Vol. 47, p. 141).

According to the Jesuit Relations many Indian peoples at that time believed that alcohol allowed their sprit to be uninhibited and like their dreams their intoxicated behaviour was the real or true nature of their spirit. Thus, due to some traditional cultural views, especially those regarding dreams and sprits, the consumption of alcohol was difficult to control (Dailey, 1968).

Alcohol Problems in Europe and Ancient Civilizations

It is interesting to note that these observations of alcohol related problems amongst North American Indians, as reported by the Jesuits, is reflective of similar problems that were observed in Europe and were historically reported for over 6,0000 years by ancient civilizations. Alcoholic beverages in the form of beer and wine were in use as early as 6,000 years ago by the ancient Babylonians and Egyptians. The Egyptian god Osiris was known by two names, the god of wine and the god of death. Early Arabian alchemists at the school of Alexandria were the first to invent the distillation process whereby alcohol is separated from its base fruit or grain mash (Poley, 1979). Following the invention of the distillation process, the Arabians introduced distilled alcohol to other countries during the Middle Ages. The use of distilled alcohol spread rapidly throughout Europe where it became revered as a medicinal agent. Distilled alcohol became associated with many special events such as births, marriages, deaths, the crowning of kings and queens, and the signing of treaties. Religious monasteries were utilized both as inns and as drinking taverns. Alcohol was also used medically as an antiseptic and an anaesthesia, and served as a base in many

prepared medications, such as salves and tonics (Zahourek, 1991). The Gaelic people believed that distilled alcohol was a remedy for all diseases and hence called it "whiskey," from a Gaelic term meaning "water of life" (Poley, 1979). By the end of the 16th century the destructive propensities of alcohol were apparent, and were referred to by writers such as William Shakespeare (1564-1616) who, for example, alluded to the problems associated with alcoholism in his play about the King of Scotland, MacBeth (as cited in Cox, 1987). *MacBeth:* Act II, Scene I.

> MacDuff and the porter speak:
>
> MacD. What three things does drink especially provoke?
>
> Port. Marry, sir, nose painting, sleep, and urine - Lechery, sir, it provokes and it unprovokes: it provokes the desire, but it takes away the performance: therefore, much drink may be said to be an equivocator with lechery: it makes him and mars him; it sets him on, and it takes him off; it persuades him, and disheartens him; makes him stand to, and not stand to: in conclusion, equivocates him in a sleep, and giving him the lie, leaves him.

Alcohol in Colonial North America

Alcohol was also used extensively by the early European immigrants in place of drinking water because local water was often contaminated. However, because of the problems associated with the consumption of alcohol, early colonial laws viewed drunkenness as a lack of good judgement on the part of the individual. Drunkards were frequently punished by whipping, public humiliation by placement in stocks, and banishment from the colony. Despite these early efforts to control drunkenness the abuse of alcohol and its associated problems expanded with the booming expansion of population of European immigrants (Health & Welfare Canada, 1985).

First Attempts to Prohibit Drinking Alcohol in America

Sales to Indians. In the late 1600's efforts were made in Canada in attempt to eradicate the destructive problems associated with alcohol use in North America by the reduction of alcohol sales to Indians. However, the British and French governments viewed the sale of alcohol as an important means of revenue and stopped attempts to prohibit its sale. In 1822, in response to governmental sanction of the sale of alcohol, the Hudson's Bay Company made an effort to reduce sales to Indians with a resolution for temperance that was presented in London under the direction of Governor Simpson. By 1826, the Hudson's Bay Company became determined to stop the selling of liquor to Indians and in 1860 set a company policy stating that liquor was no longer allowed in their trading with Indian peoples (Ray, 1974).

Temperance societies. The first public action against the use of alcohol by the general public was the formation of the Union Temperance Society in 1808 by farmers in the state of New York (Aaron, P.; & Musto, D., 1981). A general wave of temperance movements spread throughout North America from 1825 to 1855. The first Canadian "temperance society" was formed in Novia Scotia in 1828. During this temperance movement the first prohibition law was passed in 1848 in Maine. Similar prohibition laws and acts were passed in Ontario in 1853, in Quebec in 1855, in New Brunswick and Novia Scotia in 1856, and then throughout Canada. The Canadian "Temperance Act" was passed in 1878 followed by a second temperance movement between 1880 and 1890 (Health & Welfare Canada, 1985). In 1883, the US Christian Woman's Temperance Union, that was formed earlier in 1874, became a world wide organization (Gusfield, 1963).

Prohibition laws rescinded. With the onset of World War I the prediction of alcohol was greatly reduced in North America because of the need to use grain for food. Consequently there was a reduction in alcohol consumption. However, following the end of World War I the use of alcohol increased. The provinces of Ontario, Quebec, British Columbia and the Yukon Territories rescinded their prohibition laws and established policies to control the commercial sale of alcoholic beverages. The province of Manitoba followed in 1923, Alberta in 1924, Saskatchewan in 1927, Novia Scotia in 1930 and Prince Edward Island in 1948 (Health & Welfare Canada, 1985). However in the United States the sale of alcohol was prohibited from 1920 to 1933, based on the Volstead Act of 1919 (Zahourek, 1991).

First Attempts to Treat Alcoholism as a Disease

Following the failure of political, religious and social interventions to eradicate the problems associated with alcohol, the first attempt to overcome drunkenness by treating the problem as a disease was the formation of Alcoholics Anonymous (AA) in 1935 by two alcoholics, a stock broker and a medical doctor, Bill Wilson and Robert Smith. In their literature and presentations on drunkenness, the AA spoke about the disease nature of alcoholism (AA World Services, 1993).

First Medical References to Drunkenness as Disease and Addiction

By the late 1700s, the destructive characteristics of alcohol were well recognized in Europe and in America. In 1784, Benjamin Rush, a signer of the American Declaration of Independence, was the first medical doctor to refer to drunkenness as a progressive disease. Rush proposed that hospitals be established especially for the physical treatment of alcoholism (Cox, 1987). Addiction to alcohol

was first referred to as "alcoholism" in 1849 by the Swedish physician Magnus Huss. In 1884, George Harley was the first to express a connection between alcohol and organic body tissue when he wrote (Sonnedecker, 1963):

> ... hereditary insanity is due to the transmission from parent to child - not of abnormal thoughts but of the morbid brain tissue itself in which the thoughts originate. In like manner, the drunkard does not transmit to his offspring the craving for alcohol, but the abnormal organic tissue which gives rise to the craving.

In the 1920s, Dr. E.W. Adams, secretary to the Rolleston Committee, expressed the full implication of addiction, including tolerance, the abstinence syndrome and relapse (Berridge, 1990):

> ... five cardinal points by which we may recognize the presence of true addiction. First, the masterfulness of the drug used; second, the determining motive of shelter from reality, or the euphoric urge; third, the emergence of an imperious need or craving; fourth, the establishment of tolerance; and fifth, the occurrence of the abstinence syndrome.

Medical Acceptance of Alcoholism as a Disease

However, attempts to promote medical and psychological views about the aetiology of alcoholism only began when the concept of alcoholism as a disease was first acknowledged as a medical problem by the World Health Organization (WHO) in 1951. A further five years passed before the American Medical Association (AMA) accepted alcohol as an illness, in 1956. The AMA definition emphasized biochemical factors in diagnosis of addiction and viewed addiction as a physical craving that has psychological factors. The AMA followed the WHO in defining alcoholism and drug addiction under the term "dependence" (Berridge, 1990).

In 1960, Dr. E. M. Jellinek 's book *The Disease Concept of Alcoholism* was controversial among scientists and treatment professionals, and had a profound effect

on how alcoholism was viewed by the medical profession. Jellinek (1960) identified three contributing factors in alcoholism: first, a constitutional liability factor, specifically, the alcoholic's biochemical sensitivity to alcohol; second, a personality or psychological factor, such as the alcoholic's feelings of inadequacy or inferiority; and, third, social factors such as the cultural acceptability of the consumption of alcohol which allows alcoholics to relieve their stress by drinking (Begleiter & Platz, 1972).

The American Psychiatric Association (APA) first recognized alcoholism as a disease in 1965. Also in 1965, the Brain Committee adopted the following definition of alcoholism (Royce, 1981):

> ... addiction is after all a socially infectious condition and its notification may offer a means of epidemiological assessment and control. We use the term deliberately to reflect certain principles which we regard as important, viz., that the addict is a sick person and that addiction is a disease which if allowed to spread will become a menace to the community.

In 1967, the American Medical Association (AMA) presented the following definition of alcoholism: (Royce, 1989)

> Alcoholism is an illness characterized by preoccupation with alcohol and loss of control over its consumption such as to lead usually to intoxication if drinking is begun; by chronicity; by progression; and by tendency toward relapse. It is typically associated with physical disability and impaired emotional, occupational, and/or social adjustments as a direct consequence of persistent and excessive use.

Following the AMA's recognition of alcoholism as a disease, the National Institute of Alcoholism Abuse (NIAA) was formed in 1970 in the United States by the Hughes Act (Royce, 1989). The concept of the alcohol dependence syndrome arose from political motives to uphold the power of the medical profession, as stated by Shaw in 1979 (Berridge, 1990):

> ...the syndrome idea was not simply an attempt to find a substitute for the concept of alcoholism; rather it was an attempt to create a particular kind of substitute concept - one which coped with all the critiques of the disease theory of alcoholism, yet which retained all its major assumptions and implications.

Disease Effects of Alcohol

Studies of the acute effects of alcohol on psychomotor and cognitive functions occurred as early as the mid-1800s (Blane & Leonard, 1987). Alcohol is unique among psychoactive substances in having the capacity to cause widespread tissue damage and is one of the commonest causes of morbidity and mortality in the developed world (Saunders, 1991). Alcohol was classified as a sedative-hypnotic drug (Lee & Becker, 1987) and as a cerebral depressant for its ability to dampen the central nervous system (CNS) while producing relatively little analgesic effect when taken in low to moderate doses (Anthelli and Schuckit, 1991).

Liver Disease and Ethanol

Ethanol (ethyl alcohol) is the commonest form of alcohol used for beverages at the present time. Other forms of alcohol sometimes used by chronic alcoholics include methanol (methyl alcohol) and ethylene glycol (Lee & Becker, 1987). Chronic abuse of ethanol has been found to lead to a variety of serious disease states, the most common of which is alcohol induced liver disease (ALD). Most of what is known about the etiology of alcoholism is based on rates of cirrhosis mortality (Helzer & Burman, 1991). Despite the clear association between ethanol abuse and ALD and despite recent advances in the past decades in research focusing on the hepatic (relating to the liver) effects of ethanol, the precise molecular action(s) of ethanol and/or its metabolites leading to ALD remain unclear (Sultanos and Soranno, 1989).

Demographics of Alcoholism in the United States (General)

Recent estimates indicated that 17%-30% of heavy drinkers will develop ALD. Cirrhosis is the end stage of ALD and in 1985 was reported to be the eighth leading cause of death in the United States, and the fourth leading cause of death in males between the ages of 35 to 54 (Galambos, 1985). The 1994 report on *Treatment for Alcohol and Other Drug Abuse,* released by the US Department of Health and Services, indicated 80% of crimes in the U.S. were substance abuse related. The report also indicated that 78.9% of Native Americans in prisons in 1991 were alcoholics. In the general population it was found that over 3 million Americans were addicted to narcotics such as heroine (1 million) and cocaine (2 million). Approximately 5.5 million indicated they smoked marijuana more than once a week. The report further showed that over 11 million people abused prescription drugs such as tranquillizers and psychotropics, and over 18 million people abused or were addicted to alcohol (Crowe & Reeves, 1993).

Demographics of Alcoholism in Canada (General)

Reports from Statistics Canada indicated that alcohol consumption rose by approximately 33% across the nation between 1970 and 1978. The consumption of wine increased a further 27% from 1978 to 1988 (Cohen, 1989). Medical statistics on alcoholism during this period indicated that approximately 40% of alcoholics treated in hospitals had comorbidity diagnosis of affective disorders, 50% had related psychiatric disturbances, 18% had major depression, 6% had bipolar or cylcothymic disorders, and 27% had generalized anxiety disorder (Harinath, 1989).

Demographics of Alcoholism with Canadian Aboriginal Peoples

Recent research on reported demographics of Aboriginal peoples living on reserves in Canada indicated that mortality and health problems related to alcohol consumption has been difficult to estimate accurately. Mao, Moloughney, Semenciw and Morrison (1992) cautioned that while mortality data on Canadian Aboriginal peoples is in demand for purposes of health care planning, resource allocation, and research, much of the available data has limitations due to poor reporting procedures. The Department of Indian and Northern Affairs Canada (INAC) has been the major government agency responsible for obtaining data on Canada's Aboriginal peoples. A major problem exists with the validity of the statistical figures obtained from Canadian government reporting procedures, because the reporting of deaths has been voluntary for Canada's Aboriginal population living on reserves. Hence, the death rate may be underestimated because reserve funding is based on the number of band members and reports of deaths result in a loss of finances to band councils, therefore deaths on reserves often went unreported (Mao et al 1992). This fact was further complicated because the cause of death was seldom reported with mortality rates. The authors, Mao et al., emphasized that although the rates from these reports may not be accurate, because of the poor reporting procedures, the available data are considered to provide a reasonable estimate for the time period these reports represent. Also, it should be noted that from 1857 to 1951 Canadian Indians were required to enfranchise their legal title to "status Indian" in order to live off their reserves and to be allowed to drink alcohol or liquor in public, along with other general rights of citizenship (Brizinski, 1989). Therefore, the data that were collected by INAC only reflects Status Indian

peoples living on reserves and does not include non-Status Indian or Metis peoples, or Status peoples or enfranchised Indians living off-reserve. Therefore the data does not reflect all Canadian "Aboriginal" peoples.

Estimates on ALD mortality rates. Mao et al. (1992) estimated the mortality rates by cause of death for Status Indians living on-reserve. These estimates were based upon rates calculated from reports from Indian Reserves and INAC records for the years 1979-1988 in the provinces of Quebec, Ontario, Manitoba, Saskatchewan and Alberta, and 1986-1988 for the province of British Columbia. Their report indicates that the age-standardized mortality rate of male Status Indians due to alcoholism-cirrhosis, or ALD, was 33.2/100,000 (the fourth leading cause of death for this group) as compared to 14.2/100,000 in the general male Canadian population (also the fourth leading cause of death). The alcoholism-cirrhosis rate for female Status Indians was 29.8/100,000 and represented the leading cause of death for this group, while the rate for the general female Canadian population was 5.5/100,000 (representing the fifth leading cause of death for this group).

MSB alcohol related mortality estimates. Since 1962 the Medical Services Branch (MSB) of Health and Welfare Canada has been responsible for providing health services to Status Indians living on reserves and further collects information on deaths on reserves. In 1992 the MSB reported on Aboriginal health in Canada and concluded that from 1978 to 1990 the age-standardized death rate of the Status Indian population dropped from being 4.6 times greater than the rate of the general population to 1.7 times greater. This report also indicated that alcoholism was a significant contributor to Status Indian mortality caused by ALD, motor vehicle

accidents, suicide and violence, as well as contributing to depression (Health and Welfare Canada, 1992).

Theories of Alcoholism

In attempt to understand the underlying nature of alcoholism researchers have presented various theories based on biological, personality, social, and behavioral models. The following represents a short review of ten theories on alcoholism as discussed by Blane and Leonard (1987). This short review of theories, as cited from their book, is not meant to be an exhaustive study of the subject. Many theories have been presented and numerous books and articles have been written on this topic. Readers who would like a broader scope of the ten theories presented here are referred to the book Psychosocial Theories of Drinking and Alcoholism by Blane and Leonard (1987). The following discussion of these theories is intended to provide the reader with a general overview of some important theoretical postulations that were presented prior to the discovery of neurofeedback methods for the treatment of alcoholism. The theories discussed are then presented as one integrated theoretical view of underlying factors reflective of Canadian Aboriginal alcoholics.

Psychodynamic Approaches

Traditional approaches to understanding personality factors in alcohol use and abuse have their roots in classical psychoanalysis. According to psychodynamic theory, the "etiology of alcoholism" can be found in unresolved and unconscious conflicts that originated during childhood. Recent psychodynamic approaches lean toward a theory of narcissistic conflicts that alcoholics and other drug abusers experience. Advocates of the psychodynamic approach have commonly used projective

test such as the Rorschach Ink Blot Test, the Thematic Apperception Test, and the Frank Drawing Completion Test, for measuring unconscious needs and impulses. More recently, researchers studying intrapsychic processes associated with the use of alcohol have sampled other aspects of subjects' fantasies with the Thought Sampling Questionnaire (Klinger & Cox, 1986, as cited in Cox, 1987). However, the Minnesota Multiphasic Personality Inventory (MMPI) has been the single most used instrument for the multidimensional clinical assessment of personality problems associated with addictions in general and specifically with people suffering with alcoholism (Cox, 1987).

Personality Theory

A wide range of personality factors have been pursued in the search for the elusive personality of alcoholics, including; non-conformity, impulsivity, negative affect and self-esteem, cognitive-perceptual styles, and motivational determinants. Personality theories on alcoholics include dependency theories such as presented by McCord and McCord (1960) and Blane (1968), and power theory as described by David McClelland and his associates at Harvard (McClelland, Davis, Kalin, and Wanner, 1972). Their theories suggest that drinking men are often independent and aggressive, and have a need to feel powerful because of inadequate skills associated with socialization. Drinking, therefore, is seen as an activity that may help them satisfy their need for power by reducing inhibitions. The theory of "womanliness" described by Wilsnack (1974) indicates that alcoholic women have chronic doubts about their adequacy as women. Both the power theory and the theory of womanliness were

developed from analysis of subjects' responses to the Thematic Apperception Test (Cox, 1987).

Opponent Process Theory

Opponent Process Theory (OPT) was first described in the early 1970s as a theory of acquired motivational phenomena, including addictive behaviours (Solomon & Corbit, 1973a, 1973b; Hoffman & Solomon 1974). The theory assumes that the brains of all mammals are organized to oppose or suppress many types of emotional arousal and hedonic processes, regardless whether they have been generated by positive or negative reinforcers. The opposing affect of hedonic processes are automatically set in motion, by those stimulus patterns that psychologists or ethnologists have shown through defining experiments to function as Pavlovian unconditioned stimulus (UCS), operant reinforcers, or innate releasers (Solomon, 1980). Applied to alcohol: alcohol is the UCS, which when drunk elicits an unconditioned response (UCR).

As applied to alcohol abuse the OPT theory is basically a classical conditioning approach. This approach holds that the intake of alcohol has a direct effect on physiological processes. Accordingly, this has an effect that is counteracted by a homeostatic rebound mechanism that has physiological effects opposite to those of alcohol. However, this rebound mechanism overcorrects, thereby leading to a "failure of equilibrium." According to the theory, this rebound mechanism becomes stronger with repetition and diminishes the effect of alcohol such that the individual requires more alcohol than before to achieve the same effect. Addiction to alcohol occurs when the individual begins to drink to alleviate this conditioned homeostasis response (Solomon, 1980).

Social Learning Theory

Social Learning Theory (SLT) as applied to alcoholism has its roots in SLT as first expressed by Bandura (1969) and can be considered an interactionist theory (Abrams and Niaura, 1987). However, in contrast with earlier views that assume that behaviour is a product of the two-way interaction between personal dispositions and situations, SLT points to personal factors, environmental factors, and individual behaviour as interlocking determinants of each other. Causality is, therefore, multi-dimensional among these factors. The SLT model of reciprocal causal function is termed "reciprocal determinism" and states that the individual is both an agent and a recipient of behaviour patterns.

The SLT model is different from traditional behaviour theory which states that learning occurs by experiencing the effects of behaviour or the repeated pairing of stimuli and responses. Bandura (1969) stated, "alcoholics are people who have acquired through differential reinforcement and modelling experiences alcohol consumption as a widely generalized dominant response to aversive stimulation." The common behavioural approach toward alcoholism is characterised by emphasis on recording at the time of admission; the individual alcoholic's level of addiction, their pattern of alcohol consumption, their socio-economic circumstances, and personal consequences experienced when drinking. The behavioural approach further emphasizes to maintain records of the individual's experiences throughout treatment, and during their period of abstinence, as reported at the time of follow-up (Wilkinson, & Sanchez-Craig, 1991).

The SLT model presumes that the alcoholic develops a habituation to alcohol as an integral part of psychological development and socialization. Predisposing individual factors influence patterns of alcohol consumption. Alcohol becomes an increasingly important negative reinforcement in enhancing social interaction. Alcohol addiction affects the individual's capacity to cope effectively by using alcohol as a coping mechanism. Predisposing factors further affect the individual's acquired tolerance, avoidance of withdrawal symptoms, and dependence on alcohol to provide euphoria.

SLT postulates that there is no single genetic marker of alcoholic personality, nor is there a single environmental stressor. Further, there are no clearly defined "stages" of alcoholism. Recovery is dependent on learning alternative ways of coping (Abrams and Niaura, 1987). SLT assumes that all drinking from incidental social use through alcohol abuse and alcoholism is governed by similar principles of learning, cognition, and reinforcement. Abrams and Niaura explained that SLT emphasizes that these principles are developmentally arranged through psychosocial and cultural predisposing influences (Abrams and Niaura, 1987).

Expectancy Theory

Expectancy Theory (ET) centralizes the importance of cognitive factors in the initiation and maintenance of drinking behaviours. In its simplest form, ET "refers to the anticipation of a systemic relationship between events or objects" (Goldman, Brown & Chrstiansen, 1987). Expectancies are specific attitudes formed and modified by previous experiences (Shapiro & Morris, 1978, as cited in Blane & Leonard, 1987). Although there is no clearly agreed upon usage of the term expectancy, it

typically refers to intervening cognitive variables, including: information, encodings, schemas, and scripts. Applied in its simplest form to alcoholics, ET suggests that individuals consume alcohol because they believe desired outcomes are only available if they behave in this way in this context (Goldman et al., 1987).

Tension Reduction Theory

Tension Reduction Theory (TRT) was developed from the Drive Reduction Theory (DRT) of the 1940s in an attempt to understand how alcohol is reinforcing. A basic tenet of TRT states: the subordination of the individual to society generates tension; tension is painful and demands relief; this in turn creates two problems: the problem of elimination of reduction conditions which create tension, and the problem of finding a mode for relief of tension (from Jellinek, 1945, in Cappel and Greely, 1987).

Stress Response Dampening

Stress Response Dampening (SRD) is closely aligned with the reduction hypothesis and focuses upon alcohol's effects on the individual when stressed. Alcohol dampens the physiological response subjectively alleviating stress and thereby reinforcing drinking in similar stressful situations. This model considers both the psychopharmacological approach examining the physiological effects of alcohol and its relationship to other drugs, and the non-pharmacological cognitive effects (i.e. expectancies) and individual differences in sensitivity to SRD. SRD as applied to alcoholics does not follow the original premise of Tension Reduction Hypothesis (TRH) as proposed by Conger (1956) when he hypothesised that alcohol consumption is reinforced because of drive-reducing propensities.

Many reviews of SRD have found that the literature on alcohol and stress is confusing and inconclusive. Although, alcohol appears to dampen the stress response, alcohol consumption can lead to a variety of negative consequences and at times is stress inducing. Stress related drinking is determined by a variety of psychological and social factors especially the perceived ability to cope effectively with the stressor through alcohol. The continuation of drinking therefore appears to be related to contributing factors that go far beyond the stress reducing pharmacological effects (Sher, 1987).

Self-Awareness Model

The Self-Awareness Model (SAM) was presented by Hull in 1987. The model takes as a general assumption that alcohol has effects on behaviour because of disruptive cognitive processes. Central processes of cognition are impaired by intoxication. Alcohol has been found to negatively affect information input more than information retrieval and memory. The hypothesis suggests that alcohol impairs information processing by inhibiting the use of the brain's organizing strategies.

SAM, therefore, proposes that alcoholics are less self-aware of their behaviours because of the inhibition upon information processing and memory due to intoxication. It appears that alcohol has the effect of eliminating the self-conscious contribution to inappropriate social behaviour (Hull & Young, 1983a). SAM posits that alcohol affects cognitive processes, specifically the self-awareness state, rather than the physiological stress response. The four basic proportions of SAM are as follows (Hull & Young, 1983b)

1. Alcohol decreases self-awareness.

2. It does so by inhibiting higher-order cognitive processes related to the encoding of information in terms of its self-relevance.

3. By inhibiting these encoding processes and thus decreasing the individual's sensitivity to self-relevant information, alcohol consumption imposes the opposite affective and behavioural consequences of manipulations that normally increase self-awareness. It therefore: (a) decreases the correspondence of behaviour with external and internal standards of appropriate conduct, and (b) decreases self-evaluation based on past performances.

4. The fact that alcohol decreases negative self-evaluation following failure is a sufficient condition to induce and sustain alcohol consumption.

Hull (1987a) concludes that the SAM provides a useful framework within which to understand some of the personal causes and effects of alcohol consumption.

Self-Handicapping Model

The Self-Handicapping Model (SHM) has its origins in the theories of attribution and impression management. An important feature of the SHM is that it addresses a major gap in the knowledge of alcoholism in explaining alcohol abuse amongst successful individuals. SHM asserts that successful individuals are able to maintain a positive competence image by controlling the attributions drawn from their behaviour. If failure occurs under the use of alcohol then the individual's competence is not assailed since poor performance is charged to alcohol. The SHM uses many tactics for self-image protection. Anxiety is aroused by poor performance which has a direct bearing on an individual's competence image. This situation is both unpleasant and typically self-defeating (Spence & Spence, 1966). When the level of anxiety is

elevated to where image competence performance becomes hampered, the individual consumes alcohol. Alcohol then becomes the object of blame for poor performance.

Self-handicapping therefore serves two distinct functions: it reduces performance anxiety derived from evaluative interactions; and it provides concomitant esteem-maintaining defences. Self-handicapping has been found to exist to a greater extent with males than with females (Berglas, 1987).

Interactional Theory

Interactional theory (Ekehammer, 1974; Endler & Magnusson, 1976) attempts to explain the extent to which behaviour is consistent across structures or is specific to situations. Sadawa (1987) argued there was a need to examine the interactions between various theoretical models, especially the behavioural and developmental models. Interactional theory thus is more of a model than a theory (Endler & Magnusson, 1983, as cited in Cox, 1987)) which includes both the person and the environment, as well as attempts to delineate interrelationships between aspects of person and environment (Cox, 1987).

An Integrated Theoretical View of Canadian Aboriginal Alcoholism

Applying interactional theory to the other theories discussed, it could be hypothesized that Canadian Aboriginal alcoholics have developed alcoholism due to unresolved conflicts of childhood (psychoanalytic theory), and inadequate skills associated with socialization in mainstream society (personality theory). Further, they have developed a conditioned response to alcohol consumption for reducing emotional stress (operant theory), and have acquired learned responses in using alcohol to overcome aversive social situations (social learning theory). These behavioral

responses are further integrated with certain desired outcomes that are only available when consuming alcohol (tension reduction theory). Any following inappropriate social behaviors are not fully realized because of the reduction of self-conscious processing brought about by the intoxicating effect of alcohol (tension reduction theory). Poor performance can be blamed on alcohol and not on the self (self-handicapping model). Finally, alcohol is continued to be used in order to reduce associated stress (stress response dampening).

Genetic Predisposition to Alcoholism

Although the various theories have presented interesting insights on alcoholism an underlying cause has not been found. Knowledge of human genetics has provided a newer tool for helping to find causes of diseases. In recent years genetic researchers have searched for evidence of genetic predisposition to alcoholism. In 1991, Blum presented an overview of evidence of possible genetic indicators for alcoholism from his research of studies that were conducted in a number of countries. The following review, cited from Blum's studies of biogenetic research, is presented to show the important findings that are providing evidence of possible genetic marker(s) for indicating an individual's higher risk to alcoholism.

Alcoholic Children of Alcoholic Parents

Research findings on the possibility of genetic predisposition to alcoholism were presented, by Blum (1991), from previous studies conducted with adopted children of alcoholic parents. In 1972, Schuckit, Goodwin, and Winokur at the Washington University in St. Louis, USA, found that subjects reared apart from their biological parents were more likely to have problems with alcohol consumption when

their biological parent was an alcoholic. Goodwin, Schulsinger, Hermansen, Guz, and Winokur (1973), from a study of 5,483 adopted males in Denmark, found the sons of alcoholic parents were three times more likely to have problems with alcohol consumption than were the sons of non-alcoholic parents. A 1978 study, by Bohman at the Umea University in Sweden, found from a sample of 1,125 adopted males that the sons of alcoholic fathers were three times more likely to have problems with alcohol consumption than were the sons of non-alcoholic fathers, and the sons of alcoholic mothers were twice as likely to have problems with alcohol consumption (Blum, 1991).

Types of Alcoholics

Cloninger, Bohman, and Sgvardsson (1981), from a study of 862 adopted males and 913 adopted females, reported that 22.8% of the sons of alcoholic biological fathers became alcoholics compared to 14.7% from non-alcoholic fathers, and 28.1% of the males whose biological mothers were alcoholic also became alcoholics, compared to 14.7% whose biological mothers were non-alcoholic. The study further found that daughters of alcoholic mothers were 10.8% more likely to become alcoholics compared to 2.8% from non-alcoholic mothers. The study also found that the adoptive environment was not a determining factor even when an adopted parent was an alcoholic. The study further identified two distinct types of alcoholism: Type I, identifies both genetic predisposition due to parental use of alcohol and environmental influences, specifically where parents were in unskilled occupations, as the most common form of alcoholism for both males and females; Type II, identifies males who were adopted from alcoholic parents, are nine times more likely to become alcoholic

regardless of environmental factors; Type III, identifies alcoholics with anti-social personality disorders, percentages not inferred.

Genetic research has also looked for brain electrical scalp site EEG markers which show a causal relationship with alcoholism. Therefore alcoholism would be detectable from EEG recordings.

P3 Scalp Site a Possible Identifier

The P3 scalp electrode site was found to be a possible genetic marker of a predisposition to alcoholism in boys, from studies by Begleiter, Porjesz, Bihari, and Kissin (1984) at the New York State University in Brooklyn, and by Whipple, Parker and Noble (1988), at the University of California in Los Angeles. Their studies found in general there was a low magnitude of electrical activity at the P3 site, and may indicate possible risk toward alcoholism (Blum, 1991).

The Alcoholic Gene

Assuming that alcoholism is a genetically definable disease, genetic researchers believe that an alcohol-gene (Alogene) should be identifiable from the 3,000 known disease genes amongst the 100,000, or more, genes found in the human genetic chain (Blum, 1991a). In 1989, Blum reported that research had discovered a possible genetic marker referred to as "A1 allele." In 1990, Blum et al determined from small samples that other genes as well as nongenetic factors may also be involved in alcoholism, and that more research is needed before present genetic findings can be used as a diagnostic tool for identifying predisposition to the risk of alcoholism. Although one genetic study indicated that 85% of North American Indian alcoholics from the south-western region of the USA appeared to show gentic markers, Blum

(1991) also concluded that some forms of alcoholism may develop without a genetic contribution.

Treatment

Regardless of the underlying reasons for consuming alcohol, some form of treatment is required for the elimination or reduction of alcohol addiction. Treatment programs for alcoholism began in 1935 when a former alcoholic stockbroker, Bill Wilson, met an alcoholic general practice medical doctor, Dr. Robert Smith in Akron, Ohio. The two men discovered that the mutual sharing of their experiences strengthened their resolve to remain abstinent. Their mutual experience resulted in the formation of the Alcoholics Anonymous (AA) self-help group support system. The AA system emphasizes a 12 step program that must be applied for the remainder of the alcoholic's life in order to assure continued abstinence. The 12 steps and the AA traditions are described in the AA "Big Book." The emphasis of the AA program is the personal motivation of the alcoholic to get better, and his/her ability to recognize, accept, surrender and understand that alcoholism is a type of illness where continued sobriety is dependent on never consuming alcohol again (AA World Services, 1993).

Therapeutic Communities

Therapeutic communities were first developed by Maxell Jones in the 1950s, in psychiatric hospitals in Britain. Therapeutic communities for addictions in the United States began with the formation of Synanon in New York state in the early 1960s. Klein and Miller (1986) suggested that the residential therapeutic community treatment approach is most appropriate for individuals with anti-social behaviour. However, other researchers have suggested that in-patient residency in rehabilitation

centres is the best way to treat all addictions, particularly at the chronic stage. Many practitioners believe that the only effective treatment for addictions is in-patient treatment using an adaptation of the AA model in the form of a 12-Step Transformational Model (12-STM) that fosters a systemic and holistic approach.

Galizio and Maish (1985) suggested that treatment programs using a 12-STM should consider following the Minnesota Model which is based on the following 7 factors:

1. Use of drug and alcohol addiction biophysiological social disease model.
2. Use of psychiatric biopsychosocial model.
3. Treatment of the biological part of each disorder first.
4. Emphasis on the "family disease" aspects of addiction and psychiatric disorders.
5. Focus on commitment to abstinence.
6. Therapeutic progression using the 12 steps of AA.
7. Structural progress emphasizing "discipline" in recovery.

Recent Suggestions For Treatment Paradigms

Although the AA self-help group approach has helped many alcoholics, self-help groups are not found to be the answer for all alcoholics (Wells, 1991). Traditional alcoholism prevention programs utilizing treatment models such as the AA support system and aversive-conditioning were often found to be unsuccessful following a single treatment (Costello, 1975). In vivio aversive conditioning using emetic drugs (vomit producing) to evoke an unpleasant physical response when consuming alcohol was found to be moderately effective, and was also found to be associated with high relapse rates (Quinn & Henbest, 1967; Rachman & Wilson, 1981). Although aversive-

conditioning was shown to be more effective when combined with other techniques, Spiegler (1983) concluded that failure of aversive-conditioning was found when there was high resistance to all types of treatment. Lewis (1992) suggested that the abstinence-oriented recovery model of self-help programs is a treatment choice for highly motivated individuals during the early stage of addiction. However, Kaufman (1985) indicated there exists a need to treat addictions differentially and to base treatment on diversity rather than on one commonality. He concluded that alcohol problems are found to be both "systems-maintaining and systems-maintained." For example, alcohol abuse by one family member can become so central to a family's functioning that alcohol becomes the primary organizing factor in the system's structure, with the family learning to maintain its homeostasis around the individual's drinking behaviour. The family system reaches a point where it is unable to differentiate itself from the alcoholic and each member learns to react in ways that reinforce the addiction (Lewis, 1992).

Client-Centred versus Program-Centred Treatment

Ranew & Serritella (1992) believe that treatment should be client-centred rather than program-centred. They suggest that an effective treatment model should encompass multiple theories and a diversity of program and treatment strategies in order to develop individualized treatment plans. Karasen (1986) specified the need to find a relationship between specific techniques from different schools of psychotherapy to help differentiate among effective experiencing (E), cognitive Mastery (R), and Behavioural Regulation (A). He emphasized that treatment paradigms need to recognize individual differences as well as multiple causality and different levels of

analysis. Two individuals may both be labelled as alcoholic; however, they may have completely different socio-economic, familial, educational, personality, cognitive and occupational characteristics. Hence, it seems reasonable to assume that diverse treatment models are necessary (L'Abate, 1992).

L'Abate (1990) proposed there is a need for a continuum of psychological interventions based on three different recognized types of prevention: primary, secondary and tertiary. His proposed strategies distinguish three degrees of functioning/dysfunctioning: primary preventions identify non-clinical not diagnosable people at low risk; secondary preventions identify pre-clinical and diagnosable people at high risk but not critical; and tertiary preventions identify people at very high risk and diagnosed to be in a critical state.

The first major difficulty with addictions interventions is in "reality testing." How does the therapist validate the addict's perceptions and accounts without spending a significant amount of time with fabricated or distorted past and present events irrelevant to the addiction? L'Abate (1992) suggests a preferred media of treatment can be classified according to the ERAAwC model, where:

1. E = Emotionality: aligns with the existential and humanist schools which stress the importance of internal states such as, anxiety, fear, guilt, shame.
2. R = Rationality: aligns with psychodynamic and/or rational approaches such as psychoanalysis, rational-emotive therapies, reality therapies, expectancy theory and the self-handicapping model of addiction.
3. A = Activity: includes all the behavioural approaches that use classical conditioning

and reconditioning, instrumental reinforcement, and social learning interaction methods.

4. Aw= Awareness: includes Gestalt-type approaches which stress the importance of internal cues versus external cues.

5. C=Context: as stressed by the interactional schools and family therapy movement.

Detoxification

Detoxification is the first step in the treatment of alcoholics. The treatment goals are threefold: to provide symptomatic relief, to prevent withdrawal complications, and to encourage the individual to enter alcoholism treatment following withdrawal (Sellers, 1988). During detoxification, drugs are often used to treat alcohol withdrawal symptoms, to assist with the maintenance of abstinence, and to treat mental health disorders that either lead to or result from extreme drinking (Peachy, 1991).

Drug treatment for detoxification involves the basic principle of substituting another sedative-hypnotic agent for alcohol, such as benzodiazeprine (Librium or Valium), Phenobarbital (barbiturates), or paraldehyde. Benzodiazepine is also used as a detoxifying agent to help prevent alcoholic seizures (Launders, 1983). Although drugs may play a major role in the rehabilitation of the chronic alcoholic, they are adjunct to other therapies directed at the amelioration of alcohol related problems (Peachy, 1991). Therefore, drugs should be used in the context of an alcoholism treatment plan which includes all elements of therapy required by the patient (Sellers, 1988).

Non-Drug Maintenance

Non-drug treatment for health problems associated with alcoholism may include vitamin therapy such as described by Launders (1983):

1. Detoxification: Multivitamins containing zinc, taken twice a day. Zinc may ensure continual function of alcohol dehydrogenase (enzymes which catalyze oxidation-reduction reactions).
2. Wernicke-Korsakoff Syndrome (alcohol dementia): Thiamin, given intramuscularly (100 mg), and folic acid, 1 mg per day.
3. Magnesium and folate deficiency: Magnesium, 2 mL every 6 hours for 48 hours or in the form of glucoheptonate salt 15 mL 3 times per day.
4. Hypocalcemia (abnormally low levels of calcium in the blood): Calcium gluconate 7.5 mg per day.
5. Hypophosphatemia (abnormally low levels of phosphates in the blood): Potassium phosphate 20-40 mL to each litre of intravenous fluid.

Evaluating Treatment Programs

Saltzman and Norcross (1990) indicated that the variety of treatment modalities for addiction has been referred to as a "confusing and maddening morass" of psychotherapeutic interventions. Moos, Finney, and Chronkite (1990) suggested that many researchers have used a "black box" approach to evaluate treatment programs, by paying little attention to other factors that may affect alcohol abuse or addiction. Therefore, they have discovered little about the process of treatment or about ways to improve treatment. For example; researchers such as Polich, Armor and Braiker (1981) determined, from a four year follow-up study of a combination of patient and

treatment-related factors, that these factors were only able to account for approximately 4% of the variance in long term abstinence and slightly over 9% of the variance in drinking problems.

Moos et al (1990)suggested that an *"expanded biopsychosocial paradigm"* is needed to more accurately evaluate the efficacy of treatment process and treatment outcomes. Their evaluation paradigm design incorporates the following five factor method for evaluation: (A) life conditions prior to treatment, (B) client prior to treatment, (C) treatment, (D) life conditions after treatment, and (E) client during follow-up. They also emphasized that it is important for future research to determine how well recovered alcoholics function 2-10 years following treatment. Further, how are these individuals functioning psychologically, socially, and in their career field, and how stable is their course of remission and what effect does their remission have upon spouse, family, social, and career acquaintances? L'Abate (1992) suggests that a continuum of preventive interventions with a lattice-like integration of professionals and para-professionals would allow for systematic models of intervention strategies with systematic evaluation and follow-up.

Canadian Aboriginal Treatment Centre

Some of the suggestions made by Moos et al, (1990) and L'Abate (1992) for integrating preventive interventions with social paradigms were applied approximately 18 years earlier by the Aboriginal controlled alcohol and drug treatment centre located in St. Albert, Alberta. This Aboriginal administered and operated centre was founded in 1972 and is funded by the Alberta Alcohol and Drug Abuse Commission (AADAC). The centre has two separate components: the Nechi Institute, Alcohol and Drug

Education Centre for training addictions counsellors; and Poundmaker's Lodge for the treatment of addictions (Hodgson, 1992).

Nechi Institute and Poundmaker's Lodge. The Nechi Institute and Poundmaker's Lodge integrate traditional Canadian Aboriginal culture, particularly northern Cree and mid-western Aboriginal cultures, with the 12 steps of the AA program. The Medicine Wheel, which emphasizes the inter-relationship of all things, is the main cultural model that is integrated with the AA program. Traditional Aboriginal spiritual ceremonials such as sweetgrass ceremonies and sweat lodges are also integrated with the centre's augmentation of the AA model.

The Nechi Institute and Poundmaker's Lodge emphasize community awareness as integral for the addict's complete recovery. Through their combined efforts, these two centres have raised awareness of addictions and abuse problems in Aboriginal communities, from 25 participating communities in 1986 to 1165 participating communities in 1992 (Hodgson, 1992).

In 1994, Poundmaker's Lodge, in continuous effort to find effective treatment methods for people suffering with drug and alcohol abuse, was the first Canadian treatment centre to engage in co-operative research, namely this study, on the possible efficacy of neurofeedback training for the reduction of alcoholism.

Background to Neurofeedback Therapy

Neurofeedback training as a form of therapy developed out of the earlier research that focused on discoveries of the brain's electrical activity, or EEG, and the discoveries of proven feedback methods from early biofeedback studies. The integration of these two paradigms, the brain's electrical activity and biofeedback

methods, form the basis for neurofeedback training (NT). In order to understand the nature of NT it is essential to review the background on research findings of the brain's electrical properties and the findings from early biofeedback research.

Electrical-Mechanical Explanations

Origins of cerebral electrical activity having electrical-mechanical explanations of EEG recordings, such as the hypothesis that eye muscle movement and cardiac pulsations produce alpha rhythms, were presented by researchers such as Kennedy (1959), Lippold (1973), Cavonius and Estevez-Uscaryn (1974), and Castillo (1983). However, studies conducted by Hogan and Fitzpatrick (1987), with brains that were isolated from any muscle activity, found that alpha rhythms were still produced. Their study showed that electromechanical explanations on the origin and generation of EEG activity are difficult to support. Their study further indicated that biochemical sources are responsible for the electrical activity of the brain.

EEG Activity and Inhibitors of EEG Activity

Active EEG refers to the EEG that is recorded when attention to sensory stimuli produces a change in EEG amplitude. For example, Berger (1929/1969) found that the EEG of the faster beta rhythm became more active and the slower alpha rhythm was less active when consciousness was focused on a specific mental activity. Berger suggested that mental work can, therefore, be seen as an expression of the faster beta rhythm and an inhibition of the slower alpha rhythm. Hans Berger (1929/1969) first indicated that a particular sensory centre exerted a generalized inhibitory effect on the rest of the cortex during sensory stimulus and under certain circumstances can be associated with conscious mental processes. This inhibitory effect was found to

manifest with a reduction of alpha rhythm activity, suggesting that alpha is associated with passive psychological processes.

In reference to biofeedback and neurofeedback inhibition refers to behaviours that diminish feedback to the subject; whereas, activation refers to behaviours that enhance feedback to the subject (London and Schwartz, 1984).

Delta Rhythms

Following Berger's (1929/1969) use of Greek letters for identifying electrical frequencies, Walter (1937) referred to the frequency range from 0.5-4 Hz as the delta band. The lower EEG frequencies of the delta band were found to dominate during deep sleep. EEG brain wave activity recorded when subjects were at rest, was found to occur in the theta and alpha frequency ranges between 4-12 Hz. EEG activity was found to shift toward the beta frequency range above 12 Hz when subjects became excited (Berger, 1929/1969; Jasper, Solomon and Bradley, 1938).

The delta band of frequencies, or the delta rhythm, has been found to increase with emotional stimulation, such as being asked personal questions (Hoagland, Cameron, and Rubin, 1938). Also, increases in delta activity were found during word fluency tasks. However, the presence of high amplitude delta brainwave activity in the awake state is generally considered abnormal (Tucker, Dawson, Roth, and Penland, 1985).

Theta Rhythms

The Greek letter theta was assigned to the electrical brainwave activity in the 4-8 Hz frequency band. In the early 1960s, the theta rhythm was found to occur in deeper stages of meditation (Anand, China, and Singh, 1961). In 1953, Walter

reported that theta activity predominated in children between 1-6 years of age and decreases with age. He suggested that alpha waves scan for information and theta waves scan for pleasure. He further suggested there appears to be a relationship between slow theta brainwaves and performance tasks. Haulon and Royal (1977) studied the effect of theta suppression with radar control operators and determined the results of their study agreed with the conclusions of Lawrence and Johnson (1976) that the suppression of theta may be effectual in improving performance in vigilance tasks of long duration.

Winson (1990) proposed that the EEG theta rhythm occurring during the awake state can be explained as a mechanism of survival. Winson's experiment with animals found that theta brainwave activity was produced during survival behaviours, suggesting that survival stimuli activate theta frequencies.

Alpha Rhythms

The EEG frequencies in the alpha band are designated as 8-12 Hz and may record in amplitudes as high as 30-50 uV. Alpha was reported to be found in 75% of all people when awake and relaxed and increased during tasks involving reaction time (Surwillo, 1963). Other researchers such as Morgan (1965) and Brown (1974) reported that alpha rhythms can be produced by simply closing the eyes. These early studies further found alpha to be associated with a state of relaxed wakefulness that is often referred to as passive-awake. The alpha band was also found to be associated with pleasant mood experiences thus producing a state of relaxed alertness (Kamiya, 1969). However, other studies found that subjects engaged in alpha biofeedback training

reported both positive and negative subjective experiences (Cott, Pavaloski, & Goldman, 1981).

Plotkin (1976) reported that alpha activity occurs when the mind is still and awareness of perception is keen. Plotkin suggested the success in producing alpha amplitude was highly dependent upon the subjects' personal expectations. His studies determined that the individual's personal experiences from biofeedback experiments were unrelated to the level of alpha amplitude. He reported that their personal experiences were not related to alpha amplitude, but, were more related to their personal value of the method, their evaluation of pleasant feelings during the experiment, and other individual perceptions of their experience.

Relaxation was determined by Tyson and Audett (1979) to be the most common behavioural strategy for increasing alpha rhythms. Kondo, Travis, Knott, and Bean (1979) found that by using biofeedback training methods subjects were able to increase their alpha amplitude above their baseline measures at the occipital lobe. Mikuriya (1979) reported that the syncronization of right and left occipital lobe alpha rhythms appear to produce a greater degree of relaxation or quietude compared to unilateral or bilateral training. In general, a strong relationship has been reported to exist between increased alpha levels and states of reduced stress and anxiety (Tyson, 1987).

Beta Rhythms

The beta EEG activity was defined by Berger (1929/1969) as a high frequency rhythm in the range of 12-50 Hz generated at low amplitudes from 2-30 uV. This large band of beta frequencies was later separated by Jasper and Andrews (1938)

into two categories consisting of beta waves ranging from 13-30 Hz and gamma waves ranging from 30-50 Hz. This separation and the use of the term gamma is less frequently accepted or employed by researchers and is infrequent in literature (Ray, 1990). Beta rhythms from 12 Hz to 20 Hz were first recorded from the sensory motor lobe during research experiments with hungry cats trained to press a bar for a food reward. The researchers who discovered this rhythmic pattern (Roth, Sterman, & Clemente, 1967; Sterman, & Wyrwicka, 1967; Wrywicka & Sterman, 1968) referred to this range of frequencies as the Sensory Motor Rhythm (SMR) because of its location in the sensory motor cortex (Sterman, 1996).

Earlier in 1949, Jasper and Penfield reported that beta activity can be blocked by voluntary mental effort. In 1953, Walter was successful in associating increases and decreases in beta activity with emotional tension, such as occur in states of anxiety. Lindsley and Wilke (1974) suggested that beta activity was involved during the processing of positive and emotional stimuli. Vogel, Broverman and Klalber (1978) reported that reduced beta EEG activity occurred during automatized tasks such as reading color names and naming familiar objects. Beta EEG activity was reported to increase during more spatial tasks, such as embedded figures, object assembly and block design. Subjects in their study were also reported to show less beta rhythm on difficult math tasks than subjects who performed better on spatial tasks. Ray and Cole (1985) found that higher beta microvolt (uV) levels were present in the right temporal region during positive emotional stimuli, and decreased with negative emotional stimulus showing that beta rhythm was affected by tactile, auditory, and emotional stimulation. Guillon and Buchsbaum (1986) also confirmed that sensory input and

mental activity produce higher beta frequencies, and further have the effect of blocking alpha activity.

Biofeedback and Criticisms of Early Biofeedback Research

Biofeedback is a form of applied psychophysiological methods designed to provide systematic external feedback, in the form of auditory and visual signals, of a person's responses to some selected physiological state in order to effect changes to that state. Biofeedback methods rely upon the application of learning theory combined with operant conditioning and classical conditioning from behavioural therapy, as developed by early biofeedback and behavioural researchers, such as; Bandura and Walters (1963), Ullman and Krasner (1965), Wolpe (1973), and Sterman and Wyrwicka (1967).

Considerable research was conducted in the 1960s and the 1970s, in order to develop biofeedback methods for learning to control internal processes (Lubar, 1977). However, by the mid 1970s the various methods for assessing biofeedback and the reported conclusions were being questioned (Gastaut, 1975). Friedman (1976) contended that the lack of rigorous methodology limited the conclusions that could be derived from most biofeedback research projects. Hardt and Kamiya (1977) suggested that the opposing findings that were previously reported in biofeedback research were often related to vaguely defined procedures and varying approaches used in defining, detecting and measuring EEG activity.

Biofeedback a placebo. Biofeedback was also criticized as being seen as a panacea, and that it might merely be a placebo (Stroebel & Gluecck, 1973; Ruel, 1976). Passini, Watson, Dehnel, Herder and Watkin (1977) reported inconclusive

findings with alpha biofeedback and suggested the results may be merely due to a placebo effect. An 18 month follow-up of their subjects (Watson, Herder, & Passini, 1978) found that alpha biofeedback training appeared to have had an effect on subjects' anxiety. However, the results were ambiguous regarding the effect on subjects' alcohol abuse. Again they suggested the results may be due to a placebo effect. It is also interesting to note that Miller (1972) reported he and his colleagues were unable to replicate their early biofeedback experiments where animals were immobilized with curare. His later experiments did not use curare. Therefore, he suggested that the earlier results may have been due to the curare (Lynch, 1973).

However, Wyler, Lockard, Ward, and Finch (1976), in a study of five subjects engaged in an experiment with EEG operant conditioning, ruled out placebo effects when they found no EEG change in one subject, increased changes in two subjects, and decreased changes in two subjects. Kuhlman and Allison (1977) also ruled out effects. Green and Green (1978), from their studies of biofeedback at the Menninger Foundation, concluded it is difficult to have a true control group in biofeedback research. They also cautioned that it is highly unethical to use false feedback in order to create a placebo effect for research purposes.

Biofeedback: a Learned Skill

Green and Green (1978) emphasized that biofeedback is not conditioned, but rather is a learned skill. However, a previous study conducted in Germany with ten alcoholics found that, although they were able to raise their heart rates and their respiratory rates using biofeedback, they did not show learning across sessions nor with prolonged training (Cohen Kiem & Lieb, 1976). In 1981, Suter, Griffin, Smallhouse

and Whitlach concluded from a study with 14 subjects that they were able to control their EEG activity, particularly alpha, but they did not learn to control their EEG activity from session to session. Cott, Pavloski and Goldman (1981) determined that learning was not found to have taken place with subjects who were trained with the stimulus signal set below the probability of 0.50. They concluded, in order for learning to take place with biofeedback experiments, the probability of obtaining a stimulus signal has to be greater than 0.50. DeGood (1983) reported that day to day reliability of biofeedback training was found only with a combination of EMG and EEG training.

Feedback required. Yamaguchi (1980) found that subjects who were categorized as having an external locus of control, from scores obtained on a Japanese version of the Rotter Internal-External Locus of Control Scale, were able to significantly increase their alpha activity above their baseline measures. He concluded that successful alpha enhancement training, or biofeedback training, might have a cognitive component. However, in an experiment with 50 subjects and 50 controls, Gagea (1982) found that only subjects with a biofeedback signal were able to learn to increase or lower their EEG alpha levels. A study with 160 healthy males also found that control of their theta and/or alpha rhythms was contingent on receiving a related feedback signal (Chernigovskii, Markam, & Avsarkiyan, 1982).

Hemisphere EEG. Fox (1979) suggested it may be easier to learn to control alpha activity in the nondominant hemisphere. Cunningham and Murphy (1981), from a study with 24 learning disabled adolescent males, reported improvement in math skills when subjects learned to raise their EEG activity in the right hemisphere

while at the same time lowering EEG activity in the left hemisphere. In 1984, Moore reported changes in stuttering with a single subject who learned to suppress alpha in the left hemisphere. Jenkins and Moore (1985) reported EEG changes in the right and left hemispheres of subjects engaged in processing cognitive-linguistic material with the aid of a biofeedback signal. Similar EEG patterns were found with 20 male and 20 female university subjects who were all right handed; however, females were found to shift to right hemisphere dominance when producing emotionally-laden thoughts while provided with a biofeedback signal (Davidson & Schwartz, 1976).

Occipital EEG. The successful increasing of occipital alpha amplitude using feedback enhancement was demonstrated by 20 female university students (Kondo, Travis, Knott, & Bean, 1979). However, alpha feedback training at the occipital lobe was not found to be effective with 36 female college students being trained with generalized relaxation when confronted with aversive stimuli (Chisholm, DeGood, & Hartz, 1977). In contrast, 36 "normal" female university students showed greater ability to identify changes in alpha activity in the occipital lobe compared to other regions (Cincirpini, 1984). Significantly greater changes in alpha rhythm in the right hemisphere of the occipital lobe were found with eight subjects who were clinically diagnosed with anxiety disorder (Hoffman, 1980).

Learning to syncronize the right and left hemispheres of the occipital lobe was reported to produce a quietude state in over 200 subjects (Mikuriya, 1979). In 1978, DeGood and Valle reported that non-users of alcohol and tobacco were found to be able to increase their occipital alpha, however, users of these substances found this task to be more challenging. Their study suggested that individuals with poor cortical

regulatory ability might be predisposed to the negative effects of cortical activating substances such as alcohol.

Variability Due to Inattention to Signals

Mulholland, Goodman, and Boudrot (1983) cautioned that biofeedback practitioners must consider the attentiveness of the trainee in order to make more accurate decisions about the variability of the EEG recordings gathered during feedback sessions. It has been found that attentiveness to the feedback signal is shown to improve dysfunctions, whereas, inattention to the feedback signal has been shown to increase the variability in the physiological dysfunctions. They concluded that greater variability in individual EEG profiles may be associated with dysfunction; whereas, a reduction in variability is considered to be associated with improvement.

Abnormal EEG and Behavioural Disorders

In 1938, Jasper, Solomom and Bradley were successful in recording EEG activity in persons with Minimal Brain Dysfunction (MBD). Abnormal EEG levels were also recorded from children with MBD by researchers such as, Cohn and Nardini (1958), and Pavy & Metcalf (1965). In 1952, Knott, Platt, Ashby and Gottlieb reported EEG abnormalities in children with behaviour disorders and psychopathic personality disorders such as epileptogenic activity.

During the 1970s it became clear there were several different cognitive and behavioural disorders that were associated with abnormal EEG patterns, in particular: (1) Hyperkinetic Disorder (HKD), (2) Specific Learning Disabilities (LD), (3) Conduct Disorders (CD), and (4) Attention Deficit Disorder (ADD). Auditory feedback linked to recorded EEG signals was found to enhance an individual's perceived control over

his/her EEG activity, such as activating specific frequencies while inhibiting other frequencies (London & Schwartz, 1984).

Neurofeedback Training (NT) As A Therapeutic Intervention

Neurofeedback methods were developed from the earlier work with biofeedback methods that were discovered by pioneer researchers, such as Sterman and Wyrwicka (1967), Kamiya (1968), Brown (1974) and Green and Green (1977), who began EEG alpha/theta NT in the late 1960s and early 1970s. At that time NT was referred to as brainwave training or BWT. Early studies, using biofeedback methods with EEG signals in the form of brainwave training, or NT, were first successfully applied in the reduction of epileptic seizures. Studies with cats that were trained to increase SMR activity (Fairchild & Sterman, 1974; Sterman, 1976) found a change in chemically induced seizure activity. Sterman (1974) determined that increasing the SMR activity while inhibiting the lower frequencies of the theta band was successful in reducing seizure activity in four epileptics.

Following the studies with cats at the University of Los Angeles by Sterman and his colleagues (as previously cited), research by Finley (1975), Seifert and Lubar (1975), and Lubar and Bahler(1976) further determined that seizure activity in humans can be reduced by inhibiting the activity of the lower frequencies of the theta band and simultaneously increasing the activity of the SMR. In 1982, Sterman determined, from a review of a number of research studies that had been conducted by different researchers, that approximately 70% of the epileptics who received SMR training learned to reduce their seizure activity.

NT with Attention Deficit Hyperactive Children

Early ideas about children with attentional problems stated that inattentiveness was due to "wonton mischief" and was believed to be purposeful on the child's part, as suggested by Stills in 1902. In 1973, Satterfield, Lesser, Sand and Cantwell proposed that ADHD children who sought constant stimulus from sensory input did so because of their low arousal to sensory information. Their hypothesis seemed to explain the reason ADHD children responded positively to amphetamine sulphate, as was first documented by Bradley in 1937.

In 1976, Lubar and Bahler noticed improvement in attentional span and a reduction in hyperactivity with some of the epileptics who were trained to reduce their seizure activity. Consequently, he proposed that ADHD may be due to the inability of ADHD children to produce the higher beta frequencies normally found during cognitive task performance. Using NT methods, Lubar and Shouse (1976) conducted a NT case study with an 8 year 11 month old child diagnosed as hyperkinetic. Hyperkinesis was successfully reduced with NT designed to reduce theta activity and increase beta activity.

Following successful reduction of hyperkinetic activity the child was re-trained to produce theta and reduce beta activity. This procedure resulted in the recurrence of hyperkinetic behaviour. The child was again trained to reduce theta and increase beta. Again hyperkinetic activity was successfully reduced. This set of experiments served to show that the dysfunctions of ADHD could be either increased or decreased using neurofeedback training methods.

Following this pioneer study by Lubar and Shouse in 1976, research and clinical practices with ADHD children over the past twenty years have developed procedures which have proven successful in training ADHD children to reduce the symptoms of their disorders. Neurofeedback methods have, therefore, been successful in empowering ADHD children with measures of self-control over their dysfunctions.

NT and Alcoholism

Paralleling earlier beliefs about ADHD being due to uncontrolled wonton mischief, alcoholism was also thought to be due to uncontrolled wonton mischief and self indulgence (AA, 1993). This viewpoint did not change until alcoholism was finally accepted as a disease by the medical profession. However, gaining personal control over the physiological aspects of the alcohol addiction was not believed possible until biofeedback was found to be a proven method for changing physiological states (Jones & Holmes, 1976).

In 1941, Davis, Gibbs, Davis, Jetter and Trombridge, reported that alcohol consumption reduced the production of EEG activity in the 10-13 Hz range, and increases in alcohol consumption were found to produce increases in the 4-8 Hz theta activity. By 1966, research indicated that many chronic alcoholics produced lower levels of alpha activity, when not consuming alcohol, in comparison to non-alcoholics (Pollock, et al., 1983). It appeared that alcohol had a normalizing effect on the production of alpha rhythms (Propping, Kruger & Janah, 1980). Therefore, the consumption of alcohol was able to reduce the anxiety brought about by abnormally high levels of beta activity found in many alcoholics (Lipscomb, Nathan, Wilson & Abrams, 1980).

At the Menninger Institute, Green, Green, and Walters, (1970) developed an alpha/theta biofeedback technique that they used in experiments with alcoholics in the 1970s. Their alpha/theta method was combined with Autogenic Training, a form of self-hypnosis described by Schultz & Luthe (1959), and breathing exercises, preceded by finger temperature warming. In 1973, the US Navy's medical neuropsychiatric research unit at San Diego, California, initiated a study on the possible use of biofeedback in the prevention of alcoholism (Naitoh, 1973). However, a successful application of biofeedback methodology specifically for the omission of alcoholism was not reported until 1989 from studies conducted by Peniston and Kulkosy at the Veterans Administration Medical Centre in Fort Lyon, Colorado.

In 1989, Peniston and Kulkosky reported that all the EEG recordings from a sample of 15 Vietnam war veterans, diagnosed with chronic post traumatic stress disorder (PTSD) and comorbid alcohol abuse, showed low levels of alpha activity at the O1 site. They applied NT methods at the O1 site with the hope it would reduce subjects PSTD, and their associated addiction disorders. In their NT design they integrated NT with thermal biofeedback, autosuggestion, imagery, and psychotherapy techniques, similar to the methods previously described by Green, Green and Walters in 1970. From their application of this integrated technique at the O1 site, Peniston and Kulkosky reported success in helping their sample of Vietnam war veterans overcome their PTSD and associated alcohol abuse (Peniston & Kulkosky, 1989). Their sample of experimental subjects received the NT two times per day, five days per week, for a total of 26 days. The results were compared to 14 controls.

Personality Changes. From their 1988 pioneer study, Peniston and Kulkosky (1989) reported positive personality changes in the subjects treated with NT, as measured with the Minneapolis Multiphasic Personality Inventory (MMPI), the Millon Clinical Multiaxial Inventory (MCMI) and the Sixteen Personality Factor Questionnaire (16PF). Reduction in depression was recorded using the Beck Depression Inventory (BDI). Sustained abstinence of alcoholism was determined from 18 month and 36 month follow-up studies (Peniston and Kulkosky, 1991).

Contrasting Findings

In contrast to the findings reported by Peniston and Kulkosky (1989), Gurvits, Lasko, Schachter and Kuhne (1993) reported that they found no significant differences in EEG recordings between 15 controls and 27 Vietnam war veterans with PTSD and alcohol abuse. Previously in 1944, Greenblatt, Lewis, and Ferrincio found that only 5% of the alcoholics they studied had abnormal EEG patterns where alcoholism was not confounded with psychiatric or psychological factors. In 1952, Little and McAvoy reported that the EEG profiles of alcoholics showed a variety of patterns. They concluded that the poor alpha activity found in the EEG recordings of many alcoholics may be due to cerebral conditions and not due to the result of alcohol consumption.

In 1953, Funkhouser, Nagler and Walke, found, from a total of 81 alcoholics without overlapping complications, that 79.1% produced normal EEG patterns. The 20.9% who had abnormal patterns had either fast or slow EEG recordings, 53.7% had low alpha amplitude and 54.3% had high beta amplitudes. In their sample of alcoholics, 108 had psychiatric complications, 23 had allucinosis, 11

had delirium tremens, and 74 had convulsions. They concluded from their study that, although some alcoholic EEG profiles showed low alpha amplitudes and others showed high beta amplitudes, most alcoholics produced normal EEG patterns. In 1957, Delay, Verdaux, and Chanoit reported that out of 953 alcoholics only 72% had very small EEG differences. They concluded that the recorded EEG changes were constant over time and may indicate that alcohol consumption was causing organic brain damage in their sample.

A 1961 study at the New Castle State Hospital examined 645 EEG profiles from 533 chronic alcoholics. The findings revealed that only 10% of the total admissions, 601 patients, had abnormal EEG recordings and only 7.5% of the selected chronic group had abnormal EEG recordings. Gross abnormalities in these recordings were found to be related to other psychiatric factors and were not due to alcoholism. Their study concluded that uncomplicated chronic alcoholics did not have significantly different EEG profiles from normal profiles (Dryken, Grant, & White, 1961).

In 1963, Arentsen and Sindrup reported finding only a 17% difference between normal EEG profiles and the EEG profiles from 317 Danish alcoholics. He found that 32% of the alcoholics showed abnormal profiles compared to approximately 15% in the general population.

The EEG profiles of alcoholics have been found to vary greatly depending on the type of alcoholic, the severity of the disease, and how early was the onset of the addiction (Johannesson, Bergland & Ingvar, 1982). Begleiter and Platz (1972), and Volavka, Pollock, Gabrelli & Mednick (1985) also found that most abnormal EEG

patterns found with alcoholics were explained by other factors, either organicity, psychiatric or psychological.

Ehlers and Schuckit (1990) reported that a sample of 21 twenty-five year old sons of alcoholic fathers produced significantly more beta activity in the 12-20 Hz range, compared to controls with no family history of alcoholism. They concluded that an alcoholic family background might genetically modify beta production.

Frontal lobe EEG. Bauer and Hesselbrock (1993), at the University of Connecticut, found that alcoholics who were also diagnosed with antisocial personality disorder produced high levels of beta EEG activity in their frontal lobes. In 1995, Deckel, Hesselbrock and Bauer reported they found that only the EEG differences in right and left frontal lobe sites were associated with alcohol related expectancies and further suggested that "dysfunction" in the frontal lobe may be biologically determined. However, previously in 1994 Bauer reported that high beta activity at the central site of the sensorimotor cortex (CZ) was the only predictor of alcoholics who had relapsed following a treatment program.

Determining Individual Appropriateness for NT

Screening for alcoholism may determine that alcohol consumption is a problem requiring attention, but the difficulty remains in determining which alcoholic might best benefit from NT. Baseline EEG measures have not been found to reliably discriminate between low risk (LR) and high risk (HR) individuals for alcoholism (Pollock, Volvaka, Goodwin, & Mednick, 1983; Ehlers and Schukit, 1990; Cohen, Porjesz, & Begleiter, 1991; Cohen, Porjesz, & Begleiter, 1993).

Confounding Variables

A serious problem with making conclusions from the EEG recordings of alcoholics is the fact that alcoholics frequently use more than one substance. Studies on the effects of psychoactive drugs on EEG activity have shown considerable variability in outcomes. Although studies have found conclusive evidence of the effects of psychoactive substances on EEG activity the conclusions show that the EEG is definitely altered. For example, caffeine has been shown to increase both alpha and beta uV levels, also tobacco smoking has been found to increase both alpha and beta uV levels, but mostly alpha, cocaine has been found to increase beta uV levels and decreases theta uV levels, cannabis increases alpha uV levels and decreases beta uV levels, and pharmaceutical drugs have been found to have varying effects on EEG activity. Therefore, the abnormal levels in the EEG profiles of many alcoholics could be due to the consumption of other substances; (Knott, 1990). Other confounding variables occur when the alcoholic has overlapping psychiatric or psychological factors which alter EEG patterns (Greenblatt, Levin & Di Cori, 1944; Funkhouser, Nagler & Walke, 1953; Dryken et al., 1961; Begleiter & Platz, 1972; Volavk et al 1985).

Primary versus Secondary Alcoholics

Boeving (1993) suggested that as alpha waves increase, the brain starts to normalize, neurotransmitters start being secreted in the correct amounts, and the addict loses the craving for alcohol or drugs. Although this may be the case for many alcoholics, other research indicates some alcoholics already have high levels of alpha. In 1949, Funderburk determined from a study of 78 chronic alcoholics that 55 could be classified as primary alcoholics and 23 as secondary alcoholics. He found that primary

alcoholics are those whose addiction to alcohol is the primary problem, whereas those with confounding psychiatric or psychological factors experienced alcohol abuse as the secondary problem. Seventy-three percent of the primary alcoholics were found to produce higher levels of beta and lower levels of alpha EEG activity. Secondary alcoholics were found to produced higher levels of alpha activity. His study showed that low alpha and high beta in the EEG profile may indicate that alcoholism is the primary concern and a high alpha amplitude may indicate that other factors are the cause of alcohol abuse.

Conclusions From The Literature

Conclusions from the literature review suggest that since the introduction of alcohol into North America alcohol consumption has caused serious health and social problems for many people including those of Aboriginal ancestry. Successful treatment methods need to be found for people who suffer from alcohol addiction and abuse. The available literature on biofeedback and on the use of NT with alcoholism indicates that NT may be a plausible treatment technique for many alcoholics regardless of the cause of their addiction or associated complications. Galanter (1991) suggested the research that has been completed in this area indicates there is a need for designed longitudinal studies to be conducted before generalized conclusions can be made about alcoholics' EEG profiles. Although accurate genetic markers may be discovered, genetic predisposition does not necessarily appear to be true for all alcoholics. It is apparent there is more than one type of alcoholic; hence, more than one type of treatment will more than likely be required in order to help a larger number of alcoholics. As suggested by L'Abate (1992), there exists a need for a continuous search for

preventive interventions and an integration of professional and para-professional techniques, in order to develop systematic models for evaluation, intervention, and maintenance of life time sobriety for the people who suffer with alcohol addiction and abuse in general.

Research Questions

In the present study the following research questions were addressed:

1. Do measurable changes exist between pre and post-QEEG assessments obtained from the participants?
2. What descriptive information does the data show?
3. What benefits were reported by the participants?
4. What benefits were perceived by the observer?
5. Do measurable changes exist between participants' psychological characteristics as assessed by pre and post-psychometric instruments, and from participants' reports?
6. What do the measured and reported changes indicate?
7. Do differences exist in post-assessment EEG profiles between participants who completed neurofeedback training in comparison to participants who only completed the centre's augmented AA program?
8. What do the differences indicate?
9. What do the findings indicate for further research purposes?
10. What do the findings indicate for educational purposes?

CHAPTER THREE

METHODS

Overview

The research process for this study began with a personal interview with the Director of Poundmaker's Lodge, in 1993, who gave his consent to allow the study to be conduct in the Aboriginal operated alcohol and drug rehabilitation centre. All participants were drawn from volunteer alcoholics accepted to Poundmaker's Lodge following an explanatory presentation on NT as a method for treating alcohol abuse, that was given in a group setting. The participants were interviewed individually and initial researcher observations were recorded. Paper and pencil assessments were administered in a group setting to measure cognitive functioning, depression, and personality factors. These were followed by quantitative electroencephalography (QEEG) recordings to obtain neurometric measures from 19 scalp electrode sites, with each participant prior to the NT sessions. Qualitative data in the form of verbal responses to informal questions were collected from each participant during their NT sessions. Recordings of participants' changes in their EEG rhythms were also collected at the completion of each NT session. Final interviews were held after completion of the last NT session and post-assessment QEEG recordings were gathered during the final day of the 28 day treatment program. Follow-up telephone interviews and file reviews at Poundmakers' Lodge were then conducted in approximately three years (36 months plus two to four weeks) following the completion of NT in the rehabilitation centre.

Confidentiality

This research followed the ethical guidelines as set out by the Ethics Committee, Faculty of Education, University of Alberta for conducting research with human subjects. In order to meet the ethical requirements of confidentiality with human subjects, each participant was assigned a numerical code to prevent identification. Individual participants are therefore referred to by their assigned numerical code. In order to identify the NT participants from the AA participants and to provide some measure of gender differences, the participants are referred to as either male NT (those who completed NT), male NTD (those who dropped from NT), male AA or female AA (those who only completed the centre's augmented AA program).

Participants

All volunteer participants were drawn from the available sample of alcoholic clients admitted to Poundmaker's Lodge. One person volunteered to participate in the pilot study conducted in 1993 at the Cognitive Re-regulation Clinic, on campus at the Faculty of Education, University of Alberta. Twenty-four potential participants expressed their interest in participating in this study following a presentation on NT that was given to all clients residing in Poundmaker's Lodge in February 1994. Thirteen of the twenty-four potential participants decided to decline for various reasons. Six who were from a nearby correctional facility declined because they were required to complete the Minneapolis Multiphasic Personality Inventory-2 (MMPI-2). The common reason given for declining was out of fear that their MMPI-2 results and perhaps all assessment results including EEG recordings might be used against them by correctional and/or judicial authorities. They further expressed their concerns to the

other potential participants. Four others then decided against participating also out of their perceived threat of the possible misuse of the results from the assessments they were required to complete. Three changed their minds when they realized the daily commitment they were required to make. One declined when he saw the electrode paste, commenting that he would not be happy with sticky paste in his hair every day.

The final sample consisted of a total of eleven alcoholics of Canadian Aboriginal ancestry, six males and five females. One male pilot participant and ten participants from the main study location at Poundmaker's Lodge alcohol and drug rehabilitation centre. The study was restricted by the administrator of Poundmaker's Lodge to allow only male participants to receive NT.

Measures

The methods and instruments used for gathering qualitative information and quantitative measures during this study are described in the order of qualitative methods first and quantitative methods last. Qualitative data were obtained on information about participants' personal background from pre and post-interview sessions, reported experiences of their treatment, and perceived changes to their alcohol addiction. Quantitative data were obtained from the SILS, BDI, and MMPI-2 and from pre and post-EEG recordings, and from EEG recording results gathered during each NT session.

Interviews and Researcher's Observations

The initial interviews consisted of informal questions designed to gather information about each participant's family background, personal experiences from

childhood and adolescence, interpersonal relationships with spouses and children and personal experiences with problems associated with alcoholism.

Daily informal interviews were held with each participant during their one hour NT sessions. Data was gathered at the beginning of each session for approximately five to ten minutes prior to NT and immediately after the completion of each NT session. At the beginning of each session participants were asked to describe any subjective experiences they might have had following completion of their last NT session and that they believed were related to their NT. At the end of each session the participants were asked to describe any subjective experiences they might have had during the NT session that they attributed to the NT. The following open-ended questions were asked by the researcher:

1. What did you experience following completion of yesterday's session?
2. What did you experience during today's session?
3. How did you feel during the session?
4. How do you feel now?
5. What does this mean to you?
6. Can you describe anything else?

Final interviews were held to obtain information about each participant's cumulative experiences during the 28 day in-residence treatment at the rehabilitation centre, and also during the NT sessions for those who participated in this part of the study. During the final interviews information was obtained about participants' beliefs regarding their ability to abstain from alcohol without NT following release from the rehabilitation centre.

Psychometric Measures

Pre and post-assessment psychometric measures of participants' cognitive functioning were obtained from scores on the Shipley Institute of Living Scale (SILS), measures of depression were obtained from the Beck Depression Inventory (BDI), and measures of personality factors were obtained from the Minneapolis Multiphasic Personality Inventory-2 (MMPI-2). The pilot participant was also administered the Millon Clinical Multiphasic Inventory (MCMI) and the Sixteen Personality Factors (16 PF) as part of his pre-assessment battery.

Shipley Institute of Living Scale. The Shipley Institute of Living Scale (SILS) was developed by Walter Shipley (1939) to provide a time-effective means of assessing an individual's level of cognitive functioning. The SILS scoring system transforms raw scores into T-scores with a mean of 50 and a standard deviation of 10. The raw and transformed scores are plotted on profile sheets designed for this purpose. The scoring system provides a method for estimating equivalent measures of the Full Scale Global IQ on the Wechsler Adult Intelligence Scale from age-appropriate normative data. The scoring also provides methods for estimating Conceptual Quotient, Abstract Quotient, and Mental Age Equivalency. The instrument is considered to provide a fair measure of cognitive functioning in a reasonably short assessment time frame (Boyle, 1967). The data plotted on profile sheets was then entered into a word processing program and reported in the Results section.

Beck Depression Inventory. The Beck Depression Inventory (BDI) was designed as a 21 item self-report instrument to assess the severity of a variety of depression symptoms. The design structure of the instrument utilizes sets of questions

which describe clinically significant depression symptoms ranging from *Mild, Moderate,* to *Severe depression.* The subject chooses a numerical response on all items ranging from zero to three. The numerical value indicates the individual's perceived level of depression for that item: The items ask questions about the individual's perceived level of sadness, pessimism, sense of failure, dissatisfaction, guilt, self-dislike, self-harm, social withdrawal, indecisiveness, self-image change, work difficulty, fatigability, and anorexia. Raw scores are converted to T-scores with a mean of 50 and standard deviation of 10. Scores below 50 are within the normal range, 50-59 indicates mild to minimal depression, 60-69 indicates marked depression, and scores over 70 indicate extreme depression (Beck, et al 1961). The BDI is a commonly used instrument for measuring the severity of depression with alcoholics before and following detoxification (Clark, Gibbons, Haviland & Hendryx, 1993).

Minnesota Multiphasic Personality Inventory-2. The Minnesota Multiphasic Personality Inventory (MMPI) was originally devised by Hathaway and McKinley in 1940 and is currently the most widely used and researched psychometric instrument for providing an objective assessment of abnormal behaviours. Revised in 1989 by Butcher, Dahlstrom, Graham, and Tellegen, the current instrument, the Minnesota Multiphasic Personality Inventory-2 (MMPI-2), contains 567 true and false items that yield 10 clinical scales which assess major categories of abnormal behaviour, plus 4 validity scales for assessing the individual's test-taking attitudes. The 10 clinical scales assess: hypochondriasis, depression, hysteria, psychopathic deviate, masculinity-femininity, paranoia, psychasthenia, schizophrenia, hypomania, and social introversion. The four validity scales are: cannot say, lie, F (infrequency of responses), and K (a

correction factor). The results are plotted on a standard profile sheet indicating the clinical significance of elevated scores. Raw scores are converted to T-scores with a mean of 50 and standard deviation of 10 (Hathaway & McKinley, 1989).

A variety of MMPI-2 supplementary scales have also been developed for extrapolating more specific behaviours such as alcoholism, as measured by the MacAndrew Alcoholism Scale (MAC). The MAC tabulates the items that assess the individual's tendency toward continued alcohol habituation. The current MMPI-2 normative sample included 8,129 alcoholics: 6,512 white male adults, 409 white male adolescents, 1045 white female adults, 163 white female adolescents, and 297 black male adults. Although this sample is not necessarily representative of other ethnic groups, this instrument remains the most researched and valid instrument for assessing clinical personality characteristics in general, and, specifically, alcoholism (Green, 1991). The MAC has shown significant correlations with alcohol symptoms and alcohol dependence (Patton, Barnes & Murray, 1994).

Millon Clinical Multiaxial Inventory (MCMI). The MCMI was administered once only, and only to the pilot participant. The MCMI was designed to be administered in a short time period to assess pathological patterns from individual personality characteristics. The inventory requires a grade eight reading level and consists of 175 items which are answered as either true or false. The raw scores are converted to standard scores which are transferred to a profile sheet that provides base rate (BR) standard scores for eight personality patterns: schizoid, avoidant, dependent, histrionic, narcissistic, antisocial, compulsive, and passive-aggressive. The profile sheet also provides measures for three pathological personality disorders: schizotypal,

borderline, and paranoia. The profile sheet further shows nine clinical syndromes: anxiety, somatoform, hypomans, dysthymia, alcohol abuse, drug abuse, psychotic thinking, psychotic depression, and psychotic delusion. The validity of an individual profile is determined from two correction scales designed to determine the identification and adjustment of test-taking distortion. Clinically elevated scales showing standard scores over 74 are interpreted as showing the *presence of pathology*, and scores over 84 indicate the *prominence of pathology* (Millon, 1983).

Sixteen Personality Factor Questionnaire (16PF). The 16PF was also administered only once, and only to the pilot participant. The 16PF was designed to provide a complete coverage of personality in a brief time. The instrument measures 16 independent dimensions of personality and eight broader traits or secondary dimensions. The 16 dimensions or scales are independent of each other, with low correlations between each dimension. Low scores and high scores differentiate between polar opposites of the same dimension. The six dimensions or factors are identified by the letters of the alphabet from A to Q. The letter Q is further subdivided into Q1, Q2, Q3 and Q4. The letters identify the following corresponding personality factors listed in sequential order: reserved/outgoing, dull/bright, lower ego strength/higher ego strength, submissiveness/dominance, sober/happy-go-lucky, expedient/conscientious, shy/venturesome, tough-minded/tender-minded, trusting/suspicious, practical/imaginative, forthright/astute, self-assured/apprehensive, conservative/experimenting, group dependent/self-sufficient, undisciplined/controlled, relaxed/tense. The eight secondary dimensions or factors are: extroversion/introversion, low anxiety/high anxiety, sensitive/tough poise,

dependent/independent. The 16PF is a commonly used instrument for obtaining information about individual personality characteristics and is used in a wide range of settings such as school and vocational counselling, industrial and clinical psychology (Cattell, Embree, Tatsuoka, 1970).

Neurometric Measures

The neurofeedback equipment and computer software for clinical applications of EEG recordings were designed by Lexicor Medical Technology Corporation, Boulder, Colorado. Neurometric measures were taken with the Lexicor Neurosearch 24 computer system (NRS-24, 1991). The Lexicor system uses the International 10/20 electrode placement system of 19 active scalp sites (see Appendix I). The International 10/20 electrode system requires two ear references and one electromyography (EMG) reference, in accordance with standard procedures as determined by Jasper (1958). Full referential EEG recordings of the 19 scalp sites were collected using an electro-cap system designed by Electro-Cap International, Eaton, Ohio, USA (Electro-cap, 1990). All electrode sites were prepared with a special electrically conductive electro-gel (supplied by Electro-Cap). All impedance measures were maintained below 5 K ohms during pre and post-assessment EEG recordings, and below 10 K ohms during the neurofeedback training sessions. A linked ear reference monopolar montage was used for reference sites of the EEG recordings during NT.

Pre and Post-Dynamic EEG Assessments

Participants sat in a comfortable chair throughout the duration of the pre and post-dynamic EEG assessment periods. EEG assessments were completed during two

hour periods set aside for this purpose. The full referential EEG data gathered from each participant during the full cap dynamic EEG pre and post-assessments were collected and analysed by the Lexicor computer software and stored on a computer system. The data were gathered from six separate but sequentially assigned tasks, of five minutes duration each, for a total of thirty minutes. The purpose of these tasks was to assess participants' dynamic EEG activity while performing a set of common cognitive functions prior to and upon completion of their NT.

Assigned tasks. The six assigned five minute tasks were:

1. Eyes open: sitting quietly while concentrating on a single spot, a half inch red dot with crossed lines at its centre placed at eye level.
2. Eyes closed: sitting silently without concentrating on any particular event.
3. Reading silently: silently reading a story chosen by each participant from the rehabilitation centre's literature on alcoholism.
4. Listening silently: listening silently to material chosen by the participant and read by the researcher.
5. Drawing: copying geometric figures. The Bender Gestalt Visual Motor Test was used in order to provide a set of common figures.
6. Arithmetic: arithmetic questions were selected from the Canadian Achievement Test (CAT) battery at a level according to each participant's level of functioning.

EMG and EEG recordings. Simultaneous electromyography (EMG) recordings of muscle movements were taken during the pre and post-EEG neurometric assessments. One electrode was placed on the frontalis muscle between near the left eye. Another electrode was placed on the masseter muscle, approximately 3 cm below

the electrode on the frontalis muscle. A third electrode was placed 1 cm below the left eye to record eye movement artifact. The function of this procedure was to assist in the visual inspection of the EEG for facial and eye muscle movements which produce artifact signals that could interfere with the scalp site EEG recordings. Artifacting was conducted by visual inspection of the raw signal data. An assistant provided an second artifact check and visual comparisons were made to determine any significant discrepancies. The QEEG data is thereby considered to be artifact free.

The sampling rate for recording EEG activity was set at 128 samples per second. A high-pass filter, for allowing frequencies to pass above a selected value, was set at 0.5 Hertz (Hz). A low-pass filter, for stopping frequencies from passing beyond a set value, was set at 24 Hz. This method of filtering allowed only frequencies in the range from 0.5 Hz to 24 Hz to be collected and analyzed. The amplitude gain of raw incoming EEG signals was set at 3200.

Neurofeedback Training Sessions

Participants engaged in neurofeedback training sat in a comfortable chair throughout the duration of each NT session. The NT sessions with the non-resident pilot participant were scheduled for one hour periods five days per week, in cooperation with the staff of the Cognitive Re-regulation Clinic. The NT sessions with the participants at Poundmaker's Lodge were scheduled for one hour periods seven days a week at a pre-determined time for each participant, in agreement with the centre's counselling staff. The same time period was adhered to each day.

At the beginning of each NT session the participants were given instructions on guided imagery. Guided imagery instructions directed each participant to construct

visualizations of: alpha amplitude increase, alcohol rejection, abstinence scenes, ideal confident behaviours without consuming alcohol, and relaxed and mellow personality scenes. Although the instructions given were the same for all participants, the final visualizations were individually created by each participant.

Initial instructions were given, to each participant, explaining how to interpret the computer screen display of their brain wave activity, and of the audio feedback signal which indicated when they were obtaining the desired EEG changes. Following these instructions and instructions in guided visualizations of the desired scenes, which lasted approximately ten minutes, the participants were left alone for the duration of the session to produce these scenes and to bring about the desired EEG changes. Each participant in Poundmaker's Lodge was given 25 to 30 consecutive EEG alpha/theta NT sessions of 30 minutes duration each. The total number of sessions varied slightly between participants due to missed sessions. The pilot participant completed 40 NT sessions of 30 minutes each.

Neurofeedback training was conducted with the EEG electrode connected to each participant's left occipital scalp site (O1), 1 cm above and 1 cm to the left of the Inion and an ear electrode for reference. Pre-training baselines of the amplitude of the participant's theta, alpha and beta rhythms were obtained at the beginning of each training session. Baseline measures were obtained at the beginning of each session for a period of one minute prior to commencing training. The training amplitude threshold was set to a level dependent on the pre-training baseline recorded for each participant. The computer training protocol was then set so that the participant's received a feedback auditory signal when their alpha brain wave activity was above the set

threshold for the alpha range. A second and different sounding auditory signal provided feedback when the participants theta activity was below the threshold set for the theta range. Threshold settings in microvolts were adjusted so that the participant received a signal approximately 50% of the training time. Threshold settings varied according to individual baselines. Auditory tones were selected by each participant according to individual preference.

The EEG raw data were collected on removable hard disks. The EEG brain wave signal was collected and analyzed by the Lexicor Neurosearch-4 system (NRS-4, 1991). The Lexicor EEG recording system relays the data to a microcomputer for analysis. The Lexicor analysis system performs a Fast Fourier Transform (FFT) on the raw data, as explained by Dummermouth and Keller (1973). See Appendix III for samples of EEG recordings obtained from the Lexicor system. The analysed data were then stored on a removable hard disk for visual data artifacting.

Procedures

Prior to commencement, approval to conduct this study, in accordance with the guidelines set down for research with human subjects, was obtained from the Ethics Committee of the Faculty of Education, University of Alberta. This study of NT methods for the reduction of alcoholism with alcoholics of Canadian Aboriginal ancestry was conducted between August 1, 1993 and March 15, 1994. In order to gain a degree of understanding about NT methods prior to conducting the main study, a pilot study with one non-resident participant was conducted in the Cognitive Re-regulation Clinic, at the University of Alberta, between August 1, 1993 and January 30, 1994. The main study was conducted with the in-residence participants in

Poundmaker's Lodge, drug and alcohol rehabilitation centre in St. Alberta, Alberta, Canada, between March 1, 1994 and February 4, 1994. This was the time period when the rehabilitation centre agreed to allow access to volunteers for this research, and it was also a time period when the Cognitive Re-regulation Clinic was able to provide the required highly specialized EEG equipment, for off campus use.

Assessment Administration Procedures

The NT training procedures and the need for the assessments were explained to the non-resident pilot participant during a single session prior to beginning NT. Informed consent forms were then signed at the end of the information session. The lengthy time period of five months with the one participant in the pilot study was due to his relapses with drinking episodes between NT sessions. Although these relapse periods extended the time frame of the pilot study beyond the expected period, valuable information on EEG changes and related subjective experiences were obtained. A second study on NT with alcoholics was being conducted by another researcher during this same time period with participants from a different rehabilitation centre. Similar problems were reported with non-resident participants. Because of difficulties with non-resident alcoholics, it was determined that the main study should be held in the controlled environment at Poundmaker's Lodge.

A group pre-information session with all participants at Poundmaker's Lodge was held during a scheduled two hour session in which the NT training procedures and the need for the assessments were explained. Informed consent forms (Appendix II) were signed by all participants. NT sessions were started on the second day and administered on a daily basis throughout the 28 day in-residence rehabilitation

period. The final assessments were then completed with each participant during the last scheduled individual appointment.

The assessments with the pilot participant were conducted separately at the Cognitive Re-regulation Clinic, University of Alberta. The pilot participant was the only participant to be administered three personality instruments during his initial assessment. The three instruments were the: (MMPI-2, the MCMI, and the 16 PF. Because of the length of time required to administer so many instruments and the time constraints of the rehabilitation centre, the MMPI-2 was the only personality instrument administered to the to the participants in the rehabilitation centre. Also, it was the only post-assessment personality instrument administered to the pilot participant. The main reason for selecting the MMPI-2 instead of the MCMI was based on the fact that the MMPI-2 has normative data which includes Native Americans whereas the MCMI only has normative data and scoring tables for Blacks, Whites and Hispanics. Although separate scoring tables were not available for Native Americans, the pilot participant was administered the MCMI during the pre-assessment in conformance with the methods of previous research studies, and to help determine a suitable assessment battery for this Aboriginal population. Hence, the participants scores were calculated using the available norms and recorded for research purposes.

All other assessments were completed at the rehabilitation centre as follows: The psychometric assessments (SILS, BDI, and MMPI-2) were group administered, so that all participants could complete these tests at the same sitting. Pre-assessments were scheduled the day before starting NT. Post-assessments were completed on the final day scheduled for completing their NT. The administration of each instrument

followed the guidelines specified by the publisher of each instrument. The assessments were distributed in the form of assessment batteries. Differences in assessment batteries were determined by individual participant's willingness to complete each instrument. One participant in the pilot study completed all assessments and NT but not the centre's modified AA program. Two participants completed the rehabilitation centre's modified AA program as well as all assessments and NT. Two participants completed all psychometric assessments and a portion of the NT prior to early termination from the rehabilitation centre. Three participants completed all assessments and the Centre's AA program, but not NT. Three participants completed the psychometric assessments but dropped from the study without completing any NT or any post-assessments.

Data Analysis and Scoring Procedures

The qualitative and quantitative data gathered were analysed and interpreted as follows:

Qualitative data. The qualitative data gathered from personal interviews and observations were collected in hand-written form and recorded separately for each participant. The information gathered was then transferred to a computer word processing program and entered into the Results section.

Psychometric data. The psychometric data, collected from the SILS, BDI , and MMPI-2, were hand scored and recorded on individual profile score sheets provided by the publisher of each instrument. Raw scores were transformed to standard scores and entered on to the appropriate profile sheets. The results from the scored profile sheets were transferred to tables which were prepared separately for this

purpose. The tabled results of the transformed scores were then entered into a word processing program and reported in the Results section.

Dynamic pre-post EEG data. The recorded raw EEG data were examined for the following conditions: eyes open, eyes closed, and reading silently. These conditions were selected based on recent research showing they have the highest reliability of the six assigned tasks previously outlined (Graap, Janzen, Norman, and Fitzsimmons, 1994). The artifacted data are reported in magnitude, measured in peak microvolts.

Prior to quantitative analysis of the recorded raw EEG signals, the numerical values of the bands were selected in a manner that would prevent overlap between the final frequency of one band and the beginning frequency of the next band. This was done in order to avoid computational errors which occur when bands are set such that the end frequency of one band is the same as the beginning frequency of the next band. The frequency bands analysed were delta (0.5-3.9 Hz), theta (4.0-7.9 Hz), alpha (8.0-11.9 Hz), beta1 (12.0-15.9 Hz), beta2 (16.0-19.9 Hz), and beta3 (20.0- 23.9 Hz).

Ten sites were examined in order to provide measures from the front to the back regions of the brain, and from the left and right hemispheres. The ten sites and their locations were: F3 and F4 in the frontal lobe, C3 and C4 in the sensory-motor cortex, P3 and P4 in the parietal lobe, T5 and T6 in the temporal lobe, and O1 and O2 in the occipital lobe.

Statistical analysis of the quantitative EEG (QEEG) data was computed by the statistical package included in the computer software of the Lexicor Neurosearch 24. The results from the computer analysis were artifacted visually to remove

abnormally high microvolt recordings assumed to be associated with electromyography (EMG) amplitudes caused by muscle movement, and from other transient sources within the cortex.

The QEEG numerical averages of each scalp electrode site were placed onto a spreadsheet of the Excel data analysis computer program developed by Grey Matter International, Inc., (1994) and distributed by Microsoft Corporation (1994). The Excel data spreadsheets were programmed to compute descriptive measures of means and standard deviations of the QEEG results. The Excel spreadsheets were also programmed to provide bar graph displays showing the differences between the pre and post-assessment QEEG results of the NT participants and the AA participants, as shown in the Results section.

CHAPTER FOUR

RESULTS

Presentation of Results

To help the reader gain a sense of how the study progressed from beginning to end, the results from this study are presented in an order that closely follows the sequence in which the information was gathered. First, interviews were held with each participant to collect demographic and personal background information about his/her problems around their alcohol addiction. The interviews were followed by pre-assessment psychometric batteries and then pre-assessment quantitative EEG (QEEG) recordings were completed. Participants' personal experiences of their NT sessions were recorded as reported during each session. At the completion of the NT sessions post-assessment QEEG recordings were completed. These were followed by post-assessment psychometric batteries and final interviews. The qualitative data gathered from participants' background and psychometric assessments are presented in the first part of this section and the QEEG data are presented in the second part. The data are presented in summary tables showing only the information that was relative to the study. The summaries are presented in the order of qualitative data first and quantitative data second.

Grouped numerical data from individual QEEG recordings of all frequency bands for the ten sites selected, and bar graphs showing pre and post-results of NT and AA, and male and female group differences are shown in Appendix IV.

Sample Gender and Ethnic Background

The sample size of 11 participants consisted of six males and five females. Ethnic background of the total sample is shown in Table 1. Three participants reported having only native background and eight reported having a mixed native and Caucasian background. Ethnic background of each individual participant and the treatment group they were in is shown in Table 2.

Table 1

Sample Gender and Ethnic Background (N=11)

Gender	Native Only	Native plus Caucasian	Totals
Male	2	4	6
Female	1	4	5

Table 2

Participants' Gender and Ethnic Background (N=11)

Participant	Native Ethnicity	Caucasian Ethnicity	NT Plus AA	AA Only
1	Cree	French		Yes
2	Cree	French		Yes
3	Stoney			Yes
4	Cree	English		Yes
5	Cree	French		Yes
6	Cree			Yes
7	Cree	English		Yes
8	Cree			Yes
9	Cree	English		Yes
10	Cree	English		Yes
11	Cree	English		Yes

Order of Presentation of Data

Demographic data on participants' family background, alcohol background and related lifestyle factors are presented first. Psychometric data on participants' cognitive functioning, depression, education, age of onset and age at the time of the study are presented second. Grouped data on the MMPI-2 scales are reported third. Data on the number of NT sessions, changes to uV levels during NT, emotional versus non-emotional experiences, ability to construct visualizations, and the ability to produce an alpha/theta cross-over during NT training are reported fourth. Data from follow-up reports on length of sobriety following release form the rehabilitation centre, reported lifestyle changes, and career motivation are reported last.

Order of participants. The results from the male alcoholics who completed neurofeedback training (male NT) and all assessments are presented first. The results from the male alcoholics who started NT but dropped after completing only a portion of NT (male NTD) are presented next. Following these are the results of the participants who only completed the centre's augmented 28 day AA program. First in sequential order are the results of the male alcoholic who only completed pre-assessments and the AA program (male AA) followed by the male alcoholic who completed the AA program and both pre and post-pre-assessments (also, male AA). Finally the results of the female alcoholics who completed the AA program (female AA) and pre and post-assessments are presented, followed by the female AA participants who also completed the AA program but only completed the pre-assessment battery (also, female AA).

Summary of Qualitative Data

The data gathered on participants' demographic background, indicating family background, lifestyle factors, level of education, and scores obtained from the assessments of their cognitive functioning, level of depression, and psychological factors, are summarized and presented as grouped data and in tabled format, as follows.

Grouped Data on Participants' Demographic Background and Lifestyle Factors

Data gathered on participant's family background and lifestyle factors are shown in Table 1. These data show that all 11 participants were raised in an environment where alcohol was consumed. Ten reported alcohol was abused in their home environment, whether with natural or adopted parents. One participant (#4 Male NT) reported alcohol was used socially by his natural parents. All participants reported childhood and adult traumatic experiences. The MMPI-2 assessment results further revealed that five participants (#1, #2, & #3 Male NT; and #7 & # 11 Female AA) were assessed with clinically significant psychological factors. Five participants (#2, #3, & #4 Male NT; #6 Male AA & # 8 Female AA) had criminal records. Eight participants had previous attempts with rehabilitation programs, (#2 & #5 Male NT, and #10 Female AA, did not have previous rehabilitation experience). All participants smoked tobacco on a daily basis. Participant #6 (Male NT) was assessed to be in the late stage of addiction, #5 (Male NT) and #11 (Female AA) were assessed in the early stage of addiction. All other participants were assessed in the middle stage of addiction, according to the rehabilitation centre's criteria for classification.

Table 1

Grouped Data on Participants' Demographic Background and Lifestyle Factors

Participant	Natural Parent Alcoholic M	F	Step-Parent Alcoholic Environment	Childhood Traumatic Experiences	Adult Traumatic Experiences	Clinical Psych. Factors	Previous Criminal Record	Previous Rehab. Attempt	Daily Smoke Tobacco	Stage Of Addic.
# 1 (Male NT)	YES	?	NO	Separation. Adopted. Emotional trauma.	Physically-Assaulted. Intra-racial discrimination. Seizures.	YES	NO	YES	YES	MID
# 2 (Male NT)	YES	?	YES	Separation & emotional abuse.	Fights. Jail. Divorce.	YES	YES	NO	YES	MID
# 3 (Male NT)	NO	YES	N/A	Physical & emotional abuse.	Fights. Marital problems.	YES	YES	YES	YES	MID
# 4 (Male NT)	Socially		N/A	Physical & emotional Abuse.	Fights. Stabbed. Family Problems.	?	YES	YES	YES	MID
# 5 (Male NT)	YES	YES	N/A	Physical & emotional abuse.	Fights. Marital problems.	?	NO	NO	YES	EARLY
# 6 (Male NT)	YES	?	NO	Physical & emotional abuse.	Family killed MVA. Manslaughter.	?	YES	YES	YES	LATE
# 7 (Female NT)	NO	YES	N/A	Physical & emotional abuse.	Emotional & sexual abuse.	YES	NO	YES	YES	MID
# 8 (Female NT)	YES	YES	YES	Psychological & sexual abuse.	Parents - killed-MVA. Divorced. Jail.	?	YES	YES	YES	MID
# 9 (Female NT)	?	YES	YES	Physical & emotional & sexual Abuse.	Prostitution. Robery. Trafficking. Hepatitis.	?	NO	YES	YES	MID
# 10 (Female NT)	YES	?	YES	Physical & emotional & sexual abuse.	Physical & emotional-abuse. Divorce.	?	NO	NO	YES	MID
# 11 (Female NT)	YES	YES	N/A	Physical & emotional abuse.	Emotional abuse. Divorce.	YES	NO	YES	YES	EARLY

M = mother, F = father, Rehab. = rehabilitation, Addic. = addiction. N/A = not available.
? = information not available, Separation = separation from natural parents, MVA = motor vehicle accident.

Grouped Data on Cognitive Functioning, Depression, Education, and Age

The data gathered from individual participants' assessments showing cognitive functioning from scores obtained on the SILS and converted to WAIS estimates, level of pre and post-depression from scores obtained on the BDI, reported grade level of education, reported age at time of assessments, reported age at onset of addiction, and the number of years of addiction were compiled into one table for ease of comparison between participants and also to show the differences between male and female participants. Group means were calculated for the total group, for male alcoholics, and for female alcoholics. Differences between the male means and the female means were also calculated. See Table 2.

Total group means (n=11). The mean age for the total group was 31.2 years, with a range of 18 years to 51 years. The mean age at the onset of addiction was 15.5 years, and the average length of addiction was 15.6 years. The mean level of global cognitive functioning according to WAIS estimates was in the *Average* range, at a Full Scale IQ (FSIQ) of 98. The mean level of depression at the beginning of the treatment program was in the *Moderate* range (8.6/39) and in the *Minimal* range (0.9/39) at the completion of the NT and the centre's augmented AA program.

Male means. The means calculated for the male group show that the average male age at the time of assessment was 31.0 years. The mean level of education for the male group was 7.8 grade years. The mean age at onset of addiction for the males was 14.2 years, and the length of addiction averaged 16.8 years. The mean level of global cognitive functioning for the male group, according to WAIS estimates was in the *Average* range (FSIQ=90.5). Only one participant's cognitive

Table 2

Cognitive Functioning, Depression, Education, Age, Age at Onset and Years of Addiction, N=11

Participant	Cognitive Functioning	Depression Pre	Depression Post	Education Grade	Age	Age Onset	Years Addiction
Males							
# 1 (male NT)	89	14	00	10	28	14	14
# 2 (male NT)	89	11	00	5	32	16	16
# 3 (male NT)	90	09	00	5	31	14	17
# 4 (male NTD)	85	11	--	6	32	14	18
# 5 (male NTD)	107	11	--	12	26	14	12
# 6 (male AA)	83	07	05	9	37	13	24
Females							
# 7 (female AA)	104	01	00	13	35	17	18
# 8 (female AA)	107	16	03	9	26	14	12
# 9 (female AA)	104	01	00	9	18	13	05
# 10 (female AA)	108	13	00	9	27	17	10
# 11 (female AA)	112	01	00	13	51	25	26
Means							
Group Mean	98.0	8.6	0.9	9.1	31..2	15.5	15.6
Male Mean	90.5	10.5	1.3	7.8	31.0	14.2	16.8
Female Mean	107.0	6.4	0.6	10.6	31.4	17.2	14.2
Difference	F+16.5	M+4.1	M+0.7	F+2.8	F+0.4	F+3.0	M+2.6

F+ = female average greater than male average, M+ = male average greater than female average.
-- indicates no assessment.

functioning, estimated 2 points below average for a FSIQ of 83, was outside of the *Average* range. The mean level of depression for the male participants at the beginning of the rehabilitation program, was in the *Moderate* range (10.5/39) and in the *Minimal* range (1.3/39) at completion of the treatment program.

Female means. The means calculated for the female group show that the mean female age, at the time of assessment, averaged 31.0 years. The mean level of education for the female group was 10.6 grade years. The mean age of onset of addiction for the females was 17.2 years, and the length of addiction averaged 14.2 years. The mean level of global cognitive functioning for the female group according to WAIS estimates was in the *Average* range (FSIQ=107). The mean level of depression for the female participants at the beginning of the rehabilitation program, was in the *Moderate* range (6.4/39) and in the *Minimal* range (0.6/39) at completion of the program.

Male/female differences. The differences between male and female means show that the female average age (31.4) was 0.4 years older than the males (31.0). The average female level of education (10.6) was 2.8 grade years higher than the male average (7.8). The mean age at the onset of addiction for the females (17.2) was 3.0 years less than the mean age of onset of addiction for the males (14.2), and the average length of addiction for the females (14.2) was 2.6 years less than the length of male addiction (16.8). The mean global cognitive functioning for the females (107) was 16.5 points higher than the males (90.5), or slightly greater than one standard deviation. The average level of depression at the beginning of treatment for the female

group (6.4) was 4.1 points lower than the male mean (10.5) and 0.7 points lower at the completion of the treatment program (male=1.3 and female=0.6).

Grouped Data on MMPI-2 Scales

The data from the participants' MMPI-2 assessments were described under "personality traits and emotional functioning" with each participants' results. The data were also placed into tables for ease of comparison, as follows:

Validity scales and Basic Clinical scales (n=5). See Table 3 showing T-scores obtained on the Validity scales and Basic Clinical scales, for the five participants who received NT.

Supplementary scales (n=5). See Table 4 showing T-scores obtained on the Supplementary scales, for the five participants who received NT.

Selected Wiener-Harmon scales, and selected Content scales (n=5).

See Table 5 showing T-scores obtained on selected Weiner-Harmon scales, for the five participants who received NT.

Table 3

Grouped Data on MMPI-2 Scales: Validity Scales and Basic Clinical Scales, N=5

	Participants									
	#1 (Male- NT)		#2 (Male - NT)		#3 (Male - NT)	#7 (Female- AA)	#11 Female - AA
MMPI-2 Scales	Pre	Post	Pre	Post	Pre	Pre	Pre
VALIDITY SCALES							
Omissions	00	00	00	00	00	00	00
L-Lie	48	61	43	48	43	52	61
F-Frequency	**99**	55	58	51	**98**	48	36
K-Correction	30	47	41	43	30	59	**70**
Fb-Back F	**75**	63	55	51	**83**	42	00
Vrin	**88**	65	54	34	30	30	00
Trin	57	57	50	50	54	50	00
BASIC SCALES							
Hs- Hypocondraisis	**104**	42	45	37	59	30	49
D-Depression	**97**	42	**68**	45	**68**	30	44
HY-Conversion Hysteria	**89**	40	54	40	54	47	54
Pd-Psychopathic Deviate	**89**	57	**77**	64	**74**	30	58
Mf-Masculine/Feminine	48	38	50	48	40	52	50
Pa-Paranoia	**116**	53	**83**	**68**	**68**	49	49
Pa-Psychasthenia	**87**	30	**70**	55	**77**	42	53
Sc-Schizophrenia	**120**	39	**67**	56	**67**	30	30
Ma-Hypomania	**78**	**68**	**69**	**69**	56	53	49
Social Introversion	**78**	44	45	49	59	59	36

Scores marked in **BOLD** indicate clinical significance (a 'T-score' of 65 or greater).

Table 4

Grouped Data on MMPI-2 Scales: Supplementary Scales, N=5

	Participants									
	#1 (Male- NT)		#2 (Male - NT)		#3 (Male - NT)	#7 (Female- AA)	#11 Female - AA
MMPI-2 Scales	Pre	Post	Pre	Post	Pre	Pre	Pre
SUPPLEMENTARY SCALES							
A-Anxiety	57	42	**75**	63	**77**	43	35
R-Repression	47	39	34	34	45	**78**	57
Es-Ego Strength	30	30	34	49	31	51	57
Mac-R Alcoholism	**75**	48	**72**	**78**	58	30	45
GM-Gender Masculine	30	51	33	62	30	55	63
GF-Gender Feminine	38	58	49	39	41	56	32
OH-Control Hostility	37	54	48	45	38	53	63
DO-Dominance	30	48	38	45	30	37	56
Re-Soc.Responsibility	30	30	30	30	30	42	59
Mt-College Maladjustment	**91**	48	**73**	54	**81**	41	35
Pk-Post Traumatic Stress	**108**	47	**73**	55	**82**	49	39
Ps-Post Traumatic Stress	**108**	48	**69**	59	**84**	56	37
Si1-Shyness	**71**	53	51	45	56	**71**	41
Si2-Social Avoidance	62	41	36	37	41	69	47
Si3-Alienation	**80**	50	**65**	59	**80**	30	30

Scores marked in **BOLD** indicate clinical significance (a 'T-score' of **65** or greater).

Table 7

Grouped Data on MMPI-2 Scales: Selected Wiener-Harmon Scales, and Selected Content Scales, N=5

	Participants									
	#1 (Male- NT)		#2 (Male - NT)		#3 (Male - NT)	#7 (Female- AA)	#11 Female - AA
MMPI-2 Scales	Pre	Post	Pre	Post	Pre	Pre	Pre
WIENER-HARMON SCALES							
DO-Depression Obvious	**111**	44	**72**	48	**81**	46	36
DS-Depression Subtle	40	52	48	44	36	53	69
HyO-Hysteria Obvious	**120**	51	59	45	**78**	46	37
HyS-Hysteria Subtle	31	38	47	43	62	51	66
PdO-Psychopath Obvious	**103**	63	**84**	71	**90**	41	47
PdS-Psychopath Subtle	52	52	56	48	48	43	78
PaO-Paranoia Obvious	**116**	75	**70**	70	**84**	47	38
PaS-Paranoia Subtle	52	43	69	52	39	52	61
MaO-Hypomania Obvious	**91**	67	67	61	64	32	35
MaS-Hypomania Subtle	50	57	61	69	64	47	44
CONTENT SCALES							
LSE-Low Self-Esteem	**93**	41	55	48	**83**	35	40
ASP-Anti Social Practice	**83**	37	53	55	**72**	35	36
WRK-Work Interference	**94**	57	**67**	52	**74**	43	34
ANG-Anger	**74**	50	**67**	59	**67**	43	31
BIZ-Bizarre Mentation	**119**	54	51	54	**67**	39	52
FAM-Family Problems	**102**	60	60	57	**68**	43	39
TRT-Treatment Indicator	**98**	43	56	49	**71**	35	35

Scores marked in **BOLD** indicate clinical significance (a 'T-score' of **65** or greater).

Grouped Data on NT Sessions

The qualitative data gathered during participants' NT sessions describing the number of sessions completed, and whether or not changes occurred in theta, alpha and beta uV levels, attention was maintained on task, emotional experiences or non-emotional recollections were reported, personal visualizations were possible, and the crossover effect occurred with theta and alpha, are listed in Table 6.

Number of NT sessions. Participant #1 completed 40 NT sessions, #2 completed 30, #3 completed 26, and participants #4 and #5 completed 7

Changes in uV levels. Only participant #2 did not produce uV increases in his theta and alpha bands. All participants produced decreases in their beta rhythms.

Attentive during sessions. Participants #1, #3, and #5 reported being attentive to the assigned task when left alone. Participant #2 usually slept and Participant #3 generally sang powwow songs.

Visualizations. Participants #1, #3 and #5 reported they were able to produce personal created visualizations during their NT sessions. Participants #2 and #4 were unable to produce their own visualizations as required by the NT method.

Crossover effect. The recordings of participants #3 and #5 showed the desired crossover effect between theta and alpha rhythms.

Emotional versus non-emotional reports. All participants gave non-emotional reports of their childhood and adult backgrounds during the informal interviews held with each NT session. Only participants #3 and #5 showed emotional expression when reporting on previous traumatic experiences.

Table 6

Data on Number of NT Sessions, Changes to uV levels, Emotional Versus Non-Emotional Reports, Visualizations, and Crossover Effect, N=5

Participant	Number of NT Sessions	Change in uV Levels			Attentive During Sessions	Produced Personal Visualizations	Alpha/Theta Crossover Effect	Emotional Laden Reverie	Non-emotional Report of Memories
		Increase		Decrease					
		Theta	Alpha	Beta					
1	40	Yes	Yes	Yes	Yes	Yes	No	No	Yes
2	30	No	No	Yes	Slept	No	No	No	Yes
3	26	Yes	Yes	Yes	Sang	Yes	Yes	Yes	Yes
4	7	Yes	Yes	Yes	Yes	No	No	No	Yes
5	7	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Grouped Data on Follow-Up Report, Sobriety, Lifestyle, and Career Motivation

The qualitative data gathered from the three year follow-up reports on length of sobriety, lifestyle changes, and career motivation are listed in Table 7.

Follow-up reports completed. Four of the female participants (7,8,10 &11) were not able to be located for follow-up reports. All six males and one female participant (9) were located for follow-up reports.

Length of sobriety. Participant #1 reported one year plus a few days of total sobriety. Participants # 2 (male NT) and #9 (female AA) reported less than one week of sobriety. Participants #3 (male NT) and #4 (male NT) reported less than six months of sobriety. Participant #5 reported less than one month of sobriety. Participant #6 (male AA) reported 3 years continuous sobriety.

Lifestyle changes. Participants #4 (male NT) and #6 (male AA) reported they were motivated to change their lifestyle to be totally free of alcohol. Participant #5 (male NTD) believed he was able to control his drinking for business/social purposes.

Career motivation. Only the male participants #1, #3, #4, #5 and #6 reported they were motivated to enhance their careers by committing to a training program or business undertaking, after completing the 28 day rehabilitation program. Four female participants were not available for comment.

Use AA support. Only participants #4, and #6 continued to use the AA support system after completing the 28 day augmented AA program at the rehabilitation centre.

Practice visualizations. Only participants #5 reported he practised the visualizations and techniques taught during NT. Participant #3 used his spirit song.

Table 9

Data From Follow-Up Reports on Length of Sobriety, Lifestyle Change, and Career Motivation, N=11

Participant	Follow-Up Report Completed	Length of Sobriety	Motivated to Change Lifestyle	Motivated to Enhance Career	Uses AA Support	Practice Daily Visuals
1	Yes	>1 year	No	Yes	No	No
2	Yes	<1 week	No	No	No	No
3	Yes	<6 months	Some	Yes	No	No
4	Yes	<1 week	No	No	Yes	No
5	Yes	<1 month	No	Yes	No	Yes
6	Yes	>3 years	Yes	Yes	Yes	n/a
7	No	?	?	?	?	n/a
8	Yes	<1 week	No	No	No	n/a
9	No	?	?	?	?	n/a
10	No	?	?	?	?	n/a
11	No	?	?	?	?	n/a

Visuals = visualizations.
? = information not available because participant was not able to be located.
n/a = not applicable, participants were not assigned visualizations (only assigned to participants in NT).

Summary of QEEG Data

The following written descriptions of the QEEG results with corresponding tables and figures are presented in an order that first provides an overall summary of the pre-assessment QEEG results as a single group showing the sample's QEEG profile. The accumulative results are presented as a sample profile to provide evidence of specific EEG scalp sites that may serve as markers indicating a predisposition for alcoholism. The first summary presentation shows the sample QEEG profile as calculated from the averaged QEEG values that were combined from all 11 participants. The sample profile shows the highest and lowest scalp site uV levels for the six frequency bands and the Total QEEG for the three conditions reported, Eyes Closed, Eyes Open, and Reading Silently. The sample summary is followed by separate summaries of the results from the three conditions showing the averaged uV values of the six frequency bands and Total QEEG. These summary presentations of the numerical data are then followed by graphical presentations showing the sample profile in the form of a scalp site map indicating highest and lowest sites and also showing hemisphere dominance. The sample profile is followed by individual profiles mapping the highest and lowest sites and hemisphere dominance for each of the 11 participants.

Overall Summary of Pre-Assessment QEEG Recordings

The overall summary of the pre-assessment QEEG recordings in Table 8 shows the sample QEEG scalp site averages as calculated from the uV levels from all 11 participants. The sample averages show the scalp sites with the highest and lowest QEEG mean values for the six bands and the three conditions assessed.

Eyes Closed. The results for the Eyes Closed condition show that the highest uV level was produced in the Alpha band at scalp electrode site P4 (7.03), and the lowest uV level was produced in the Beta3 band at site P3 (1.77). The highest level of Total QEEG activity was found at site C3 (30.22), and the lowest level of Total QEEG activity was found at scalp site T5 (21.17).

Eyes Open. The results for the Eyes Open condition show that the highest uV level was produced in the Delta band at site F3 (6.25), and the lowest uV level was produced in the Beta3 band at site P3 (2.01). The highest level of Total QEEG activity was found at site F3 (27.62), and the lowest level of Total QEEG activity was found at site T6 (18.61).

Reading Silently. The results for the Reading Silently condition show that the highest uV level was produced in the Delta band at site F4 (6.82), and the lowest uV level was produced in the Beta3 band at site P3 (2.24). The highest level of Total QEEG activity was found at site O1 (29.05), and the lowest level of Total QEEG activity was found at site T5 (23.40).

Means. The Delta band showed the highest uV level of QEEG activity (6.56) over all others and the Beta3 band showed the lowest (2.01), as averaged from all three conditions combined to show the accumulative means of the sample. Alpha band high and low averages were both higher than the high and low averages for the three beta bands.

Table 10

Sample Pre-Assessment QEEG Averages Showing Sites With Highest and Lowest QEEG Values For The Six Bands And The Three Conditions Selected, N=11

Condition	Delta		Theta		Alpha		Beta1		Beta2		Beta3		Total	
& Value	H	L	H	L	H	L	H	L	H	L	H	L	H	L
EC	F4	T5	F4	T5	P4	T5	P3	T5	O1	T5	P4	*P3*	C3	T5
Value	6.62	4.27	5.25	3.33	7.03	4.80	3.37	2.16	2.88	1.84	2.45	*1.77*	30.22	21.17
EO	F3	T5	F4	T5	C4	T5	C4	T5	C4	T5	C4	*P3*	F3	T6
Value	6.25	4.20	4.62	2.92	3.85	2.90	2.75	2.21	2.65	2.17	2.64	*2.01*	27.62	18.61
RS	F4	T5	F4	T5	C4	T5	O1	T5	O1	T5	O1	*P3*	O1	T5
Value	6.82	4.47	4.90	3.25	3.84	3.12	2.95	2.52	2.86	2.34	2.90	*2.24*	29.05	23.40
Mean	**6.56**	4.31	4.92	3.17	4.91	3.61	2.99	2.30	2.80	2.12	***2.68***	2.01	29.00	21.10
S.D.	0.29	0.14	0.32	0.22	1.84	1.04	0.33	0.20	0.13	0.25	0.25	0.24	1.30	2.40

Note: Total values include higher beta frequencies recorded but not reported.
H = highest value for site indicated, L = lowest value for site indicated.
Values in Bold (**5.04**) show band with the highest value for that site.
Values in Bold Italics (***2.83***) show band with the lowest value for that site.
Double underlined numbers (3.84) show site with highest value for that band.
Single underlined numbers (3.12) show site with lowest value for that band.

Summary of Averaged QEEG uV Values for Eyes Closed

The data listed in Table 11 show the averaged numerical values calculated for six bands and total QEEG means for the ten sites for the Eyes Closed condition. Means and standard deviations for each band and total QEEG values are also listed.

Delta band. Delta was highest at site F4 (6.62) and lowest at T5 (4.27).

Theta band. Theta was highest at site F4 (5.25) and lowest at T5 (3.33).

Alpha band. Alpha was highest at site P4 (7.03) and lowest at T5 (4.80). Alpha at site P4 was also higher than at all other sites. Alpha was also highest at all sites compared to all three beta bands.

Beta1 band. Beta 1 was highest at site P3 (3.37) and lowest at T5 (2.16)

Beta2 band. Beta2 was highest at site O1 (2.88) and lowest at T5 (1.84).

Beta3 band. Beta3 was highest at site P4 (2.45) and lowest at P3 (1.77). Beta3 at site P3 was also the lowest compared to all other sites.

Highest total QEEG. The site with the highest total QEEG activity compared to all other sites was C3 (30.22).

Lowest total QEEG. The site with the lowest total QEEG activity compared to all others was T5 (21.17).

Range. The range of the total QEEG activity was 8.01 uV (30.22-21.17).

Highest mean. Alpha showed the highest mean QEEG overall (5.68).

Lowest mean. Beta3 showed the lowest (2.17).

Table 11

Grouped Pre-assessment QEEG Averaged Data For The Six Bands And Total Values From The Ten Sites Selected For The Eyes Closed Condition, Grouped N=11

Site	Delta	Theta	Alpha	Beta1	Beta2	Beta3	Total
F3	**6.51**	5.03	5.66	2.58	2.48	***2.35***	28.49
F4	**6.62**	5.25	5.69	2.63	2.54	***2.36***	28.98
C3	**6.55**	4.89	6.20	2.72	2.56	***2.41***	30.22
C4	**6.11**	4.82	6.02	2.78	2.64	***2.43***	30.00
P3	**5.44**	4.54	5.17	3.37	2.07	***1.77***	27.70
P4	5.47	4.57	**7.03**	2.80	2.53	***2.45***	28.32
T5	4.27	3.33	**4.80**	2.16	1.84	***1.81***	21.17
T6	4.65	3.56	**5.44**	2.38	2.17	***1.95***	23.51
O1	4.59	3.73	**5.30**	2.58	2.88	***2.03***	24.78
O2	4.97	3.64	**5.45**	2.64	2.33	***2.12***	25.50
Mean	5.52	4.34	**5.68**	2.66	2.40	***2.17***	26.87
S.D.	0.89	0.70	0.62	0.31	0.31	0.27	3.00

Note: Total values include higher beta frequencies recorded but not reported.
Values in Bold **(6.51)** show band with the highest value for that site.
Values in Bold Italics ***(2.35)*** show band with the lowest value for that site.
Double underlined numbers (5.25) show site with highest value for that band.
Single underlined numbers (3.33) show site with lowest value for that band.

Summary of Averaged QEEG uV Levels for Eyes Open

The data listed in Table 12 show the averaged numerical values calculated for six bands and total QEEG means for the ten sites for the Eyes Open condition. Means and standard deviations for each band and total QEEG values are also listed.

Delta band. Delta was highest at site F3 (6.25) and lowest at T5 (4.20). Delta F3 was also the highest over all other sites.

Theta band. Theta was highest at site F4 (4.62) and lowest at T5 (2.92)

Alpha band. Alpha was highest at site C4 (3.85) and lowest at T5 (2.90).

Beta1 band. Beta 1 was highest at site C4 (2.75) and lowest at T5 (2.21).

Beta2 band. Beta2 was highest at site C4 (2.65) and lowest at P3 (2.17).

Beta3 band. Beta3 was highest at site C4 (2.63) and lowest at P3 (2.01). Beta3 was also the lowest over all other sites.

Highest total QEEG. The site with the highest total QEEG activity over all other sites was F3 (27.62).

Lowest total QEEG. The site with the lowest total QEEG activity over all others was T6 (18.61).

Range. The range of the total QEEG activity was 9.01 uV (27.62-18.61).

Highest mean. Delta showed the highest mean value QEEG overall (5.31).

Lowest mean. Beta3 showed the lowest (2.29).

Table 12

Grouped Pre-Assessment QEEG Averaged Data For The Six Bands And Total Values From The Ten Sites Selected For The Eyes Open Condition, Grouped N=11

Site	Delta	Theta	Alpha	Beta1	Beta2	Beta3	Total
F3	**6.25**	4.49	3.84	2.62	2.59	***2.44***	27.62
F4	**6.21**	4.62	3.78	2.54	2.47	***2.39***	27.12
C3	**6.14**	4.08	3.72	2.61	2.41	***2.34***	26.01
C4	**6.01**	4.05	3.85	2.75	2.65	***2.63***	27.30
P3	**5.27**	3.77	3.51	2.51	2.17	***2.01***	23.63
P4	**5.08**	3.60	3.71	2.52	2.20	***2.07***	23.75
T5	**4.20**	2.92	2.90	2.21	2.30	***2.02***	20.62
T6	**4.34**	2.97	3.37	2.35	2.27	***2.21***	18.61
O1	**4.60**	3.48	3.52	2.60	2.36	***2.34***	24.35
O2	**4.95**	3.33	3.56	2.66	***2.39***	2.40	24.96
Mean	**5.31**	3.73	3.58	2.54	2.38	***2.29***	24.40
S.D.	0.80	0.58	0.29	0.16	0.16	0.20	2.94

Note: Total values include higher beta frequencies recorded but not reported.
Values in Bold (**6.25**) show band with the highest value for that site.
Values in Bold Italics (***2.44***) show band with the lowest value for that site.
Double underlined numbers (4.62) show site with highest value for that band.
Single underlined numbers (2.92) show site with lowest value for that band.

Summary of Averaged QEEG uV Levels for Reading Silently

The data listed in Table 13 show the averaged numerical values calculated for six bands and total QEEG means for the ten sites for the Reading Silently condition. Means and standard deviations for each band and total QEEG values are also listed.

Delta band. Delta activity was highest for all ten sites and highest over all other sites at the frontal lobe site F4 (6.82). Delta was lowest at site T5 (4.47).

Theta band. Theta was highest at site F4 (4.90) and lowest at T5 (3.25).

Alpha band. Alpha was highest at site C4 (3.84) and lowest at T5 (3.12).

Beta1 band. Beta 1 was highest at site O1 (2.95) and lowest at T5 (2.52).

Beta2 band. Beta2 was highest at site O1 (2.86) and lowest at T5 (2.34).

Beta3 band. Beta3 was highest at site O1 (2.90) and lowest at P3 (2.24). Beta3 was also the lowest over all other sites except O1 (2.86), where Beta 2 was the lowest, and was the same value as Beta2 for site O2 (2.83).

Highest total QEEG. The site with the highest total QEEG activity over all other sites was O1 (29.05).

Lowest total QEEG. The site with the lowest total QEEG activity over all others was T5 (23.40).

Range. The range of the total QEEG activity was 5.65 uV (29.05-23.40).

Highest mean. Delta showed the highest mean value QEEG overall (5.60).

Lowest mean. Beta3 showed the lowest (2.45).

Table 13

Grouped Pre-Assessment QEEG Averaged Data For The Six Bands And Total Values From The Ten Sites Selected For The Reading Silently Condition, Grouped N=11

Site	Delta	Theta	Alpha	Beta1	Beta2	Beta3	Total
F3	**6.45**	4.73	3.53	2.60	2.43	***2.35***	27.58
F4	**6.82**	4.90	3.70	2.71	2.46	***2.36***	28.34
C3	**6.62**	4.35	3.81	2.71	2.48	***2.37***	27.81
C4	**6.03**	4.42	3.84	2.85	2.67	***2.51***	28.24
P3	**5.45**	3.97	3.81	2.67	2.32	***2.24***	25.70
P4	**5.47**	4.14	3.79	2.70	2.45	***2.26***	26.11
T5	**4.47**	3.25	3.12	2.52	2.34	***2.31***	23.40
T6	**4.70**	3.56	3.36	2.60	2.43	***2.37***	24.75
O1	**4.96**	4.29	3.81	2.95	***2.86***	2.90	29.05
O2	**5.04**	4.04	3.67	2.86	***2.83***	***2.83***	28.31
Mean	**5.60**	4.17	3.64	2.72	2.53	***2.45***	26.93
S.D.	0.84	0.50	0.30	0.13	0.19	0.23	1.85

Note: Total values include higher beta frequencies recorded but not reported.
Values in Bold (**6.45**) show band with the highest value for that site.
Values in Bold Italics (***2.35***) show band with the lowest value for that site.
Double underlined numbers (4.90) show site with highest value for that band.
Single underlined numbers (3.25) show site with lowest value for that band.

Grouped Summary Showing Alpha Higher Compared to Beta for Eyes Closed

Table 14 shows where alpha was found to be higher than beta for the ten sites and for the eyes closed condition, for all participants. Only one participant had at least one site that showed at least one beta band where the QEEG magnitude was higher than for the alpha band. The other ten participants showed that alpha was higher at all sites compared to all three beta bands. Higher sites and numerical values were observed from the data shown in Appendix IV. The numerical value shown in brackets indicates the amount in microvolts that beta was higher than alpha.

Participant #2 (male NT). Beta was higher at C3 (0.4), C4 (0.2), P3 (0.9), P4 (0.6), and T5 (0.4).

Beta compared to Alpha at site O1. Beta was not shown to be higher compared to alpha at site O1 for any of the participants for the eyes closed condition.

Table 14

Pre-Assessment QEEG Recordings Showing Alpha Higher or Lower Magnitude Compared to Beta for Eyes Closed, N=11

Participant	F3 | F4	C3 | C4	P3 | P4	T5 | T6	O1 | O2
#1 (male NT)	A | A	A | A	A | A	A | A	A | A
#2 (male NT)	A | A	**B** (0.4) | **B** (0.2)	**B** (0.9) | **B** (0.6)	**B** (0.4) | A	A | A
#3 (male NT)	A | A	A | A	A | A	A | A	A | A
#4 (male NT)	A | A	A | A	A | A	A | A	A | A
#5 (male NT)	A | A	A | A	A | A	A | A	A | A
#6 (male AA)	A | A	A | A	A | A	A | A	A | A
#7 (female AA)	A | A	A | A	A | A	A | A	A | A
#8 (female AA)	A | A	A | A	A | A	A | A	A | A
#9 (female AA)	A | A	A | A	A | A	A | A	A | A
#10 (female AA)	A | A	A | A	A | A	A | A	A | A
#11 (female AA)	A | A	A | A	A | A	A | A	A | A

A shows alpha higher than beta, and **B** shows beta higher than alpha. Numbers in brackets (0.1) indicate the amount in uV that beta was higher than alpha.

Grouped Summary Showing Alpha Higher Compared to Beta for Eyes Open

Table 15 shows where alpha was found to be higher than beta for the ten sites and for the eyes open condition, for all participants. Three participants had at least one site that showed at least one beta band where the QEEG magnitude was higher than for the alpha band. The other eight participants showed alpha was higher at all sites. Higher sites were observed from the data shown in tables 1 to 30. The numerical value in brackets indicates the amount in microvolts that beta was higher than alpha.

Participant #2 (male NT). Beta was higher at P3 (0.1), P4 (0.1), T5 (0.6), and T6 (0.8).

Participant #4 (male NT). Beta was higher at all sites, ranging from 0.2 to 4.4 microvolts.

Participant #8 (female AA). Beta was higher at T5 (0.1).

Beta compared to alpha at site O1. Beta was shown to be higher compared to alpha at site O1 for only one participant, participant #4 (male NT) and showed only 0.2 microvolts higher.

Table 15

Pre-Assessment QEEG Recordings Showing Alpha Higher or Lower Magnitude Compared to Beta for Eyes Open, N=11

Participant	F3 | F4	C3 | C4	P3 | P4	T5 | T6	O1 | O2
#1 (male NT)	A | A	A | A	A | A	A | A	A | A
#2 (male NT)	A | A	A | A	**B** (0.1) | **B** (0.1)	**B** (0.6) | **B** (0.8)	A | A
#3 (male NT)	A | A	A | A	A | A	A | A	A | A
#4 (male NT)	**B** (4.4) | **B** (2.7)	**B** (2.7) | **B** (4.4)	**B** (0.6) | **B** (0.6)	**B** (1.0) | **B** (0.7)	**B** (0.2) | **B** (0.4)
#5 (male NT)	A | A	A | A	A | A	A | A	A | A
#6 (male AA)	A | A	A | A	A | A	A | A	A | A
#7 (female AA)	A | A	A | A	A | A	A | A	A | A
#8 (female AA)	A | A	A | A	A | A	**B** (0.1) | A	A | A
#9 (female AA)	A | A	A | A	A | A	A | A	A | A
#10 (female AA)	A | A	A | A	A | A	A | A	A | A
#11 (female AA)	A | A	A | A	A | A	A | A	A | A

A shows alpha higher than beta, and **B** shows beta higher than alpha. Numbers in brackets (0.1) indicate the amount in uV that beta was higher than alpha.

Grouped Summary Showing Alpha Higher Compared to Beta for Reading Silently

Table 16 shows where alpha was found to be higher than beta for the ten sites and for the reading silently condition, for all participants. Five participants had at least one site that showed at least one beta band where the QEEG magnitude was higher than for the alpha band. The other six participants showed that alpha was higher at all sites. Higher sites were observed from data shown in Appendix IV. The numerical value in brackets indicates the amount in microvolts that beta was higher than alpha.

Participant #2 (male NT). Beta was higher at C3 (0.1), T5 (0.3), and T6 (0.1).

Participant #3 (male NT). Beta was higher at F3 (0.4), F4 (0.2), C3 (0.2), C4 (0.1), and O1 (0.1).

Participant #4 (male NT). Beta was higher at F3 (0.4), F4 (0.2), C3 (0.3), C4 (1.0), P4 (0.1), T5 (1.0) and T6 (0.4).

Participant #8 (female AA. Beta was higher at P3 (0.1), T5 (0.1), and O1 (0.1).

Participant #11 (female AA). Beta was higher at O1 (0.4).

Beta compared to alpha at site O1. Beta was shown to be higher compared to alpha at site O1 for only three participants, participant #3 (male NT) for 0.1 uV higher, participant #11 (female AA) for 0.1 uV higher, and participant #11 (female AA) for 0.4 uV higher.

Table 16

Pre-Assessment QEEG Recordings Showing Alpha Higher or Lower Magnitude Compared to Beta for Reading Silently, N=11

Participant	F3 | F4	C3 | C4	P3 | P4	T5 | T6	O1 | O2
#1 (male NT)	A | A	A | A	A | A	A | A	A | A
#2 (male NT)	A | A	A | A	A | A	**B** (0.3) | **B** (0.1)	A | A
#3 (male NT)	**B** (0.4) | **B** (0.2)	**B** (0.2) | **B** (0.1)	A | A	A | A	**B** (0.1) | A
#4 (male NT)	**B** (0.4) | **B** (0.2)	**B** (0.3) | **B** (0.1)	A | **B** (0.1)	**B** (1.0) | **B** (0.4)	A | A
#5 (male NT)	A | A	A | A	A | A	A | A	A | A
#6 (male AA)	A | A	A | A	A | A	A | A	A | A
#7 (female AA)	A | A	A | A	A | A	A | A	A | A
#8 (female AA)	A | A	A | A	**B** (0.1) | A	**B** (0.1) | A	**B** (0.1) | A
#9 (female AA)	A | A	A | A	A | A	A | A	A | A
#10 (female AA)	A | A	A | A	A | A	A | A	A | A
#11 (female AA)	A | A	A | A	A | A	A | A	**B** (0.4) | A

A shows alpha higher than beta, and **B** shows beta higher than alpha. Numbers in brackets (0.1) indicate the amount in uV that beta was higher than alpha.

Summary Showing QEEG Profile Maps

The grouped data showing a sample QEEG profile map are presented first to provide an overall view of scalp site and hemisphere dominance for this sample. The summary results show the probability of overall hemisphere dominance, as well as high and low scalp site locations that might serve as indicators for predisposition to alcoholism. Individual participants' QEEG profiles as calculated from all six bands for the ten electrode sites and three conditions selected, are presented last. The data are presented in the order of Eyes Closed, Eyes Open and Reading Silently.

Site Markers.

Highlighted capital letters are used in the graphical figures to show the site with the highest Total QEEG for paired sites. The capital letter H shows the higher site and bold **H** shows the site that had the highest Total QEEG compared to all others. Bold **L** shows the lowest Total QEEG value compared to all other sites. The marker B shows where beta was higher than alpha. The number zero (0) indicates no measurement was available due to equipment failure at site P3 for post-assessment only.

Site and hemisphere dominance.

Written descriptions describe the electrode sites with the highest and lowest averaged QEEG activity and hemisphere dominance for the three conditions selected. Following the written descriptions are graphical figures showing the sites with the highest and lowest QEEG activity for paired sites indicating right versus left hemisphere dominance, and also, the site with the highest and lowest activity over all others. The sample profile is presented first followed by the profiles of individual

participants, describing each participants' high and low electrode sites and indicating hemisphere dominance.

Sample Profile Showing Hemisphere and Scalp Site Dominance

Figure 1 shows the sample QEEG profile indicating where alpha is higher than beta and showing hemisphere dominance. Highest and lowest sites are marked with capital letters and with bold letter markers.

Hemisphere dominance. The right hemisphere showed higher levels of averaged QEEG activity compared to the left hemisphere for all three conditions. The right hemisphere showed eleven higher sites compared to four in the left hemisphere, but three of the four sites in the left hemisphere were highest over all other sites. Alpha was higher than beta for all sites.

Eyes Closed. Three areas (sensory/motor, parietal and occipital) of the right hemisphere showed higher levels of activity for the Eyes Closed condition. F3 in the left hemisphere of the frontal lobe was highest over all others. Alpha was highest at P4, and lowest at site T5.

Eyes Open. Four areas of the right hemisphere showed higher levels of activity compared to one area (sensory/motor) of the left hemisphere for the Eyes open condition. C3 in the left hemisphere of the sensory/motor cortex was highest over all others. Alpha was higher than beta and highest at site P4, and lowest at site T5.

Reading Silently. Four areas of the right hemisphere showed higher levels of activity for the Reading Silently condition. O1 in the left hemisphere of the occipital lobe showed the highest activity over all others. Alpha was higher than beta and highest at site C4, and lowest at site T5.

Frontal lobe. F4 in the right hemisphere of the frontal lobe showed the highest level of activity over all other sites for the Eyes Closed and Reading Silently conditions. F3 was the higher for the paired sites F3/F4 for Eyes Open. Alpha was dominant over beta for all three conditions and highest at site F4.

Sensory/motor cortex. C3 in the left hemisphere of the sensory motor cortex showed the highest level of activity over all other sites for the Eyes Open condition. C4 was the higher for the paired sites C3/C4 for the Eyes Closed and the Reading Silently conditions. Alpha was dominant over beta and highest at site C3 for Eyes Closed and at site C4 for Eyes Open and highest over all for Reading Silently.

Parietal lobe. Right hemisphere P4 showed the higher level for the sites P3/P4 for all three conditions. Alpha was dominant over beta for all conditions and highest over all at site P4 for Eyes Open and Reading Silently conditions.

Temporal lobe. T6 in the right hemisphere of the temporal lobe showed the higher level of activity for the paired sites T5/T6 for the Eyes Closed and Reading Silently conditions and lowest for Eyes Open. T5 in the left hemisphere showed the lowest level of activity over all other sites for the Eyes Closed and Reading Silently conditions. Alpha was dominant over beta for all conditions and highest at T6.

Occipital lobe. O1 in the left hemisphere of the occipital lobe showed the highest level of activity over all other sites for the Reading Silently condition. O2 showed the higher level of activity for the paired sites O1/O2 for the Eyes Closed and the Eyes Open conditions. Alpha was dominant and highest at O2 for Eyes Closed and Eyes Open and highest at O1 for Reading Silently.

Figure 1

Sample Pre-assessment Profile Showing Site and Hemisphere With the Highest and Lowest Total QEEG Values, and Alpha Higher Compared to Beta, N=11

Eyes Closed

Total QEEG						Alpha / Beta				
	|	H			frontal lobe (F3 | F4)	A	|	**A**	
H	|				sensory motor cortex (P3 | P4)	**A**	|	A	
L	|	|	H	| H	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA	| A	| **HA**	| **A**
	|	H			occipital lobe (O1 | O2)	A	|	**A**	

Delta highest at sites F3/F4/C3/C4/P3. Alpha higher than delta at sites P4/T5/T6/O1/O2. Beta3 lowest at all sites.

Eyes Open

Total QEEG						Alpha / Beta				
H	|				frontal lobe (F3 | F4)	**A**	|	A	
	|	H			sensory motor cortex (P3 | P4)	A	|	**A**	
H	|	|	H	| **L**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA	| A	| **HA**	| **A**
	|	H			occipital lobe (O1 | O2)	A	|	**A**	

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Reading Silently

Total QEEG						Alpha / Beta				
	|	H			frontal lobe (F3 | F4)	A	|	**A**	
	|	H			sensory motor cortex (C3 | C4)	A	|	**HA**	
L	|	|	H	| H	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA	| A	| **A**	| **A**
H	|				occipital lobe (O1 | O2)	**A**	|	A	

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

H shows highest total uV level for paired sites, H | H = same value, **H** = site with highest uV level compared to all other sites.
L shows site with the lowest uV level compared to other sites. A = alpha higher than beta, **A** = higher for paired sites.
LA = lowest alpha compared to all sites, **HA** = highest alpha compared to all sites.

Individual Participants' Hemisphere and Scalp Site Dominance

Hemisphere dominance showing the hemisphere with the highest QEEG values are described first. Changes in hemisphere dominance between pre and post-assessments are described next. Finally higher alpha QEEG activity compared to beta activity is described in a format that gives preference to alpha dominance. Where alpha is also higher than beta only alpha is indicated. Alpha is shown as higher than beta with the marker capital letter A, the site with the higher alpha for paired sites where alpha is higher than beta for both sites is marked with a bold **A**. Beta is therefore shown to be lower than alpha when a beta marker is absent. When beta is higher than alpha then beta is represented with the beta marker B. The site with the highest alpha compared to all other sites is marked with a bold **HA**, and the site with the lowest alpha compared to all other sites is marked with LA. When beta is highest over all sites then the highest beta site is marked with a bold **B**.

Participant #1 (male NT). The results in Figure 2 for the pre-assessment show left hemisphere dominance for all three conditions. The results further show a shift to a right hemisphere dominance for the post-assessment for all three conditions.

The pre-assessment results also show alpha was dominant over beta for all three conditions. For the eyes closed condition, alpha was highest for site O1 over all other sites, and lowest at sites F3 and F4. For the eyes open condition, alpha was highest sites for the three sites P3, T5, and O1, having the same value over all other sites. Further, for both conditions alpha was the lowest at site T6. For the reading silently condition, alpha was highest at site O2, and lowest at site T6.

Figure 2

Case #1 (Male NT): Right Versus Left Hemisphere Highest and Lowest Alpha EEG Value Compared to Beta

Pre-Alpha / Beta	Eyes Closed	Post-Alpha / Beta
LA | LA	frontal lobe (F3 | F4)	A | **A**
A | A	sensory motor cortex (P3 | P4)	A | **A**
A | **A** | A | A	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA | 0 | **HA** | **A**
HA | A	occipital lobe (O1 | O2)	**A** | A

Delta highest over all bands at F3/F4/C3/C4/P3, Alpha highest over all bands at P4/T5/T6/O1/O2.

Pre-Alpha / Beta	Eyes Open	Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	A | **A**
A | A	sensory motor cortex (P3 | P4)	A | **A**
HA | **HA** | A | LA	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA | 0 | **HA** | **A**
HA | A	occipital lobe (O1 | O2)	**A** | A

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Pre-Alpha / Beta	Reading Silently	Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	LA | **A**
A | A	sensory motor cortex (C3 | C4)	LA | **A**
B | **A** | A | LA	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA | 0 | **A** | **HA**
A | **HA**	occipital lobe (O1 | O2)	**A** | B

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

A = alpha higher than beta, LA = lowest alpha for all sites, **A** = highest alpha for paired sites, **A** | **A** = same value alpha, **HA** = site with highest alpha compared to all other sites, B = beta higher than alpha, **B** = highest over all sites. #0 = no recording.

The post-assessment results show that alpha was dominant over beta for all three conditions, with the single exception of a higher beta value at site O2 for the reading silently condition. For both the eyes closed and the eyes open conditions, alpha was highest at site P4 over all other sites, and lowest at site T5. For the reading silently condition, alpha was highest at site T6, and lowest at sites F3, P3, and T5, all showing the same value.

Participant #2 (male NT). The results in Figure 3 for the pre-assessment show right hemisphere dominance for all three conditions. The results further show a shift to a left hemisphere dominance for the post-assessment for all three conditions.

The results for the pre-assessment for the eyes closed condition also show that beta was higher than alpha for all sites except F4 and T6. Further, for the eyes closed condition beta was highest at site P4 over all other sites. Alpha was highest at site F4 over all other sites and lowest at site T5. For the eyes open condition, beta was dominant for the parietal lobe and the temporal lobe, with the highest beta at site T6. Alpha was highest at sites F3 and O1, having the same value over all other sites. Further, alpha was the lowest at site T5. For the reading silently condition, alpha was dominant over beta and was the highest at site C4, and lowest at site T5. Alpha was dominant at both sites O1 and O2, with the same value for both sites.

The post-assessment results for the eyes closed condition show that beta was dominant over alpha for the parietal lobe, temporal lobe and occipital lobe. Further, beta was highest over all others at site O1. Alpha was also highest at site O1, but dominated by beta. For the eyes open condition alpha was dominant over beta, but

Figure 3

Case #2 (Male NT): Right Versus Left Hemisphere Highest and Lowest Alpha EEG Value Compared to Beta

Eyes Closed

Pre-Alpha / Beta		Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	**A** | **A**
B | B	sensory motor cortex (P3 | P4)	**A** | A
B/LA | B | **B** | **HA**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	B | 0 | B | B/LA
B | B	occipital lobe (O1 | O2)	**B/HA** | B

Delta highest over all bands at F3/F4/C3/C4/P3, Alpha highest over all bands at P4/T5/T6/O1/O2.

Eyes Open

Pre-Alpha / Beta		Post-Alpha / Beta
A | **HA**	frontal lobe (F3 | F4)	**A** | **A**
A | **A**	sensory motor cortex (P3 | P4)	**A** | A
B/LA | B | B | **B**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	B | 0 | A | B/LA
HA | A	occipital lobe (O1 | O2)	**HA** | **HA**

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Reading Silently

Pre-Alpha / Beta		Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	**A** | A
B | **HA**	sensory motor cortex (C3 | C4)	**A** | A
B/AL | A | **A** | B	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	B/**HA** | 0 | A | B/LA
A | **A**	occipital lobe (O1 | O2)	**A** | A

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

A = alpha higher than beta, LA = lowest alpha for all sites, **A** = highest alpha for paired sites, **A** | **A** = same value alpha, **HA** = site with highest alpha compared to all other sites, B = beta higher than alpha, **B** = highest over all sites. #0 = no recording.

beta shows a highest value over all others at site T5. Alpha was highest at sites O1 and O2, with the same value for both sites. Further, alpha was lowest at site T6. For the reading silently condition the results show that alpha dominated over beta, except for the temporal lobe sites T5 and T6. Alpha was highest at site T5, but beta was higher than alpha, and beta was highest overall. Alpha was lowest at site T6.

Participant #3 (male NT). The results in Figure 4 for the pre-assessment show right hemisphere dominance for all three conditions. The results also show there was a shift to a left hemisphere dominance for the post-assessment for the eyes closed and the eyes open condition. The right hemisphere remained dominant for the reading silently condition.

The results for the pre-assessment for the eyes closed condition also show that alpha was dominant over beta for all sites. Further, for the eyes closed condition alpha was highest at site T6, over all other sites, and lowest at site T5. For the eyes open condition alpha was dominant for all sites, and was highest at site T6, and lowest at site C3. For the reading silently condition, beta was dominant over alpha for the sensory motor area, the temporal lobe and the occipital lobe. Alpha was dominant and highest over all others at sites P3 and P4, with the same value for both sites. Alpha was lowest at site F3.

The post-assessment results show that alpha was higher than beta for all sites for all three conditions. Further, for eyes closed alpha was highest overall at site T6, lowest at the O1 site. For eyes open alpha was highest over all at T6, and lowest at T5. For reading silently alpha was highest at site T6, and lowest at site O1.

Figure 4

Case #3 (Male NT): Right Versus Left Hemisphere Highest and Lowest Alpha EEG Value Compared to Beta

Pre-Alpha / Beta	Eyes Closed	Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	A | **A**
A | **A**	sensory motor cortex (P3 | P4)	A | **A**
LA | A | **A** | **HA**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	A | 0 | **A** | **HA**
A | A	occipital lobe (O1 | O2)	LA | **A**

Delta highest over all bands at F3/F4/C3/C4/P3, Alpha highest over all bands at P4/T5/T6/O1/O2.

Pre-Alpha / Beta	Eyes Open	Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	**A** | **A**
LA | **A**	sensory motor cortex (P3 | P4)	A | **A**
A | A | **A** | **HA**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA | 0 | **HA** | **A**
A | **A**	occipital lobe (O1 | O2)	**A** | A

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Pre-Alpha / Beta	Reading Silently	Post-Alpha / Beta
LA | **A**	frontal lobe (F3 | F4)	**A** | A
B | B	sensory motor cortex (C3 | C4)	**A** | A
B | **HA** | **HA** | B	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	A | 0 | A | **HA**
B | **A**	occipital lobe (O1 | O2)	LA | **A**

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

A = alpha higher than beta, LA = lowest alpha for all sites, **A** = highest alpha for paired sites, **A** | **A** = same value alpha, **HA** = site with highest alpha compared to all other sites, B = beta higher than alpha, **B** = highest over all sites. #0 = no recording.

Participant #4 (male NTD). Right and left hemisphere changes were not available because this participant did not complete a post-assessment.

The results in Figure 5 for the eyes closed condition show a left hemisphere dominance for the pre-assessment. The results for the eyes closed condition also show that alpha was higher than beta for all sites. Alpha was highest at site P3, and lowest at site T6.

The results for the eyes open condition show that beta was dominant for all sites and was highest overall at site F3. The results further show that alpha was highest at site P4, but was lower than beta. Also, alpha was lowest at site T6.

The results for the reading silently condition show that beta was dominant over all others and was highest overall at site T5. The results further show that alpha was highest at site O1, but lower than beta. Also, alpha was lowest at site T6.

Participant #5 (male NTD). Right and left hemisphere changes were not available because this participant did not complete a post-assessment. However, The results in Figure 6 show there was a right hemisphere dominance for all three conditions.

The results for the eyes closed condition show that alpha was highest over all others at site F4 and lowest at site T5

The results for the eyes open condition show that alpha was highest over all others at site P3 and lowest at site T5.

The results for the reading silently condition show that alpha was highest over all others at site T6, and lowest at site T5.

Figure 5

Case #4 (Male NT): Right Versus Left Hemisphere Highest and Lowest Alpha EEG Value Compared to Beta

Pre-Alpha / Beta	Eyes Closed	Post-Alpha / Beta
A | A	frontal lobe (F3 | F4)	|
A | **A**	sensory motor cortex (P3 | P4)	|
A | **HA** | A | LA	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
A | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at F3/F4/C3/C4/P3, Alpha highest over all bands at P4/T5/T6/O1/O2.

Pre-Alpha / Beta	Eyes Open	Post-Alpha / Beta
B | B	frontal lobe (F3 | F4)	|
B | A	sensory motor cortex (P3 | P4)	|
B | B | B/**HA** | B/LA	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
B | B	occipital lobe (O1 | O2)	|

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Pre-Alpha / Beta	Reading Silently	Post-Alpha / Beta
B | B	frontal lobe (F3 | F4)	|
B | B	sensory motor cortex (C3 | C4)	|
B | **A** | B | B/LA	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
HA | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

A = alpha higher than beta, LA = lowest alpha for all sites, **A** = highest alpha for paired sites, **A** | **A** = same value alpha, **HA** = site with highest alpha compared to all other sites, B = beta higher than alpha, **B** = highest over all sites. #0 = no recording.

Figure 6

Case #5 (Male NT): Right Versus Left Hemisphere Highest and Lowest Alpha EEG Value Compared to Beta

Pre-Alpha / Beta	Eyes Closed	Post-Alpha / Beta
A | **HA**	frontal lobe (F3 | F4)	|
A | **A**	sensory motor cortex (P3 | P4)	|
LA | **A** | **A** | **A**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
A | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at F3/F4/C3/C4/P3, Alpha highest over all bands at P4/T5/T6/O1/O2.

Pre-Alpha / Beta	Eyes Open	Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	|
A | **A**	sensory motor cortex (P3 | P4)	|
LA | **HA** | A | **A**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
A | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Pre-Alpha / Beta	Reading Silently	Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	|
A | **A**	sensory motor cortex (C3 | C4)	|
LA | **A** | A | **HA**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
A | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

A = alpha higher than beta, LA = lowest alpha for all sites, **A** = highest alpha for paired sites, **A** | **A** = same value alpha, **HA** = site with highest alpha compared to all other sites, B = beta higher than alpha, **B** = highest over all sites. #0 = no recording.

Participant #6 (male AA). The results in Figure 7 for the pre-assessment show left hemisphere dominance for all three conditions. The results also show there was a shift to a right hemisphere dominance for the post-assessment for the eyes closed and the eyes open condition. The right hemisphere remained dominant for the reading silently condition.

The results for the pre-assessment show that alpha was higher than beta for all sites for all three conditions. The results for the eyes closed condition show alpha was highest at site F4, over all other sites, and lowest at site T5. For the eyes open condition alpha was highest at site F4, and lowest at sites T5 and T6, with the same value for both sites. For the reading silently condition alpha was highest over all others at site O1, and lowest at site T5.

The post-assessment results show that alpha was higher than beta except at site T5 for the eyes open condition and at sites T5 and O1 for the reading silently condition. The results for the eyes closed condition show that alpha was highest overall at site O1, and lowest at site T5. For the eyes open condition alpha was highest over all others at sites F3 and F4, with the same value for both sites. Further, alpha was lowest overall at sites T5 and T6, with the same value for both sites. For the reading silently condition the results show that alpha was highest over all others at site O1, but beta was higher than alpha. Further, beta was higher than alpha at site T5, and alpha was lowest at site T6.

Figure 7

Case #6 (Male AA): Right Versus Left Hemisphere Highest and Lowest Alpha EEG Value Compared to Beta

Eyes Closed

Pre-Alpha / Beta		Post-Alpha / Beta
A | **HA**	frontal lobe (F3 | F4)	**A** | **A**
A | A	sensory motor cortex (P3 | P4)	A | **A**
LA | **A** | A | A	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA | 0 | **A** | **A**
A | A	occipital lobe (O1 | O2)	**HA** | A

Delta highest over all bands at F3/F4/C3/C4/P3, Alpha highest over all bands at P4/T5/T6/O1/O2.

Eyes Open

Pre-Alpha / Beta		Post-Alpha / Beta
A | **HA**	frontal lobe (F3 | F4)	**HA** | **HA**
A | **A**	sensory motor cortex (P3 | P4)	A | **A**
LA | **A** | A | LA	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	B/ LA | 0 | **A** | LA
A | A	occipital lobe (O1 | O2)	**A** | A

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Reading Silently

Pre-Alpha / Beta		Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	**A** | A
A | A	sensory motor cortex (C3 | C4)	**A** | **A**
LA | **A** | A | **A**	temporal lobe/parietal lobe (T5 | P3 | P4 | T5)	B | 0 | A | LA
HA | A	occipital lobe (O1 | O2)	**B/HA** | A

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

A = alpha higher than beta, LA = lowest alpha for all sites, **A** = highest alpha for paired sites, **A** | **A** = same value alpha, **HA** = site with highest alpha compared to all other sites, B = beta higher than alpha, **B** = highest over all sites. #0 = no recording.

Participant #7 (female AA). Right and left hemisphere changes were not available because this participant did not complete a post-assessment. However, the pre-assessment results in Figure 8 show there was a right hemisphere dominance for all three conditions.

The results further show that alpha was highest over all others at site P3, and lowest at site T6.

The results for the eyes open condition show that alpha was highest over all others at site C3, and lowest at site O2.

The results for the reading silently condition show that alpha was highest over all others at site P3, and lowest at site T6.

Participant #8 (female AA). The results in Figure 9 for the pre-assessment show left hemisphere dominance for all three conditions. The results further show there was a shift to right hemisphere dominance for the post-assessment for all three conditions

The results for the pre-assessment for the eyes closed condition also show that alpha was higher than beta for all sites. Further for the eyes closed condition, alpha was highest at site F3, over all other sites, and lowest at site T5. For the eyes open condition alpha was higher than beta for all sites except for site T6 where beta was higher than alpha. Alpha was highest over all others at site F3, and lowest at site T5. For the reading silently condition, alpha was higher for all sites except for site O1 where beta was higher than alpha. Alpha was highest over all others at sites F3 and lowest at sites T5 and P3, with the same value for both sites.

Figure 8

Case #7 (Female AA): Right Versus Left Hemisphere Highest and Lowest Alpha EEG Value Compared to Beta

Pre-Alpha / Beta	Eyes Closed	Post-Alpha / Beta
A | A	frontal lobe (F3 | F4)	|
A | A	sensory motor cortex (P3 | P4)	|
A | **HA** | A | LA	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
A | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at F3/F4/C3/C4/P3, Alpha highest over all bands at P4/T5/T6/O1/O2.

Pre-Alpha / Beta	Eyes Open	Post-Alpha / Beta
A | A	frontal lobe (F3 | F4)	|
HA | A	sensory motor cortex (P3 | P4)	|
A | A | **A** | **A**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
A | LA	occipital lobe (O1 | O2)	|

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Pre-Alpha / Beta	Reading Silently	Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	|
HA | A	sensory motor cortex (C3 | C4)	|
A | **A** | A | LA	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
A | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

A = alpha higher than beta, LA = lowest alpha for all sites, **A** = highest alpha for paired sites, **A** | **A** = same value alpha, **HA** = site with highest alpha compared to all other sites, B = beta higher than alpha, **B** = highest over all sites. #0 = no recording.

Figure 9

Case #8 (Female AA): Right Versus Left Hemisphere Highest and Lowest Alpha EEG Value Compared to Beta

Pre-Alpha / Beta	Eyes Closed	Post-Alpha / Beta
HA | A	frontal lobe (F3 | F4)	A | **A**
A | A	sensory motor cortex (P3 | P4)	A | **A**
BL | **A** | A | **A**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA | 0 | **HA** | **A**
A | A	occipital lobe (O1 | O2)	**A** | A

Delta highest over all bands at F3/F4/C3/C4/P3, Alpha highest over all bands at P4/T5/T6/O1/O2.

Pre-Alpha / Beta	Eyes Open	Post-Alpha / Beta
HA | A	frontal lobe (F3 | F4)	A | **HA**
A | A	sensory motor cortex (P3 | P4)	A | **A**
B/LA | **A** | A | **A**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA | 0 | **A** | **A**
A | A	occipital lobe (O1 | O2)	**A** | A

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Pre-Alpha / Beta	Reading Silently	Post-Alpha / Beta
HA | A	frontal lobe (F3 | F4)	A | **HA**
A | A	sensory motor cortex (C3 | C4)	A | **A**
LA | LA | **A** | **A**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	B/LA | 0 | A | **A**
B | **A**	occipital lobe (O1 | O2)	A | **A**

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

A = alpha higher than beta, LA = lowest alpha for all sites, **A** = highest alpha for paired sites, **A** | **A** = same value alpha, **HA** = site with highest alpha compared to all other sites, B = beta higher than alpha, **B** = highest over all sites. #0 = no recording.

The post-assessment results show that alpha was higher than beta for all sites for all three conditions, except for site T5 for the reading silently condition where beta was higher than alpha. Further for the eyes closed condition alpha was highest over all others at site P4, and lowest at site T5. For the eyes open condition alpha was highest over all others at site F4, and lowest at site T5. For the reading silently condition the results show that alpha was highest over all others at site F4, and lowest at site T5.

Participant #9 (female AA). The results in Figure 10 for the pre-assessment show right hemisphere dominance for the eyes closed condition and for the reading silently condition. The was no dominant hemisphere for the eyes open condition. The results further show there was a shift to right hemisphere dominance for the post-assessment for all three conditions.

The results for the pre-assessment show that alpha was higher than beta for all sites and for all three conditions. The results for the eyes closed condition further show that alpha was highest at site P4, over all other sites, and lowest at site P3. For the eyes open condition alpha was highest over all others at site C3, and lowest at site O1. For the reading silently condition, alpha was highest over all others at site C4, and lowest at sites T5 and O1, with the same value for both sites.

The post-assessment results show that alpha was highest over all others at site O2, and lowest at site T5. For the eyes open condition alpha was highest over all others at site P4, and lowest at site T5. For the reading silently condition the results show that alpha was highest over all others at site P4, and lowest at site T5.

Figure 10

Case #9 (Female AA): Right Versus Left Hemisphere Highest and Lowest Alpha EEG Value Compared to Beta

Eyes Closed

Pre-Alpha / Beta		Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	**A** | A
A | **A**	sensory motor cortex (P3 | P4)	A | **A**
A | LA | **HA** | **A**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA | 0 | **A** | **A**
A | **A**	occipital lobe (O1 | O2)	A | **HA**

Delta highest over all bands at F3/F4/C3/C4/P3, Alpha highest over all bands at P4/T5/T6/O1/O2.

Eyes Open

Pre-Alpha / Beta		Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	A | **A**
HA | A	sensory motor cortex (P3 | P4)	A | **A**
A | **A** | A | **A**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA | 0 | **HA** | **A**
LA | **A**	occipital lobe (O1 | O2)	A | **A**

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Reading Silently

Pre-Alpha / Beta		Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	**A** | A
A | **HA**	sensory motor cortex (C3 | C4)	A | **A**
LA | A | **A** | **A**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	LA | 0 | **HA** | A
LA | **A**	occipital lobe (O1 | O2)	A | **A**

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

A = alpha higher than beta, LA = lowest alpha for all sites, **A** = highest alpha for paired sites, **A | A** = same value alpha, **HA** = site with highest alpha compared to all other sites, B = beta higher than alpha, **B** = highest over all sites. #0 = no recording.

Participant #10 (female AA). Right and left hemisphere changes were not available because this participant did not complete a post-assessment. See Figure 11.

The results for the eyes closed condition show a balance between right and left hemispheres for the sensory motor area, the parietal lobe and the temporal lobe. The results further show that alpha was highest over all others at site F3, and lowest at sites T5 and T6, with the same value for both sites.

The results for the eyes open condition mixed dominance between hemispheres. The results further show that alpha was highest over all others at site F3, and lowest at site T5.

The results for the reading silently condition show a left hemisphere dominance. The results further show that alpha was highest over all others at site F3, and lowest at sites T5 and T6, with the same value for both sites.

Figure 11

Case #10 (Male NT): Right Versus Left Hemisphere Highest and Lowest Alpha EEG Value Compared to Beta

Pre-Alpha / Beta	Eyes Closed	Post-Alpha / Beta
A | **HA**	frontal lobe (F3 | F4)	|
A | **A**	sensory motor cortex (P3 | P4)	|
LA | **A** | **A** | LA	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
A | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at F3/F4/C3/C4/P3, Alpha highest over all bands at P4/T5/T6/O1/O2.

Pre-Alpha / Beta	Eyes Open	Post-Alpha / Beta
A | **HA**	frontal lobe (F3 | F4)	|
A | **A**	sensory motor cortex (P3 | P4)	|
LA | **A** | A | **A**	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
A | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Pre-Alpha / Beta	Reading Silently	Post-Alpha / Beta
A | **HA**	frontal lobe (F3 | F4)	|
A | A	sensory motor cortex (C3 | C4)	|
LA | **A** | A | LA	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
A | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

A = alpha higher than beta, LA = lowest alpha for all sites, **A** = highest alpha for paired sites, **A** | **A** = same value alpha, **HA** = site with highest alpha compared to all other sites, B = beta higher than alpha, **B** = highest over all sites. #0 = no recording.

Participant #11 (female AA). Right and left hemisphere changes were not available because this participant did not complete a post-assessment. However, the results in Figure 12 show there was a right hemisphere dominance for all three conditions.

The results show that alpha was higher than beta for all sites for all three conditions, except at site O1, for the reading silently condition.

The results for the eyes closed condition show that alpha was highest over all others at site P4, and lowest at site O1.

The results for the eyes open condition show that alpha was highest over all others at site P4, and lowest at site O1.

The results for the reading silently condition show that alpha was highest over all others at site O1, but beta was higher than alpha for this same site. The results further show that alpha was lowest at sites T5 and F3, with the same value for both sites.

Figure 12

Case #11 (Female AA): Right Versus Left Hemisphere Highest and Lowest Alpha EEG Value Compared to Beta

Pre-Alpha / Beta	Eyes Closed	Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	|
A | **A**	sensory motor cortex (P3 | P4)	|
A | A | **HA** | A	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
LA | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at F3/F4/C3/C4/P3, Alpha highest over all bands at P4/T5/T6/O1/O2.

Pre-Alpha / Beta	Eyes Open	Post-Alpha / Beta
A | **A**	frontal lobe (F3 | F4)	|
A | **A**	sensory motor cortex (P3 | P4)	|
A | A | **HA** | A	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
LA | **A**	occipital lobe (O1 | O2)	|

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta2 lowest at O2.

Pre-Alpha / Beta	Reading Silently	Post-Alpha / Beta
LA | **A**	frontal lobe (F3 | F4)	|
A | **A**	sensory motor cortex (C3 | C4)	|
LA | A | **A** | A	temporal lobe/parietal lobe (T5 | P3 | P4 | T6)	| | |
B/**HA** | A	occipital lobe (O1 | O2)	|

Delta highest over all bands at all sites. Beta3 lowest compared to all bands except Beta 2 same at O1/O2 and lowest at O1.

A = alpha higher than beta, LA = lowest alpha for all sites, **A** = highest alpha for paired sites, **A** | **A** = same value alpha, **HA** = site with highest alpha compared to all other sites, B = beta higher than alpha, **B** = highest over all sites. #0 = no recording.

CHAPTER FIVE

DISCUSSION

This study explored the use of neurofeedback training (NT) as a therapeutic method for helping alcoholics of Canadian Aboriginal ancestry to reduce their craving for alcohol consumption. The sample was made up of volunteer participants Poundmaker's Lodge drug and alcohol rehabilitation centre, St. Alberta, Alberta, Canada. NT at scalp site O1 consisted of hearing different auditory tones when raising alpha (8-12 Hz) and inhibiting beta (16-20 Hz) frequency bands, and as viewed on a computer monitor screen. Data gathered from psychometric measures, participants' backgrounds, their experiences of NT and the centre's AA program, and from three year follow-up interviews are discussed. Quantitative data collected from pre and post-assessment electroencephalograph (EEG) recordings were analysed for descriptive measures showing changes in averaged QEEG values and showing hemisphere and scalp electrode site dominance.. The results from scalp sites F3, F4, C3, C4, P3, P4, T5, T6, O1 & O2 and from NT are discussed in reference to alcoholism. Due to limitations of the study only a descriptive analysis is provided.

Limitations of The Study

External Validity

This study utilized a non-random sampling design, specifically volunteer participants. As has been reported, volunteer participants may be more motivated than non-volunteer participants, thus posing a threat to external validity (Association for Advanced Training in the Behavioral Sciences, 1994). Also, the participants in this

study who were from correctional centres may have had ulterior motives for attending the treatment centre and for volunteering for NT. The small sample size of the study may also limit its generalizability and did not allow for the use of inferential statistics.

Internal Validity

Selection. Participants were not randomly assigned to treatment groups within this study. Some differences existed between groups in terms of familial background, personal lifestyles, socio-economic status, educational achievement level, and cognitive functioning level. Also, some participants appeared to be more interested in the NT sessions as compared to other participants and differences in interest may have affected their degree of focus during the NT sessions. These individual differences may have acted as extraneous variables confounding the study's results.

History. The procedures of the treatment centre's augmented AA program and the centre's counselling and training staff were not under the control of the researcher. The treatment centre's procedures assigned the participants to different AA treatment groups and to different AA counsellors. These extraneous variables may also have threatened internal validity.

Attrition. Two participants in the NT treatment group dropped in the first quarter of the study. To control for the potential effects of attrition, a researcher can determine whether systematic differences exist on pre-treatment measures between participants who drop out of a study and the remaining participants. Systematic differences can introduce the possibility of attrition bias (Association for Advanced Training in the Behavioral Sciences, 1994). In this study, analysis of pre-treatment

data of the two participants who dropped out did not reveal systematic differences as compared to the remaining participants.

Representative Sample

The biographical profiles of the participants in this study indicate this sample is not representative of the general population of Canadian Aboriginal peoples. This sample of alcoholics from the Canadian Aboriginal population is more representative of adults of Canadian Aboriginal background who have a lower level of education than what is expected for a person of Canadian Aboriginal ancestry. Also, the participants are more representative of alcoholics of Canadian Aboriginal ancestry who come from severely troubled families where traditional Canadian Aboriginal culture and spiritual practices were not taught or practised, where alcohol was abused by parents, and in which they frequently experienced emotional abuse and/or physical abuse and/or sexual abuse. Further, they are representative of adults with Canadian Aboriginal ancestry who use chemical substances as a means for coping with a difficult and often traumatic adult lifestyle.

Qualitative Data Supportive of These Conclusions

The background data (summarized in Table 1) show the participants were in the age range between 18 and 51 years, with an average age of 29.5 years (males averaged 31.0 years and females averaged 27.6 years). The cognitive functioning level of the sample was estimated as *Average* (group mean FSIQ=98). In this sample of Canadian Aboriginal peoples, 73% had grade nine or less education and 18% had completed some education beyond grade 12. In comparison, only 17% of the

Aboriginal population of Canada had grade nine or less education (Aboriginal Peoples Survey, Statistics Canada, 1991b), and 33% had some post-secondary education.

Family background and substance abuse. Although the participants in this study were classified as *Status Indians*, according to the federal government's classification system, the participants were generally of mixed Indian and Caucasian origins. The background data show that all participants had experienced traumatic events during their childhood, such as emotional abuse, physical abuse, sexual abuse, and separation from their natural parents. Also, all participants reported traumatic adult experiences, such as emotional abuse, physical abuse, sexual abuse, incarceration, physical fights, divorce, and the loss of loved ones from violent causes.

Ten participants reported the abuse of alcohol in their familial environment. Only one stated he believed his parents were social drinkers, while the other ten reported alcohol was abused by either their natural or their step-parents. In comparison, 86% of adults in the total Canadian Aboriginal population reported using alcoholic beverages, but only 61% reported alcohol as a problem in their communities, and 39% reported family violence as a problem (Statistics Canada, 1991a).

All participants reported they had used other forms of mood altering substances including pharmaceutical products. In comparison, only 48% in the total Canadian Aboriginal population reported drug abuse was a problem. The data also show that all participants smoked tobacco on a daily basis, compared to 44% in the total Canadian Aboriginal population (Statistics Canada, 1991a).

Traditional aboriginal cultural determinants. Although it is interesting to speculate that the participants may have been influenced to consume alcohol by

traditional cultural factors, such as reported in the Early Jesuit Relations (Dailey, 1968). The Jesuits reported that traditional cultural views, particularly that alcohol released one's spirit, were frequently instrumental in bringing about the abuse of alcohol by many of the Indian people during the early colonial period of North America. However, since only one participant in this study reported having minimum traditional cultural experience, traditional Aboriginal cultural background can be ruled out as a possible determinant for alcoholism for the participants in this study.

Conclusions from Background Data

The background data and comparative statistics show that the participants in this study were more representative of adults of Canadian Aboriginal ancestry with mixed Indian and Caucasian origins who come from a background of difficult and traumatic childhood experiences within troubled families, and who also have fewer opportunities in the labour market because of lower education. Vaillant (1983), from studies on genetic loading, determined that an unstable environment is an important predictor for determining individual loss of control of alcohol at an early age and for the development of multiple symptoms. Familial influences and peer group pressures were found to be paramount to the onset of addiction in adolescence due to the powerful effect of role models (Belle Glass & Marshall, 1991). Research on Canadian children in general who are reared in troubled families and in step-families indicates there is a greater chance for life long problems because of troubled childhood, poorer education, and less opportunities in the labour market (Statistics Canada, 1997).

Psychological Factors

Depression. All participants reported experiencing depression prior to starting their treatment program. The average level of depression was in the minimal range with only two reporting moderate to severe levels of depression. Although the depression scores were greatly reduced at the end of the 28 day treatment program it is difficult to know what might have contributed to this reduction. During the period of this study, the participants resided in a favourable environment where they were free of all of their usual stressors. Therefore the change in depression scores may have been solely because these stressors were absent during their 28 day treatment program. Also the centre's treatment program taught relaxation techniques and dealt with many of their personal problems associated with their alcoholism. Hence, it is not possible to draw accurate conclusions regarding the observed changes in participants depression scores.

MMPI-2. The data showing the scores obtained on the MMPI-2 (Tables 33, 34, and 35) by five of the participants (#1, #2, #3, #7 & #11) indicate that the three male participants (#1, #2 & #3) who were taking the NT as well as the augmented AA program reported more clinically significant psychological problems than were reported by the two female participants who were only taking the AA program. The common themes from the Basic Scale for the three male NT participants were: *Depression, Psychopathic Deviate, Paranoia and Schizophrenia. College Maladjustment, Post-Traumatic Stress and Alienation* were their common themes on the supplementary scales. *Depression-Obvious, Psychopath-Obvious, Paranoia-Obvious, and Hypomania-Obvious* were their common themes on the Wiener-Harmon Scales.

Lastly, their common themes on the Content Scales were: *Work Interference and Anger*. Only four clinically significant scores were reported by the two female participants who completed the pre-assessment. These were: *Repression* and *Shyness* on the Basic Scales (participant # 7) and *Hysteria Subtle* and *Psychopath Subtle* (participant #11).

Post-assessment MMPI-2. There was a tendency to report a general reduction in symptoms on the post-assessment by the two participants who completed them. However, the post-assessment results from participant #1 indicate that he continued to show clinically significant scores for *Hypomania* on the Basic Scales, and *Paranoia-Obvious and Hypomania-Obvious* on the Wiener-Harmon Scales. Participant #2 also showed clinically significant scores on his post-assessment for: *Depression and Paranoia* on the Basic Scales, *Mac-R-Alcoholism* on the Supplementary Scales, and *Psychopath-Obvious and Paranoia-Obvious* on the Wiener-Harmon Scales.

MMPI-2 with North American Indians. Although the original MMPI and the MMP-2 have been the most frequently used instruments for assessing the level of psychopathology with clinical populations, common difficulties have been reported (Graham, 1987; Green, 1991) with its use in general and specific difficulties were found when using this instrument with North American Indian peoples. Problems exist when using this instrument with North American Indians because of the weak representation in the normative reference group. Specifically, there were no North American Indian subjects in the original MMPI normative reference group and the normative reference group of the revised edition of the MMPI-2 contained only 77 North American Indians in the 2,600 subjects drawn from centres across the United States. Further, the 77 Indians in the

normative reference group, representing less than 3.0% of the total reference group, were all alcoholics. Data from the MMPI-2 profiles of American Indians are therefore difficult to interpret because there is no non-alcoholic data available for comparison.

Other problems exist because non-alcoholic American Indians were generally found to score higher on most clinical scales compared to their Caucasian counterparts. Hence, any scores obtained by Indian people are liable to be higher, therefore over representing possible pathology. Also, no differences were found between Indians and Caucasians on the Validity scales. This report is generally considered unusual (Green, 1991) since no consistent patterns have been found between other ethnic samples and similar trends are not found with samples of psychiatric patients or substance abusers. However, it is possible that this finding is accurately reporting "there are no differences." Therefore, North American Indians may have honestly reported that they have higher levels of psychopathology. Further, there have not been any studies of American-Indian versus Caucasian differences at the item level of the MMPI or the MMPI-2 (Green, 1991). Hence, caution needs to be exercised when making conclusions about the results obtained from Aboriginal MMPI/MMPI-2 profiles in general. Hence, caution needs to be exercised when making conclusions about the results obtained from Aboriginal MMPI/MMPI-2 profiles in general.

Over-reporting and under-reporting of pathology are common problems that affect the accuracy of the MMPI-2 for indicating reported pathology. Further, the instrument has been found to be only slightly better than 60% accurate for most clinical patients. Gripshover and Dacey, 1994, found that the MAC-R did not discriminate high risk alcoholism and seemed to be a better discriminator of female alcoholism.. Also,

there are no studies on its use with the general population. Gottesman and Prescott (1989) emphasized there are ethical concerns regarding the use of the MAC in screening for alcoholism. Other difficulties encountered with the accuracy of MMPI/MMPI-2 reports are the interpretation of items, language difficulties, and general confusion about the test items (Green , 1991). Therefore, ethnic differences and differences between clinical and normal profiles are difficult to confirm.

Conclusions from MMPI-2 Scores

Based on the reports from the previous research presented and the results from this sample indicating possible over or under-reporting, it is difficult to conclude outcomes from this sample's measures on the MMPI-2. Given the lower level of education of the male participants who completed the MMPI-2 assessments, it is possible they may have experienced some confusion with this instrument. The information provided by the MMPI-2 results is therefore considered valuable for helping to guide future research choices in selecting assessment instruments.

Theoretical View

Although the various theoretical views all point to different factors which appear to fit with the backgrounds of many alcoholics, the background data gathered from the participants in this study are more suggestive of Social Learning Theory (SLT) (Bandura, 1969; Abrams & Niaura, 1987). A number of the participants reported that alcohol was their means of dealing with the stress they were experiencing in their life. It appears that the participants in this study might have learned to use alcohol as a strategy for coping with their problems due to observed role modelling. This is most reflective of SLT which suggested there is no single marker for alcoholism

and emphasized that alcoholics are people who have learned to consume alcohol as a coping strategy from interactions and observations of significant others in their environment.

Conclusions of Theoretical Views

As suggested by SLT, it is possible that this small sample of alcoholics is more representative of Canadian Aboriginal people who become alcoholics solely because they have learned to use a chemical substance as a mechanism for coping in a difficult and unfavourable environment.

Genetic Predisposition

Almost in contrast to the view of SLT, it is equally possible that a genetic predisposition might exist for the participants of this study. All participants reported their parents used alcohol. An overview of the findings from biogenic research (Blum, 1991) indicated there is a greater risk that children of alcoholic parents will become alcoholics themselves, compared to children who come from non-alcoholic parents. Further, Blum's genetic research findings on North American Indians in the southern regions of the USA indicated they were 85% more likely to become alcoholics, than was expected in the non-native population.

Conclusions of Genetic Indicators

Because there were no DNA samples from the participants in this study, it is not possible to come to any conclusions regarding genetic predisposition. Although biogenic research seeks to find markers for alcoholism in the human DNA chain, Blum (1991) also reported that other factors might be the cause. Therefore, it can only be concluded from the data of their family backgrounds that the participants appear to

have had a greater genetic risk for alcoholism. However, it is possible that other factors, such as environmental influence, peer group influence, and psychological factors, potentially might have been causal factors in their alcoholism.

QEEG Measures

The QEEG recordings from ten scalp sites were examined: F3 and F4 of the frontal lobe, C3 and C4 of the sensory motor lobe, P3 and P4 of the parietal lobe, T5 and T6 of the temporal lobe, and O1 and O2 of the occipital lobe. The qualitative electroencephalograph (QEEG) profiles complied from the participants' averaged pre-assessment QEEG recordings were grouped to show a sample QEEG profile. The sample QEEG profile indicated a cluster of scalp electrode site markers that might predict alcoholism. The sites indicated were:

1. Delta and theta frequency bands showed the highest uV amplitudes at sites F3 and F4 of the frontal lobe, for all three conditions.
2. Alpha frequency band was found to show higher averaged uV amplitudes than the three beta bands, in general, for all ten sites and the three conditions.
3. Alpha showed the highest averaged uV amplitude at site P4 of the parietal lobe for the Eyes Closed condition, and at site C4 of the sensory motor cortex for the Eyes Open and the Reading Silently conditions.
4. Beta1 showed the highest averaged uV amplitude at site P3 of the parietal lobe for the Eyes Closed condition, at site C4 of the sensory motor cortex for the Eyes Open condition, and at site O2 of occipital lobe for the Reading Silently condition.

5. Beta2 showed the highest averaged uV amplitude at site O1 of the occipital lobe for Eyes Closed and the Reading Silently conditions, and also at site C4 of the sensory motor cortex for the Eyes Open condition.

6. Beta3 showed the highest averaged uV amplitude at site P4 of the parietal lobe for the Eyes Closed condition, and at site C4 of the sensory motor cortex for the Eyes Open and the Reading Silently conditions.

7. Site T5 showed the lowest averaged uV amplitudes for all frequency bands, except Beta3, for all three conditions.

8. Site P3 showed the lowest uV averaged amplitude for the Beta3 band, for all 3 conditions.

9. There was also a tendency toward right hemisphere dominance.

Sample QEEG Profile Indicating Predisposition to Alcoholism

It appears from these findings that possible scalp electrode site markers might be indicated when highest and lowest QEEG uV amplitudes are found to exist together and in the order indicated by this sample's QEEG profile, as determined from the grouped QEEG pre-assessment data. The QEEG profile of this sample that might indicate a predisposition for alcoholism shows the following group of QEEG markers from the ten sites and three conditions assessed:

1. Delta and theta show the highest uV amplitudes at F3 and F4.
2. Alpha shows higher uV amplitudes compared to beta 12-24 Hz, in general.
3. Delta, theta and alpha show lowest uV amplitudes at T5.
4. Beta shows lowest uV amplitude at T5, in general.
5. Beta shows highest uV amplitude at C4, but lower than alpha.

6. Beta1 shows highest uV amplitudes at P3, O1, but lower than alpha.
7. Beta2 shows highest uV amplitudes at O1, and O2, but lower than alpha.
8. Beta3 shows highest uV amplitudes at P4, but lower than alpha.
9. There is also a tendency toward right hemisphere dominance.

Previous research reports have indicated that EEG markers for alcoholism could be determined from higher beta amplitude (Ehlers & Schuckit, 1990; Ehlers & Schuckit, 1993), or from high beta and low alpha at the O1 site (Peniston & Kulkosky, 1989). Bauer and Hesselbrook (1993) suggested that higher EEG activity at the frontal lobe might indicate a predisposition to alcoholism, particularly when there is antisocial personality disorder. Bauer (1994) also concluded that higher beta activity at the CZ site was a predictor of alcoholics who are prone to relapse. Further, Deckel, Hesselbrock and Bauer (1995) suggested "dysfunction" of the EEG rhythm in the frontal lobe of alcoholics might be biologically determined. Other studies also indicated the frontal lobe sites F3 and F4, and parietal lobe site P4 (Cohen, Porjesz & Begleiter 1993) and P3 (Whipple, Parker & Noble,1987) were indicators of alcoholism. Begleiter, Porjesz, and Tenner (1979) reported that studies of the P3 site indicated both statistically significant abnormalities and non-significant findings. They concluded that abnormal amplitudes at the P3 site might indicate alcoholics who are unable to inhibit responses to irrelevant stimuli. A study at the Salk Institute in San Diego (Nevile, et al., 1991) found significant differences at the P3 site with the sons of alcoholic parents regardless if they consumed alcohol or not.

The results from this study show that all of these sites are possible indicators, particularly, when found in the collective order that is shown by the sample

QEEG profile. Duffy, Albert, McAnulty, & Garvey, (1984) concluded that not just one region of the brain is activated during performance tasks, but rather multiple regions are activated when subjects engage in specific behavioural tasks.

Conclusion from the Sample QEEG Profile

It can be seen from the sample QEEG profile of the participants in this study that there was no single marker, as suggested by previous researchers (Begleiter, Porjesz, & Tenner 1979; Whipple, Parker & Noble,1987; Peniston & Kulkosky, 1989; Ehlers & Schuckit, 1990; Ehlers & Schuckit, 1993; Bauer and Hesselbrook, 1993). Rather, there appears to be a cluster of scalp electrode site markers.

However, it is difficult to conclude if the sample QEEG profile is actually a predictor of alcoholism or not. It is possible that the other factors were the cause of alcoholism and the QEEG profiles developed because the participants had been consuming alcohol for an extended period of time. Also, all participants used other mood altering substances at some time and all participants smoked tobacco on a daily basis. Previous research reports (Dryken, Grant, White, 1961; Begleiter & Platz, 1972, Volavka, et al 1985) concluded it is difficult to determine what may have caused the discrepancies found in alcoholics EEG profiles when other chemical substances are used and when psychological factors are indicated.

The following three logical questions can be asked about the participants' individual QEEG profiles. (1) Did the participants have the QEEG profiles prior to their consumption of alcohol that started in their teenage years (sample mean 14.7 years)? (2) Or, as suggested by Ehlers and Schuckit in 1990, did this QEEG profile develop because of their extended period of consuming alcohol (sample mean 14.7

years)? (3) Also, what differences might the smoking of tobacco and the periodic use of other chemical substances have had in the development of their QEEG profiles?

Because QEEG profiles of the participants prior to their consuming alcohol are not known, conclusions can only be drawn from the reported data. According to the available data, a logical conclusion is: the sample QEEG profile of the participants in this study suggest this profile might indicate alcoholics with overlapping socioeconomic and psychological factors who have a high risk for chronic alcoholism and who also have a high risk for relapse. A possible QEEG profile suggested by the findings from this study would be one where: alpha rhythm is higher than beta in general, and where delta and theta dominate in the frontal lobe, and where all frequency bands in general show the lowest uV amplitudes at T5, and where Beta3 is lowest at P3. Also, beta is lower than alpha but highest at C4, P4, O1 and O2.

Type of Alcoholic

The background data and QEEG profiles indicate the sample is more representative of *Secondary Alcoholics*, as described by Funderburk (1949) where consuming alcohol is deemed to be a secondary problem because there are other factors that are the primary problem. The individual QEEG profiles of the participants in this study all showed higher amplitudes of alpha activity for almost all ten sites and for the three conditions assessed. According to their alpha dominant profiles and their psychological factors, they more closely represent Funderburk's classification for *Secondary Alcoholism* (Funderburk, 1949).

Also, according to Cloninger's (1981) *Alcoholic Types*, the participants in this study might meet the classification for *Type I Alcoholic*, where genetic factors

coincide with environmental factors. Although the environmental factors reported by the participants can be taken at face value it cannot be stated with any degree of certainty that their parents were necessarily alcoholics simply because they were observed to consume alcohol. Therefore, genetic factors can only be assumed. Further, The Type I and Type II classification have not been found to correlate with alcohol related social factors such as age of onset but were found to correlate with anti-social personality disorder (Blum 1991).

Hence, the classification of *Secondary Alcoholism* as defined by Funderburk in 1949 appears to more closely fit the sample profile of this study. The QEEG results, therefore, indicate that the classification of *Secondary Alcoholism* for alcoholics of Canadian aboriginal ancestry might be identifiable from a similar QEEG profile.

Individual QEEG Profiles

Although the sample QEEG profile shows promise in providing a tool in the form of a set of scalp electrode site markers for indicating a predisposition for alcoholism, the individual QEEG profiles of each participant do not support all of the sample indications. Individual profiles showed variations in hemisphere dominance and in dominant scalp sites. With the exception of one participant (#4), the only consistent findings across the other 10 participants was alpha showed higher than beta for almost all sites and for all three conditions and delta was generally higher than all other bands for all three conditions, together with wide variability in EEG amplitude across the EEG spectrum. Therefore the individual profiles of the 10 alpha dominant participants suggest that an alpha dominant profile, where delta is also generally higher than all

bands, found together with other factors such as psychological factors and negative-parental and negative-peer role modelling, would indicate *Secondary Alcoholism.*

Participant #2 and #3 showed a rather mixed profile where beta was higher than alpha at some sites. However, the microvolt differences between beta and alpha bands were generally less than one microvolt (0.1 uV), and most frequently beta was in the range from 0.1 to 0.4 microvolts higher than the alpha band. Only participant #4 showed microvolt differences that were over one microvolt, with a range of 0.2 to 4.4 microvolts, and for the eyes open condition only. Participant #4 showed 17 sites with higher beta compared to alpha, out of the 30 sites that assessed. Participant #4 was the only participant to show higher beta at the O1 site but only for the eyes open condition, and the difference in microvolts was only 0.2 uV higher than alpha. Given his general tendency for higher beta, participant #4 might be classified as a *Primary Alcoholic.*

The QEEG profiles shows that only three participants had some sites with higher beta over alpha and only for the reading silently condition. Higher beta microvolt values ranged from 0.1 uV for participants #3 and #8 and 0.4 uV for participant #11. Only participant #4 showed a higher uV value for beta compared to alpha for the eyes open condition. Beta was not higher at site O1 for any of the participants for the eyes closed condition.

EEG Variability

Previous research reports (Mulholland, Goodman, & Boudrot, 1983; Ochs, 1991) concluded it is the greater variability in the uV amplitude of the EEG signal across the frequency range that indicates dysfunction, and that less variability in amplitude is associated with improvement in the dysfunctional symptoms. The post-

assessment QEEG recordings of the participants in this study who completed NT show there was a general trend toward a reduction in the variability of their uV amplitudes across all frequency bands. Whereas the participants who only completed the AA program showed an increase in the variability of their EEG activity.

Assuming that a greater reduction in variability is instrumental in improving dysfunction, it is possible that if the participants in this study had more NT sessions they might have had greater success. One participant had 40 sessions, one had 32, one had 26, and two had only seven sessions. The follow-up reports from the pilot participant who completed 40 NT sessions indicated he was sober for more than one year. His follow-up report further indicates that it was his lifestyle preference that caused him to return to drinking in order to be included with his selected social group.

Lifestyle and Sobriety

One participant (#5) who only had seven NT sessions reported benefit from practising the visualizations techniques he was taught during the sessions. In his follow-up report he commented that although he was using alcohol in social situations, he found benefit from periodic practise of the techniques he was taught. Participant # 3 reported benefit from periodic practice of the spirit song he recalled during his NT session. However, he was still having difficulty with drinking with his friends and was incarcerated for impaired driving. Also, the three year follow-up report of participant # 1 found that he did not make a lifelong commitment to sobriety. He did not attend AA meeting, nor did he practice the visualizations or other techniques that helped him during his NT sessions. Although he experienced a distaste for alcohol during his NT sessions and was no longer craving a drink, he was not able to give up the lifestyle he was

accustomed to and which was associated with the consumption of alcohol. He did not attend AA meeting, nor did he practice the visualizations or other techniques that helped him during his NT sessions. Although he experienced a distaste for alcohol during his NT sessions and was no longer craving a drink, he was not able to give up the lifestyle he was accustomed to and which was associated with the consumption of alcohol. His follow-up report indicated that he went to the beer parlor because he was afraid to be abandoned by his girl friend and would suffer feelings of loneliness. His pattern of drinking behaviours was based on these fears which developed during his early childhood when his father was incarcerated and his mother abandoned him. Following this, he grew up in a small town of mostly Ukrainian people where he was the only adopted Native in that community and where he experienced loneliness because he was not well integrated with his peer group.

In support of AA teachings emphasizing that the alcoholic must make a lifelong commitment, Vaillant (1983), from extensive research with alcoholics, found that regardless of the treatment method the alcoholic must be willing to change. He further found that it is easier to treat alcoholism during the early stage. However, he also concluded that recovery is unlikely until the individual's subjective pain is severely symptomatic. In general, the relapse reports from the participants in this study appear to endorse his conclusions.

Relapses and Attempts To Quit Drinking

The data gathered from interviews with the participants indicates that eight of the participants had attempted to quit drinking prior to volunteering for this study. Therefore, this was the first attempt at rehabilitation for only three of the participants

(# 2 male NT, # 5 male NT, and # 10 female NT). Recent research (Simpson, Joe & Lehman, 1986) indicates that relapses are to be expected with alcoholics in general, especially with their first attempt to rehabilitate. It is not unusual for alcoholics to relapse within one month to one year. It is expected that approximately 54% will relapse once and 33% are expected to have multiple relapses. Research further indicates that less than 33% of addicts are expected to achieve permanent abstinence following a single attempt to rehabilitate (Gorski, Kelly & Havens, 1993).

Based on these research findings, it would be expected that at least three of the participants (33%) should have achieved sobriety following only the augmented AA program at Poundmaker's Lodge. It would further be hoped that a greater percentage of those taking the NT as well as the AA program would have obtained sobriety.

Of the participants who were able to be reached for the three year follow-up, only participant # 6 (male AA) reported remaining sober during the three year period. The pilot participant was sober for slightly more than a year, one other participant for approximately six months, one for approximately one month, and three reported less than one week. Because only one female participant was able to be located for the follow-up report, it is not possible to conclude the outcomes from this study on a percentage basis reflecting all participants.

Indications from NT

Although the pre-assessment QEEG results did not indicate that the left occipital scalp site O1 was a significant site in comparison to all other sites, this site was used for the NT sessions in this study in keeping with traditional practices in working with substance abuse. Electrode scalp site O1 has traditionally been used as a

training site in clinical practice and research studies with EEG feedback methods because this site has been reported to be effective in facilitating imagery and insight found with positive life changes as first reported by researchers such as Japer (1958), and Green and Green at the Menninger Institute (1977).

The results from participant # 1 (male NT) indicate that NT had helped this individual to reduce his craving and need for the consumption of alcohol. This participant was the only participant to engage in the NT as an outpatient in order to provide a pilot study prior to the main study in the rehabilitation centre. A major difficulty with outpatient alcoholics is their tendency to relapse when access to alcohol is readily available (Vaillant, 1983; Gorski, Kelly & Havens, 1993). However, because this participant did experience relapses it was possible to determine to some extent how the NT was progressing and how the NT was affecting his alcoholism at different stages of the NT program. His self-reports show the NT intervention reduced his need for alcohol and that it was his lifestyle preference that caused him to relapse even though he had remained sober for over a year.

His self-reports further provided some empirical evidence of the stages that were observed with the changes in his alcohol craving as brought about during NT. Based on the reports by the pilot participant, it appears that during the early NT (approximately 1-9 sessions) the alcoholic may experience a reduction in the craving for alcohol and will also begin to recall unresolved traumatic events. During the second stage (approximately 10-18 sessions) the alcoholic may begin to experience feelings of nausea from relatively small amounts of alcohol and will continue to recall past traumatic incidents. During the third stage (approximately 19-25 sessions) there is

an experience of unpleasant taste sensations and an advanced level of reduction in the craving for alcohol. During the fourth stage (approximately 26 to 32 sessions) there may be a greater reduction in the craving for alcohol with increased desires for a pleasant and successful lifestyle free from alcohol and the recollection of pleasant scenes from childhood. During the final stage (sessions 32 and beyond) the individual gains greater freedom from craving alcohol and is able to clearly visualize living a life of sobriety.

Alpha/Theta Crossover Effect

The reports from the participants who engaged in NT show some were fully attentive to the process and others were not. The reports also revealed that the two participants who were most attentive in the process, participants # were able to produce a crossover effect with theta and alpha rhythms. When this crossover effect occurs, theta and alpha bands produce the same uV amplitude at the same time. Therefore, theta and alpha act as one larger band where the frequency range of the cumulative band is from 4 Hz to 12 Hz. This cumulative frequency range seems to have the effect of allowing unresolved and unconscious intrapsychic conflicts to be brought to consciousness.

Previous research studies found that the theta frequency band is associated with the dream state in which scenes from the unconscious mind come to semi-consciousness while asleep, and has been reported to scan for pleasure (Walter, 1953) as well as being instrumental in survival (Winson, 1990). The alpha frequency band has been reported to scan for information (Walter, 1953) and to produce a state of passive awareness (Morgan, 1965) or relaxed alertness (Kamiya, 1969) in which the

individual experiences feelings of calm and peacefulness. Therefore, the alpha band is associated with a passive state of awareness where the individual is aware of the activity that is happening in the environment but does not respond physically or cognitively. It appears that during the alpha/theta crossover period, the deeper state of awareness of reaching into the unconscious is integrated with the passive awareness state.

This coupling of the theta and alpha bands appears to produce a wider window of awareness, thereby making it easier to recall events from the unconscious (theta scanning) in what is perceived to be a safe environment (alpha scanning). Therefore, forgotten memories, such as the suppressed traumatic events of childhood, are able to surface into the theta dream-like state produced by the theta rhythms and, at the same time, recollection of these events is perceived to be safe due to the passive awareness produced by the alpha rhythms. When the NT session ends, the participant then returns to full consciousness, or beta activity. At this point the individual becomes fully conscious of what took place during the semi-dream like state produced by the alpha/theta crossover effect. The momentary shock of recalling the suppressed traumatic events brings about the emotional reaction that is more commonly referred to as catharsis or abreaction.

The following recent conclusions by Lee (1996), of research studies on brain activity regarding the resolution of traumatic memories supports these views of the alpha/theta crossover effect. Difficulty in communications between the right hemisphere and left hemisphere is believed to be a component of post-traumatic stress disorder (PTSD). Research on the retrieval of emotional laden memories has shown

that the verbal left hemisphere (Henry, 1992) does not have immediate access to the emotional laden information that has been found to be stored in the right hemisphere (Joseph, 1993) and in deeper recesses of the limbic system (Taylor, Bagby, & Parker, 1991). Also, the left hemisphere has been found to mediate positive emotional states whereas the right hemisphere mediates negative states (Davidson & Fox, 1988; Davidson & Eckman, 1990; Henry, 1992) and is dominant in the storage and recall of negative emotional memories (Joseph, 1993). Therefore, the negative emotional memories of past traumas that are mediated by the right hemisphere have been found to respond to therapeutic interventions that use metaphorical and visual imagery as modes of communication (Winner & Gardner, 1977; Danesi, 1989) or what might be called a visual-spatial language (Dean, 1984; Pavino, 1986; Eviatar, Menn, & Zaidel, 1990).

Primitive automatic survival responses and ritualistic habits are found to be activated within the primitive "Reptilian Brain" portion of the Triune Brain found in humans (MacLean, 1990). Emotional responses, memory, bonding and social behaviors are activated within the limbic system of the Paleomamilian Brain. The amygdala plays a major role in the limbic system's response mechanisms. The amygdala receives information from cortical structures within the neocortex including the hippocampus and the thalmus. Thalmic input to the amygdala mediates primitive emotional responses, such as anger, which are output back to cortical structures such as the prefrontal cortex resulting in conscious emotional experiences (Kandle, Schwartz, & Jessell, 1991).

The amygdala has been found to process emotional laden content and does not differentiate input sources but responds to all input information as if they are real

(Lee, 1996). It is this aspect of the amygdala as an information processing unit that is instrumental in causing past traumatic memories to seem real during the time of recall (Damasio, 1994; & Lee, 1996). According to Lee (1996) the human "Primitive Brain," which includes the Reptilian and Paleomamilian Brains, needs to experience closure and mastery with respect to traumatic memories in order to no longer perceive and react to them as life threatening. Because the primitive brain and the right hemisphere of the neocortex process non-verbal symbolic forms of communication, traumatic memories are found to respond to therapeutic techniques that use a symbolic structure, such as art therapy, play therapy, and hypnotic techniques that use visualized therapeutic symbols. Closure is brought about by creating a scene in which the subject masters the threatening situation of the past traumatic incident. Although this visualized scene is not a real event, the Primitive Brain perceives the symbolic closure as if it were real (Lee, 1996).

Because NT techniques with alcoholics use visualizations, such as visualizing alpha rising and scenes of confidence as described by Peniston and Kulkosky (1989), these metaphorical messages in symbolic form communicate with the unconscious emotional laden content of the right hemisphere, the amygdala, and deeper recess of the limbic system. Therefore the crossover effect between theta and alpha, as produced during NT, integrated with visualizations allows the individual to access the emotional laden traumatic memories stored in the right hemisphere and deeper structures of the limbic system. The participants in the present study visualized themselves in the form of an eagle rising above the physical earth (memories of traumatic physical experiences rising out of the unconscious) and soaring upward to

higher reaches of the clear blue sky (memories rising to clear consciousness). Therefore the participants in the present study who were able to produce the crossover effect experienced recollection of their past traumas and produced emotional reactions (shock of realizing this actually happened to them) which brought about a degree of resolution of their past traumatic incidents. They were also taught to visualize scenes of being confident without alcohol. During the three year follow-up, one participant (#5) reported that using this visualization technique helped him to reduce his anxiety associated with his business interactions.

Conclusions from NT

It is interesting to note that although participant #1 did not produce the theta/alpha cross over effect, that NT was, however, effective in bringing about "flu like symptoms" that occurred when he consumed alcohol, similarly as reported by Peniston and Kulkosky in 1989. It appears that this *internal aversive symptom (IAS)*, as produced from NT, is effective in bringing about changes in alcohol consumption.

Equally interesting are the follow-up reports of participants #3 and #5 indicating they gained some control over the consumption of alcohol and over the frequency of drinking. Participant #3 reported he was able to abstain for long periods but still had trouble refusing his friends. Participant #5 reported he was able to drink socially for business reasons. Their reported experiences during NT show that the theta/alpha crossover resulted in abreaction to their previous traumatic incidents. This psychological effect appears to be efficacious in bringing about a reduction for the craving of alcohol. Peniston & Kulkosky (1989) concluded the crossover effect is "instrumental to the alcoholic's recovery". Rosenfeld (1997) emphasized it is this

particular aspect of NT training that is most helpful in the alcoholic's recovery to total abstinence and can be viewed as an "important adjunct to psychotherapy." Therefore, it might be the psychotherapeutic effect from the theta/alpha crossover together with the effect that visual metaphors has on the alcoholic that is the most instrumental in helping to bring about sobriety.

Conclusions from these findings suggest that NT has the potential to produce two positive effects: psychotherapeutic and *IAS*, as well as effecting changes in frequency production. These two effects appear to help alcoholics gain greater self-control over their addiction when either of them takes place. It may be assumed that if both effects were to occur during NT then the individual experiencing both effects would be expected to have even greater self-control over their addiction to alcohol. Lubar (1997) also emphasized the importance of combining NT with behavioral management techniques, individual or group psychotherapy, and with educational interventions.

Indications for Education

The literature indicates that since the formation of AA, education has been a key factor in the alcoholics recovery (AA World Services, 1993). Also, the first effort put forth by the medical profession following the American Medical Association's acceptance of alcoholism as a disease was to establish informative teaching about alcoholism and alcoholics (Berridge, 1990). Education has therefore been seen as essential in attempting to treat the alcoholic and to reduce the suffering caused by alcohol abuse.

The literature on research studies with alcoholics further indicates that the age of onset of alcoholism was generally found to occur during the early teenage years. Sheenan, et al. (1988) found that in the general population 92% of adult addicts (drug and alcohol) reported their addiction started prior to the age of nineteen. Further research by Helzer, et al (1988) and by Statistics Canada (1991a, 1991b) shows this to be true regardless of race, culture, gender, or socio-economic status. It is apparent that the time of onset of alcoholism generally occurs in the secondary school period during junior high and senior high school years.

Research by (Goodwin, 1983) also indicates that many alcoholics experienced academic difficulty during their school years and often have a history of associated AD/HD, or conduct disorder. It is further interesting to note that the alcoholics in this sample were found to have similar EEG profiles as found with children diagnosed with attention deficit disorder and/or hyperactive disorder (AD/HD). Therefore, it is possible that some of the participants in this study were attention deficit or both attention deficit and hyperactive when they were children.

Considering the lower level of education of 73% of the participants in this study, together with their QEEG profiles which are similar to AD/HD profiles, it seems reasonable to assume that had NT been available during their grade school years, NT might have helped some of them to improve their academic skills and also to avoid more serious problems with alcoholism. Hence, it seems reasonable that NT training should be more readily available during the grade school years, inclusive from primary through to the secondary school years. Using NT methods during a child's developing years prior to the age of onset for alcoholism might prevent many of the associated

problems that occur later in their lives, and are shown to be costly in economic terms to the general population. The evidence from previous research demonstrates that NT can help many individuals with AD/HD (Lubar, 1997), and can also help many alcoholics and drug addicts to gain greater control over their addictions (Peniston & Kulkosky, 1991).

Recommendations for Future Research

The outcomes from this study indicate that future research needs to address a wide range of research questions and possible uses for NT. The literature review and the results of this study suggest future research should focus on exploring NT to discover a variety of methods which would be beneficial for helping to reduce the suffering created by alcohol addiction for a greater range of alcoholic types. The following suggestions are presented with this aim in mind.

1. There is a need to find definitive scalp electrode site markers which would indicate QEEG profiles that would identify different types of alcoholics.
2. There is a need to discover different NT methods or protocols that would match the different QEEG profiles of the various alcoholic types.
3. There is a need to discover NT methods that can be administered under outpatient as well as inpatient conditions, for cost effective purposes.
4. There is a need to determine appropriate psychometric instruments for conducting research with alcoholics in general and and specifically with aboriginal populations.
5. There is a need to research the connection between AD/HD children and children at greater risk for alcoholism who might be identified from their QEEG profiles.

6. There is a need to research the application of NT at the grade school level during and prior to the period of general onset for alcoholism.

Final Conclusions

The outcomes of this explorative study on NT for the reduction of alcoholism with alcoholics of Canadian Aboriginal ancestry show the participants engaged in the study were not representative of the general population of these peoples. The participants are more representative of people of Canadian Aboriginal ancestry who have a lower level of education than is expected from this population, and who learned to use alcohol as a mechanism for coping with problematic life experiences, and where traditional cultural views were not followed.

The QEEG results did not confirm with previous research conclusions found in the literature and described by and Kulkosky (1989) where alcoholism was determined from high beta and low alpha amplitudes at the O1 site. The QEEG profiles obtained from this sample indicates the participant alcoholics in this study are more representative of *Secondary Alcoholics* who become alcoholics because of psychological and environmental influences and who are found to produce higher amplitudes of alpha rhythms, in comparison to *Primary Alcoholics* who produce higher amplitudes of beta rhythms. The QEEG assessments indicate a QEEG profile showing alpha dominant over beta for almost all sites together with psychological and environmental factors is more representative of *Secondary Alcoholism* than would be a single site marker. Ten of the eleven participants in this study met the classification for *Secondary Alcoholism* and only one participant met the classification for *Primary Alcoholism.*

The outcomes from the participants who completed NT showed that the crossover effect, where theta and alpha produce the same amplitude at the same time, is instrumental in helping to resolve intrapsychic conflicts, thereby serving as a viable adjunct to psychotherapy. The results of the NT also showed that NT has promise for helping some alcoholics to reduce their craving for alcohol. However beneficial NT might be, it is apparent that alcoholics must want to change their lifestyles which are associated with the consumption of alcohol. Further, it is apparent the alcoholic must be willing to commit to a lifetime practice of methods that help to support the changes brought about by NT. Previous research findings and the background information provided by this study suggest that NT might prevent many children from becoming alcoholics in their adult lives if this form of therapy was more readily available during their developing years in grade school.

In final conclusion, the results of this study indicate it would not be possible to predict that the participants were alcoholics from their pre-assessment QEEG profiles if information on their alcoholic backgrounds was not available. However, the results indicate that the use of NT shows promise for helping alcoholics of Canadian Aboriginal ancestry to gain greater control over their addictions and should be further researched with larger samples under both inpatient and outpatient conditions to determine its use for a variety of alcoholic types as well as specific populations. The results shows that NT was beneficial as an adjunct to psychotherapy. The study also indicates that NT should be applied and researched with young adolescents during their grade school years, the most common age of onset for alcoholism.

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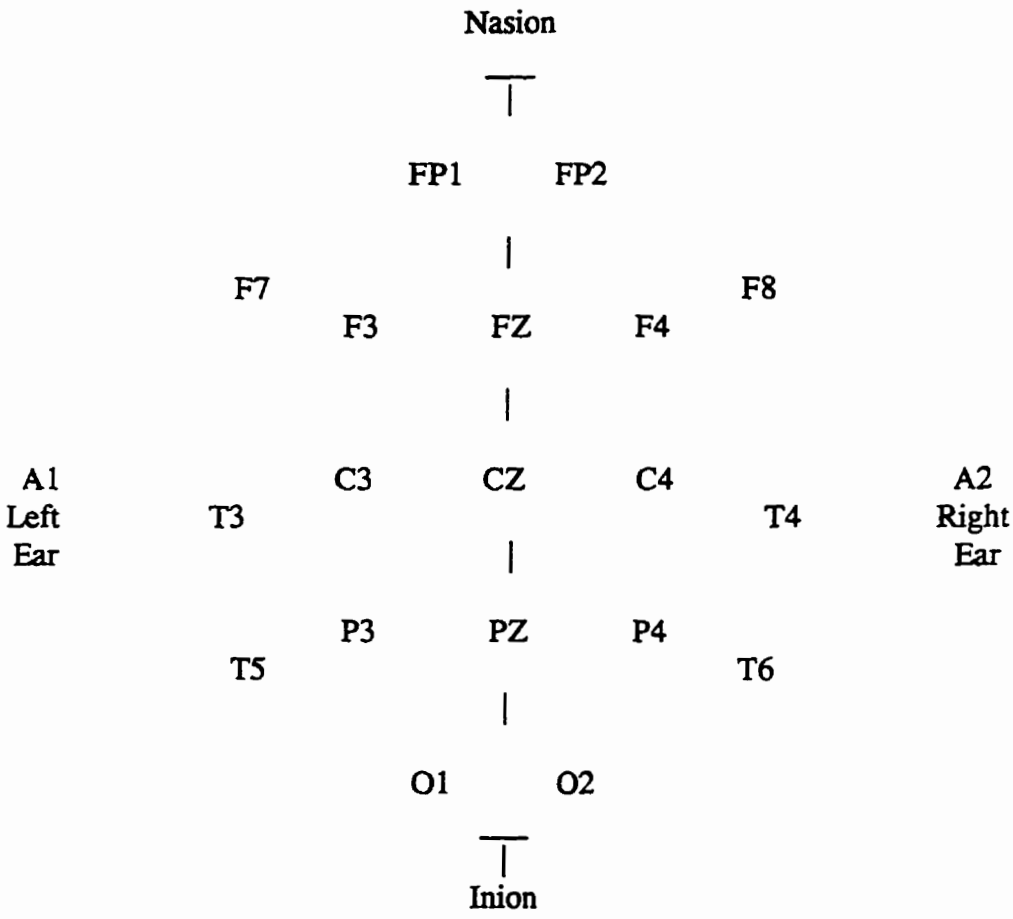
APPENDIX I

International 10/20 Scalp Electrode Placement System



(Alpha/Numerical references indicate the common scalp electrode sites.)

APPENDIX II

Sample Informed Consent Form

ALPHA-THETA EEG BIOFEEDBACK
BRAIN WAVE TRAINING FOR THE REDUCTION OF ALCOHOLISM
RATIONALE AND PROCEDURES FOR EEG BRAIN WAVE TRAINING

Addiction to alcoholism is difficult to overcome. Recent investigations have found that chronic alcoholics, even after abstinence, have lower levels of Alpha and Theta brain wave activity on background cortical electroencephalographic readings. These readings were further found to often have abnormally high levels of Beta brain wave activity. It has also been found that Alpha levels increase after alcohol consumption. These findings suggest that some individuals may have a predisposition to the development of alcoholism due to abnormal brain wave levels.

The application of Alpha-Theta EEG Brain Wave Training has been found by recent researcher's to have a long term efficacious effect in the reduction of the habitual consumption of alcohol with chronic alcoholics. Chronic alcoholics who received the EEG training and who previously experienced a number of relapses following contemporary forms of treatment reported they no longer desire to drink and/or are able to drink due to the effect of nausea following consumption of small amounts of alcohol.

EEG Alpha-Theta brain wave training is a biofeedback technique utilizing a computer screen and audio signals to provide feedback information to the individual in the form of visual and auditory stimulus. This technique provides the individual with immediate feedback information about the levels of Alpha-Theta brain wave activity. The individual learns through this feedback process to control the brain wave frequency that is giving the feedback. Together with visualization training the participants learn to increase their Alpha and Theta levels. During the training sessions and following an increase in Alpha-Theta levels the participants reported induced feelings of relaxation, and an enjoyable, tranquil, calm and serene state. Participants reported continuous abstinence during 18 month and three year follow-up investigations that were conducted by the early pioneer researchers of this technique. EEG Alpha-Theta biofeedback brain wave training is being conducted in a number of centres in the United States and reports indicate similar results.

Some participants who desired to consume alcoholic beverages following their brain wave training reported successfully overcoming the training effects by consuming increasing amounts of alcohol. These reports suggest that the training effect can be reversed if desired.

Although EEG Brain Wave training has been found to be efficacious for the reduction of alcoholism at other research centres no claims are made to that effect by the present researchers or by the University of Alberta.

As a volunteer participant in EEG brain wave training I have read the above information and understand that there are no claims made by the researchers or by the University of Alberta that participation in brain wave training will reduce my alcohol habituation, I further do not hold the researchers or the University of Alberta responsible for any changes which may occur in my behaviour or brain wave activity during nor following the training period. I understand that all changes made to my brain wave patterns are under my control at all times during the training sessions and that I may withdraw from training at any time. I further release the researchers and the University of Alberta from any responsibility to my person while engaged in research activities.

Experimental Participant's Signature Date

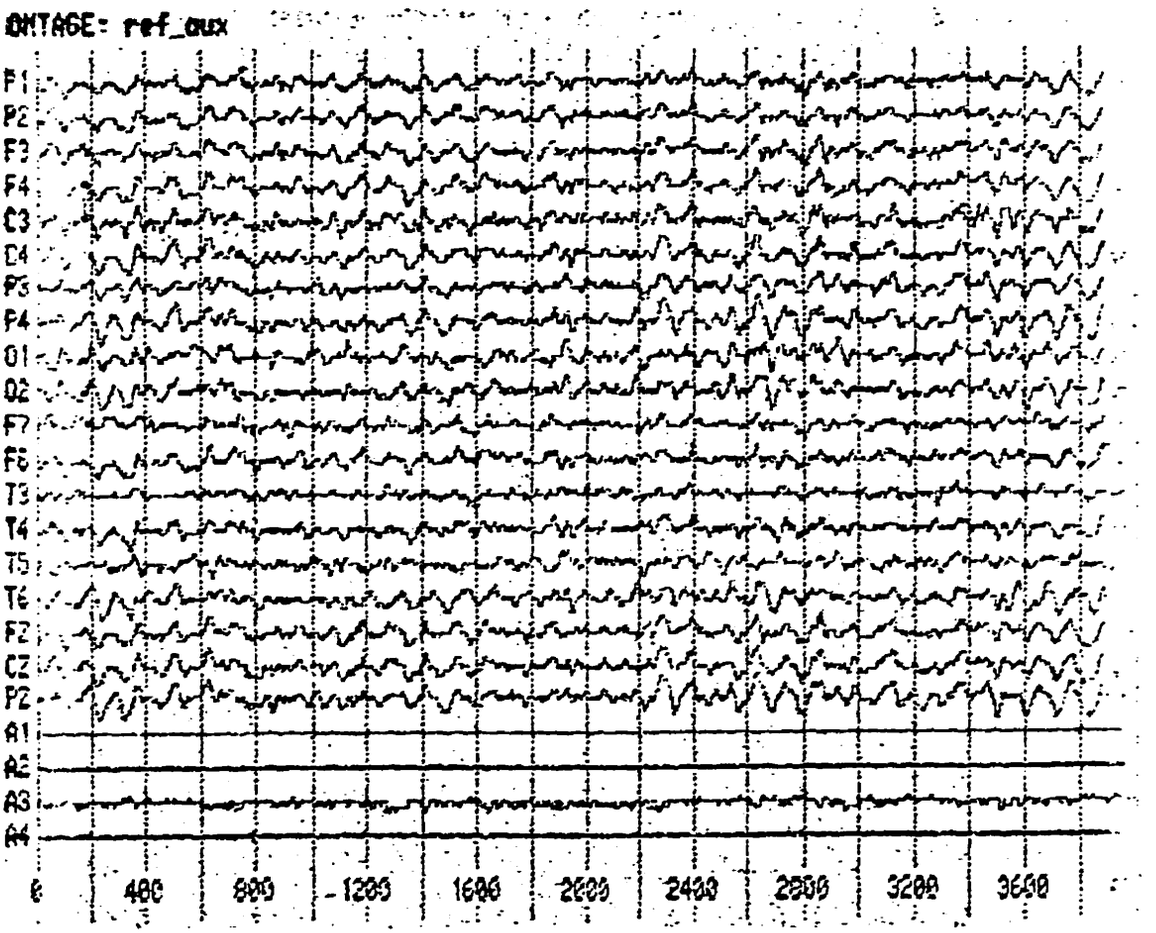
Witness's Signature Date

APPENDIX III

Samples of EEG recordings from the Lexicor NRS-24 system.

1. EEG Line Graph recording.
2. EEG Spectral Analysis recording.
3. EEG Topograph Map recording.

1. EEG Line Graph recording

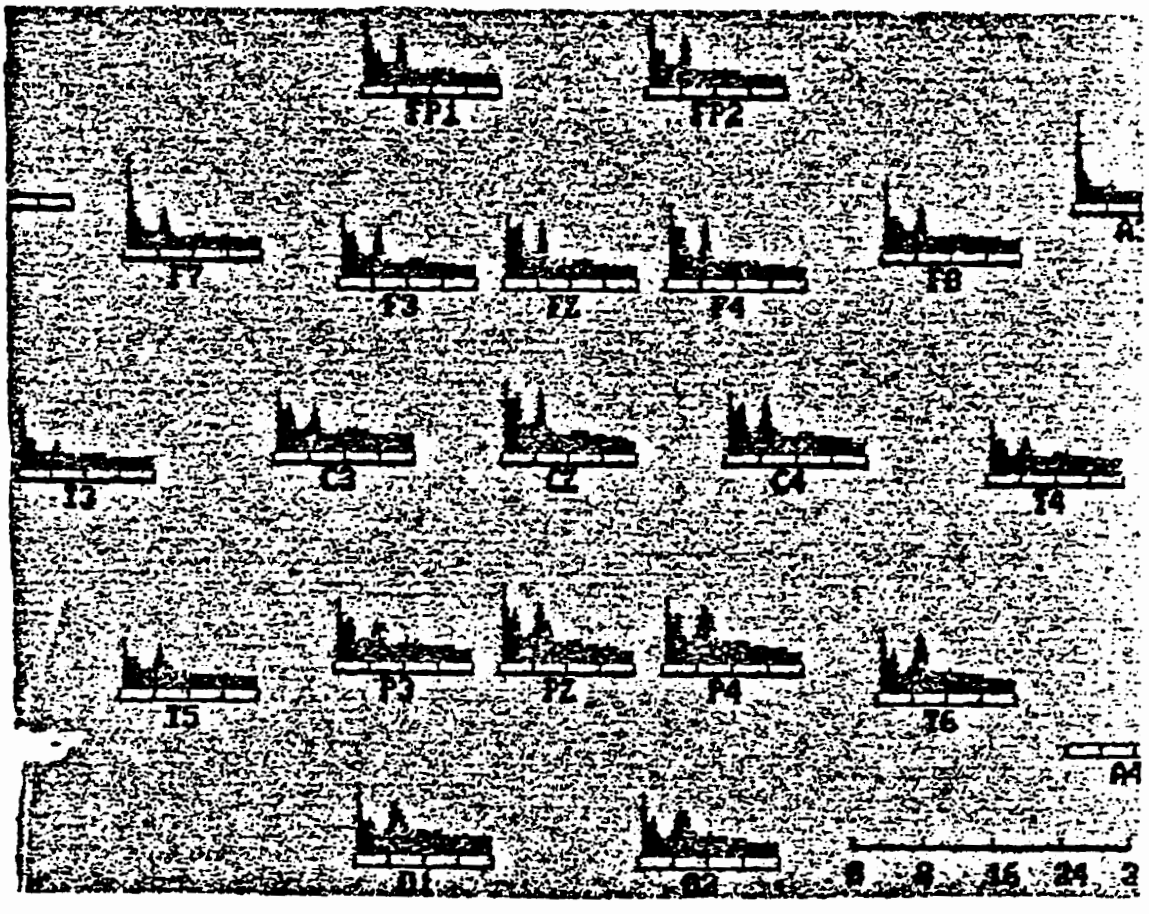


Participant #3 (male NT): Pre-assessment EEG recording of the eyes closed condition.

Chart shows averaged magnitude over 150 epochs or 5 minutes of artifacted data.

(Gain 32K, sample rate 128/sec.)

2. EEG Spectral Analysis Recording

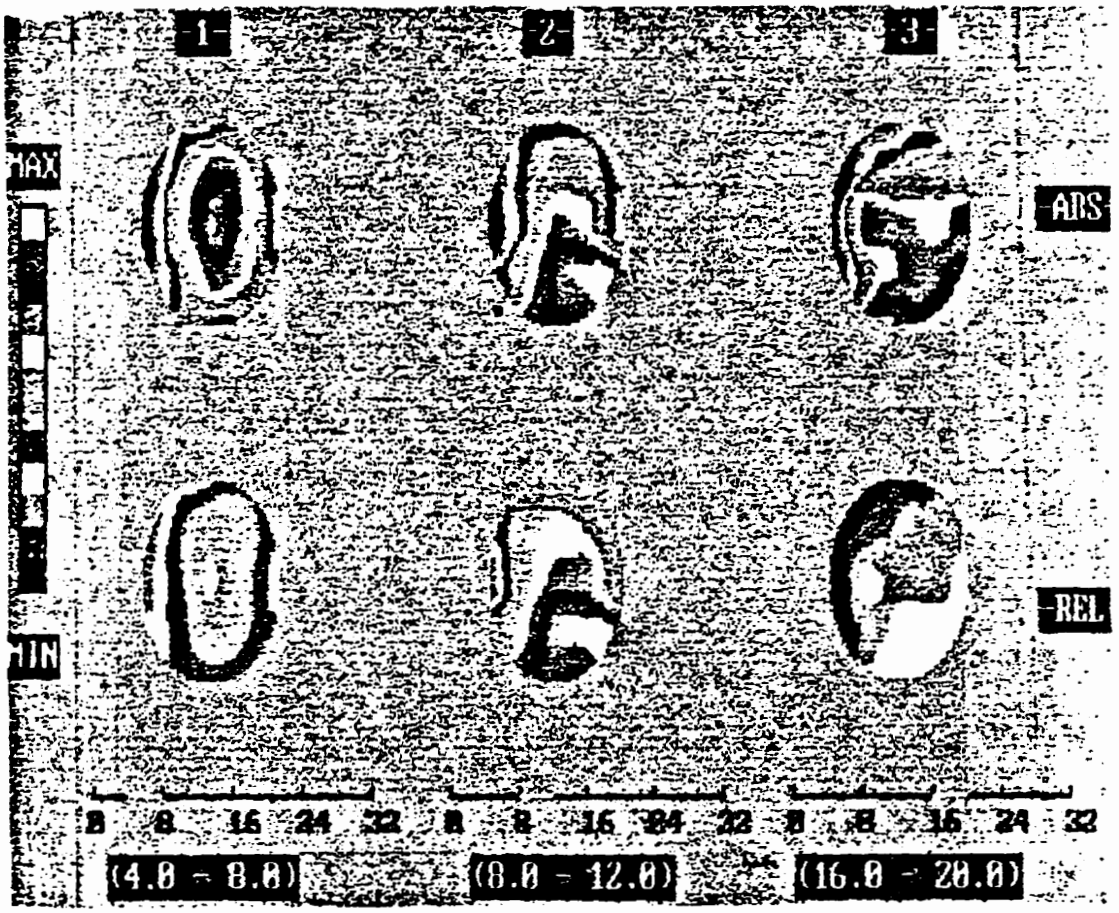


Participant #3 (male NT): Pre-assessment EEG recording of the eyes closed condition.

Chart shows averaged magnitude over 150 epochs or 5 minutes of artifacted data.

(Gain 32K, sample rate 128/sec.)

3. EEG Topograph Map recording



Participant #3 (male NT): Pre-assessment EEG recording of the eyes closed condition.

Chart shows averaged magnitude over 150 epochs or 5 minutes of artifacted data.

(Gain 32K, sample rate 128/sec.)

APPENDIX IV

Individual and Grouped QEEG Results

The charts and accompaning figures showing pre and post-assessment averaged QEEG magnitudes are presented in the order of eyes closed condition, eyes open, and reading silently. The results from the ten scalp sites recorded are in the following order:

1. F3 scalp electrode site.
2. F4 scalp electrode site.
3. C3 scalp electrode site.
4. C4 scalp electrode site.
5. P3 scalp electrode site.
6. P4 scalp electrode site.
7. T5 scalp electrode site.
8. T6 scalp electrode site.
9. O1 scalp electrode site.
10. O2 scalp electrode site.

Site F3: Eyes Closed Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.4	2.8	5.0	1.7	4.9	1.8	2.6	0.7	2.1	0.7	2.1	0.7	29.2	5.2
1-Post	3.5	0.9	3.3	0.9	4.1	2.0	1.2	0.4	1.2	0.5	1.1	0.3	19.7	3.6
2-Pre	5.9	2.1	5.0	1.8	3.2	1.2	3.1	1.6	2.3	0.8	2.2	0.7	25.0	4.8
2-Post	4.2	1.7	4.3	1.5	2.5	0.8	2.0	0.9	1.6	0.7	1.6	0.5	18.8	3.5
3-Pre	5.5	1.9	4.6	1.4	6.3	2.9	2.7	1.0	3.0	1.1	2.7	0.8	29.0	4.9
3-Post	6.8	2.7	5.8	1.9	4.5	2.7	2.2	0.8	2.2	1.3	1.9	0.8	25.7	5.7
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings Only)														
4-Pre	5.0	1.5	3.8	1.1	3.8	1.6	2.0	0.9	2.4	1.2	2.7	1.6	29.2	8.8
5-Pre	9.3	5.7	3.6	1.3	4.0	1.5	2.0	0.6	1.8	0.5	1.5	0.5	24.3	6.6
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	5.4	2.5	4.0	1.2	3.5	1.7	2.3	0.8	1.7	0.6	1.8	0.8	21.5	4.3
6-Post	7.7	2.6	4.2	1.2	3.3	2.0	2.3	0.7	1.9	0.7	2.0	0.9	24.8	6.4
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	6.2	1.9	6.4	2.4	11.8	3.8	3.4	1.2	3.1	0.8	3.8	1.1	39.3	6.3
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	5.8	2.0	5.5	1.9	8.0	3.3	2.9	1.0	3.7	1.6	3.0	1.0	32.2	6.6
8-Post	6.4	2.6	7.4	2.6	9.8	3.4	3.2	1.1	4.9	1.6	3.9	0.9	40.2	7.1
9-Pre	6.8	2.4	6.1	2.1	6.4	2.6	2.2	0.8	2.4	0.9	2.0	0.7	28.5	5.4
9-Post	9.8	4.3	6.2	2.0	4.2	2.1	2.2	0.7	2.1	0.7	1.9	0.6	29.0	5.7
AA Female Alcoholics (Pre-Assessment QEEG Recordings Only)														
10-Pre	6.3	2.3	4.8	2.9	3.1	1.2	2.0	0.7	1.8	0.7	1.7	0.6	22.8	4.2
11-Pre	6.0	2.4	6.5	2.8	7.3	3.3	3.2	1.5	3.0	1.2	2.4	0.7	32.4	8.5

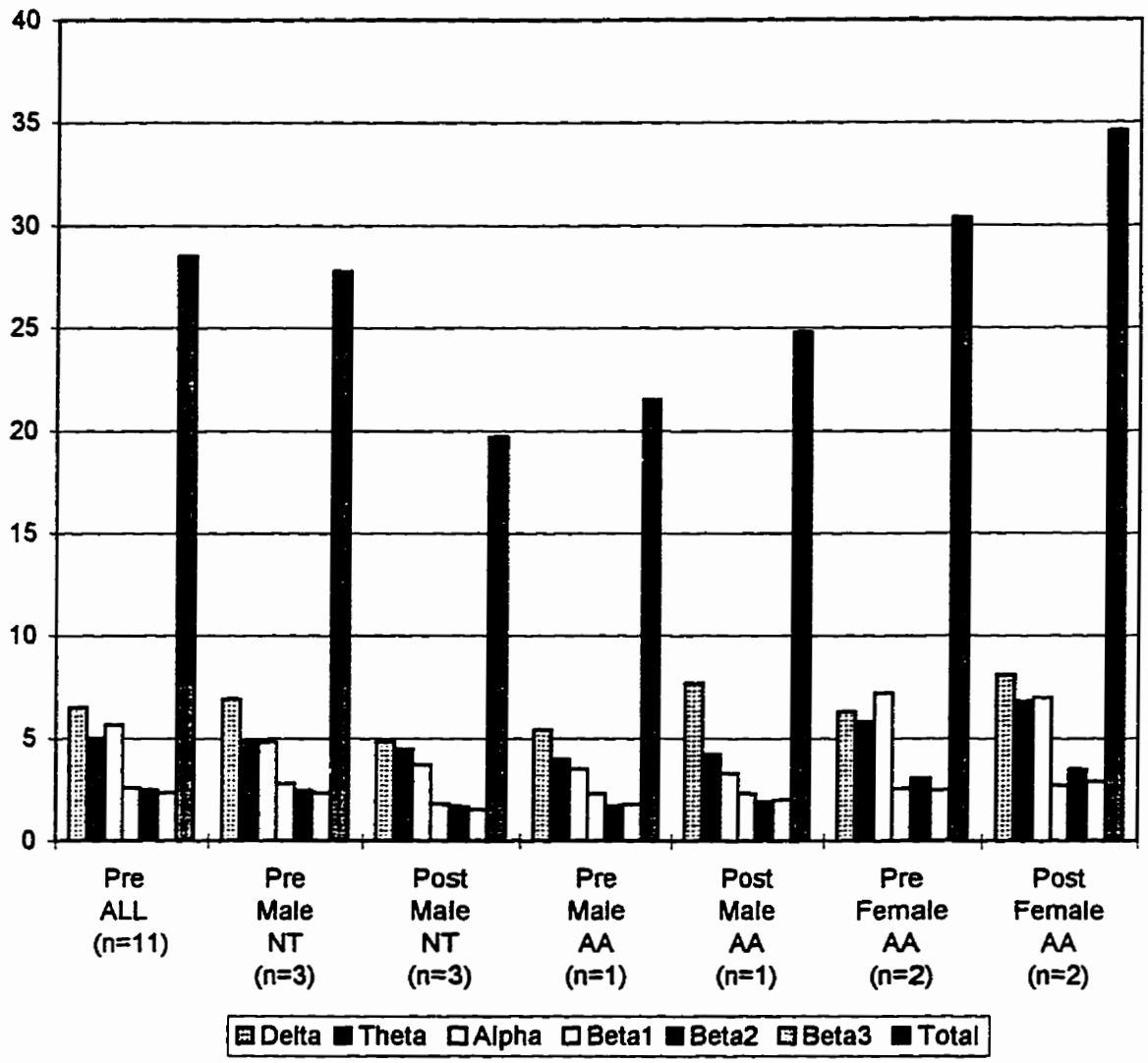
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site F4: Eyes Closed Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	8.8	2.7	4.8	1.7	4.9	1.8	2.8	0.9	2.2	0.8	2.0	0.7	28.7	5.5
1-Post	4.1	1.4	3.2	1.2	4.4	1.6	1.4	0.3	1.5	0.5	1.2	0.4	22.1	3.7
2-Pre	6.3	1.7	5.1	1.3	3.7	1.4	3.4	2.2	2.6	0.7	2.5	0.6	27.4	4.8
2-Post	4.3	1.6	4.3	1.4	2.5	0.8	1.9	0.8	1.6	0.6	1.6	0.5	18.9	3.4
3-Pre	6.9	2.5	5.6	1.5	6.7	3.1	2.9	1.1	3.2	1.1	2.8	1.0	32.4	5.0
3-Post	6.5	2.5	5.6	1.6	4.6	2.8	2.2	0.8	2.3	1.2	2.0	0.7	25.5	5.4
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings Only)														
4-Pre	4.8	1.7	3.9	1.2	3.7	1.7	1.7	0.5	2.1	0.8	2.2	0.9	26.2	5.9
5-Pre	8.5	4.4	4.1	1.6	5.0	2.0	2.2	0.8	1.9	0.6	1.5	0.5	25.2	6.1
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	5.5	2.3	4.2	1.2	3.7	1.7	2.3	1.0	1.9	0.7	1.8	0.7	22.2	4.4
6-Post	7.7	3.1	3.9	1.0	3.2	2.0	2.3	0.8	1.7	0.5	1.7	0.5	23.7	6.1
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	5.8	2.0	6.1	2.1	10.2	3.5	3.2	1.0	3.0	1.0	3.8	1.1	37.3	6.0
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	5.6	2.6	5.1	2.5	6.9	3.5	2.5	1.1	3.3	1.7	2.7	1.1	29.6	7.0
8-Post	6.7	2.6	7.6	2.5	10.2	3.5	3.1	1.1	4.7	1.7	4.0	1.1	41.2	7.0
9-Pre	7.3	2.9	6.9	2.4	7.0	2.9	2.5	1.1	2.7	1.0	2.2	0.7	31.3	6.2
9-Post	10.4	4.1	5.6	1.9	3.8	2.1	2.1	0.7	2.0	0.7	1.7	0.5	28.2	5.8
AA Female Alcoholics (Pre-Assessment QEEG Recordings Only)														
10-Pre	7.3	2.5	5.4	2.0	3.5	1.4	2.1	0.7	2.0	0.9	2.1	0.9	25.8	4.5
11-Pre	6.0	2.5	6.5	2.9	7.3	3.4	3.3	1.3	3.0	1.0	2.4	0.7	32.5	8.5

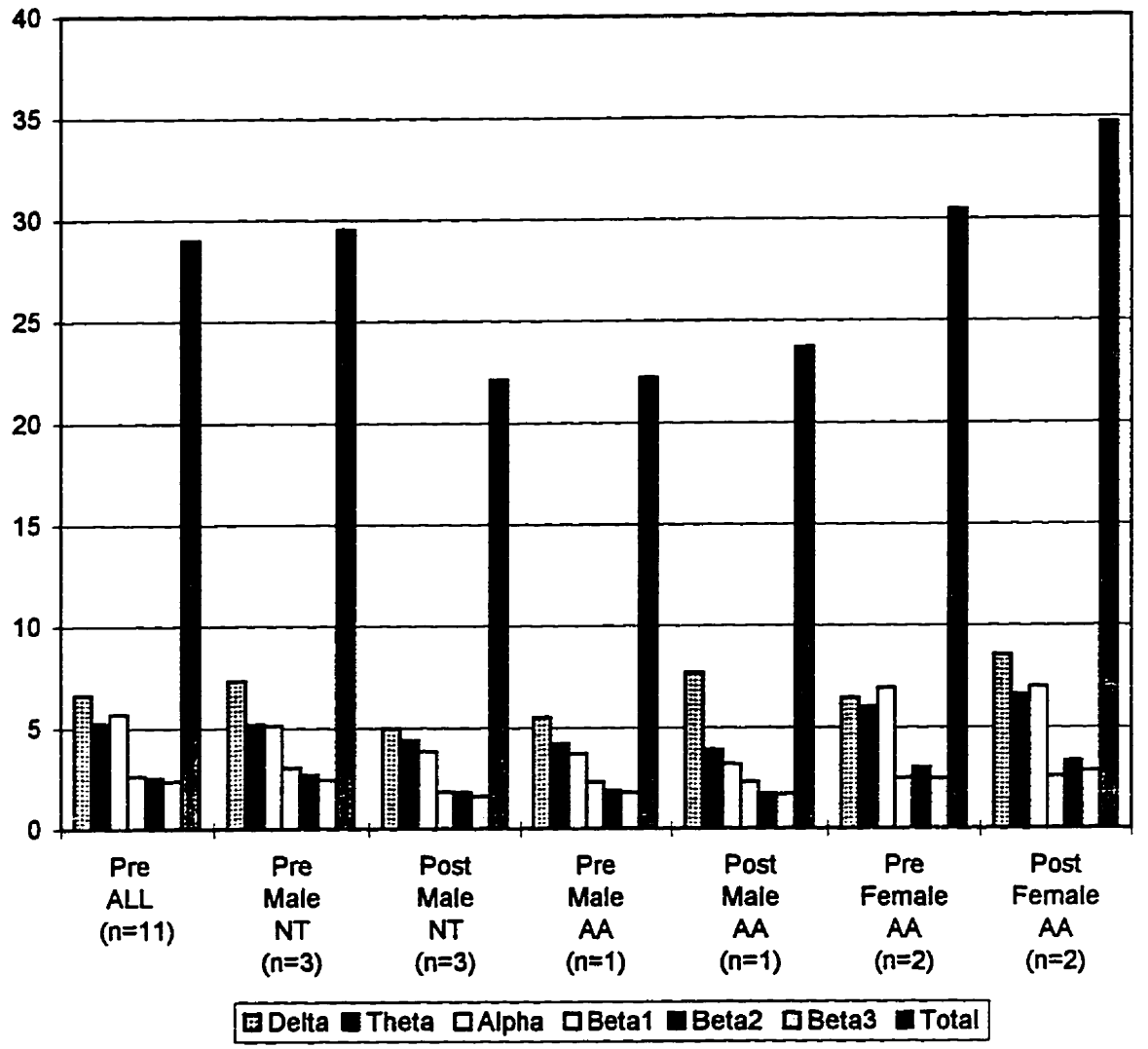
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site F3: Eyes Closed Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site F4: Eyes Closed Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site C3: Eyes Closed Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	8.6	2.8	4.8	1.4	5.5	2.0	2.7	0.8	2.1	0.8	2.1	0.8	28.9	5.3
1-Post	2.8	0.9	2.6	1.0	4.2	1.0	1.3	0.4	1.3	0.5	1.0	0.3	18.8	3.2
2-Pre	5.3	1.9	4.3	1.4	2.8	1.1	3.2	1.4	2.2	0.8	2.1	0.6	22.9	4.1
2-Post	3.9	1.5	4.0	1.3	2.4	0.8	2.3	0.9	1.7	0.6	1.5	0.5	18.2	3.2
3-Pre	5.6	1.8	4.6	1.5	6.3	2.9	3.5	1.4	4.1	1.8	3.4	1.2	44.7	9.8
3-Post	5.7	2.3	5.0	1.8	4.0	2.1	2.0	0.8	1.9	0.9	1.7	0.7	23.0	5.0
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings Only)														
4-Pre	4.4	1.6	3.4	1.0	3.7	1.5	1.9	0.9	2.2	1.0	2.5	1.3	26.5	8.0
5-Pre	11.7	6.1	4.0	1.4	3.7	1.4	2.0	0.6	1.8	0.6	1.4	0.4	26.6	6.7
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	4.9	2.2	3.7	1.1	3.6	1.8	2.3	0.9	1.8	0.7	1.7	0.7	20.6	4.3
6-Post	6.7	2.6	3.6	2.6	3.1	0.9	2.2	2.0	1.6	0.7	1.8	0.5	22.1	5.0
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	7.7	2.2	7.5	2.9	16.8	4.6	4.0	1.4	3.3	0.9	4.4	1.4	49.0	8.0
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	4.9	1.6	4.6	1.6	6.9	2.8	2.7	1.0	3.2	1.3	2.7	1.0	27.9	5.9
8-Post	6.4	2.4	7.0	2.5	10.2	3.4	3.8	1.2	4.0	1.6	4.2	1.1	41.1	6.7
9-Pre	6.4	2.3	5.9	2.1	8.3	3.0	2.4	0.8	2.7	1.1	2.1	0.7	30.3	5.8
9-Post	10.6	4.4	5.4	1.7	4.4	2.9	2.0	0.7	1.8	0.7	1.6	0.6	27.8	6.7
AA Female Alcoholics (Pre-Assessment QEEG Recordings Only)														
10-Pre	6.5	2.9	4.4	1.7	2.9	1.1	1.9	0.7	1.7	0.7	1.6	0.6	21.8	4.5
11-Pre	6.1	2.3	6.6	2.6	7.7	3.4	3.3	1.4	3.1	1.1	2.5	0.9	33.2	8.2

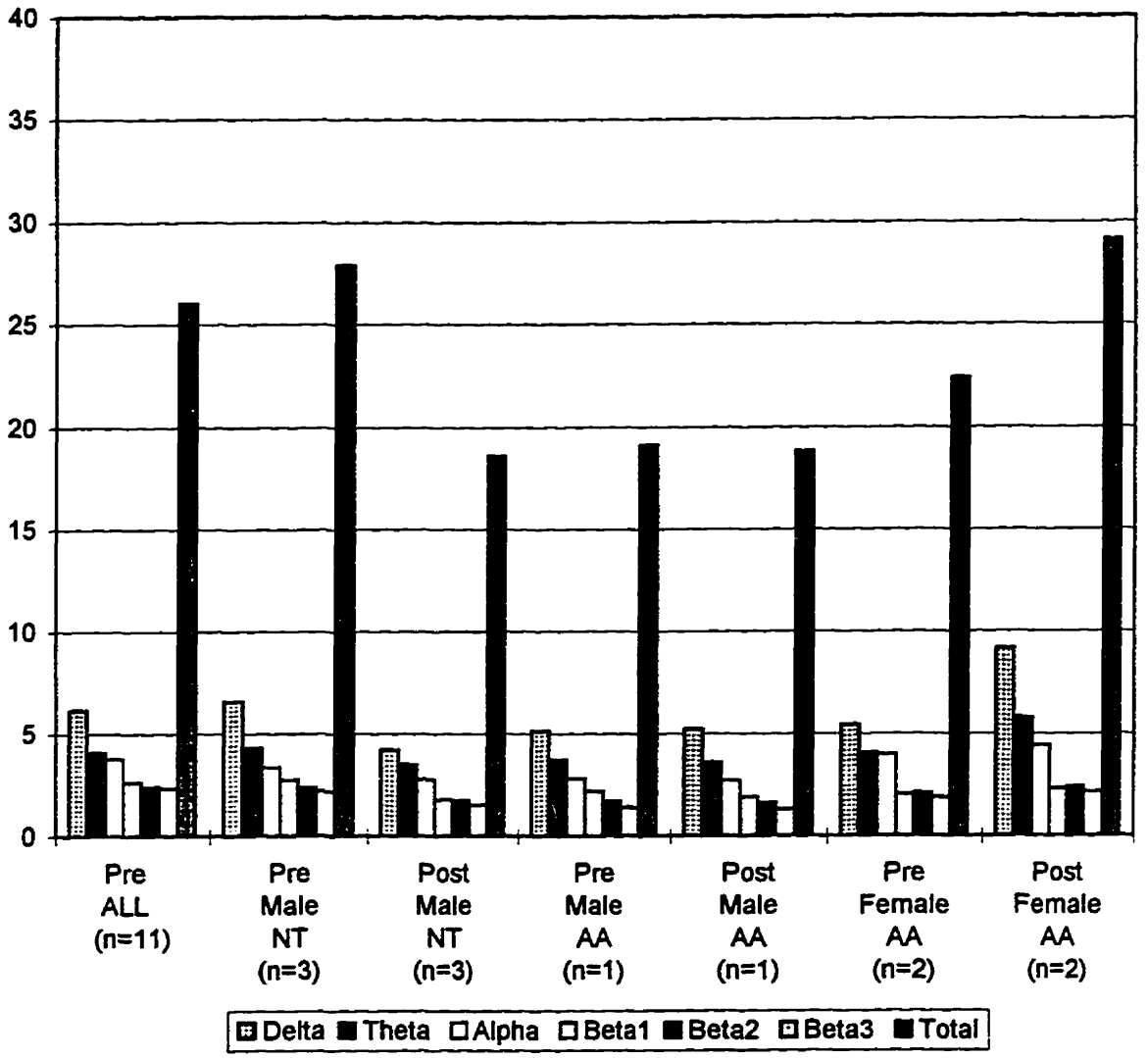
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site C4: Eyes Closed Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	8.2	2.7	4.5	1.3	5.4	1.8	2.8	0.9	2.2	0.8	2.0	0.7	28.3	4.5
1-Post	3.4	1.1	2.6	0.8	5.2	1.9	1.5	0.5	1.6	0.5	1.3	0.4	22.3	3.5
2-Pre	5.7	1.9	4.6	1.4	3.3	1.1	3.5	1.6	2.5	0.8	2.3	0.7	25.5	4.2
2-Post	4.1	1.5	3.8	1.3	2.3	0.7	2.0	0.9	1.5	0.6	1.4	0.4	17.6	3.2
3-Pre	5.9	2.1	5.0	1.9	7.9	4.0	3.5	1.1	3.7	1.3	3.3	1.4	45.1	9.7
3-Post	5.9	2.4	5.0	1.5	4.6	2.7	2.5	0.8	2.3	1.0	1.9	0.7	24.9	5.1
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings Only)														
4-Pre	4.2	1.5	3.4	1.0	3.8	1.7	2.3	1.3	2.9	1.9	3.0	2.0	30.6	12.8
5-Pre	6.1	3.1	3.8	1.3	4.7	1.7	2.1	0.7	1.8	0.5	1.4	0.5	21.9	4.7
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	5.0	2.0	3.7	1.1	3.5	1.7	2.4	0.9	2.0	0.8	1.6	0.6	20.8	4.3
6-Post	6.1	3.0	3.5	0.8	3.1	1.9	2.3	0.8	1.7	0.5	1.9	0.6	21.6	5.4
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Post	5.5	1.9	6.3	2.3	12.7	3.7	3.6	1.2	3.1	1.1	4.3	1.2	41.9	7.9
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	6.6	3.5	4.2	1.4	5.6	2.3	2.2	0.7	2.8	1.2	2.4	0.8	26.9	5.7
8-Post	7.6	3.1	7.3	2.3	11.1	3.3	4.1	1.4	4.7	1.7	4.4	1.2	44.1	6.2
9-Pre	6.7	2.5	6.5	2.1	8.3	3.4	2.6	0.9	3.0	1.4	2.3	0.9	32.6	6.1
9-Post	9.5	3.7	6.1	2.0	4.8	2.9	2.4	0.9	2.3	1.0	2.0	0.8	29.8	6.2
AA Female Alcoholics (Pre-Assessment QEEG Recordings Only)														
10-Pre	7.4	2.7	4.3	1.5	2.9	1.2	1.8	0.6	1.7	0.7	1.7	0.7	22.5	3.9
11-Pre	5.9	2.4	6.7	3.0	8.1	3.9	3.8	1.8	3.3	1.1	2.4	0.7	33.9	8.5

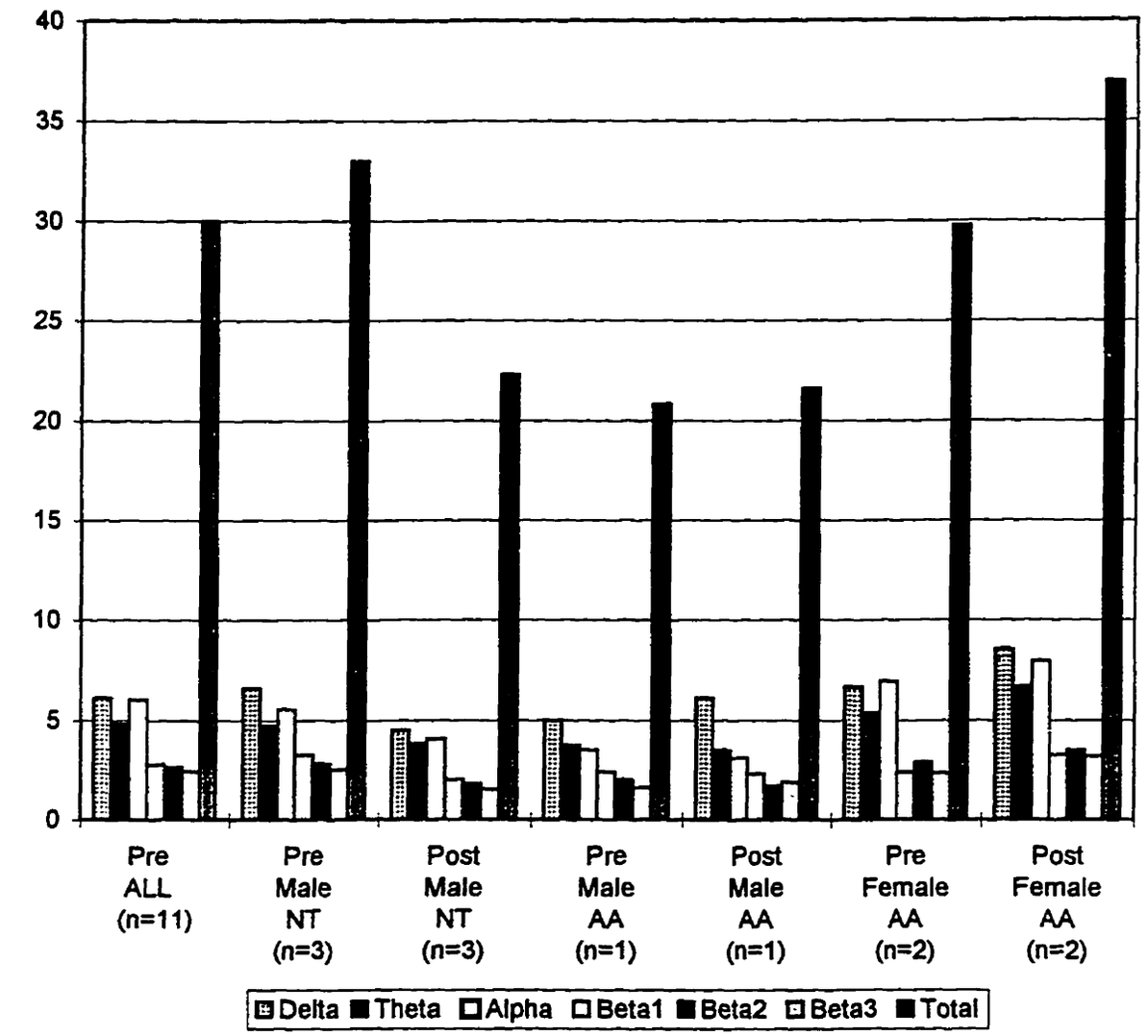
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site C3: Eyes Closed Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site C4: Eyes Closed Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site P3: Eyes Closed Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	8.4	2.9	4.6	1.2	6.7	2.7	3.1	0.9	0.9	2.2	0.8	2.2	30.2	5.9
1-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2-Pre	4.9	1.6	3.6	1.1	2.8	1.1	3.7	1.8	2.0	0.7	1.8	0.5	21.7	4.1
2-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
3-Pre	5.5	1.7	4.6	1.5	6.0	2.8	3.3	1.1	3.3	1.1	2.7	0.9	30.0	5.2
3-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings Only)														
4-Pre	4.0	1.6	3.1	1.0	4.7	3.3	1.8	0.8	1.8	0.6	2.0	1.0	24.1	6.9
5-Pre	5.9	2.4	4.3	1.2	4.6	1.7	2.4	0.6	1.9	0.5	1.4	0.4	22.3	3.3
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	3.9	1.5	3.3	1.1	3.6	1.7	2.4	0.9	1.8	0.6	1.6	0.6	19.3	3.9
6-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	00.0	0.0
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	5.9	1.9	7.3	2.9	20.1	5.1	3.7	1.2	3.4	1.0	5.3	1.6	50.7	9.2
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	4.7	1.6	4.4	1.5	7.1	2.8	2.9	1.1	3.1	1.3	2.7	1.0	27.9	6.2
8-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	00.0	0.0
9-Pre	5.5	2.1	4.2	1.5	5.6	2.2	1.7	0.6	1.9	0.7	1.7	0.5	22.9	4.2
9-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	00.0	0.0
AA Female Alcoholics (Pre-Assessment QEEG Recordings Only)														
10-Pre	5.0	1.8	3.9	1.5	2.8	1.1	1.9	0.7	1.8	0.7	1.6	0.6	19.9	3.9
11-Pre	6.1	2.0	6.6	3.0	10.5	4.5	3.5	1.5	3.4	1.3	2.3	0.7	35.7	9.4

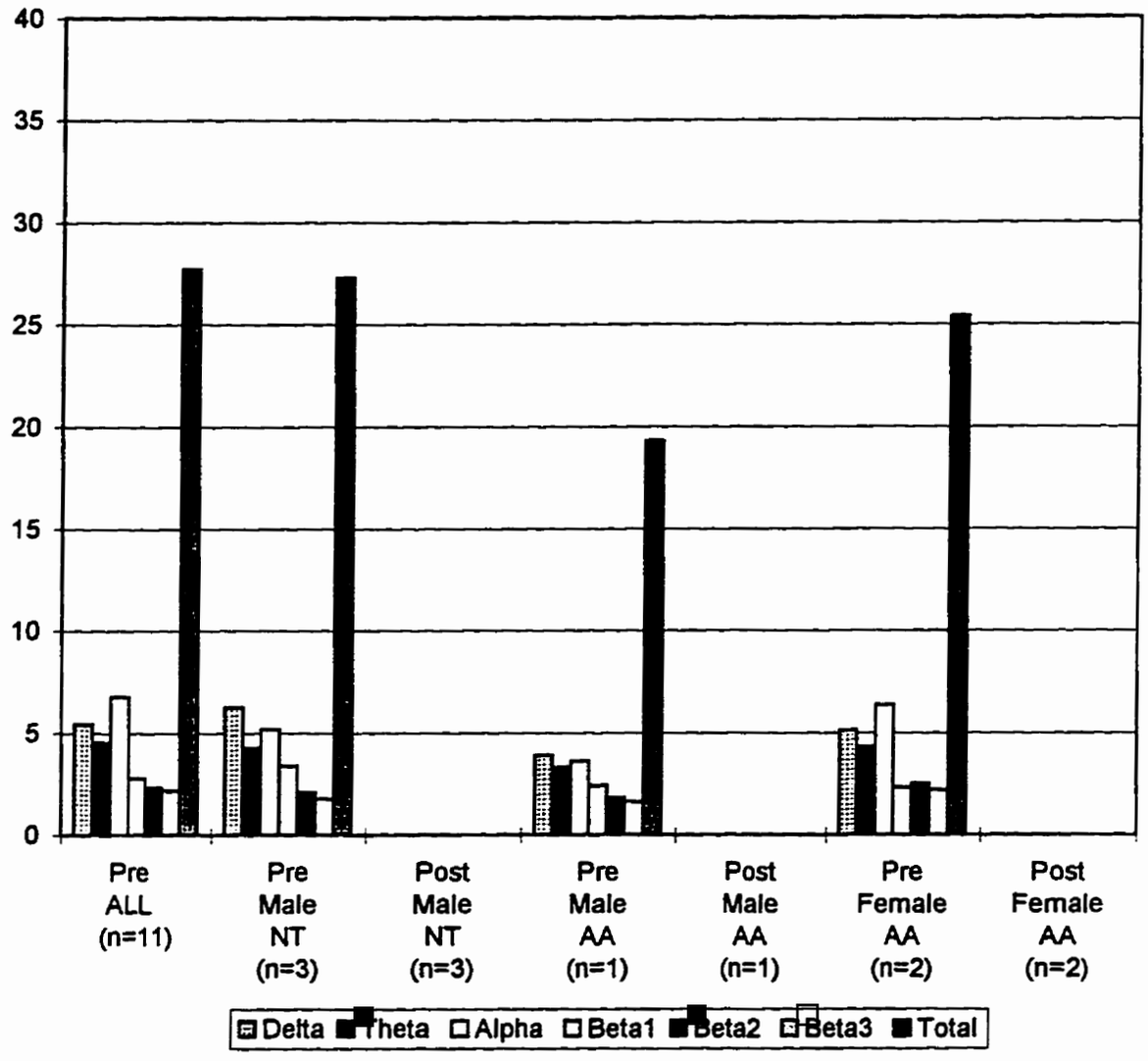
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site P4: Eyes Closed Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	8.0	2.7	4.4	1.3	6.4	2.6	2.9	0.9	2.2	0.8	2.2	0.8	29.2	5.7
1-Post	3.1	1.0	2.5	0.9	6.6	2.7	1.6	0.5	1.7	0.6	1.4	0.5	23.7	4.6
2-Pre	5.4	1.7	4.1	1.3	3.3	1.4	3.9	2.2	2.1	0.7	2.0	0.6	23.9	4.8
2-Post	3.9	1.3	3.6	1.2	2.7	1.3	2.8	1.6	1.4	0.5	1.3	0.5	17.2	3.7
3-Pre	6.1	2.0	5.1	1.8	8.1	4.9	3.7	1.2	3.9	1.3	3.0	0.9	34.4	7.6
3-Post	4.8	1.7	4.0	1.2	3.9	2.3	2.3	0.8	2.1	0.8	1.7	0.6	21.1	4.5
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings Only)														
4-Pre	3.8	1.4	2.9	0.8	3.8	2.4	1.7	0.6	1.8	0.7	1.8	0.9	22.3	5.7
5-Pre	5.6	2.6	3.6	1.1	4.6	1.7	2.1	0.7	1.7	0.5	1.3	0.4	20.7	3.9
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	4.0	1.6	3.2	1.1	3.5	1.8	2.5	1.1	1.9	0.7	1.8	0.6	20.2	4.3
6-Post	5.4	2.7	3.3	0.9	3.3	1.7	2.3	0.7	2.1	0.7	2.3	0.7	22.7	4.6
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	5.4	1.9	6.5	2.6	16.8	4.5	3.5	1.1	3.2	1.0	4.3	1.3	45.2	8.2
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	4.0	1.4	3.7	1.3	5.0	2.1	2.1	0.7	2.5	1.0	2.2	0.8	22.5	4.7
8-Post	7.2	2.5	6.9	1.9	11.5	4.1	4.3	1.2	4.2	1.3	4.7	1.2	43.5	6.0
9-Pre	6.4	2.5	5.5	1.9	10.2	4.5	2.6	0.8	2.9	1.1	2.2	0.8	33.0	7.5
9-Post	9.2	3.7	6.0	2.1	5.9	4.4	2.6	0.9	2.3	1.0	1.9	0.8	30.7	8.5
AA Female Alcoholics (Pre-Assessment QEEG Recordings Only)														
10-Pre	5.0	1.8	3.9	1.5	2.8	1.1	1.9	0.7	1.8	0.7	1.6	0.6	19.9	3.9
11-Pre	6.5	2.4	7.3	3.4	12.8	5.3	3.9	1.4	3.8	1.6	2.4	0.8	40.2	10.7

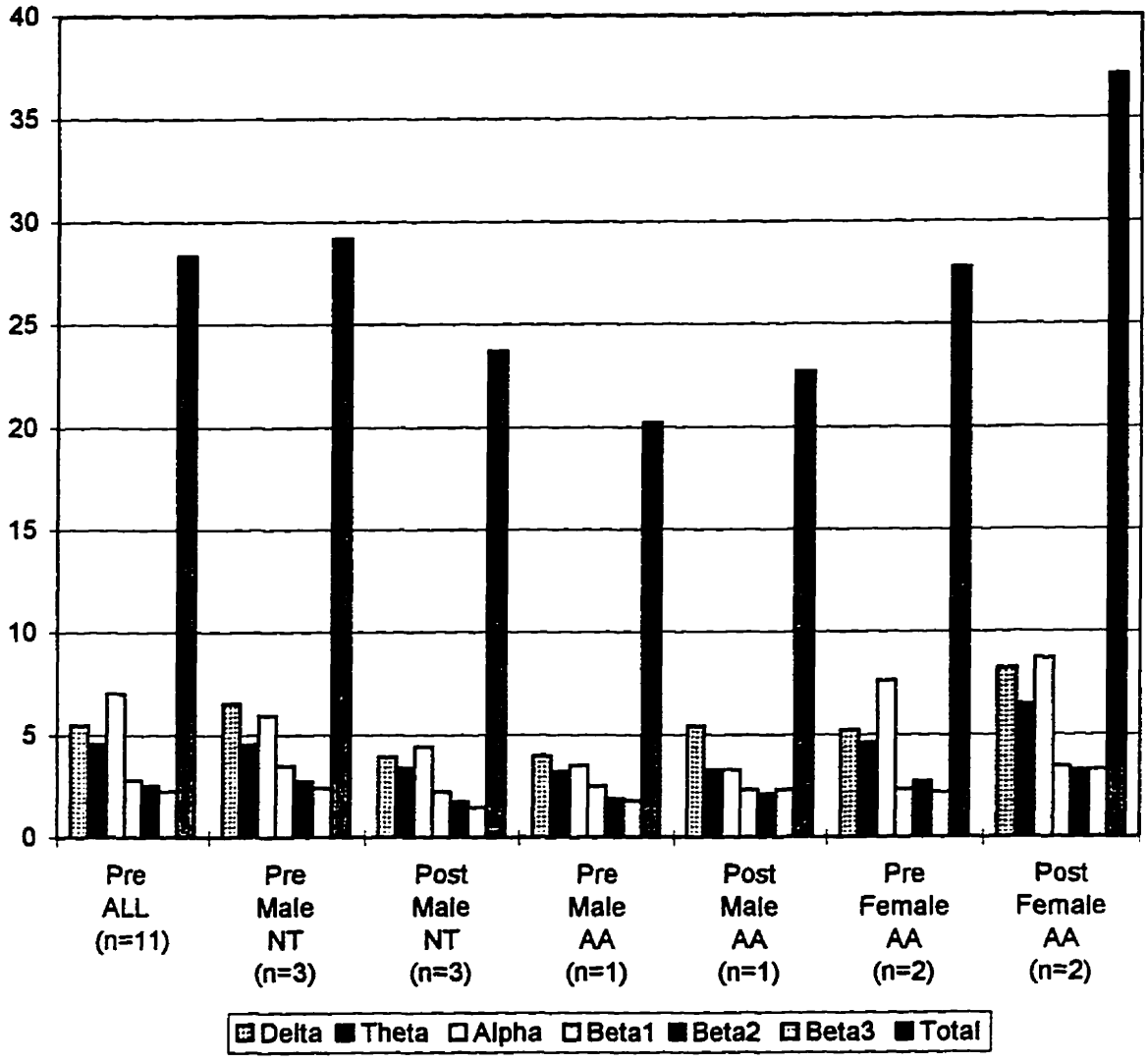
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site P3: Eyes Closed Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site P4: Eyes Closed Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta=0.5-3.9 Hz, Theta=4-7.9Hz, Alpha=8-11.9Hz, Beta1=12-15.9Hz, Beta2=16-19.9 Hz, Beta3=20-23.9Hz, Total=24-32Hz, M=Mean, SD=Standard Deviation. Magnitude values are in microvolts.

Site T5: Eyes Closed Pre and Post-Assessment QEEG Averages, N=11

Case	Delta		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	8.9	3.0	4.6	1.4	5.5	2.5	2.9	1.0	2.0	0.8	1.9	0.6	28.7	5.9
1-Post	2.2	0.8	1.6	0.6	2.5	1.2	0.9	0.3	0.9	0.3	0.7	0.2	12.5	2.5
2-Pre	3.9	1.2	2.6	0.8	2.2	0.9	2.6	1.2	1.5	0.5	1.5	0.4	16.8	2.9
2-Post	2.6	1.0	3.8	1.0	2.7	0.7	2.8	0.9	2.2	0.6	1.8	0.5	18.7	2.7
3-Pre	4.1	1.3	3.8	1.0	5.6	2.3	2.9	1.0	2.6	0.9	2.6	0.9	25.9	4.6
3-Post	4.1	2.1	3.4	1.0	2.6	1.2	1.6	0.6	1.4	0.5	1.1	0.4	16.1	3.4
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings Only)														
4-Pre	3.1	1.1	2.5	0.8	3.6	2.7	1.6	0.6	1.4	0.5	1.6	0.6	19.4	4.0
5-Pre	4.8	2.2	3.0	0.8	2.6	0.9	1.7	0.4	1.3	0.3	1.0	0.3	15.7	2.9
AA Group Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	2.8	1.2	2.3	0.8	2.4	1.2	2.1	0.8	1.5	0.4	1.6	0.5	15.5	2.6
6-Post	4.0	1.2	2.8	0.9	2.8	1.1	2.7	1.0	2.3	0.8	2.4	0.6	21.5	3.9
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	4.1	1.3	4.4	1.6	12.5	3.4	2.9	1.0	2.8	0.8	3.8	1.2	34.7	5.8
AA Female Alcoholics (Pre and Post -Assessment QEEG Recordings)														
8-Pre	2.6	1.2	1.9	0.7	2.6	1.0	1.2	0.4	1.4	0.5	1.3	0.4	12.6	2.7
8-Post	6.4	2.2	5.0	1.3	6.7	2.1	2.8	0.8	2.9	0.8	2.8	0.7	30.0	4.6
9-Pre	4.4	1.7	3.4	1.1	6.1	2.5	1.8	0.7	1.8	0.7	1.5	0.5	21.0	4.6
9-Post	15.1	6.8	5.3	1.8	3.2	1.5	1.8	0.6	1.5	0.5	1.3	0.5	29.9	8.5
AA Female Alcoholics (Pre-Assessment QEEG Recordings Only)														
10-Pre	3.7	1.5	2.9	1.1	1.9	0.7	1.4	0.5	1.3	0.4	1.2	0.4	14.3	2.9
11-Pre	4.6	1.6	5.2	2.3	7.8	3.4	2.7	1.1	2.6	1.1	1.9	0.6	28.3	7.0

Delta=0.5-3.9 Hz, Theta=4-7.9Hz, Alpha=8-11.9Hz, Beta1=12-15.9Hz, Beta2=16-19.9 Hz, Beta3=20-23.9Hz, Total=24-32Hz, M=Mean, SD=Standard Deviation. All values are in micro volts.

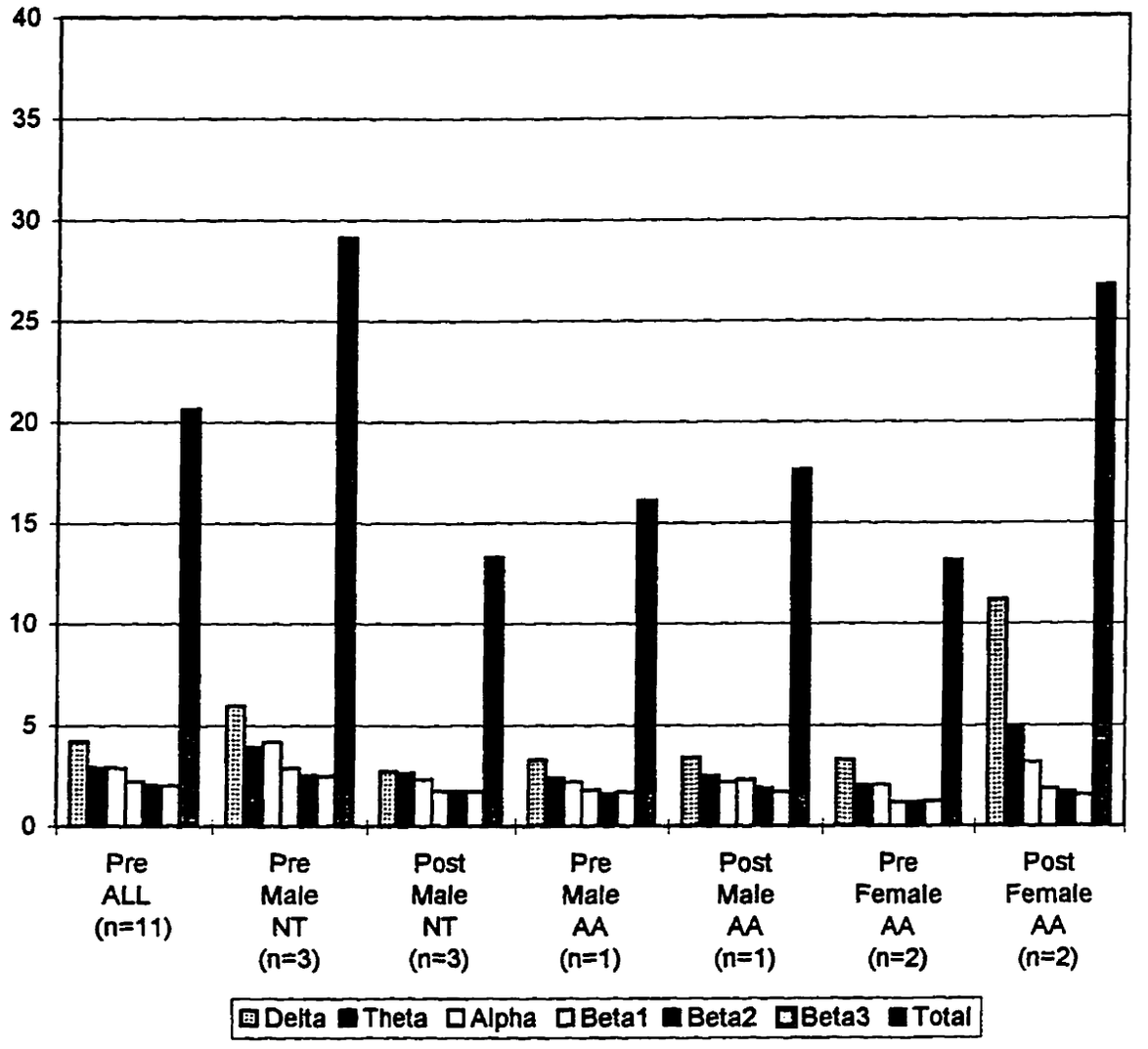
Site T6: Eyes Closed Pre and Post-Assessment QEEG Averages, N=11

Case	Delta		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Post	8.0	2.7	4.1	1.3	5.2	2.1	2.5	0.8	2.0	0.8	2.0	0.6	26.5	4.6
1-Post	2.7	0.8	2.0	0.8	5.0	2.3	1.4	0.5	1.5	0.5	1.4	0.4	20.2	4.2
2-Pre	5.1	1.5	3.6	1.1	3.0	1.3	2.9	1.3	2.1	0.6	1.9	0.5	21.7	3.7
2-Post	2.6	0.9	2.3	0.7	1.5	0.6	1.7	0.9	1.1	0.4	1.0	0.3	11.8	2.4
3 -Pre	5.3	1.8	4.4	1.4	8.4	4.9	3.7	1.3	4.0	1.4	3.0	0.9	33.5	7.9
3-Post	4.8	1.6	3.9	1.2	5.0	3.2	2.7	0.9	2.6	1.0	2.1	0.7	24.0	5.6
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings Only)														
4-Pre	2.9	1.1	2.2	0.6	3.0	1.9	1.5	0.5	1.5	0.5	1.4	0.5	17.9	3.9
5-Pre	6.5	3.6	3.1	0.9	4.4	1.6	1.9	0.6	1.5	0.5	1.2	0.4	20.3	4.8
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	2.6	1.0	2.2	0.7	2.6	1.4	2.2	1.0	1.8	0.6	1.8	0.6	16.8	3.3
6-Post	4.6	2.0	2.6	0.7	3.0	1.2	2.4	0.8	2.6	0.7	2.2	0.7	21.7	3.3
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	3.5	1.3	3.5	1.2	9.9	3.3	3.1	1.0	2.9	0.9	3.5	1.1	32.4	5.4
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	3.2	1.2	2.8	1.0	4.6	2.0	2.0	0.7	2.0	0.7	1.8	0.7	19.0	4.5
8-Post	6.5	2.3	6.2	2.1	9.6	3.3	3.7	1.1	3.8	1.3	4.0	1.4	38.2	6.6
9-Pre	5.2	2.1	4.4	1.4	7.2	3.0	2.0	0.6	2.2	0.8	1.8	0.6	25.4	5.0
9-Post	8.7	3.3	4.7	1.5	3.8	2.3	2.0	0.7	1.7	0.7	1.5	0.5	24.7	5.6
AA Female Alcoholics (Pre-Assessment QEEG Recordings Only)														
10-Pre	3.4	1.2	2.6	0.8	1.9	0.8	1.2	0.5	1.1	0.4	1.1	0.5	13.1	2.5
11-Pre	5.5	2.4	6.3	3.4	9.6	4.3	3.2	1.1	2.8	1.0	1.9	0.7	32.0	9.4

Delta=0.5-3.9 Hz, Theta=4-7.9Hz, Alpha=8-11.9Hz, Beta1=12-15.9Hz, Beta2=16-19.9 Hz, Beta3=20-23.9Hz, Total=24-32Hz, M=Mean, SD=Standard Deviation. All values are in micro volts

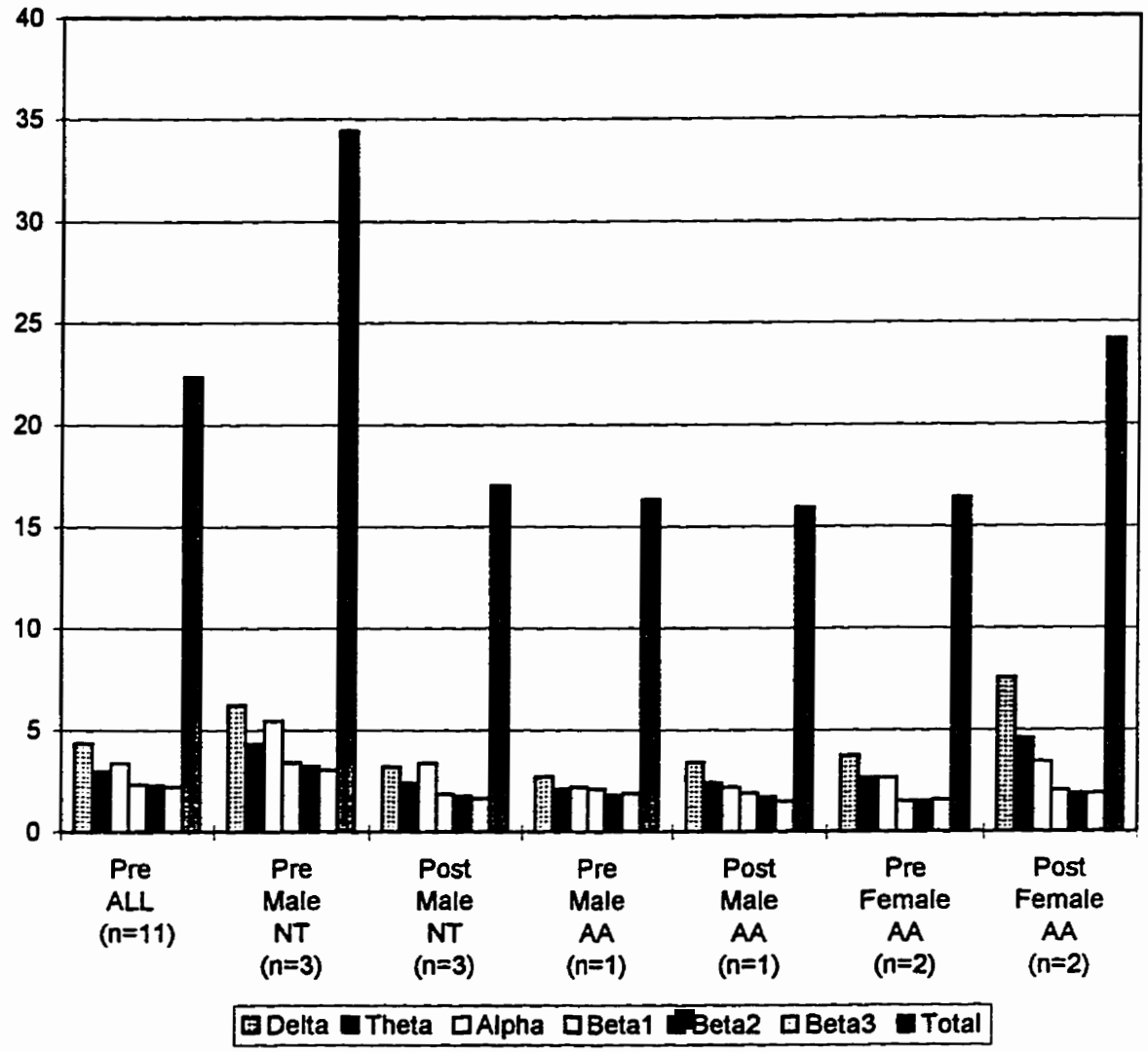
Figure 12

Site T5: Eyes Closed, Pre and Post-Assessment Average QEEG Magnitude



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site T6: Eyes Closed, Pre and Post-Assessment Average QEEG Magnitude



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Table 9

Site O1: Eyes Closed Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	8.9	3.0	4.7	1.4	7.2	3.9	3.1	1.0	2.2	0.9	2.1	0.8	31.3	6.9
1-Post	3.0	1.1	2.1	0.8	5.3	2.2	1.5	0.5	1.4	0.5	1.4	0.4	21.1	4.0
2-Pre	4.5	1.7	3.0	1.1	3.3	1.2	3.2	1.3	2.5	0.8	2.6	0.7	24.0	4.0
2-Post	3.0	1.1	4.3	1.0	3.0	0.9	3.1	1.2	2.1	0.6	1.7	0.6	19.8	3.3
3 - Pre	5.2	1.6	4.4	1.4	7.4	3.6	3.9	1.2	3.8	1.3	3.4	1.0	34.0	6.2
3-Post	4.4	2.2	3.3	1.0	2.5	1.2	1.7	0.6	1.5	0.6	1.2	0.5	16.7	3.4
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings Only)														
4-Pre	3.0	1.0	3.1	1.0	3.4	1.6	1.9	0.8	1.7	0.6	1.6	0.5	21.0	1.2
5-Pre	5.4	2.2	4.3	1.0	3.8	1.2	2.3	0.6	1.7	0.4	1.3	0.4	20.7	3.0
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	2.8	1.0	3.0	1.0	3.3	1.5	2.6	1.1	2.2	1.0	2.3	1.2	20.7	5.9
6-Post	4.5	1.8	3.6	1.4	4.9	1.7	3.6	1.1	4.0	1.6	4.6	1.2	34.4	1.9
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	4.0	1.2	4.6	1.5	12.3	3.8	3.4	1.0	3.2	1.0	4.4	1.3	37.9	6.4
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings Only)														
8-Pre	3.5	1.2	3.2	1.2	4.8	1.9	2.1	0.8	2.3	0.9	2.1	0.8	20.8	4.3
8-Post	5.7	2.0	5.4	1.4	9.3	2.9	3.4	1.0	3.4	1.0	3.3	0.8	34.2	5.0
9-Pre	4.6	1.6	3.4	1.1	4.7	1.8	1.5	0.5	1.6	0.5	1.5	0.5	19.6	3.4
9-Post	9.4	3.8	5.2	1.7	5.3	4.7	2.2	0.8	1.9	0.8	1.7	0.9	28.1	8.8
AA Female Alcoholics (Pre-Assessment QEEG Recordings Only)														
10-Pre	4.6	1.7	3.5	1.4	2.4	0.9	1.8	0.6	1.6	0.6	1.5	0.5	18.0	3.5
11-Pre	4.0	1.4	3.8	1.7	5.7	2.4	2.6	1.0	2.3	0.8	2.1	0.7	24.6	5.1

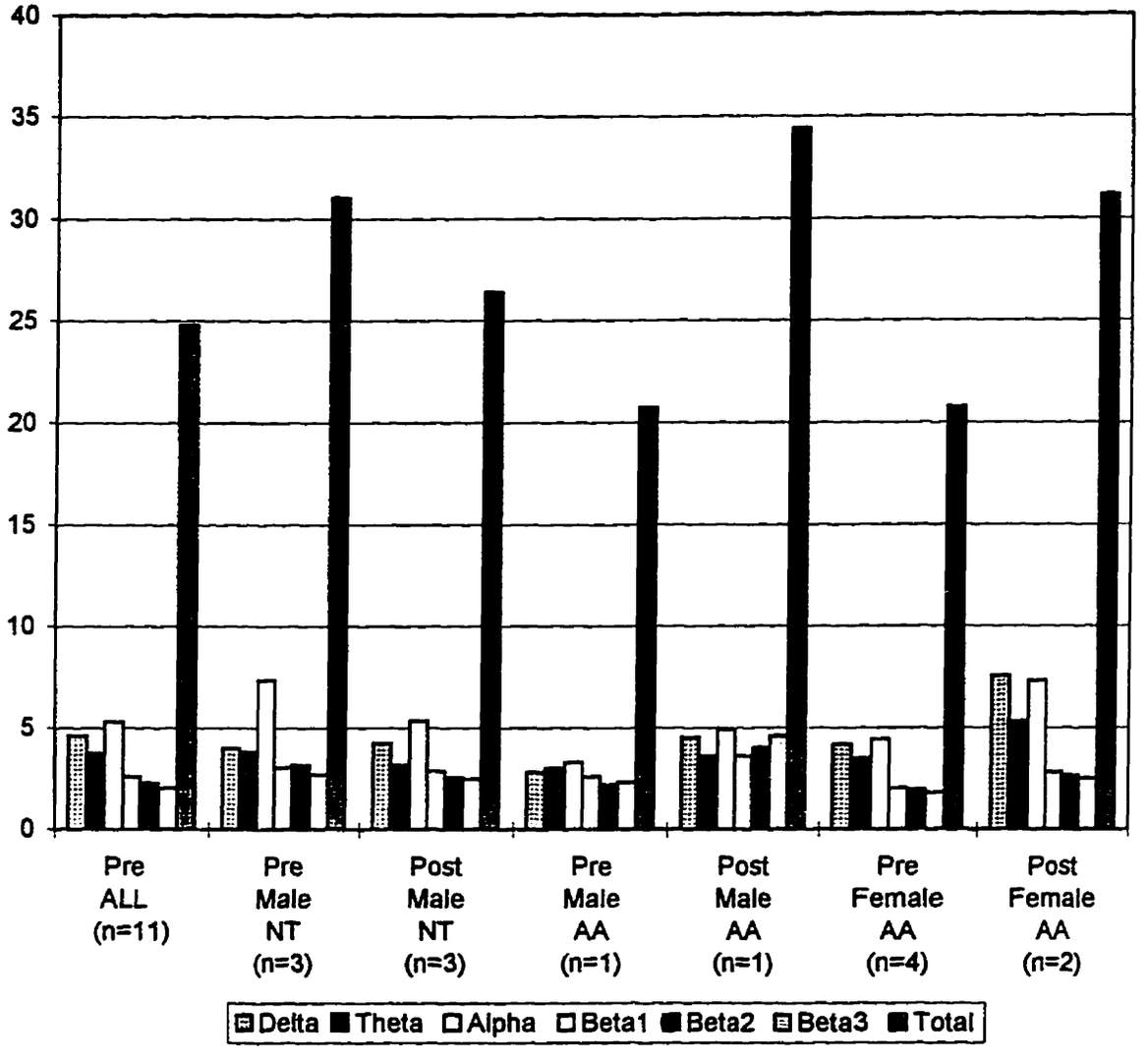
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in microvolts.

Site O2: Eyes Closed Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1 -Pre	8.1	2.8	4.3	1.3	6.9	3.9	3.2	1.0	2.3	0.9	2.7	1.1	31.5	7.1
1 -Post	2.6	0.8	1.8	0.7	5.2	2.1	1.6	0.5	1.7	0.6	1.7	0.5	22.4	3.9
2 -Pre	4.7	1.5	3.0	1.0	2.8	1.1	2.8	1.4	1.8	0.5	1.9	0.5	20.0	3.7
2 -Post	2.9	1.1	3.4	0.9	2.2	0.7	2.4	1.1	1.4	0.4	1.2	0.4	15.2	2.6
3 -Pre	5.7	2.0	4.3	1.7	7.1	4.2	3.8	1.3	3.8	1.3	3.0	0.9	32.7	7.6
3 -Post	4.4	1.6	3.7	1.3	4.0	2.3	2.5	0.7	2.3	0.8	1.9	0.6	21.7	4.5
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings Only)														
4-Pre	2.7	0.9	2.7	0.9	3.1	1.4	1.8	0.7	1.6	0.6	1.4	0.5	19.1	4.3
5-Pre	7.5	4.0	3.8	1.1	4.0	1.3	2.1	0.6	1.6	0.5	1.3	0.4	22.1	4.9
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	2.9	1.1	2.6	0.9	3.1	1.3	2.6	1.0	2.2	0.9	2.6	1.1	21.3	5.0
6-Post	4.5	2.1	3.3	1.1	3.8	1.4	2.7	0.8	3.0	1.1	3.2	0.8	27.1	4.1
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	4.4	1.5	4.2	1.5	10.8	3.2	4.1	1.3	3.7	1.1	4.7	1.5	40.8	5.6
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings Only)														
8-Pre	3.5	1.2	3.1	1.1	4.2	1.7	1.9	0.7	2.2	0.8	2.0	0.7	19.6	4.1
8-Post	6.0	2.2	5.7	1.8	10.2	3.6	3.7	1.1	3.6	1.1	3.5	0.9	39.5	5.8
9-Pre	5.5	2.0	4.5	1.6	9.4	4.5	2.6	0.8	2.7	0.9	2.5	0.8	31.2	6.7
9-Post	9.0	3.6	6.0	2.1	6.2	5.7	2.5	1.0	2.3	1.0	2.1	1.0	31.5	10.1
AA Female Alcoholics (Pre-Assessment QEEG Recordings Only)														
10-Pre	5.3	1.9	3.2	1.1	2.2	0.9	1.5	0.6	1.4	0.6	1.3	0.6	17.1	3.6
11-Pre	4.4	1.7	4.3	2.1	6.3	2.6	2.6	0.9	2.3	0.8	1.8	0.6	25.1	5.7

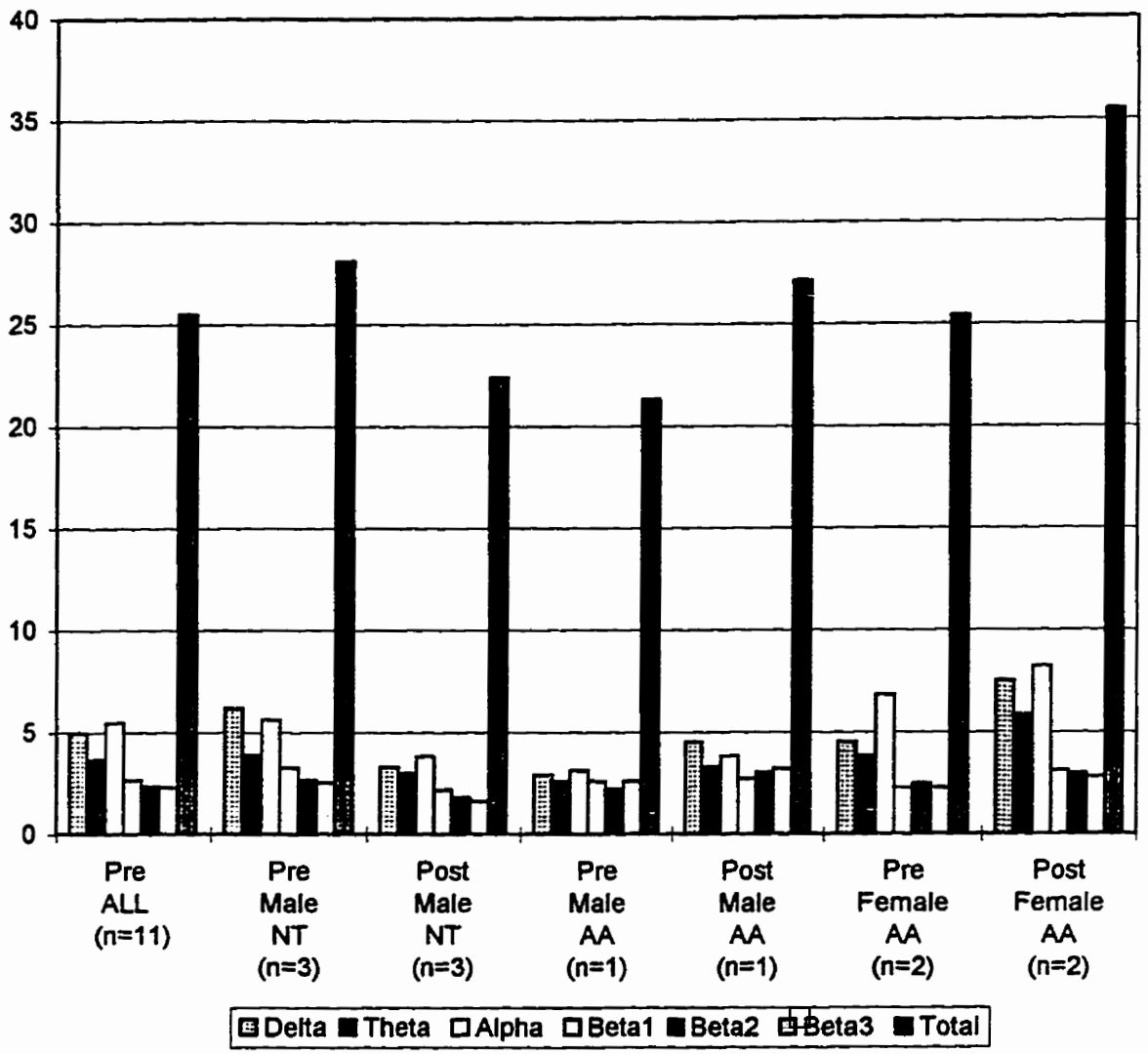
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in microvolts.

Site Q1: Eyes Closed Pre and Post-Assessment Averaged QEEG Magnitudes



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site O2: Eyes Closed, Pre and Post-Assessment Averaged QEEG Magnitudes



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site F3: Eyes Open Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.8	4.7	5.6	1.7	3.9	2.0	3.2	2.4	2.6	1.2	2.3	0.6	30.8	8.6
1-Post	6.2	7.5	4.1	3.5	3.0	2.3	1.3	0.4	1.3	0.5	2.3	0.5	23.6	11.8
2-Pre	5.5	1.9	4.6	1.3	2.9	0.9	2.6	0.8	2.2	0.7	2.2	0.7	23.4	3.0
2-Post	4.2	1.7	4.3	1.5	2.5	0.8	2.0	0.9	1.6	0.7	1.6	0.5	18.8	3.5
3-Pre	5.9	2.1	4.9	1.7	7.0	3.2	2.8	1.0	3.2	1.2	2.9	0.9	40.2	8.6
3-Post	4.7	1.4	4.9	1.9	3.9	1.5	1.9	0.7	2.1	0.8	2.1	0.7	22.8	0.5
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	5.3	1.7	4.0	1.5	3.2	1.2	4.2	2.5	6.6	4.3	7.6	5.2	45.1	20.3
5-Pre	6.9	3.5	3.3	0.9	2.9	1.0	1.9	0.6	1.3	0.4	1.2	0.4	19.5	4.3
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	6.4	4.6	4.3	3.5	2.8	2.3	2.3	0.8	1.6	0.5	1.4	0.5	21.3	6.8
6-Post	6.0	2.5	3.9	1.3	2.8	1.1	2.0	0.9	1.6	0.6	1.5	0.5	20.4	4.1
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	6.3	2.2	4.6	1.6	5.8	2.3	3.0	1.1	2.2	0.7	2.8	0.9	29.0	5.2
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	5.0	1.7	5.0	1.8	3.3	1.1	2.2	0.7	2.5	0.9	2.6	0.7	25.2	3.6
8-Post	6.1	2.0	7.4	2.3	5.0	1.8	2.4	0.8	2.8	1.0	2.6	0.8	30.1	4.7
9-Pre	7.0	3.2	4.7	1.4	4.1	1.3	2.0	0.7	1.9	0.7	1.8	0.5	24.4	4.4
9-Post	11.9	6.9	5.9	2.0	3.9	.3	2.0	0.7	1.9	0.6	1.6	0.5	29.5	8.8
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	5.6	3.0	4.4	1.6	3.1	1.2	2.0	0.6	1.8	0.6	1.9	0.5	22.1	4.7
11-Pre	5.0	1.7	4.0	1.2	3.2	1.3	2.6	1.2	2.6	1.0	2.3	0.8	23.6	4.0

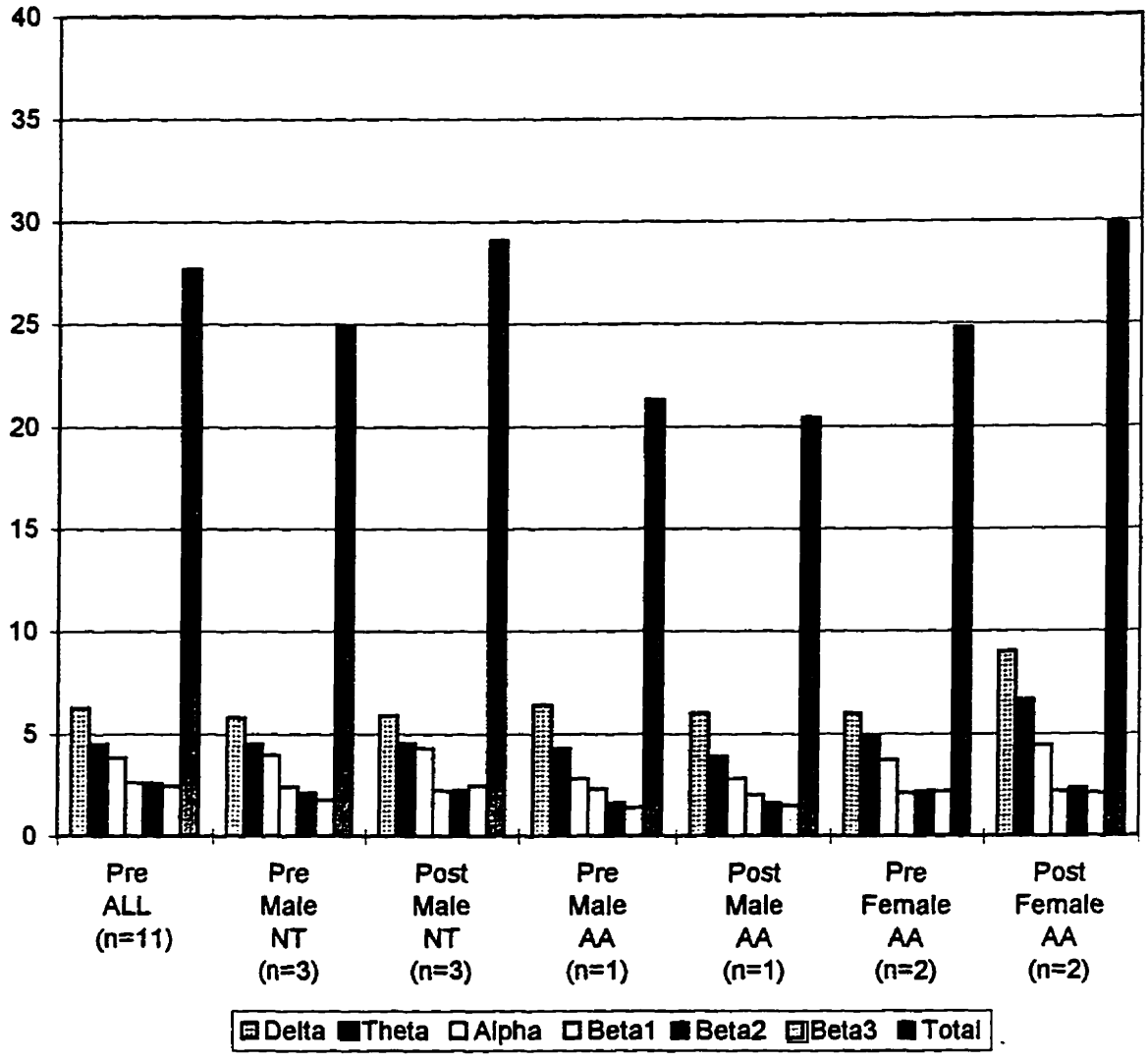
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site F4: Eyes Open Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.3	4.4	5.6	1.7	4.0	1.8	3.3	2.3	2.7	1.3	2.3	0.8	30.7	8.3
1-Post	5.7	5.3	3.9	2.5	3.3	2.5	1.2	0.4	1.4	0.4	1.4	0.4	23.5	9.3
2-Pre	5.6	1.9	4.8	1.3	3.3	0.8	3.1	0.8	2.9	0.8	2.8	0.7	27.8	3.3
2-Post	4.3	1.6	4.3	1.4	2.5	0.8	1.9	0.8	1.6	0.6	1.6	0.5	18.9	0.4
3-Pre	7.2	2.7	5.5	2.0	7.4	3.5	3.0	1.0	3.4	1.2	2.9	1.1	43.2	7.4
3-Post	5.5	1.4	4.8	1.9	3.9	1.5	2.2	0.7	2.3	0.8	2.1	0.7	24.1	4.0
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	4.8	1.7	4.1	1.5	2.8	1.0	3.7	2.0	4.9	2.7	5.5	3.4	37.8	15.1
5-Pre	7.4	2.4	3.6	1.0	3.4	1.4	2.0	0.7	1.4	0.4	1.2	0.4	21.0	3.3
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	6.6	4.3	4.5	1.7	3.0	1.5	2.4	0.8	1.7	0.6	1.5	0.5	22.1	6.4
6-Post	5.8	2.8	3.8	1.3	2.8	1.0	2.1	0.9	1.7	0.5	1.5	0.6	20.2	4.2
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	3.8	1.4	4.2	1.2	3.5	1.0	1.8	0.5	1.7	0.5	2.2	0.7	20.7	2.9
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	4.9	1.6	4.5	1.6	3.0	1.0	1.8	0.5	2.0	0.7	1.9	0.5	22.0	3.4
8-Post	6.1	2.1	7.8	2.5	5.1	1.8	2.4	0.8	2.9	1.0	2.5	0.7	30.7	4.7
9-Pre	7.1	3.4	5.1	1.6	4.3	1.4	2.1	0.7	2.2	0.7	2.0	0.6	25.7	5.0
9-Post	14.6	9.6	6.0	2.3	3.5	1.5	2.0	0.6	1.8	0.5	1.6	0.5	31.9	11.9
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	6.5	2.7	5.0	1.8	3.4	1.2	2.0	0.7	1.7	0.6	1.7	0.5	23.4	4.3
11-Pre	5.1	1.4	3.9	1.2	3.5	1.4	2.7	1.1	2.6	0.8	2.3	0.6	23.9	3.7

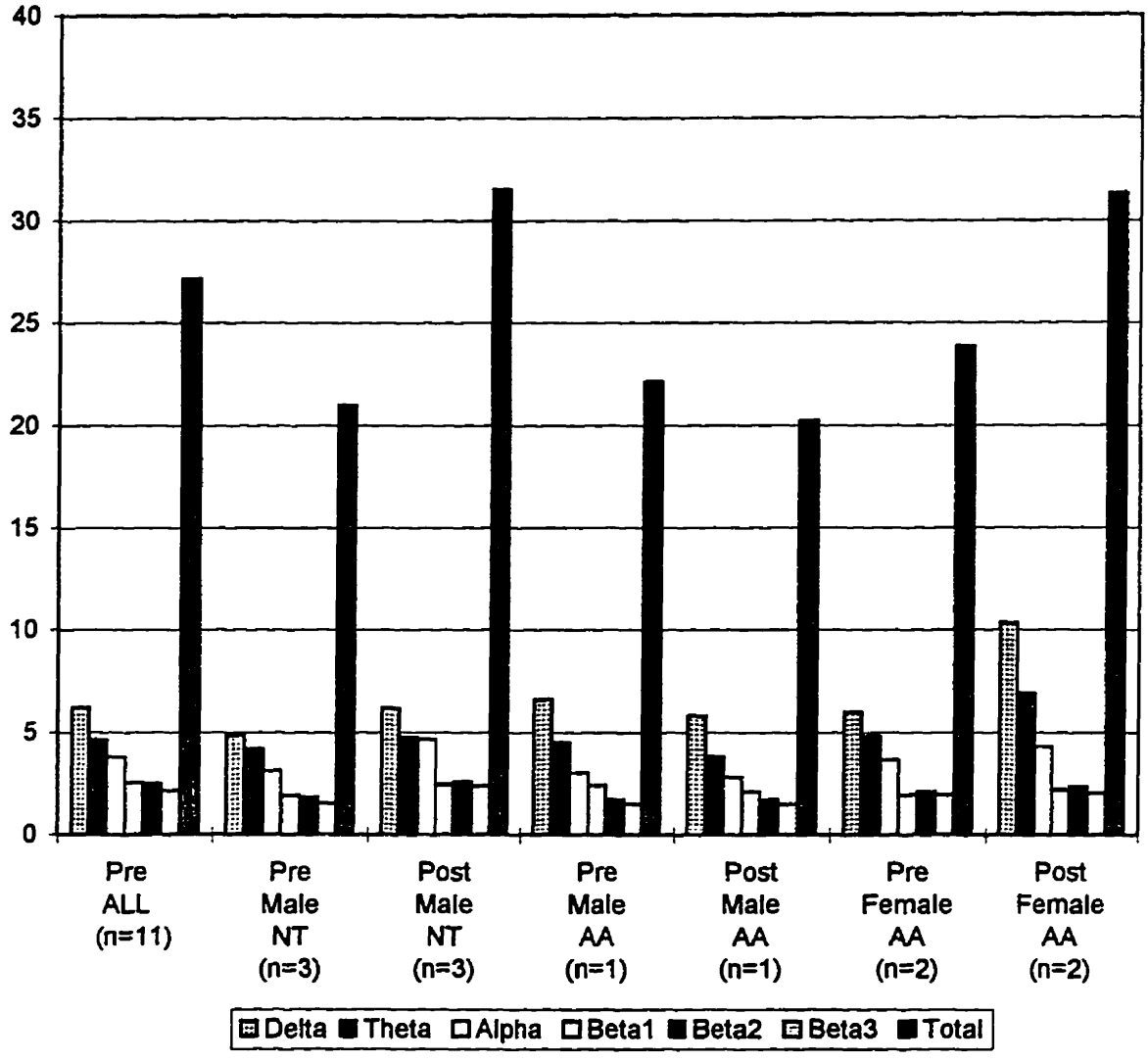
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site F3: Eyes Open Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta=0.5-3.9 Hz, Theta=4-7.9Hz, Alpha=8-11.9Hz, Beta1=12-15.9Hz, Beta2=16-19.9 Hz, Beta3=20-23.9Hz, Total=24-32Hz, M=Mean, SD=Standard Deviation. Magnitude values are in microvolts.

Site F4: Eyes Open Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site C3: Eyes Open Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.3	4.4	5.7	1.7	4.2	2.1	3.2	2.4	2.5	1.2	2.1	0.7	30.6	8.6
1-Post	3.9	3.0	2.7	1.7	2.5	1.8	1.3	0.4	1.3	0.4	1.1	0.4	18.6	6.2
2-Pre	5.2	1.8	3.9	1.2	2.6	0.9	2.6	0.9	2.1	0.6	2.0	0.6	21.9	3.0
2-Post	4.0	1.4	4.1	1.3	2.3	0.7	1.9	0.6	1.7	0.5	1.5	0.4	18.0	2.7
3-Pre	5.2	1.8	3.3	1.1	3.2	1.2	2.4	0.8	2.5	1.3	2.5	1.3	31.2	9.0
3-Post	4.7	1.3	3.7	1.1	3.5	1.5	2.1	0.7	2.2	0.8	2.0	0.7	21.6	4.2
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	4.8	1.7	3.5	1.2	2.9	1.1	4.1	2.4	5.5	3.5	5.6	3.7	37.5	16.0
5-Pre	9.2	6.1	3.7	1.4	3.0	1.0	2.0	0.6	1.3	0.4	1.2	0.3	22.4	7.5
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	5.1	2.2	3.7	1.3	2.8	1.3	2.2	0.8	1.7	0.7	1.4	0.5	19.1	3.7
6-Post	5.2	2.2	3.6	1.3	2.7	1.1	1.9	0.9	1.6	0.6	1.3	0.5	18.8	3.4
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	7.0	2.4	4.8	1.9	8.6	3.8	3.7	1.5	2.5	0.8	3.2	1.0	34.5	6.9
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	4.6	1.6	4.0	1.3	2.8	1.0	1.9	0.6	2.1	0.8	2.0	0.6	20.6	3.3
8-Post	5.9	2.0	6.5	2.1	4.6	1.9	2.6	0.7	3.0	1.3	2.9	0.9	29.2	4.5
9-Pre	6.2	2.2	4.1	1.2	5.2	2.1	2.2	0.7	2.1	0.7	1.8	0.7	24.2	4.1
9-Post	12.4	6.3	5.0	1.9	4.2	1.9	2.0	0.7	1.8	0.7	1.4	0.5	29.0	8.3
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	5.4	2.7	4.0	1.4	2.8	1.1	1.8	0.6	1.6	0.6	1.7	0.4	20.2	4.1
11-Pre	5.5	1.8	4.2	1.2	3.4	1.5	2.6	1.1	2.6	1.1	2.2	0.8	23.9	4.2

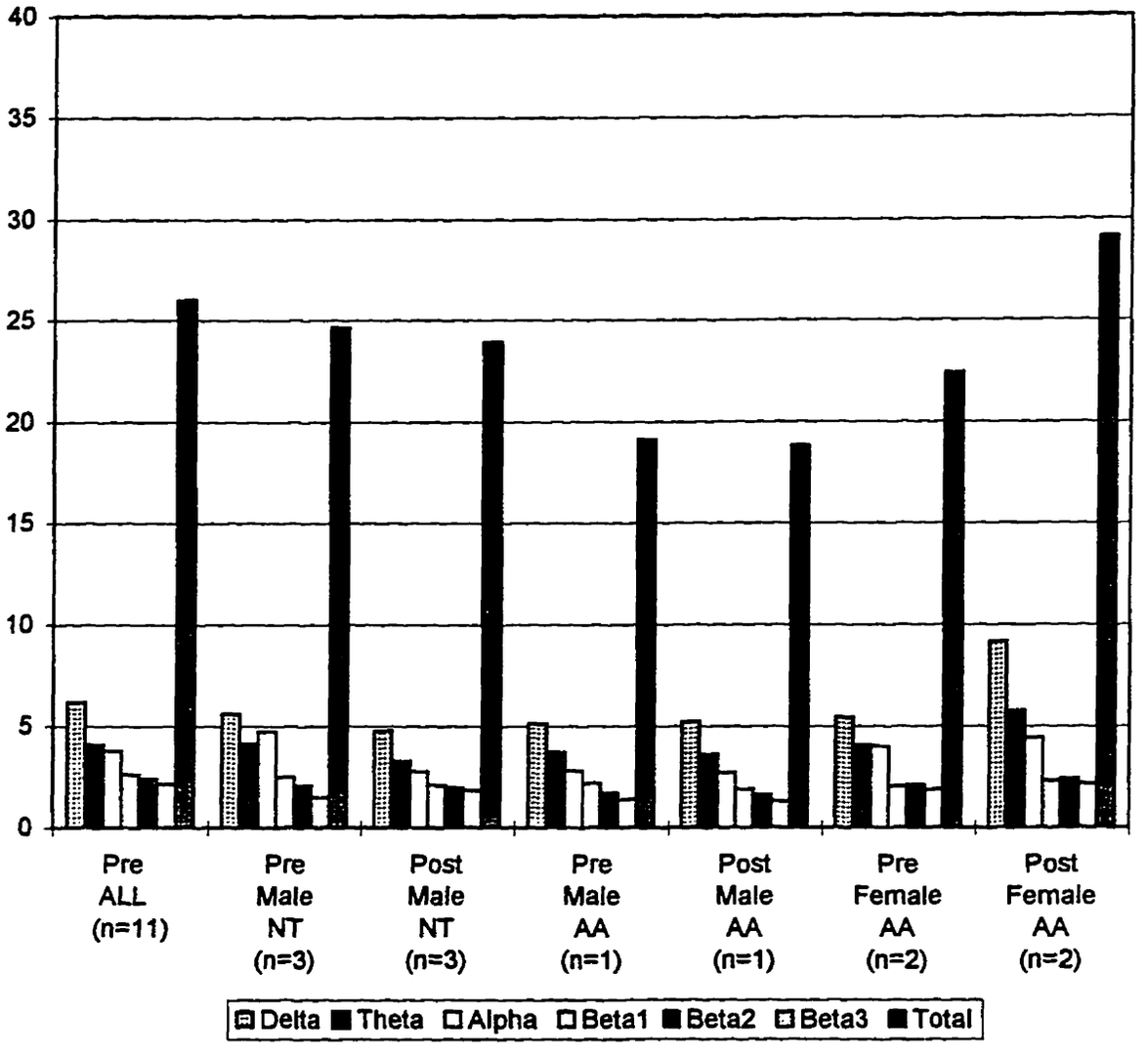
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site C4: Eyes Open Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.0	4.4	5.5	1.5	3.9	1.8	3.2	2.3	2.6	1.1	2.2	0.8	30.0	8.4
1-Post	3.8	1.9	2.7	1.3	3.4	2.5	1.2	0.4	1.2	0.5	1.3	0.4	19.5	5.3
2-Pre	5.5	1.9	4.2	1.1	3.0	0.8	2.9	0.9	2.6	0.8	2.5	0.7	24.5	3.1
2-Post	4.2	1.5	3.9	1.2	2.1	0.6	1.7	0.7	1.6	0.6	1.4	0.4	17.2	3.0
3-Pre	5.2	1.8	3.6	1.2	3.9	1.5	2.7	0.8	2.7	0.9	2.6	0.9	32.8	5.8
3-Post	5.0	1.5	3.8	1.2	4.2	2.0	2.4	0.8	2.4	0.9	2.1	0.8	23.3	4.7
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	4.6	1.7	3.5	1.3	3.3	1.5	5.6	3.4	7.1	4.6	7.7	4.8	48.1	22.9
5-Pre	6.3	2.7	3.3	0.9	3.3	1.3	2.0	0.6	1.3	0.4	1.1	0.4	19.4	3.4
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	5.1	2.0	3.7	1.1	2.8	1.4	2.2	0.8	1.8	0.8	1.5	0.5	19.7	3.4
6-Post	5.3	2.1	3.6	1.2	2.6	0.9	2.1	1.0	1.8	0.7	1.5	0.6	19.3	3.3
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	5.4	1.8	4.3	1.8	7.8	2.9	3.2	1.1	2.5	0.9	3.3	1.1	32.2	5.7
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	5.6	5.0	3.7	1.3	2.5	0.9	1.6	0.5	1.8	0.6	1.8	0.5	20.5	5.7
8-Post	6.9	2.0	7.2	2.1	4.9	1.7	2.8	0.9	3.1	.2	2.9	0.9	31.7	4.6
9-Pre	6.4	2.4	4.7	1.4	4.9	1.7	2.3	0.7	2.3	0.9	2.2	0.9	25.8	4.4
9-Post	8.5	3.1	5.4	1.8	4.6	1.8	2.3	0.8	2.2	0.7	2.0	0.7	27.5	5.3
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	7.6	3.5	4.0	1.4	2.9	1.1	1.6	0.6	1.5	0.5	1.5	0.4	21.6	4.8
11-Pre	5.4	1.7	4.1	1.3	4.0	1.9	3.0	1.2	3.0	1.0	2.5	0.8	25.7	4.4

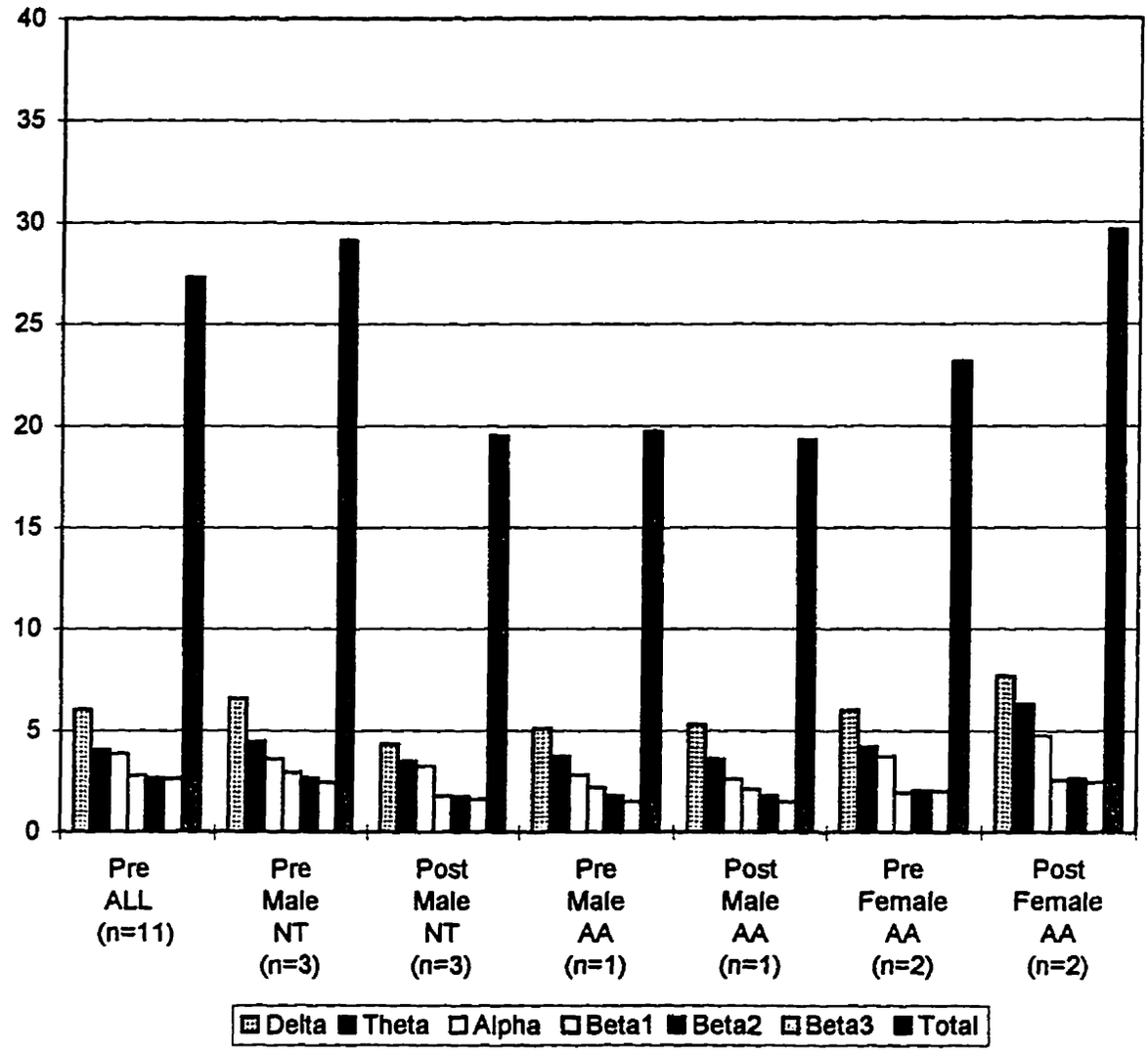
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site C3: Eyes Open, Pre and Post-Assessment Averaged QEEG Magnitudes



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site C4: Eyes Open Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta=0.5-3.9 Hz, Theta=4-7.9Hz, Alpha=8-11.9Hz, Beta1=12-15.9Hz, Beta2=16-19.9 Hz, Beta3=20-23.9Hz, Total=24-32Hz, M=Mean, SD=Standard Deviation. Magnitude values are in microvolts.

Site P3: Eyes Open Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.4	4.1	5.7	1.7	4.4	2.1	3.3	2.4	2.5	1.2	2.1	0.7	30.9	8.2
1-Post	3.2	2.3	2.2	1.1	2.4	1.6	1.2	0.5	1.0	0.5	0.9	0.3	15.9	5.2
2-Pre	4.8	1.9	3.3	1.0	2.6	0.9	2.7	0.9	2.0	0.5	2.1	0.6	21.3	3.3
2-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
3-Pre	5.8	1.9	4.4	1.5	6.6	3.3	3.7	1.6	3.5	1.3	2.7	1.0	41.6	9.4
3-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	4.3	1.5	3.0	1.0	2.6	1.0	2.8	1.3	3.0	1.4	3.2	1.6	25.5	7.1
5-Pre	5.5	1.9	4.0	0.8	3.7	1.1	2.4	0.7	1.5	0.4	1.3	0.4	20.3	2.6
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	4.0	1.4	3.1	0.9	2.7	1.1	2.2	0.8	1.7	0.6	1.4	0.5	17.8	2.8
6-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	3.5	1.3	3.2	0.9	3.3	1.0	2.4	0.9	2.0	0.7	2.4	0.8	20.5	2.9
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	4.4	1.7	3.7	1.1	2.6	1.0	1.9	0.6	2.0	0.7	2.0	0.6	19.8	3.2
8-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
9-Pre	5.9	2.7	3.3	0.9	3.5	.2	1.7	0.5	1.6	0.5	1.4	0.5	19.7	3.6
9-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	4.6	2.3	3.7	1.3	2.9	1.3	1.7	0.5	1.6	0.6	1.6	0.4	18.7	3.9
11-Pre	5.8	2.0	4.1	1.2	3.7	1.5	2.8	0.9	2.5	0.9	1.9	0.7	23.8	4.0

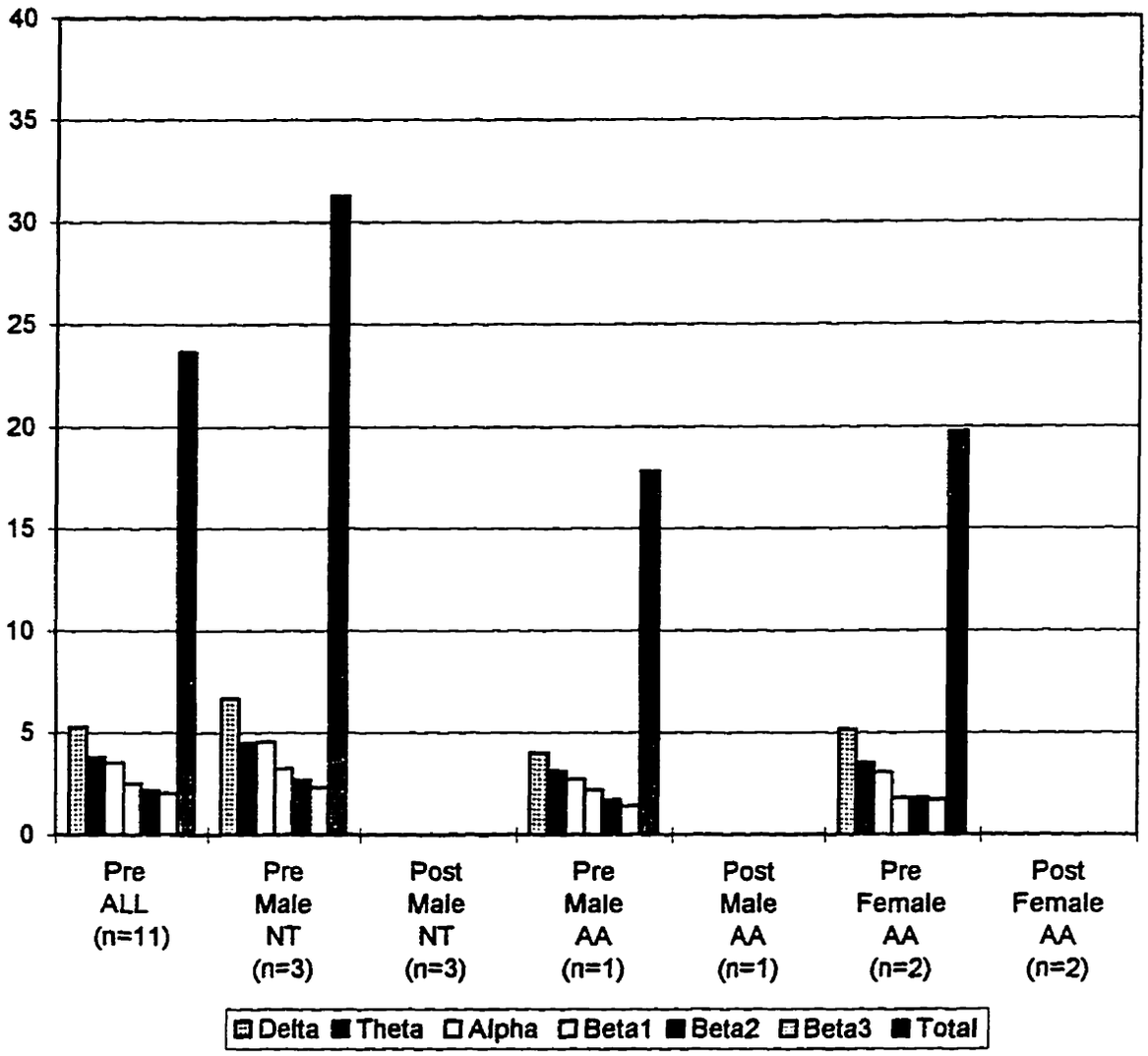
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site P4: Eyes Open Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	8.8	3.9	5.5	1.5	4.1	1.9	3.2	2.3	2.5	1.1	2.2	0.8	29.7	8.2
1-Post	3.2	1.1	2.3	1.0	3.9	3.0	1.4	0.6	1.2	0.5	1.0	0.4	18.6	6.1
2-Pre	5.2	2.0	3.7	1.1	2.8	0.8	2.9	1.0	2.3	0.7	2.3	0.6	23.2	3.3
2-Post	3.9	1.4	3.5	1.1	1.9	0.5	1.8	0.8	1.4	0.5	1.1	0.4	15.5	2.7
3-Pre	6.3	2.1	5.2	1.9	9.8	5.7	4.1	1.6	3.9	1.4	3.2	1.2	48.6	11.8
3-Post	3.9	1.3	3.0	0.8	4.2	2.4	2.3	0.7	2.2	0.8	1.8	0.7	20.5	4.5
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	4.0	1.7	2.9	1.0	2.7	1.1	2.8	1.3	3.0	1.4	3.3	1.7	25.6	7.6
5-Post	5.8	2.4	3.1	0.9	3.4	1.3	2.1	0.6	1.4	0.4	1.1	0.4	19.0	3.1
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	4.2	1.3	3.1	0.9	2.6	1.2	2.4	0.9	1.9	0.7	1.7	0.5	19.1	3.0
6-Post	4.4	1.7	3.1	1.1	2.4	0.9	2.0	0.8	1.8	0.7	1.5	0.5	18.0	2.9
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	3.5	1.2	3.2	0.9	3.5	1.1	2.8	1.0	2.4	0.8	2.7	0.9	22.3	2.9
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	3.9	1.7	3.2	1.1	2.3	0.8	1.5	0.4	1.7	0.6	1.7	0.5	17.3	3.0
8-Post	6.6	2.5	6.1	1.9	4.8	1.9	2.6	0.7	2.8	1.1	3.1	1.0	29.7	4.9
9-Pre	3.8	1.3	2.2	0.7	2.9	1.3	1.4	0.4	1.3	0.4	1.2	0.4	14.8	2.8
9-Post	8.2	2.9	5.2	2.0	5.9	2.9	2.6	0.9	2.4	1.1	1.9	0.6	28.7	6.5
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	4.6	2.3	3.4	1.3	2.6	1.1	1.4	0.5	1.3	0.4	1.3	0.4	16.8	3.8
11-Pre	5.8	2.2	4.1	1.3	4.1	1.8	3.1	1.0	2.6	0.9	2.1	0.6	24.9	4.4

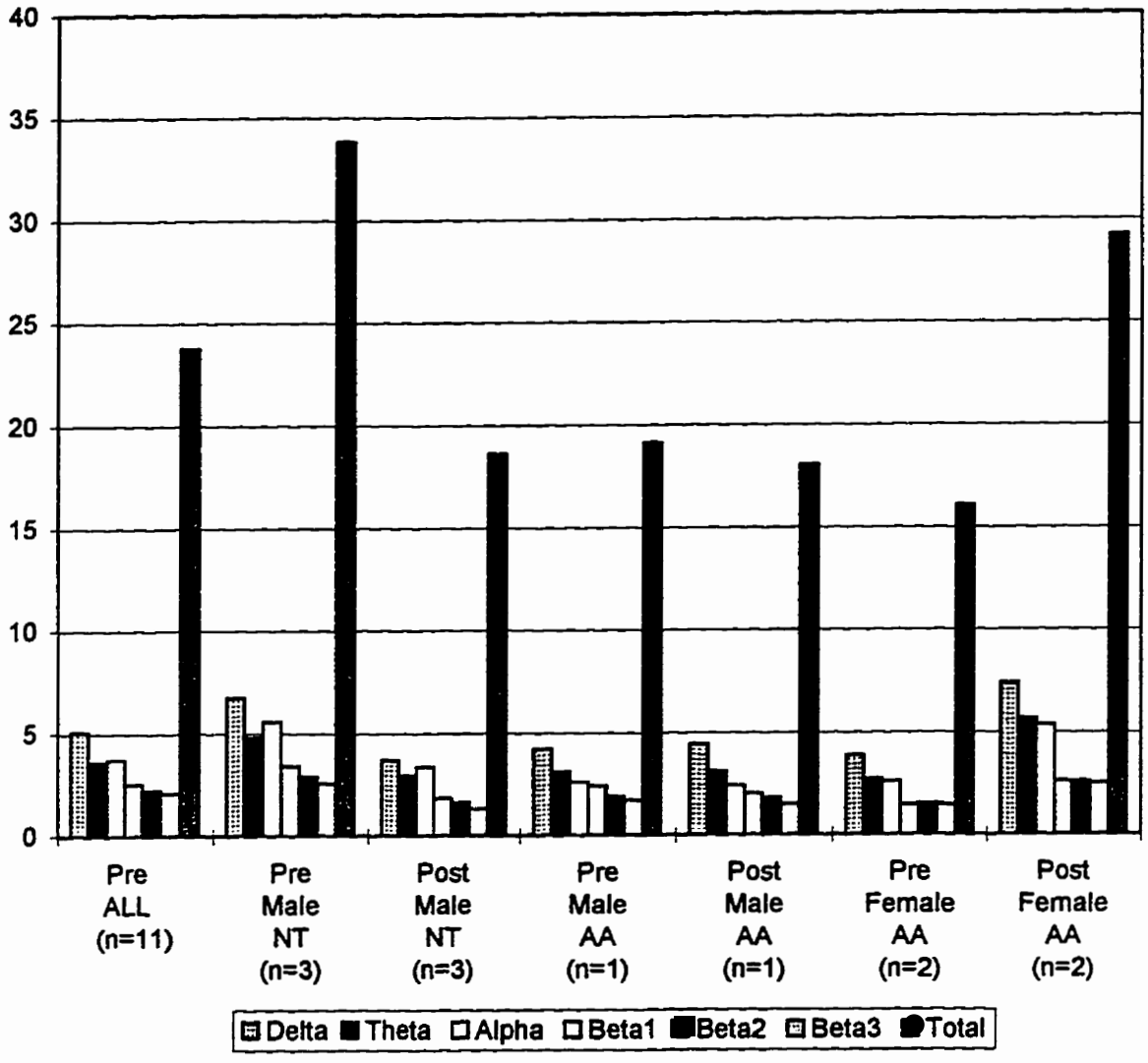
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site P3: Eyes Open, Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site P4: Eyes Open Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site T5: Eyes Open Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.8	4.3	5.8	1.9	4.4	2.0	3.4	2.6	2.7	1.2	2.2	0.7	32.0	8.9
1-Post	2.5	1.7	1.5	0.5	1.8	0.9	1.0	0.3	1.0	0.4	0.9	0.2	13.3	3.5
2-Pre	3.8	1.5	2.4	0.9	2.1	0.7	2.2	0.7	2.2	0.7	2.7	0.8	20.1	3.5
2-Post	2.9	1.0	3.7	1.0	2.7	0.6	2.7	0.7	2.8	0.8	2.9	0.9	22.3	3.0
3-Pre	4.3	1.5	3.6	1.0	6.0	2.6	3.1	1.1	2.7	1.0	2.6	0.9	35.2	7.3
3-Post	2.8	1.2	2.7	0.9	2.4	0.9	1.5	0.5	1.4	0.5	1.4	0.5	14.5	2.8
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	3.5	1.1	2.7	0.8	2.8	1.2	3.4	1.6	3.7	1.8	3.8	1.9	27.9	9.7
5-Pre	4.9	2.0	3.2	0.7	2.6	0.6	1.9	0.5	1.2	0.3	1.0	0.3	16.5	2.3
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	3.3	2.5	2.4	1.3	2.2	1.3	1.8	0.8	1.6	0.7	1.7	0.6	16.1	6.2
6-Post	3.4	6.0	2.5	2.3	2.2	1.3	2.3	1.0	1.9	0.7	1.7	0.8	17.6	11.5
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	2.8	1.1	2.5	0.8	3.2	1.0	2.7	1.0	2.6	1.1	3.0	1.1	21.7	4.6
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	2.8	1.9	1.8	0.8	1.2	0.5	1.0	0.3	1.1	0.4	1.3	0.4	11.5	2.8
8-Post	7.3	3.0	4.5	1.5	3.1	0.9	1.9	0.5	1.9	0.6	1.9	0.6	23.2	4.4
9-Pre	3.8	1.3	2.2	0.7	2.9	1.3	1.4	0.4	1.3	0.4	1.2	0.4	14.8	2.8
9-Post	15.1	6.1	5.4	1.9	3.2	1.2	1.8	0.5	1.5	0.5	1.2	0.3	30.2	7.6
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	3.3	1.5	2.7	0.9	1.9	0.8	1.3	0.4	1.2	0.4	1.1	0.3	13.3	2.6
11-Pre	3.9	1.4	2.8	0.8	2.6	1.1	2.1	0.8	2.0	0.7	1.6	0.6	17.7	3.4

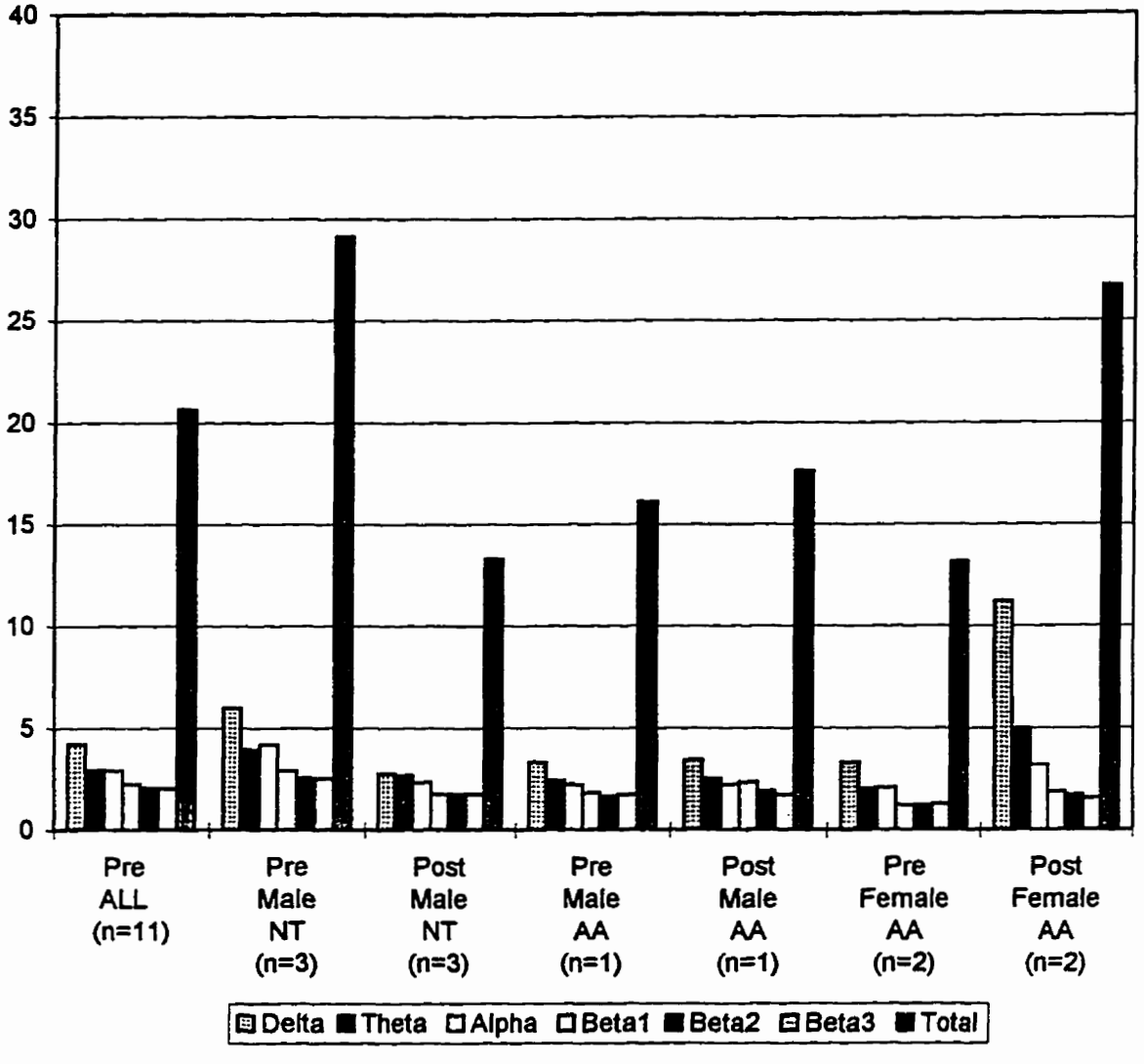
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site T6: Eyes Open Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	8.5	3.7	5.2	1.5	3.8	2.0	3.0	2.2	2.4	1.1	2.2	0.8	28.6	8.2
1-Post	2.8	0.9	1.9	0.7	3.2	2.5	1.3	0.5	1.0	0.3	1.2	0.4	17.0	5.2
2-Pre	4.7	1.7	3.4	1.0	2.9	0.8	3.2	1.0	3.3	0.8	3.7	1.2	27.5	4.5
2-Post	2.7	1.0	2.3	0.7	1.4	0.4	1.5	0.5	1.6	0.5	1.6	0.5	14.0	2.2
3-Pre	5.4	1.8	4.4	1.5	9.6	5.2	4.0	1.4	4.0	1.4	3.2	1.1	47.1	11.0
3-Post	4.1	1.3	3.0	1.0	5.5	3.8	2.8	0.8	2.7	0.9	2.1	0.7	23.7	6.0
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	3.1	1.1	2.3	0.8	2.5	0.9	2.6	1.1	3.1	1.2	3.2	1.6	22.9	6.1
5-Pre	5.9	2.7	2.5	0.7	2.9	1.1	2.0	0.6	1.4	0.4	1.2	0.3	18.0	3.0
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	2.7	0.9	2.1	0.6	2.2	1.0	2.1	0.7	1.8	0.5	1.9	0.5	16.3	2.6
6-Post	3.4	1.1	2.4	0.8	2.2	0.9	1.9	0.7	1.7	0.6	1.5	0.5	15.9	2.4
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	2.6	0.9	2.3	0.8	3.3	1.1	2.7	0.9	3.0	1.1	3.2	1.0	22.2	0.8
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	2.8	0.9	2.2	0.7	1.7	0.5	1.3	0.4	1.4	0.5	1.5	0.4	13.8	1.8
8-Post	5.6	2.1	4.6	1.4	3.3	1.1	2.1	0.6	2.1	0.7	2.4	0.8	23.3	3.5
9-Pre	4.7	1.8	3.1	0.9	3.6	1.5	1.7	0.5	1.6	0.6	1.6	0.5	19.0	3.2
9-Post	9.5	3.7	4.5	1.5	3.6	1.4	2.0	0.7	1.7	0.6	1.4	0.4	25.0	5.2
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	3.4	1.4	2.4	0.9	2.0	0.9	1.1	0.4	1.0	0.3	1.0	0.3	12.5	2.5
11-Pre	3.9	1.4	2.8	1.4	2.6	0.8	2.1	1.1	2.0	0.8	1.6	0.7	17.7	3.4

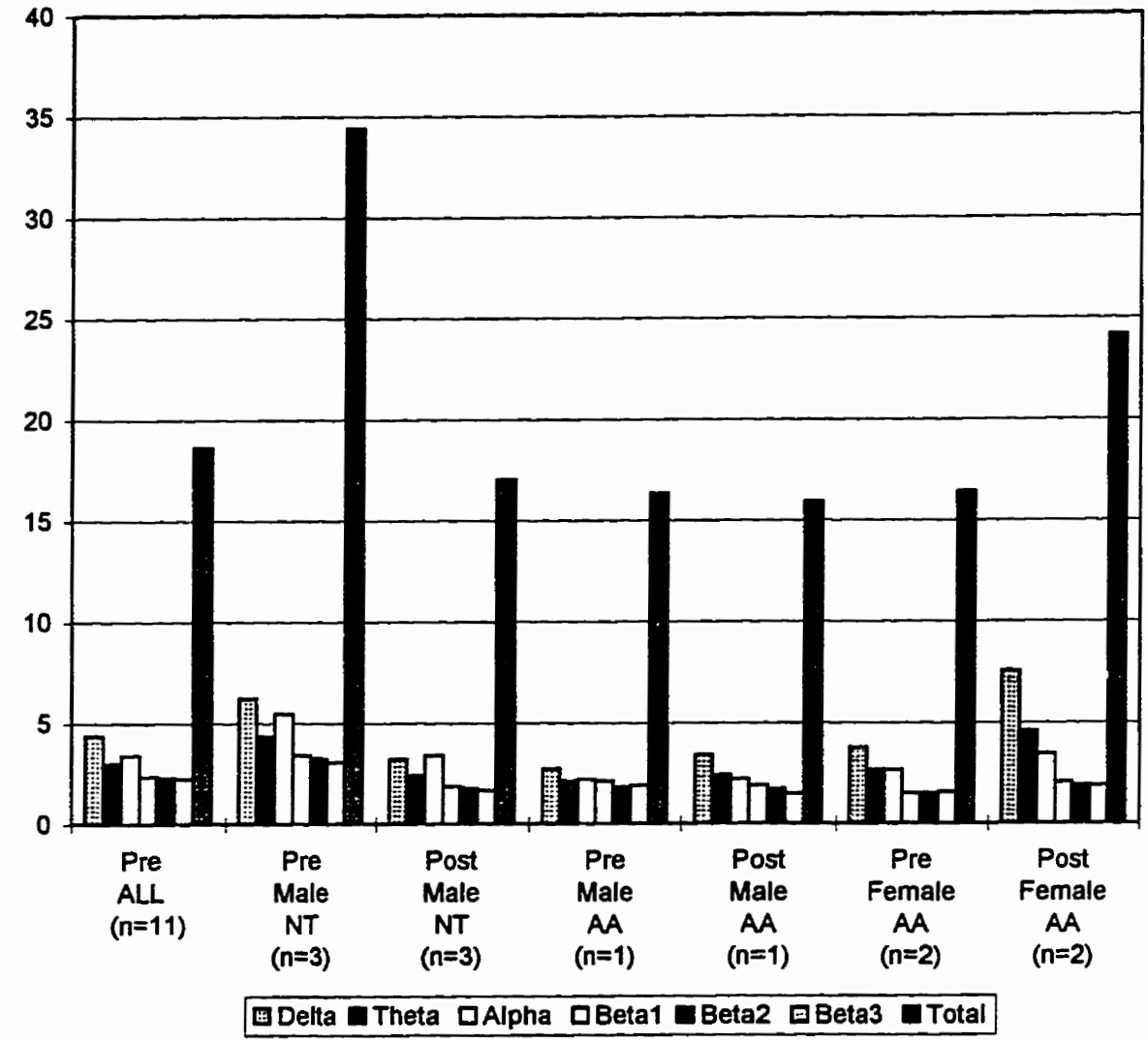
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site T5: Eyes Open Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site T6: Eyes Open Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site O1: Eyes Open Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	10.3	4.2	5.9	1.9	4.4	2.1	3.5	2.4	2.7	1.2	2.4	0.9	33.3	8.9
1-Post	3.0	1.2	2.0	0.8	3.2	3.1	1.6	0.6	1.3	0.7	1.3	0.4	19.1	6.9
2-Pre	4.2	1.7	2.9	1.0	3.3	1.5	3.1	1.1	2.8	1.1	3.0	1.1	25.1	6.5
2-Post	3.2	1.1	4.0	1.0	2.8	0.7	2.6	0.6	2.0	0.5	1.6	0.5	18.6	2.6
3-Pre	5.6	1.8	4.4	1.4	8.5	4.1	4.5	1.7	4.3	1.9	3.7	1.4	50.2	11.8
3-Post	3.3	1.2	2.7	0.7	2.5	1.0	1.7	0.5	1.6	0.5	1.5	0.5	16.0	2.7
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	3.2	1.1	3.3	1.0	3.1	1.5	3.2	1.2	3.1	1.1	3.3	1.2	26.0	5.8
5-Pre	5.0	1.8	4.3	0.8	3.5	0.9	2.5	0.6	1.6	0.4	1.3	0.4	20.2	2.3
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	3.1	1.2	2.7	0.8	2.8	0.9	2.4	0.8	2.4	0.7	2.3	0.7	20.3	3.0
6-Post	3.4	1.3	3.0	1.1	2.6	1.0	2.2	0.8	2.0	0.7	1.9	0.8	18.9	3.4
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	3.2	0.9	3.0	0.8	3.4	0.9	2.7	0.8	2.7	0.8	3.2	1.0	24.3	2.9
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	3.4	1.3	2.9	0.9	2.1	0.7	1.6	0.5	1.7	0.6	1.9	0.6	16.8	2.5
8-Post	5.2	1.8	4.3	1.5	3.2	1.1	2.0	0.6	2.1	0.6	2.2	0.7	22.4	3.2
9-Pre	4.6	2.3	2.6	0.8	2.8	1.0	1.4	0.4	1.3	0.5	1.3	0.5	16.4	3.1
9-Post	9.0	3.3	4.2	1.5	4.2	2.0	2.1	0.7	1.8	0.7	1.5	0.4	24.9	5.2
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	4.0	2.1	3.3	1.1	2.5	1.0	1.6	0.5	1.5	0.5	1.5	0.4	16.8	3.4
11-Pre	4.0	1.4	3.0	1.0	2.3	0.7	2.1	0.6	1.9	0.7	1.8	0.7	18.4	2.9

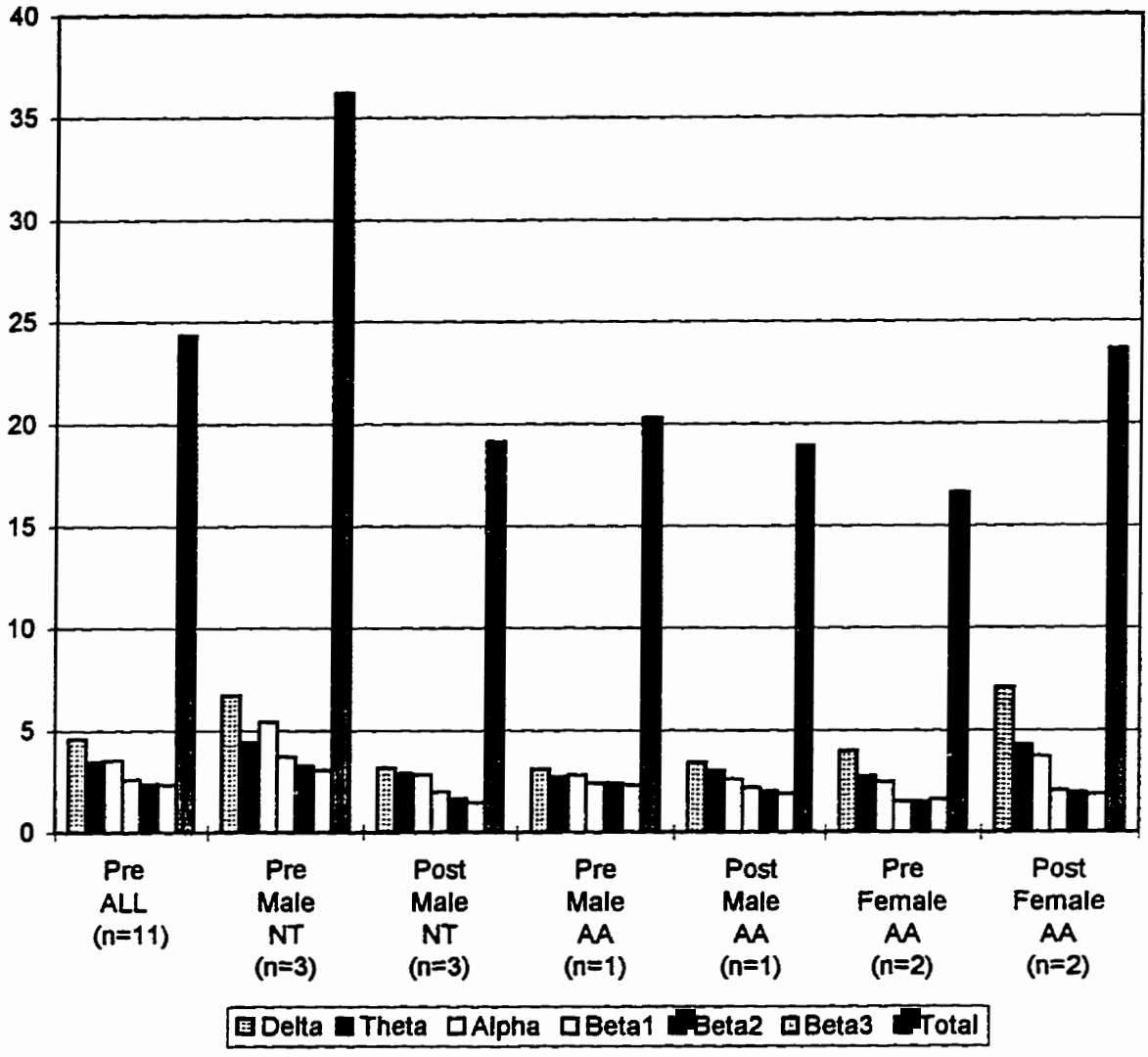
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts

Site O2: Eyes Open Pre and Post-Assessment QEEG Averages, N=11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.2	3.8	5.5	1.5	4.3	2.1	3.4	2.2	2.8	1.1	2.7	0.9	32.5	8.4
1-Post	2.5	0.7	1.8	0.8	3.1	2.8	1.5	0.5	1.5	0.5	1.5	0.5	19.0	6.0
2-Pre	4.5	1.7	3.0	1.0	3.2	1.1	3.2	1.1	2.7	1.0	2.9	1.0	24.9	4.9
2-Post	3.2	1.1	4.0	0.8	2.8	0.5	2.6	0.6	2.0	0.4	1.6	0.3	18.6	2.1
3-Pre	5.6	1.8	4.4	1.4	8.5	4.1	4.5	1.7	4.3	1.9	3.7	1.4	50.2	11.8
3-Post	3.6	1.1	3.0	0.9	4.8	3.0	2.7	0.8	2.5	0.7	2.1	0.7	22.2	5.1
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	3.2	1.2	3.0	1.0	3.2	1.3	3.3	1.4	3.2	1.2	3.6	1.6	26.7	6.0
5-Pre	7.4	3.9	3.5	0.8	3.3	1.1	2.4	0.7	1.6	0.5	1.3	0.4	21.9	4.5
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	3.5	1.3	2.4	0.7	2.6	1.0	2.6	0.9	2.3	0.7	2.6	0.7	21.4	2.8
6-Post	3.9	1.3	2.8	1.1	2.4	0.9	2.1	0.7	1.9	0.7	1.8	0.7	18.3	2.9
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	3.2	1.0	2.8	0.8	3.1	1.0	2.5	0.7	2.6	0.7	2.7	0.9	22.6	2.5
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	3.6	1.9	2.8	0.9	2.0	0.7	1.5	0.4	1.6	0.5	1.8	0.5	16.6	3.0
8-Post	5.4	1.9	4.5	1.5	3.3	1.2	2.1	0.7	2.2	0.6	2.4	0.7	23.6	3.2
9-Pre	4.7	1.7	2.9	0.8	4.1	1.7	2.2	0.6	2.0	0.7	2.2	0.6	21.8	3.4
9-Post	7.9	2.9	4.2	1.4	4.9	2.2	2.5	0.8	2.3	0.9	1.8	0.6	26.6	5.2
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	5.3	2.5	3.2	1.1	2.3	1.0	1.4	0.5	1.2	0.4	1.2	0.4	16.7	3.5
11-Pre	4.3	1.5	3.1	1.1	2.6	0.8	2.3	0.7	2.0	0.7	1.7	0.6	19.3	2.8

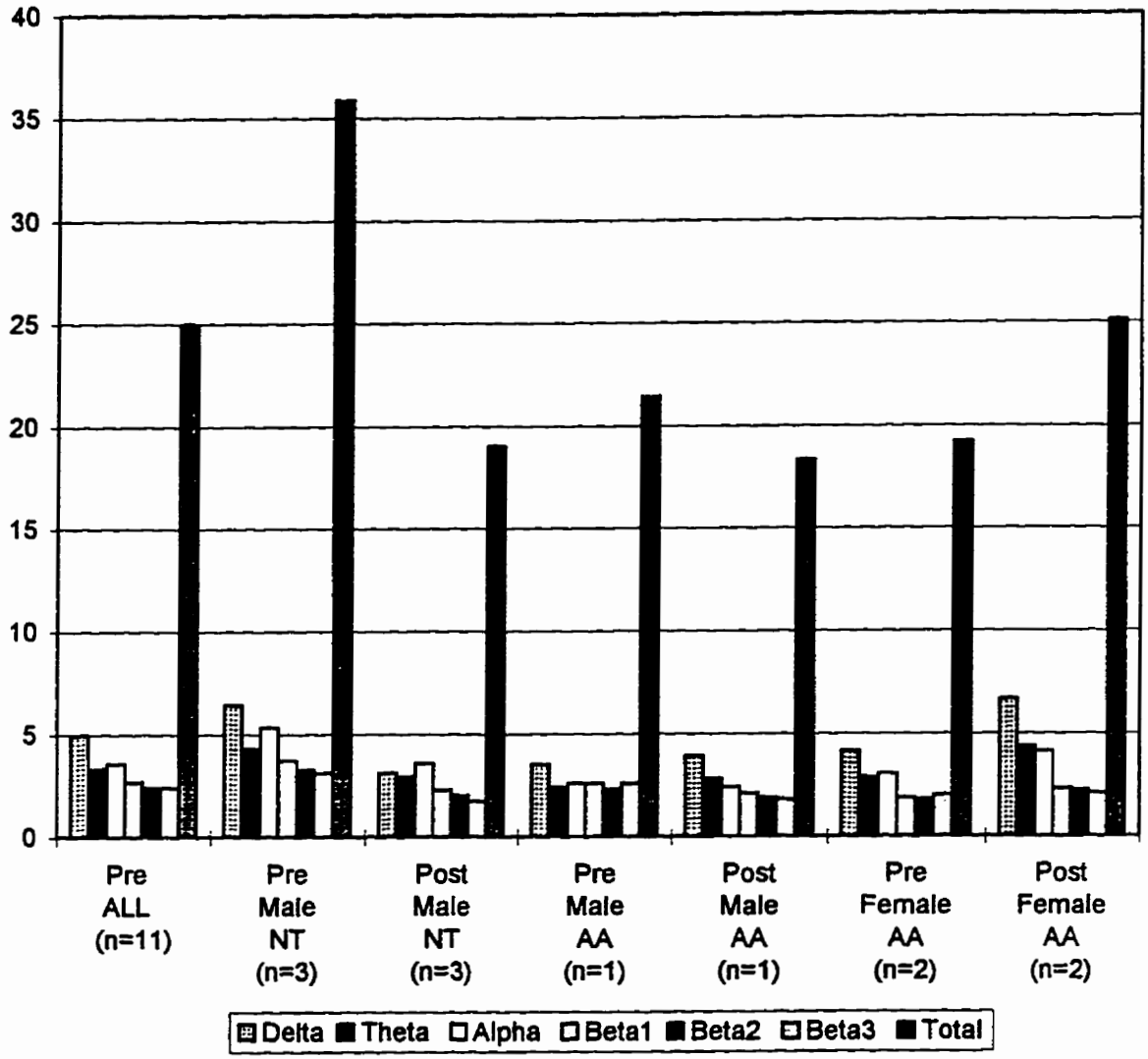
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site O1: Eyes Open Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta=0.5-3.9 Hz, Theta=4-7.9Hz, Alpha=8-11.9Hz, Beta1=12-15.9Hz, Beta2=16-19.9 Hz, Beta3=20-23.9Hz, Total=24-32Hz, M=Mean, SD=Standard Deviation. Magnitude values are in microvolts.

Site O2: Eyes Open Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz,

Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site F3: Reading Silently Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.7	4.3	5.7	2.1	4.6	2.1	3.1	1.5	2.5	1.9	2.3	1.7	31.6	11.4
1-Post	3.0	1.3	2.5	0.8	1.7	0.6	1.3	0.4	1.3	0.3	1.5	0.5	18.1	2.7
2-Pre	5.8	2.0	4.9	1.5	3.0	1.1	2.9	1.3	2.6	0.8	2.3	0.7	25.2	4.1
2-Post	4.1	1.5	4.5	1.4	2.7	0.8	1.8	0.9	1.9	0.8	1.8	0.8	19.9	3.9
3-Pre	6.1	1.9	4.9	1.5	3.1	0.9	2.9	0.9	3.5	0.9	3.5	1.0	40.2	5.0
3-Post	5.5	1.7	5.6	1.7	3.8	1.6	2.7	1.2	2.6	0.8	2.5	0.8	27.0	4.3
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	4.7	1.4	3.6	1.0	2.3	0.6	2.2	0.8	2.4	0.9	2.7	0.9	28.8	5.2
5-Pre	7.6	6.4	4.6	3.7	3.0	1.5	2.3	1.1	1.8	0.8	1.8	1.0	24.3	12.3
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	5.0	1.9	3.9	1.0	3.1	1.4	2.8	1.1	1.9	0.6	1.5	0.4	20.8	4.1
6-Post	5.7	3.2	4.0	1.6	2.9	1.3	2.4	0.7	1.6	0.5	1.5	0.6	21.0	5.1
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	6.5	1.6	5.0	1.9	5.8	2.8	2.9	1.2	2.2	0.9	2.8	1.1	29.9	6.1
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	6.3	2.3	5.2	1.7	3.6	1.1	2.2	0.7	2.7	0.8	2.9	0.8	28.3	4.3
8-Post	7.6	2.6	7.3	2.3	4.5	1.4	2.7	1.0	2.9	1.1	2.9	1.0	32.2	5.4
9-Pre	7.6	2.9	5.6	1.7	4.0	1.2	2.4	0.6	2.2	0.8	1.9	0.7	26.6	5.0
9-Post	10.8	5.3	6.3	2.0	4.2	1.4	2.4	0.7	2.3	0.7	1.9	0.6	30.6	6.8
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	6.4	3.1	4.7	1.6	2.8	1.0	2.0	0.7	1.9	0.6	1.9	0.7	23.1	5.4
11-Pre	5.3	1.8	3.9	1.2	3.5	1.2	2.9	1.0	3.0	0.9	2.2	0.8	24.6	3.7

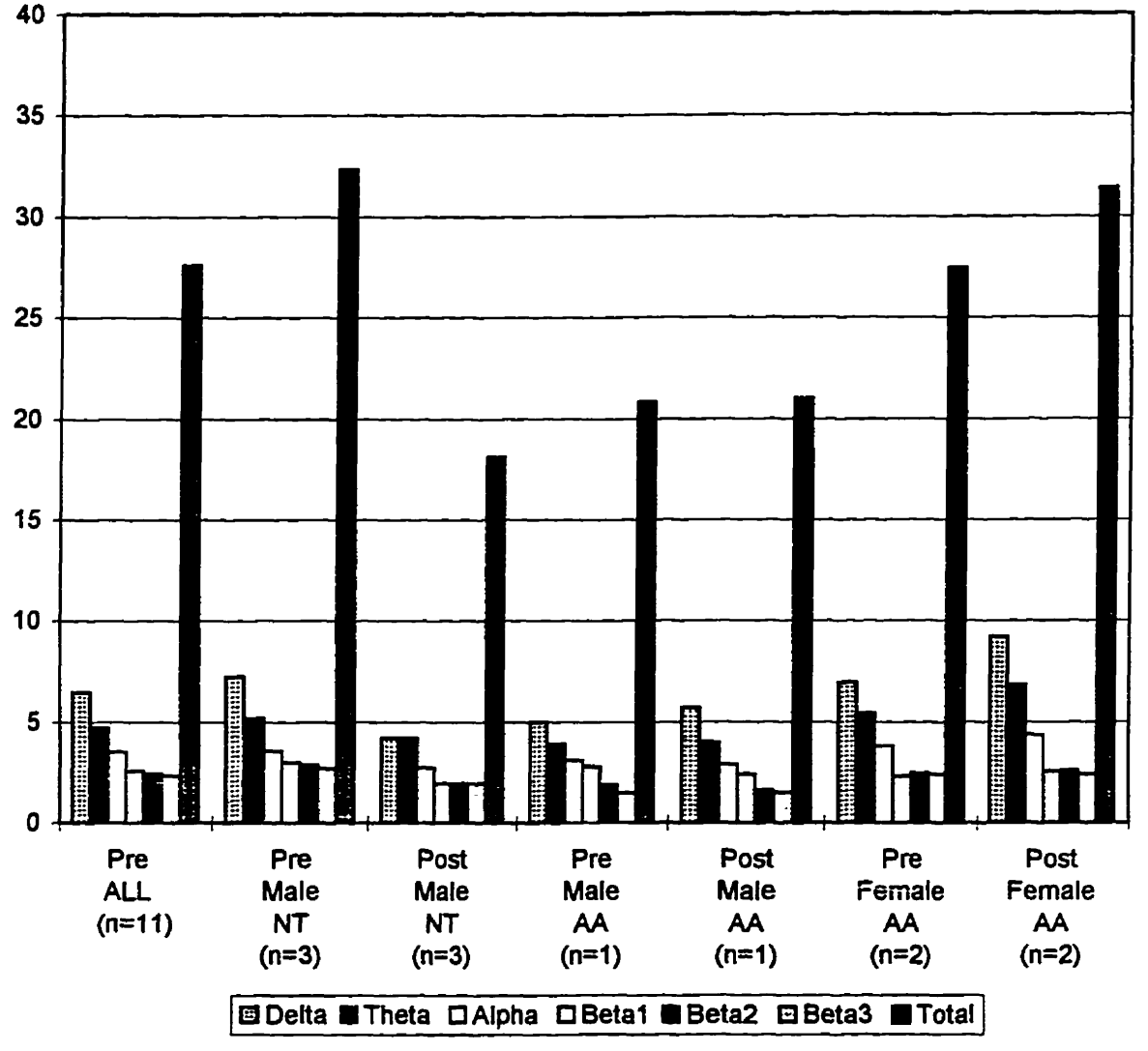
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site F4: Reading Silently Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.6	4.2	5.7	2.0	4.6	2.6	3.1	1.7	2.6	1.9	2.5	1.7	32.0	11.0
1-Post	3.1	1.1	2.8	1.0	2.0	0.7	1.5	0.5	1.6	0.5	1.7	0.7	20.3	3.5
2-Pre	7.2	2.8	5.5	1.8	3.6	1.3	3.6	1.5	3.1	1.0	2.9	0.9	30.9	5.5
2-Post	4.4	1.4	2.7	1.4	2.6	0.9	2.1	1.4	2.0	0.7	2.0	0.8	26.9	5.6
3-Pre	7.5	2.2	5.2	1.7	3.5	0.8	3.1	1.1	3.7	1.2	3.6	1.1	43.6	6.5
3-Post	6.4	2.5	5.1	1.7	3.7	1.1	2.5	0.8	2.4	0.6	2.1	0.6	25.7	3.7
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	4.6	1.9	3.9	1.2	2.2	0.6	2.0	0.6	2.2	0.8	2.4	0.8	26.7	4.3
5-Pre	7.4	2.4	4.6	1.5	3.4	1.3	2.4	1.0	1.8	0.8	1.6	0.9	24.0	6.3
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	4.9	1.8	4.0	1.4	3.1	1.0	2.8	0.9	1.9	0.5	1.6	0.5	20.9	3.7
6-Post	6.0	3.4	4.0	1.5	2.8	1.3	2.4	0.8	1.7	0.6	1.5	0.5	21.4	5.3
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	6.6	2.2	4.9	1.6	5.5	2.5	2.9	1.0	2.4	0.8	3.0	1.0	30.0	5.2
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	6.8	2.9	4.7	1.7	3.2	1.0	2.0	0.7	2.0	0.6	2.1	0.6	24.7	4.7
8-Post	8.0	2.5	7.9	2.8	5.1	1.7	2.8	1.6	3.1	1.0	2.7	0.8	33.6	6.4
9-Pre	8.1	2.9	6.0	1.8	4.6	1.3	2.6	0.7	2.4	0.8	2.1	0.8	28.7	4.5
9-Post	11.6	6.8	6.0	2.3	3.8	1.3	2.2	0.7	2.2	0.7	2.0	0.6	30.8	8.8
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	6.6	3.0	5.2	1.8	3.0	0.9	2.2	0.7	1.9	0.7	1.9	0.7	24.2	4.9
11-Pre	5.7	2.2	4.2	1.3	4.0	1.3	3.1	1.0	3.1	1.0	2.3	0.7	26.0	4.2

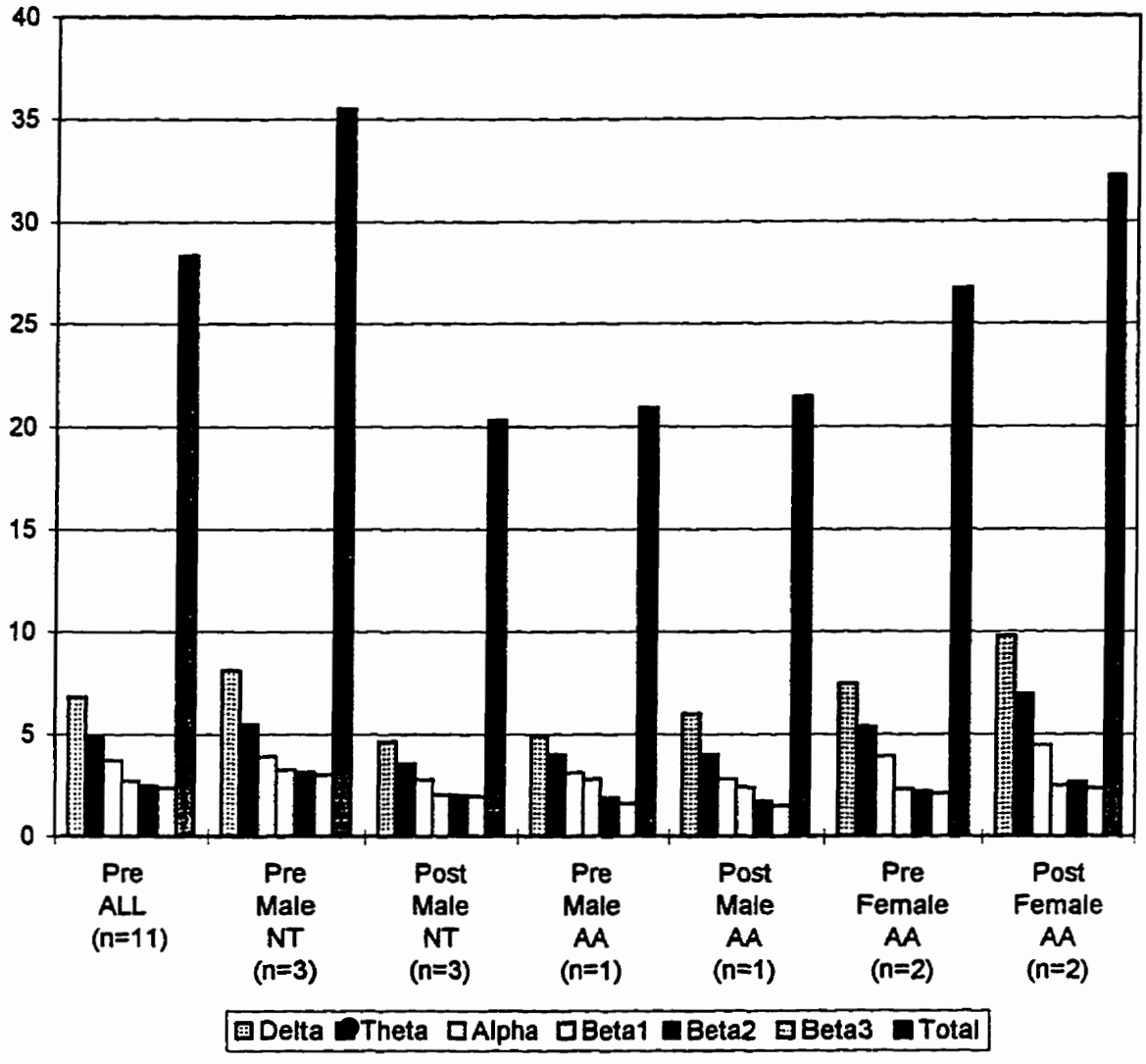
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site F3: Reading Silently Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta=0.5-3.9 Hz, Theta=4-7.9Hz, Alpha=8-11.9Hz, Beta1=12-15.9Hz, Beta2=16-19.9 Hz, Beta3=20-23.9Hz, Total=24-32Hz, M=Mean, SD=Standard Deviation. Magnitude values are in microvolts.

Site F4: Reading Silently Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site C3: Reading Silently Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.5	3.9	5.7	2.0	4.7	2.3	3.1	1.5	2.5	1.8	2.3	1.6	31.5	10.2
1-Post	2.9	1.1	2.2	0.6	1.7	0.6	1.2	0.4	1.2	0.4	1.2	0.4	16.3	2.5
2-Pre	5.5	1.8	4.2	1.3	2.6	1.0	2.7	1.1	2.4	0.8	2.2	0.7	23.1	4.0
2-Post	3.9	1.6	3.7	1.2	2.4	0.8	2.1	0.8	1.8	0.7	1.7	0.6	18.5	3.5
3-Pre	5.4	1.9	4.3	1.6	3.4	0.9	3.0	0.9	3.6	1.4	3.0	1.3	38.2	7.1
3-Post	4.4	1.5	4.2	1.2	4.0	1.5	3.1	1.1	3.0	1.1	2.5	0.9	26.4	4.8
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	4.6	1.3	3.2	0.9	2.4	0.7	2.3	0.7	2.4	0.9	2.7	0.8	28.3	5.1
5-Pre	12.3	6.0	4.9	2.9	3.1	1.3	2.4	1.0	1.8	0.8	1.8	0.9	29.4	10.5
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	4.9	2.0	3.5	1.2	3.0	1.4	2.8	1.2	2.1	0.6	1.6	0.4	20.7	4.0
6-Post	5.0	2.0	3.6	1.1	2.8	1.4	2.4	0.8	1.7	0.6	1.5	0.5	19.7	3.7
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	7.0	1.7	4.6	1.5	8.4	4.1	3.7	1.3	2.5	1.1	3.5	1.3	35.0	7.0
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	5.1	2.4	3.9	1.3	3.2	0.8	2.4	0.7	2.7	0.7	2.9	0.7	26.2	3.6
8-Post	7.6	2.7	6.2	1.7	4.1	1.3	2.6	0.9	3.0	1.0	3.2	1.0	31.4	4.5
9-Pre	6.8	2.5	5.2	1.6	4.1	1.4	2.3	0.7	2.2	0.7	1.9	0.7	25.1	4.4
9-Post	10.8	5.4	5.4	1.7	3.8	1.5	2.1	0.6	2.0	0.7	1.6	0.5	28.0	6.6
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	6.1	2.8	4.1	1.4	2.7	1.1	1.9	0.6	1.9	0.6	1.9	0.6	22.0	4.9
11-Pre	5.6	1.6	4.3	1.3	4.3	1.5	3.2	1.2	3.2	0.9	2.3	0.8	26.4	3.7

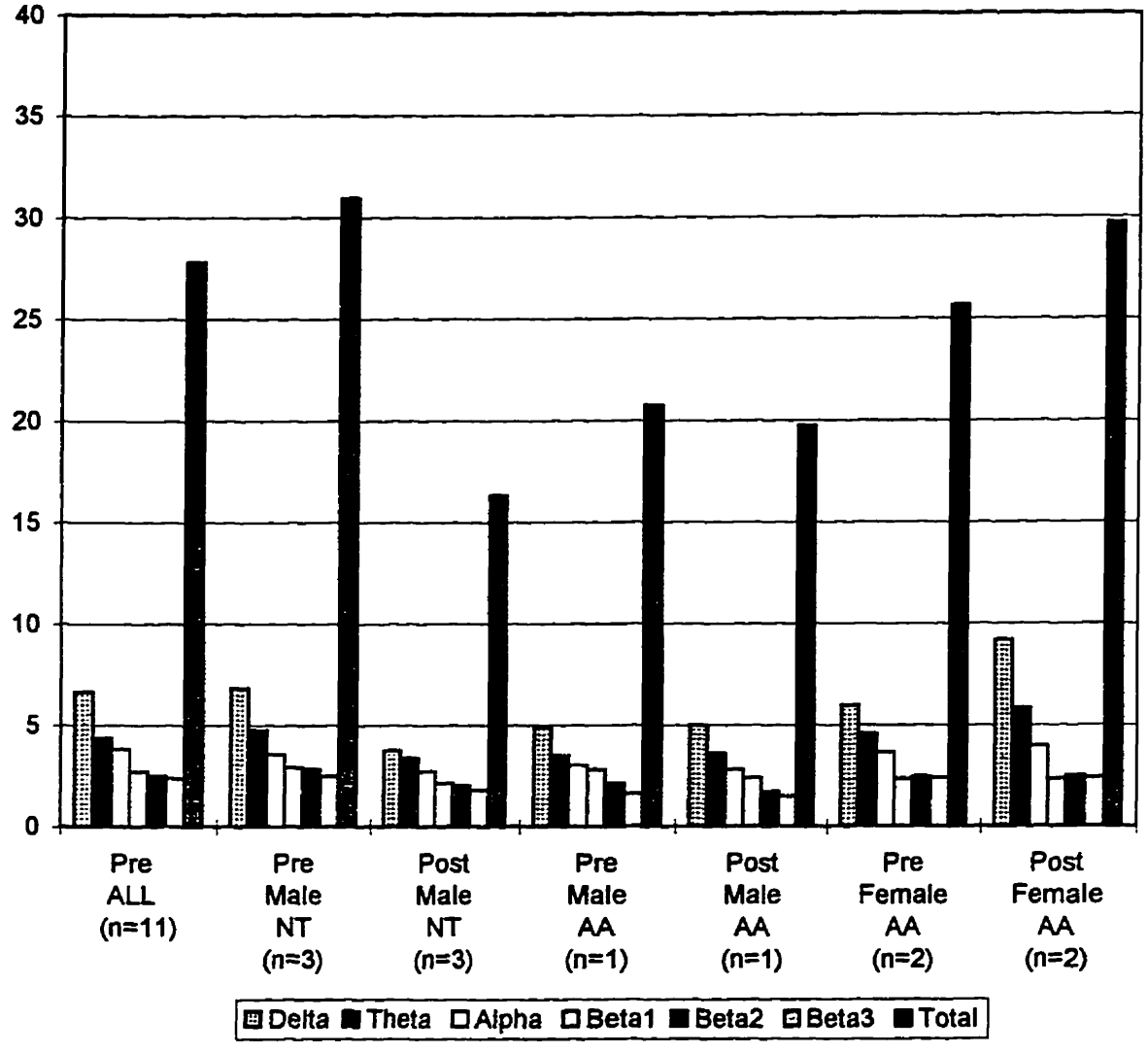
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site C4: Reading Silently Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.3	3.8	5.6	1.8	4.6	2.5	3.0	1.5	2.6	1.7	2.4	1.5	31.2	9.8
1-Post	3.0	1.3	2.3	0.7	2.1	1.0	1.5	0.4	1.4	0.4	1.3	0.4	18.1	2.7
2-Pre	6.5	2.4	4.9	1.7	3.4	1.4	3.4	1.4	2.8	1.0	2.6	0.8	27.7	5.5
2-Post	4.1	1.6	3.8	1.4	2.3	0.9	2.1	1.0	1.8	0.7	1.7	0.6	18.6	3.7
3-Pre	5.3	1.7	4.5	1.6	3.5	1.1	3.2	0.8	3.6	1.2	3.4	1.2	39.8	5.7
3-Post	4.8	1.4	4.0	1.2	3.9	1.5	2.5	0.7	2.3	0.7	2.0	0.6	23.3	2.9
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	4.4	1.4	3.1	1.0	2.3	0.7	2.7	0.8	3.5	1.6	3.3	1.3	33.6	7.0
5-Pre	6.1	3.0	4.3	1.8	3.3	1.5	2.4	1.1	1.8	0.7	1.7	0.8	22.4	7.2
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	4.5	1.3	3.6	1.1	2.9	1.2	2.7	1.1	2.1	0.7	1.7	0.5	20.7	3.3
6-Pre	5.2	2.1	3.7	1.1	2.8	1.4	2.5	0.8	1.9	0.7	1.7	0.5	22.7	4.3
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	5.2	1.8	4.3	1.5	7.0	3.3	3.8	1.3	3.0	1.2	3.8	1.4	33.4	6.5
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	4.9	2.0	3.4	1.2	2.6	0.8	1.8	0.6	1.9	0.5	2.2	0.7	21.4	3.4
8-Post	8.4	2.9	7.4	2.3	4.8	1.6	2.9	1.2	3.3	1.2	3.2	0.9	34.4	6.0
9-Pre	7.6	2.6	6.0	1.9	4.8	1.6	2.8	0.9	2.6	1.0	2.3	0.9	29.2	5.6
9-Post	8.8	3.2	5.9	1.7	4.5	1.7	2.5	0.8	2.3	0.8	2.1	0.7	29.0	4.7
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	6.3	2.9	4.1	1.4	2.6	0.9	1.9	0.6	1.8	0.6	1.7	0.6	21.6	4.6
11-Pre	6.2	2.2	4.8	1.6	5.2	1.8	3.7	1.6	3.7	1.1	2.5	0.7	29.6	4.3

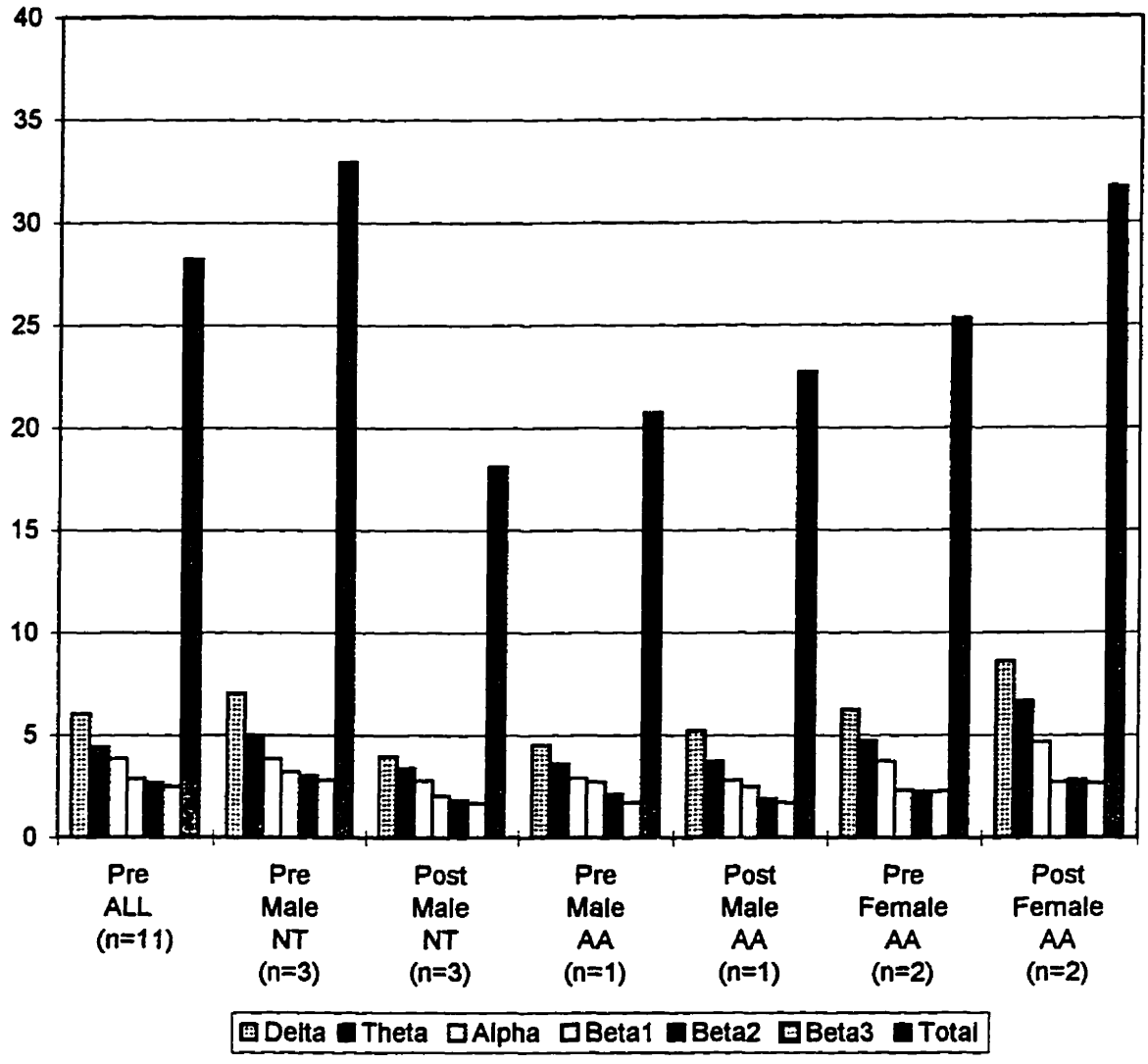
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site C3: Reading Silently Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site C4: Reading Silently Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site P3: Reading Silently Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.3	3.7	5.8	2.1	4.7	2.2	3.3	1.4	2.6	1.7	2.4	1.5	32.2	9.3
1-Post	3.0	1.1	2.0	0.5	1.6	0.5	1.2	0.3	1.2	0.3	1.1	0.3	15.3	2.1
2-Pre	5.0	1.8	3.7	1.2	2.5	0.9	2.5	1.0	2.1	0.7	2.1	0.6	21.5	3.5
2-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
3-Pre	5.4	1.7	4.1	1.5	4.5	1.6	3.2	0.9	3.5	1.1	3.0	1.2	38.7	5.7
3-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	4.3	1.4	3.1	0.9	2.3	0.9	2.1	0.6	2.0	0.6	2.1	0.5	24.6	3.4
5-Pre	5.7	2.2	4.7	1.6	3.5	1.3	2.8	1.1	1.9	0.7	1.8	1.0	23.6	6.9
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	4.2	1.5	3.2	0.9	3.1	1.4	2.8	1.0	2.2	0.6	1.8	0.6	20.5	3.2
6-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	5.3	1.5	4.8	1.4	9.2	4.3	4.2	1.6	2.8	0.8	3.6	1.4	35.3	7.4
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	3.6	2.7	2.3	1.5	1.7	0.9	1.4	0.5	1.6	0.5	1.8	0.6	16.4	5.5
8-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
9-Pre	6.4	2.7	3.9	1.2	2.9	1.0	1.7	0.5	1.7	0.5	1.5	0.5	20.3	4.3
9-Post	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	5.0	2.1	3.7	1.2	2.9	1.3	2.1	0.8	2.1	0.8	2.1	0.8	22.0	4.9
11-Pre	5.8	1.9	4.4	1.4	4.6	1.6	3.3	1.1	3.0	0.8	2.4	0.7	27.6	3.7

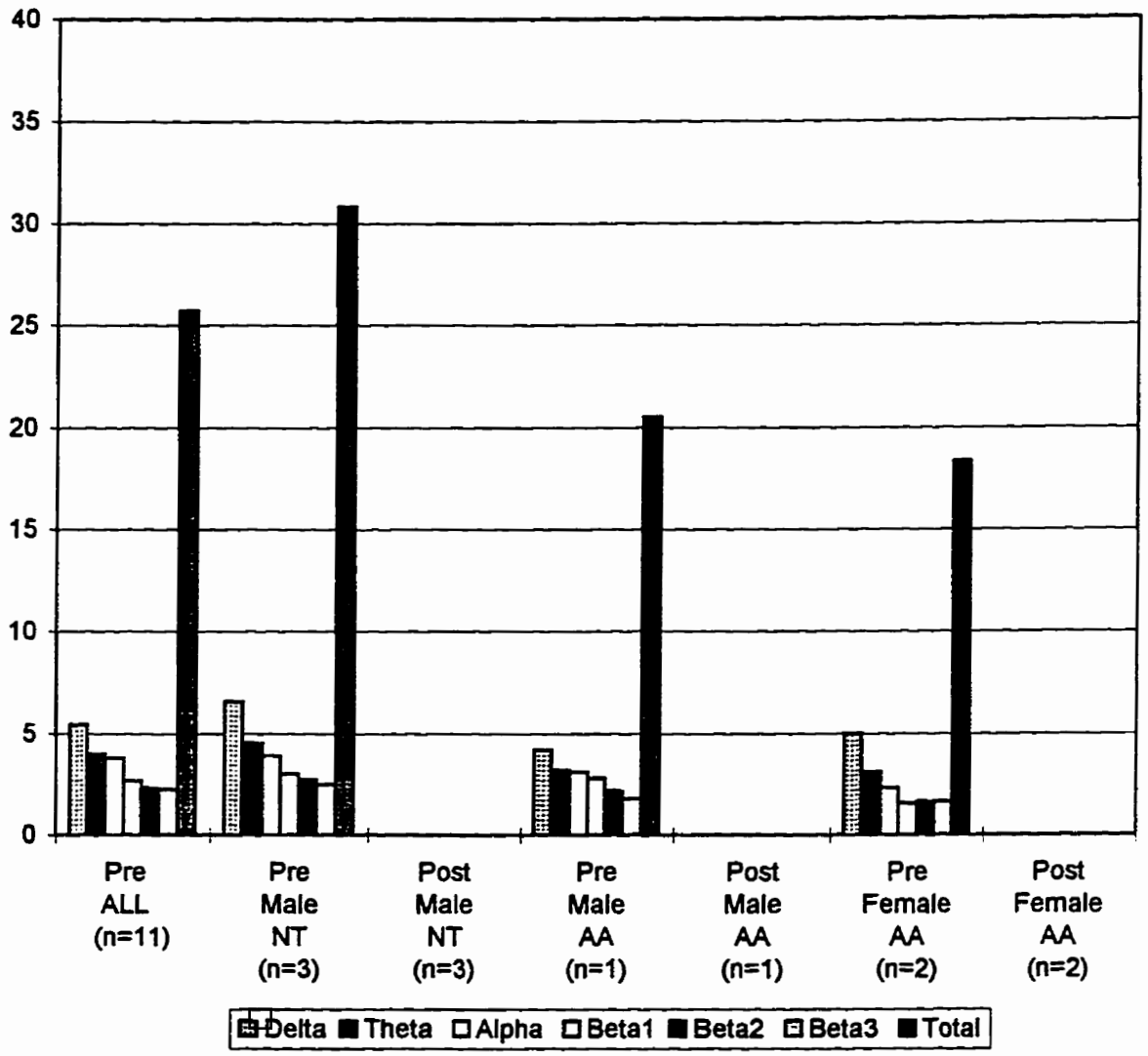
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site P4: Reading Silently Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.0	3.6	5.6	2.0	4.5	2.3	3.2	1.5	2.7	1.6	2.4	1.5	31.5	9.4
1-Post	3.2	1.2	2.1	0.6	2.0	0.9	1.5	0.4	1.4	0.4	1.3	0.4	18.3	2.4
2-Pre	5.8	2.1	4.6	1.5	3.1	1.1	3.0	1.3	2.5	0.8	2.4	0.8	25.1	4.6
2-Post	3.9	1.7	3.4	1.2	2.2	0.8	2.0	0.9	1.6	0.6	1.4	0.4	17.0	3.3
3-Pre	5.4	1.7	4.1	1.5	4.5	1.6	3.2	0.9	3.5	1.1	3.0	1.2	38.7	5.7
3-Post	4.0	1,3	3.0	0.8	3.6	1.6	2.2	0.6	1.9	0.6	1.7	0.4	19.3	2.6
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	4.0	1.4	2.9	1.0	2.2	0.8	2.0	0.6	2.2	0.7	2.3	0.7	24.8	3.4
5-Pre	5.9	2.4	4.2	1.5	3.2	1.3	2.5	1.0	1.9	0.8	1.9	0.9	23.1	6.3
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	3.9	1.2	3.2	0.8	2.9	1.1	2.5	0.9	2.2	0.7	1.8	0.5	19.9	2.9
6-Post	4.3	1.7	3.1	1.0	2.8	1.4	2.6	0.9	2.1	0.7	2.0	0.6	20.8	3.9
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	4.7	1.7	4.9	1.5	7.7	4.0	3.6	1.1	2.6	0.8	2.9	1.1	31.9	6.5
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	3.8	1.5	2.6	0.9	2.3	0.7	1.8	0.6	2.0	0.5	2.1	0.6	18.7	2.7
8-Post	8.4	2.7	6.1	1.9	4.8	1.6	2.9	0.8	3.1	1.3	3.4	0.9	33.3	5.1
9-Pre	6.7	2.5	5.3	1.6	4.3	1.7	2.9	1.0	2.5	0.8	2.3	0.7	27.9	5.3
9-Post	8.4	3.0	5.6	1.7	4.8	2.3	2.7	0.8	2.4	0.8	2.0	0.6	28.9	4.8
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	4.2	1.9	2.9	1.6	2.0	1.0	1.5	0.6	1.4	0.6	1.3	0.4	15.8	4.4
11-Pre	6.3	2.2	5.2	1.9	5.0	1.8	3.5	1.0	3.4	0.9	2.5	0.7	29.8	4.1

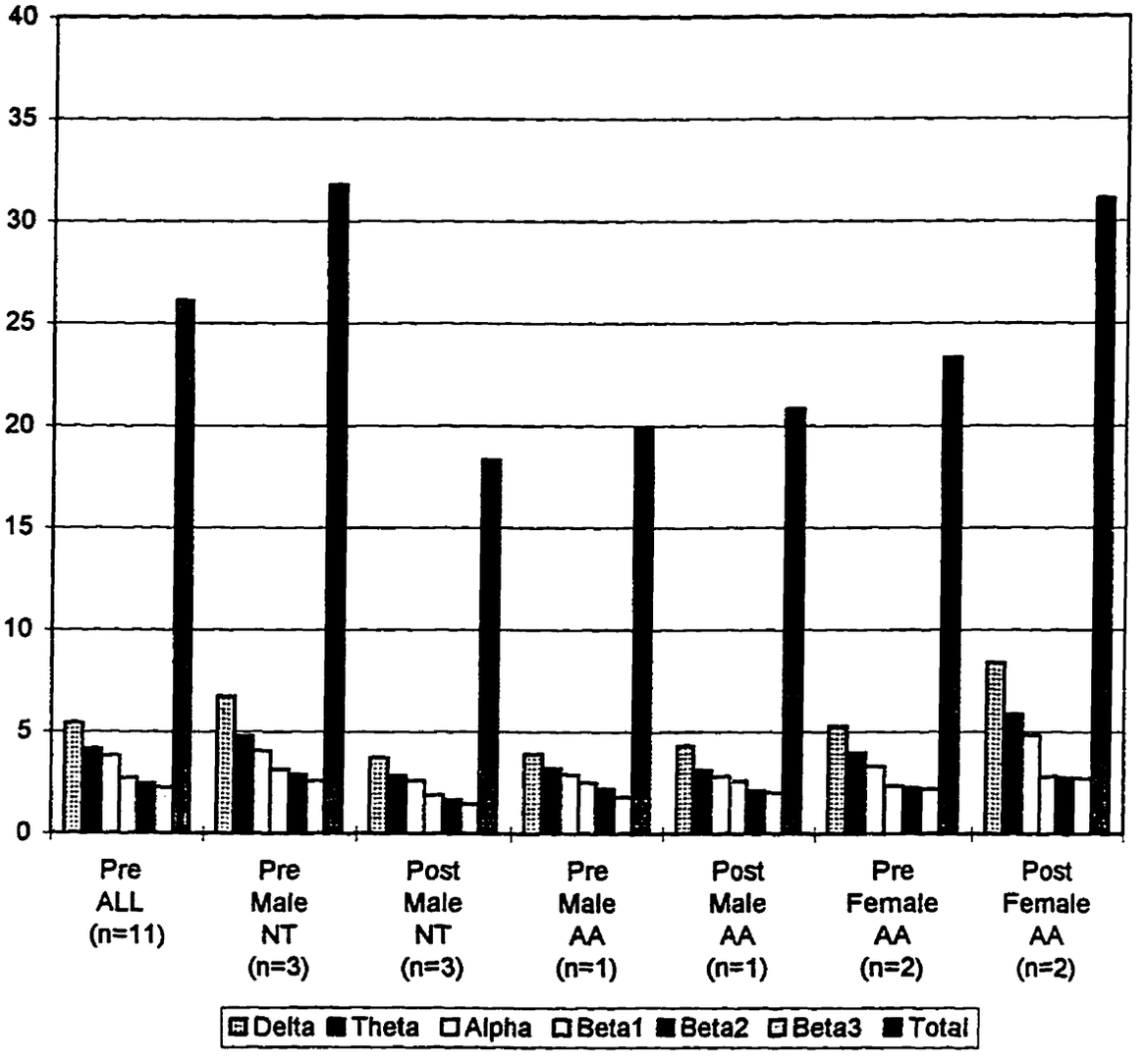
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site P3: Reading Silently Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site P4: Reading Silently Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta=0.5-3.9 Hz, Theta=4-7.9Hz, Alpha=8-11.9Hz, Beta1=12-15.9Hz, Beta2=16-19.9 Hz, Beta3=20-23.9Hz, Total=24-32Hz, M=Mean, SD=Standard Deviation. Magnitude values are in microvolts.

Site T5: Reading Silently Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.5	4.3	5.8	2.1	4.7	2.5	3.3	1.5	2.7	1.9	2.5	1.7	32.6	0.9
1-Post	2.5	1.0	1.6	0.4	1.7	0.5	1.8	0.5	1.8	0.5	1.9	0.6	20.2	2.7
2-Pre	3.9	1,3	2.7	0.8	2.3	0.7	2.3	0.8	2.4	0.7	2.6	0.8	20.6	3.0
2-Post	2.8	1.1	3.7	0.9	3.0	0.8	3.1	1.0	2.8	0.9	2.8	1.0	23.1	4.5
3-Pre	4.2	1.5	3.3	1.1	3.5	1.6	3.2	0.8	2.7	0.9	2.6	0.7	32.4	4.2
3-Post	3.1	1.2	2.7	0.7	2.8	1.1	1.7	0.6	1.4	0.5	1.2	0.3	15.0	2.7
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	3.7	1.1	3.1	0.8	2.8	0.9	3.3	1.0	3.4	1.1	3.8	1.3	34.2	5.7
5-Pre	5.2	2.3	3.8	1.4	2.6	1.2	2.1	1.1	1.4	1.0	1.4	1.3	18.9	9.0
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	3.1	1.2	2.3	0.8	2.5	1.1	2.4	0.9	1.8	0.5	1.9	0.6	17.5	3.0
6-Post	2.8	1.0	2.4	0.7	2.7	1.3	3.2	1.0	2.4	0.8	2.6	0.8	21.7	3.5
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	4.2	1.3	4.0	1.1	6.3	2.9	3.9	1.3	3.0	0.8	3.4	1.0	30.5	4.9
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	3.6	2.7	2.3	1.5	1.7	0.9	1.4	0.5	1.6	0.5	1.8	0.6	16.4	5.5
8-Post	7.2	2.3	4.3	1.3	3.2	0.8	2.3	0.8	2.9	0.8	3.3	1.0	29.4	4.4
9-Pre	4.5	1.6	2.9	1.0	2.5	0.8	1.7	0.6	1.4	0.5	1.3	0.4	16.3	2.9
9-Post	13.7	6.1	5.0	1.7	2.9	1.1	1.7	0.5	1.5	0.5	1.2	0.4	27.8	7.3
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	3.3	1.6	2.4	0.7	1.9	0.7	1.4	0.5	1.5	0.5	1.4	0.5	14.5	3.2
11-Pre	4.0	1.3	3.2	1.0	3.5	1.2	2.7	0.9	2.7	0.9	2.7	0.7	23.5	3.4

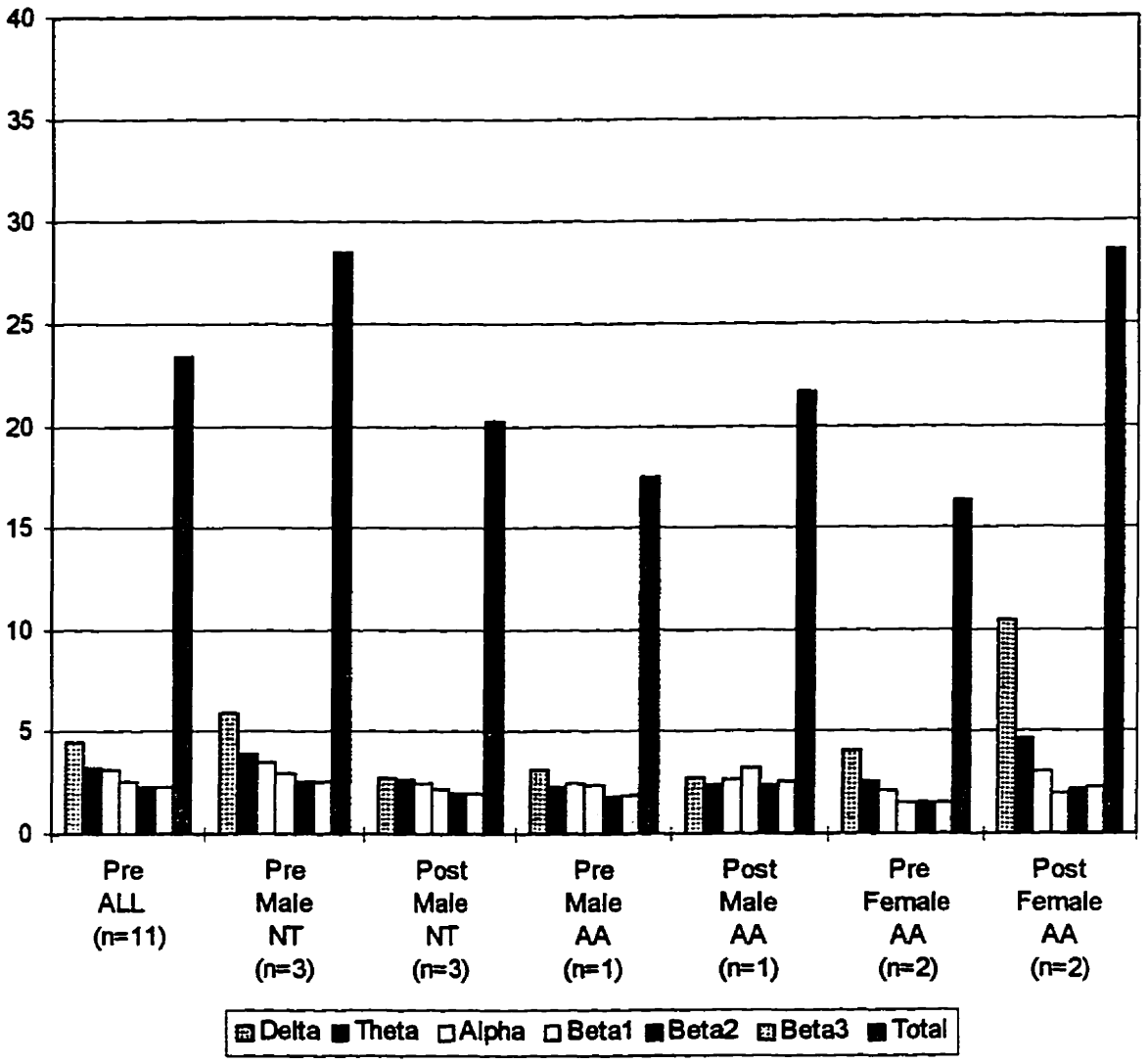
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site T6: Reading Silently Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	8.6	3.6	5.3	2.0	4.3	2.2	3.2	1.5	2.8	1.7	2.4	1.5	30.7	9.3
1-Post	2.7	0.9	1.9	0.6	2.3	0.8	2.3	0.8	2.4	0.7	2.2	0.6	25.3	3.7
2-Pre	5.7	1.0	4.3	0.9	3.3	0.5	3.4	0.8	3.2	0.7	3.2	0.8	28.9	5.1
2-Post	2.9	1.0	2.6	0.9	1.6	0.5	1.8	0.7	1.8	0.7	1.8	0.8	15.8	3.3
3-Pre	5.0	1.5	4.0	1.2	4.4	1.5	3.6	1.0	3.8	1.1	3.3	1.2	40.5	1.0
3-Post	3.9	1.2	3.3	1.0	4.4	2.1	2.6	0.7	2.2	0.6	2.0	0.5	21.5	3.8
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	3.2	1.1	2.8	0.9	2.8	0.9	2.6	0.8	2.7	0.7	3.2	1.1	28.8	4.3
5-Pre	5.3	2.5	3.4	1.1	2.8	1.3	2.2	0.9	1.8	0.8	1.9	0.9	20.4	6.3
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	2.5	0.7	2.2	0.6	2.6	1.1	2.2	0.6	2.0	0.6	2.1	0.7	17.3	2.5
6-Post	3.6	1.4	2.3	0.9	2.6	1.2	2.6	0.8	2.2	0.8	2.2	0.7	19.6	3.8
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	3.5	1.2	4.0	1.1	4.9	2.2	2.8	1.0	2.3	0.7	2.4	0.7	24.7	3.6
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	3.8	1.5	2.6	0.9	2.3	0.7	1.8	0.6	2.0	0.5	2.1	0.6	18.7	2.7
8-Post	7.3	2.4	4.6	1.5	3.7	1.2	2.7	0.8	3.0	1.1	3.2	0.9	29.3	3.8
9-Pre	6.3	4.2	4.4	1.6	3.5	1.3	2.5	0.8	2.1	0.7	2.0	0.6	24.3	6.9
9-Post	8.5	3.4	4.5	1.6	3.5	1.3	2.2	0.7	1.9	0.6	1.7	0.5	25.3	4.8
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	3.3	1.6	2.4	0.7	1.9	0.7	1.4	0.5	1.5	0.5	1.4	0.5	14.5	3.2
11-Pre	4.5	1.7	3.8	1.3	4.2	1.6	2.9	1.0	2.5	0.8	2.1	0.6	23.5	3.5

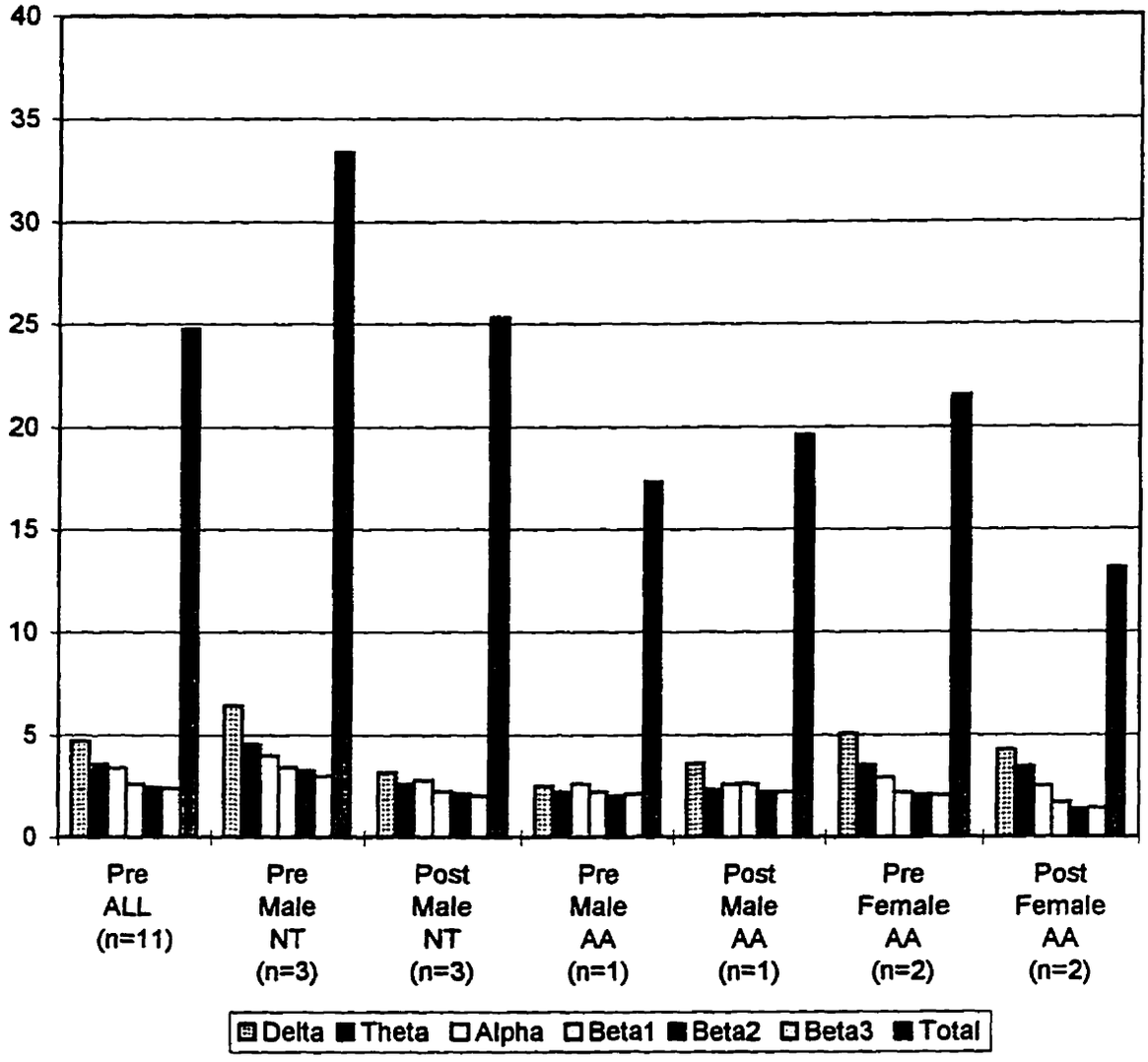
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site T5: Reading Silently Pre and Post-Assessment Average QEEG Magnitudes, N=11



Delta=0.5-3.9 Hz, Theta=4-7.9Hz, Alpha=8-11.9Hz, Beta1=12-15.9Hz, Beta2=16-19.9 Hz, Beta3=20-23.9Hz, Total=24-32Hz, M=Mean, SD=Standard Deviation. Magnitude values are in microvolts.

Site T6: Reading Silently Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site O1: Reading Silently Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	9.6	4.1	6.1	2.1	4.7	2.4	3.4	1.4	2.9	1.8	2.7	1.5	34.3	9.6
1-Post	3.1	1.1	2.0	0.6	1.9	0.6	1.6	0.4	1.7	0.5	1.6	0.8	20.3	2.7
2-Pre	4.6	1.7	3.5	1.1	2.4	0.8	2.1	0.8	2.0	0.6	1.9	0.6	20.0	3.4
2-Post	3.4	1.2	4.3	1.0	2.9	0.7	2.7	0.7	2.1	0.5	1.8	0.6	19.8	3.1
3-Pre	4.9	1.5	4.3	1.4	4.2	1.2	4.3	1.3	4.1	1.5	3.7	1.2	45.1	5.7
3-Post	3.4	1.3	2.8	0.7	2.7	0.9	1.8	0.6	1.5	0.4	1.4	0.4	16.0	2.8
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	3.4	1.0	4.0	1.2	3.0	1.0	2.6	0.9	2.3	0.8	2.4	0.6	27.5	4.3
5-Pre	5.3	2.3	5.1	1.5	3.6	1.3	2.8	1.1	2.0	0.8	1.9	1.1	24.1	7.1
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	3.0	1.2	3.2	0.9	3.8	1.2	3.1	1.1	3.4	1.1	3.4	1.0	27.1	4.0
6-Post	3.4	1.1	2.8	0.9	3.6	1.3	3.7	1.3	3.1	1.0	3.8	1.2	27.8	3.5
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	4.5	1.3	7.3	1.7	6.9	2.3	4.5	1.5	3.4	0.9	3.9	1.5	37.5	4.8
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	4.1	1.7	3.3	1.1	3.0	0.8	2.4	0.8	2.8	0.8	3.1	0.9	25.6	3.6
8-Post	7.1	2.4	4.8	1.4	3.9	1.1	2.6	0.8	3.1	0.9	3.2	1.0	31.4	4.3
9-Pre	5.4	1.9	3.3	1.1	2.5	0.8	1.6	0.5	1.5	0.5	1.3	0.5	17.8	3.5
9-Post	8.2	3.1	4.6	1.3	3.9	1.6	2.5	0.8	2.0	0.7	1.7	0.5	25.7	4.2
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	5.1	2.4	3.4	1.2	2.6	1.1	1.9	0.7	2.0	0.7	2.0	0.7	20.7	5.1
11-Pre	4.7	1.5	3.7	1.2	5.2	1.5	3.7	1.2	5.1	1.7	5.6	1.6	39.8	6.1

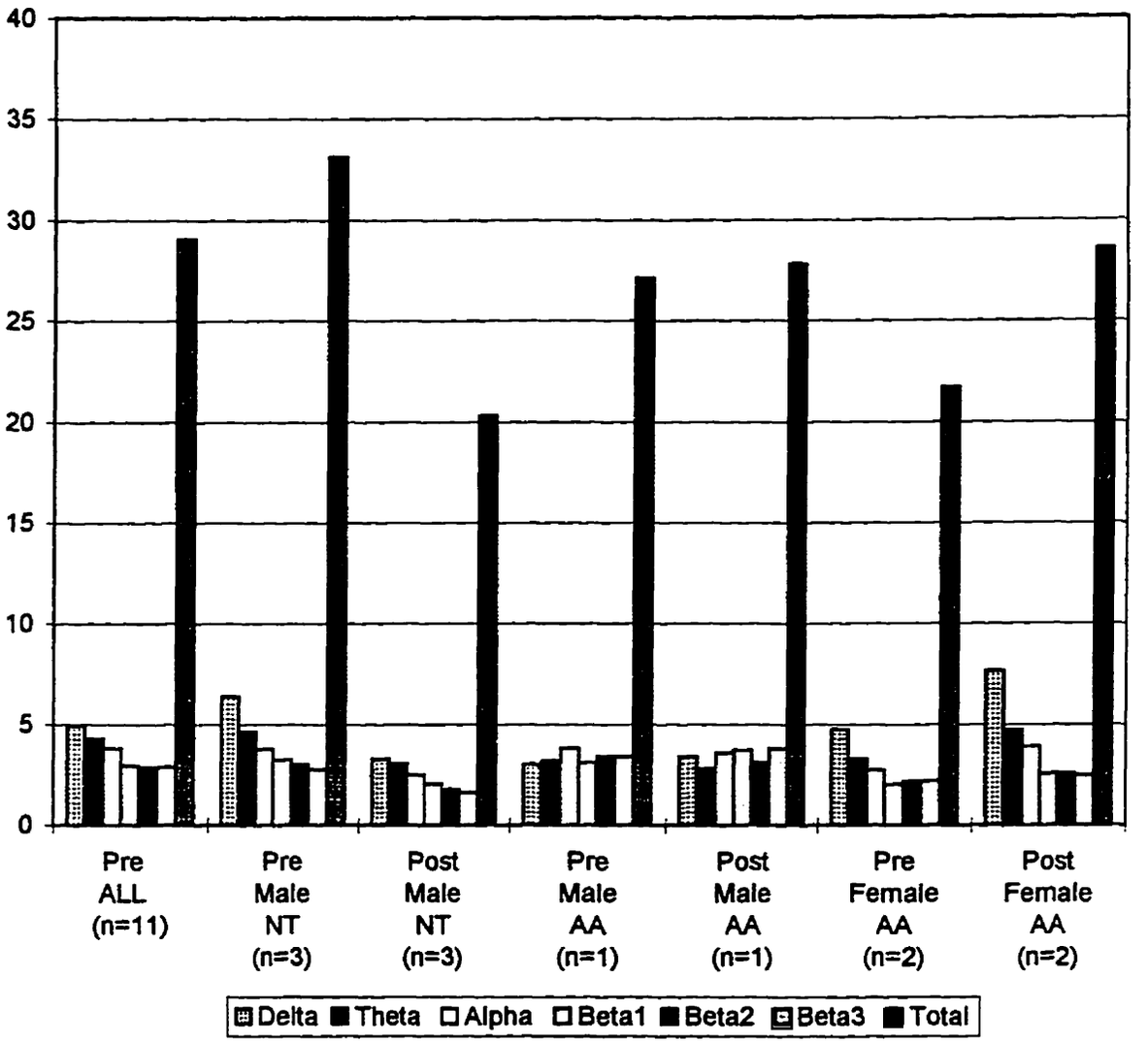
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site O2: Reading Silently Pre and Post-Assessment QEEG Averages, N = 11

Case	Delta1		Theta		Alpha		Beta1		Beta2		Beta3		Total	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Neurofeedback Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
1-Pre	8.9	3.7	5.8	2.0	4.8	2.3	3.5	1.4	3.4	1.6	3.5	1.6	36.2	8.7
1-Post	2.7	1.0	1.9	0.6	1.9	0.5	1.9	0.6	2.2	0.6	2.0	0.6	23.1	2.9
2-Pre	4.9	1.8	3.7	1.2	2.4	0.8	2.2	0.9	2.1	0.6	2.1	0.7	20.9	3.9
2-Post	3.2	1.3	3.5	1.0	2.1	0.6	1.9	0.6	1.6	0.4	1.4	0.5	15.9	2.7
3-Pre	4.8	1.5	3.9	1.4	4.1	1.5	3.9	1.2	4.0	1.6	3.6	1.3	42.0	6.6
3-Post	3.4	1.1	3.4	1.0	3.8	1.7	2.6	0.6	2.1	0.6	1.8	0.4	20.0	3.2
Neurofeedback Male Alcoholics Dropped (Pre-Assessment QEEG Recordings only)														
4-Pre	2.9	0.9	3.5	1.0	3.1	1.1	2.8	0.8	2.9	0.8	2.9	0.8	29.8	4.0
5-Pre	6.3	2.5	4.7	1.3	3.8	1.3	2.9	1.1	2.4	0.9	2.5	0.9	27.1	6.0
AA Male Alcoholics (Pre and Post-Assessment QEEG Recordings)														
6-Pre	3.1	1.4	2.6	0.9	2.8	0.9	2.3	0.8	2.5	0.7	2.5	0.7	20.7	2.7
6-Post	3.6	1.4	2.6	0.9	2.9	1.1	2.9	0.9	2.5	0.8	2.8	0.9	22.8	3.4
AA Male Alcoholics (Pre-Assessment QEEG Recordings Only)														
7-Pre	4.3	1.0	6.6	1.4	5.9	1.8	4.0	1.4	3.2	0.8	3.2	1.1	34.1	3.9
AA Female Alcoholics (Pre and Post-Assessment QEEG Recordings)														
8-Pre	4.4	1.7	3.1	1.0	2.9	0.9	2.1	0.7	2.3	0.6	2.7	0.8	23.2	3.4
8-Post	7.2	2.6	5.0	1.5	4.0	1.2	2.7	0.8	2.7	0.8	3.1	0.9	30.2	4.3
9-Pre	5.7	2.1	4.1	1.5	4.2	1.6	3.2	1.1	3.0	0.9	2.9	0.8	29.0	5.1
9-Post	7.7	2.8	4.5	1.4	4.6	1.9	2.9	0.9	2.6	0.9	2.5	0.8	29.3	4.2
AA Female Alcoholics (Pre-Assessment QEEG Recordings only)														
10-Pre	5.1	1.9	3.0	0.9	2.6	1.1	1.9	0.7	2.1	0.8	2.0	0.7	20.7	4.5
11-Pre	5.0	1.4	3.4	1.1	3.7	1.0	2.7	1.0	3.2	1.1	3.2	1.4	27.7	5.3

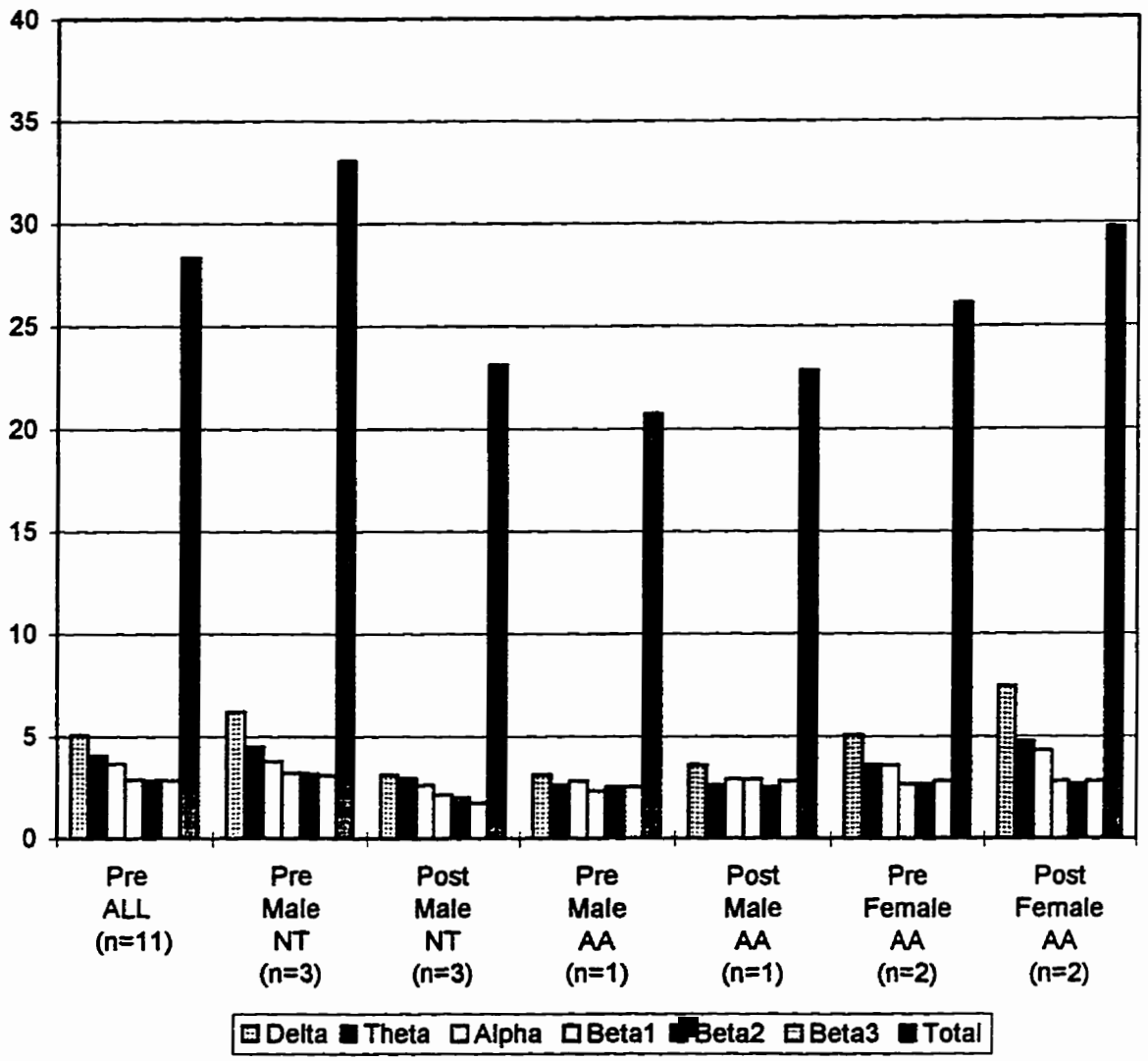
Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. All values are in micro volts.

Site O1: Reading Silently Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.

Site O2: Reading Silently Pre and Post-Assessment Averaged QEEG Magnitudes, N=11



Delta = 0.5-3.9 Hz, Theta = 4-7.9Hz, Alpha = 8-11.9Hz, Beta1 = 12-15.9Hz, Beta2 = 16-19.9 Hz, Beta3 = 20-23.9Hz, Total = 24-32Hz, M = Mean, SD = Standard Deviation. Magnitude values are in microvolts.