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FEEDBACK ELECTROENCEPHALOGRAPHY

TECHNIQUES AND CLINICAL APPLICATION

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ABSTRACT

A series of investigations into feedback training of the human electroencephalogram (EEG) is presented.

Firstly, a number of studies of methodological and theoretical issues, using feedback training of the 8-12 Hz occipital alpha rhythm as a paradigm, are reported. It is evident that some subjects are able both to suppress and to enhance their alpha activity from baseline levels; but, because individual differences are large, these training effects are difficult to demonstrate by the analysis of group data.

Secondly, an examination of the detection and feedback training of the 12-16 Hz, so-called, sensorimotor rhythm (SMR) in the rolandic EEG is described. The evidence for the existence of this EEG pattern in man is seen to be lacking. The techniques that are used to detect such EEG signals are demonstrated to have a crucial influence on the results obtained.

Thirdly, a clinical study to assess claims that SMR feedback training is an effective treatment for epileptic seizure disorders is reported. This study involved a comparison between feedback training of rolandic EEG in the SMR frequency range and several control procedures, using three adult patients each with a history of drug-resistant, generalised seizures. A marked clinical improvement for all three patients was obtained; but, although specific changes in the EEG did occur, this was not related to the training of any particular EEG component.

Some physiological mechanisms for the training and clinical effects observed in these studies are proposed.

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1:1

HISTORICAL BACKGROUND

1:1:1 Feedback Electroencephalography

Feedback electroencephalography is concerned with the experimental modification of human electroencephalographic (EEG) activity through a training procedure involving the provision of sensory feedback information (Mulholland, 1968). In this chapter the historical origins of this particular area of research, and the claims that have been made for its application to the treatment of epilepsy, will be briefly outlined.

The experimental modification of EEG activity began when a number of early investigators observed that the characteristic desynchronization of the 8-12 Hz occipital alpha rhythm by visual stimuli could be established as a conditioned response. It was found that this alpha blocking response could be elicited by various types of previously neutral stimuli through repeated pairing, and that many features of classical Pavlovian learning could be demonstrated (Loomis et al., 1936; Jasper and Shagass, 1941; Knott and Henry, 1941; Shagass, 1942; Albino and Burnand, 1964). The alterations of EEG activity obtained through these procedures, however, quickly extinguished. Moreover, they were concerned only with the suppression of alpha rhythm. Results of much wider interest were presented by Kamiya (1962; 1967; 1968; 1969) who claimed that if individuals were given precise feedback information about the occurrence of alpha rhythm in their EEG, then they were able to learn to enhance and suppress its abundance.

Sensory feedback of the EEG has also an early history since it was utilized in the original studies of Adrian and Matthews (1934a) that verified the existence of the EEG. These workers converted the alpha

rhythm into an auditory signal in order to investigate the effect of opening the eyes in the dark, and to correlate subjective impressions of visual attention with the presence or absence of the rhythm. Several other pioneer workers similarly attempted to delimit the subjective states associated with alpha activity¹. Feedback loops were also employed in a number of investigations concerning the effects of various stimuli on the alpha rhythm (Walter and Walter, 1949; Mulholland and Runnals, 1962; Mulholland and Evans, 1966).

Kamiya's technique was to convert the alpha rhythm into a tone signal and then to simply ask his subjects to attempt to keep the tone on, or to attempt to keep it off. The exact experimental procedures and results of the Kamiya studies are rather poorly documented. He does report, however, that in one study a group of ten subjects demonstrated a significant difference in their abundance of alpha activity between a twenty-minute period in which they attempted to enhance the occurrence of alpha, and a twenty-minute period in which they attempted to suppress it (Kamiya, 1968). Furthermore, Kamiya (1968) noted that, "80-90% of some one hundred subjects investigated were able to successfully develop some control over the appearance of their alpha rhythm after feedback training"; and, on the basis of his results, concluded that, "People can be taught conscious control of their brain activity in a relatively short time".

There have been many subsequent accounts of the selective modification of EEG alpha rhythm through the use of feedback training (Dewan, 1966; 1967; Hart, 1967; Mulholland, 1968; Peper, 1970; Peper and Mulholland, 1970; Beatty, 1971; Brown, 1971; Hord and Barber, 1971; Nowlis and Kamiya, 1971; Peper, 1971a; 1971b; Beatty and Kornfeld, 1972; O'Mallery and Connors, 1972; Utz and Bankikiotes, 1973; Travis et al., 1974a; 1974b; 1975a; 1975b; Kondo et al., 1975; Kuhlman and Klieger, 1975; Fehmi and Selzer, 1976;

¹Bates, J.H.V., *personal communication* (1976).

Hardt and Kamiya, 1976b; Plotkin, 1976a; 1976b; Prewett and Adams, 1976). It has also been demonstrated that subjects who receive contingent feedback information exhibit significantly greater changes in their alpha activity during training than do subjects who receive noncontingent, or no, feedback information (Beatty, 1971; Travis et al., 1974a; 1974b; Kondo et al., 1975; Kuhlman and Klieger, 1975; Plotkin, 1976a). However, the data, in general, indicate that the claims made for the effects of feedback training by Kamiya were somewhat overstated. Moreover, other workers have failed to obtain any evidence for the enhancement of alpha activity by feedback training, or have found that feedback and control groups exhibited similar learning curves (Paskewitz et al., 1970; Cleeland et al., 1971; Grusche et al., 1973; Strayer et al., 1973; Podlesny and Raskin, 1974). It has also been suggested that although subjects can learn to suppress alpha activity, they cannot enhance it above normal baseline levels (Lynch and Paskewitz, 1971; Paskewitz and Orne, 1973; Walsh, 1974; Orne and Wilson, 1976; Johnson, 1976). Much of the conflict of data may be attributed to methodological differences between the various studies. The manner in which feedback is presented, the techniques employed to quantify the data, and the conditions under which the training is given, have all varied considerably (Travis et al., 1974b). Furthermore, as will be demonstrated in this thesis, there is a strong possibility that some of the results obtained were spurious, since the primary data were derived by quite complex apparatus and the relationship of the readings taken from the apparatus to the object of enquiry is not always immediate.

At the same time as the findings on EEG feedback training appeared, it was reported that many visceral and glandular responses could be brought under experimental control by similar techniques.

In a number of rodent studies remarkably specific and independent changes in a wide variety of autonomic functions, including heart rate,

blood pressure, peripheral blood flow, intestinal motility, and rate of urine formation, were obtained using feedback in the form of conventional operant procedures, and a curarized preparation (DiCara and Miller, 1968a, 1968b; 1968c; Miller and Banuazizi, 1968; Miller and DiCara, 1968; Miller, 1969; DiCara, 1970; Slaughter et al., 1970). Some of the impressive results claimed in these investigations have since proved to be very difficult to replicate (Miller and Dworkin, 1974). Nevertheless, the traditional belief that autonomic responses are not open to influence by reinforcement in the same manner as skeletal responses (Skinner, 1938; Mowrer, 1947) is no longer accepted to be tenable (Brener, 1974). The training of animal EEG activity by conventional operant conditioning techniques has also been extensively documented (Olds, 1965; 1967; Wyrwicka and Serman, 1968; Dalton, 1969; Fetz, 1969; Miller, 1969; Black et al., 1970; Black, 1971; Glazer, 1974).

With human subjects the control of many other physiological functions that, in addition to the EEG, are usually assumed to be involuntary, has been reported. For example, it has been claimed that individuals can learn to selectively modify their heart rate (Engel and Melmon, 1968; Weiss and Engel, 1971; Stephens et al., 1972), blood pressure (Benson et al., 1971; Schwartz, 1972; Shapiro et al., 1972), vasodilation (Snyder and Nobel, 1968), galvanic skin responses (Fowler and Kimmel, 1962; Quay and Kubiak, 1974), and stomach acid secretion (Welgan, 1974). Human subjects have also been able to influence their electromyographic (EMG) activity (Green et al., 1969; Jacobs and Fenton, 1969), including very precise and specific control of the discharge rates of individual motor units (Harrison and Mortensen, 1962; Basmajian et al., 1965). It should be noted, however, that the effects obtained in many of these studies are quite small and that control procedures were not always carried out.

These somewhat different lines of enquiry came to be associated, and this led to the concept of a completely new research field concerned with the regulation of bodily processes. The name of biofeedback was coined in 1969 for this field; a term which has since come to be applied generally to any techniques that utilize external stimulus signals to provide information about internal states that are to be brought under experimental control (Stoyva, 1976).

The principle of biological feedback is in itself not new, it is found at all levels of ontogenetic and phylogenetic organisation from intracellular processes to social communication patterns. Moreover, there are many historical antecedents of the concept of learned physiological control. Eastern mystics have long claimed to be able to influence their bodily processes through the practice of certain physical, mental, and spiritual disciplines. These claims, however, have generally been regarded with scepticism in the West, although some of them have recently been substantiated (Anand et al., 1961; Wenger and Bagchi, 1961). There are also several instances of early workers who investigated voluntary control techniques (Jacobson, 1938; Schuitz and Luthe, 1959). Probably the earliest example is that of Bair (1901) who employed a system of mechanical amplification, combined with visual feedback presented by a smoked drum, to successfully teach subjects the ability to wiggle their ears. However, according to Stoyva (1976), the origin of biofeedback as a distinct discipline stems from the beginning of this decade when there was an explicit recognition that, "A wide-ranging new principle of learning was, in fact, involved".

The possibility of learning to bring physiological functions, and in particular the activity of the brain, under direct voluntary control elicited a wave of enthusiasm and naive speculation in both the popular and scientific press (Maslow, 1969; Brown, 1974; Karlins and Andrews, 1975).

The reports of EEG feedback training appeared concurrently with a renewal of interest in Eastern religions and hallucinogenic drugs. Consequently, alpha feedback was seen by some authors to offer an alternative, 'scientific', technique by which to expand conscious experience and achieve self-attainment (Maslow, 1969; Nideffer, 1973; Brown, 1974; Karlins and Andrews, 1975). Others proposed that EEG training could provide mentalistic and introspective traditions with a new objective tool with which to identify and explore the phenomenological states associated with particular EEG patterns (Kamiya, 1969; Stoyva and Kamiya, 1968; Brown, 1970; 1971; 1974; Peper, 1972).

There have also been a profusion of studies on the potential clinical applications of biofeedback, with the result that it has been hailed as a panacea for a whole gamut of disease states (Miller, 1969; Shapiro and Schwartz, 1972; Blanchard and Young, 1974). A particular claim with which we will be concerned in this thesis is that feedback training of the EEG is a powerful new therapeutic tool for the treatment of epilepsy.

1:1:2 Feedback Electroencephalography in the Treatment of Epilepsy

Epilepsy is a manifestation of a "Periodic and excessive discharge of cerebral neurons, which may result in a loss of consciousness, involuntary movements, abnormal sensory phenomena, increased autonomic activity, and a variety of psychic disturbances" (Gilroy and Meyer, 1975). There is not, however, a single disease state of epilepsy, but a group of related seizure disorders - the epilepsies - which have different symptoms and etiologies (Niedermeyer, 1974).

The nosology of the epilepsies is still a matter of considerable debate. Two main groups, however, have been distinguished¹.

¹*Detailed proposals for a classification of epileptic disorders are provided by Gastaut (1970) and Merlis (1970).*

The first consists of primary generalised seizures, with bilaterally symmetrical clinical and EEG disturbances and the absence of a localised onset (Sutherland et al., 1974). The clinical symptoms range from transient lapses of consciousness in petit mal seizures, through impairment of consciousness with varying involvement of clonic movements, to deep coma with tonic-clonic motor events in grand mal seizures. The EEG shows diffuse, high voltage, synchronous and symmetrical spike discharges and slow wave activity. These seizures are currently believed to originate in the brain stem. They may also be associated with a genetically transmitted low seizure threshold (Niedermeyer, 1974).

The second group of seizures includes all those that have a functionally localised etiology and onset. These are referred to as partial seizures. Focal high voltage spikes and slow waves may be evident in the EEG; the motor, sensory, or psychic events that are seen clinically depend on the anatomical origin of the epileptogenic discharge and the manner in which it is propagated (Penfield and Jasper, 1954). All forms of partial seizures can result in a generalized seizure secondary to the initial ictal event. These seizures are usually related to a variety of localised brain lesions; constitutional factors may be again involved, however.

There have been a number of attempts made to inhibit seizure activity by the use of various behavioural conditioning techniques.

Efron (1956; 1957) reported the case of one patient whose uncinat seizures could be inhibited by a powerful olfactory stimulus. This effect was transferred to a previously neutral stimulus, a silver bracelet, by classical conditioning procedures. It is reported that eventually merely thinking of the bracelet was sufficient to arrest the development of a seizure and the patient became almost seizure free.

Stevens (1961) employed a wide range of sensory stimuli in an effort

to desynchronize focal and generalised seizure activity in sixty-six patients. However, she found that there was a tendency for the epileptic activity to be augmented after stimulation. Tassinari (1968) similarly met with only limited success in the suppression of focal discharges by somaesthetic stimuli. Stevens (1960; 1962; Stevens et al., 1967) also sought to effect a suppression of epileptic activity through the development of a conditioned generalization or avoidance response, using epileptiform EEG discharges as an endogenous stimulus; but she generally failed to obtain much clinical improvement.

In contrast, Ounsted et al. (1966) report the successful treatment of one intractable petit mal patient by the desynchronization of epileptiform discharges in the EEG with photic stimulation. Moreover, although the conditioning procedure was given for a total of only sixteen days, it is reported that the effects generally continued over a follow-up period of seven years.

Forster and colleagues (Forster and Campos, 1964; Booker et al., 1965; Forster, 1966; 1969; Forster et al., 1965, 1969) developed a technique of progressive desensitization for the treatment of patients whose seizures were precipitated by specific sensory stimuli. Sub-threshold stimuli were repeatedly presented until habituation of the seizure response was obtained. This technique of conditioned inhibition proved to be effective in photosensitive, musicogenic, pattern, voice, and startle reflex epilepsies. However, it was found that extinction of the inhibition rapidly occurred unless reinforcement of the conditioned response was maintained. One solution that was devised for the treatment of photosensitive epilepsy was to pair the conditioned inhibition to an auditory stimulus. Special eyeglasses were then constructed which generated this auditory stimulus whenever flickering light was encountered in the environment. Forster et al. (1965) report that the precipitation of

seizure activity was greatly diminished in six patients with this method.

Naquet (1961) also investigated the experimental modification of the precipitation of seizures in photosensitive patients through the use of photic stimulation, but concluded that it would be difficult to apply these techniques clinically.

These early studies on the treatment of epilepsy by conditioning all focused on the suppression of epileptiform activity. With the advent of biofeedback, however, came a totally different approach: that of training patients to produce patterns in their EEG that are naturally inimical to seizure activity.

This work was pioneered by Sterman and colleagues in Los Angeles. Sterman (1974) relates that it came about through a process of serendipity during investigations of a 12-16 Hz EEG pattern in the cat, which was given the name of sensorimotor rhythm (SMR) since it is seen over the feline sensorimotor cortex during states of motionlessness (Roth et al. (1967)). It was demonstrated that the animals could be trained to enhance the abundance of the SMR activity in their EEG through traditional operant methods. Furthermore, Sterman (1974; 1976c; 1977b) reports that what was particularly seminal about this work was the discovery that the SMR training apparently resulted in an increased resistance to drug-induced seizure activity. The SMR trained animals were given intraperitoneal injections of monomethylhydrazine in convulsant doses as part of a separate study. It was found that the onset of seizure activity was significantly delayed, in comparison with a group of cats who did not receive SMR training, even though other symptoms of toxicity were displayed (Sterman et al. 1969). This finding was replicated in a subsequent study involving thirty animals (Sterman 1976a); however, the effects of other convulsant agents on SMR trained animals have not yet been reported.

These results led Sterman and colleagues (Sterman 1973a; 1973b; 1974; Sterman et al., 1974) to conclude that SMR training results in a raised seizure threshold through the facilitation of a central motor inhibitory system. Hence the possibility of using SMR feedback training to treat epileptic patients was explored.

The first patient investigated had an eight-year history of drug-refractory tonic-clonic seizures. She was provided with feedback of 12 Hz EEG activity recorded from over the rolandic region for three thirty-minute training sessions per week. Sterman and Friar (1972) report that within three months an abrupt reduction in the frequency of clinical seizures was obtained. Over the subsequent two years the incidence of seizures was diminished from some two per month to one in six months (Sterman, 1974).

Three more patients, with tonic-clonic, akinetic, and petit mal seizure types, were then added to the study. After periods of SMR training ranging from eight to eighteen months Sterman et al. (1974) report that for all patients, "There was a significant reduction of EEG and clinical epileptic manifestation as indicated by clinical EEG recordings, EEG spectral analysis and seizure logs". Sterman (1973a) further claims that "The lowest rates of clinical seizure activity in the history of their respective disorders were uniformly achieved within several months of the initiation of training".

These impressive results following feedback training of SMR were corroborated by several other independent workers.

Finley and colleagues (Finley, 1974; Finley et al., 1975) provided one adolescent epileptic with thrice weekly feedback training sessions of rolandic 12 Hz EEG. They report that, at the end of seven months of treatment, the frequency of the patient's atonic seizures was reduced from approximately eight per hour to one per hour.

Lubar and colleagues (Lubar and Seifert, 1975; Seifert and Lubar,

1975; Lubar and Bahler, 1976) investigated the effects of SMR feedback in six patients who presented a cross-section of epilepsies, including tonic-clonic, myoclonic, akinetic, focal, and psychomotor types. After enhancement training of rolandic 12-14 Hz activity for three sessions per week for some three months, five of the patients are reported to have demonstrated a clinical improvement (Lubar and Seifert, 1975; Seifert and Lubar, 1975). Two more patients were added to the group, and with further training for periods of up to nine months, some of the patients are said to have become completely free from seizures (Lubar and Bahler, 1976).

Not surprisingly, these findings have aroused a considerable amount of attention. It has been suggested that feedback training of SMR offers the hope for severely epileptic patients that their seizures might be controlled when anticonvulsant medication is not sufficient; and that with patients whose seizures are adequately controlled by medication, the drug therapy might be reduced or even eliminated (Seifert and Lubar, 1975).

Unfortunately, such optimism is premature since the work on SMR training in the treatment of epilepsy is open to several serious methodological criticisms. Firstly, in all of the studies the therapeutic effects have been directly attributed to the enhancement of SMR. However, as will be apparent in this thesis, the evidence for a relationship between the clinical changes and the acquisition of the SMR response, or even for the existence of an SMR in man, is extremely slender. Secondly, the techniques used for the detection and feedback of SMR activity in the EEG have typically failed to take into account the complex nature of the EEG signal or the characteristics of frequency selective circuits. Finally, there have been very few attempts in any of these studies to control for placebo, or other effects not specific to SMR training, that might be responsible for the clinical improvements.

OBJECTIVES OF THIS RESEARCH

This project was set up in order to investigate the validity of the EEG feedback training phenomenon, and the claims for its successful application to the treatment of epilepsy. The objectives of this research were:

- (1) To examine the evidence for a selective modification of the EEG alpha rhythm through the provision of sensory feedback;
- (2) to study the methodology of EEG feedback training, and, in particular, the relationship between the techniques that are employed to process the EEG signal and the results that are obtained;
- (3) to replicate the reports of SMR training in man; and
- (4) to determine whether or not SMR training does offer a unique clinical tool for the treatment of epilepsy.

Because there is a strong tendency for oversimplified models to be used to account for EEG phenomena, the next chapter is concerned with a discussion of the neurophysiological basis of EEG potentials and the manner in which these waveforms become synchronized to form distinct rhythmic patterns, in particular, the alpha rhythm and SMR. The possible physiological significance or function of these rhythms is also considered.

In Chapter Three the setting up of the initial system in the laboratory for investigating feedback training of the alpha rhythm is described, together with the early studies that were carried out on alpha feedback training.

The modification of the equipment in order to detect SMR activity, and the investigation of SMR training in normal subjects is reported in Chapter Four.

One of the greatest difficulties that all investigators of SMR training have faced is that of designing equipment to accurately register the presence of SMR in the EEG, particularly in that of the epileptic. In

Chapter Five the development of a SMR detection and feedback system for the clinical study, based on the experience gained in the laboratory studies, is discussed. A brief comparison of the systems used by other workers is provided as an Appendix.

The clinical study, which involved a comparison of the effects of SMR training and several control procedures using three epileptic patients, is reported in Chapter Six. The overall results of this study, and a physiological model to account for the effects observed, are discussed in Chapter Seven.

Finally, the general conclusions from this research concerning a number of methodological and theoretical issues are summarized in Chapter Eight, and some proposals for further work are outlined.

In recent years there have been a number of reports that individuals can be taught to modify their EEG spectra through the provision of sensory feedback. The validity of these claims, however, is a matter of considerable controversy. Discrepancies in the data from different laboratories partly arise as a result of differences in the techniques used, and the conditions under which training was given. This research was therefore initiated in order to investigate EEG training in relation to such factors.

The reports of EEG training became encompassed within a broad concept of learned voluntary control over internal states, which was given the name of biofeedback. Biofeedback has been proposed as a therapy for a wide range of clinical problems. Of particular relevance to this work are claims that EEG feedback training is an effective technique for the treatment of drug-resistant epilepsy. Earlier attempts to apply EEG conditioning techniques to suppress epileptic seizures met with only limited success. The feedback training approach differs from these in that it focuses on the trained enhancement of a 12-16 Hz pattern in the EEG, known as SMR. This pattern is hypothesised to be refractory to seizure activity on the basis of an animal model. However, there have been very few attempts to verify that the therapeutic effects are, in fact, associated with the SMR feedback training procedure. A clinical study was therefore carried out in order to investigate the evidence for these claims.

A brief outline of the scope of this presentation is given.

2:1 ELECTROGENESIS OF THE EEG

2:1:1 Neuronal Generators of Surface Slow Waves

The existence of fluctuating potentials recorded from the brain was first demonstrated by Caton (1875). Beck (1890) confirmed this and concluded that these potentials reflect active regions within the cortex. By recording from needle electrodes in trephined human subjects, Berger (1931) observed that the brain potentials were more prominent on the cortex than within the underlying white matter, indicating that the generator of EEG waves must reside within the cortex itself. Many investigators have subsequently established a cortical origin for surface slow waves (Andersen and Andersson, 1968; Elul, 1968; Creutzfeldt and Houchin, 1974).

It was initially thought that the gross EEG represented the temporal summation and complex phase interaction of cortical action potentials (Adrian and Matthews, 1934b; Bishop, 1936). However, this view was demonstrated to be erroneous when Li and Jasper (1953) showed that the EEG persisted after the abolition of action potentials by anoxia. An alternative hypothesis, which has continued to gain support, is that the EEG is an expression of innumerable fluctuations of the graded membrane potentials on the somata and dendrites of cortical neurons (Bremer, 1949; Eccles, 1951; Kiloh et al., 1972). These fluctuations can be due to synaptic excitation or inhibition, or may be engendered by pathological membrane changes. There is also some evidence of quite substantial spontaneous oscillations of membrane potentials (Andersen and Andersson, 1968).

With the introduction of intracellular micro-electrode recording techniques, it was qualitatively demonstrated that the time course of

transient changes of the resting potential of cortical neurons was of the same order of magnitude as that of the surface slow waves of the EEG (Elul, 1962; Purpura and Cohen, 1962; Klee and Offenloch, 1964; Creutzfeldt et al., 1966a; Frost et al., 1966). For example, Creutzfeldt and Houchin (1974) cite the time constant of excitatory post-synaptic potentials (EPSPs) as 10-30 msec and of inhibitory post-synaptic potentials (IPSPs) as 70-150 msec for cortical cells.

Although we have much evidence in favour of cortical post-synaptic potentials being a main source of the EEG, a causal link between surface slow waves and neuronal membrane potentials is difficult to conclusively establish. Some workers have reported close relationships between the intracellular or extracellular potentials of single units (individual neurons) in the cortex and surface EEG waves (Fromm and Bond, 1964; Jasper and Stephanis, 1965; Creutzfeldt et al., 1966a; 1966b; Frost and Gol, 1966). However, Elul (1972) has pointed out that such correlations between gross and cellular activity are ephemeral and dependent on factors such as the state of arousal and depth of anaesthesia. Clear data relating gross surface activity to cellular activity and microanatomy are only available for a few types of post-synaptic potentials.

Various laboratories conflict over the types of surface positive or surface negative waves that they report to be associated with excitatory or inhibitory cellular activity. Creutzfeldt and colleagues have proposed a tentative model to account for the generation of these different types of EEG waves based on their analysis of the cross- and auto-correlations between various features of the surface EEG and neuronal events within the motor cortex of anaesthetised cats (Creutzfeldt et al., 1966a; 1966b). Creutzfeldt suggests that slow membrane transients are in phase with surface potentials; whereas fast membrane transients may be phase shifted or reversed. Thus surface positive slow waves could result from cortical

superficial inhibition or deep excitation, and surface negative slow waves from superficial excitation or deep inhibition (Creutzfeldt and Houchin, 1974).

Creutzfeldt's model, however, is based on the hypothesis that the EEG results from electrotonic conduction of the activity in parallel columns of pyramidal cells arranged as perpendicular dipoles (Calvet et al., 1964; Creutzfeldt et al., 1966a). The concept of an electrical dipole is used to represent extracellular current flow between sources and sinks in the upper and lower layers of the cortex. This concept has also been used in many theoretical attempts to relate surface potentials of the brain to an equivalent generator inside an irregular volume conductor (Brody, 1968). Regan (1972) has commented on the inadequacy of the dipole as a mathematical model; and there is some evidence now that the generator elements of the EEG also do not resemble simple cortical dipoles. A dipole must have a point where the potentials become reversed in phase. Some types of cortical waves show a clear phase reversal within the cortex; others have homogenous potentials widely distributed throughout the depth of the cortex, and a mixture of both occurs during epileptic activity (Creutzfeldt and Houchin, 1974). Laminar analysis of cortical potentials has established that the hypothetical concept of vertical layers of cellular dipoles is insufficient to account for all the phase shifts and reversals observed (Petsche and Rappelsberger, 1969).

Spatio-temporal contour maps of the distribution of the potential fields corresponding to various surface slow waves can be constructed by stratigraphic analysis of intra-laminar activity, using two-dimensional arrays of microelectrodes. This method has revealed the existence of a great number of small generators of different levels, positions, strengths and mutual interaction (Petsche and Rappelsberger, 1969; Petsche, 1975). These investigations were carried out on the rabbit cortex, which has a

similar histological structure to the human cortex. Although they mostly involved experimentally induced epileptiform activity, Petsche (1975, personal communication) has stated that a similar phenomenon can be observed for spontaneous activity.

Offenloch et al. (1973) computed the power spectra of the spontaneous electrical activity recorded at the surface and in various laminar layers of the sensorimotor cortex of the cat. They found that although the frequency content of the laminar spectra was practically identical to that of the surface electrocorticogram, the intercorrelations between spectra were extremely low (Offenloch, 1975a). This implies a general oscillatory property of independent elements throughout the depth of the cortex. During strong synchronous input, however, the coherence between laminar spectra grows (Offenloch, 1975b).

Stratigraphic analysis and cortical incision experiments, nevertheless, suggest that the functional organisation of the EEG generators is a vertical one, with perhaps small column-like generator zones of different lengths and positions situated one upon another at different levels of the cortex (Petsche and Rappelsberger, 1969). This model is illustrated in Figure 2.1. These morphological elements of synchrony differ from dipoles by virtue of their unstationariness: thus the pattern of electrical activity is determined by the rising, changing and moving of potential fields within the cortex.

It has been reported that a pair of microelectrodes in the cortex show independent, and not synchronous, activity between them when the microelectrode tips are as close together as 100 μm (Petsche and Rappelsberger, 1969), or even 30 μm (Elul, 1968). This indicates that the fundamental generator elements must be close in size to individual neurons in the horizontal plane. Elul (1972) has suggested that the unitary generators may be relatively small patches of cellular membrane, including

several synaptic groups, which share the same synaptic input ("synaptic functional units"), rather than complete neurons, since it is known that different regions of the soma are not equipotential during spontaneous wave activity. From a study of the depth distribution and anatomy of various types of surface potentials, using the trans-cortical bipolar electrode recording technique, Calvet et al. (1964) have suggested that the actual form of the surface waves will depend upon the location of the synaptic action on the neurons concerned.

Intracellular waveforms bear only a remote similarity to the patterns of the EEG. Therefore apart from the question of synchronization, which is

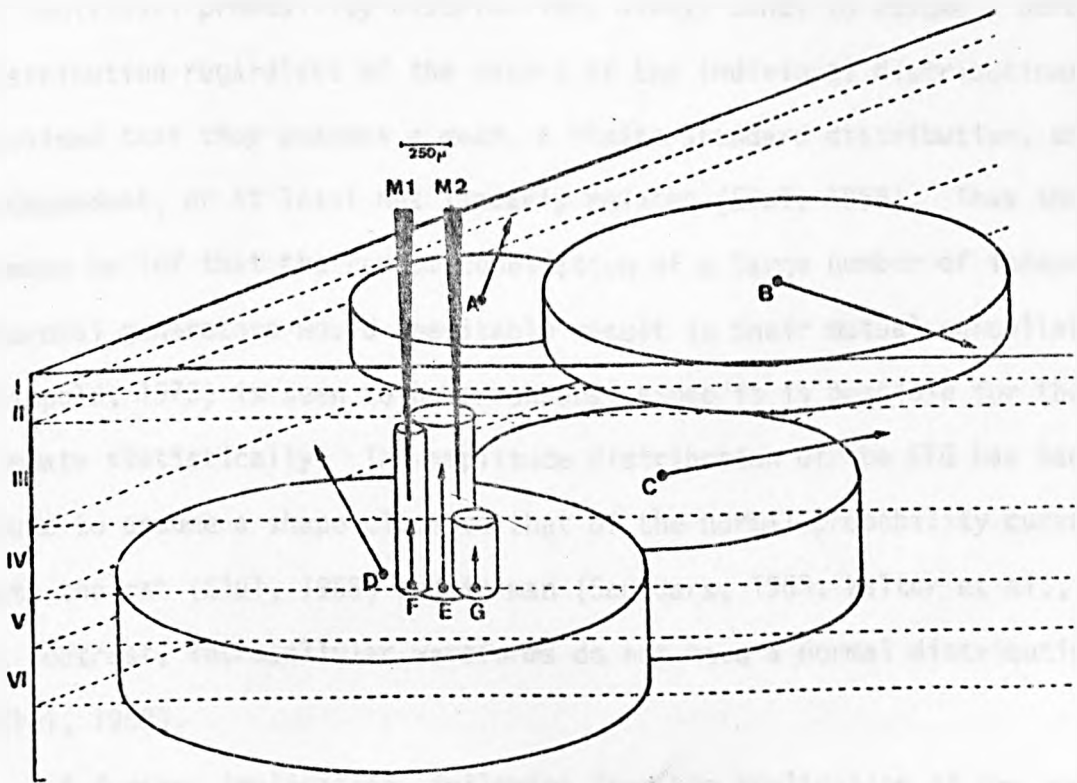


Figure 2.1

Diagrammatic representation of dynamic functional units of synchronized activity in the laminae of the cortex. These generator zones of the EEG are symbolized by the cylinders A - G. At the surface (A,B), and in the deep layers (C,D), the generator zones are extensive and spread horizontally in various directions as depicted by the arrows. In the region of cellular apical dendritic bundles, the generator zones are narrow vertical columns (E,F,G) and their spread is mainly corticopetal. M1 and M2 indicate microelectrodes positioned 250 μm apart.

After Petsche (1975).

discussed below, we need to specify a mechanism whereby the individual generators are compounded to form the gross EEG.

It is usually assumed that the EEG simply represents the linear summation of synchronous neuronal activity (Creutzfeldt et al., 1966a). However, the long-term coherence between gross and neuronal waves is insufficient to support this view (Elul, 1972 *supra*). An alternative proposition, advanced by Elul, is that the EEG may be accounted for as the normally distributed output ensuing from combination of the activity of many independent or non-linearly related neuronal generators according to the *central limit theorem*. This theorem states that the sum of a large number of individual probability distributions always tends to assume a normal distribution regardless of the nature of the individual distributions, provided that they possess a mean, a finite standard distribution, and are independent, or at least not linearly related (Elul, 1968). Thus the very common belief that the random combination of a large number of independent neuronal generators would inevitably result in their mutual cancellation (Lippold, 1973) is seen to be erroneous, since it is possible for them to summate statistically. The amplitude distribution of the EEG has been found to assume a shape close to that of the normal probability curve in both the cat (Elul, 1968) and in man (Saunders, 1963; Walter et al., 1966). In contrast, intracellular waveforms do not have a normal distribution (Elul, 1968).

A further implication, following from the application of the *central limit theorem* to the electrogenesis of the EEG, is that the change from the complete independence of all neuronal generators to their total synchronization should result in an enormous increase in amplitude of the gross EEG. This is because amplitude in the first instance is a function of the square root of the number of generators; and in the second, a function of the total number of generators. Elul argues from this that the

concept of EEG synchronization becomes redundant, since this could easily result from the interaction of only a small part of the total neuronal population (Elul, 1968). Instead, he sees the surface recorded EEG as the intermittent and constant flux of synchrony involving just relatively small groups of neurons (Elul, 1972). This model assumes, however, a constant non-linear relationship between the neuronal generators; whereas Offenloch (1975b) found that coherence values between different cortical sites increased when strong synchronizing input was applied, and coherence is normally interpreted to indicate a linear relationship. Similarly, large cortical coherences at distances up to several millimetres have been observed in dogs when the alpha rhythm is present (Lopes da Silva, 1973a). Moreover, the EEG amplitude probability distribution departs from Gaussian during slow wave sleep (Weiss, 1973) and during hypersynchrony (Petsche and Rappelsberger, 1969). This suggests that during EEG synchronization there is a transient linear summation of cellular generators in addition to their statistical summation.

In conclusion, it seems that the answer to the question "What is the EEG?" is that the EEG represents a complex spatial and temporal average of the extracellular potential fields of spontaneous and evoked membrane changes on a large number of individual cortical neurons or groups of neurons. It follows that a definitive close correspondence between on-going EEG activity and single unit activity will be very difficult to establish.

The appearance of sub-cortical potentials in the overall EEG is unlikely to be significant, but possibly volleys of action potentials do contribute as, for example, in the genesis of epileptiform spikes. The influence of glial cells on the EEG is obscure, but it is thought that they may add to the standing potential and slow DC shifts of the brain, and perhaps also have some significant effects on the final waveforms of the EEG (Regan, 1972).

2:1:2 Scalp Recorded EEG

Potentials recorded from the scalp are much smaller than those recorded on the surface of the cortex (De Lucchi et al., 1962; Cooper et al., 1965; Pfurtscheller and Cooper, 1975). In humans the attenuation can be as great as 35000:1 (Cooper et al., 1965). Furthermore, the recorded waveforms of scalp activity can be considerably different from those of the underlying cortex (Cobb and Sears, 1960). The scalp and intervening tissues have complex electrical characteristics; and the low resistance cerebrospinal fluid, in particular, has the effect of "smearing", or averaging, the cortical activity in addition to attenuating it. De Lucchi et al. (1962) demonstrated that there was little in common between activity recorded from a needle electrode placed in the scalp of a cat and from a single electrode lying on the cortex directly underneath it. When the scalp record was compared to the average of six interconnected cortical electrodes, however, similarity between the recordings was increased. The results from hemispherectomy studies have also suggested that the cerebrospinal fluid, skull and scalp can play a very substantial part in the spread of cerebral potentials fields (Cobb and Sears, 1960).

Synchrony in the cortex can be highly localised, with differences between waveforms recorded from electrodes as little as 1 mm apart (De Lucchi et al., 1962; Andersen and Andersson, 1968); although, at the same time, substantial coherences can be found for distances up to several centimetres (Lopes da Silva et al., 1973a). This situation stems from the scattered zones of synchrony associated with different thalamo-cortical functional units (see 2:2:1). During transmission to the scalp surface this localised activity tends to be averaged out.

High amplitude fast activity (above 15 Hz) is much more prominent on the cortex than on the scalp. This is not merely the result of a low pass filter effect since, as Pfurtscheller and Cooper (1975) found, all

frequencies are similarly attenuated. These workers suggest that the characteristic inverse relationship between EEG amplitude and frequency may instead be partly due to temporal delays and complex phase interactions occurring during transmission to the scalp.

2:2:1 Thalamic Control of Cortical Synchrony

Patterns of synchronized cortical activity are strongly determined by subcortical afferent input. In several species it has been demonstrated that undercutting of the cortex markedly interferes with spontaneous or evoked rhythmical activity (Bremer, 1949; Jasper, 1949; Echlin et al., 1952; Andersen et al., 1967a). There is much evidence that the essential subcortical structure responsible for EEG rhythms is the thalamus.

Persistent rhythmic activity can still be recorded from the thalamus and from exposed white matter after decortication (Adrian, 1941; Andersen and Andersson, 1968; Andersson and Manson, 1971). Conversely, thalamectomy abolishes normal cortical synchrony (Jasper, 1949; Kristiansen and Courtois, 1949; Andersen et al., 1967a). The flattening of the EEG after injection of tetrodotoxin into the ventricles (Elul, 1972) and the reduction in frequency of cortical barbiturate spindles produced by localised cooling of the thalamus (Andersen et al., 1967a; Andersen and Andersson, 1968), or by localised thalamic injection of penicillin (Ralston and Ajmone-Marsan, 1956) also serve to illustrate the thalamic control of cortical rhythmical activity.

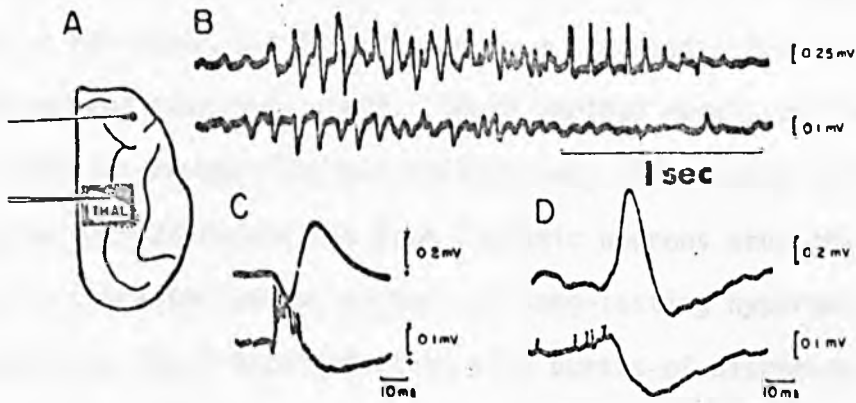
A number of workers have found very close correspondence between rhythmical activity in the thalamus and in the appropriate thalamic projection area of the cortex (Andersen et al., 1967b; Howe and Sterman, 1972; Bouyer et al., 1974; Ganes and Andersen, 1975; Ganes, 1975). Andersen and Andersson (1968) proposed that the rhythmicity is topographically imposed on the cortex via the thalamo-cortical specific projection system. Lopes da Silva et al. (1973a) criticized this hypothesis on the grounds that they were unable to find sufficiently large thalamo-cortical coherences to support the concept of the thalamus as

a primary pacemaker of cortical rhythms. However, very strong thalamo-cortical coherences, with point-to-point wave correspondence, can be observed if suitable precautions are taken to ensure that the computations are based on a comparison between a thalamic relay nucleus and its exact cortical projection area. Andersen and Ganes, (1975) delimited functionally corresponding zones in the nucleus ventralis posterolateralis (VPL) of the thalamus and its primary projection area, the somatosensory cortex, by means of automatic statistical analysis of responses evoked by contralateral cutaneous stimulation. Barbiturate spindles simultaneously recorded from these thalamic and cortical zones were highly cross-correlated; whereas a small displacement of the cortical electrode reduced the thalamo-cortical wave synchrony. This experiment is illustrated in Figure 2.2. The data indicate that there is a strict topographical relation between synchronized activity in the thalamus and cortex, and that thalamic cell groups serve as pacemakers for the cortical rhythmic activity.

2:2:2 Determinants of Thalamic Rhythmical Activity

An early hypothesis for the basis of EEG synchrony was that this was due to the cyclic movement of activity between the cortex and the thalamus (Bishop, 1936). This was refuted by the effects of decortication (Adrian, 1941). Morison and co-workers introduced the concept that the medial nucleus of the thalamus acted as a pacemaker of the EEG, on the basis of their discovery that stimulation of this area could produce widespread rhythmical activity in the cortex (Dempsey and Morison, 1942). Jasper (1949) elaborated this theory to include an intrathalamic stage to mediate the spread of rhythmic activity. Subsequent investigation has revealed that the specific relay nuclei of the thalamus are also able to exhibit rhythmical activity. This led Andersen and Andersson (1968) to advance the facultative pacemaker theory which holds that all major thalamic

(a)



(b)

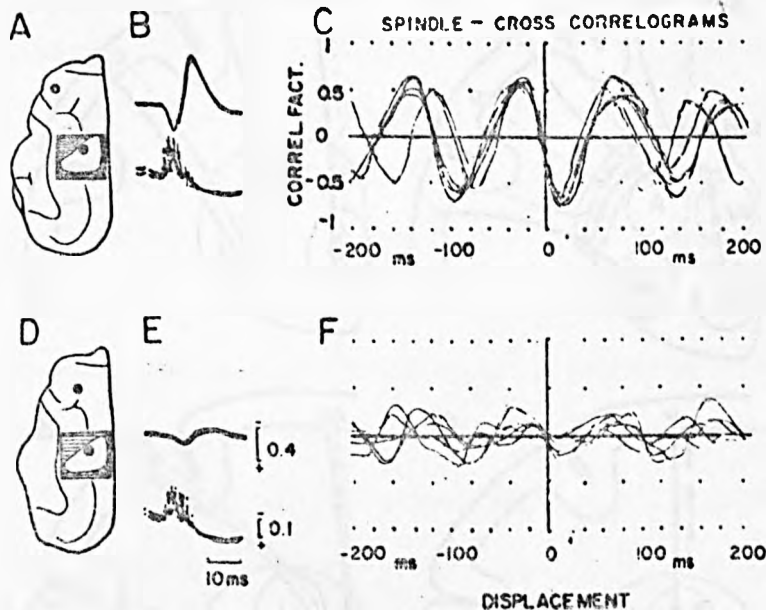


Figure 2.2

(a) Barbiturate spindle activity and sensory evoked potentials recorded from the thalamic VPL nucleus and the corresponding cortical projection area. A: Recording situation. B: Cortical (upper) and thalamic (lower) spindles. C: Short latency potentials in the cortex and thalamus evoked by contralateral forepaw stimulation. D: Excerpt of spontaneous spindles recorded from the same locations.

(b) Cross-correlograms of thalamic and cortical spindles recorded as functionally on-line and off-line. A: The thalamic and cortical electrodes are on-line as indicated at B by the size of the evoked potentials. C: Cross-correlograms of five thalamo-cortical spindles are shown superimposed. D: The cortical electrode was moved 3 mm medially. E: Amplitude of the evoked cortical response was small indicating that the electrodes are off-line. F: No consistent wave synchrony is present in the cross-correlograms.

After Ganes and Andersen (1975).

nuclei have the ability to produce rhythmic activity and control a corresponding part of the cortex; and that the locus of rhythmicity in the thalamus is not fixed, but is governed by a stochastic process according to the afferent and intrinsic input. These various models of the basis of thalamo-cortical rhythmicity are collectively illustrated in Figure 2.3.

Microelectrode recordings from thalamic neurons show that rhythmical activity is characterised by periods of long-lasting hyperpolarisation terminated by a short depolarisation with bursts of discharges. Most workers are agreed that the modulation of deep inhibition is the essential

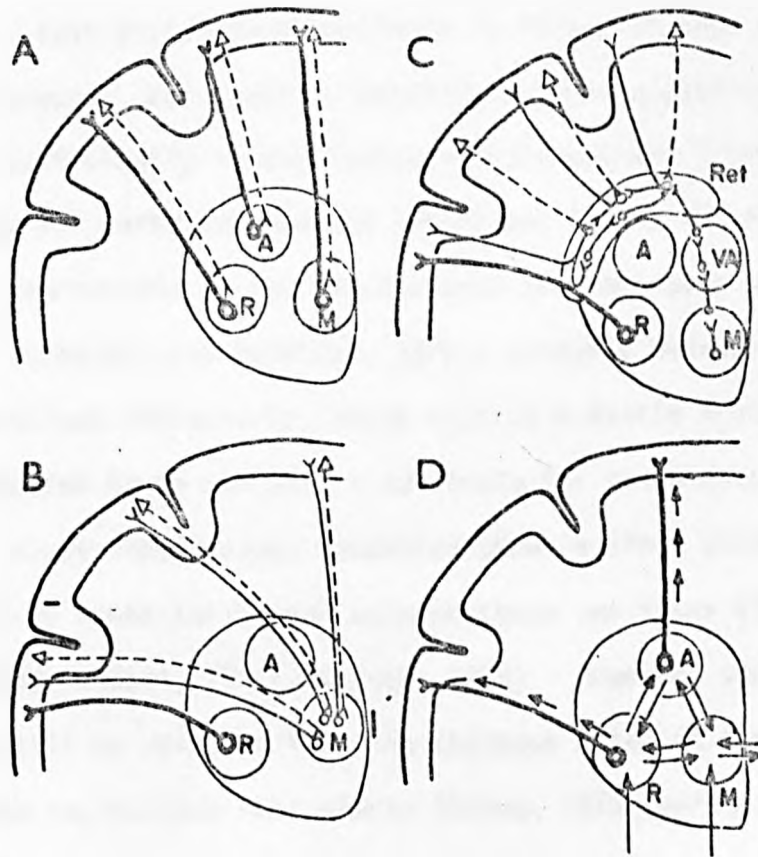


Figure 2.3

Schematic representation of various models of the origin of thalamo-cortical rhythmicity. A: Thalamo-cortical reverberating circuits. B: Medial thalamic pacemaker. C: Thalamic midline pacemaker and non-specific projection system. D: Facultative pacemakers in all major thalamic nuclei. Abbreviations: R, thalamic relay nucleus; A, thalamic association nucleus; M, medial thalamic nucleus; VA, anterior thalamic nucleus; Ret, thalamic reticular nucleus.

From Andersen and Andersson (1974).

mechanism underlying thalamic rhythmicity. But the means by which this inhibition is mediated, or the reason for the grouped discharges following inhibition, is still in dispute.

Andersen and Andersson (1968; 1974) suggest that a very large IPSP results from a recurrent axon collateral of a thalamic relay cell which activates an inhibitory interneuron. This interneuron then inhibits the relay cell and several other neighbouring cells. At the end of the IPSPs these cells all tend to discharge at the same time thus involving more inhibitory interneurons. The process of recurrent inhibition and burst discharge then repeats itself.

There is some histological evidence in favour of this theory, but it is still equivocal. Recurrent collaterals have been difficult to demonstrate sufficiently in the thalamus (Scheibel and Scheibel, 1970); although they are certainly present (Andersen, 1975), and thalamic inhibitory interneurons of the Renshaw type have not been universally established (Scheibel and Scheibel, 1970), possibly because of their small size. The nucleus reticularis, which sits as a mantle around the thalamus, has been proposed as an anatomical substrate for the widespread thalamic inhibition, since simultaneous recording shows a close association of bursting and of tonic inhibition between these two sites (Scheibel and Scheibel, 1970; Waszak, 1974; Massion, 1975). However, spontaneous spindle bursts can still be recorded from the thalamus after it has been surgically isolated from the nucleus reticularis (Ganes, 1975, personal communication) and it is very likely that some common mechanism is responsible for the close correspondence of activity. Recently, dendro-dendritic coupling has been discovered in several parts of the thalamus and this could well be involved in thalamic inhibitory phasing as an alternative to the hypothetical recurrent pathway (Andersen and Andersson, 1974).

Originally, Andersen and Andersson (1968) suggested that the

synchronous discharge of thalamic cells after inhibition resulted from an increased excitability, or post-anodal exaltation, of the thalamic neurons, which they assumed to be a consequence of changes in membrane conductance. This hypothesis of a post-inhibitory rebound has been criticised on the grounds that it has not been demonstrated in injury-free thalamic neurons (Purpura, 1970; Andersson and Manson, 1971). These latter workers favour instead the interaction of both excitatory and inhibitory interneurons for the thalamic synchronizing process. However, this must also remain speculative until the existence of the excitatory interneurons is experimentally verified.

Ganes, (1975, personal communication) believes that spontaneous changes in the membrane potential of the thalamic neurons may initiate the action potentials necessary for triggering a spindle. He found that, not only could rhythmic activity be recorded from a fully denervated thalamus, but also from small (2 mm) cubes of tissue which were fully isolated from the rest of the thalamic complex. These data support the view that thalamic cell groups have perhaps a unique autogenic ability for generating synchronized activity.

Andersson (1975) has proposed that the appearance of spindle burst activity in the thalamus is related to changes in the depolarising pressure on the projection cells. As the IPSP starts to diminish, impulse bombardment causes synaptic depolarisation which eventually brings about a discharge. The effect of afferent input upon the thalamic recurrent loop is thus to both excite the projection cell and to inhibit the inhibitory interneurons. The balance between these two effects controls the discharge pattern and frequency of the projection cell (Andersson et al., 1971a). In the absence of external excitatory drive (for example, after extensive lesions of the brain stem or complete thalamic isolation), Andersson et al. (1971a) suggest that excitatory interneurons have the inherent property of

discharging spontaneously. However, even if rhythmic activity can still occur in isolated thalamic tissue, it does not mean that it is normally independent of afferent input. Neither can we exclude the possibility that chronic experimental deafferentiation could result in a spread of cellular injury discharges within the closed-chain, self-exciting neuronal networks equivalent to the effects of external excitatory drive.

Lopes da Silva and colleagues demonstrated that when neuronal networks of the type postulated by Andersen and Andersson (1968), with a recurrent inhibitory circuit, are presented with a noise input they could generate rhythmic activity simply by a band-pass filter effect (Lopes da Silva et al., 1974). They set up a computer model based on histological data from the VPL nucleus. The model produced rhythmic oscillations in the mean membrane potentials of the population of simulated neurons which were very similar to those obtained empirically from the thalamus of the dog (Lopes da Silva et al., 1973a). When the influences of various parameters upon the rhythmic activity were examined mathematically, it was shown that a family of spectral curves could be obtained which simulated the development of the EEG as a function of age: that is, from a predominantly low frequency spectrum to one centred at alpha frequency. This was found mainly to depend on the feedback introduced into the system by the degree of interconnectivity. Alpha frequencies have been shown to correlate with age (Friedlander, 1958), and there is a structural and functional relationship between the maturation of the EEG and that of neuronal dendrites and synapses (Scheibel and Scheibel, 1964) and of interneurons (Creutzfeldt and Houchin, 1974). Thus the filter properties of the thalamic nuclei, which depend on the duration of IPSPs (in the order of 100 msec) and the extent of synaptic coupling, could account for the change in the dominant EEG rhythms seen during ontogenesis.

Since Lopes da Silva and colleagues do not accept the notion of the

thalamic control of cortical rhythms (*supra*), they went on to argue that cortical rhythms are produced independently by the same form of neural networks as in the thalamus. There is, however, no evidence for similar powerful recurrent inhibitory phasing in the cortex.

Andersen and Andersson (1974) suggest abandoning the term "pacemaker" in favour of a more neutral term such as "oscillators" to signify the stochastic nature of the thalamic rhythm generators. It might be simpler, however, to adopt the concept of frequency selective neuronal networks, since whether there is an inherent autorhythmicity unique to thalamic circuits (Andersen and Andersson, 1968), or whether they require to be continually stimulated into synchrony by afferent or intrinsic bombardment (Andersson et al., 1971a), is merely a question of the degree of "damping" of the frequency-tuned circuits. Several other workers have likened the CNS generally to a filter network (Prast, 1949; Lowenberg, 1959; Walter, 1959; Saunders, 1968). It has also been said that the alpha rhythm in man represents the natural resonant frequency of the brain (Wooldridge, 1963); and that the burst of waves at alpha frequency evoked by a single bright flash closely resembles the transient response of a narrow bandpass filter (Prast, 1949). However, it should be borne in mind that there is not just one "filter" but a multitude, and that these are unstationary in terms of both their neuronal elements and their relationships to one another. Thus the rhythmical entity should not be looked upon as consisting of a fixed group of neurons but as a functional unit which may change its size and characteristics from one moment to another (Andersson and Manson, 1971).

2:2:3 The Spread of Synchrony

Thalamic synchronization appears to be achieved by changes in the balance between excitatory effects on the thalamic relay cells and inhibition of the interneurons of the recurrent inhibitory pathway, as a

result of a decrease in afferent input (Andersson et al., 1971a; 1971b). During low excitatory drive the interneurons are disinhibited and recurrent inhibitory phasing can occur. Due to the widespread effect of the inhibitory interneurons an increasing number of projection cells are involved in the rhythms during each successive burst (Andersen and Andersson, 1968). The focus of synchrony can be highly localised and there is no evidence of a central pacemaker (Andersen and Andersson, 1974). However, from a study of the effects of specific lesions and reversible deafferentation, it was found that the medial thalamic nuclei may act as a gating mechanism modifying the excitability of the rhythm generators in other parts of the thalamus via a balance between the activity in inhibitory and excitatory connections (Andersson et al., 1971a; 1971b; Andersson, 1975). There appears to be no driving of rhythmicity from the medial thalamus to other nuclei, as Dempsey and Morison (1942) suggested, but rather a deprivation of the desynchronizing effects from the unspecific system.

Microelectrode recording from thalamic nuclei in both anaesthetized and unanaesthetized animals has shown that the area of synchrony in the thalamus is quite often limited to one particular nucleus, and that various independent synchronizing foci can be operating simultaneously at different frequencies (Verzeano and Negishi, 1960a; 1960b; Andersson and Manson, 1971). Zones of synchrony as small as 100-200 μm have been found (Verzeano et al., 1965; Andersen and Andersson, 1968), but coherence measures indicate that the individual regions are co-ordinated into larger functional units (Andersen and Andersson, 1974). Simultaneous widespread gating effects mediated by the medial thalamic nuclei could be partly responsible for this co-ordination (Andersson et al., 1971a). In addition, it has been suggested that distributor neurons aid the spread of synchrony (Andersen and Andersson, 1968). Because of the nexus between the nucleus reticularis

and the thalamus (Waszak, 1974), with the vast majority of reticularis cell axons projecting caudally upon the thalamus and upper brain stem (Scheibel and Scheibel, 1970), this structure may also play a role in the distribution of synchronization.

It has been argued that the travelling wave phenomenon of scalp surface potentials, as shown by spatio-temporal Fourier, or topographic analysis, demonstrate that EEG synchrony is not imposed upon the cortex, but is of cortical origin (Nunez, 1974; 1975; Petsche, 1975). However, it is possible that the travelling waves of the cortical EEG are determined by subcortical afferents, since Verzeano and Negishi (1960a; 1960b) found that rhythmic activity in the thalamus could be propagated in a form of spiralling through neuronal loops inside a given thalamic region. It would be interesting to see whether spatio-temporal Fourier analysis would provide evidence of travelling waves in the thalamus similar to those found by Nunez (1974) in the cortex.

Although the thalamo-cortical connections are very specific (Ganes and Andersen, 1975), the multiple projections from a single functional rhythmic unit are funnel-shaped (Andersen, 1975, personal communication), so that various small scattered zones of the cortex can be controlled and brought into synchrony. There is also evidence that bilateral synchrony is primarily mediated at the subcortical level through the massa intermedia and subthalamic interconnections (Andersen and Andersson, 1974).

The cortex itself is to some extent able to generate rhythmic slow waves (Kristiansen and Courtois, 1949; Andersen et al., 1967a), although the characteristic electrical pattern of cortical areas that have been completely isolated from all neuronal input is one of low voltage activity interrupted by paroxysmal discharges (Echlin et al., 1952; Andersen et al., 1967a). The apparent conflict between data from various laboratories studying the properties of the isolated cortex largely arises because of

differences in preparations and in the size of the cortical slab studied (Andersen and Andersson, 1968). It is likely that under normal conditions the cortex also has frequency selective properties which act to perpetuate and spread rhythmic subcortical input. Although recurrent inhibitory circuits are found in the cortex (Scheibel and Scheibel, 1970), it is also very probable that there is greater "damping", in the form of inhibition, in the closely coupled cortical networks, which would mean that they are less likely to produce spontaneous rhythmicity, and the frequency and distribution of the cortical rhythms would be largely determined by thalamo-cortical projections, except perhaps when some pathological mechanism causes a breakdown in normal inhibition. Petsche and Rappelsberger (1969), for example, have shown that during seizure activity the cortex plays a very important role in the propagation of synchrony.

The synchronization of the EEG thus appears to be strongly dependent on a rhythmic thalamo-cortical influence. Andersen and Andersson (1968) were unable to record barbiturate spindles from brain stem structures other than the thalamus. Albe-Fessard (1975, personal communication) reports finding a clear association between rhythmic activity in the caudate nucleus and the human alpha rhythm; but Gusselnikov et al. (1973) found that in the rat the caudate rhythm generating mechanism was secondary and reflected excitation from the thalamus. Input from extrathalamic subcortical structures must, however, combine with the thalamo-cortical input to influence the appearance, form, and distribution of the surface EEG.

2:3:1 Relevance of Animal Data

Alpha has been defined as a "rhythm, usually with frequency 8-13 Hz in adults, most prominent in the posterior areas, present most markedly when eyes are closed and attenuated during attention, especially visual" (Storm van Leeuwen et al., 1966). Activity similar to the human alpha rhythm is found in many species, although it may not fulfill all these criteria.

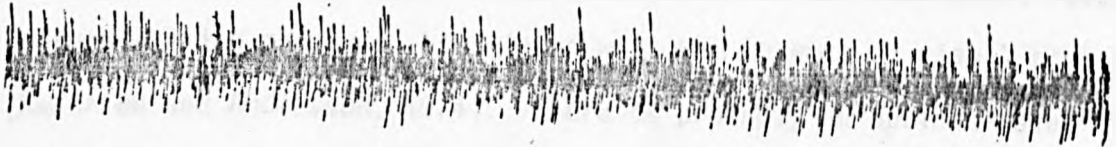
Much of the experimental evidence reviewed above on the neurogenesis of brain rhythms has been gathered from barbiturate anaesthetized preparations. Andersen and Andersson (1968; 1974) argue that barbiturate spindles provide a simplified analogue of spontaneous EEG rhythms, and that the basic rhythm generating mechanisms are similar. Lopes da Silva et al. (1973b) found differences in spectra and topographic distribution between alpha and barbiturate rhythms. However, from a study of the alpha rhythm in the unanaesthetized boxer dog, which conforms to the strict definition of the human alpha rhythm (Storm van Leeuwen et al., 1967), they conclude that there is sufficient evidence to extend the barbiturate spindle model to the standard alpha rhythm (Lopes da Silva et al., 1973a). Other workers have also recorded activity in the thalamus similar to barbiturate spindles from the unanaesthetized cat (Gjerstad and Skrede, 1970; Andersson et al., 1971a), or monkey (Verzeano et al., 1965).

Typical rhythmic activity has been recorded from the thalamus of conscious patients during neurosurgery for Parkinsonism (Albe-Fessard et al., 1966; Jasper and Bertrand, 1966). Albe-Fessard (1975, personal communication) reports observing a close correspondence between the activity in a part of the pulvinar nucleus of the thalamus and alpha rhythms recorded from the scalp. Gastaut (1949) recorded human alpha rhythms from the vicinity of the subcortical optic projections and concluded that, "The EEG develops principally in a system of closed cortico-thalamic circuits

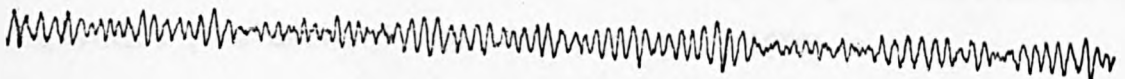
orientated radially in the occipital lobe". Gastaut also confirmed that the subcortical activity varied greatly and the surface EEG represented a synthesis of the individual rhythms.

The human alpha rhythm shows many similarities to the effects of a narrow band filter with noise input (Figure 2.4). The amplitude probability densities of normal alpha rhythm and noise bandpass filtered at 9 Hz are both Gaussian (Saunders, 1968). Thus it appears that surface alpha rhythm contains a random component, which most probably reflects the stochastic rhythm generating processes in the thalamus and the complex interaction of the individual elements from which the EEG is compounded. The characteristic spindling of alpha rhythm could be an expression of this resonance effect, as is clear from Figure 2.4. There seems little need to invoke the concept of beating between various alpha patterns suggested by

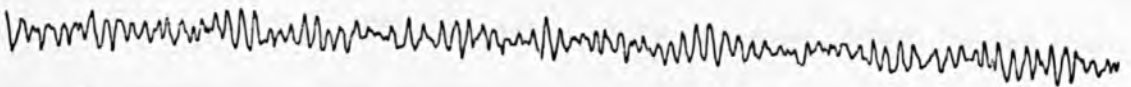
(a)



(b)



(c)



50 μ V [

1 sec

Figure 2.4

Similar characteristics of alpha rhythm and a frequency-tuned circuit presented with a random input.

(a) Recording of white noise. High frequency cut: 150 Hz; time constant: 0.3 sec.

(b) White noise after passage through a resonant filter (AIM, Model PFO 166B) with a centre frequency of 10 Hz and a Q-factor of 10 (i.e. a bandwidth of 1 Hz).

(c) An example of occipital alpha rhythm recorded from a normal adult female.

Walter (1950).

Barbiturate spindling can exhibit much variation in frequency throughout the thalamus depending on the anaesthetic level (Andersson, 1975, personal communication). In the unanaesthetized decorticate cat Andersson and Manson (1971) found that three characteristic rhythms could be recognised, largely related to the ventro-basal, the dorso-lateral and the medial regions of the thalamus. Andersen and Andersson (1974) suggest that such regional differences in the frequency and topography of EEG rhythms might reflect the relative importance of various connections influencing the excitability of the neurons involved in the thalamo-cortical rhythms, together with the functionally localised effects of changes in depolarising pressure in the specific afferent system.

The frequency stability of human alpha rhythm and its dominance in the occipital regions is perhaps related to a uniform and consistent reduction in depolarising pressure associated with the absence of visual processing (Andersen and Andersson, 1974). There is probably an optimum level of afferent input for the widespread production of alpha rhythm, which could account for the production of "paradoxical alpha" by stimulation during drowsiness (Kreitman and Shaw, 1965; Morrell, 1966).

The human alpha rhythm of the EEG is not a universal feature (Golla et al., 1943). However, alpha 'rhythm' has been distinguished from alpha 'activity' (Bartley, 1940; Lindsley, 1952; Harding, 1968), which may exist when a recordable alpha rhythm has been obscured by other on-going activity. The reason why alpha activity is more synchronized in some individuals than in others to produce a distinctive summated signal is unknown. Different attenuation factors may also be involved, for example, Leissner et al. (1970) found alpha amplitude to be correlated with skull thickness as measured by ultra-sonic techniques; and the various transmission characteristics of the intervening tissue and cerebrospinal fluid must also

be taken into account. There is, of course, not just one alpha rhythm for an individual, but many differing in frequency, phase, waveform and location in relation to the recording electrodes (Rémond, 1968; Mulholland, 1969; Maynard, 1972).

It appears to be justified to assume that the basic neurophysiological mechanism underlying the human alpha rhythm is essentially similar to that proposed for the generation of rhythmic EEG patterns in animals. However, many questions remain unanswered, and the appearance of the alpha rhythm is without doubt a complex phenomenon, very likely involving other processes in addition to frequency selective thalamo-cortical networks (Magoun, 1964).

Much less work has been done with regard to the other classical rhythms of the EEG. Beta rhythm could perhaps reflect the activity of separate, motoric, thalamo-cortical functional units; and typical theta activity has been recorded from the postero-medial region of the baboon thalamus during drowsiness (Bouyer et al., 1972). During delta slow waves in the EEG there appears to be a marked depression of neuronal activity in the thalamus (Howe and Sterman, 1972), reflecting perhaps a general decrease in depolarising pressure. Extra-thalamic subcortical structures are likely to be differentially involved in the genesis of these rhythms.

2:3:2 Alpha Rhythm as a Physiological Artifact

The etiology of the alpha rhythm has remained a controversy that has persisted since its discovery by Hans Berger; and in recent years some authors have again raised doubts concerning its neuronal origin (Brumlik, et al., 1966; Bell, 1972; Lippold, 1973).

A number of cardiobalistic and mechanical theories have been advanced : the theory of Kennedy (1959) being the most well known. He proposed that the alpha rhythm was a consequence of the mechanical oscillation of the

charged brain gel through arterial pulsation of the cerebrospinal fluid. The evidence for this theory was his claim that alpha waves were absent in a trephined subject until intracranial pressure was restored by means of a rigid skull covering. This curious result was rebutted by the examples of alpha rhythm recorded from the widely exposed surface of the brain during surgery (Rosner, 1961).

Another argument for the peripheral origin of alpha rhythm has been voiced by Lippold (1973). He has persisted in claiming that alpha rhythm arises from the orbit of the eye and is related to tremor in the extraocular muscles. Originally he proposed that the corneoretinal potential (a standing potential across the eyeball, thought to stem from the regeneration of visual purple) was electronically conducted by the extraocular muscles to be recorded from the occipital poles. The alpha waveform came from resistance changes in the shunt path caused by tonic tremor (Lippold, 1970; Lippold and Novotny, 1970; Ennever et al., 1971). In support of his thesis, he maintains that there is a strong correlation between translational eye movements and the alpha rhythm, and that experimental manipulation of tremor frequency causes a corresponding change in alpha frequency (Lippold and Novotny, 1970). In addition, Lippold and colleagues report a decrease in alpha amplitude associated with decreased corneoretinal potential produced by a reduction in the level of ambient illumination (Ennever et al., 1971); which logically implies that alpha rhythm should be much attenuated in darkness!

Alpha rhythm in eyeless patients has been reported by several laboratories (Abbott and Dymond, 1970; Shaw et al., 1970; Upton et al., 1970). Undaunted, Lippold proposed extra-retinal potential sources of unknown origin in the orbit (Lippold and Shaw, 1971), together with fluttering of the muscle stumps and conjunctival lining (Lippold, 1973). Butter and Glass (1970) investigated the ocular tremor hypothesis and found

no evidence that the alpha rhythm was imposed by eye tremor, although they recorded rotational eye movements and not axial eye movements. A critical experimental test of the hypothesis was carried out by Chapman et al. (1971) who recorded alpha rhythm from two patients who had undergone complete unilateral exenteration, in which the entire contents of one orbit were removed. No marked asymmetries were found in the alpha rhythm over the two hemispheres (Cavonius^V, 1973). Lippold himself found that alpha rhythm, defined exactly as in man, was still present in the monkey (*Macaca Mulatta*) after bilateral enucleation. To this he concedes, "It therefore appeared that the waves did in fact originate in the substance of the brain" (Lippold, 1973).

Morrell (1967) has provided strong evidence for the neuronal basis of the alpha rhythm. He carried out intracellular recording from the human occipital cortex and found that there were clear, regular and coherent relationships between intracellular potentials and selected wave sequences of the EEG for some cortical neurons. It is unlikely that a closer relation of the complex surface rhythm to individual cortical neuronal function could ever be found. Furthermore, Morrell (1967) demonstrated the localised desynchronization of cortical alpha rhythms by the illumination of small sectors of the visual field, which was in accordance with the known anatomical distribution of fibres in the primary afferent pathway for the area stimulated. However, it still remains to be conclusively demonstrated that the human alpha rhythm has a thalamo-cortical origin by a precise experiment of the sort carried out by Ganes and Andersen (1975, *supra*), and it is to be hoped that an opportunity for this to be done during neurosurgery will be found.

2:3:3 Efferent Oculomotor Processes and the Alpha Rhythm

The blocking of the alpha rhythm has been classically associated with visual afferent and attentive processes (Storm van Leeuwen et al., 1966). Mulholland (1968; 1969; 1972) has pointed out that there is ample evidence, both anatomical and experimental, that efferent and integrative processes related to moving, positioning and focusing the eyes are also important. For example, these functions have extensive representation in areas 17, 18, and 19 of the visual cortex, and various ocular fixation and pursuit tracking tasks have demonstrated alpha blocking associated with oculomotor control processes (Mulholland, 1972). Visual attention to a uniform visual field alone does not produce alpha blocking, as Lehtonen and Lehtonen (1967) demonstrated with flash counting. Mulholland and Peper, (1971) suggest that alpha blocking probably only occurs in the overall process of pursuit tracking, convergence and lens accommodation through a combination of large afferent inflow and extensive oculomotor control processes. Wertheim (1974) takes the argument further by differentiating between oculomotor control monitored by visual input and that monitored by internal events such as memory functions; but there is no experimental data to support his view. It is possible that the well known blocking of alpha by mental exertion, and also with the startle reaction (Mundy-Castle, 1957), could be explained by a concurrent increase in oculomotor activity (Lorens et al., 1962).

Butler and Glass (1970) found evidence for a weak cross-correlation between alpha rhythm and eye movement or eyelid flutter. They proposed the counter-hypothesis to Lippold (*see* 2:3:2) that eye movements may be modulated by extracellular current flow at alpha rhythm frequencies in the brain stem invading the oculomotor abduceus and trochlear nuclei. Other workers have also noted the juxtaposition of the oculomotor system and the

reticular system in the same zone of the brain stem tegmentum (Bender, 1969; Mulholland, 1972). Hence it is possible that there is some cross-talk between the systems at this level, although it is more probable that the eye movement related activity would have a small "phase-locking" effect on the stochastically determined alpha rhythms. Slow wave potentials, associated with eye movements but not dependent on the type of movement, have in fact been observed in the thalamic lateral geniculate nucleus of the alert monkey in darkness (Feldman and Cohen, 1968).

It has been reported that alpha rhythm can be enhanced by extreme elevation of the eyes (Mulholland and Evans, 1965; Dewan, 1967; Kris, 1968; Mulholland, 1968; Bender, 1969; Fenwick and Walker, 1969; Mulholland, 1972). Fenwick and Walker (1969) demonstrated increased alpha activity by extreme eye positioning even when this manoeuvre was carried out in total darkness, indicating that this phenomenon is independent of visual stimulation. Parametric investigation showed that the maximum induced alpha activity was attenuated sharply at about 20° either side of the vertical visual axis (Fenwick and Walker, 1969). Dewan (1967) reports using forced eye elevation to enable subjects to achieve a high level of voluntary control over their alpha rhythm, although since subjects were also allowed to use eye closure and imagery to influence their alpha rhythms, his results are difficult to interpret. Some workers have failed to find a relationship between eye position and alpha amplitude (Fenwick, 1966; Chapman et al., 1970); and not all subjects show this effect, although it is reliable in those who do (Mulholland, 1968). Extreme forced elevation of the eyes, often sufficient to engender discomfort, is said to be necessary to produce this effect (Mulholland, 1968; Fenwick and Walker, 1969). It has been suggested that it results mainly from a tendency for the eyes to defocus and relax convergence (Dewan, 1967), although extreme eye elevation produces many other complex changes in the ocular system (Fenwick and Walker, 1969).

2:4:1 Thalamo-cortical Origin

Synchronized electrical activity of the sensorimotor cortex during behavioural immobility or internal inhibition has been described in a number of species (Gastaut, 1952; Kruger and Henry, 1957; Kogan, 1960; Anokhin, 1961; John et al., 1961; Rowland, 1961; Donhoffer and Lissak, 1962; Roth et al., 1967; Sterman and Wyrwicka, 1967; Chase and Harper, 1971; Howe and Sterman, 1972; Rougeul et al., 1972; Bouyer et al., 1974).

In the cat a characteristic rhythm of some 12-18 Hz is seen to arise from the region of the coronal gyrus, ranging in amplitude from 50-500 μ v (Chase and Harper, 1972; Rougeul et al., 1972). This was neologized "sensorimotor rhythm" by Roth et al. (1967) since they believed its anatomical substrate to be the sensorimotor cortex. Subsequent macroelectrode studies have localised SMR specifically to the post-cruciate gyrus, which corresponds to the somatosensory projection area from the trunk and limbs of the cat (Howe and Sterman, 1972; Rougeul et al., 1972). SMR correlates have also been found generally within the subcortical structures of the somatosensory system and particularly in the thalamic nucleus ventralis posterior lateralis (VPL), which is a primary somatosensory relay nucleus (Harper, 1973). During epochs of SMR, high voltage rhythmic activity has been observed in the VPL which closely parallels cortical SMR activity (Sterman et al., 1970; Howe and Sterman, 1972).

Extracellular recording revealed that individual VPL neurons display characteristic firing patterns during SMR activity. These patterns usually consist of recurrent bursting and silence, but occasionally instances of enhanced discharge are encountered, as shown by the examples provided in Figure 2.5. The bursting seen in the VPL nucleus during the development of

spontaneous SMR is very similar to that accompanying barbiturate spindle activity. Thus it would seem justified to assume that the basic inhibitory phasing model, which we have discussed for the generation of EEG synchronization patterns, applies also to the rhythmic activity recorded from the sensorimotor cortex. This is further supported by the fact that cortical SMR and correlated VPL activity are disrupted simultaneously by phasic motor activity (Howe and Sterman, 1972), and that a lesion in the thalamus abolishes SMR activity in the corresponding region of the cortex (Bouyer et al., 1974).

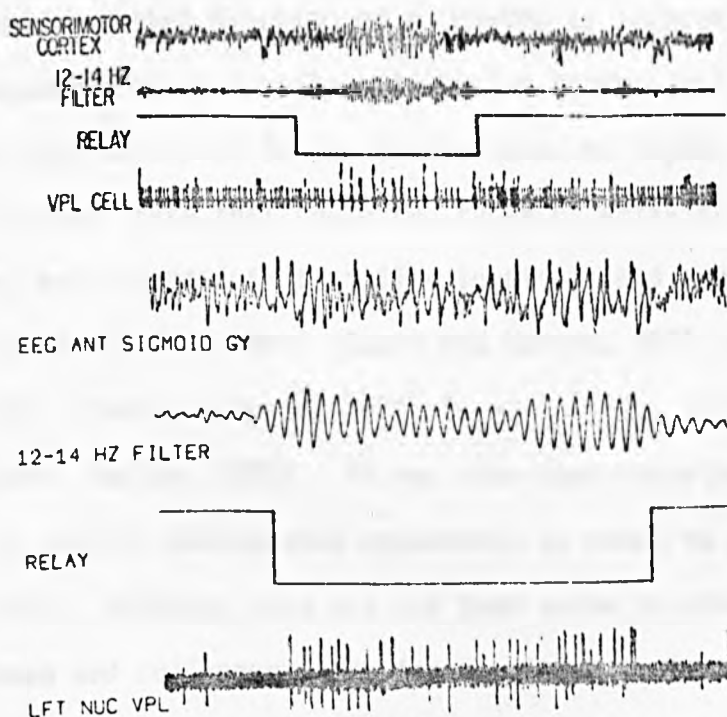


Figure 2.5

The two basic discharge patterns recorded extracellularly from neurons in the VPL thalamic nucleus of the cat during trained production of SMR. The majority of units exhibit the shift from irregular high frequency discharge to a burst and silence pattern during SMR activity, as exemplified in the upper example. The lower, recorded on a faster time-base, shows a shift from low baseline rates to a specific rapid discharge associated with the SMR which characterises other VPL neurons. (From Sterman (1973a).

2:4:2 Sensorimotor Rhythm and Mu Rhythm

An EEG pattern in man that bears many topographical and functional similarities to the SMR recorded in the cat is the mu rhythm, also known as 'rythme rolandic en arceau', 'comb rhythm', or 'wicket rhythm'. This rhythm was discovered during electrocorticographic recording by Gastaut (1952). He described it as, "... formée d'ondes asymétriques, à phase positive acérée et à phase négative arrondie; ... sa fréquence se situe autour de 9 c/s et sa répartition spatiale et temporelle est des plus originales puisqu'il occupe la région rolandique des deux côtés".

The mu rhythm was found to occur as localised synchrony specifically related to a lack of movement in the relevant muscle group for the corresponding area of cortical representation. It has subsequently been well established that blocking of mu rhythm is induced by passive and active movements or by tactile stimulation (Yamada and Kooi, 1975).

The relationship of feline SMR to human mu rhythm is controversial. Several authors state that these two types of activity are synonymous, since they are recorded from similar locations and are both associated with a cessation of movement (Chase and Harper, 1971; Rougeul et al., 1972; Lanoir, 1973; Babb and Chase, 1974; Bouyer et al., 1974; Harper, 1973; Rougeul, 1974; Kaplan, 1975). It has also been reported that the cat SMR exhibits a similar saw-toothed appearance to human mu rhythm (Chase and Harper, 1971), although this has not been noted by other workers.

Sterman and colleagues take issue. These workers hold that human mu rhythm is not the equivalent of feline SMR, but that the SMR represents an independent neural process in both cat and man (Sterman et al., 1974). Their contention is based on four main points:

- (1) The lower frequency human mu occurs spontaneously and is related most directly to a state of relaxed wakefulness. Conversely, SMR in man

emerges as a specific consequence of biofeedback training and is related to a more focused state of wakefulness.

(2) There is little evidence for a mu rhythm in the cat, the motionless state being characterised by the higher frequency SMR.

(3) SMR in man develops over both frontal and central cortex, in contrast to the strictly rolandic localisation of mu.

(4) In both cat and man the SMR pattern is not always disrupted by small localised movements, as is the human mu rhythm.

Their argument is weak on several grounds however. Firstly, even though a specific enhancement of 12-16 Hz rolandic EEG activity can be obtained through feedback training, it does not, of course, necessarily follow that this activity is therefore SMR; especially since there has been no evidence yet presented to indicate that these frequencies in man bear any relationship to a motionless state. Secondly, their topographic distinction between SMR and mu is at variance with the reports of other workers that mu rhythm is seen in frontal areas (Klass and Bickford, 1957; Yamada and Kooi, 1975); the distinction is made more tenuous by the fact that SMR in the cat is localised to the post-central cortex. Finally, Chase and Harper (1971) have observed that the disruption of feline SMR can be highly localised in the cortex and thus not readily be detected by gross electrodes. The blocking of human mu rhythm by movement has similarly been found to be functionally localised at the cortical level (Gastaut, 1952).

There is some neurophysiological evidence to suggest that mu rhythm is primarily linked with the rolandic motor cortex (pre-central gyrus), which contrasts with the region responsible for the formation of SMR in the cat (Howe and Sterman, 1972 *supra*). Albe-Fessard (1975, personal communication) found that thalamic cells in the nucleus ventralis lateralis (VL) and nucleus ventralis intermedius (Vim) (which receive motor and

cerebellular afferents) exhibit bursting during mu rhythm, rather than cells in the VPL somatosensory nucleus, and the rhythmic bursting was disrupted only in the Vim nucleus during contralateral fist clenching.

These thalamo-cortical differences could prove to be grounds for distinguishing SMR from mu activity. On the other hand, Rougelet et al. (1972) have noted that rolandic motor rhythms in the monkey, which were very similar to human mu, were found on closer investigation to originate from post-central regions; and that the thalamic loci of these rhythms were a function of the state of arousal (*see below*). At the cortical level the motor and sensory systems are closely intertwined and are both likely to be involved in the appearance and blocking of rolandic rhythms.

Depth recordings have indicated spontaneous beta rhythms in the human pre- and post-central cortex that are related to motor inhibition, but which are generally much higher in frequency than either mu or SMR activity. Thus Jasper and Penfield (1949) recorded a rhythm of 18-30 Hz, which was blocked by voluntary movement specifically in the cortical area of representation of the portion of the body being used. They proposed that this beta rhythm was characteristic of the motor cortex in a manner analogous to the alpha rhythm for the occipital cortex. Sem-Jacobsen et al. (1956) similarly found that when subjects were relaxed high amplitude rhythms of 25 Hz dominated the electrocorticogram, whereas activation produced an increase to 50 Hz or more. Conversely, depth recordings have also revealed mu rhythm in some subjects (Chatrian et al., 1959).

The relationship between the human mu and beta rhythms has not yet been adequately resolved. It has been proposed that the mu waveform might represent halving of the beta rhythm resulting from the complex interaction of phase relationships (Gastaut et al., 1954). However, Yamada and Kooi (1975) found in a group of twenty subjects, with well defined mu and beta activity, that the beta rhythm was seldom an exact second harmonic of the mu

waveform, but tended for most subjects to be slightly faster. Hence they suggested that mu may reflect a mixture of higher order harmonics.

An important recent contribution to this debate has been made by Rougeul and her associates, who found that somatosensory rhythmic patterns associated with behavioural immobility are a function of the state of alertness (Rougeul et al., 1972; Bouyer et al., 1974; Rougeul, 1975). On the basis of their studies on the cat and the monkey (*Papio papio*) they distinguished mu activity into three separate types.

(i) *Rythmes d' hypervigilance* (RHV): ranging in frequency from 20-40 Hz, with a mean of 36 Hz, in the cat; and from 12-18 Hz, with a mean of 18 Hz, in the monkey. These patterns are seen whilst the animal is motionless but attentive.

(ii) *Rythmes de veille immobile* (RVI): ranging in frequency from 12-18 Hz, with a mean of 14 Hz, in the cat; and from 8-13 Hz, with a mean of 8 Hz, in the monkey. These patterns are equated with SMR since they appear when the animal enters a quiet, relaxed, motionless state.

(iii) *Rythmes d' assoupissement antérieurs* (RAA): occurring when the animal is drowsy, but not sleeping. The frequency of these patterns varies continuously in both the cat and the monkey, indicating an interaction of RVI with a slower component.

Rougeul (1975) reports that similar rhythms to RVH, RVI, and RAA have also been observed during corresponding behavioural states in the squirrel monkey, and in children. These rhythms are all disrupted by movement, excepting small localised motoric activity such as eye movements. In both the cat and the monkey the three types of mu activity were found to exhibit a close correspondence to single unit activity in the thalamus. They are each associated with thalamo-cortical systems which are independent at the thalamic level but interact in the cortex (Rougeul et al., 1972; Bouyer et al., 1974).

Sterman's argument that rolandic 12-16 Hz components in man are equivalent to SMR recorded from the cat, is based on the inherent assumption that the human EEG is homologous to that of the cat. It would seem more reasonable to suppose, however, that the human EEG bears a phylogenetic similarity to that of other primates. Hence the data of Rougeul's group strongly suggest that the 12-16 Hz feline SMR is paralleled in man by mu activity; which generally ranges in frequency between individuals from 7-13 Hz (Klass and Bickford, 1957; Storm van Leeuwen, 1966), although Albe-Fessard (1975, personal communication) reports that characteristic mu spindles of up to 25 Hz have occasionally been observed.

A distinct mu rhythm is a relatively rare occurrence in the scalp EEG of the majority of human subjects. Cohen et al. (1964) suggest that this be due to a constant barrage of somaesthetic stimuli which disrupt its appearance. Hence they endeavoured to enhance mu activity by the minimisation of peripheral sensation, but without any success. It is also quite possible that the mu waveform is rare simply because it could depend on a particular harmonic relationship of patterns in the rolandic cortex, as many workers have suggested; but these patterns themselves vary as a function of the level of somatosensory afferentation and of the state of alertness.

In addition, there is much evidence that efferent processes are involved in the regulation of central cortical synchronization, as with the occipital alpha rhythm. Several workers have reported that the mu and beta rhythms are disrupted by imagined or intentional movement (Jasper and Penfield, 1949; Gastaut, 1952; Gastaut and Bert, 1954; Magnus, 1954; Penfield and Jasper, 1954; Klass and Bickford, 1957). It was found, for example, that mu rhythm is blocked by the attempt of amputees to move a phantom limb (Chatrian et al., 1959; Gastaut et al., 1965). Similarly, Rougeul et al. (1972) report that in the cat RVI is seen to block a short time before any muscle contraction is indicated in the electromyogram.

These observations thus demonstrate that peripheral input is not essential for the desynchronization of the somatosensory rhythms.

It has been reported that the central rhythms are sometimes blocked by non-somaesthetic stimuli (Gastaut and Bert, 1954; Chatrian et al., 1959). This effect might be a result of small covert movements, or possibly of efferents representing feed-forward information processing in readiness for a motor response.

2:4:3 Sensorimotor Rhythm and Sleep Spindles

During light sleep, bursts of rhythmic activity of approximately 14 Hz, which closely resemble feline SMR, are seen to develop over the sensorimotor cortex in many species (Stermann et al., 1970). The single unit activity of the VPL thalamic nucleus during these sleep spindles has also been demonstrated to be very similar to that seen during conditioned SMR (Harper, 1973).

Biofeedback training of SMR has been claimed to increase the amplitude and incidence of sleep spindles in both cats and humans (Lucas and Stermann, 1974; Stermann, 1974); and that motor disturbances during sleep are diminished in SMR trained animals, and the overall time spent in sleep reduced (Lucas and Stermann, 1974). Moreover, the variations in somatomotor and visceromotor activity that accompany spindle sleep are in general similar to those observed for epochs of SMR (Chase and Harper, 1971).

Because of the considerable functional and neurophysiological resemblances between SMR and sleep spindles, Stermann (1974; 1976c) proposes that there is a common neural basis for these two phenomena, and that a specific relationship exists between inhibitory mechanisms concerned with the suppression of motor inhibition in the awake and in the sleeping state. However, Johnson et al., (1976) found that there was no significant

relationship between spindle bursts and the onset or the rate of motor activity in sleep. It has also been experimentally demonstrated that SMR and sleep spindles have their origin in separate thalamo-cortical systems. SMR is found optimally in the somatosensory projection system - the VPL nucleus and the post-cruciate region (Harper, 1973); whereas sleep spindles have a thalamic focus primarily in the VL nucleus and are recorded maximally from the pre-cruciate area (Sobieszek, 1968; Howe and Sterman, 1972), which implies that their affinity is more with the motor system. Howe and Sterman (1972) report that there is no correlation between sleep spindles and similar activity in the VPL, and that a separate SMR could sometimes be recorded simultaneously from that location. Rougeul and co-workers also found that the thalamo-cortical system for sleep spindles was distinct from that for their three types of mu activity (Rougeul et al., 1972; Bouyer et al., 1974), and Albe-Fessard reports a similar observation in man (1975, personal communication).

The conclusion, therefore, is that the neurophysiological as well as the behavioural characteristics of SMR and sleep spindles are distinct, although these rhythms might share some similar links with mechanisms associated with motor inhibition. As such, SMR in the cat does not represent sleep spindles occurring in the awakening state, as has been suggested by Sterman (1974; 1976c). If this proposal were true, then there would be good grounds for arguing that a similar SMR should exist in man, especially since the frequency band of sleep spindles happens to be identical in both man and cat. However, the evidence unequivocally indicates that these are independent phenomena and that their relationship is secondary.

2:5:1 Alpha Rhythm

The majority of experimental studies that have attempted to investigate the function of synchronized EEG activity have concentrated on the dominant alpha rhythm. Early authors favoured the idea that the function of the alpha rhythm might be to maintain the cortex in an active state during periods in which attention is directed away from visual attention. Correlates between the alpha rhythm and various indices of visual processing or imagery have been reported by many workers (Jasper and Cruickshank, 1937; Golla et al., 1943; Slatter, 1960). These, in general, were interpreted to support the hypothesis that alpha rhythm is closely involved with visual attention. Conversely, other workers have produced results which are not consistent with this hypothesis (Walsh, 1953). Most studies have taken no account whatsoever of oculomotor activity, which plays a primary role in the appearance and blocking of the alpha rhythm (see 2:3:3), and so no firm conclusions can be drawn from the data.

It has been postulated that the alpha rhythm expresses a correlate of the activity of a neuronal mechanism for the gating and scanning of visual information (Nunn and Osselton, 1974). Thus the alpha rhythm has been likened to a television raster (Walter, 1953), or to a memory "readout" system (Andersen and Andersson, 1968). However, there is little evidence to raise these hypotheses above the level of speculation. On the contrary, some investigations have found that the concept of a perceptual scanning system is made rather unlikely by the experimental data (Walsh, 1952; MacKay, 1953).

Another theory which has proved to be quite popular is that the alpha rhythm represents an excitability cycle of the central nervous system (CNS).

There have been a number of claims that the phase of the alpha rhythm is correlated with visual reaction times (Lansing, 1957; Callaway, 1962; Dustman and Beck, 1965). However, since it is well known that the phase of alpha rhythm measured at different leads can be quite different, and that the phase relationships between the leads are not constant but variable (Walsh, 1953), such reports are virtually meaningless. Several investigators have found evidence for a strong correlation between the frequency of the alpha rhythm and response time or decision time (Denier et al., 1959; Mundy-Castle and Sugarman, 1960; Surwillo, 1963; 1964). Boddy (1971), using on-line power spectral analysis, was unable to confirm these results, however, and suggested that they were most likely due to shifts in the EEG frequency bands associated with changes in the level of arousal.

A common proposal is that cortical cells tend to synchronize when they are "idle" (Adrian and Matthews, 1934b), and that the alpha rhythm and similar EEG patterns represent an "idling cortex" (Chase and Harper, 1971). It is now clear, however, that there is not simply an abrupt removal of afferent impulses to the cortex in the absence of sensory input (the optic pathway, for example, is considerably occupied with the "dark discharge"), but that EEG synchronization is mediated by the thalamus and involves a shift in the complex balance of excitation and inhibition (see 2:2:1).

At the thalamic level, synchronized EEG activity appears to have important consequences for information processing in the CNS. Because of the long lasting hyperpolarization of the relay cells associated with rhythmic behaviour, the synaptic transmission of thalamic afferents to the cortex is impeded (Andersen et al., 1967b; Howe and Serman, 1973). Many thalamo-cortical circuits are involved through the thalamic distribution of rhythmic activity, which results in the functional deafferentation of diverse cortical areas. In addition, widespread lateral inhibition in the

thalamo-cortical projection systems from the recurrent inhibitory process could lead to a sharpening and accuracy in the analysis of incoming sensory information. Steriade and Deschenes (1974) have demonstrated powerful surround inhibition of pyramidal tract neurons during stimulation of the VL nucleus; and Massion (1975) found evidence of a feedback mechanism whereby stimulation of the cortex evokes localised rhythmic activity in thalamic nuclei, which he suggests might function to enhance the signal to noise ratio of specific afferents. This differential modulation of the sensitivity of particular thalamo-cortico-thalamic feedback loops might also suggest a possible neurophysiological mechanism for the focusing of 'attention'.

Andersen and Andersson (1968) pointed out that synchronized EEG behaviour, such as the alpha rhythm, could be an epiphenomenon - an unavoidable process that goes with powerful thalamic recurrent inhibition, which has a special purpose of its own, such as the lateral inhibition of sensory information. Adrian and Matthews (1934b) also expressed the view that the EEG rhythms have no function but arise as a consequence of the way in which neurons are interconnected. I am also in favour of assigning a passive role to the EEG synchronization patterns. Although synchronized brain activity appears to be closely related to functional inhibition, the synchrony can be seen as a secondary phenomenon that follows from the frequency selective properties of neural networks- in particular those associated with sensory integrative and gating mechanisms in the thalamus.

2:5:2 Sensorimotor Rhythm

Sensorimotor synchronization has been proposed as the cortical correlate of Pavlovian "internal inhibition" (Anokhin, 1961; John et al., 1961; Rowland, 1961; Donhoffer and Lissak, 1962; Roth et al., 1967).

According to Pavlov (1927), when a positive conditional stimulus is not reinforced a negative conditional reflex develops which results in internal inhibition, manifested as a cessation of activity, drowsiness and sleep. However, since SMR can arise when cats are given reinforcement for simply remaining motionless (Chase and Harper, 1971) it is unlikely that internal inhibition, following from processes of differentiation, extinction or delay, is a direct causal antecedent.

The behavioural immobility concurrent with SMR is most often accompanied by a reduction in somatic processes, although there need not be a generalized cessation of motor activity, since SMR has been also recorded during sustained tonic contraction. (Donhoffer and Lissak, 1962). Bursts of SMR are accompanied by a decrease in the tone of the nuchal musculature (Chase and Harper, 1971), together with a suppression of the masseteric jaw reflex (Babb and Chase, 1974). There are also changes in respiratory and cardiac activity that are time-locked to the period of the SMR burst. The respiratory pattern tends to become more rhythmic and stable, and it has been further claimed that there is a high correlation between the onset of each epoch of SMR and the inspiratory phase of the respiratory cycle; and similarly between the termination of each epoch and the expiratory phase (Chase and Harper, 1971). Heart rate increases slightly just before the onset of SMR, followed by a marked decrease of up to 30% (Chase and Harper, 1971). A synopsis of these physiological correlates of SMR is provided in Figure 2.6.

The changes in somatic, respiratory and cardiac activity concomitant with SMR are parasympathetic in nature and are said to be similar to those seen during internal inhibition and sleep. This led Sterman and Wyrwicka (1967) to propose that the changes express a "central state of inhibition" which is responsible both for the development of SMR and for the suppression of phasic motor behaviour. However, much more neurophysiological

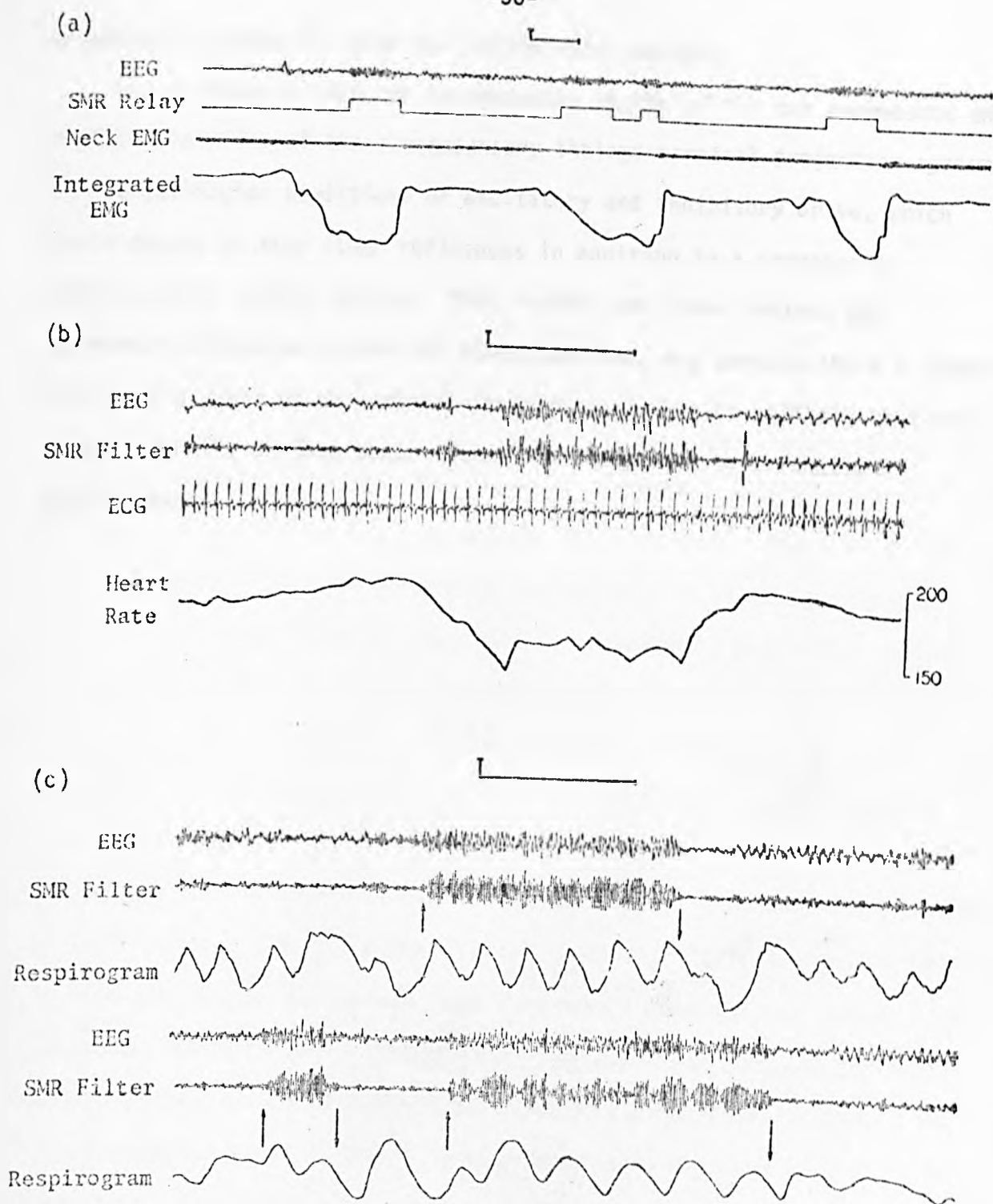


Figure 2.6

(a) Depression of neck electromyogram (EMG) during conditioned SMR in the cat. Note that the reduction in integrated EMG is greater during the latter part of the SMR bursts than at the beginning.

(b) Electrocardiogram (ECG) during SMR. Heart rate increases slightly before the onset of SMR epochs, followed by a pronounced decrease that is time-locked to the period of the burst.

(c) Two examples of the stabilization of respiratory activity during SMR episodes. The SMR spindle develops during inspiration (upward deflection) and terminates during expiration (downward deflection)

Calibration bars: 50 μ V and 5 sec.
After Chase and Harper (1971).

evidence is needed in order to justify this concept.

It is probable that the sensorimotor rhythm of the cat represents the rhythmic activity of the somatosensory thalamo-cortical projection system during particular conditions of excitatory and inhibitory drive, which could depend on many other influences in addition to a decrease in proprioceptive sensory inflow. This rhythm, and other related EEG phenomena such as mu rhythm and sleep spindles, may perhaps share a common link with a state of behavioural inhibition; but it is unlikely that this link is causal, or that these rhythms have any important functional significance.

It has been suggested that the recorded EEG mainly results from both the statistical and linear summation of potential changes on the membranes of cortical neurons. Independent sub-cellular generator elements are dynamically interrelated to form vertically oriented functional zones of synchronized potential. The activity can be much attenuated, modified and averaged during transmission to the cortex.

Stochastic rhythmic foci in the thalamus impose synchronized activity upon the cortex via strictly topographical but diffuse projections. Thalamic rhythmicity is associated with the recurrent phasing of inhibition which arises from changes in the balance of excitatory and inhibitory modulation and perhaps also from intrinsic properties of the neuronal membranes. Various mechanisms are discussed for the initiation, gating and spread of synchrony. The behaviour of thalamic, and to a lesser extent, cortical neural networks is likened to damped frequency-tuned circuits.

There is considerable evidence to suggest that the human alpha rhythm of the EEG has a similar neuronal basis, and very little to support the various alternative mechanisms that have been argued for its origin. The frequency, waveform and distribution of the alpha rhythm could primarily be a consequence of particular conditions of diminished excitatory drive upon thalamic nuclei, although further structures and processes might be involved in its appearance. Efferent oculomotor functions are very closely linked to the gating and desynchronization of the alpha rhythm.

Central cortical patterns of synchronization that are associated with motor inhibition have been demonstrated to be a function of the state of alertness. Feline SMR for the same behavioural conditions in primates is paralleled by activity at classic mu rhythm frequencies. Sleep spindles have a neurophysiological origin distinct from SMR, although both these

EEG patterns might perhaps share a similar functional relationship to hypothetical central inhibitory mechanisms.

Synchronization of the EEG is seen as a secondary phenomenon exhibited by dynamic thalamo-cortical neuronal networks involved in the high-level filtering and integration of information within the CNS.

Chapter Three FEEDBACK TRAINING OF THE EEG ALPHA RHYTHM

3:1 ALPHA DETECTION AND FEEDBACK

3:1:1 Introduction

In order to carry out an investigation of feedback electroencephalography the general specifications of a system for the detection of alpha rhythm in the EEG, and its conversion to a form of sensory feedback, were derived from an appraisal of the extant literature. I then designed and constructed a suitable system using facilities in the Department of Communication. The setting up of this system, and some of the many problems that were encountered, are described below.

The study of feedback training of the alpha rhythm began with a replication and closer analysis of the results reported by Kamiya (1967; 1968; 1969) and other early workers. This led to a further investigation of the influence of the level of arousal on alpha rhythm, and to an exploratory study into the use of analogue modes of feedback. The possibility of adopting avoidance training techniques for the enhancement of alpha rhythm was later investigated with the assistance of a colleague.

3:1:2 Development of Apparatus

A synoptic diagram of the complete alpha detection and feedback system is provided in Figure 3.1. The EEG was recorded using a sixteen-channel electroencephalogram (Beckmann, Model TC). One occipital channel was fed into an analogue band-pass filter in order to isolate 8-12 Hz alpha activity from the raw EEG. A programmable filter-oscillator (AIM, Model PFO 166B) was first tested for this purpose, but was found to be unsuitable because of instability. A variable frequency band-pass filter (Barr and

Stroud, Model EP2) was therefore borrowed to replace it. The frequency response of the band-pass filter is flat within 3 dB between the selected cut-off frequencies of 8 Hz and 12 Hz, and the filter attenuation slope a nominal 48 dB/octave.

Some workers have used filtered alpha rhythm to drive directly various forms of feedback (Brown, 1974). However, in order to establish whether or not any modification of alpha abundance actually does occur with feedback training, it is necessary that there be some means of identifying and registering the alpha activity, so that it can be quantified. One popular and convenient method is to use rectification and smoothing to produce a DC voltage, varying in proportion to the alpha activity, which is then fed to a level-sensitive trigger circuit set to operate a relay system whenever alpha activity above a certain amplitude occurs (Boudrot, 1972; MacDonald and Nowlis, 1969; Pasquali, 1971). As Paskewitz (1971) has pointed out,

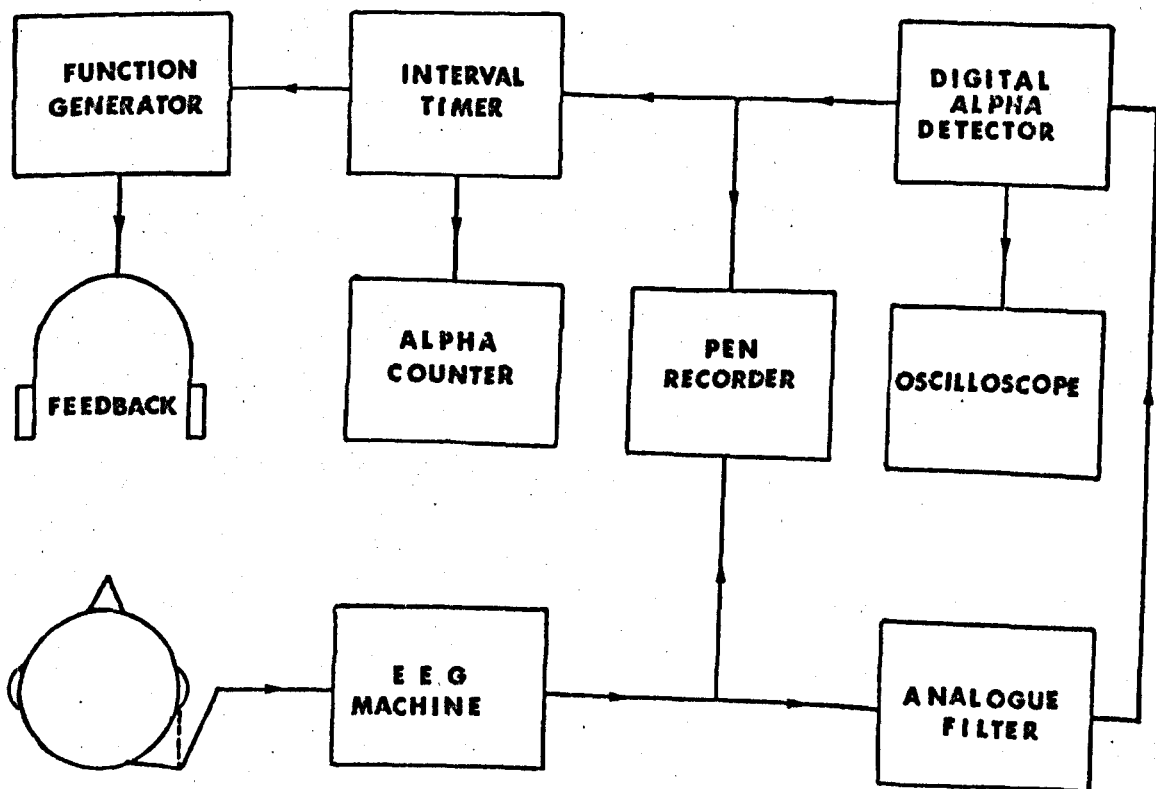


Figure 3.1
Schematic outline of the alpha detection and feedback system.

however, there are two major sources of inaccuracy inherent in this method of alpha detection. The most obvious is that relatively high amplitude activity outside of the alpha band, in particular artifacts, may be insufficiently attenuated by the band-pass filter to prevent false triggering of the detector circuit. Merely increasing the attenuation slope of the filter does not solve this problem, since this will in turn increase the likelihood of the filter itself generating a spurious signal in the form of "ringing" evoked by transients in the EEG (*see* 5:1:2).

The second drawback of this technique concerns the time constant of the detector circuit. A short time constant will cause the detector to be too sensitive to amplitude fluctuations of alpha activity. Conversely, a long time constant will slow the response of the detector, with the result that, on the one hand, the detector will be held on for a period after the cessation of a high amplitude burst of alpha activity; and, on the other, a corresponding delay in the activation of the detector occurs, with the possibility that short low amplitude bursts could be missed altogether.

An alternative approach for the identification of alpha activity in the EEG is to utilize digital period analysis. This enables step-function cut-offs at 8 Hz and 12 Hz to be obtained which are independent of amplitude changes in the EEG. I decided to adopt digital techniques in my system for detecting and quantifying the alpha activity, in preference to the analogue methods used by most other workers in extant studies because they offer increased power to discriminate against artifact.

Paskewitz (1971) describes a circuit for a digital alpha detector which operates by pulse-width discrimination as illustrated in Figure 3.2. This circuit was constructed with a number of minor modifications. The detector is provided with a threshold control so that no output is produced unless the filtered EEG exceeds a criterion amplitude. The threshold value in the original circuit was in the order of 100 mV, but, since the EEG power-

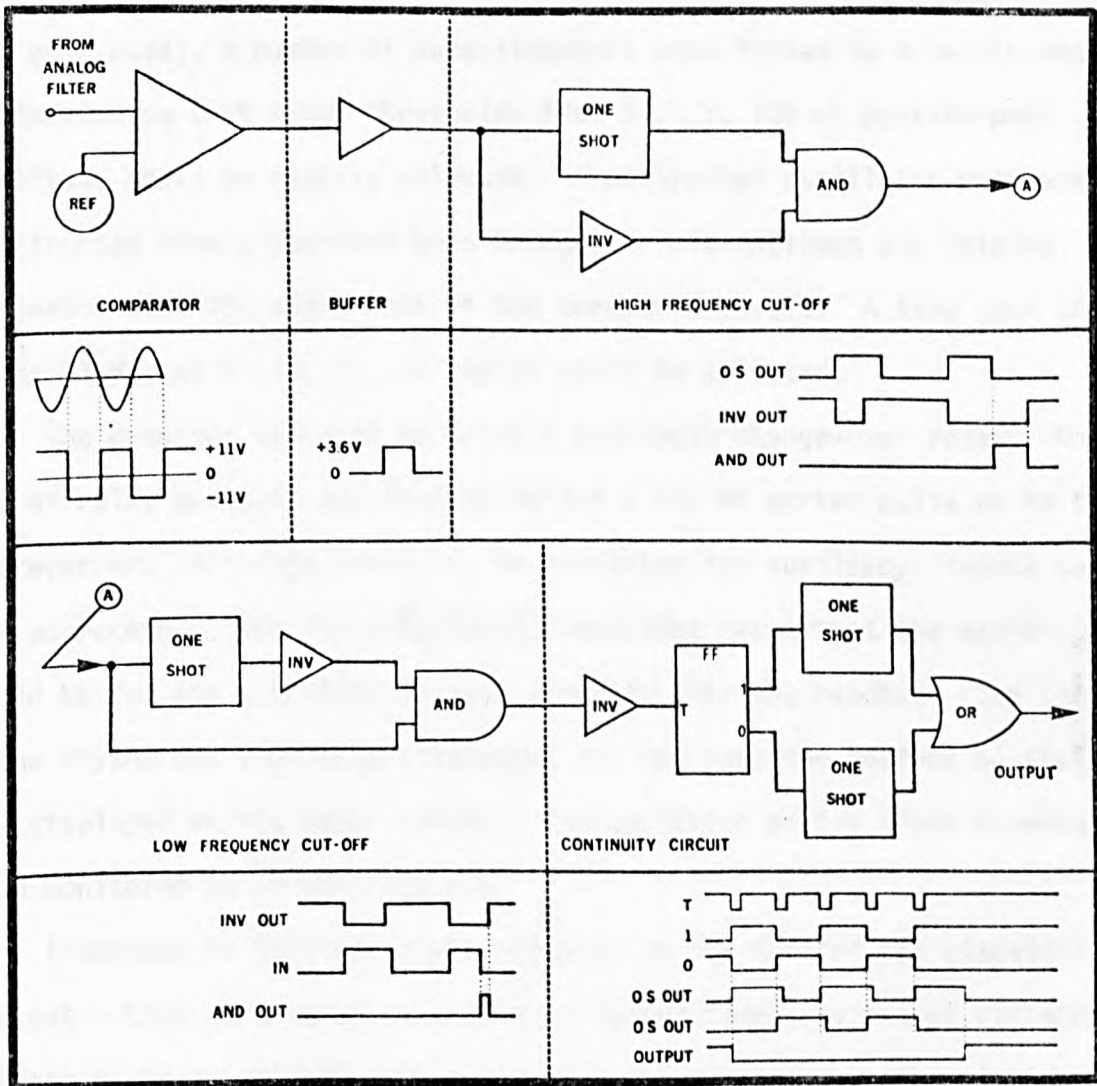


Figure 3.2

Diagrammatic summary of the operation of the digital alpha detector. The filtered EEG signal, if it exceeds threshold amplitude as set by the reference voltage, is converted into a square wave train in which the period is determined by the zero-crossing points of the signal. Each positive pulse, corresponding to the half-wavelength interval, is then gated with the output of a monostable (one shot) set to half the period of the upper frequency limit (41.66 msec for a 12 Hz cutoff). If the input wave is below the upper frequency, a difference pulse is produced which is similarly gated with the output of another monostable that represents the maximum temporal difference between the upper and lower frequency limits. An output pulse indicates that the input signal lies within the selected frequency band. A continuous output for as long as the criterion frequency is present is maintained by a flip-flop and two monostables: each set to slightly overlap the full-wave period of the lowest frequency passed.

From Paskewitz (1971).

amplifiers produced more than 2 V for a 50 μ V signal at the headbox (at the gain used), a number of potentiometers were fitted to a switch and calibrated so that alpha thresholds from 5 μ V to 100 μ V peak-to-peak amplitude could be readily selected. A calibrated oscillator unit was constructed from a standard Wein Bridge circuit (Millman and Halkias, 1972) to enable accurate adjustment of the threshold levels. A sine wave of 10 Hz or 13 Hz, at 5, 10, 50, or 100 μ V could be selected.

The detector was used to drive a four-pole change-over relay. One set of relay contacts was used to switch a 100 mV marker pulse on to the penrecorder. Although there was no provision for auxiliary inputs to the EEG penrecorder, the facility for DC recording meant that the marker pulse could be fed via a resistive chain directly into the headbox. The filtered alpha rhythm was similarly attenuated and fed into the headbox so that it was displayed on the paper record. The operation of the alpha detector was also monitored on an oscilloscope.

I decided to quantify alpha activity in the form of the classical percent - time measure so that data collected from sessions of different lengths could be readily compared. First, I connected a digital timer (Colne Instruments, Model DT1) to operate directly from the alpha detector relay. However, the digital timer proved to be very sensitive to stray switching transients, even after filters were fitted to the power and input leads, with the result that the timer would sometimes reset itself during the course of measurement. Hence this timer was replaced by an electro-mechanical print-out counter that became available. The print-out counter (Sodeco, Model 532) was clocked with ten per second pulses from a pulse generator (Lyons, Model PG71N). These pulses were gated through the relay of the alpha detector to give a real time measure of alpha activity ($\text{sec} \times 10^{-1}$).

An interval timer was incorporated into the system to prevent

inaccuracies in the comparison of alpha scores during an experiment arising from delay and error in enabling the alpha processing circuits for the fixed trial period. A predetermined counter (Sodeco Model, 535), in the same unit as the print-out counter, was used for this purpose. One per second time-marker pulses generated by the penrecorder were externalised and amplified to operate the predetermined counter so that time intervals from 1-9999 seconds could be reliably established. When the predetermined number of seconds was reached, a six-pole relay was switched to block counter and feedback signals. The beginning and end of the time interval were also registered on the penrecorder by a pulse on the time-marker channel.

The print-out counter and interval timer were arranged so that the experimental session could be controlled automatically. This ruled out any opportunity for experimenter bias in control over trial periods, and had the additional advantage that I could easily carry out preliminary experiments using myself as the subject. The automation was achieved by means of interconnected relay circuitry so that at the end of each trial period the score on the alpha counter was printed out, the counter reset, and the predetermined counter recycled to start the next trial. The required switching delays were obtained by the use of transistorised time constant networks.

I decided to use auditory feedback to provide subjects with instantaneous information on the occurrence of alpha activity in the EEG, as this would allow feedback training in both eyes-open and eyes-closed conditions. I chose a low-volume 400 Hz triangular wave provided by a function generator (Wavetek, Model 112). The tone was amplified by a transistorised audio-mixer circuit and relayed to the subject through a pair of stereo headphones (Alpha, Model SDH-105).

The alpha feedback apparatus is pictured in Plate I. To the right of the photograph can be seen the Beckmann EEG amplifiers and penrecorder, to

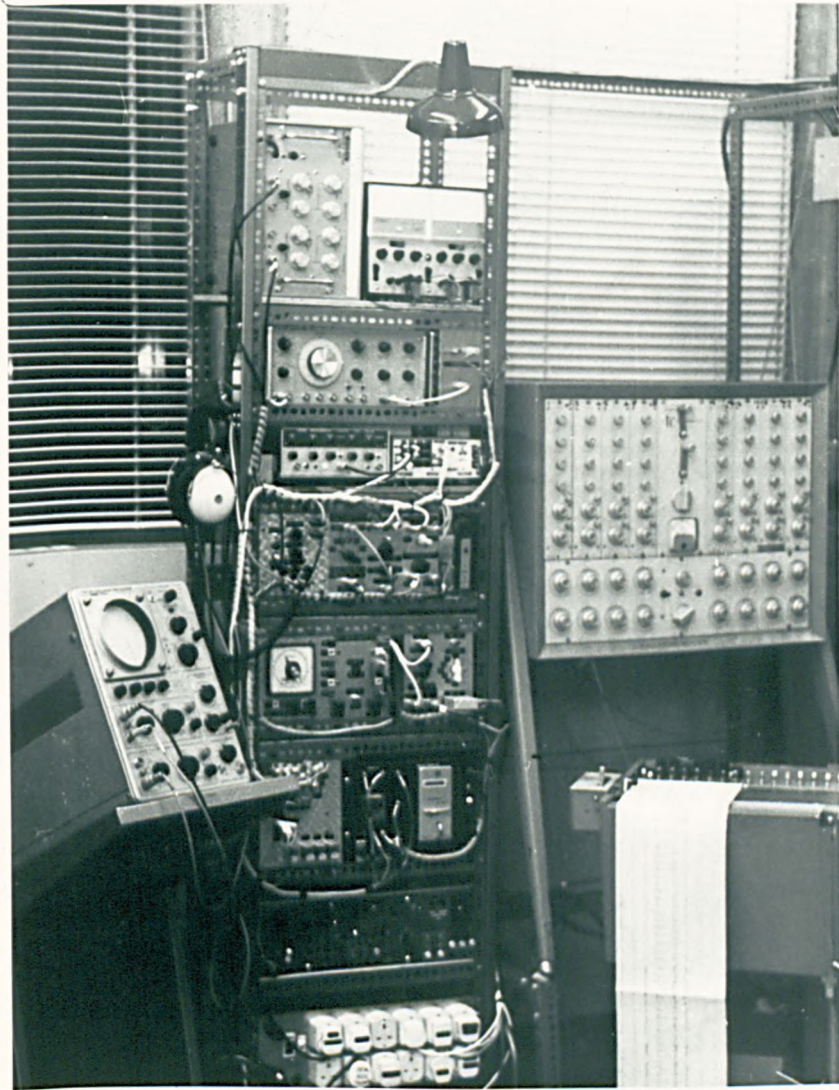


Plate I.

The original apparatus built by the author to investigate feedback training of the EEG alpha rhythm.

the left the oscilloscope. The rack in the centre contains the alpha processing equipment and power supplies. Connection cables from the rack were carried to an adjacent audiometric test chamber (Industrial Acoustics, Model 1204A), in which all the feedback training laboratory studies were carried out. This chamber consisted of a heavily sound-insulated and electrically shielded 9 ft x 9 ft room, illuminated by a single 15 watt bulb and forced-air ventilated. A comfortable reclining chair was provided for the subjects, fitted with a headrest to reduce muscle artifact and eye movement (Milnarich, 1958). A microphone was connected to the audio-mixer to enable the experimenter to communicate with the subject via the headphones.

3:1:3 Pilot Study

A small pilot study was carried out to enable the operation of the complete alpha detection and feedback system to be thoroughly tested, and the various recording and detection parameters adjusted.

First, the electrode positions for recording the alpha rhythm were selected. Previous workers in the field used alpha activity from a considerable variety of derivations, both referential and bipolar, over the central, parietal, temporal, or occipital regions of either hemisphere, or along the midline. I decided to employ referential recording from occiput to mastoid as I was concerned with maximising the amplitude function of alpha, rather than with topographical or phase relationships (Cooper et al. 1969). I also examined various bipolar channels during some preliminary EEG recordings, but the simple referential recording both proved to be the most convenient, and often exhibited the highest amplitude alpha activity. Electrodes were sited on the occiput at points O_1 and O_2 (measured by reference to theinion, nasion, and pre-auricular points) according to the *International Ten-Twenty System* (Jasper 1958, see Figure 4.1). The right occipital electrode was selected for alpha feedback training since, although

Mulholland (1968) found no evidence that the effect of feedback differed depending on whether right or left EEG is processed, it has been noted that there is a tendency for more alpha to be exhibited on the right side for the population in general (Hill and Parr, 1963; Kiloh et al., 1972). A reference electrode was placed over the right mastoid bone, and a ground electrode at the vertex.

Electrode sites were marked with a chinagraph pencil and then cleaned thoroughly by abrasion with cotton wadding soaked in surgical spirit. Silver / silver-chloride 9 mm disc electrodes (Standard Laboratory Equipment, Type B1/9) were attached with collodion and filled with sodium chloride electrolyte. A small impedance meter was constructed to measure the scalp-electrode contact, as DC meters can cause electrode polarization and hence attenuation and distortion of EEG activity (Hill and Parr, 1963). The epidermis was scraped away at the centre of the electrode with a notched, blunted needle to reduce the impedance below 5 K Ω so as to minimize interference from the mains-supply field (Cooper, et al., 1969).

In addition to the occipital EEG channels, two eye movement channels were recorded. This was for several reasons:

(1) To enable eyes-open and eyes-closed conditions to be monitored. The aqueous humour of the eye is electropositive to the vitreous humour; and, since the eyeballs flick upwards when the eyes are closed (Bell's phenomenon), eye closure and eye blinks are easily recorded.

(2) It has been reported that alpha rhythm is enhanced by extreme ocular deviation in some individuals (Bender, 1969; Fenwick and Walker, 1969; Mulholland, 1972, *see* 2:3:3).

(3) Slow rolling eye movements can provide an indication of the onset of an early sleep stage (Kooi, 1971).

The eye electrodes were placed on the orbit above each eye and referenced to the electrode on the right mastoid. The area was cleaned and abraded with surgical spirit and silver / silver-chloride disc electrodes

attached with strips of surgical tape (this method was found to be more satisfactory than the use of either collodion or adhesive discs). The electrodes were filled with sodium chloride electrolyte.

The occipital EEG was recorded at the convenient gain of 50 μ V per centimetre. A time constant of 0.3 sec and high frequency filtering above 30 Hz were selected for the pre-amplifiers, since I was primarily concerned with optimising the clarity of alpha activity rather than the fidelity of recording over the entire EEG band. The eye movement channels were recorded with a time constant of 1.0 sec or DC-coupled amplifiers, at a reduced gain

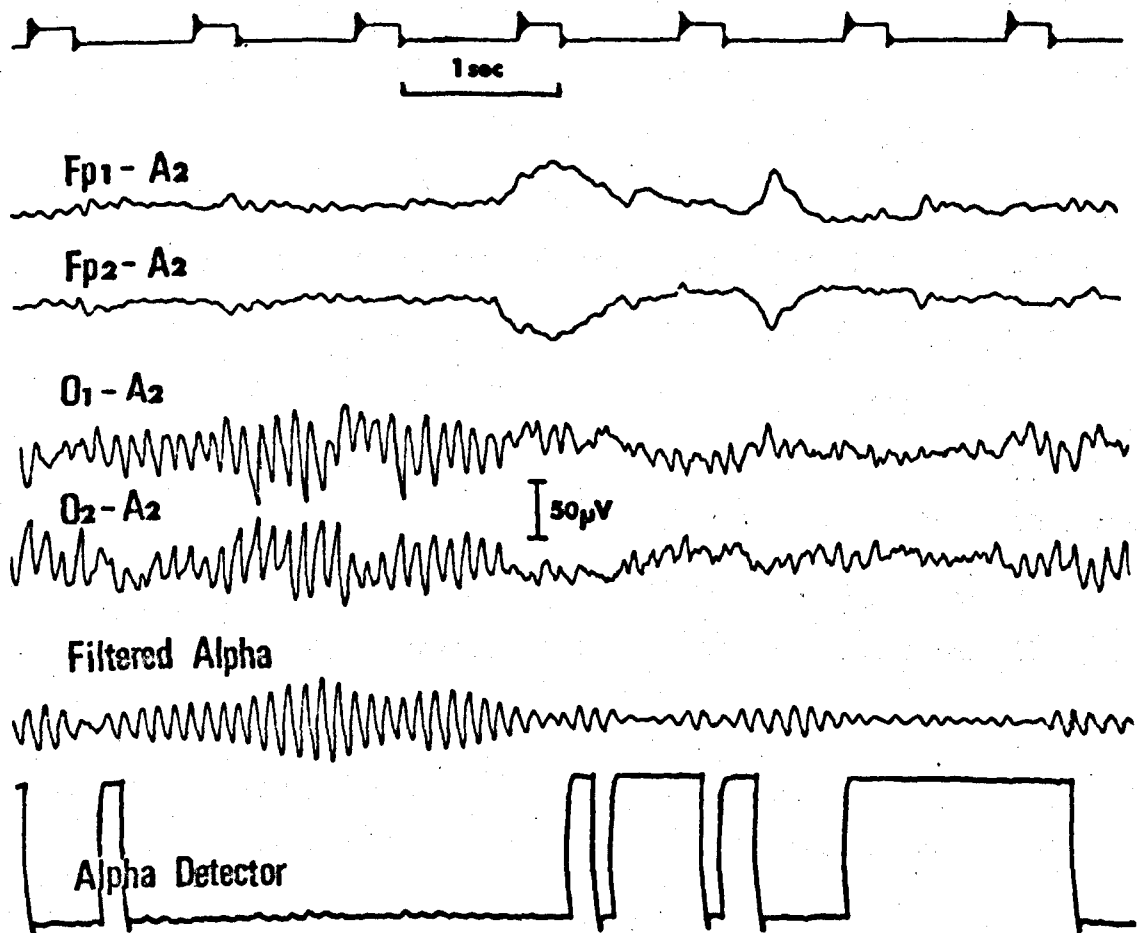


Figure 3.3

Penrecorder output showing the response of the 8-12 Hz band-pass filter and the digital alpha detector (a downward deflection indicating the presence of alpha activity above threshold level) to the right occipital EEG signal (O_2-A_2). Also shown are left occipital EEG (O_1-A_2), right (Fp_1-A_2) eye movement channels, and a 1 Hz time-marker signal. The phase reversal of the EEG and eye movement channels is introduced at the pre-amplifier stage.

of 250 μ V per centimeter.

An example of the paper record is provided in Figure 3.3. Spindle bursts of alpha rhythm can be seen in both the raw EEG and the filter channels, together with the corresponding step-function response of the digital alpha detector (set at a threshold amplitude of 25 μ V). Note also the delay, which is in the order of 300 msec, in the response of the alpha detection system. This results from the phase shift introduced by the band-pass filter, and the turn-on time of the detector relay. Previous workers have generally chosen criterion levels for the detection of alpha activity in the range of 10-20 μ V (Brown, 1971; Nowlis and Kamiya, 1970; Paskewitz and Orne, 1973). It soon became apparent during the course of the pilot study however, that the use of a fixed criterion amplitude for all subjects meant that, on the one hand, high alpha subjects received almost continuous feedback and, on the other, that subjects with flat EEGs or low alpha densities received virtually none. Therefore neither type is afforded with much opportunity for the trained enhancement of alpha activity. An illustration of such differences in alpha activity is given in Figure 3.4.

Eight subjects were used in the pilot study. They were each given ten three-minute feedback training trials, alternating between alpha enhancement and alpha suppression. For the enhancement trials the subjects were asked to attempt to keep the feedback tone on as much as possible; and for the suppression trials to keep the tone off as much as possible. They were instructed to keep their eyes closed throughout the course of the session.

One difficulty soon encountered was that it became evident that subjects' scores would sometimes drop dramatically. It was discovered that the digital alpha detector was responsible for this phenomenon, since it was extremely sensitive to changes in the DC level of the input signal. By a fractional adjustment of the DC offset trim on the Barr and Stroud

band-pass filter the scores could be made to sharply increase once more. On examination, it was found that when the input signal was near to threshold level a DC bias of as little as 5 mV could prevent alpha from being detected, as it gives rise to a non-symmetrical square wave train. This problem was further exacerbated by a gradual DC drift at the output of the band-pass filter occurring over the course of several minutes. The input impedance of the alpha detector circuit is in the order of 10 K Ω , hence for it to be AC-coupled to the band-pass filter, a large capacitor is necessary so as to prevent attenuation of the alpha band¹. Therefore I

¹Time constant (T secs) = resistance (R Ohms) x capacitance (C Farads). 3 dB attenuation occurs at a frequency given by $1/6.3 T$. A capacitor in the region of 3-4 μF would be required in this instance.

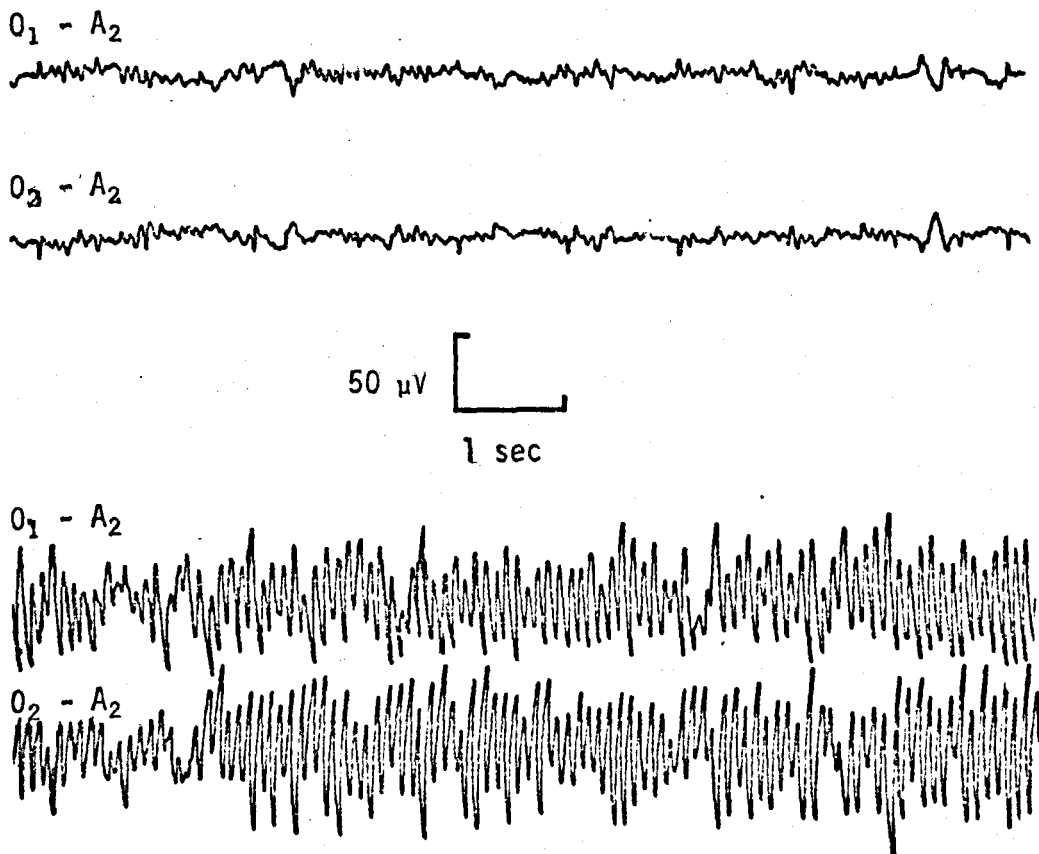


Figure 3.4

Occipital EEG recorded at the same gain from two subjects, illustrating the wide differences in amplitude of the alpha rhythm that were observed.

built a voltage-follower buffer amplifier to which the band-pass filter was capacitively coupled. A FET operational amplifier with low DC drift characteristics was chosen. This successfully prevented any further aberrant DC influences on the digital alpha detector.

Owing to the deleterious effects of DC drift (and also because the alpha thresholds were varied during sessions to study the effect on the level of feedback), the alpha scores from the pilot study were not suitable for formal analysis. Nevertheless, it appeared that the subjects were able to exert some influence over the occurrence of the feedback tone. One subject apparently increased alpha density from 20% to 39% within thirty minutes of feedback training. However, since considerable muscle activity was often evident in his EEG recording, as can be seen in Figure 3.5, much of this increase was probably artifactual (see 4:1:4).

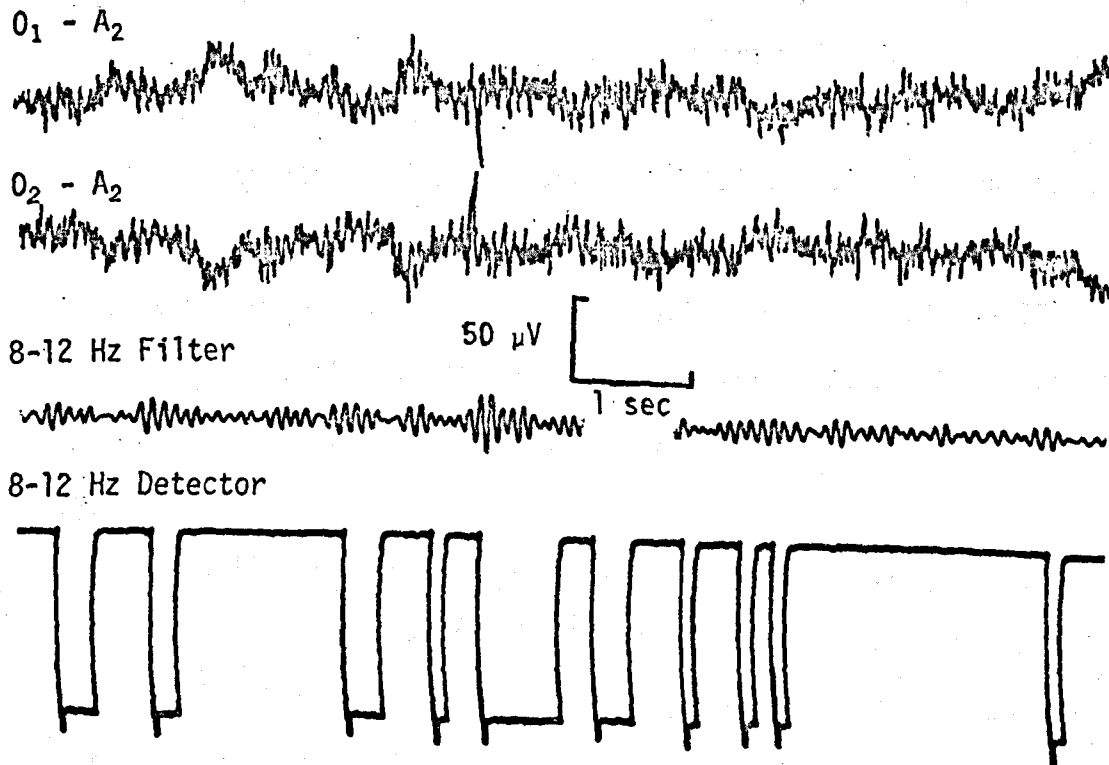


Figure 3.5

EEG trace from a subject who demonstrated considerable enhancement of alpha scores. Much high frequency muscle artifact is apparent in the recording, which might have been responsible for spurious signals at the output of the analogue filter and consequent false triggering of the alpha detector.

3:2:1 Introduction

The principal aim of this investigation was to replicate the results of the early alpha feedback workers who reported that feedback training enabled subjects to show a significant difference in percentage alpha between trials in which they attempted to enhance alpha, and trials in which they attempted to suppress alpha (Hart, 1967; Kamiya, 1967; 1968; 1969; Nowlis and Kamiya 1970).

A second aim was to examine changes in percentage alpha during training in relation to the baseline levels of alpha, since the early studies have been criticised on the ground that the impressive differences reported between enhance-alpha and suppress-alpha trials are the result of unidirectional control. It has been argued that, although the studies demonstrate that alpha activity can be voluntarily modified, they do not prove that subjects are able to exclude factors which inhibit alpha activity to the same extent that they are able to utilize these factors to block alpha activity (Lynch and Paskewitz, 1971; Paskewitz and Orne, 1973; Peper and Mulholland, 1970; Walsh, 1972). Moreover, it has been proposed that alpha activity "never goes above baseline level" (Walsh, 1972; 1974) and that "significant increases in alpha densities occur when circumstances have lowered alpha densities below optimal baseline levels" (Paskewitz and Orne 1973).

If a baseline measure is obtained to give a more credible indication of the ability of subjects to both enhance and suppress alpha then it becomes difficult to establish optimal baseline conditions from which to measure, since nearly all baseline measures are said to be to some extent contaminated by influences such as apprehension or the novelty of the situation (Lynch and Paskewitz, 1971). Furthermore, any attempt to follow

the spontaneous variation in baseline alpha by the use of rest periods interspersed throughout the training trials could be confounded by the carry-over effects from the feedback training, or by changes in arousal and motivation inherent in defining the task as a "rest" period (Paskewitz and Orne, 1973). Therefore, although in a number of studies the tabulated data shows that at least some of the subjects have exhibited increases in alpha above their eyes-closed baseline densities (Hart, 1967; Nowlis and Kamiya, 1970; Peper, 1970; Peper and Mulholland, 1970), often the baseline measure was one obtained from an initial recording and thus it could be argued that it was suppressed below optimum by situational effects.

It has not been empirically established, however, that baseline alpha measures are either suppressed initially, or influenced by preceding feedback trials. The experimental data is conflicting: sometimes a spontaneous rise in baseline alpha densities is reported to accompany alpha feedback training (Beatty and Kornfield, 1972; Strayer et al., 1973), sometimes a decrease (Beatty, 1971), and sometimes no change (Hart, 1967). Therefore I sought to investigate spontaneous changes in baseline alpha occurring during the course of this feedback study by taking several baseline measures, and also to examine these measures for possible carry-over effects from the alpha enhancement and suppression tasks.

3:2:2 Method

Design and Procedure

The experiment was based on that described by Kamiya (1969), but a number of improvements were made in the design.

(1) Alpha suppression and alpha enhancement trial blocks were alternated according to a split-half design so as to balance out any order effects, and any contamination of the baseline measures by carry-over effects.

(2) Longer trial blocks were used than by Kamiya, since subjects in the pilot study had complained of the difficulty of frequently alternating between alpha enhancement and alpha suppression tasks.

(3) Subjects were asked at the end of each trial block to recount the strategies that they had employed in their attempts to regulate the tone, rather than at the end of the session, so that a close record of the correspondence between alpha score and introspective recall was obtained.

(4) The subjects were informed of their alpha scores at the end of each trial as a supplement to the feedback tone, since there is some evidence that subjects who are provided with such augmented feedback learn faster than those who are merely given contingent feedback (Brener and Hothersall 1966; Nowlis and Kamiya, 1970).

Subjects were first informed of the general nature of the experiment and presented with written instructions as follows:

In order to assist you to recognise and control your alpha activity, the alpha rhythm will be fed back to you by means of a tone signal which is switched off whenever alpha occurs. You should try to control the presence of the tone as much as you can by whatever means seems to work best. This is an individual matter which only you can discover. However, relaxation, whilst remaining alert, may assist you to turn the tone off; and visual imagery or mental effort may help you to turn the tone on.

The experiment will begin with a one-minute rest period in which the baseline level of your alpha rhythm is determined. There will then be four alpha feedback trial blocks, each one lasting seven minutes, followed by another one-minute rest period. The experimenter will inform you over the intercom when each trial block begins and ends; and will instruct you when you are to try to keep the tone off, and when you are to try to keep the tone on. You will also be told your alpha scores at one-minute intervals during each trial block.

During rest periods there will be no feedback tone. Do not try to control your alpha activity in any way during these periods, just sit quietly and wait for the next instructions. Please do not move your arms, legs, or head about, since any movement can easily interfere with the equipment. Once you are told the experiment is to begin, please keep your eyes closed at all times until the end. This is extremely important.

Twenty volunteer undergraduate and postgraduate students served as subjects. The split-half design was such that ten of the subjects began with a seven-minute alpha suppression trial block then, after the subject

had been asked to report methods used in attempting to control the feedback tone, a baseline measure was taken. This was then followed by a seven-minute enhancement trial block. After another report and baseline measure, the sequence was repeated. The other ten subjects began with the alpha enhancement task and followed a similar pattern. A preliminary one-minute baseline recording was taken for both groups of subjects.

Apparatus

The apparatus for this study was the modified alpha detection and feedback system as described above. A standard criterion threshold for alpha amplitude was not adopted in this study because of the problem of gross differences in alpha amplitude, and therefore in the amount of feedback received, that was found in the pilot study. Instead, the threshold amplitude was set at 10 μV for those subjects who exhibited low alpha, 25 μV for those with high alpha, and 15 μV for those intermediate. The appropriate threshold was selected for each subject before the session began so as to some extent optimise the density of feedback that they received.

Since some of the subjects in the pilot study had complained that they found that the feedback tone was distracting when they were attempting to maintain alpha, the feedback contingency was switched so that alpha rhythm was indicated by the absence of the tone, although Hord and Barber (1971) reported that either tone-on or tone-off is equally effective as a feedback signal.

The same preparation and recording techniques were used as described previously.

3:2:3 Results

The mean data of the twenty subjects for the enhance-alpha, suppress-alpha and baseline trials is presented in Figure 3.6. It is clear that there is a very marked difference between the enhance-alpha and suppress-

alpha scores, especially in the second seven-minute trial block. It can also be seen that the suppress-alpha scores are far more reduced below the baseline scores than the enhance-alpha scores are raised above their corresponding baseline measures. The highest enhance-alpha trial score and the highest baseline trial score for each subject are listed in Table 3.1.

Before carrying out statistical comparisons on the data, the shapes of the distributions of the measures were examined to determine whether parametric statistics were appropriate. The skewness and kurtosis of the distributions were calculated from moments about the mean. The data was generally platykurtic (mean enhance: $g^2 = -1.56$; mean suppress: $g^2 = -1.63$; mean baseline: $g^2 = -1.59$), but only slightly skewed (mean enhance: $g^1 = -0.027$; mean suppress: $g^1 = 0.08$; mean baseline: $g^1 = -0.23$).

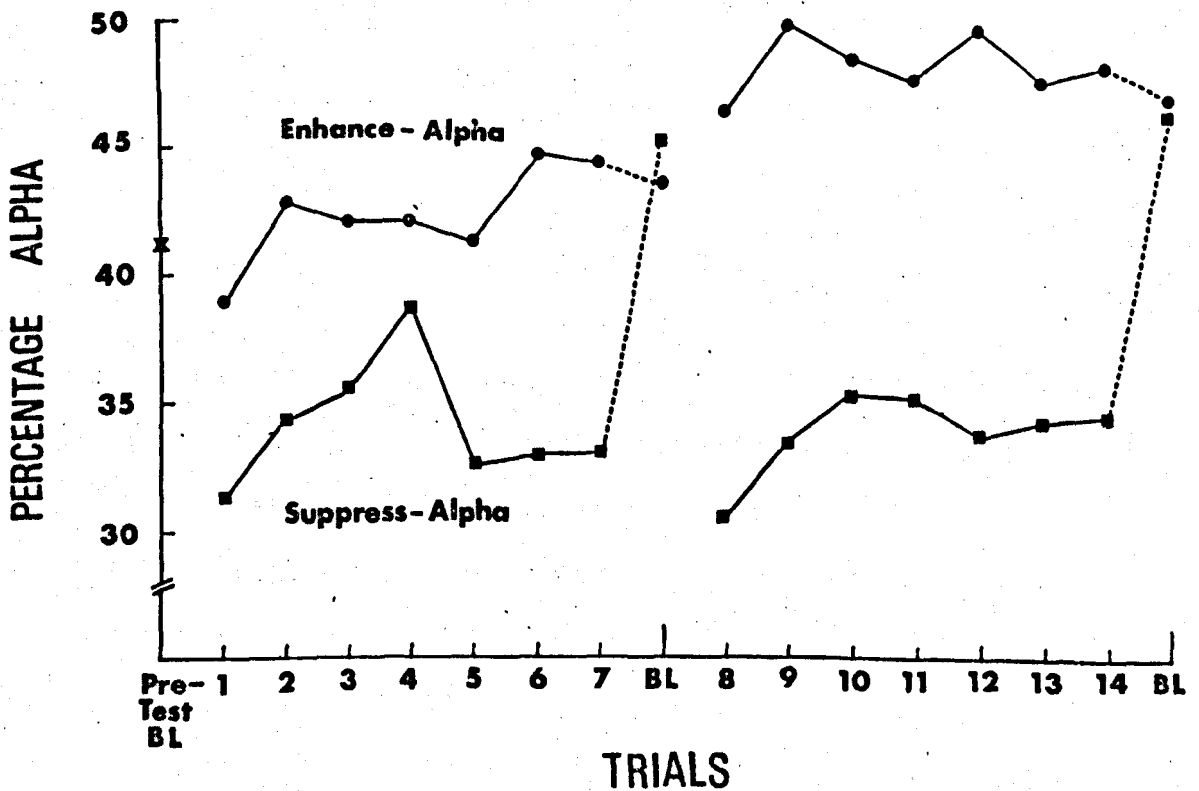


Figure 3.6

Mean percentage alpha per minute of the twenty subjects for the enhance-alpha and two suppress-alpha trial blocks within the split-half design. The mean initial baseline and the baseline recorded at the end of each trial block are also plotted.

Table 3.1. Comparison of highest enhance-alpha trial score and highest baseline trial score for each subject.

Subject	Highest baseline alpha score	Highest enhance alpha score	Percentage increase
S.H.	62.0	56.5	-8.9
E.K.	5.3	5.1	-3.8
R.W.	68.3	64.3	-5.8
H.A.	84.1	81.0	-3.7
A.E.	84.5	78.3	-7.3
K.S.	78.6	88.5	+12.6
A.R.	39.3	32.8	-16.5
J.G.	82.8	81.6	-1.4
A.B.	59.3	69.1	+16.5
I.S.	46.6	35.8	-23.2
C.B.	21.6	50.6	+134.3
N.B.	28.0	23.1	-17.5
R.R.	14.6	15.1	+3.4
C.J.	76.3	80.1	-5.0
R.H.	79.6	78.6	-1.2
C.C.	34.5	33.5	-3.0
R.Q.	25.1	27.8	+10.8
A.A.	84.5	82.8	-2.0
R.P.	78.3	81.8	+4.5
J.S.	58.1	57.5	-0.7
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Mean	55.6	56.2	+4.6
St. Dev.	26.5	26.3	32.1

Accordingly, I decided that it would be justified to use parametric analysis since it has been demonstrated that this is robust against even quite marked violations of the underlying assumptions with sample sizes of as little as fifteen: only in extreme cases, such as those which involve distributions differing markedly in skewness, are larger sample sizes or non-parametric statistics necessitated (Boneau, 1960).

The enhance-alpha and suppress-alpha scores across the trials were compared by a two-way analysis of variance. Since the experimental design involved repeated measures on both factors, a factorial block analysis was employed (Kirk, 1968) with one factor being Task and the other being Trials.

The main effect for Task was extremely significant ($F(1,513) = 146.3$, $p < 0.001$), but the main effect for Trials just failed to reach significance ($F(13,513) = 1.72$, $p > 0.05$), and there was no significant Task x Trials interaction ($F(13,513) = 1.18$, $p > 0.05$). To enable a closer analysis of the pattern of changes within the enhance-alpha and suppress-alpha trials the data for these were further examined individually.

There was no significant difference between the mean enhance-alpha scores and the mean baseline scores ($t = 0.2$, $df = 19$, $p > 0.5$)¹. A one-way analysis of variance of the enhance-alpha scores, however, produced a very significant Trials effect ($F(13,247) = 3.43$, $p < 0.005$). A test for linear trend² was very significant ($F(1,247) = 36.4$, $p < 0.0001$), confirming that there was a progressive increase in alpha across trials, as indicated by Figure 3.6.

In contrast, the difference between the mean suppress-alpha and the mean baseline scores was highly significant ($t = 3.79$, $df = 19$, $p < 0.01$), but a one-way analysis of variance of the suppress-alpha scores was non-significant ($F(13,247) = 0.78$, $p > 0.1$). This perhaps reflects a one-trial learning paradigm in which the subjects quickly demonstrated a suppression of alpha during the first one-minute period of each suppress-alpha trial block, as is evident in Figure 3.6. The subjects in fact reported that they found it relatively easy to learn how to suppress alpha but difficult to maintain the necessary effort.

The five baseline measures were compared by a one-way analysis of variance (with repeated measures). There was no significant effect ($F(4,76) = 1.121$, $p > 0.05$); neither did the initial pretest baselines

¹The two-tailed alternative is adopted for all comparisons between means in this thesis, unless otherwise stated.

²By the method of orthogonal polynomials (Winer, 1962).

differ significantly from the final baselines when compared using a t-test for correlated samples ($t = 1.14$, $df = 19$, $p > 0.2$). Thus no significant spontaneous increase in the baseline levels of alpha occurred during the session.

A comparison of the enhance-alpha scores and the suppress-alpha scores of the ten subjects in each group of the split-half design, using a t-test for independent samples, produced no significant differences ($t = 0.46$, $df = 19$, $p > 0.5$), indicating that no order effects occurred. Similarly, the baseline measures following enhance-alpha trial blocks were compared with the baseline measures following suppress-alpha trial blocks ($t = 1.5$, $df = 39$, $p > 0.1$) and no significant difference found. This demonstrates that the baseline measures were not influenced by carry-over effects from the trial periods, as Kamiya (1969) suggests.

A summary of the strategies that each subject reported using in their efforts to control the feedback tone, and hence their alpha rhythm, is provided in Table 3.2. Also listed are the ratios of the mean enhance-alpha and suppress-alpha scores to mean baseline for each subject, in order that their reports can be judged in relation to their actual success.

It can be seen that the mental state most generally reported for producing alpha activity is a relaxed, effortless one. Similarly, fixed concentration, visual imagery and tracking of visual phosphenes were often reported to be the most effective techniques for suppressing alpha, as would be anticipated. Three subjects report using eye-elevation to facilitate alpha, but there was no consistent evidence in the records of any of the subjects of increased alpha time-locked to eye movement, which is in accordance with the view that forced ocular deviation is necessary to produce alpha enhancement (Fenwick and Walker, 1969).

Table 3.2. Synopsis of the strategies that each subject reported using to turn off the feedback tone (enhance alpha) and to turn on the feedback tone (suppress alpha). The ratio of the mean enhance-alpha trial scores to mean baseline scores, and of the mean baseline to mean suppress-alpha trial scores are also listed for each subject.

<u>SUBJECT</u>	<u>SCORE RATIO TO BASELINE (E/BL)</u>	<u>METHOD REPORTED FOR CONTROLLING ALPHA RHYTHM</u>
<u>ALPHA ENHANCEMENT</u>		
S.H.	1.1	Concentrating on sound of breathing. Attempting to keep attention away from visual Imagery.
E.K.	1.0	Flicking eyes up and down.
C.B.	2.4	Feeling tense and concentrating on an image of violent motion.
N.B.	0.8	Mentally practising multiplication tables as fast as possible.
R.W.	0.9	Attempting to keep the mind blank.
R.R.	1.1	Imagining nature scenes such as fields of corn.
C.J.	1.1	Daydreaming of horse-riding and jumping on a bed of feathers.
H.A.	0.9	Thinking of feeling very relaxed.
R.H.	1.0	Daydreaming and trying to ignore feedback tone.
A.E.	1.0	Relaxing, slow breathing and feeling pleased when tone goes off.
C.C.	0.7	Relaxing, deep breathing and concentrating on feedback tone.
K.S.	0.9	Relaxing, focusing on the top of the head and keeping the mind blank.
R.Q.	0.9	Attempting to ignore experiment and induce a state of reverie.
A.R.	0.9	Attempting to ignore feedback tone.
A.A.	1.0	Thinking about very pleasant situations.
J.G.	1.1	Attempting to keep the mind blank by listening to sound of breathing.
R.P.	1.1	Letting the mind drift. Looking upwards and to the left.
J.S.	1.0	Looking upwards with both eyes as much as possible.
A.B.	1.1	Attempting to hold the mind in the same state as when alpha is indicated as being present.
I.S.	1.5	Reverie and feeling amused.
<u>ALPHA SUPPRESSION</u>		
<u>(BL/S)</u>		
S.H.	2.4	Fixating on visual phosphenes.
E.K.	2.4	Relaxing and keeping the mind blank.
C.B.	1.9	Crossing the eyes and looking upwards.
N.B.	1.1	Alternate relaxation and concentration.
R.W.	1.2	Mental arithmetic and counting.
R.R.	0.8	Thinking of noisy and active scenes.
C.J.	1.1	Recalling unpleasant situations. Fixating on visual phosphenes.
H.A.	1.4	Attempting to 'look' through the eyelids.
R.H.	1.0	Concentrating on the feedback tone.
A.E.	1.2	Visual Imagery.
C.C.	4.0	Thinking of frightening and stressful situations. Following visual phosphenes.
K.S.	1.8	Crossing eyes and fixating on visual phosphenes.
R.Q.	2.3	Intense concentration. Forced accommodation of eyes. Holding breath.
A.A.	2.0	Imagery and daydreaming.
J.G.	1.2	Visualising shapes.
R.P.	1.2	Crossing eyes and looking upward.
J.S.	2.1	Focusing both eyes on a spot in the visual field.
A.B.	1.0	Tracking visual phosphenes.
I.S.	2.9	Maintaining fixed concentration on the feedback tone.
A.R.	1.5	Mental arithmetic and counting.

3:2:4 Discussion

Both the graphic and the statistical analyses establish that the subjects were able to effectively modify their alpha densities during feedback training. Thus the findings of Kamiya (1969), and of other early workers were replicated. Both analyses also indicate that alpha control is strongly undirectional: i.e. subjects can quickly learn to suppress their alpha activity below baseline levels, but the enhancement of alpha is much more difficult to achieve. This was, in fact, noted by many of the subjects.

Furthermore, the results of this study indicate that alpha for the group was not significantly enhanced above baseline levels. This appears to confirm the hypothesis (Paskewitz and Orne, 1973; Walsh, 1974) that alpha feedback training does not lead to alpha levels exceeding those seen under optimum resting conditions. However, a closer examination of the data reveals gross inter-subject differences in performance that have confounded the analysis of the group data.

In Table 3.1 it can be seen that, although the mean increment for the group is only 4.5% above baseline, for six of the twenty subjects the highest enhance alpha score exceeded the highest baseline score - the mean increment for these six being 30.5%. Similarly, Plotkin (1976b) found that integrated alpha scores during enhance trials were only "about 8% above the baseline measure, but that there were sixteen individuals whose highest enhance scores were between 25% and 74% above baseline".

Such individual performances, including an apparent enhancement of 134% in the case of subject (S.H.) in this present study, are totally obscured within the analysis of group mean data. It is evident from Table 3.1 that this is because the highest enhance alpha scores were suppressed below baseline scores for the majority of the subjects.

The initial depression of alpha levels shown by most subjects is likely to be a consequence of their efforts to gain control over the feedback tone.

As Peper (1972) has noted, "The major difficulty in these studies is that the subjects are unable to stop trying". Similarly, Travis (1937), in an early study of the alpha-rhythm, documented that, "Any form of mental effort such as attempts to refrain from thinking, to ignore external and internal stimuli, to keep from falling asleep, or to relax, can have an attenuating effect on alpha". Many other studies have emphasised the need for a passive state of unfocused attention in order to augment alpha levels (Brown, 1970; Hart, 1967; Kamiya, 1969; Lynch and Paskewitz, 1971), although this may not be true for all individuals (see 3:3:1). The effect of the feedback training paradigm, in which the subject is provided with sensory feedback information, can thus be to cause a drop in alpha below baseline levels. For some subjects alpha can rise above baseline as they become less problem-oriented, but for others the demand characteristics of the task continue to produce an inappropriate response, *viz.* an effect opposing that sought by training.

This inappropriate response to alpha feedback is a major confounding influence on the interpretation of the data. Nowlis and Kamiya (1970), for example, report that in the eyes-closed condition only five of their sixteen subjects showed appropriate change for both enhance alpha and suppress alpha trials: seven subjects showed increases for both trials, whilst four showed decreases for both. Hart (1967) and Kuhlman and Klieger (1975) similarly report that the performance of individual subjects in response to feedback varied greatly; and Hart notes that, "The differences between conditions are overridden by the variability within each group".

It is clear that the interpretation of pooled data as to the efficacy of feedback training, and in particular whether it is possible to train alpha to exceed optimum baseline conditions, is meaningless when it is confounded by high inter-subject variance, especially when there are subjects who exhibit an inappropriate response. With the small samples that are

usually employed in this work, this inter-subject variance markedly increases the likelihood of a *Type II* statistical error. It is apparent that, even with the short time of training in this present study, increases of 10% or more above the highest baseline measure were obtained for four of the subjects. It is not suggested that the highest baseline measures here represent optimum baseline levels (since this was the first session for the subjects, and they were not in total darkness, this is highly unlikely). However, in the face of such individual differences in performance, categorical statements such as: "It now appears that alpha density never goes above optimal baseline (eyes-closed resting in the dark) during feedback training" (Walsh, 1974); or, "Subjects can acquire volitional control over alpha activity only under conditions which normally lead to decreased densities" (Paskewitz and Orne, 1973), made on the basis of pooled data with comparatively small population samples, are totally unfounded.

3:3:1 Introduction

The study described above indicated that there are considerable individual differences in the type of behavioural strategies that subjects feel to be associated with alpha activity (see Table 3.2). Furthermore, although almost since the time of Berger alpha activity has been said to be generally associated with a state of relaxation (Milnarich, 1958), there were a few subjects who indicated that they produced more alpha during intense concentration, tension, or the performance of cognitive tasks. There are many other similar cases to be found in the literature.

Plotkin (1976a) found that among the strategies reported by subjects who were successful at significantly enhancing their alpha activity above baseline level are "multiplication problems", "counting", "maths equations", and "feelings of anger, fear, paranoia, frustration or sadness". Plotkin also notes that, "Four persons did explicitly mention relaxation as a suppression strategy". Peper and Mulholland (1970) similarly indicate that half of their eight subjects maintained alpha by "being tense" or "concentrating", and decreased alpha by relaxing. Also Brown (1970), in her study of alpha training using visual feedback, reports that, "Half of the subjects either felt relaxed by the experience or attempted actively to relax", but the use of this technique to maintain a selected subjective state was *not* necessarily accompanied by the higher levels of alpha abundance for the subject group.

Several workers have reported that alpha is enhanced during tension or stress for some individuals. Lemere (1936), in one of the first studies on individual differences in the ability to produce alpha, reports that, "Several reliable subjects stated that they were the most tense... during the trial that showed the "best" alpha waves. There was also a general

tendency for the waves to get "worse" with subsequent trials although relaxation became better". Orne and Wilson (1976) found that, although there was no group mean change in alpha density during the anticipation of electric shock, striking individual differences were hidden within the group data. Thus, whilst one subject decreased alpha by 70% below baseline level, there were two subjects who exhibited an increase of 40%. Similar individual differences in the response of alpha to induced anxiety have been reported by Simonova (1968).

In addition, there are reports of various problem-solving tasks producing a facilitation of alpha for some people in contrast to the general trend (Mundy-Castle, 1957; Werre, 1957; Lorens and Darrow, 1962). Kreitman and Shaw (1965), in a specific investigation of the enhancement of alpha by task performance, report that the average change in percentage alpha during mental computation for a group of sixteen subjects was -10%; but that an enhancement of alpha occurred for six of the subjects, with a mean change of +71%! Orne and Wilson (1976) recently found that subjects could be classified as "blockers" or "augmenters" on the basis of their alpha change pattern. Moreover, the individual patterns of alpha response appear to be remarkably stable over tasks and time.

In view of these individual differences in the relation of alpha to the level of activation, I decided to carry out a small study to determine whether simply asking subjects to relax or to tense themselves would produce different alpha change patterns across subjects.

3:3:2 Method

Occipital EEG was recorded from sixteen subjects during both relaxation trials and trials in which they were instructed to concentrate on making themselves feel aroused and tense (but to refrain from making any muscular movement). These tasks were carried out in both the eyes-closed

and eyes-open conditions. Each trial was in the order of three minutes. Percent-time alpha was measured as described above and, in order to obtain some indication of the degree of arousal during the activation trials, ECG and EMG were recorded. ECG was taken from a pair of stainless steel plates attached to the wrists by rubber straps. The gain was reduced to 250 μ V per centimetre, with high (30 Hz) and low (0.03 sec time constant) filtering. The standard neck lead was chosen for EMG (semispinalis capitis muscle and splenius capitis muscle) as described by Lippold (1967). This was recorded at a gain of 50 μ V per centimetre with no high frequency filtering, but a short time constant (0.03 sec) so as to prevent the pre-amplifier from being "blocked" by head movement. No equipment was available at this time for quantifying the EMG signal.

3:3:3 Results and Discussion

The change in alpha between the relaxation and activation trials for each of the sixteen subjects are listed in Table 3.3.

It can be seen that many subjects exhibited an increase in alpha during the activation trials. Moreover, the group mean percentage alpha was significantly greater for the activation trials than for the relaxation trials in the eyes-open condition ($t = 4.15$, $df = 15$, $p < 0.001$), but not in the eyes-closed condition ($t = 1.99$, $df = 15$, $p > 0.05$). The heart rates recorded during the relaxation and activation trials were also compared and found to be significantly higher for the activation trials in both the eyes-open ($t = 3.89$, $df = 15$, $p < 0.001$), and the eyes-closed ($t = 2.87$, $df = 15$, $p < 0.01$) conditions (1-tailed tests). The heart rates for the eyes-open and eyes-closed activation trials did not differ significantly ($t = 0.31$, $df = 15$, $p > 0.5$); neither did those for the two relaxation trials ($t = 1.38$, $df = 15$, $p > 0.1$). The changes in heart rate during activation trials thus

Table 3.3. Change in mean percentage alpha from relaxation to activation trials for eyes-open and eyes-closed conditions.

<u>Subject</u>	<u>Eyes-open</u>	<u>Eyes-closed</u>
S1	+2.6	-2.1
S2	+16.5	-14.2
S3	+17.0	+3.0
S4	+12.6	+10.0
S5	+38.1	+31.3
S6	+7.1	+3.5
S7	+20.3	+2.7
S8	+3.7	+2.0
S9	+9.0	+3.1
S10	-8.4	-0.3
S11	+33.3	-0.1
S12	+9.2	-0.6
S13	+7.1	+24.1
S14	0.0	-2.7
S15	+24.9	0.0
S16	+10.6	+21.5
---	-----	-----
Mean	+12.7	+5.1
St. Dev.	11.7	11.0

confirm that an increase in the overall level of psychophysiological arousal did in fact occur. Similarly, a substantial enhancement of the amplitude of the EMG trace was typically observed during the activation trials.

The greater enhancement of alpha with activation in the eyes-open condition than in the eyes-closed, even though the general increase in arousal appears to be the same, therefore argues against the most immediate explanation of this phenomenon, which is that the results were due to muscle artifact and in-band noise causing spurious triggering of the alpha detector. It is most improbable that such artifact would occur consistently across subjects in the eyes-open trials. Moreover, the EEG records provided no indication that artifact was enhanced during the eyes-open activation trials.

Most work on the paradoxical enhancement of alpha during arousal has been concerned with the appearance of alpha evoked by stimulation during states of hypoarousal (Morrell, 1966). This phenomenon led to the proposal that the interaction between alpha and arousal is not monotonic, but follows a bell-shaped function, so that there is said to be an optimal level of arousal for alpha (Malmo, 1959; Mackworth, 1969). Hence it has been suggested that the increase in alpha that some subjects exhibit during activating tasks is a consequence of the subjects being drowsy (Peper and Mulholland, 1970; Hardt and Kamiya, 1976a). Since alpha feedback training commonly involves subjects being seated with their eyes closed, in a comfortable chair, in a quiet darkened room (as in the alpha feedback study described above), it is indeed quite probable that some subjects do become drowsy, and voluntarily aroused themselves to enhance alpha. However, in many of the other studies in which a paradoxical enhancement of alpha was found, there was no evidence that the subjects were underaroused (Kreitman and Shaw, 1965, *supra*). Similarly, in this present study, since the enhancement of alpha by arousal was more marked for the eyes-open condition than for the eyes-closed, it is unlikely that it reflected an alerting response.

Moreover, the concept of a bell-shaped interaction between alpha and arousal has been recently criticised by Plotkin (1976a) on the basis of the increasing evidence that oculomotor processing plays a very important role in the attenuation of alpha activity (Mulholland, 1972, *see* 2:3:3). Plotkin distinguishes between "behavioural arousal" and "oculomotor arousal", and argues that the desynchronization of occipital alpha is primarily associated with oculomotor activation whether or not there is a concomitant increase in behavioural arousal. Hence an explanation of the enhancement of alpha during the activation trials in this study might be that it results from some reduction in visual and oculomotor processing.

Thus it is possible that the concentration on the task of becoming aroused might lead to a tendency for defocusing in the eyes-open condition, and of refraining from trying to 'see in the dark' in the eyes-closed condition.

3:4:1 Introduction

Some of the subjects in the alpha feedback replication study reported that they found the feedback tone to be disturbing, even though the feedback system was arranged so that the tone was switched off whenever alpha activity occurred, and that this prevented them from enhancing their alpha scores. The sudden appearance of the feedback tone can have an alerting function. Moreover, for some subjects the tone occurs in very short and frequent bursts. Thus it can seriously impede the attainment of a state of unfocused attention.

As an alternative to binary feedback, I decided to investigate the use of analogue feedback in the form of a continuous signal modulated by the intensity of the alpha rhythm. The purpose of this small study was to compare the efficacy of two types of modulation: frequency (FM) and amplitude (AM).

3:4:2 Method

Frequency-modulated feedback was provided by a tone which varied in pitch in proportion to the absolute amplitude of the alpha rhythm. The alpha signal was isolated from the EEG by a 8-12 Hz band-pass filter and buffer amplifier, as before, and then rectified and smoothed to produce a steady negative-going DC voltage. This voltage was applied to the input of a voltage controlled oscillator (Farnell, Model FG1). In the absence of alpha activity the subject heard a soft 200 Hz sine wave. As alpha spindles occurred the tone sank in pitch (I found a low pitch tone to be more pleasant to listen to for long periods, than a high one) such that a 50 μ V, 10 Hz signal at the headbox produced a tone of approximately 100 Hz.

For the alternative form of analogue feedback, a white noise signal was amplitude-modulated so that it varied in intensity in direct proportion to the intensity of the alpha rhythm. I decided to employ white noise in preference to a tone because a constant tone varying in loudness is likely to prove very distracting. A suitable device was constructed which mixes the alpha activity with white noise so that the intensity of the white noise fluctuates at alpha frequency to produce a soft, restful, "chuff-chuff" sound (Bollen, 1973). The input to the white noise generator and mixer was provided from the band-pass filter and buffer amplifier, and the gain of the system adjusted to provide a suitable range of modulation.

Both of these modes of feedback were given to a group of six subjects. The subjects were asked to attempt to enhance alpha for four two-minute trials, and to attempt to suppress alpha for four two-minute trials, with each mode. For three of the subjects the tone from the voltage-controlled oscillator was provided first; and for the other three, the modulated white noise. A two-minute baseline recording was taken at the end of each task for both modes of feedback. The subjects were instructed to keep their eyes closed for all trials, and at the end of the session they were asked to report which of the two modes of feedback they preferred.

3:4:3 Results and Discussion

Half of the six subjects preferred the frequency-modulated tone as feedback, and half the amplitude-modulated white noise. Four of the subjects complained that they found the tone irksome, and that the white noise was much more pleasant to listen to. Three of these said that the white noise was the preferable form of feedback because it was less obtrusive and enabled them to relax without being distracted. Conversely, the fourth, and the two remaining subjects, preferred the modulated tone

because they felt it gave them more information about their alpha activity than the modulated white noise, which was too "imprecise" and lacked a sufficient range of perceived modulation to make an effective metric of the amount of alpha activity.

The group percentage alpha scores corresponding to the alpha enhancement and alpha suppression trials for both the modalities of feedback are plotted in Figure 3.7. The enhance-alpha and suppress-alpha trials for frequency-modulated feedback were compared using the distribution-free Wilcoxon matched-pairs test. The rank differences were not significant ($T = 6$, $N = 6$, $p > 0.05$). Similarly there was no significant difference between the enhance-alpha and suppress-alpha scores for amplitude-modulated feedback ($T = 3$, $N = 6$, $p > 0.05$). The differences between the enhance and suppress alpha scores for each mode of feedback were also compared but the

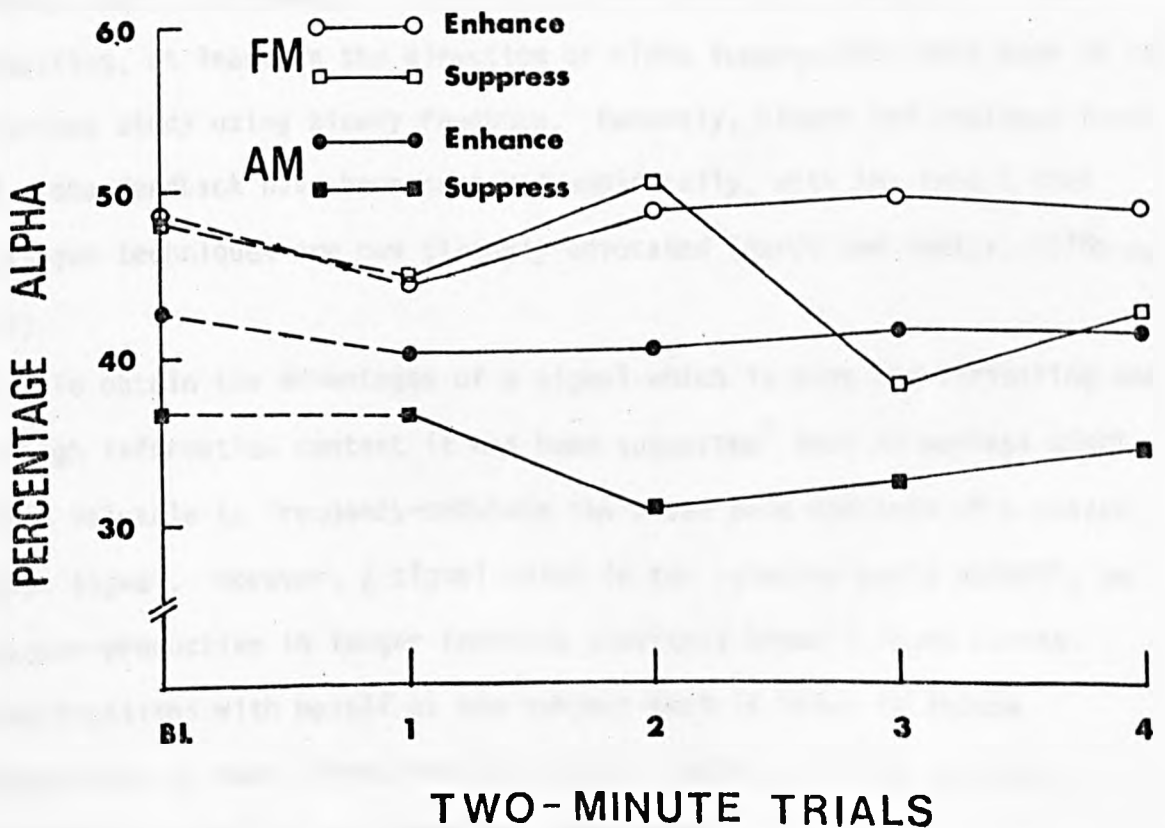


Figure 3.7

Alpha enhancement, alpha suppression, and baseline (BL) group mean percentage alpha trial scores for frequency-modulated (FM) and amplitude-modulated (AM) modes of feedback.

result was non-significant ($T = 5$, $N = 6$, $p > 0.5$). The data therefore indicates that both forms of feedback were equally ineffective in producing significant changes in alpha activity within the short time period of this experiment. However, with a larger subject sample and further feedback training it is quite possible that the two modes of feedback might operate differentially, perhaps in accordance with the predilections of the subjects.

This study emphasizes again the predominance of individual differences in alpha feedback training, and illustrates that no one form of feedback is likely to be satisfactory for all subjects. Hence to optimise feedback training it might be valuable to allow subjects an opportunity to select from a choice of several different modes of feedback.

The importance of moment-by-moment feedback information was apparent with all subjects, and this would suggest the superiority of analogue to binary forms of feedback. Conversely, more immediate changes in alpha densities, at least in the direction of alpha suppression, were seen in the previous study using binary feedback. Recently, binary and analogue forms of alpha feedback have been compared empirically, with the result that analogue techniques are now strongly advocated (Hardt and Kamiya, 1976b *see* 8:1).

To obtain the advantages of a signal which is both non-irritating and of high information content it has been suggested¹ that it perhaps might prove valuable to frequency-modulate the broad band spectrum of a shaped noise signal. However, a signal which is too relaxing would possibly be counter-productive in longer feedback sessions; since I found during investigations with myself as the subject that it tends to induce drowsiness, or even sleep, and therefore a decrease in the dominant frequency of the EEG away from the alpha range.

¹*MacKay, D.M., personal communication (1974).*

3:5:1 Introduction

Conventional operant procedures, with reinforcement through food or direct-brain reward, have produced some impressive demonstrations of bi-directional control of animal EEG activity (Wyrwicka and Sterman, 1968; Dalton, 1969; Fetz, 1969; Miller, 1969; Black et al., 1970; Black, 1971; Glazer, 1974; Wyler and Fetz, 1974). The results from the training of human EEG patterns, however, are generally not as definitive as those from the animal studies. This is perhaps partly because the reinforcers employed in the human biofeedback situation are typically weak and of a secondary nature. Miller (1974) drew attention to this problem in his review of biofeedback research and concluded that, "There is an urgent need for further study of the use of different types of feedback, or of a less cognitive, more primitive type of reward than money or the knowledge of success.... This is a new area in which investigators should be bold in what they try, but cautious in what they claim".

Powerful positive reward is of course difficult to arrange for human subjects. An alternative form of reinforcement, extensively employed in the therapeutic application of operant procedures (Rachman and Teasdale, 1969), is the avoidance of an aversive stimulus. A major disadvantage of avoidance training, however, is that the aversive stimulus itself can produce large unconditioned effects on physiological activity. Nevertheless, avoidance training has been successfully employed to effect control over a number of human physiological functions, such as heart rate (Frazier, 1966) and spontaneous skin resistance responses (Quy and Kubiak, 1974). Avoidance

¹*This investigation was carried out in the Spring of 1976 in conjunction with K.M. Sullivan as a project for his B.A. degree examination in Psychology. He was responsible for the EEG recordings, under my supervision.*

training has also been used in animals to enhance localised EEG patterns (Black et al., 1970) and to establish conditioned EEG desynchronization (Izquierdo et al., 1965).

An investigation of the efficacy of avoidance training for the experimental modification of human EEG activity was suggested by the recent finding of Orne and Paskewitz (1974) that the anticipation of harmless, but quite painful, electric shock did not lead to any significant attenuation of the EEG alpha rhythm, even though marked increases in heart rate and skin conductance responses were observed. Moreover, only a transient drop in alpha densities occurred when the shocks were actually administered. This present study was therefore set up to examine empirically the effects of a shock-avoidance contingency, as an alternative methodology for training the human alpha rhythm.

3:5:2 Method

A free-operant avoidance paradigm (Sidman, 1953) was adopted. Shocks were presented at regular intervals, with a predetermined delay in the occurrence of the next shock whenever a criterion alpha response was detected. The criterion response was defined as a burst of alpha activity greater than 20 μ V in amplitude, and exceeding a prescribed duration.

Apparatus

Occipital EEG was recorded from saline pad electrodes (Standard Laboratory Equipment type A1.P) placed at 3 cm above the inion on the midline, and on the left mastoid. Alpha activity was detected by band-pass filtering and digital period analysis, as described above. Criterion responses were automatically determined by duration logic circuitry which was developed for investigating the feedback training of sensorimotor rhythm (see 5:1:5). When a criterion response occurred, a trigger pulse was

delivered to a specially designed avoidance training unit (Quy, 1976). This was programmed to provide a shock-shock interval of 20 sec and a response-shock interval of 40 sec. The shocks were of 350 msec duration and were derived from a constant-current, 225 V isolated-battery source. They were delivered to the right index finger through two silver disc electrodes coated with electrolyte. A synchro-slide recorder (Phillips, Model L 1011) was modified to record and replay shock schedules. The occurrence of criterion responses and shocks was indicated on the EEG paper record by marker pulses.

In addition to scoring criterion responses, alpha activity was quantified as a time integral, as it has been recently suggested that integrated alpha provides a more sensitive index of alpha change than criterion measures (Travis et al., 1974b; Hardt and Kamiya, 1976b *see* 8:1). The output from the alpha band-pass filter was fed to a zero-resetting integrator (Emde and Shipton, 1974, *see* 4:2:2), but the signal was first gated by the relay of the digital alpha detector in order to prevent contamination of the data by insufficiently attenuated out-band activity, in particular movement and muscle artifact. Output pulses from the integrator were scored and automatically printed by the electro-mechanical counter system described previously (*see* 3:1:2).

A block diagram of the alpha feedback and detection system, as it was modified and extended for this experiment is provided in Figure 3:8.

Procedure

A yoked-control group was included in the study in order for a comparison to be made between the effects of shock-avoidance training and non-contingent shocks. The subjects in the control group received the shock schedule of paired shock-avoidance subjects. Sixteen volunteer undergraduates (nine male, seven female) served as subjects: eight in each group.

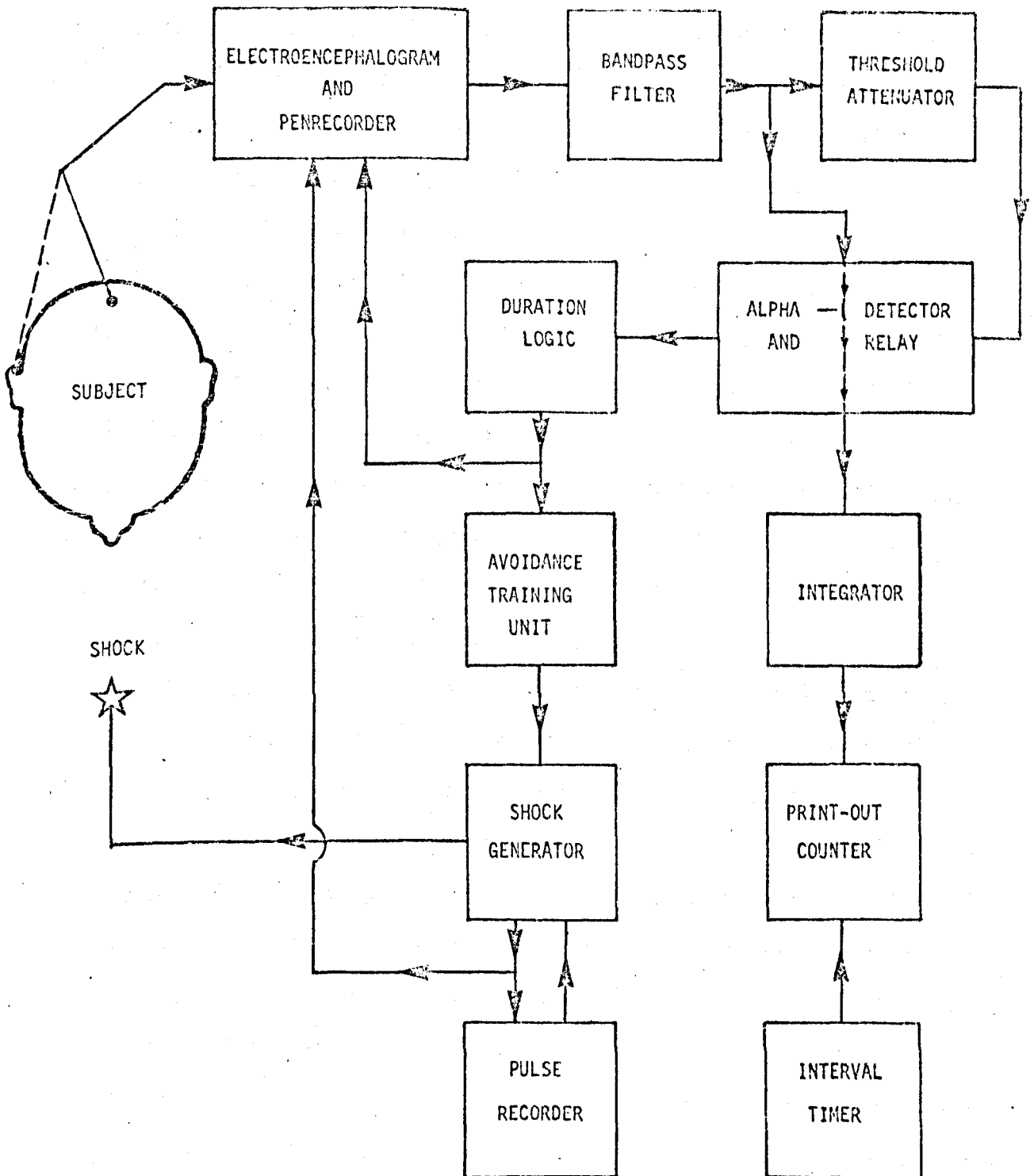


Figure 3.8

Flow diagram of the apparatus used in the investigation of avoidance training of the alpha rhythm.

The experiment involved two sessions: the first was used to find the appropriate criterion response duration for each subject, and to match subject pairs for the second session in which the training was given. Both sessions were carried out with the subjects seated in total darkness (to avoid the confounding effects on alpha rhythm of eye-opening and ambient light) in a sound-insulated chamber.

In the first session the subjects were simply asked to sit quietly for a ten-minute baseline recording. The criterion response was adjusted for each subject so that, although there was considerable intra-subject variability in response rate, there were, on average, three responses a minute registered. This provided both high and low alpha density subjects with a suitable opportunity for learning. Subjects were paired according to their mean integrated alpha scores and then randomly assigned to the shock-avoidance and yoked-control groups.

At the beginning of the second session, the subjects in both groups were instructed that they were to receive a number of unpleasant but harmless electric shocks, and that the incidence of shocks was related to the activity of the brain. Then the shock intensity was gradually adjusted for each subject (by varying series-resistance) so as to be uncomfortable, but tolerable. A three-minute baseline was then recorded, followed by a forty-minute training period. Criterion responses and integrated alpha were scored at one-minute intervals. The integrated alpha scores were expressed as a percentage of the mean integrated alpha recorded during the baseline measure at the beginning of the session, in order to minimise the influence of inter-subject variance in alpha amplitudes.

3:5:3 Results

The mean number of criterion responses per minute of training for the

shock-avoidance and yoked-control groups are shown in Figure 3.9a. These were compared within a two-way analysis of variance (Groups by Time, with repeated measures on the Time factor). There was a very significant effect for Time ($F(39,546) = 1.65, p < 0.01$), but not for Groups ($F(1,14) = 2.45, p > 0.05$), nor for the Group by Time interaction ($F(39,546) = 0.75, p > 0.5$).

It is clear from Figure 3.9a that, although the yoked-control group tended to emit more responses overall, the shock-avoidance group exhibited

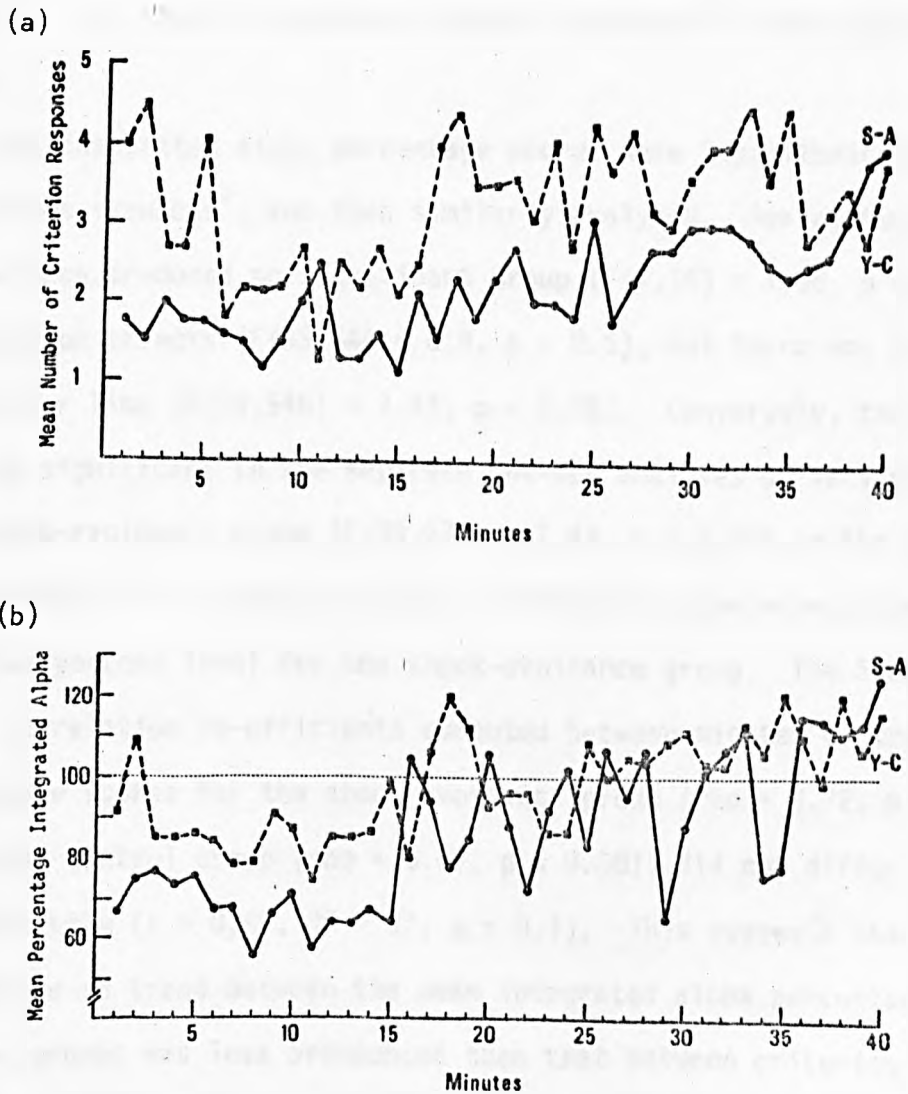


Figure 3.9

(a) Mean number of alpha responses exceeding criterion duration against minutes of training for the eight subjects in the shock-avoidance (S-A) and the yoked-control (Y-C) groups.

(b) Mean integrated alpha scores against minutes of training for the shock-avoidance (S-A) and yoked-control (Y-C) groups. The score for each subject is expressed as a percentage of his initial baseline level.

a stronger upward trend in response rate. Therefore the data for each group was further examined separately. A one-way, repeated measures, analysis of variance produced a significant Time effect for the shock-avoidance group ($F(39,273) = 1.44, p < 0.05$), but not for the yoked-control group ($F(39,273) = 1.08, p > 0.05$). Spearman rank-order correlation co-efficients were also calculated between criterion responses and minutes of training for the two groups. The correlation was twice as large for the shock avoidance group ($\rho = 0.77, p < 0.05$), indicating that the rate of increase in criterion responses was much greater for the shock-avoidance group.

The integrated alpha percentage scores were logarithmically transformed to correct skewness¹, and then similarly analysed. Again, two-way analysis of variance produced no significant Group ($F(1,14) = 1.05, p > 0.5$) or interaction effects ($F(39,546) = 0.8, p > 0.5$), but there was a significant effect for Time ($F(39,546) = 1.43, p < 0.05$). Conversely, the Time effect was not significant in the separate one-way analyses of variance for either the shock-avoidance group ($F(39,273) = 1.42, p > 0.05$) or the yoked-control group ($F(39,273) = 0.86, p > 0.05$), although it approached significance at the five percent level for the shock-avoidance group. The Spearman rank-order correlation co-efficients computed between minutes of training and percentage scores for the shock avoidance group ($\rho = 0.72, p < 0.001$) and the yoked-control group ($\rho = 0.65, p < 0.001$) did not differ significantly ($t = 0.65, df = 37, p > 0.1$). This suggests that the difference in trend between the mean integrated alpha percentage scores for the two groups was less pronounced than that between criterion responses.

The mean integrated alpha per minute for the training period was

¹Logarithmic and square root transformations were compared for their effect on the mean data. The former provided the least skewness ($g^1 = 0.26$) and kurtosis ($g^2 = -1.5$).

compared with that recorded during the baseline period at the beginning of the training session. Neither the shock-avoidance group ($t = 1.51$, $df = 7$, $p > 0.1$) nor the yoked control group ($t = 0.25$, $df = 7$, $p > 0.5$) showed a significant difference. From Figure 3.9b it can be seen that both groups exhibited an initial suppression of alpha below baseline level (as indicated by the low percentage scores), followed by a gradual increase across the session. The pooled data were examined for individual differences within the groups. Two of the eight subjects in the shock-avoidance group exhibited mean alpha levels for the training period which exceeded those in the baseline period (average of the two = 153%), and so did three subjects in the yoked-control group (average = 131%).

One surprising result that emerged from this study was that unconditioned blocking effects of electric shock on alpha rhythm were much less than were expected. The mean level of integrated alpha recorded in the baseline period that followed shortly after the shock intensities had been established at the beginning of the training session, did not differ significantly from that recorded during the first baseline session for either the shock-avoidance group ($t = 1.26$, $df = 7$, $p > 0.1$) or the yoked-control group ($t = 0.73$, $df = 7$, $p > 0.2$). This corroborates the finding of Orne and Paskewitz (1974) that anticipation of electric shock failed to cause depression of alpha levels. Moreover, when the raw EEG traces were perused, it was evidence that the electric shock itself produced no, or only slight, desynchronization of the alpha spindles, even if the shock caused the subject to respond with body or head movement, as illustrated in Figure 3.10.

3:5:4 Discussion

As Group and Interaction effects in the criterion and integral measures were absent, and alpha was not enhanced above initial baseline levels, it

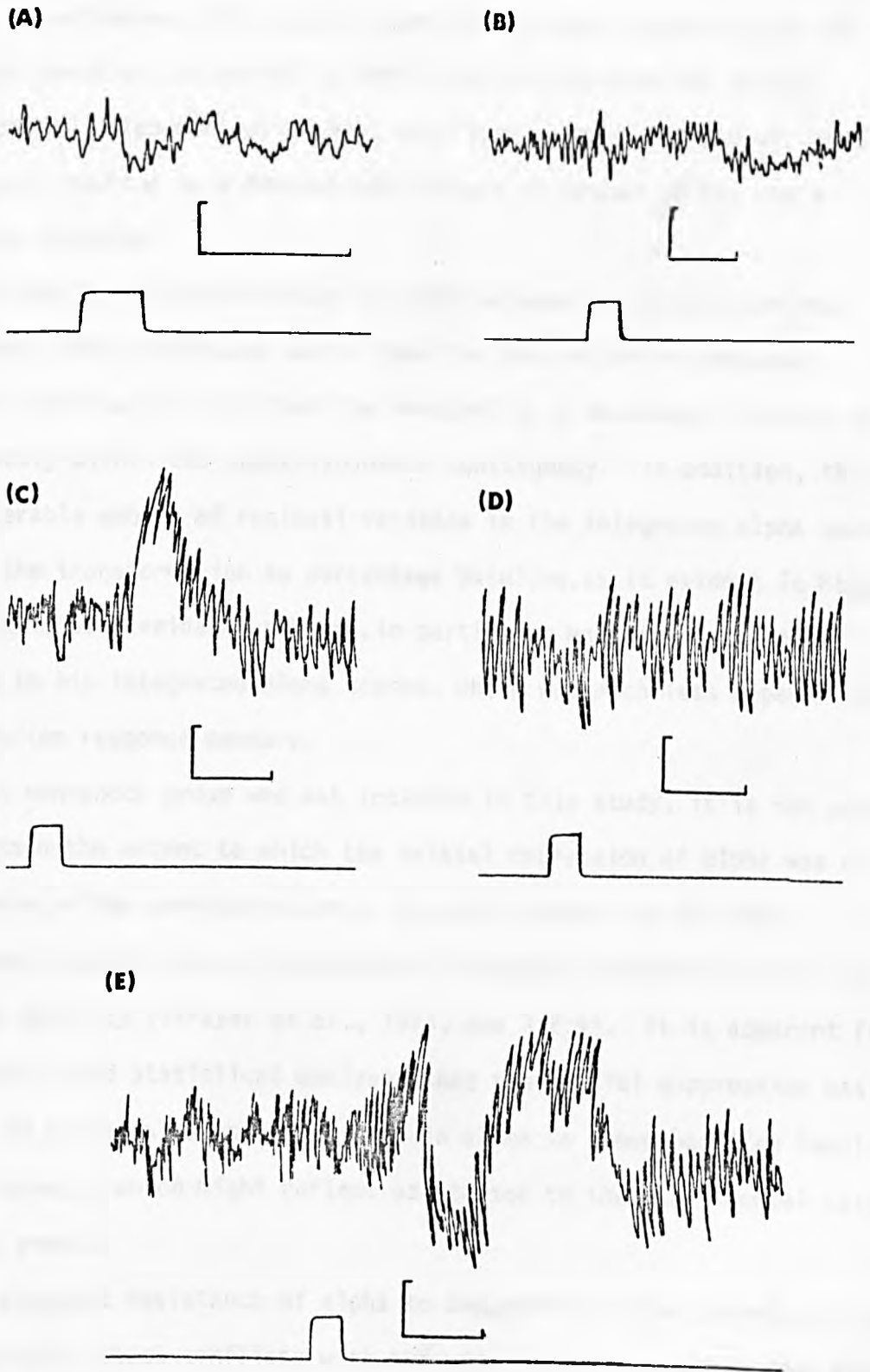


Figure 3.10

Occipital EEG recordings from different subjects during delivery of mild electric shock to the finger (indicated by marker pulse). A-D: Examples where the shock appears to produce very little desynchronization of alpha activity in the EEG, even when the shock is followed by pronounced movement artifact (C). E: An instance where alpha rhythm is apparently elicited by the shock.

Calibration bars: 50 μ V and 1 sec.

cannot be concluded that alpha activity was successfully trained in this study. Nevertheless, the general trend of the data suggests that the avoidance paradigm was operating differentially between the shock-avoidance and yoked-control groups, such that a longer period of training might have resulted in a demonstrable effect in favour of the shock-avoidance subjects.

The smaller differentiation in trend between the groups for the integrated alpha percentage scores than for the criterion responses possibly reflects the fact that the integral as a dependent variable was not directly within the shock-avoidance contingency. In addition, there was a considerable amount of residual variance in the integrated alpha scores despite the transformation to percentage baseline, as is evident in Figure 3.9b. One shock-avoidance subject, in particular, exhibited extreme lability in his integrated alpha scores, which was much less apparent in the criterion response measure.

As a non-shock group was not included in this study, it is not possible to ascertain the extent to which the initial depression of alpha was a consequence of the administration of electric shocks, or of other situational factors such as the subjects' attempts to exert control over their EEG activity (Strayer et al., 1973; see 3:2:4). It is apparent from the graphical and statistical analyses that the initial suppression was followed by a gradual spontaneous rise in alpha to above baseline levels for both groups, which might reflect adaptation to the experimental setting or to the shocks.

The apparent resistance of alpha to desynchronization during delivery of the electric shock conflicts with the widely-held assumption that alpha rhythm blocks when an awake subject is presented with a strong stimulus (Mackworth, 1969). It has been suggested, however, that the blocking of alpha is primarily associated with oculomotor arousal as part of the

orienting response (Plotkin, 1976a). Hence it is possible that, since the subjects in this present study were seated in total darkness and the stimulation was presented haptically, the oculomotor arousal produced by the shock might not have been very great. There was no evidence for a gradual habituation of alpha blocking during the session, and subjects generally reported that they did not subjectively experience any decrease in shock intensity over the training period. The shock was occasionally even observed to evoke a burst of alpha rhythm from a background of desynchronized activity, as illustrated in Figure 3.10 (E). The appearance of stimulus-induced alpha when the level of arousal is low has been well documented (Morrell, 1966, *see* 3:3:1), but, in view of the shock avoidance procedure, it is very unlikely that the subjects became drowsy in this study.

The absence of marked unconditioned effects of shock on alpha activity suggests that shock-avoidance training of other animal and human EEG components would be worth investigating. Possibly, avoidance training would prove to be more effective than present biofeedback techniques for the experimental modification of the EEG, especially as it is not uncommon for subjects to become bored or drowsy when training sessions are extended. In addition, the motivational variables involved in EEG biofeedback training are difficult to define and control. There is always the possibility of experimenter-subject interaction and expectancy effects, since much of the reinforcement stems from feeling of success at the task, and from other subtle reinforcers established by the instructions and the background of the subject (Orne, 1962; Black, 1973). Avoidance training would enable performance motivation to be more readily brought under direct experimental control. Finally, it has been suggested that faster response acquisition may sometimes be achieved if subjects are left naive to the exact response-reinforcement contingency (Hefferline et al., 1959; Quay and

Kubiak, 1974). The avoidance training paradigm would allow this to be easily arranged without producing confounding effects on motivation.

A system was constructed for the detection and feedback training of EEG alpha rhythm.

EEG from the right occiput was recorded with reference to the mastoid using standard electroencephalographic procedures. Alpha activity was detected by band-pass filtering and digital period analysis. Binary feedback of the occurrence of alpha activity was given by means of a tone signal. The cumulative incidence of alpha was quantified by an electro-mechanical counter system, and then converted to a percent-time measure. A pilot study was carried out to enable the system to be thoroughly tested and calibrated.

A study was set up to replicate reports that EEG alpha activity could be modified through feedback training, and to examine the relationship between changes in alpha with training and baseline alpha levels. Twenty subjects were asked to enhance alpha for two seven-minute trial blocks and to suppress alpha for two seven-minute trial blocks. A very marked difference in percentage alpha between enhancement and suppression trial blocks was evident, confirming the reports of other workers. However, although alpha was considerably reduced below baseline levels throughout the suppression trial blocks, the group mean alpha for the enhancement trial blocks did not differ significantly from baseline, indicating that alpha modification is strongly unidirectional. Conversely, there was a highly significant linear trend in enhancement scores over the session, whilst there was no significant spontaneous increase in the baseline alpha densities. Inter-subject differences in performance were marked and, although four of the subjects exhibited enhancement of alpha above baseline, this was masked by other subjects who showed a suppression of alpha during the enhancement trials.

A number of subjects appeared to enhance alpha by strategies associated

with concentration and tension rather than relaxation, in contrast to the general trend. This was further investigated in sixteen subjects. Mean alpha for trials in which the subjects were aroused was significantly greater than for trials in which they were relaxed in the eyes-open condition, although individual differences were again very apparent.

The binary feedback tone was found to distract some subjects from producing a mental state conducive to alpha enhancement. Hence analogue feedback, in the form either of a frequency-modulated tone or of amplitude-modulated white noise, was explored as an alternative. Although no modification in mean alpha densities was obtained within eight minutes of enhancement and eight minutes of suppression training, the results indicated that some modes of feedback might be more effective for some subjects than for others. The inherent conflict between the informative and obtrusive properties of sensory feedback was apparent in the subjects' descriptions of their experience.

Avoidance training of alpha activity was later attempted, following the report of Orne and Paskewitz (1974) that alpha was not attenuated by mild electric shock. Eight subjects in a shock-avoidance group did not differ significantly from yoked-control subjects in mean criterion response or integrated alpha measures over forty minutes of training. However, the increase in response rate over time was significantly greater for the shock-avoidance group, suggesting that with extended training shock-avoidance might be an effective technique for the experimental modification of EEG activity. Unconditioned blocking effects of the electric shock on the alpha rhythm were observed to be surprisingly absent.

4:1 PRELIMINARY ASSESSMENT OF SENSORIMOTOR RHYTHM TRAINING

4:1:1 Introduction

The emphasis of this research was shifted from the alpha rhythm to SMR at the end of the first year of the work in order that the potential application of SMR training to the treatment of epilepsy might be explored. However, before the effects of SMR training on patients with epilepsy were investigated, a study of SMR training with normal subjects was carried out. The purposes of this work were: firstly, to investigate methodological problems involved in the detection of low voltage SMR in the EEG; secondly, to investigate whether subjects could learn to enhance EEG components in the SMR frequency range through feedback training; and thirdly, to investigate the hypothesis that these components represent a functional homologue of the feline SMR.

The work began with a preliminary study of whether SMR activity could be detected in the EEG. In order to do this, the alpha detection and feedback system was adapted so that it would respond to EEG activity in the 12-16 Hz frequency band. A number of feedback training sessions were then carried out, as described below. On the basis of the results from this preliminary study, several modifications and additions to the equipment were made before the main study of SMR feedback training in normal subjects was initiated.

4:1:2 Method

The frequency cut-offs of the variable band-pass filter were adjusted to 12 Hz and 16 Hz; and the upper and lower frequency limits of the digital alpha detector were similarly altered. A threshold amplitude of 5 μ V was selected for the digital detector, since Sterman (1973a; 1974) notes that

SMR can only be detected with a system that is set to respond to signals in the order of 3-6 μ V.

The incidence of 12-16 Hz components in the EEG was quantified and automatically printed out at one-minute intervals as described previously (*see* 3:1:2). Feedback to the subject was provided in two ways. First, by a 400 Hz tone signal which was switched on whenever criterion activity occurred, rather than off, as in alpha training, since the occurrence of 12-16 Hz bursts was relatively infrequent. Secondly, since visual stimulation is reported to have no attenuating effect on SMR (Stermán, 1974), a digital timer (Colne Instruments, Model DT1) was placed in front of the subject to provide a continuous display of the cumulative duration of criterion 12-16 Hz activity.

EEG was recorded with silver / silver-chloride disc electrodes from various placements over the peri-rolandic region. These electrode placements are defined according to the *International Ten-Twenty System* (Figure 4.1). In some instances a bipolar lead was recorded from one electrode placed midway between sites C_z and C_3 to another placed midway between sites C_3 and T_3 (Stermán, 1973a; Stermán et al., 1974). Alternatively, the electrodes were placed in the anterior-posterior plane, one midway between sites F_3 and C_3 , and the other midway between sites C_3 and P_3 (Stermán and Friar, 1972). Sometimes referential recording was used, with the active electrode midway between F_3 and C_3 and the reference on the left mastoid (Finley, 1974; Finley et al., 1975). In addition, occipital EEG and eye-movements were recorded for some subjects, as described previously (*see* 3:1:3).

Recording sessions were carried out with four different subjects. They were asked to attempt to turn the feedback tone on as much as possible and to increase their score on the digital timer. Each session lasted approximately thirty minutes.

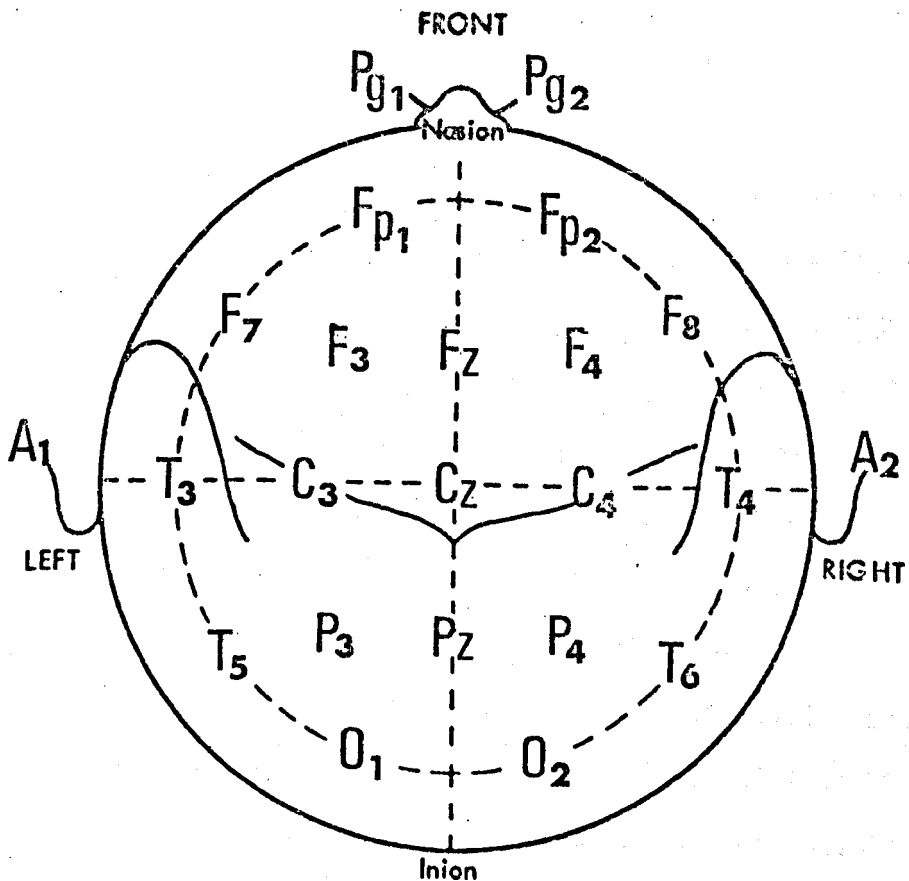
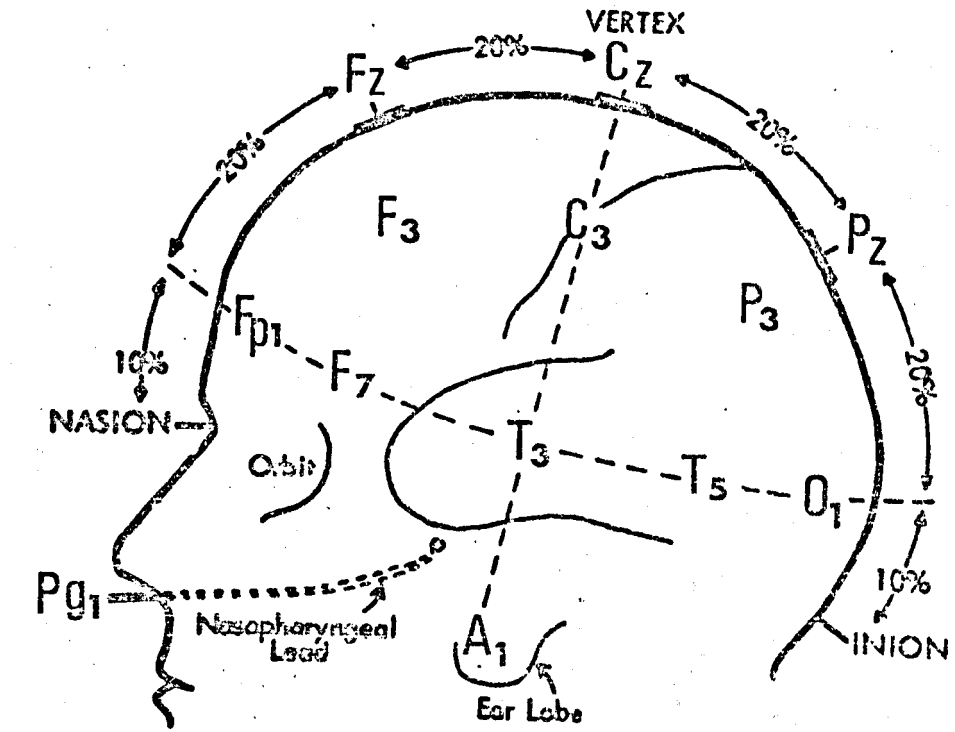


Figure 4.1

The International Ten-Twenty System for the placement of EEG electrodes.

After Jasper (1958).

4:1:3 Results and Discussion

All of the subjects exhibited some 12-16 Hz activity in their rolandic EEG, as indicated by the output of the digital detector, even though the detection system was probably biased against indicating the presence of low amplitude 12-16 Hz components because of the masking effect of background EEG activity (*see* 5:1:2). Generally the 12-16 Hz pattern only occurred as very short bursts; but after three training sessions one subject started to demonstrate spindles of up to two seconds, which were also clearly visible in the raw EEG (Figure 4.2). It became evident during these preliminary training sessions, however, that much of the 12-16 Hz activity registered as SMR was probably *not* from the rolandic cortex, but reflected rostrally conducted occipital potentials, or the effect of artifact. These two sources of error will be considered in turn.

Confounding Effects of Occipital Spindles

For some subjects, the bursts of 12-16 Hz activity in the central channel appeared to parallel a burst of alpha rhythm in the occipital channel as depicted in Figure 4.3. Moreover, these subjects consistently obtained higher 12-16 Hz scores with the eyes closed than with them open, corresponding to increased alpha densities. These findings suggest that high frequency (12-13 Hz) alpha waves are conducted forward to the rolandic region and hence detected as 'SMR'. In subjects with a high amplitude alpha rhythm it is not uncommon for alpha to be seen in the EEG as far forward as in the eye movement channels, and thus readily exceed the 5 μ V amplitude threshold for detection in the rolandic EEG. The long burst shown in Figure 4.2 therefore may be alpha rhythm, especially since it was recorded from an anterior-posterior electrode pair (occipital EEG was not recorded in this instance). Sterman (personal communication, 1974) found that some of his subjects produced more SMR during periods with the eyes closed, indicating that occipital sources were also a confounding factor in

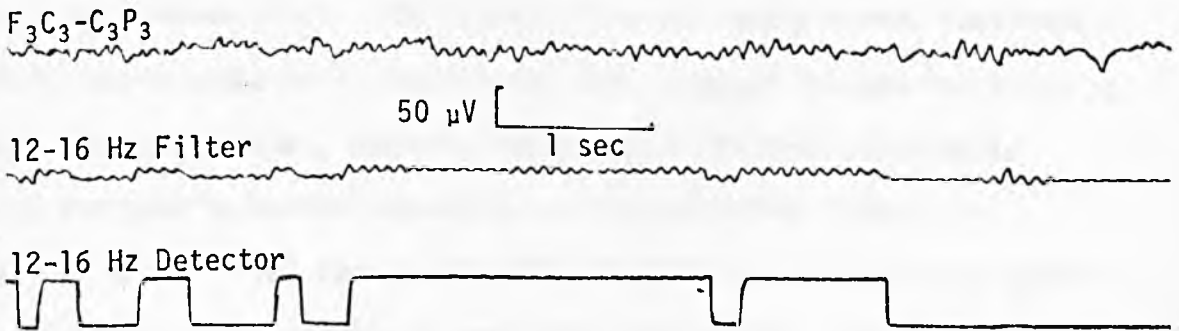


Figure 4.2

Long burst of 12-16 Hz activity seen in the rolandic EEG of one subject. The threshold amplitude of the digital detector was set at 5 μV .

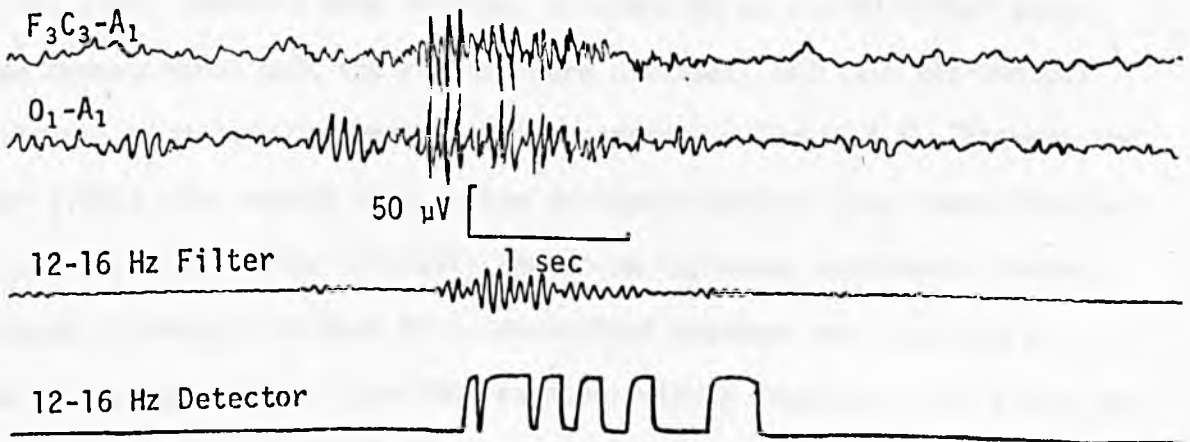


Figure 4.3

12-16 Hz activity in the rolandic EEG channel coinciding with a high voltage burst of 'sharp' alpha waves in the occipital channel.

his data.

The normal frequency range of alpha rhythm is defined as 8-13 Hz (Storm van Leeuwen et al., 1966), but a few apparently normal individuals exhibit stable alpha up in the classic beta range of frequencies (Harding, 1968). Sterman (1973a), however, reports that 12-14 Hz activity is mainly confined to central channels; and when EEG from frontal or occipital areas is fed through the SMR detection system then very little is registered. I was unable to replicate this result. It was found that considerable 12-16 Hz activity could be detected in the occipital EEG, even if the dominant alpha rhythm is of a lower frequency (Figure 4.4). These higher frequency components that are present in addition to the fundamental rhythm reflect the complex morphology of the EEG waveform and probably are a result of the stochastic nature of the interaction between the individual rhythm generators (*see* 2:2:2).

Despite the suspicions raised by the effects of occipital spindles, there was evidence in some subjects of an independent 12-16 Hz rhythm in the rolandic EEG. In one individual, in which it appeared that the central and occipital channels were related, a recording at a much higher paper speed demonstrated that the rhythms were distinct, and that the central rhythm was of higher frequency, as illustrated in Figure 4.5. Sterman and Friar (1972) also report that in one epileptic patient they found that both SMR and occipital alpha initially showed an increased occurrence during feedback training, but that this generalized increase was replaced by a selective enhancement of the SMR, together with a suppression of alpha, by the twelfth session. Similarly, in non-epileptic subjects, power spectral plots indicated a shift in the dominant peak from 9-11 Hz to 12-16 Hz over central and frontal areas, following training, whilst parietal 9-11 Hz activity was attenuated (Sterman et al., 1974).

It is possible that an apparent coincidence of SMR and alpha rhythm

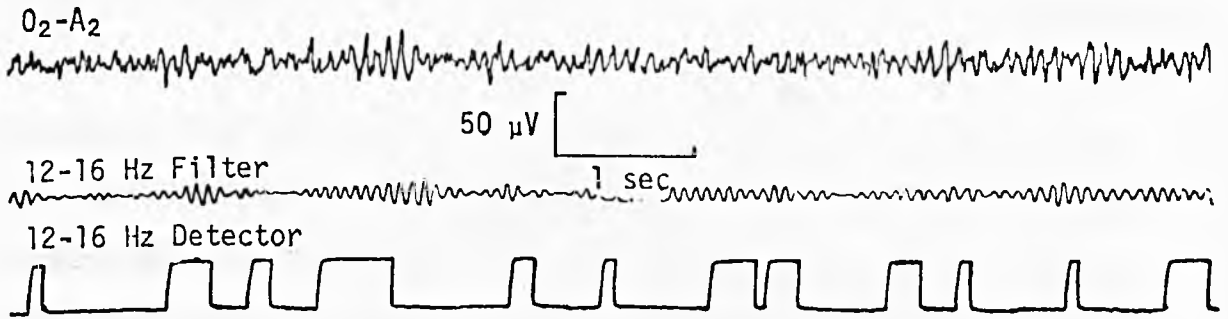


Figure 4.4

Output of 12-16 Hz detection system with occipital EEG input. Note that the digital detector, with a low frequency cutoff at 12 Hz, only responds to some sections of the filtered alpha spindles.

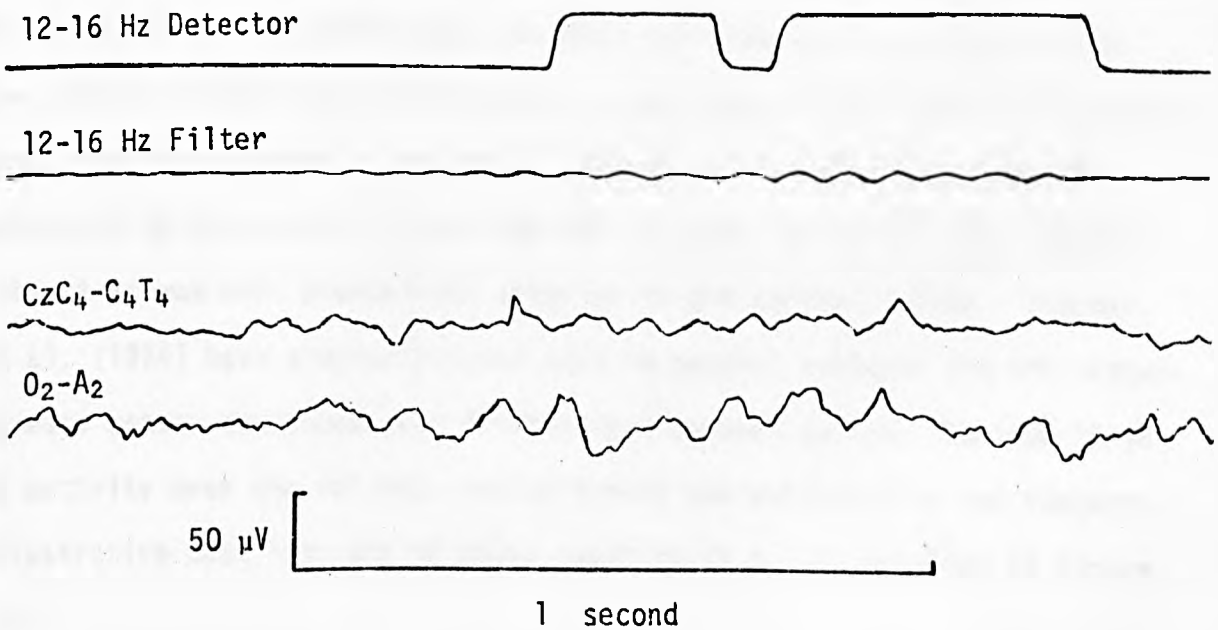


Figure 4.5

Central and occipital EEG traces at a paper speed of 120 mm/sec. The two signals appear to be independent. The average period of the central rhythm is in the region of 75 msec (13.3 Hz), and that of the occipital rhythm in the region of 106 msec (9.4 Hz).

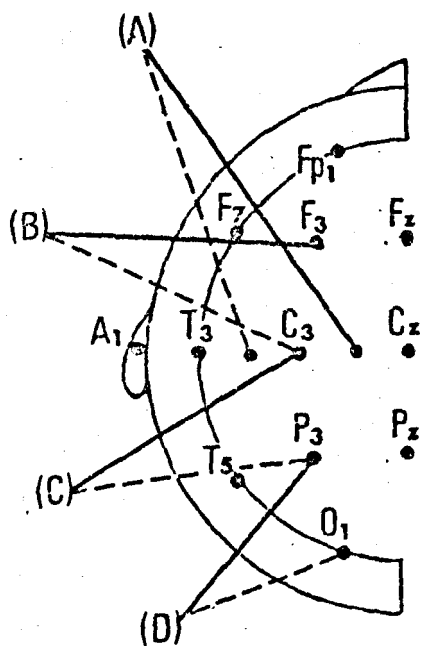
initially arises because the behavioural states required for their production are superficially similar. Thus the quiet, relaxed state said to be associated with SMR is also likely to be conducive to the generation of alpha. According to this hypothesis, a tendency for a shift in the balance of excitatory and inhibitory drive, such as produced by a gross modulation of arousal and habituation to inhibitory situational factors, might predispose toward both central and occipital synchrony, but more localized changes in afferent and efferent processes produce independent and localized activity (*see* 4:2:4).

A study of the topographical distribution of 12-16 Hz and 8-10 Hz in a group of normal subjects was attempted at a later stage of this research. Electrodes were placed across the scalp in the left peri-rolandic region at 2 cm intervals, and both bipolar and referential recordings were made. The EEG was recorded on a twelve-channel FM recorder (Thermionic, Model T4000) and analysed with the apparatus described in Chapter Five. Because of frequent hardware failures with the recorder, however, this study had to be abandoned. Nevertheless, the data that was obtained did indicate the extent of individual differences in the locus of the 12-16 Hz frequency band: for three subjects the highest abundance of 12-16 Hz was obtained posterior to the central sulcus and the occiput; conversely, for another subject it was more predominant anterior to the central sulcus. Sterman et al. (1974) have similarly noted that in several subjects the SMR pattern appears better developed over frontal than central cortex. Maximum 12-16 Hz activity over the rolandic region itself was exhibited by two subjects. Illustrative data from one of these subjects (B.S.) is provided in Figure 4.6.

In addition, an attempt was made to use phase reversal points as a means to delimit the probable site of the cortical generator of the 12-16 Hz activity (Cooper et al., 1969). However, because no clear 12-16 Hz

rhythm exists in the raw signal, adjacent channels were fed into a pair of band-pass filters. This technique indicated that the primary generator for subject B.S. was localized to an area situated posterior and slightly medial to C_3 , which corresponds to the somatosensory cortex. However, although these filters were identical models, it is not known how closely their phase response was matched, and so the results cannot be considered to be very reliable.

These individual differences in the focus and distribution of 12-16 Hz activity are most probably due to small variations in the parameters of the thalamo-cortical networks responsible for synchrony, and in the propagation of the functional rhythmic units (*see* 2:2:3). Similarly, the extent to which rostrally conducted occipital spindles contribute to the 12-16 Hz activity detected by centrally placed electrodes is likely to be a function both of their amplitude and of the frequency spectrum of the



CHANNEL	12-16 Hz DENSITY	8-10 Hz DENSITY
A	313	107
B	39	6
C	180	118
D	243	387

Figure 4.6

Comparison of 12-16 Hz and 8-10 Hz scores for four equi-distant bipolar derivations simultaneously recorded over a 17 min. epoch from subject B.S. 12-16 Hz activity is seen to be maximal in the transverse lead across the central sulcus. A subsequent determination of phase reversal points suggested that the generator site lay in the region bounded by C_z , C_3 , P_z , and P_3 .

individual's alpha rhythm. Clearly, further investigations are required, using techniques such as cross-correlation, before it can be concluded that 12-16 Hz sensorimotor activity universally exists in man as an electroencephalographically distinct and significant phenomenon. The presence of these frequency components within the alpha rhythm of some subjects suggests in the meantime that, in order to minimize their confounding effects on the data during investigations of SMR feedback training, it would be advisable to instruct subjects to keep their eyes open.

Confounding Effects of Artifact

During the preliminary investigations of SMR feedback training it became evident that artifact was a major source of error. When the chart records were perused, many instances of the false triggering of the detector system could be found. The effect of several different types of artifactual signals is illustrated in Figure 4.7.

For a number of extended training sessions I used myself as the subject, in an attempt to discover strategies for enhancing 12-16 Hz. During these sessions, I found that the feedback tone could be produced by simply swallowing hard or by jaw clenching. Moreover, this response could be refined so that a specific movement of the tongue slowly and gently across the roof of the mouth (even with the jaw completely relaxed) was sufficient to generate long bursts of feedback. The EEG exhibited a low amplitude, saw-tooth waveform, during this manoeuvre. When the exercise was repeated, and a simultaneous recording made with all high-cut filtering removed, it was revealed that large motor unit discharges were responsible for the apparent superfluity of 12-16 Hz EEG activity, as shown in Figure 4.8. These spikes originate from the temporalis muscle, a fan-shaped muscle lying on the surface of the scalp directly over the lateral peri-rolandic area (Hamilton, 1966). The periodicity of the spikes is highly labile and dependent on the tonus of the muscle. As I learned to

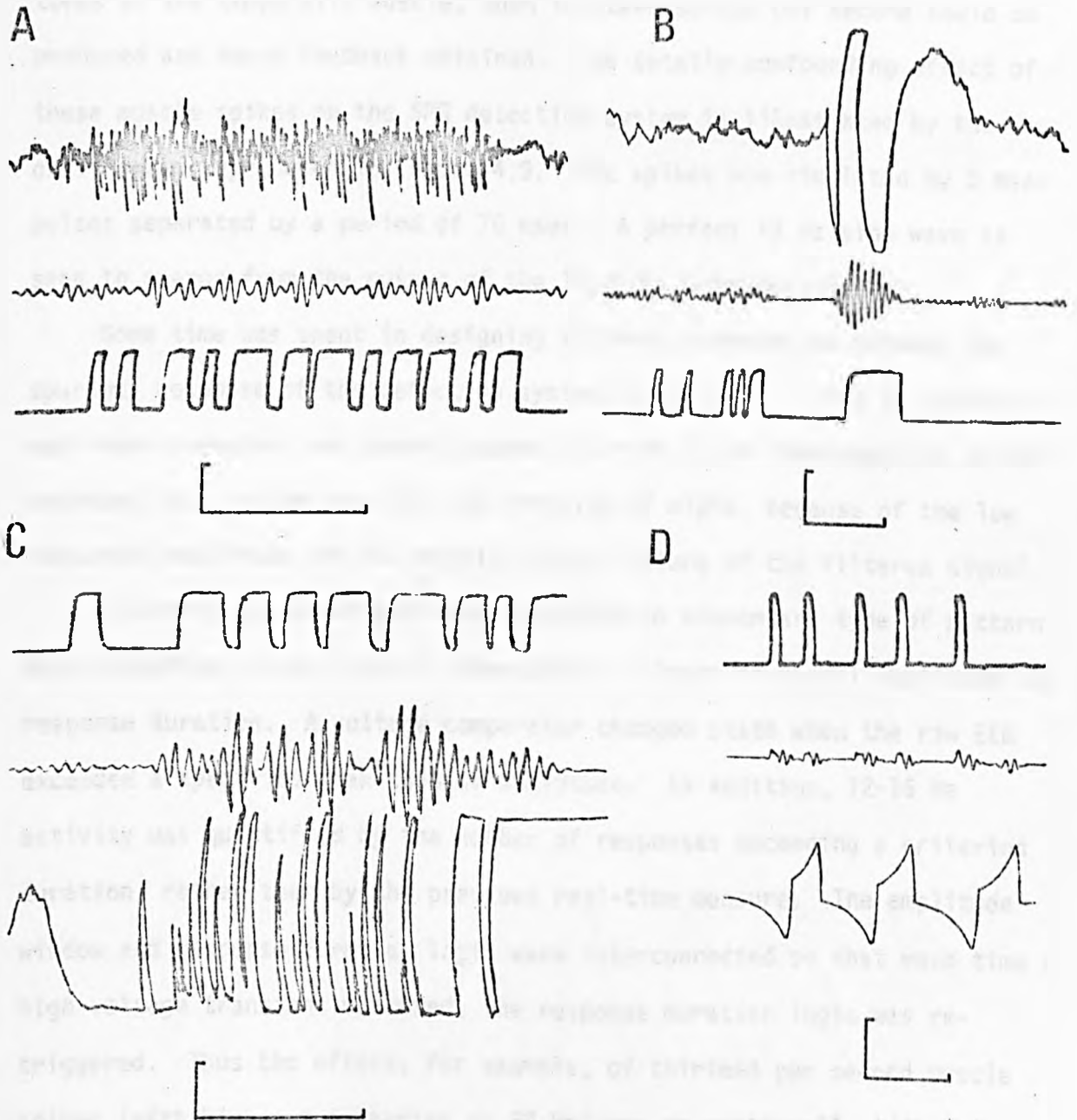


Figure 4.7

Spurious activation of the 12-16 Hz detection system by various types of artifactual signals. A: Swallow accompanied by jaw clenching. B: Transient produced by electrode movement. C: Electrode 'splashing' caused by head movements. D: Calibration pulses.

In all examples, an upward deflection of the marker pulse indicates the detection of criterion 12-16 Hz activity. Calibration bars: 50 μ V and 1 sec.

differentiate the response and exert some voluntary control over the tonus of the temporalis muscle, then thirteen spikes per second could be produced and hence feedback obtained. The totally confounding effect of these muscle spikes on the SMR detection system is illustrated by the oscillogram reproduced in Figure 4.9. The spikes are simulated by 5 msec pulses separated by a period of 76 msec. A perfect 13 Hz sine wave is seen to emerge from the output of the 12-16 Hz band-pass filter.

Some time was spent in designing suitable hardware to prevent the spurious response of the detection system to artifact. This is clearly a much more prevalent and potent source of error in an investigation of SMR training than is the case for the training of alpha, because of the low threshold amplitude and the highly derived nature of the filtered signal.

Circuitry was developed which provided an elementary type of pattern discrimination in the form of interacting analyses of signal amplitude and response duration. A voltage comparator changed state when the raw EEG exceeded a specified peak-to-peak amplitude. In addition, 12-16 Hz activity was quantified by the number of responses exceeding a criterion duration, rather than by the previous real-time measure. The amplitude window and response duration logic were interconnected so that each time a high voltage transient occurred, the response duration logic was re-triggered. Thus the effect, for example, of thirteen per second muscle spikes (with high-cut filtering at 50 Hz) was to continually 'jitter' the duration logic so that a criterion response could not be produced. The criterion measure also has the added advantage that it offers a ready means by which the response required for reinforcement can be shaped. A fuller description of the operation of the response discrimination apparatus, as it was later included in the unit built for the clinical study, will be given in the next chapter.

A number of test sessions were carried out with the modified system

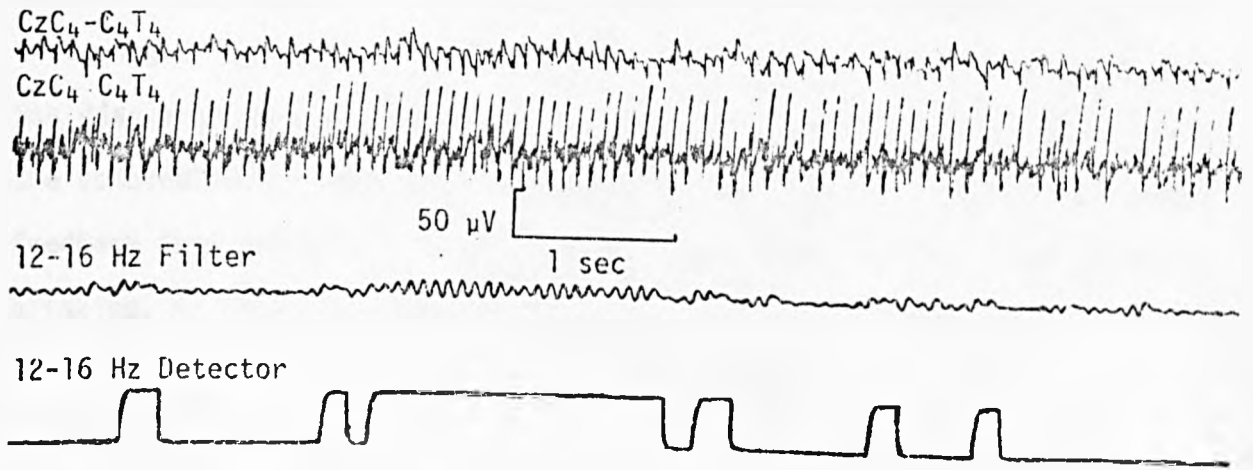


Figure 4.8

Spurious 12-16 Hz activity produced by small movements of the tongue. The rolandic EEG channel recorded with the usual high-frequency filtering shows low amplitude, saw-tooth waveforms (top trace); the same channel without the high-frequency filtering reveals the presence of motor unit discharges from the temporalis muscle (second trace). As can be seen, lengthy spurious responses result from these muscle spikes when they occur with the appropriate temporal displacement.

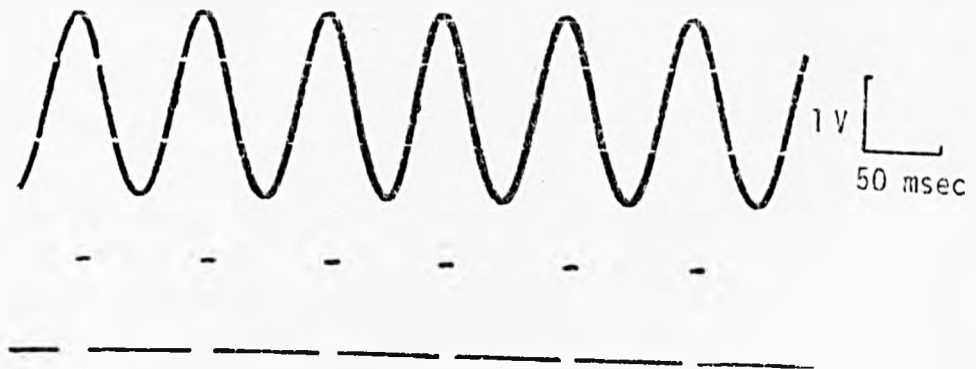


Figure 4.9

Oscillogram showing the output of the 12-16 Hz band-pass filter to a series of 3 V, 5 msec pulses at a 13 per second, simulating the effect of muscle spikes.

using myself as the subject. The feedback tone was connected to the duration logic so that a 500 msec 'bleep' was produced whenever a response exceeding criterion duration occurred. The cumulative number of responses was displayed on a digital timer as auxiliary feedback. It was found that the combination of amplitude and duration analysis satisfactorily prevented feedback from being falsely obtained by jaw clenching, head shaking, rapid blinking, or other such devices.

4:2 INVESTIGATION OF SENSORIMOTOR RHYTHM ENHANCEMENT IN NORMAL SUBJECTS

4:2:1 Introduction

The successful enhancement of SMR in non-epileptic subjects (two normal and two quadriparetic) has been reported by Sterman and colleagues (Sterman, 1973a; Sterman et al., 1974). They provided feedback training of 12-14 Hz central EEG for three twenty-minute training sessions per week over several months. The results indicated that, "Three of the four nonepileptic subjects showed a progressive increase in the rate of relay activation accompanied by the emergence of clear trains of 12-16 Hz activity in the EEG" (Sterman et al., 1974). However, no actual data, either graphical or tabulated, is given to support this claim that a progressive increase in criterion SMR occurred with feedback training. Similarly, although the EEGs were subjected to power spectral analysis, no quantification of the results across sessions was carried out to support the assertion that, "Dominance often shifted to peaks at 12 to 14 Hz, or to 14 to 16 Hz, or both" (Sterman et al., 1974). In short, as Kaplan (1975) concludes: "An increase in 12 to 14 Hz activity could be demonstrated convincingly with objective evidence such as a significant increase in the energy in that range of the EEG, or an increase in the percentage of time that activity is present, but the data reported by Sterman et al. (1974) have not been subjected to such statistical analysis". Therefore this study was set up in order to determine whether any reliable demonstration of the enhancement of 12-16 Hz rolandic EEG through feedback training could be obtained.

In addition, I sought in this study to test the assumption that 12-16 Hz rolandic activity in man represents the feline SMR by examining the relationship between the occurrence of this activity and motor inhibition. Thus it has been reported that behavioural quiescence always precedes bursts

of SMR in the cat (Stermán et al., 1974). Furthermore, a statistically significant depression of neck EMG tonus accompanying SMR has been demonstrated (Chase and Harper, 1971). With human subjects, Stermán and colleagues similarly report that, "During production of this pattern the trained subject was relaxed, attentive, oriented to the task and relatively motionless... Respiration was regular and sometimes increased in depth, while muscle tone as indicated by the chin EMG was low and stable" (Stermán et al., 1974). However, again, no data have yet been provided to give evidence of any direct link between motor suppression and the production of human 12-16 Hz rolandic EEG which would support the hypothesis that it is functionally equivalent to feline SMR.

4:2:2 Method

Apparatus and Recording

The 12-16 Hz detection and feedback system, with the modifications for artifact discrimination and response duration analysis as described in the previous section, was used for this study. An amplitude window from 5 μ V for the 12-16 Hz threshold to 50 μ V artifact threshold was prescribed. Criterion duration was initially set at 250 msec (as few subjects appeared to exhibit longer bursts of 12-16 Hz) and was increased in increments of 50 msec during training in order to shape the response.

Rolandic EEG was recorded from a bipolar derivation, with one electrode placed midway between C_2 and C_3 (10% of the interaural distance lateral to the vertex) and the other midway between C_3 and T_3 (30% of the interaural distance) (Stermán, 1973a; 1974; Stermán et al., 1974). Occipital EEG (O_1-A_1) and eye-movement (Fp_1-A_1) channels were also recorded. Silver / silver-chloride disc electrodes were used as before.

During some preliminary recordings, a number of different electromyographic leads were examined. Electrodes were placed over the

chin, neck, forehead and forearm (flexor and extensor) muscle groups in the positions described by Lippold (1967). In order for the EMG levels to be meaningfully quantified, it is necessary to differentiate between muscle tonus and the large transitory increases in motor unit firing that accompany contractile movements. The forehead (frontalis muscle) lead was found to be the least susceptible to phasic increases due to movements, and hence was selected for this study. A very low time constant of 0.03 sec was used for the EMG recording to minimize eye movement and electroencephalic artifact. Although some EEG activity could often be discerned in this EMG signal, it is a much smaller and more consistent source of contamination than the effects of muscle contraction. Silver / silver-chloride disc electrodes were attached with self-adhesive discs 1 inch up from the eyebrow and 1 inch either side of the midline. A gain of 100 μ V per centimetre and minimum high-cut filtering (150 Hz) were selected for the recording.

A zero-resetting integrator was constructed to enable the EMG signal to be quantified. The integrator was based on a circuit provided by Emde and Shipton (1974). The signal is passed through a full-wave rectifier (to give a non-zero mean value) to a Miller feedback circuit with a time constant of 20 msec. When the analogue integral equals a reference voltage the circuit is reset and an output pulse produced. This pulse was used to drive an electro-mechanical counter. The number of pulses per unit time is thus a direct index of the mean absolute amplitude of the signal on an arbitrary, but linear, scale. This integrator has a number of advantages over similar, older designs. First, the use of stable, high-gain operational amplifiers in the device greatly reduces errors arising from non-linearity and drift (Pearce and Shaw, 1965). Secondly, an optical coupler is employed to reset the integrator in place of the more common relay, which eliminates problems of contact bounce and transition time.

In addition, rectified and smoothed EMG signal was displayed on the

paper record as a fluctuating DC voltage, so that any depression of muscle tone accompanying bursts of 12-16 Hz EEG would be apparent. A high gain and 0.7 sec time constant were used to emphasize small variations in the absolute amplitude of the signal. Forearm and chin EMG leads were sometimes used for this display, as well as the frontalis EMG, because of their greater range of sensitivity.

Subjects and Procedure

Six volunteer undergraduate students (three male and three female) served as subjects. It was not possible to use a larger sample, nor to include a separate control group, since EEG facilities were only available for one day per week at this time.

Each subject received one training session per week for five weeks (the time remaining before the end of the University term). Each session consisted of six five-minute training trials, together with one five-minute baseline period at the beginning of the session, and one at the end. Feedback was not provided during the fifth session so that a comparison of control over 12-16 Hz activity without feedback information could be made.

After the subjects were given a general outline of the nature of the investigation, they were instructed to attempt to turn this feedback tone on as much as possible, and to obtain as high a score of criterion responses on the digital display in front of them as they could. During the course of the sessions they were encouraged to try various strategies so as to find the most effective means of enhancing SMR. If any prolonged episodes of 12-16 Hz activity occurred the subjects were interrupted and asked to recount their subjective experience.

4:2:3 Results

The product of the number of criterion responses and the criterion duration was computed to give a score (in seconds) of criterion 12-16 Hz

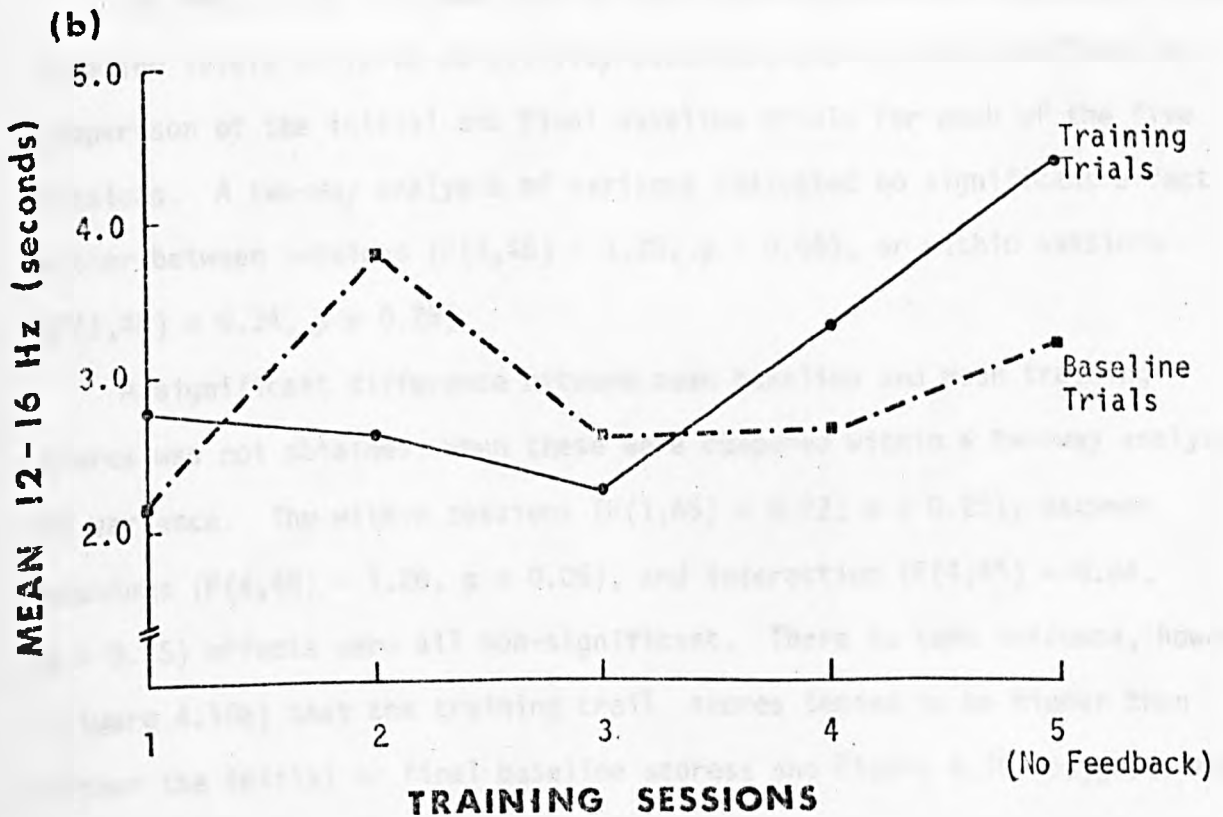
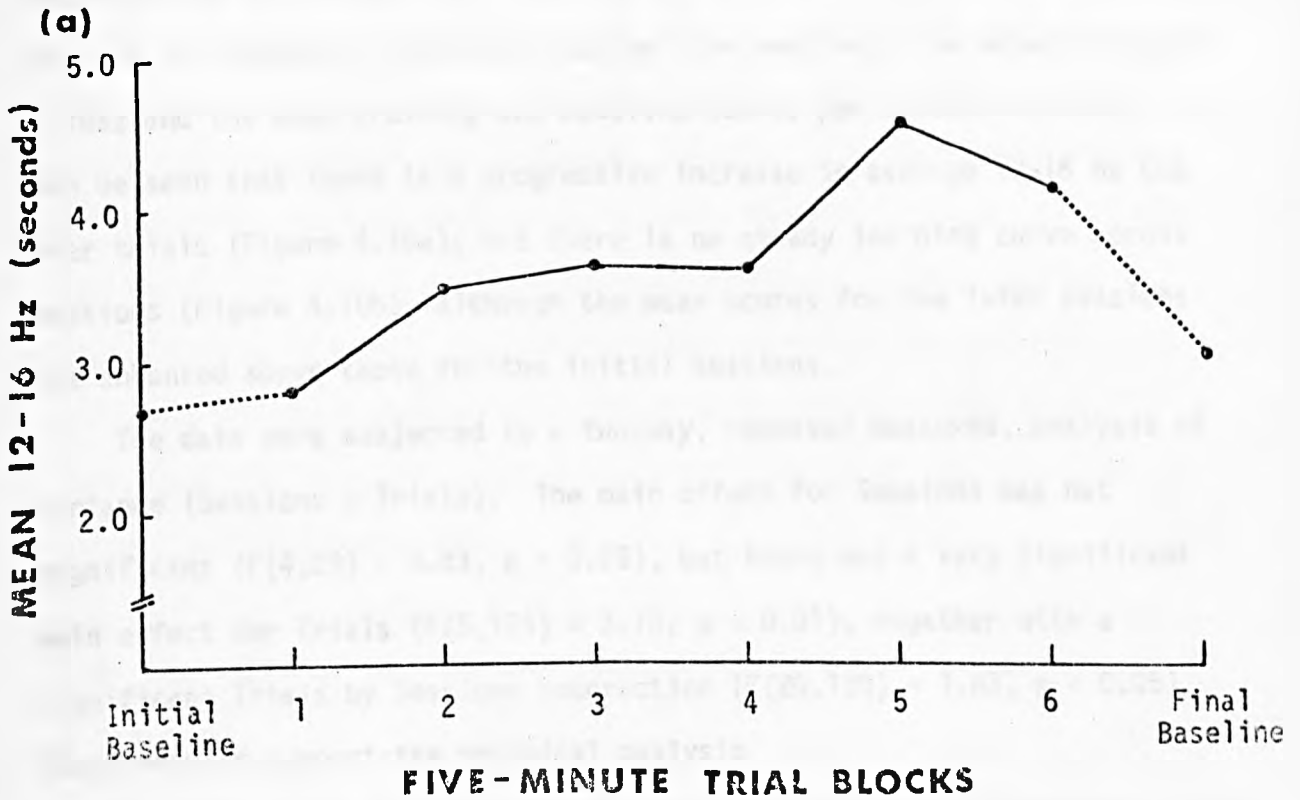


Figure 4.10

(a) Group mean abundance of 12-16 Hz activity (number of criterion responses \times criterion duration) against trial blocks, averaged over the five training sessions

(b) Mean baseline and training trial scores for the five sessions.

activity for each five-minute training and baseline trial. The mean scores for the six subjects, pooled across the five sessions, are shown in Figure 4.10a; and the mean training and baseline scores per session in 4.10b. It can be seen that there is a progressive increase in average 12-16 Hz EEG over trials (Figure 4.10a); but there is no steady learning curve across sessions (Figure 4.10b), although the mean scores for the later sessions are enhanced above those for the initial sessions.

The data were subjected to a two-way, repeated measures, analysis of variance (Sessions x Trials). The main effect for Sessions was not significant ($F(4,29) = 0.83, p > 0.25$), but there was a very significant main effect for Trials ($F(5,125) = 3.18, p < 0.01$), together with a significant Trials by Sessions interaction ($F(20,125) = 1.83, p < 0.05$). These results support the graphical analysis.

The data plots indicate that no consistent spontaneous increase in the baseline levels of 12-16 Hz activity occurred, and this was confirmed by a comparison of the initial and final baseline trials for each of the five sessions. A two-way analysis of variance indicated no significant effect either between sessions ($F(4,45) = 1.38, p > 0.05$), or within sessions ($F(1,45) = 0.34, p > 0.25$).

A significant difference between mean baseline and mean training scores was not obtained when these were compared within a two-way analysis of variance. The within sessions ($F(1,45) = 0.22, p > 0.25$), between sessions ($F(4,45) = 1.26, p > 0.05$), and interaction ($F(4,45) = 0.84, p > 0.25$) effects were all non-significant. There is some evidence, however, (Figure 4.10a) that the training trial scores tended to be higher than either the initial or final baseline scores; and Figure 4.10b suggests that this effect mainly arose during the last two sessions.

When the data were examined on an individual subject basis, it was revealed that there were large inter-subject differences in performance, as

found with alpha feedback training, sufficient to account for the lack of a statistically significant difference between the pooled training and baseline scores of the group. In Table 4.1 the baseline and training scores for each subject are presented, together with percentage change. Three subjects are seen to have exhibited a negative average change from baseline levels during training, whereas subject B.S. demonstrated an average change of +478%. The data further indicates that intra-subject differences between sessions are also very pronounced. None of the subjects demonstrated all appropriate, or all inappropriate, responses to training across the sessions.

Table 4.1. Average baseline and training trail scores per session, together with percentage change, for each of the six subjects.

SESSION	CONDITION	SUBJECT					
		B.S.	S.L.	P.M.	J.H.	J.T.	K.S.
1	Baseline	1.40	1.62	1.25	2.27	2.97	3.62
	Training	4.00	2.72	2.08	2.56	1.25	4.04
	% Change	+185.7	+67.9	+66.4	+12.8	-57.9	+11.6
2	Baseline	4.85	3.10	7.65	4.00	0.85	2.25
	Training	2.49	2.97	5.38	2.55	1.14	1.96
	% Change	-48.6	-4.2	-29.7	-36.3	+34.1	-12.9
3	Baseline	0.97	4.50	4.62	2.82	1.50	1.00
	Training	2.00	2.10	5.12	2.16	1.00	1.12
	% Change	+106.2	-53.3	+10.8	-26.9	-33.3	+12.0
4	Baseline	0.52	4.20	2.85	3.85	1.00	3.45
	Training	5.09	2.70	4.82	2.55	1.37	3.35
	% Change	+878.8	-35.7	+64.1	-33.8	+37.0	-2.9
5	Baseline	0.30	3.15	9.15	2.70	0.37	3.37
	Training	4.11	3.55	11.90	2.05	0.42	4.08
	% Change	+1270.0	+12.7	+30.0	-24.1	+13.5	+21.1
Means :	Baseline	1.61	3.31	5.10	3.12	1.34	2.74
	Training	3.54	2.81	5.86	2.35	1.04	2.71
	% Change	+478.4	-2.5	+29.3	-21.7	-1.3	+28.9

Evidence for SMR

Clear episodes of 12-16 Hz rhythm could occasionally be found in the raw EEG of subjects B.S. and P.M. During one session for subject B.S. the spindles exceeded 25 μ V in amplitude and stood out clearly from the background EEG, as shown in Figure 4.11a. Sterman (1973a; 1973b) has similarly reported bursts of SMR greater than 20 μ V in the raw EEG of one non-epileptic (quadriparetic) subject. Unfortunately, a fuse blew in the pre-amplifier of the occipital EEG channel during this session, and so it is not known the extent to which this high amplitude burst of central 12-16 Hz might reflect rostrally conducted high frequency alpha components. However, when this subject was asked to close her eyes in a later session, it was clear that the incidence of central 12-16 Hz activity was independent of the amplitude of the occipital alpha, as demonstrated in Figure 4.11b, and that the latter was of a slower frequency. Furthermore, subject B.S. was one of the subjects later selected for the study of the topographic distribution of 12-16 Hz activity (*see* 4:1:3), and her data indicated that the primary source was in the post-rolandic region (Figure 4.6). No durable link between occipital and central EEG spindles was observed for any of the other subjects in this investigation.

When the EMG data were examined, there was no evidence of any correlation between the abundance of rolandic 12-16 Hz activity and the integrated frontalis EMG scores. The Pearson product-moment correlation between mean 12-16 Hz and EMG scores per session was extremely low ($r = -0.009$). In addition, the high gain, absolute amplitude recording did not show any transient decrease during the presence of 12-16 Hz bursts, even when these were of high amplitude as in the EEG of subject B.S.

Subjective Reports

The behavioural strategies that subject B.S. associated with her marked enhancement of 12-16 Hz activity superficially suggested motoric

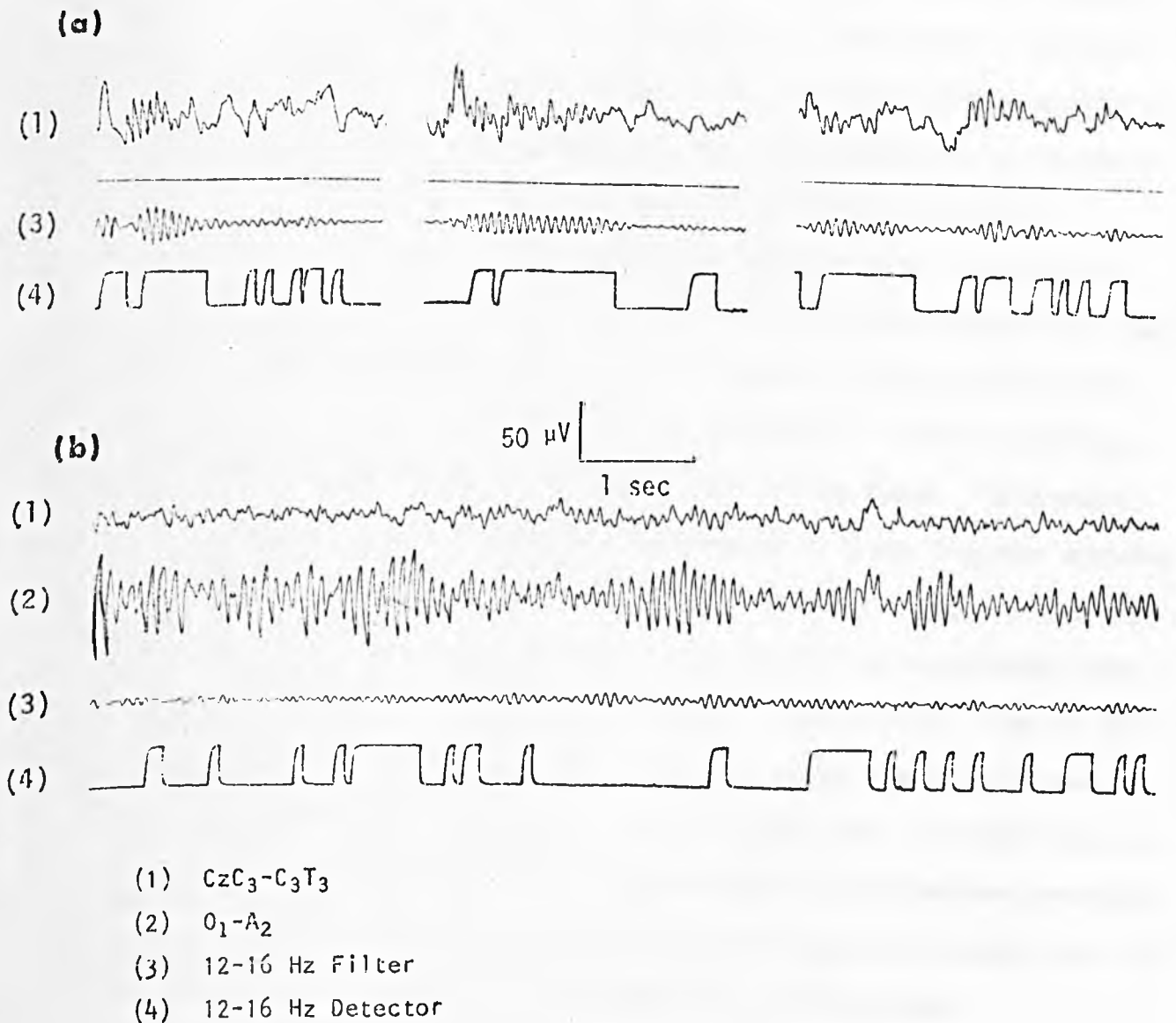


Figure 4.11

(a) High amplitude bursts of 12-16 Hz rhythm in the rolandic EEG of subject B.S. (The occipital channel was inoperative for this session.)

(b) Record from a later session demonstrating that the incidence of 12-16 Hz activity in the rolandic EEG is unrelated to the amplitude of the occipital alpha spindles.

involvement. She reported that she achieved control over the feedback tone by imagining such activities as "swimming, skiing, horse-riding, or sex". She emphasized that it was necessary for her to obtain a state of mind in which she could "feel" herself participating in these activities, and not merely passively thinking of them. The subjective correlates of SMR have been similarly stated by Sterman (1974) to be mentally experiencing particular motoric events involved in activities such as horse-riding, playing tennis, and skiing. However, when subject B.S. was questioned more closely, she noted that it was not the actual imagined movements that were associated with the increase in criterion activity, but rather the "whole scene and atmosphere" of the image. Furthermore, she stated that she had to continuously change from one imagined activity to another in order to maintain the effect, and that thinking about any complex situation or events in this manner was effective, provided that she could maintain a "sufficiently detached frame of mind". Two of the other subjects similarly reported that the strategy required to produce feedback was a relaxed, detached state, together with "flicking from one thought to another" (J.H.), or "switching ideas quickly between unrelated topics" (K.S.). They too said that daydreaming was not enough, but that they had to be "immersed in the thoughts" or "feeling them".

Four of the six subjects claimed that they could recognise the subjective state associated with the criterion responses, but found it very difficult to voluntarily produce this state. Generally, these subjects reported that they had to be able to relax and to forget about the experiment in order to be able to influence their scores. Subject P.M., for example, stated that he attempted to "hold the state of mind that accompanied the bursts", but that he was distracted by the feedback tone because it reminded him of the task. The subjects were also observed to exhibit some oscillation in their trial scores, which apparently stemmed

from a tendency of the subjects to try to analyse their behaviour if they had achieved a substantial increment in feedback (this was, in fact, encouraged by the experimenter).

In the fifth session, when the subjects were asked to attempt to enhance their scores without the aid of feedback, the data indicate that the subjects, on average, performed better than during the preceding sessions (Figure 4.10b); and only one subject (J.H.) exhibited a negative change from 12-16 Hz baseline level. It was apparent from the reports of the subjects that they generally felt less problem oriented and more relaxed during this session.

4:2:4 Discussion

These results confirm that central 12-16 Hz EEG components can be experimentally enhanced through feedback training. Although there was no consistent progressive increase in the abundance of 12-16 Hz across sessions for any of the subjects, one subject evidenced a very pronounced ability to augment her 12-16 Hz above baseline levels, and with further training sessions it is probable that inter- and intra-subject variance would be diminished so that a more stable trend could be demonstrated.

It is debatable, however, whether these 12-16 Hz components represent a homologue of feline SMR, or are just one aspect of central cortical synchronization. The presence of 12-16 Hz waves in the EEG does not necessarily imply that they represent a functionally discrete EEG entity such as the alpha rhythm, or that they arise from an "independent neural process" (Sterman et al., 1974). A band-pass filter set to *any* frequencies within the normal EEG range will produce some spindling output, due to the complex morphology of the EEG waveform¹; a fact which appears to be

¹*Practically all physically realisable time-varying signals can be decomposed into various elementary sinusoidal components (see 5:2:2).*

consistently overlooked by those workers, such as Sterman, who wish to attach special significance to "invisible" rhythms filtered from the EEG. Moreover, the demonstration of the enhancement of 12-16 Hz activity by feedback training does not, of itself, afford this activity with any special functional significance; since it only shows that subjects can learn to selectively modify the psychophysiological substrate that gives rise to a particular periodicity of thalamo-cortical traffic.

At the neurophysiological level, the 12-16 Hz activity represents a particular localized shift in the complex balance of thalamic excitation and inhibition (*see* 2:2:2). It is quite probable that the imagination of various motor activities during a state of reverie would involve strong inhibition within the CNS in order for the subject to remain motionless. There is no reason *prima facie* why this functional inhibition should be equivalent to the motor inhibition that accompanies the occurrence of SMR in the cat. On the contrary, the rolandic mu rhythm of man, which is similarly associated with inhibition of sensory and somatomotor representation in the sensorimotor cortex (Gastaut et al., 1965, *see* 2:4:2), is disrupted by the imagination of motor activity (Gastaut et al., 1965; Chatrian et al., 1959).

A characteristic behavioural correlate of feline SMR is motor quiescence (Roth et al., 1967), but there is no evidence in this study of an equivalent function for central 12-16 Hz. Sterman similarly reports that he failed to observe any concomitant changes in either EMG or heart rate during the production of SMR in humans (personal communication, 1974). One difference between the human and the cat SMR training studies is that the bursts of 12-16 Hz activity are far smaller in amplitude and duration than those recorded from the cortex of the cat, and hence any related changes in other physiological variables might therefore be less apparent (Sterman, 1974). One approach by which this problem might be overcome would be to employ averaging techniques, with the signal averager triggered

from the SMR detection system. This technique could perhaps be similarly used to look for small, time-locked variations in heart rate, galvanic skin response, or other EEG leads.

In addition, EMG is usually recorded from electrodes implanted in the neck musculature of the cat, whereas various other surface leads have been employed in the human studies, and Malmo (1962) has shown that EMG from one lead may not necessarily relate to that from another. Nevertheless, if the integrated frontalis EMG scores in this study are regarded merely as a crude index of motor excitability, then they indicate that this is not related to the abundance of central 12-16 Hz activity, nor has any other empirical support for such a relationship yet been offered elsewhere.

The reports given by the subjects indicated that the appropriate behavioural response required for the generation of the 12-16 Hz activity is a relaxed, detached state, combined with a certain level of focused attention. Although it would be unwise to attach too much credence to subjective reports in the feedback training paradigm because of the problem of 'superstitious learning', nevertheless, it is possible that a combination of relaxation and strategies such as switching attention from one topic to another might enable a subtle regulation of arousal so as to provide the appropriate level of localized thalamic drive for 12-16 Hz synchronization in the rolandic cortex. This hypothesis is supported by the observation that the subjects in this study often tended to experience long periods during which they obtained very few criterion responses; but if a long burst of feedback suddenly occurred, a train of further criterion responses would then follow, as if the subject had aroused himself from passive reverie and restored concentration. This effect was still seen even if the initial long burst of feedback was obtained falsely (such as when I decreased the criterion duration), indicating that the subjects had not merely switched to a successful behavioural strategy.

Similarly, the effect of the demand characteristics of the feedback training paradigm could be to cause a disruption in the overall delicate balance of psychophysiological arousal, and so produce the frequent inappropriate responses that confounded the statistical analyses of the group data, as with training of the alpha rhythm. Presumably, some subjects find it easier to become less problem-oriented than others, although the performance also varied within the individuals across the sessions.

The data from the fifth training session demonstrated that the subjects were generally able to perform as effectively, or more so, without feedback information than with it. Travis and co-workers in a study of alpha training (Travis et al., 1975a) similarly report that there was no tendency for alpha to diminish when feedback was withheld, provided that the subjects were forewarned that feedback would be discontinued. A number of workers have suggested that feedback information serves only to supplement instructions to modify EEG activity (Beatty, 1972; Hord and Barber, 1971; Prewett and Adams, 1976). Hence an empirical determination of the extent to which feedback itself is involved in the acquisition of control over the alpha rhythm was recently carried out by Plotkin (1976a). Groups of subjects were given either oculomotor instructions (to blur and not focus), cognitive instructions (to relax, "let go", become "non-critical" and allow themselves to review pleasant experiences), or contingent feedback; whilst other groups received both feedback of the alpha rhythm and one or the other set of instructions. It was found that there was a highly significant difference in the enhancement of alpha between subjects who had received feedback and those who had not, and this was independent of the type of instructions that accompanied the feedback. Moreover, the subjects who received the oculomotor instructions alone were as successful as those who received feedback alone, and significantly more successful than those who received cognitive instructions alone; but the

subjects who received both feedback and oculomotor instructions were much more successful than those who had received only one or the other.

These results illustrate that feedback training, by providing knowledge of results, enables the individual to more effectively gain control over a covert response. However, the data from the present study, and the alpha feedback study described previously (*see* 3:2:4), also illustrate that, in the case of the EEG, the feedback training procedure itself tends to induce a reaction opposing an increase in synchrony which must be first overcome in order for the feedback information to be of any use. Furthermore, although feedback can indicate the most effective strategies for producing the desired response, it does not follow that the individual is thereby necessarily able to voluntarily perform those strategies, as noted in this study. Thus the process of learned control of an EEG response involves both the perceptual differentiation of the behavioural state associated with the EEG response, and the development of skill in selectively attaining that state.

A pilot study of SMR feedback training was carried out with the alpha detection and feedback system modified to respond to 12-16 Hz activity of 5 μ V or more. EEG was recorded from electrodes placed over the rolandic area in a number of subjects. In all cases 12-16 Hz activity was detected. However, it was not clear the extent to which this activity represented an independent EEG entity, and not merely rostrally conducted occipital EEG components. Also, there was considerable contamination of the data by artifact, owing to the low threshold amplitude. To minimise this problem, response discrimination circuitry was added to the SMR detection system.

An investigation was carried out with a group of normal subjects to determine firstly, whether the incidence of central 12-16 Hz could be enhanced by feedback training; and secondly, whether this activity is related to motor quiescence as in the cat, by simultaneous measurement of integrated frontalis EMG and high gain, absolute amplitude EMG recording.

Six subjects received one thirty-minute feedback training session a week for five weeks. A five-minute baseline recording was also taken at the beginning and end of each session. Feedback was given by a tone signal and digital display whenever a 12-16 Hz burst, that exceeded amplitude and duration criteria, occurred in the rolandic EEG.

There was a very significant enhancement of mean 12-16 Hz within sessions, together with a variable increase across sessions. However, the mean level of 12-16 Hz during training did not differ significantly from baseline, even though there were no consistent changes in the mean baseline measures, either within or between sessions. The individual data showed that there were large inter-subject differences in performance, and also intra-subject differences across sessions. A suppression of 12-16 Hz activity below baseline levels was often evident.

One subject demonstrated an average enhancement of 12-16 Hz EEG of more than 400% above baseline levels; bursts of 12-16 Hz could also be sometimes seen in the EEG of this subject, and appeared to be quite independent of occipital spindles. The primary generator source was shown to lie in the post-rolandic cortex by a subsequent topographical study. However, in other subjects it was found that the locus of 12-16 Hz activity was in anterior or posterior regions.

No evidence was found of a relation between central 12-16 Hz activity and motor inhibition or behavioural quiescence, as indicated by EMG measures, to support the hypothesis of a functional similarity to feline SMR.

It is concluded that the 12-16 Hz components are most likely an epiphenomenon of the EEG and not the manifestation of a functionally distinct neuronal process. It is suggested that subjects enhance the 12-16 Hz activity recorded from their rolandic EEG by a subtle modulation of the level of psychophysiological arousal, so as to bring about a state predisposing toward thalamo-cortical synchrony at this particular frequency. This hypothesis is supported by the type of behavioural strategies that subjects felt to be associated with an increase in feedback; and by the fact that the incidence of 12-16 Hz was greater when the subjects were less problem-oriented, such as in the final training session when feedback was absent.

5:1 DESIGN OF EEG FEEDBACK TRAINING UNIT

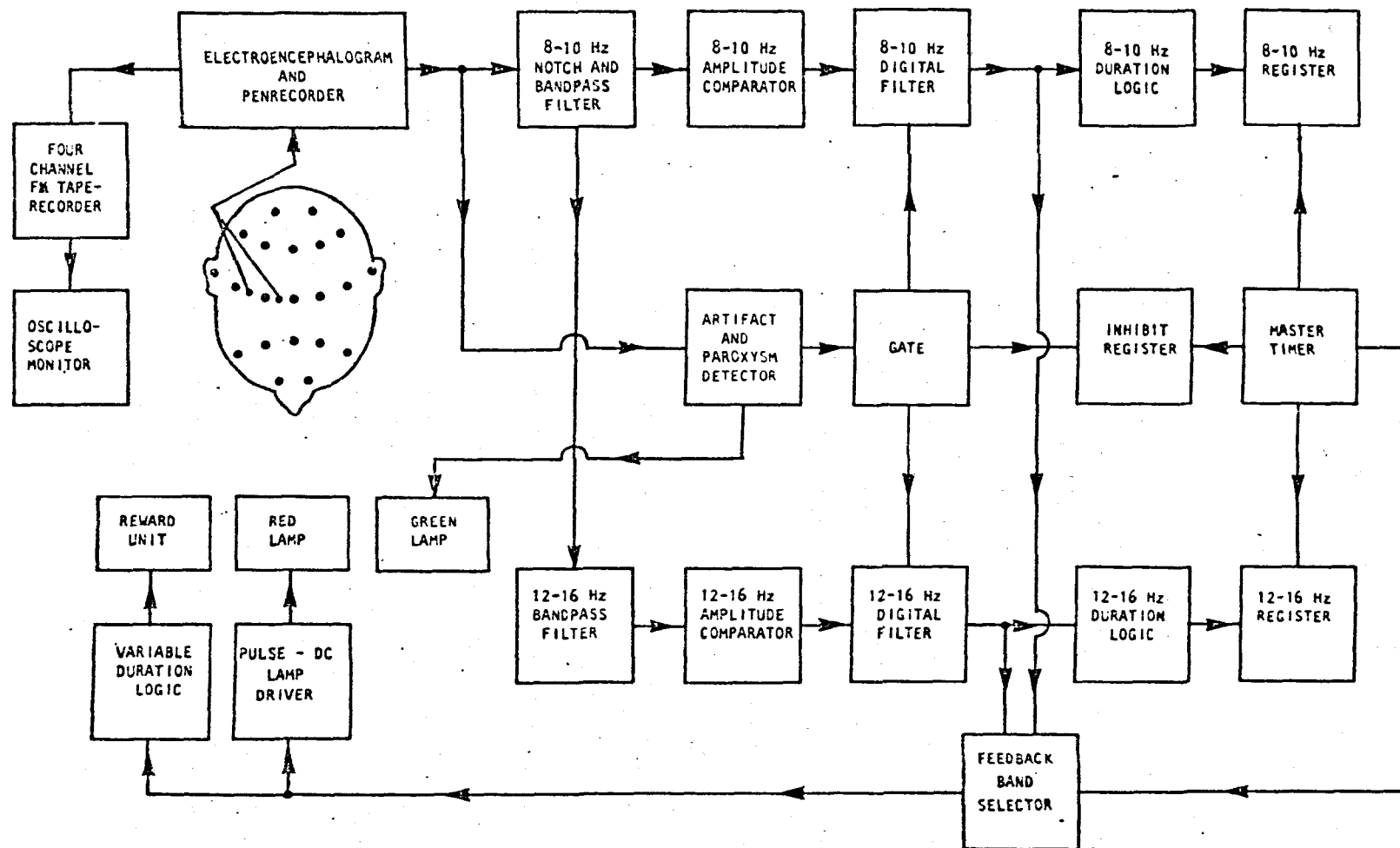
5:1:1 Introduction

For the clinical study of the effects of SMR feedback training on epileptic subjects, it was necessary to assemble other feedback training apparatus, since much of the essential equipment used in the previous studies could not be removed from the laboratory. The filter, quantification and feedback systems were designed completely 'from scratch', drawing on the experience gained from the previous work, in order to ensure that the response of the apparatus would accurately reflect the presence of frequencies within the SMR range in the EEG.

A dual-band filter and detection network was developed that simultaneously quantified both 12-16 Hz and 8-10 Hz EEG activity so that the specificity of the effects of SMR training could be investigated (*see* 6:5:1). Several devices were incorporated for the presentation of feedback of either frequency band, and of high voltage artifactual or paroxysmal activity. In contrast to the flexible rack-mounted modular form of the laboratory apparatus, the feedback training unit for the clinical study was designed to be compact and portable. Front panel controls were provided for the selection of system parameters, and various penrecorder and tape outputs externalized on the rear panel. The various considerations involved in the design of this apparatus will be discussed below. The development of computer facilities to supplement the analysis of the EEG data will also be described.

A block diagram of the complete recording, detection, and feedback system used for the clinical study is provided in Figure 5.1. A comparison

Figure 5.1. Flow diagram of the apparatus used for the clinical study. The EEG signal is fed into specially designed 8-10 Hz and 12-16 Hz analogue filter networks. Each frequency band is then further processed digitally and quantified by duration logic circuitry. Continuous feedback of the occurrence of 12-16 Hz (or 8-10 Hz) activity in the EEG is given by a red lamp. In addition, a discrete reward is given whenever the response exceeds a criterion duration. The detection and feedback circuits are inhibited during the presence of artifactual or paroxysmal activity above a prescribed amplitude; feedback of this activity is provided by a green lamp. All the registers and feedback devices are controlled by a master timer, which is used to precisely determine experimental intervals.



of the detection and feedback systems employed by other workers for investigating the effects of SMR feedback training on epilepsy is included as an appendix to this thesis. In several instances the equipment is open to criticism because of excessive frequency selectivity, or susceptibility to the effects of artifact.

5:1:2 Analogue Filters

The design of a filter to accurately distinguish low amplitude 12-16 Hz activity in the EEG and discriminate against much higher voltage waveforms of only a few hertz difference, is a difficult exercise, complicated by the plethora of transient signals in the EEG, particularly in that of epileptic patients, and by the problem of finding a suitable compromise between various opposing characteristics of analogue filters, each of which can be responsible for producing misleading data¹. A signal is modified in both amplitude and phase when passed through a filter network so that a delay in the response is introduced (Figure 5.2a). The time taken for the filter to reach full amplitude is inversely related to the bandwidth, which means that the time constant is increased for a narrow filter, and that it will not respond fully to bursts of input below a certain duration. Furthermore, it is an inherent characteristic of frequency tuned circuits that there is both a steady-state and a transient response. Thus a band-pass filter will tend to resonate or 'ring' at the peak frequency in response to any sudden change in the input level, as illustrated in Figure 5.2b. The transient response is also a function of the bandwidth of the filter so that a narrower filter will oscillate for much longer in response to a transient input than a broader band filter.

¹*For an analysis of the properties of analogue filters see Magrab and Blomquist (1971).*

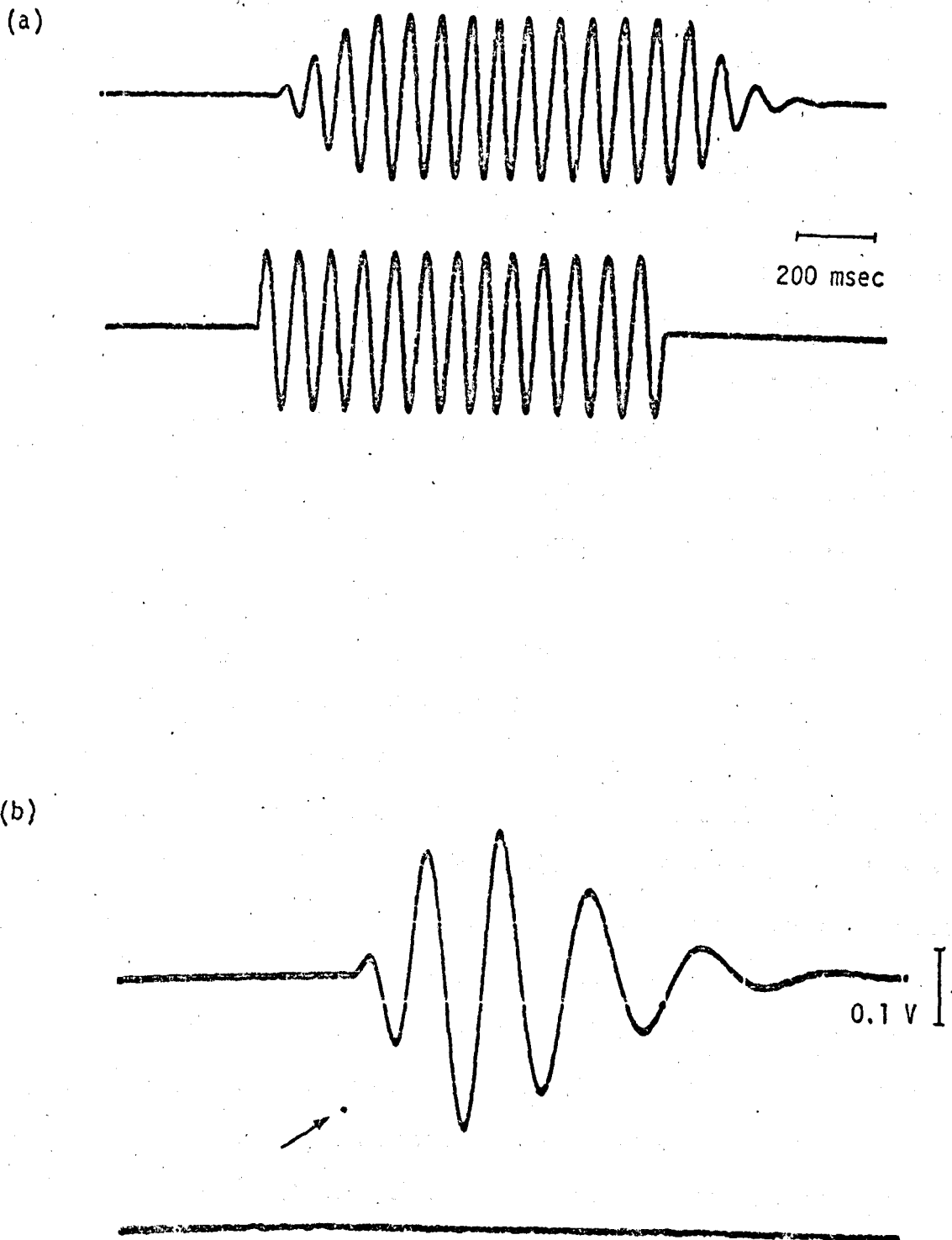


Figure 5.2

(a) Oscillogram depicting phase lag and delay in amplitude response, for a 1 sec burst of 13 Hz sine wave, introduced by the 12-16 Hz analogue filter that was incorporated in the equipment for the clinical study.

(b) Oscillogram illustrating the transient response ('ringing') of the 12-16 Hz filter to a 1 msec, 10 V pulse at the input (arrowed).

For non-ideal filters, the amplitude response is not constant, but varies with frequency. The bandwidth is therefore defined as those frequencies at which the steady-state amplitude response has fallen to $1/\sqrt{2}$, or 3 dB, of its mean value. As the attenuation slope of a practical filter is increased so that it more closely approximates the rectangular shape of a hypothetical ideal filter, the effective bandwidth is correspondingly reduced. Hence, the greater the attenuation slope of a filter, so also greater is the response time and the transient response.

It therefore follows that if a band-pass filter for the detection of SMR is made with a very sharp rate of attenuation in order to prevent high amplitude out-band signals from contributing to the output, the filter is thereby more susceptible to generating spurious output to activity such as muscle spikes, epileptiform sharp waves, and electrical artifact. As shown in Figure 5.2b, it is possible for just one short impulse to produce a signal of several cycles at the output of the filter, which is indistinguishable from genuine in-band activity.

When the specifications of the SMR detection apparatus used by other groups are closely examined (*see* Appendix), it is found that the design of the SMR filter units is often such that strong suspicions are raised about the validity of the data. This is for two main reasons:

(1) The SMR filters often have steep attenuation slopes and high Q-factors so that considerable contamination of the output from the effects of sharp waveforms would be expected. Some workers have included no protection in their systems against such spurious signals; others have only protection against high voltage slow wave activity.

(2) The bandwidth of the filter that is used to detect SMR is in some cases quite inappropriate. In the cat the frequency band of SMR has been defined as 12-20 Hz (Sterman and Wyrwicka, 1967; Wyrwicka and Sterman, 1968), 12-18 Hz (Bouyer et al., 1974), 12-16 Hz (Roth et al., 1967; Howe

and Sterman, 1972; 1973), or 12-14 Hz (Babb and Chase, 1974; Chase and Harper, 1971). In human studies the frequency range of filtered SMR has been described as 12-16 Hz (Sterman, 1974; 1976b), 12-15 Hz (Lubar, 1976; Sterman 1976c; 1977a), 12-14 Hz (Sterman et al., 1974; Kaplan, 1975; Seifert and Lubar, 1975), or 11-13 Hz (Sterman and Friar, 1972; Finley et al., 1975). These various bandwidth specifications are very misleading, however, since often they bear no relation at all to the conventional definition of bandwidth (*supra*), and obscure the fact that the SMR filters were tuned to a much narrower frequency band. Finley et al. (1975), for example, state that their SMR filter was tuned to respond sharply to $12 \text{ Hz} \pm 1 \text{ Hz}$; but they also provide the frequency response curve of the filter and note that, "According to the curve a 11 Hz signal would have to achieve a voltage of approximately 10 times that of the peak frequency in order to activate the feedback apparatus". Similarly, Sterman (1976b) gives the frequency response characteristics of the "12-14 Hz" filter employed by Sterman et al., (1974), which indicates that there was a four-fold (12 dB) attenuation at the 12 Hz and 14 Hz points. It is clearly confusing to talk of training 11-13 Hz or 12-14 Hz activity when feedback is, in fact, given for a very narrow peak frequency, even at signal amplitudes two or three times above threshold level.

It is also very improbable that any discrete rhythm in the EEG should consistently appear within such narrow frequency bands across different sessions and subjects. A specific investigation of the training of narrow band EEG in normal subjects was carried out by Sterman et al., (1975), using filters that were tuned to respond to 10 Hz, 13 Hz and 15 Hz, with a rolloff of 12 dB at $\pm 1 \text{ Hz}$ ¹. They report that, "The relatively high

¹Note that this is the same bandwidth as Sterman et al. (1974) apparently used for SMR training in their epileptic patients.

voltage typically associated with occipital 8-13 Hz activity (alpha) was not obtained when narrow band 10 Hz activity was required from the occipital EEG". They add that the voltages were reduced even further for 10 Hz, 13 Hz and 15 Hz narrow band activity from the central area. After providing three training sessions per week for three months, they found that, "Few subjects showed any marked increments in rewarded activity"; and concluded that, "The central nervous system has difficulty in generating such narrow frequency bands".

These results, plus the nature of the relationship between the transient and steady-state behaviour of a band-pass filter (*supra*), suggest that broad-band filtering is more appropriate for a study of feedback training of the EEG than highly selective filters tuned to a particular peak frequency. In addition, Serman et al. (1974) report that peaks of 12-14 Hz, or 14-16 Hz, or both appeared in the EEG spectrum following feedback training; and changes in 13 Hz and 15 Hz activity are said to be generally related (Serman, 1974; 1977a). Therefore, I decided that, in view of the uncertainty in the literature concerning the frequency range of SMR, the most suitable approach would be to filter activity in the 12-16 Hz range. However, to require a band-pass filter to have a flat response over a wide frequency band, with sharp cut-offs on either side and a minimum transient response, is to ask for the characteristics of the ideal filter! Hence a digital filter, as described in the next section, was added to obtain an approximately rectangular frequency response between 12-16 Hz.

It should be made clear that, even if digital filtering is used to obtain precise frequency cutoffs, preliminary band-pass filtering is still necessary, particularly if the digital techniques are based on period discrimination. This is because the EEG half-wavelength intervals are defined by the time between zero-crossing points (i.e. when the two electrodes are iso-electric), and thus baseline shifts and large slow

oscillations must be excluded. Furthermore, low amplitude rhythms superimposed on larger waveforms are ignored, so that any SMR in the EEG would be easily masked by normal background activity, unless this is filtered out. Some subjects exhibit 10 Hz alpha rhythms of 50 μ V or more even over the central regions, so that a preliminary band-pass filter with a sharp initial rolloff is demanded in order for SMR activity in the region of 5 μ V to be detected by zero-crossing analysis. Thus some genuine activity probably remained undetected in the preliminary laboratory studies. Fortunately, the alpha rhythm of epileptics tends to be at lower frequencies, which makes the task of discriminating 12-16 Hz in the EEG easier. On the other hand, epileptics can have diffuse theta peaks in the EEG which can be very large: in two of the subjects used in the clinical study to be described later, for example, 7 Hz waves exceeding 100 μ V were often observed in the rolandic EEG (*see* 6:3).

Commutating, or 'N-path', filters were first explored during the development of the analogue filters. These types of filters enable a sharp cutoff to be achieved, and have the added advantages that they are very easily tunable and do not have the same transient response as conventional resonant filters (Broeker, 1971; Roberts, 1972). The band-pass response is generated by switching between a number N of low-pass filter sections at a rate N times the centre frequency. A 'comb' filter response results with passbands at zero frequency and at various harmonics. By cascading several filters a single response at the desired frequency band can be obtained (Roberts, 1972). A 12-16 Hz filter network was therefore constructed from 8-path, 4-path and 3-path sections. It was found to have very good broad band selectivity; but, unfortunately, it also suffered from a tendency to 'beat' when the input signal was near to the centre frequency. This rendered it unsuitable for this particular application, and so the technique was eventually abandoned.

A system of cascaded second-order filter sections (Sallen and Key,

1955) was developed as an alternative. Three low-pass filters and three high-pass filters were connected in series to provide a band-pass function (Johnson and Hilburn, 1975). The filter sections were individually stagger-tuned so that a flat response between 3 dB points at approximately 11.5 Hz and 15.5 Hz, was obtained. The calibration of the filters was facilitated by supplying a signal from a wobulator and tracing the frequency response (after rectification and smoothing of the filter output) on an X-Y plotter. The overall attenuation slope of the filter was a modest 32 dB/octave; but a specially designed notch filter was also included which steepened the initial low frequency roll-off of the filter to approaching 80 dB/octave, without appreciably increasing the transient response. This notch filter consisted of an 8-10 Hz band-pass filter (formed from four cascaded low-pass and high-pass sections) placed in the feedback loop of a differential amplifier. At resonant frequency the input and output of the band-pass filter are in phase, and hence the output of the differential amplifier is at a minimum. The notch filter was made to simultaneously function as the band-pass filter for 8-10 Hz activity, with an attenuation slope of approximately 16 dB/octave.

The amplitude-frequency characteristics of the complete 12-16 Hz analogue filter network that was used in the feedback unit is plotted in Figure 5.3. The oscillograms reproduced in Figures 5.2a and 5.2b respectively depict the phase and transient response.

5:1:3 Digital Filters

The analogue band-pass filter was insufficient in itself to accurately isolate low amplitude 12-16 Hz EEG. It is evident from Figure 5.3, for example, that an output exceeding 5 μ V could be produced by 10 Hz components as small as 20 μ V (i.e. the attenuation factor is approximately 11 dB). To enable abrupt cutoffs that are independent of amplitude to be

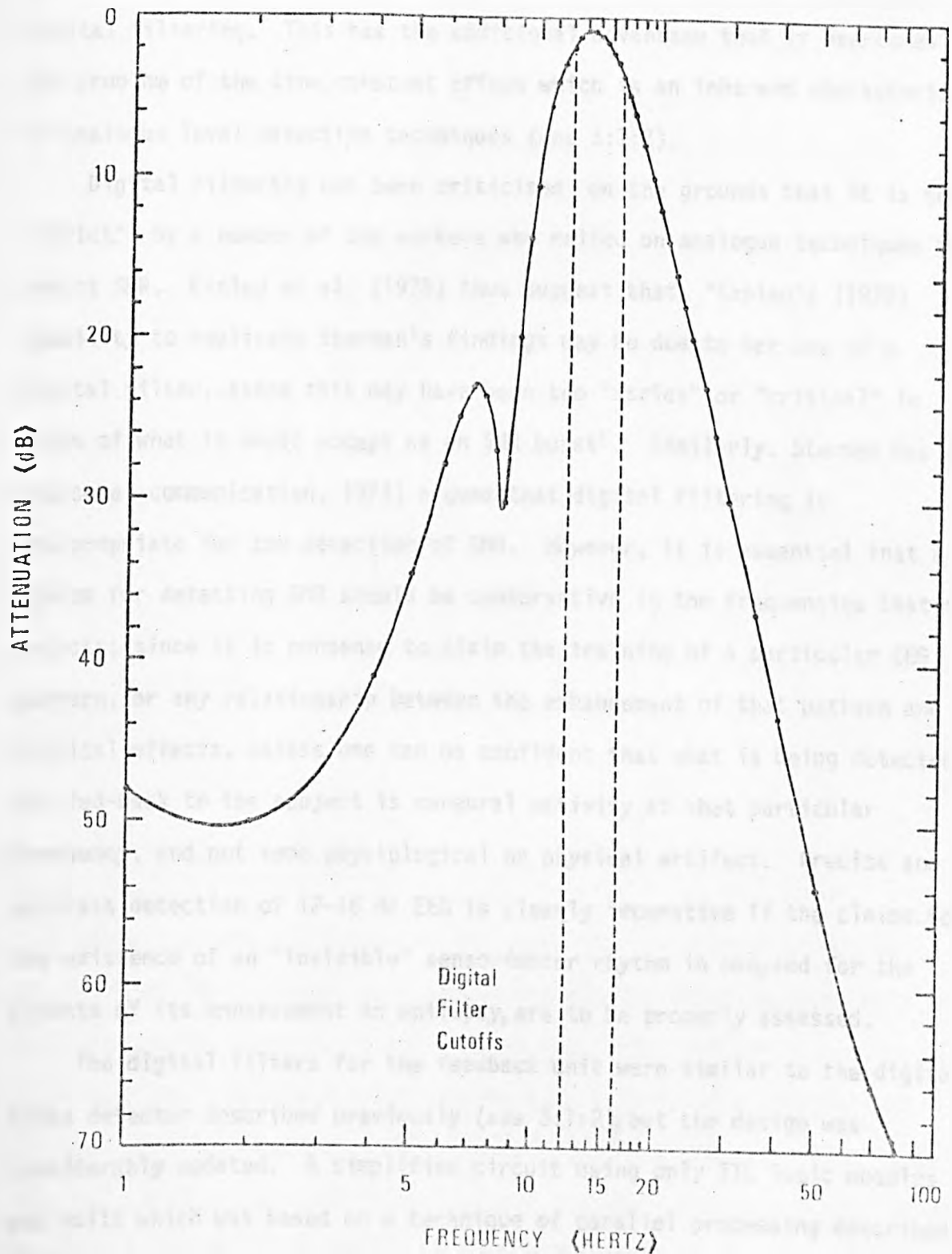


Figure 5.3

The amplitude response of the 12-16 Hz analogue filter. The high initial rolloff away from 12 Hz is produced by the 8-10 Hz notch filter that is included in the network. The dotted lines indicate the step-function cutoff points of the digital filter that was fed from the analogue filter.

obtained, it was therefore necessary to further process the signal by digital filtering. This has the additional advantage that it overcomes the problem of the time constant effect which is an inherent characteristic of analogue level detection techniques (*see* 3:1:2).

Digital filtering has been criticised on the grounds that it is too "strict", by a number of the workers who relied on analogue techniques to detect SMR. Finley et al. (1975) thus suggest that, "Kaplan's (1975) inability to replicate Sterman's findings may be due to her use of a digital filter, since this may have been too "strict" or "critical" in terms of what it would accept as an SMR burst". Similarly, Sterman has also (personal communication, 1974) argued that digital filtering is inappropriate for the detection of SMR. However, it is essential that a system for detecting SMR should be conservative in the frequencies that it selects; since it is nonsense to claim the training of a particular EEG pattern, or any relationship between the enhancement of that pattern and clinical effects, unless one can be confident that what is being detected and fed-back to the subject is cerebral activity at that particular frequency, and not some physiological or physical artifact. Precise and accurate detection of 12-16 Hz EEG is clearly imperative if the claims for the existence of an "invisible" sensorimotor rhythm in man, and for the effects of its enhancement on epilepsy, are to be properly assessed.

The digital filters for the feedback unit were similar to the digital alpha detector described previously (*see* 3:1:2), but the design was considerably updated. A simplified circuit using only TTL logic modules was built which was based on a technique of parallel processing described by Hosek and Wilson (1973). As before, EEG is converted by a voltage comparator into a square wave train whenever it exceeds a prescribed threshold amplitude. The leading edge of each wave is used to enable two retriggerable monostables which provide pulses equal to the maximum and

minimum half-wavelength intervals of the selected frequency range (41.67 msec and 31.25 msec for the 12-16 Hz band). If the input waveform falls within this logic time window an output pulse is generated. This pulse triggers another monostable set at 130 msec, so as to provide a constant output for as long as the criterion frequency is present.

The digital filters were calibrated using a precision sine wave generator, with the period of the signal monitored by a digital timer. In practice, the crisp 12 and 16 Hz cutoffs of the idealized characteristic are virtually impossible to achieve, especially since the calibration procedure involved the simultaneous adjustment of interacting, timing, symmetry and threshold parameters. Nevertheless, a quite close approximation was obtained over a very large amplitude range, with cutoffs at approximately 12-14 Hz, at slightly above the 5 μ V threshold level; widening to 11-16 Hz at 32 dB above threshold (200 μ V), as illustrated in Figure 5.4. A similar digital filter was included for the detection of 8-10 Hz EEG.

5:1:4 Amplitude Discrimination

Because of the low threshold amplitude that is necessary for the detection of 'SMR' in man, an analogue and digital filter network will be very susceptible to inaccuracies arising from transients and noise in the background EEG. As demonstrated above, (see 4:1:3), physiological and physical artifacts can very easily produce apparent SMR activity. These spurious signals are a result both of the transient characteristics of the analogue filter, and of in-band harmonic components of high voltage artifactual waveforms. Therefore, if the data are to have any validity, some means of limiting the response of the SMR detection system is clearly required. The problem is exacerbated in an investigation of the training of SMR in epileptic patients, since the epileptiform EEG is typically full

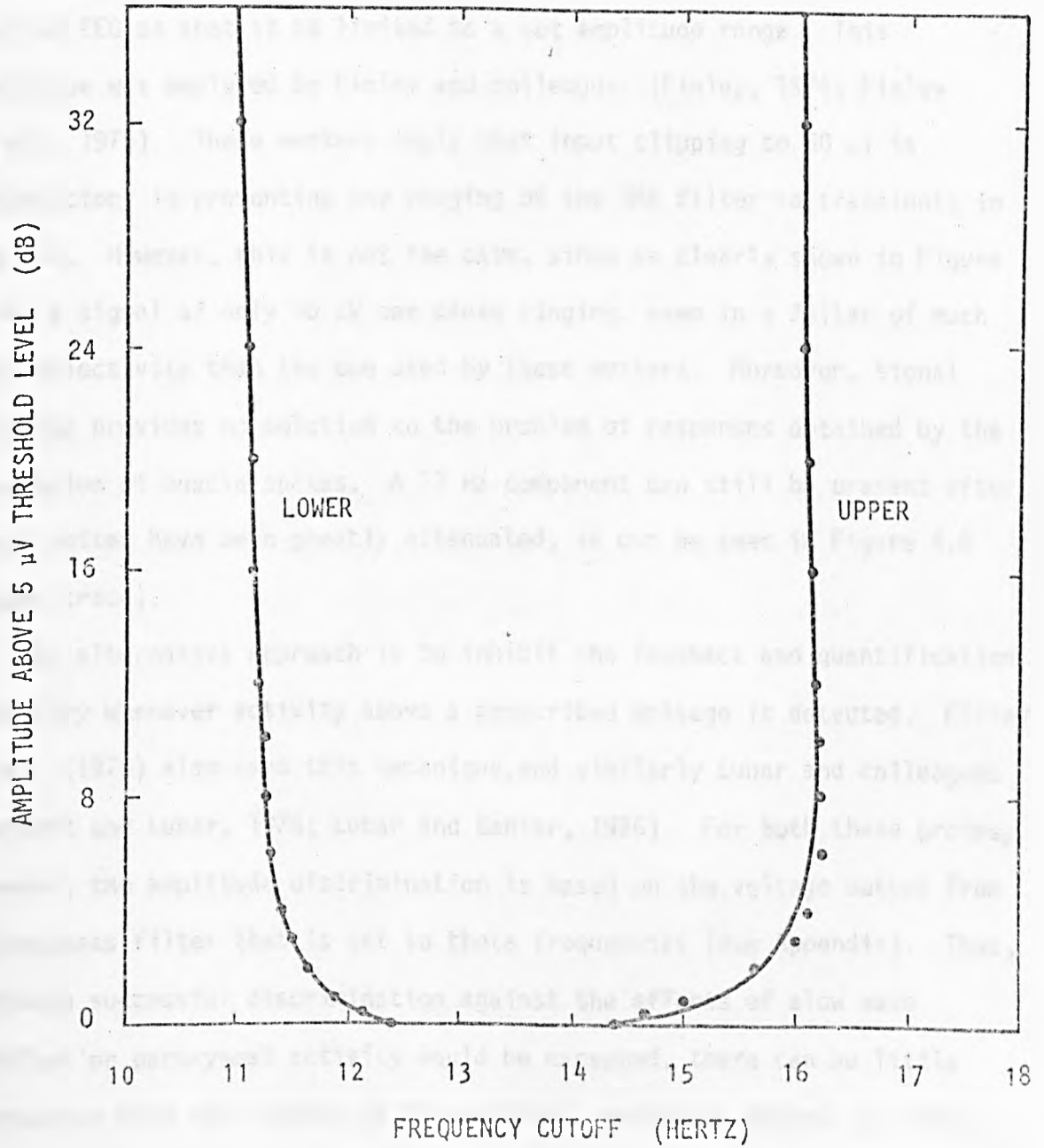


Figure 5.4. The bandwidth of the 12-16 Hz digital filter as a function of amplitude.

of high voltage spikes and paroxysmal discharges.

One seemingly obvious way in which the spurious response to large artifactual and paroxysmal signals could be curtailed is to simply 'clip' the raw EEG so that it is limited to a set amplitude range. This technique was employed by Finley and colleagues (Finley, 1974; Finley et al., 1975). These workers imply that input clipping to 60 μ V is satisfactory in preventing any ringing of the SMR filter to transients in the EEG. However, this is not the case, since as clearly shown in Figure 4.7d, a signal of only 50 μ V can cause ringing, even in a filter of much less selectivity than the one used by these workers. Moreover, signal clipping provides no solution to the problem of responses obtained by the generation of muscle spikes. A 13 Hz component can still be present after these spikes have been greatly attenuated, as can be seen in Figure 4.8 (upper trace).

An alternative approach is to inhibit the feedback and quantification circuitry whenever activity above a prescribed voltage is detected. Finley et al. (1975) also used this technique, and similarly Lubar and colleagues (Seifert and Lubar, 1975; Lubar and Bahler, 1976). For both these groups, however, the amplitude discrimination is based on the voltage output from a band-pass filter that is set to theta frequencies (see Appendix). Thus, although successful discrimination against the effects of slow wave artifact or paroxysmal activity would be expected, there can be little protection from the effects of EMG artifact, epileptic spikes, or other high frequency waveforms. Both Finley and Lubar claim that their inhibit circuitry indicated the presence of epileptiform spikes. However, since an epileptic spike by definition has a duration of 80 msec or less (Storm van Leeuwen et al., 1966), and it has been empirically demonstrated that the spike duration is mostly below 65 msec (15 Hz) (Celesia and Chen, 1976), it is difficult to believe that spikes, poly spike-wave complexes, and other

similar transients did not result in some spurious data.

A simpler and more effective technique is to operate the amplitude discrimination circuitry from the raw EEG so that the response of the SMR detection and feedback system to *any* activity above a prescribed amplitude is inhibited. Both methods, of course, also prevent the detection of any true 12-16 Hz activity that might occur simultaneously with high voltage slow wave artifact or background activity. However, this is desirable in a clinical investigation since, otherwise, it is quite possible that one may inadvertently train an epileptic patient to produce epileptogenic or high voltage synchronous activity. Lubar and Bahler (1976) thus emphasized the importance of providing suitable inhibit circuits on the basis of their observations that if some feedback of SMR was allowed to occur in conjunction with the presence of high voltage slow wave activity, then either a lack of seizure decrease, or even a transient increase, in seizure rate was obtained.

In view of the above considerations, the artifact discrimination circuitry that was developed for the laboratory and clinical studies was based on monitoring the peak amplitude of the raw EEG waveform. The EEG signal was full-wave rectified and fed to a variable threshold voltage comparator. The output of this comparator was interconnected with duration logic, as described in the next section, and also externalized to gate a digital counter which registered the cumulative time that high voltage artifactual or paroxysmal activity was present. A calibrated control was provided on the front panel of the feedback unit which, together with the digital filter threshold controls, enabled an amplitude window for the detection of 12-16 Hz and 8-10 Hz EEG to be accurately established over a wide range.

5:1:5 Duration Analysis

The amplitude discrimination circuitry was initially tested during the preliminary training sessions with the output pulse used to directly gate feedback and counter circuits. It was found, however, that signals with rapid changes of voltage gradient, such as EMG artifact, could still pass through the filter and detection system and thus be registered as 'SMR'. To overcome this problem, response duration analysis, as used by several other workers (Sterman et al., 1974; Finley et al., 1975; Lubar and Bahler, 1976), was added, so that a spindle was required to exceed a criterion duration in order to be registered as a response. The amplitude and duration discrimination circuits were made to interact so that both the small responses generated by transients below the threshold amplitude of the artifact detector, and the long spurious spindles accompanying high voltage waveforms were ignored.

The principle of the response duration analysis is similar to that of the digital filters. A logic pulse representing the duration of the 12-16 Hz spindle is compared with another pulse equal to the criterion duration. If the spindle exceeds the criterion duration then an output pulse is produced. The input logic pulse is reset by inhibit pulses from the amplitude discriminator so that the duration analysis is re-initiated. In addition, the duration logic automatically resets so that a series of output pulses are produced during a continuous input. A fixed duration of 100 msec was chosen for the feedback unit as a compromise between limiting the response to small transients; and being able to quantify short bursts of genuine 12-16 Hz in the EEG of the epileptic. A 10 Hz pulse train was thus constantly output during the occurrence of criterion 12-16 Hz activity. This was amplified and registered by an electromechanical counter. A similar system was provided for the quantification of 8-10 Hz EEG.

Feedback of criterion responses was controlled by a separate duration

analysis circuit which could operate from either frequency band. The criterion duration for this circuit was continuously variable to allow shaping of the response required to obtain feedback.

When the equipment was first used with epileptic patients, it was soon found that the retriggering of the duration logic was ineffectual during very high voltage slow wave complexes. This problem was solved by feeding the output of the amplitude discriminator to a retriggerable monostable multivibrator, which held the inhibit circuitry on for 1 sec after the occurrence of any high voltage activity. This satisfactorily completed the armour of the feedback unit against the various spikes, peaks, humps, and crags of the epileptic EEG.

5:1:6 Feedback Devices

Auditory feedback was reported to be very distracting by many subjects in the previous laboratory studies of both alpha and SMR training. Visual feedback, on the other hand, is inappropriate for alpha training, as the alpha rhythm characteristically blocks to visual stimulation. However, this is not the case in the training of central 12-16 Hz EEG, and so a number of visual feedback displays were used in the unit for the clinical study rather than tone signals:

(1) A digital display was provided which exhibited the cumulative number of criterion responses, since subjects in the laboratory study of SMR training generally reported that the digital timer was quite helpful in indicating performance.

(2) The study of analogue modes of feedback demonstrated the importance of moment-by-moment information about performance (*see* 3:4:3), hence a red lamp was included which glowed in proportion to the abundance of 12-16 Hz (or 8-10 Hz) in the EEG. This gave the subjects information

about their performance between the occurrence of criterion responses, and was found, in practice, to be quite effective in maintaining their efforts. The lamp was not driven directly from the output of the analogue filter - since the subjects would then also receive feedback during epileptiform and artifactual activity; but was operated instead by rectified and smoothed pulses from the duration logic, so that only feedback for genuine criterion activity was obtained.

(3) A green lamp was connected to the output of the amplitude discriminator to indicate the presence of high voltage artifactual and paroxysmal activity. This functioned as a 'time-out' signal in that the red lamp and digital display were inhibited whenever the green lamp was switched on. Hence it proved to be very useful in signalling to the subjects the presence of muscle artifact, such as that produced by jaw clenching. It also afforded the epileptic patients with the opportunity to learn to suppress pathological discharges in the EEG.

As the clinical study was planned to continue for over a year, it was decided to add some form of reinforcement in order to maintain the interest and motivation of the subjects in the training procedure. It was originally supposed that the study might involve child patients, and so an automatic *Smarty* dispenser was built, since other workers have shown *Smarties* to be a simple and effective form of positive reinforcement for children (Bijou and Sturges, 1959). It was found during some informal preliminary trials with undergraduate students, however, that the *Smarty* dispenser could produce a surprising amount of subject zeal even in adults! Hence it was included in the apparatus for the clinical study, and the subjects were given the choice of either *Smarties* or half-pennies as the reward. It is interesting that the two eldest patients in the clinical study chose the *Smarties* (see 6:2:3), even though they could have earned a quite significant amount with the half-pennies owing to their low income. The *Smarties* were automatically dispensed whenever a criterion

response occurred and stored in a small tray. The half-pennies (which were kept to within economic proportions by expedient use of the reward duration logic!) were counted out at the close of each training session according to the score registered by the digital display.

5:2:1 Introduction

An alternative to the analysis of time-amplitude relations in the EEG is analysis in the frequency domain. This provides an estimate of the average intensity as a function of the frequency spectrum of the signal. Since the early attempts by Grass and Gibbs (1938) - who developed an automatic photo-electric technique for processing small samples of the EEG - spectral analysis has proved to be a powerful tool for the quantitative treatment of the EEG waveform (Burch, 1959; Dumermuth, 1971).

Facilities for carrying out spectral analysis of the EEG were developed to supplement the data obtained from the EEG feedback unit during the clinical investigation. There were several reasons for this:

(1) A disadvantage of criterion duration measures of EEG frequency bands is that information about amplitude changes above the threshold level is lost. Conversely, the integration techniques, which are sometimes used as an alternative measure, are influenced by broad band changes (*see* 8:1). With the use of spectral analysis, a quantification of both absolute and relative power of the frequency bands can readily be obtained.

(2) Spectral analysis enables an evaluation of overall changes in the EEG that might accompany feedback training and underlie clinical effects. For example, it has been reported by Sterman and co-workers that sequential power spectra over 6-18 months of SMR feedback training showed a reduction of abnormal EEG activity (Sterman, 1973a; Sterman, 1973b; Sterman et al., 1974). Similarly, Kuhlman (1976) reports that extensive power spectral analysis showed reliable increases in EEG frequency toward normal limits as a function of feedback training, together with decreases in abnormal slow activity. Thus spectral analysis might provide one objective measure by which the degree of improvement of epileptic patients

could be assessed.

(3) Sterman and associates report that a degree of EEG asymmetry was often demonstrated by power spectral analysis following feedback training with "the trained hemisphere more desynchronized and showing greatly reduced abnormal frequencies" (Sterman, 1973b). This claim for a unilateral normalization of the EEG is of course a powerful argument against placebo, or other situational effects being the *causa causans* of the therapeutic benefits of feedback training for epilepsy. Hence I felt that it would be important to obtain a similar analysis of any such changes in the EEG during the clinical study.

A PDP/11 digital computer, complete with an analogue to digital converter and a spectral analysis software package, was planned for the department. Whilst waiting for this to become available, however, the opportunity arose to use a low-frequency wave analyser, situated at the University of Aston in Birmingham. This analyser is a modern development of the Burden Neurological Institute EEG analyser designed by Walter and colleagues (Walter, 1943). A bank of active resonant filters are tuned at 1 Hz intervals over a frequency band of 2-21 Hz. The output of these filters is integrated over a ten-second epoch, and the resultant voltages measured sequentially by a digital voltmeter. The information is punched on to paper tape for subsequent off-line computer processing (Bailey and Harding, 1966; Harding, 1971). The computer output lists the absolute abundance of the individual frequency bands and also quotient scores, which express the percentage contribution of each frequency to the total abundance. Further statistical processing of the data is also presented.

EEG data for analysis were stored on a four-channel FM tape recorder¹

¹Coding of the EEG by frequency modulation of a carrier signal is required since the voltage response induced across the magnetic reproduce head at low frequencies falls below the inherent noise level of normal tape systems.

(Thermionic, Model T3000), which was then transported to Aston. The replay of the data was made via a penrecorder so that the signal could be monitored during analysis. A series of calibration signals was recorded on the tape to allow the gains between channels, and between analyses, to be equalized. A number of preliminary EEG spectral analyses were made using the Aston Analyser.

5:2:2 Fourier Analysis

Almost any complex waveform can be represented by an appropriately weighted combination of sinusoidal and cosinusoidal elements. Fourier analysis is a mathematical tool by which these components can be obtained. A plot of the square of the Fourier coefficients yields a power spectrum of the waveform (Jenkin and Watts, 1968).

Dietsh (1932) attempted to apply Fourier analysis to the EEG, but the calculation involved was so extremely laborious that the technique had little practical value. A breakthrough in the analysis of complex signals was made when Cooley and Tukey (1965) introduced an iterative algorithm for the calculation of Fourier coefficients, which became known as the Fast Fourier Transform as it greatly reduced the amount of computation involved. With the advent of modern digital computers it thus became possible to perform power density spectral analyses in seconds rather than days.

An inherent assumption in the concept of spectral analysis is that the signal is stationary, *viz.* the time-varying waveform periodically and indefinitely repeats itself. This assumption is clearly violated in the case of the EEG. In addition, the waveform is characterized as the linear sum of sinusoidal elements, which is not necessarily a true description of the EEG (*see* 2:1:1). It further follows from these assumptions that spectral analysis is insensitive to transient phenomena in the EEG. Nevertheless, spectral estimates using Fourier analysis have over the last

decade substantially assisted the processing and interpretation of EEG data (Bickford et al., 1971; Joy et al., 1971).

When the PDP/11 computer became operational, a programme was written¹ so that power spectral analysis of EEG data, either on-line or off-line, could be carried out in the laboratory. The library software of the PDP/11 (*Lab Applications II*) included a Fast Fourier Transform module. This converts each point in the data time series by the operation

$$A_r = \sum_{k=0}^{n-1} X_k \exp(-2\pi jrk/n), r = 0 \dots n-1.$$

Where A_r is the r^{th} coefficient of the Fourier transform, X_k is the k^{th} complex data point from the sample consisting of n points, and j is the complex operator $\sqrt{-1}$.

An additional module performs a sum of squares of the complex Fourier coefficients to produce the power spectral values.

The parameters for the Fourier analysis were decided as follows:

(1) The resolution of the power spectrum is determined by the reciprocal of the epoch sampled. A resolution of 0.5 Hz was thought to be quite adequate.

(2) The frequency range of the spectrum is limited to half the sampling rate. A frequency limit for the initial analysis of 32 Hz was selected.

(3) The Fourier transform module operated on a maximum of 1024 complex points in steps equal to the powers of 2. For a 32 Hz cutoff the sampling rate was 64 per second. Over an epoch of 2 sec this gave 256 data points.

(4) The processing time and core store needed for the analysis is a function of the number of points transformed. 256 points were the minimum

¹The programme was developed in conjunction with Mr. S. Forrest. He was responsible for assembling the data sampling and analysis software on the PDP/11; and I for the final data processing and plotting programmes run on the University 4130 computer.

number for an analysis covering the EEG frequency range. A higher frequency range or resolution was not necessary, and would reduce the total period of the sample that could be analysed.

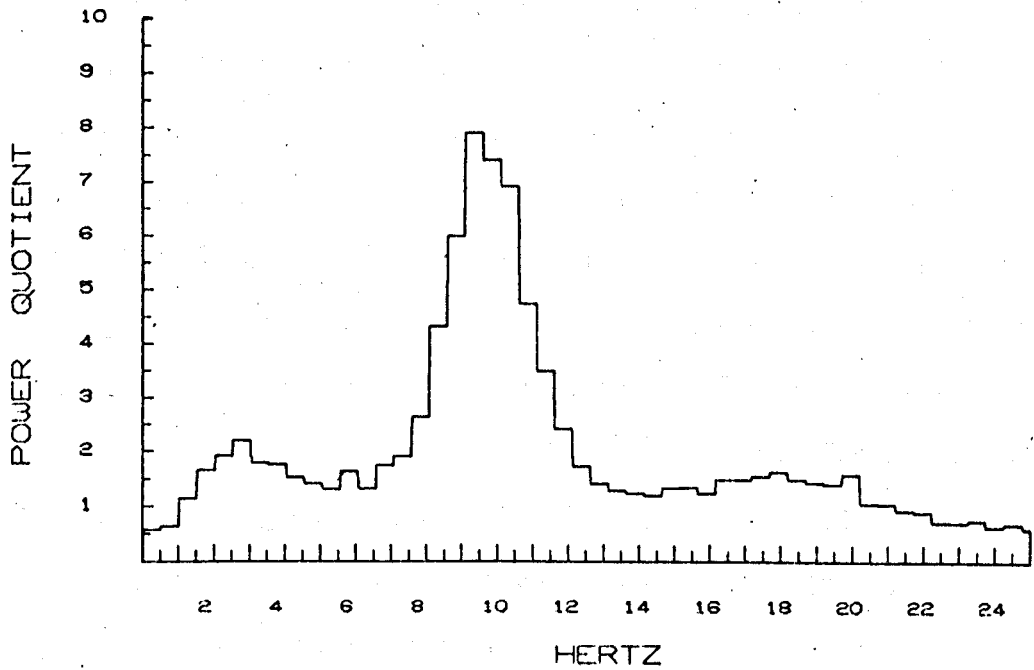
Since the EEG is a non-stationary, aperiodic signal the analysis of just one two-second epoch is, of course, meaningless. Hence a loop was included in the programme so that a running sum of spectra was obtained. The use of such summed or average spectra 'smooths' some of the error arising from violation of the assumption of stationarity¹, and allows a much larger sample to be analysed before the computer store is exceeded than if the analysis epoch is simply extended. Similar techniques have been used by many other workers (Mason, 1970). However, it should be noted that the final spectrum still only describes the total EEG epoch sampled, and it is a matter of faith that this sample can be regarded as representative of the EEG as a whole.

A maximum of 75 two-second epochs can be analysed at one time. The summed power spectral values are printed out on a teletype and also on paper tape for further processing on the University 4130 computer, which is equipped with data plotting facilities. The final output lists:

- (1) The absolute power of frequencies 0.5-25.0 Hz in steps of 0.5 Hz.
- (2) The power quotient of each frequency. This indicates the relative contribution of the frequency bands when the total power is normalized to 100%. The quotient score was adopted since it is independent of changes in the average amplitude of the signal and of the number of epochs sampled.
- (3) The mean power, mean power quotient, and mean frequency of the classic EEG bands: delta (1.0-3.5 Hz), theta (4.0-7.5 Hz), alpha (8.0-12.5 Hz), beta I (13.0-17.5 Hz), beta II (18.0-25.0 Hz); and also of the total 0.5-25.0 Hz bands, and the 12.0-16.0 Hz, 8.0-10.0 Hz, and 2.0-7.0 Hz

¹Regan, D., *personal communication* (1975).

RQ 02-A2 EC



RQ 02-A2 EO

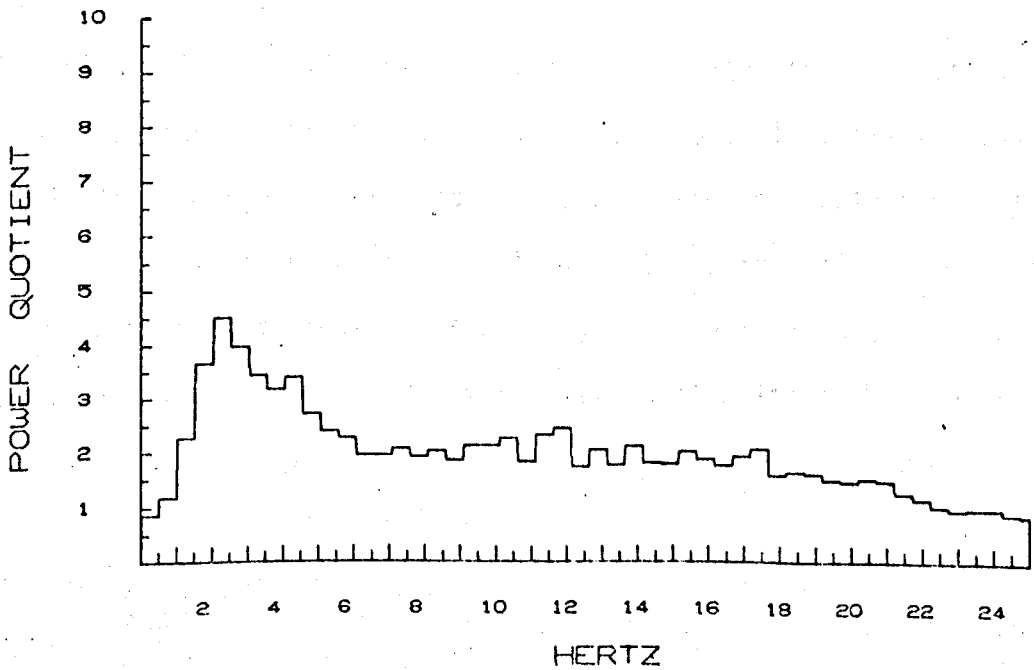


Figure 5.5
Normalised power spectral histograms of the author's occipital EEG. Note the strong peak at 9-11 Hz of the alpha rhythm in the eyes-closed (EC) condition; which is absent in the eyes-open (EO) condition.

frequency bands that were pertinent to the clinical study.

In addition, the normalized (power quotient) spectrum was automatically plotted by the computer for each analysis. In Figure 5.5 some examples showing the change in the spectrum of the occipital EEG between eyes-open and eyes-closed condition are reproduced.

Data stored on magnetic tape for analysis were played into the analogue to digital converter of the PDP/11 via a small amplifier that was adjusted to give a maximum voltage resolution. The amplifier was also used to equalize the amplitude of calibration signals between samples by monitoring the output on a penrecorder or oscilloscope. The EEG signal was passed through a band-pass filter with cutoff points at 1.0 Hz and 30.0 Hz (Krohn-Hite, Model 8700; 24 dB/octave), in order to attenuate DC switching transients, and to avoid aliasing errors¹.

The system was checked using calibration signals from a high quality oscillator with the output frequency monitored on a digital timer. Reliability coefficients were also computed by correlating the spectra obtained from several separate analyses of the same EEG signal. These are shown in Table 5.1.

The reliability coefficients are seen to be high; those obtained using power quotient scores are even greater. The small variation that there is in the spectra from run to run results from noise in the system. One form of noise is introduced by small variations in the speed of the tape recorder transport ('wow and flutter'). Noise is also generated by the tape itself, and by the frequency modulation circuitry. Within the computer there is the possibility of some inaccuracy arising from

¹Aliasing is an inherent feature of data sampling. Frequencies which are greater than half of the sampling rate (Nyquist frequency) are transposed to signals which are indistinguishable from the real lower frequency components (Magrab and Blomquist, 1971). In this case, high voltage noise spikes generated by the magnetic tape, and transient signals in the EEG itself, are a potent source of error without high frequency filtering.

Table 5.1. Reliability coefficients derived from product-moment correlation of absolute power spectra obtained from four separate off-line analysis of the same EEG signal.

<u>Run Number</u>	<u>2</u>	<u>3</u>	<u>4</u>
1	0.950	0.975	0.974
2		0.975	0.966
3			0.984

quantization errors in the analogue to digital conversion, and from other sources such as round-off errors involved in the mathematical computations.

The development of the apparatus used for a study of the effects of SMR feedback training on patients with epilepsy is described. One of the greatest difficulties was to obtain a SMR filter that gave maximum attenuation of out-band activity, on the one hand; and minimum response to transient waveforms, on the other. This was achieved by a hybrid 12-16 Hz analogue and digital filter network. A similar filter system was included for the detection of 8-10 Hz activity. The filters were followed by circuitry for amplitude discrimination of the signal and response duration analysis to limit the response of the system to epileptiform and artifactual signals. Lamp signals were arranged to provide continuous feedback of the occurrence of activity in either the 12-16 Hz or 8-10 Hz frequency bands, and of high voltage activity. These were supplemented by a digital display that was used in conjunction with monetary or confectionary reward to reinforce the appearance of an EEG response of selected frequency, amplitude, and duration criteria.

A programme for computerized power spectral analysis of the EEG was developed to provide additional data on the absolute and relative abundances of various frequency bands; and to allow claims for overall changes in the EEG during feedback training to be investigated. The programme parameters were selected to output spectra from 0.5 - 25 Hz, at a resolution of 0.5 Hz, from EEG samples of up to 150 sec duration. The reliability of the computer analysis was found to be very high.

6:1

INTRODUCTION

A clinical study was set up in an attempt to replicate and further investigate the reported therapeutic benefit of SMR training in epilepsy (*see* 1:1:2). Since the evidence for an SMR in man is weak (*see* 4:2:4), I sought in this study to examine the proposal that the observed effects on epilepsy are specifically related to the selective enhancement of EEG of SMR frequencies, and to determine whether there is evidence of other factors that might be involved.

In the first report of this work, Sterman and Friar (1972) state that, "Biofeedback training of the sensorimotor rhythm resulted in a striking enhancement of this rhythm's occurrence, differentiation from simultaneously recorded alpha activity, and a marked suppression of seizures". Similarly, Sterman et al. (1974), in a subsequent presentation of the results from four epileptic patients report that, "These patients seldom showed the clear 12-14 Hz or 14-16 Hz patterns noted in control subjects... Nevertheless, they did demonstrate a marked and localized increase in the filter-selected occurrence of the SMR pattern, and occasionally showed clear trains of low-voltage SMR activity during performance". However, as with their report that non-epileptic subjects showed a progressive increase in SMR with feedback training (*see* 4:2:1), no actual data is provided by Sterman and his colleagues to support their claims, other than a few sample EEG recordings and power spectra.

Lubar and Bahler (1976) also recently report that seven of their eight patients showed a reduction in seizures during SMR training; "furthermore, the successful patients demonstrated an increase in the amount of SMR during the course of the training period". To this Lubar (1976) adds:

"Those that showed the greatest seizure reduction also had the greatest degree of acquisition of activity in the 12-15 Hz bandpass". Again, however, no quantitative analysis was carried out to demonstrate that there was any significant relationship between the abundance of 12-15 Hz activity in the EEG and the clinical changes. Moreover, in an earlier paper, Seifert and Lubar (1975) provide plots of the percentage 12-14 Hz activity in the EEG, which they propose "represents the SMR". Despite their statement in the abstract to this report that, "Several patients showed increased percentage of SMR when feedback was provided", the data plots indicate that the only subjects to demonstrate a consistent increase in this activity were two non-epileptic controls.

Finley (1974; 1976) and co-workers (Finley et al., 1975) are the only group to offer any statistical evidence for a relationship between the acquisition of the SMR response and the effects on epilepsy. They report a strong positive correlation ($r = 0.64$, $p < 0.0005$) between the percentage of SMR and the number of training trials. Conversely, the acquisition curve for atonic seizure rate per hour (as determined by the patient's parents) was found to be almost a mirror image of the SMR curve ($r = -0.65$, $p < 0.0005$). The percentage of SMR and seizure rate were, correspondingly, negatively correlated ($r = -0.54$, $p < 0.0005$). Similarly, there was a strong negative correlation ($r = -0.54$, $p < 0.0005$) between the percentage of SMR and the percentage of epileptiform activity in the EEG, as indicated by the slow wave inhibit circuit. The positive relationship between percentage of SMR and training trials was maintained when the effects of the inhibit circuit were statistically partialled out ($r = 0.56$, $p < 0.0005$), which indicates that the percentage of SMR did not increase over trials merely because epileptiform discharges decreased (Finley et al., 1975). However, as discussed previously, Finley and colleagues relied on an extremely narrow band filter for their data, and, although they might have

obtained an increase in the 12 Hz output of this filter with feedback training, it is very difficult to accept that this signal could represent a homologue of the SMR of the cat (*see* 5:1:2).

A study to evaluate the relationship between feedback training of 12-14 Hz rolandic EEG and the effects on epilepsy was carried out with two patients by Kaplan (1974; 1975). She was unable to obtain any changes in seizure incidence, or the proportion of 12-14 Hz in the EEG, as indicated by spectral analysis, for either subject. However, training was maintained for only four months and her filter system, with no protection against spurious feedback generated by epileptiform activity or artifact, is also open to criticism (*see* Appendix). Thus it can be argued that her failure to replicate the findings of the other workers was due to methodological shortcomings.

The purposes of this study therefore were: firstly, to determine whether a statistically significant reduction in the manifestation of epileptiform activity, as indicated by seizure incidence or EEG abnormality, could be induced by feedback training of 12-16 Hz rolandic EEG; secondly, to determine whether there is any evidence of a relationship between any such therapeutic effects and the selective enhancement of this frequency band; and thirdly, to employ a number of control procedures to determine whether the therapeutic effects are specifically dependent on the feedback training paradigm.

Facilities for this study were arranged at the David Lewis Centre for Epilepsy, situated some thirty miles north of Keele. This Centre is an independent organization for the residential treatment of chronic epileptics who, because of severe and frequent seizures or emotional and behavioural disturbances, are unable to function adequately in the community. At the present time, there are about two-hundred and fifty adult and eighty-five child patients at the Centre. Thus it provided a very suitable and

convenient pool of subjects for this clinical study.

The design of the study was to use a small group of subjects as their own controls in a longitudinal analysis, since the inclusion of more than three patients, or a separate control, group was ruled out by several practical considerations¹. Although this form of approach might not be as elegant as matched groups in the Fisherian tradition, it is nevertheless one which has enjoyed a long history in clinical research (Gottman, 1973). The sample size is also comparable to that used by other researchers for this work, which, of course, requires a considerable commitment of time by both the patients and the experimenter.

The study was organized into three stages. In the first the effects of feedback training of rolandic 12-16 Hz EEG were investigated; in the second and third a number of control procedures were carried out. These control procedures consisted of feedback training of 8-10 Hz rolandic EEG in the second stage; and of random feedback for one subject, and suppression training of high voltage activity for the other two subjects in the third stage. The 12-16 Hz feedback training was maintained for six months, so as to provide time for any clinical effects to become apparent equivalent to that in which they were observed in other studies. The control procedures were then restricted to the following six months, owing to the time constraints of the project.

Each of those training conditions will be reported below. The overall results and implications of the clinical study, together with a model of possible underlying neurophysiological mechanisms will be discussed in the next chapter.

¹ (i) More than one training session was required per week for the patients to receive comparable SMR training to the subjects in other studies.
(ii) The general regimen of the Centre permitted a maximum of three patients to be trained per day.
(iii) Transport to the Centre was initially available only on two days per week.

6:2:1 Subjects

The selection of subjects for this study was carried out in conjunction with the Director (Dr. Grant) and the Physician (Dr. Barot) of the David Lewis Centre. It was based on several requirements:

- (1) It was necessary that the patients should be domiciled at the Centre in order that a longitudinal study could be carried out. This eliminated all the child patients as they were resident only on a termly basis.
- (2) The patients should have had seizures which had proved to be intractable to conventional drug treatment.
- (3) There were no clinical grounds which would indicate against their staying on a constant drug regime throughout the course of this study.
- (4) The patients should manifest more than one seizure per month so that there would be sufficient data for clinical changes to become apparent.
- (5) The seizures should be clearly distinct events and not compounded by hysterical attacks.
- (6) There should be no history of progressive brain disease or severe mental handicap.
- (7) The patients should be co-operative and likely to persevere with the training programme.

Three patients who fulfilled these requirements were chosen. A synopsis of their case histories follows¹.

¹These case histories are based on information supplied by the director of the David Lewis Centre, and the patients' medical files.

P.C.

This patient was a twenty-three year old male (born 15.11.53). The onset of epileptic seizures occurred at the age of 2 years. The only indication of a familial history of epilepsy was with a cousin of the patient's mother. At three years he was admitted to the Maudsley Hospital presenting both generalised tonic-clonic and psychomotor seizures. At fifteen years he was investigated at the Royal Bethyl Hospital, where widespread EEG abnormalities, including a focal abnormality in the right temporal region, were described.

The patient developed increasing behavioural difficulties and was excluded from a number of residential schools because of violent outbursts. He was admitted to the David Lewis Centre in 1971, where he gradually became less aggressive and more mature in behaviour. At the time of study he was satisfactorily settled at the Centre, although he was described as being "attention-seeking".

The patient has been treated with various regimes of anticonvulsants, including phenytoin, primidone, phenobarbitone, pheneturide, diazepam, carbamazepine, modazepam, clonazepam, and sodium valproate. At the commencement of this study his daily medication consisted of 3,000 mg of sodium valproate (Epilim) and 800 mg of carbamazepine (Tegretol). An intravenous injection of 10 mg of diazepam (valium) was also sometimes given to ameliorate the severity of his grand mal seizures.

Since 1974, his seizure rate averaged about three to four tonic-clonic seizures per month. The patient also often experienced myoclonic jerks, sometimes throwing him to the ground, but not always culminating in a seizure. Other than these jerks, the patient experienced no warning of the onset of a major attack. He usually fell backwards without injury, and exhibited severe cyanosis. The ictus lasted 2-3 minutes, and he usually slept afterwards for several hours. He sometimes experienced two

attacks on the same day with continuous myoclonic jerking between.

The EEG of this patient typically exhibited generalised large slow waves of 3-4 Hz and bursts of theta activity of 4-7 Hz, often occurring in paroxysms. Random spikes and sharp waves were seen in all areas, with focal spiking over the right posterior temporal and parietal regions. The alpha rhythm was of 8-9 Hz and irregular, but responded normally to eye-opening. The epileptic condition of this patient has been diagnosed according to the *International Classification of the Epilepsies* (Gastaut, 1970) as generalised partial seizures with multiple foci.

The intelligence quotient of the patient was assessed in 1970 as 78 (W.A.I.S. score). At the time of this study he was employed within the Centre as a postman.

B.H.

This patient was a forty-nine year old female (born 22.2.27). Her epilepsy first appeared at the age of sixteen; and she was admitted to the David Lewis Centre at the age of twenty-seven years suffering from frequent tonic-clonic and psychomotor seizures.

Despite the administration of various drug regimes over the twenty-year period of her treatment at the Centre, frequent seizures continued to occur, and in 1965 the patient experienced major status epilepticus. At the time of this study, her daily medication was 250 mg of phenytoin (Epanutin); her major seizures were stabilised at a rate of about two per month, tending to occur in pairs, and psychomotor seizures had ceased.

The patient usually experienced no warning of the onset of attacks, although they were occasionally preceded by episodes of myoclonic jerks. Generally she fell backwards without injury. The ictus lasted from 5-10 minutes, and she slept afterwards for some thirty minutes.

Her EEG typically consisted of low voltage fast activity, with beta and barbiturate rhythms in the central and frontal areas. Spikes and

sharp waves were frequently seen, mostly over the right hemisphere, but becoming dominant over the left during hyperventilation. A normal alpha rhythm of 8 Hz was present. The condition of this patient has been diagnosed according to the *International Classification of the Epilepsies* as primary generalised epilepsy.

No psychometric data was available for this patient. She gave the impression of rather low intelligence, but not sufficient to be regarded as mental subnormality. She has been happily employed in the laundry of the Centre since the time of her admission.

N.L.

This patient was a thirty-nine year old male (born 10.2.37). The onset of his epilepsy occurred at the age of eight years in association with an undiagnosed illness. He began to exhibit tonic-clonic seizures, predominantly involving the left side of the body, and also psychomotor and absence attacks.

In 1956 the patient was investigated at the London Hospital. Angiograms were normal, but pneumoencephalograms indicated a slight enlargement of both ventricles, particularly on the right. Serial EEG recordings demonstrated a predominantly right-sided temporal lobe focus. A temporal lobectomy was performed in 1957 in which a 7 cm area of the right anterior inferior-temporal cortex was incised. The surgical intervention was not successful, and frequent grand mal and psychomotor seizures continued to occur.

This patient was admitted to the David Lewis Centre in 1964. His treatment consisted of various manipulations of phenobarbitone, primidone and phenytoin. At the commencement of this study, his daily medication was 750 mg of primidone (Mysoline), 400 mg of Phenytoin (Epanutin) and 90 mg of phenobarbitone (Phenobarbital). His seizure frequency was variable between 5-10 minor (psychomotor) and 10-20 major (tonic-clonic)

attacks per month.

This patient's minor seizures were preceded by an aura consisting of a "buzzing sound" and a "muzzy feeling". They took the form of transient absences that were sometimes associated with falling and sometimes with automatisms. There was no warning of the onset of a major attack, and the patient usually fell very heavily forwards. As a result, he sustained many lacerations of his face and scalp and so wore a protective helmet. The grand mal ictus lasted about 30-40 seconds, after which the patient remained confused for up to several hours.

EEG recordings showed paroxysmal delta and theta activity of 2-7 Hz and also beta and barbiturate fast activity of 17-25 Hz in all areas, together with frequent diffuse spikes and sharp waves. A symmetrical alpha rhythm of 8 Hz, which responded to eye-opening, was present. Occasionally bilaterally synchronous spike and wave episodes of 2-3 Hz were observed, with the highest voltages over the frontal areas and predominantly on the right side. These sub-clinical episodes typically continued for 10-20 seconds, but the patient usually exhibited no behavioural signs other than slight slowness and confusion. The epileptic condition of the patient has been diagnosed according to the *International Classification of the Epilepsies* as secondary generalised partial seizures with complex symptomatology.

The intelligence quotient of this patient was stated in 1968 to be 78, according to an unspecified verbal scale. He appeared at the time of this study to be rather dull and slow-witted, although he chatted very freely. He was employed making paper hats in the Industrial Therapy Unit of the Centre.

These three patients were interviewed both by the Director of the Centre and myself. They were told that this form of treatment had worked for some patients but not others, so that there could be no guarantee that

it would help them. They were also told that, since we were going to experiment to find the most effective ways for our machines to be connected up, their condition could sometimes even become worse. All three patients firmly expressed the wish to participate in the study, however, which perhaps gives some indication of the despairing plight of the chronic epileptic.

6:2:2 Apparatus and Recording Techniques.

The EEG feedback unit that was discussed in the last chapter formed the core of the apparatus for the clinical study. The unit was interfaced with a sixteen-channel electroencephalograph (Standard Laboratory Equipment, Model 186), situated at the David Lewis Centre. An oscilloscope was provided for the purposes of calibration and signal monitoring, and a four-channel FM tape-recorder (first a Thermionic, Model T3000; later a Phillips, Datalog cassette recorder) to store the EEG for subsequent power spectral analysis. The equipment used in the clinical study is pictured in Plate II.

EEG Recording Placements

The electrode derivations employed by the different workers in the extant SMR studies have varied considerably. Thus Sterman and associates originally used a longitudinal bipolar lead with the anterior electrode intermediate between sites C_3 and P_3 (Sterman and Friar, 1972). In later studies, the electrodes placements were shifted to the medial-lateral plane, with one electrode at a point 10% of the inter-aural distance down from the vertex and the other, either at a point "just lateral to C_3 " (Sterman, 1973a), or at a point 30% down from the vertex (Sterman et al., 1974). Lubar's group used a similar lead (Seifert and Lubar, 1975), or a slightly more lateral one, with electrodes placed at sites C_4 (20% down from the vertex) and T_4 (40% down from the vertex) (Lubar and Bahler, 1976).



Plate II.

A feedback training session in the clinical study. The EEG is recorded by the electroencephalogram in the foreground of the picture. In the centre is the EEG feedback training unit (large box) that was designed specifically for the clinical study, together with ancillary equipment for quantification of the data. To the left is the head of the subject and, beyond it, the Smarty dispenser and feedback display.

Referential recording was adopted by Finley et al. (1975), with the active electrode placed intermediate between sites C_3 and F_3 and the reference electrode on the right mastoid. Kaplan (1975) also chose referential recording, but placed the active electrode "1 cm posterior to C_4 " and the reference electrode on the left mastoid.

There appears to be no obvious theoretical reason for preferring any one of these EEG derivations to another, and no explanation has been offered by any of the workers why their particular electrode positions were chosen. Therefore, since it was found previously that individuals could vary considerably in the topographic distribution of 12-16 Hz (see 4:1:3), I decided to determine the electrode placements for the subjects in this study empirically.

Electrodes were placed over the frontal, central, parietal, and temporal regions of the right hemisphere at 10% of the inter-aural distance in the transverse plane, and 10% of theinion to nasion distance in the anterior-posterior plane. Because of limited facilities at the David Lewis Centre, standard saline pad electrodes were used for these, and for all other, recordings made in the clinical study. Electrolyte was applied to the scalp until contact impedances were reduced to the order of 5 k Ω .

Various montages were recorded onto the four-channel FM tape-recorder and replayed into the feedback unit. One lead was kept constant across all montages so as to enable some comparison of more than four channels.

No evidence of a consistent optimum recording position for 12-16 Hz activity could be found in any of these subjects, within the limitations of the techniques used. However, there was some evidence that for subjects P.C. and N.L. most 12-16 Hz activity was recorded from electrodes in the transverse plane over the rolandic and frontal areas, whereas for B.H. the maximum scores were seen in longitudinal leads across the parietal and rolandic regions. Therefore it was decided to adopt the bipolar

electrode pairs which corresponded closest to the EEG derivations employed in previous studies. Thus, the electrodes were placed at 10% above and 10% below C_4 for subjects P.C. and N.L., as in the investigation of Sterman et al. (1974); and at 10% anterior and 10% posterior to C_4 for subject B.H., as in the investigation of Sterman and Friar (1972).

The threshold parameters of the various detection systems of the feedback unit were then adjusted for each subject, so as to provide a suitable level of feedback in accordance with the amplitude of their EEG. These parameters are listed in Table 6.1.

Table 6.1. Threshold amplitudes for each subject (μV)

<u>PARAMETER</u>	<u>SUBJECT</u>		
	<u>P.C.</u>	<u>N.L.</u>	<u>B.H.</u>
12-16 Hz	10	5	5
8-10 Hz	10	10	5
Inhibit	50	50	40

Occipital EEG and eye movement channels were recorded in addition to the central EEG. The electrode placements are summarised in Figure 6.1.

Other Physiological Measures

Because of the reports that somatic, cardiac and respiratory activity exhibit characteristic changes during SMR in the cat (Chase and Harper, 1971), and also, to a lesser extent, in human subjects (Sterman, 1974), several other physiological measures were recorded.

(1) Frontalis EMG was recorded and integrated as described previously (see 4:2:2). The integrator was gated by the master timer of the feedback unit so that the count was automatically halted along with the other registers at the end of the training intervals.

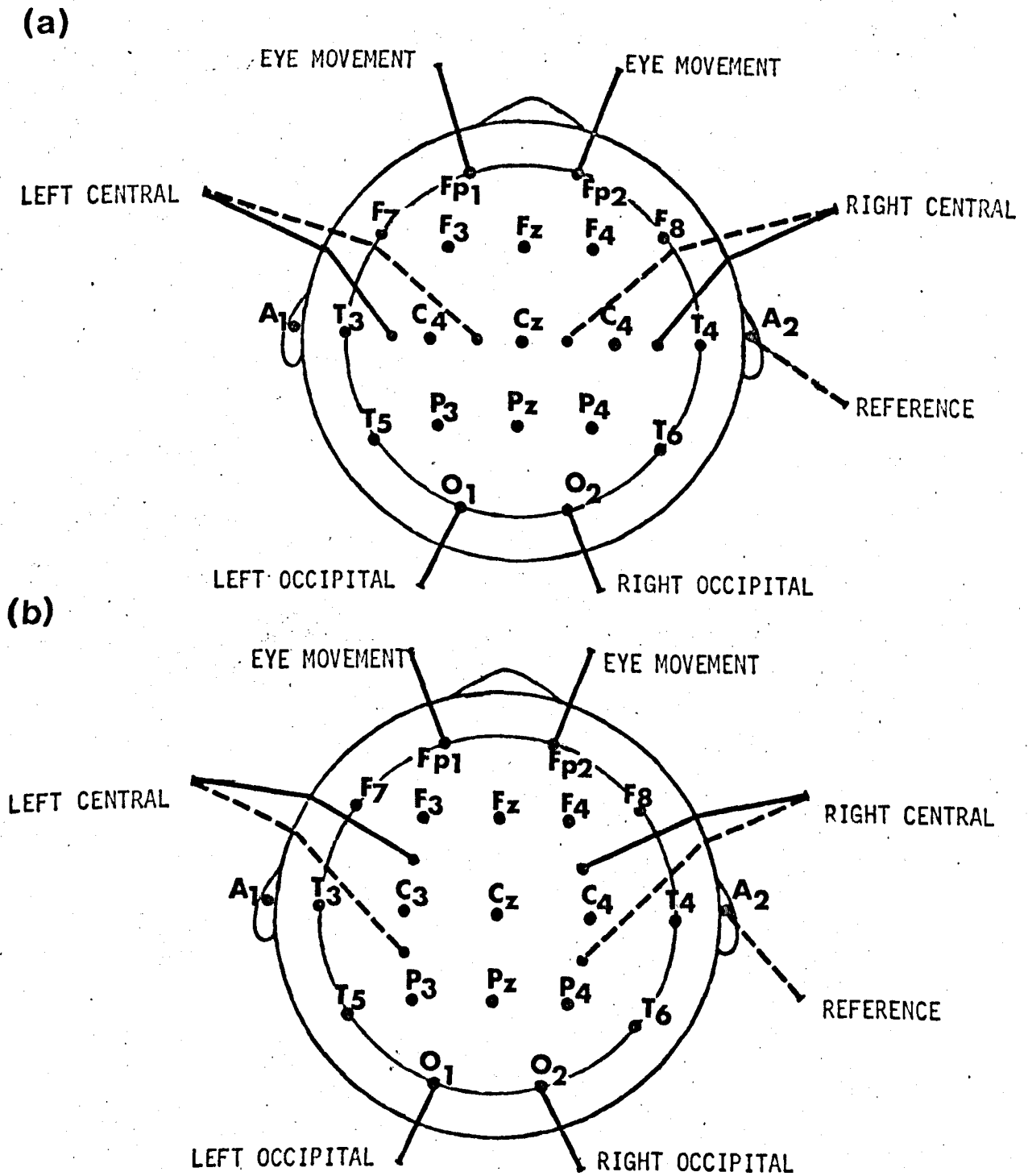


Figure 6.1

(a) Electrode placements for subjects P.C. and N.L.
 (b) Corresponding electrode placements for subject B.H.
 The right central EEG was used for feedback training in all cases.

(2) ECG was recorded from stainless steel plates attached by rubber straps to the patient's wrists. A time constant of 1.0 sec, high-cut filtering above 50 Hz, and minimum gain were selected.

(3) A respirogram was recorded using a thermistor taped beneath the patient's nostril. The maximum time constant (1.0 sec.) and high-cut filtering (25 Hz) available on the pre-amplifier were used.

Both heart rate and respiration rate were quantified manually from the paper chart (taking the average of several minutes sampled across the record) as no tachometer was available.

(4) An attempt was also made to record and quantify galvanic skin response (GSR), using an instrument made for this purpose (Electronic Developments, Self-Balancing Psychogalvanometer). However, this had to be abandoned since, even after several modifications were made, the instrument could not be connected to the patient at the same time as the electroencephalogram without producing excessive artifact.

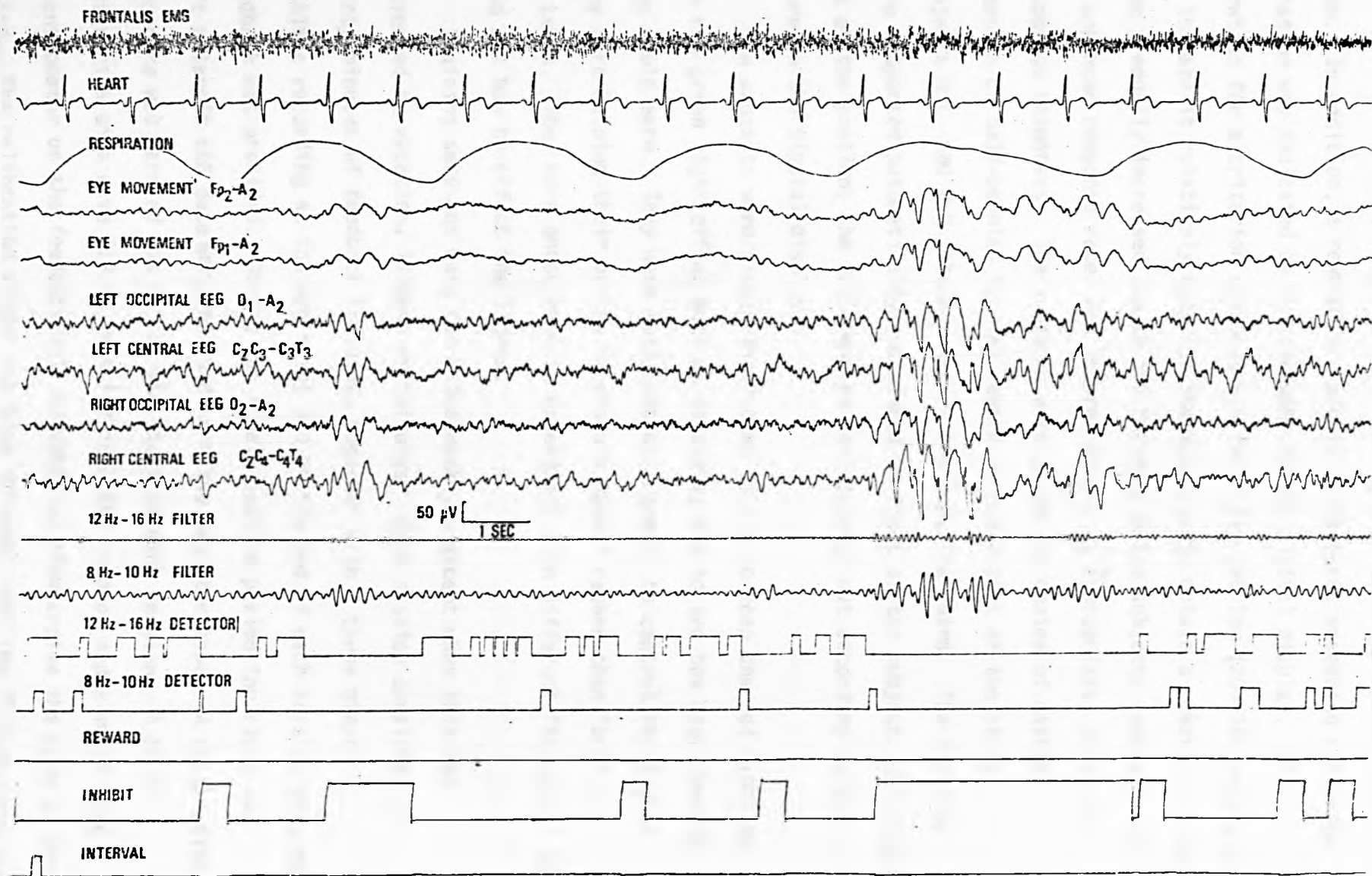
In addition to the various physiological parameters that were recorded, the outputs of the 12-16 Hz and 8-10 Hz analogue filters of the EEG feedback unit were displayed on the paper record, together with a series of marker pulses. These marker pulses indicated the following events: the detection of 12-16 Hz and 8-10 Hz activity above threshold level, the presentation of a reward for a criterion response, the activation of the inhibit circuit by waveforms exceeding the prescribed amplitude window, and the operation of the master interval timer. An example of the complete chart recording is reproduced in Figure 6.2.

6:2:3 Procedure

Feedback Training

Feedback of the moment-by-moment occurrence of 12-16 Hz activity was provided by the intensity of the red lamp on the feedback unit, and of the

Figure 6.2. Example of chart record showing the various physiological parameters recorded and the output signals from the feedback training unit. Note the high voltage paroxysmal discharges in the EEG and the response of the inhibit circuitry.



operation of the inhibit circuit to high voltage activity by the green lamp. In addition, a reward for each 12-16 Hz burst exceeding criterion duration was indicated by an increment of the digital display. The duration for a criterion response was initially set low (200-300 msec), so as to make it relatively easy for the subjects to obtain a reward. It was then gradually increased (up to 500-800 msec) as the subjects demonstrated an enhanced response rate, and lowered again, as appropriate, so as to encourage responses. The subjects were given the choice of either *Smarties* or half-pennies for the reward at the outset of the study. Subjects N.L. and B.H. chose *Smarties* and P.C. the coins. The *Smarties* were dispensed automatically and stored in front of the subject until the end of the session; the half-pennies were counted out according to the score on the digital display.

The subjects were simply instructed to try to keep the red light on and the green light off as much as possible, and to see how many rewards they could earn. They were told that by learning to control the lights they were helping their brains to produce "good" rather than "bad" patterns. They were encouraged to experiment with different "thoughts" to find out how to affect the lights.

Training sessions were run twice weekly - except when this was prevented by vacation, illness or seizures. Each session consisted of thirty minutes of feedback training, together with a three minute baseline recording at the beginning and at the end of each session when no feedback was provided. During the final baseline period the right and left rolandic EEG data were recorded onto the magnetic tape. A calibration procedure was carried out before the start of each session. A 50 μ V calibration sine wave (13 Hz) was fed into the headbox and a multi-turn potentiometer on the feedback unit adjusted to standardise the gain of the system. The calibration signal was also recorded onto the FM tape-recorder

after similar gain adjustments. With initial preparation and calibration, each training session lasted some ninety minutes. The 12-16 Hz training was maintained for twenty-four weeks from July, 1975.

Seizure Logging

Charts of seizure manifestation for the patients at the Centre were maintained by the nursing sister in charge of each residence. Since the patients worked within the Centre and slept in open-plan dormitories, they were under almost constant surveillance. Thus, these charts can be considered to be a reliable data source. However, the problem with this method of seizure logging is that there can be considerable discrepancy in the description and classification of seizures between different observers. This confusion never occurred for subjects P.C. and B.H. since their seizure type was confined to very distinct tonic-clonic attacks. However, the psychomotor seizures (often accompanied by falling) and short grand mal ictus of subject N.L. were not always easily distinguished by the non-medical staff, and were variously classified as major, akinetic, psychomotor, or focal attacks. Hence a score of the total incidence of seizures for this subject was kept for the purpose of this study.

The average seizure rate over the six month period preceding this study was used to provide a baseline measurement for the evaluation of the clinical effects of the feedback training procedures.

Anticonvulsant Medication

It is clearly desirable that drug therapy should remain constant during a study of this nature. Hence the resident physician at the Centre agreed to maintain the patients on the same drug dosage unless their condition indicated to the contrary. In practice, the only changes that were prescribed over the twelve month period of the study were a reduction of phenytoin from 400 mg to 375 mg and of phenobarbitone from 90 mg to 60 mg for subject N.L., owing to signs of toxicity; and, similarly, a reduction

of sodium valproate from 3000 mg to 2400 mg for subject P.C. These alterations were made for both subjects during the second (8-10 Hz) feedback training condition.

The medication was administered by the nursing staff of the Centre, and all three patients had a record of good drug compliance. Nevertheless, in order that any unusual changes of anticonvulsant levels present in the blood plasma of the patients might be apparent, advantage was taken of the serum level assays (by gas-liquid chromatography) that were a routine procedure at the Centre.

Psychometric Assessment

Sterman and Friar (1972) report that their subject demonstrated a number of personality changes as a corollary of the SMR training therapy. They observe that "Having previously been a quiet and unobtrusive individual, she progressively became more outgoing, showed increased personal confidence, and enhanced interest in her appearance". Since it is possible that such changes might reflect placebo effects that are responsible for the clinical improvements, it was decided in this study to attempt to obtain some objective measure of any changes in the psychological state of the patient over the period of training. Thus it was arranged for the *Wittenborn Psychiatric Rating Scale* (Wittenborn, 1955) to be completed at monthly intervals for each patient by the Director of the Centre. For this purpose, preliminary ratings were provided by the nursing sister in charge of the patients' residence and amended, as necessary, by the Director after the patients were interviewed.

By the end of the twenty-four weeks of 12-6 Hz feedback training, subject P.C. showed the most encouraging results, with a 52.3% decrease in mean seizure rate compared to the preceding six months. At one stage, he experienced the lowest seizure rate recorded for him for the last six years. A median test confirmed that the distribution of clinical attacks in the baseline and feedback training periods differed significantly ($\chi^2 = 4.6$, $p < 0.5$). The other two subjects exhibited very little change in mean seizure rate. For B.H. there was a non-significant decrease of 4.5% ($\chi^2 = 0.5$, $p > 0.5$), and for N.L. a non-significant increase of 4.1% ($\chi^2 = 0.006$, $p > 0.5$). The seizure rate per week over the six month baseline and 12-16 Hz training periods is plotted for each of the three subjects in Figure 6.3.

The data from the 12-16 Hz, 8-10 Hz and inhibit registers of the EEG feedback unit were then examined. It was found that there were individual differences in performance, as with the laboratory studies described previously, and intra-subject variance across the forty training sessions was also considerable on all of the measures.

Since the small sample size precludes a meaningful analysis of variance, a regression model was adopted for each subject individually. Clearly, caution is needed in the interpretation of such single-case statistics (Johnson, 1976); and no generalisation is possible other than that the effect holds for a particular individual. Nevertheless, the regression model can be very useful for a descriptive analysis of the data by indicating whether there is any tendency for a variable to change progressively across training sessions, and has the advantage that the response pattern of each subject can be evaluated individually. Linear regression lines were therefore fitted to scatterplots

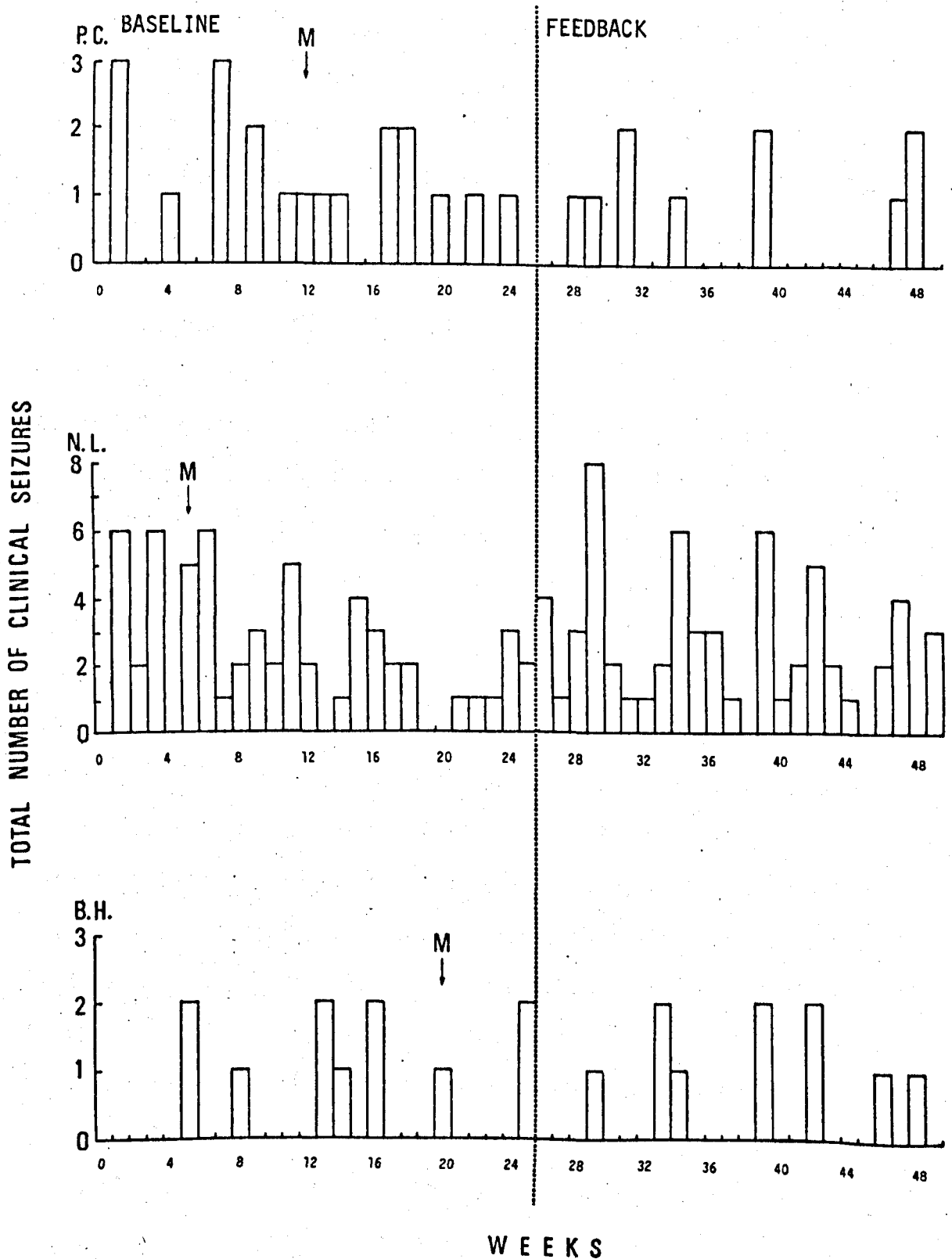


Figure 6.3
Total number of seizures per week for the three subjects (tonic-clonic seizures for P.C. and B.H., and tonic-clonic and psychomotor seizures for N.L.) during the baseline and the 12-16 Hz feedback training conditions. M denotes change of medication.

of the data to provide a measure of the general trends. Some data points were missing for some sessions, owing to electrical, mechanical or human failure. However, these represent only a small proportion of the overall data from the total of two hundred and forty training sessions involved in the clinical study.

Percent-time 12-16 Hz activity against training sessions is plotted for the three subjects in Figure 6.4. It can be seen that, whereas both subjects N.L. and B.H. demonstrated a significant progressive increase in 12-16 Hz EEG, subject P.C. did not. Thus, from these data alone it is evident that the significant decrease in seizure activity experienced by subject P.C. was not accompanied by any increase in the 12-16 Hz components of the rolandic EEG. Conversely, for subjects N.L. and B.H. the increase in 12-16 Hz activity did not result in any clinical improvement.

The subjects were questioned during each session about their strategies for manipulating the feedback. It was apparent that none of them formulated any clear response association. Moreover, there was no evidence of a consistent selective enhancement of 12-16 Hz activity above baseline levels for any of the subjects. The ratio of the amount of 12-16 Hz activity during contingent training to baseline levels (expressed as a percentage) is plotted for each of the subjects in Figure 6.5. It is clear that they all exhibited considerable variability in their performance across sessions. Matched-pair t-tests indicated that the mean percent-time training and baseline levels of 12-16 Hz did not differ significantly for any of the subjects (P.C.: $t = 0.42$, $df = 39$, $p > 0.9$; N.L.: $t = 0.49$, $df = 35$, $p > 0.6$; B.H.: $t = 1.18$, $df = 38$, $p > 0.2$).

Toward the end of the training programme, subject N.L. produced extended bursts of 12-16 Hz activity of up to one second in some sessions. In addition, some waveforms in this frequency range could occasionally be discerned in the raw EEG trace, as shown in Figure 6.6. However, no

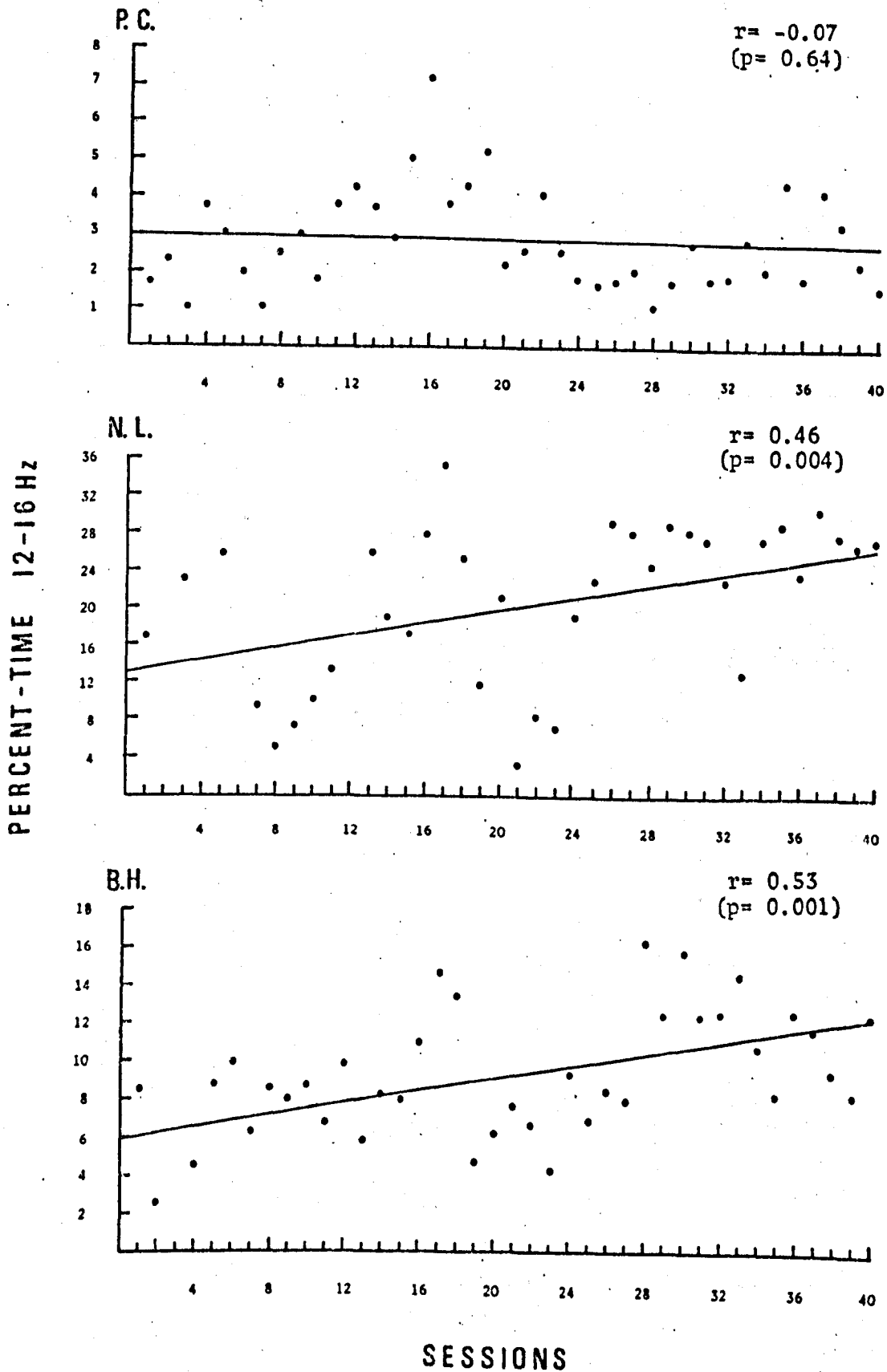


Figure 6.4
Percent-time 12-16 Hz activity against sessions of training for the three subjects. Linear regression lines are fitted to the data plots. The correlation coefficients are also listed together with their probability values.

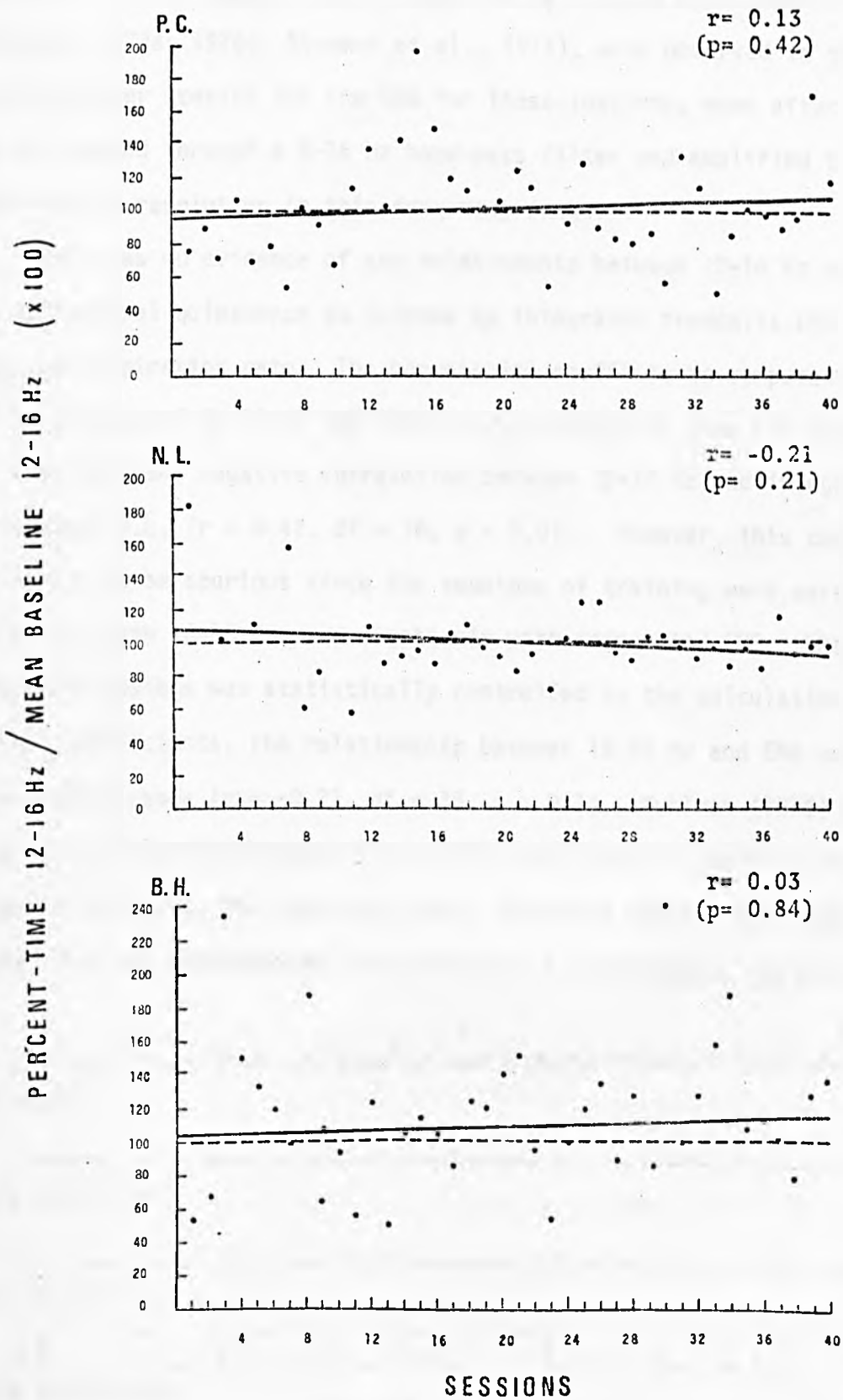


Figure 6.5

Ratio of training to mean baseline levels of 12-16 Hz activity (expressed as a percentage) against training sessions for the three subjects.

dominant 12-16 Hz peaks, such as reported by Sterman and co-workers (Sterman, 1973a; 1976c; Sterman et al., 1974), were observed in the averaged power spectra for the EEG for these sessions, even after the EEG was passed through a 8-16 Hz band-pass filter and amplified to maximise the resolution in this frequency range.

There was no evidence of any relationship between 12-16 Hz activity and behavioural quiescence as indexed by integrated frontalis EMG, heart rate, or respiration rate. The correlation coefficients computed between the percentage of 12-16 Hz and these various measures were all low, except for a significant negative correlation between 12-16 Hz and integrated EMG for subject N.L. ($r = 0.42$, $df = 36$, $p < 0.01$). However, this correlation was likely to be spurious since the sessions of training were correlated positively with 12-16 Hz, and negatively with integrated EMG. When the effect of sessions was statistically controlled by the calculation of partial coefficients, the relationship between 12-16 Hz and EMG was reduced below significance ($r = -0.21$, $df = 35$, $p > 0.1$). Kuhlman (1976) similarly found no relationship between forearm EMG and 12-16 Hz activity in five epileptic subjects, and concluded that, "Repeated testing provided no support for the hypothesised involvement of a sensorimotor rhythm, motor

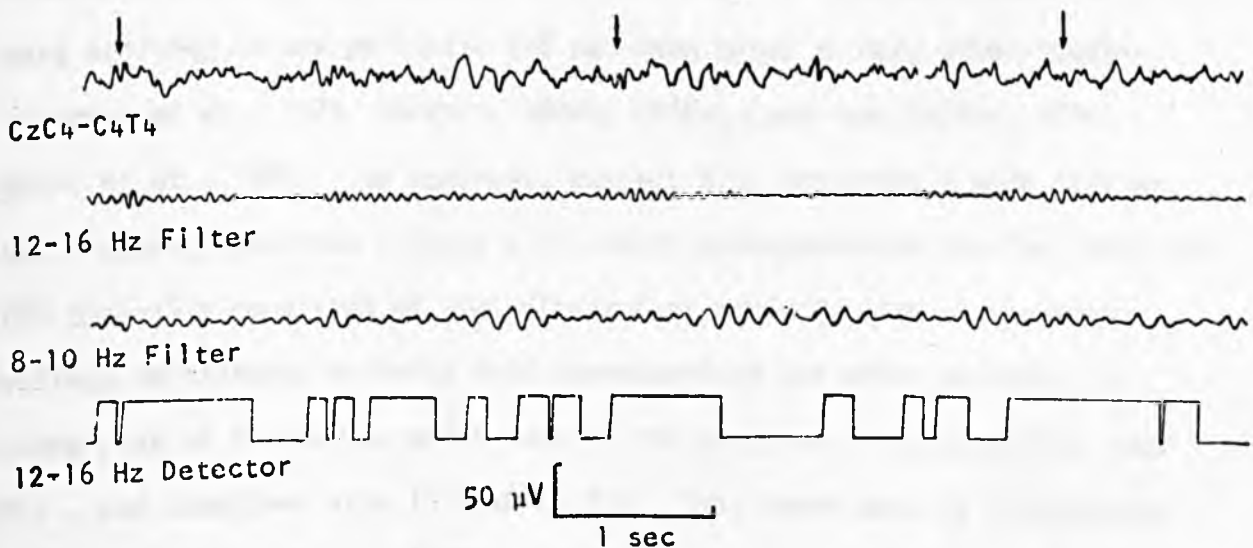


Figure 6.6

Section of record from subject N.L. showing long bursts of 12-16 Hz activity. In this instance, there is also some evidence of corresponding waveforms in the raw EEG trace (arrowed).

inhibition, or behavioural quiescence in the training effects".

Subject P.C. exhibited a significant reduction of EMG scores over sessions as indicated in Figure 6.7. The mean EMG levels of baseline and training periods did not differ significantly ($t = 0.59$, $df = 35$, $p > 0.5$). For subjects B.H. and N.L. there were no progressive changes in EMG levels across sessions, but B.H. presented significantly lower mean scores during training periods than during baseline periods ($t = 8.2$, $df = 3.5$, $p < 0.0001$), and N.L. significantly more ($t = 5.0$, $df = 37$, $p < 0.0001$).

All three subjects demonstrated a very significant progressive increase in 8-10 Hz activity over the training period; as shown in Figure 6.8. For subject B.H. this activity was significantly augmented above baseline levels ($t = 2.92$, $df = 35$, $p < 0.01$); conversely, for subject N.L. it was significantly suppressed below baseline levels ($t = 2.11$, $df = 34$, $p < 0.05$). There was no significant difference between training and baseline 8-10 Hz levels for subject P.C. ($t = 0.02$, $df = 36$, $p > 0.9$).

When computer facilities later became available, a spectral analysis was performed on the final baseline EEG data for each session. The results showed that the spectra for both subjects N.L. and P.C. were dominated by slow wave activity, as depicted in Figure 6.9. This preponderance of slow wave activity in the epileptic EEG has been noted in many other studies (Sterman et al., 1974; Sterman, 1976b; 1976c; Lubar and Bahler, 1976; Wyler et al., 1976). In contrast, subject B.H. presented a much flatter EEG frequency spectrum (Figure 6.9), which corresponds to the fact that her EEG typically consisted of low voltage fast activity without the high voltage delta-theta activity that characterised the other patients. A sharp peak at 6-7 Hz was often seen in the spectrum of subjects N.L. and P.C., and sometimes also in that of B.H. This theta peak is illustrated in Figure 6.10. Sterman (1973a) similarly reports that a dominant spectral peak at 6-8 Hz was characteristic of his epileptic patients.

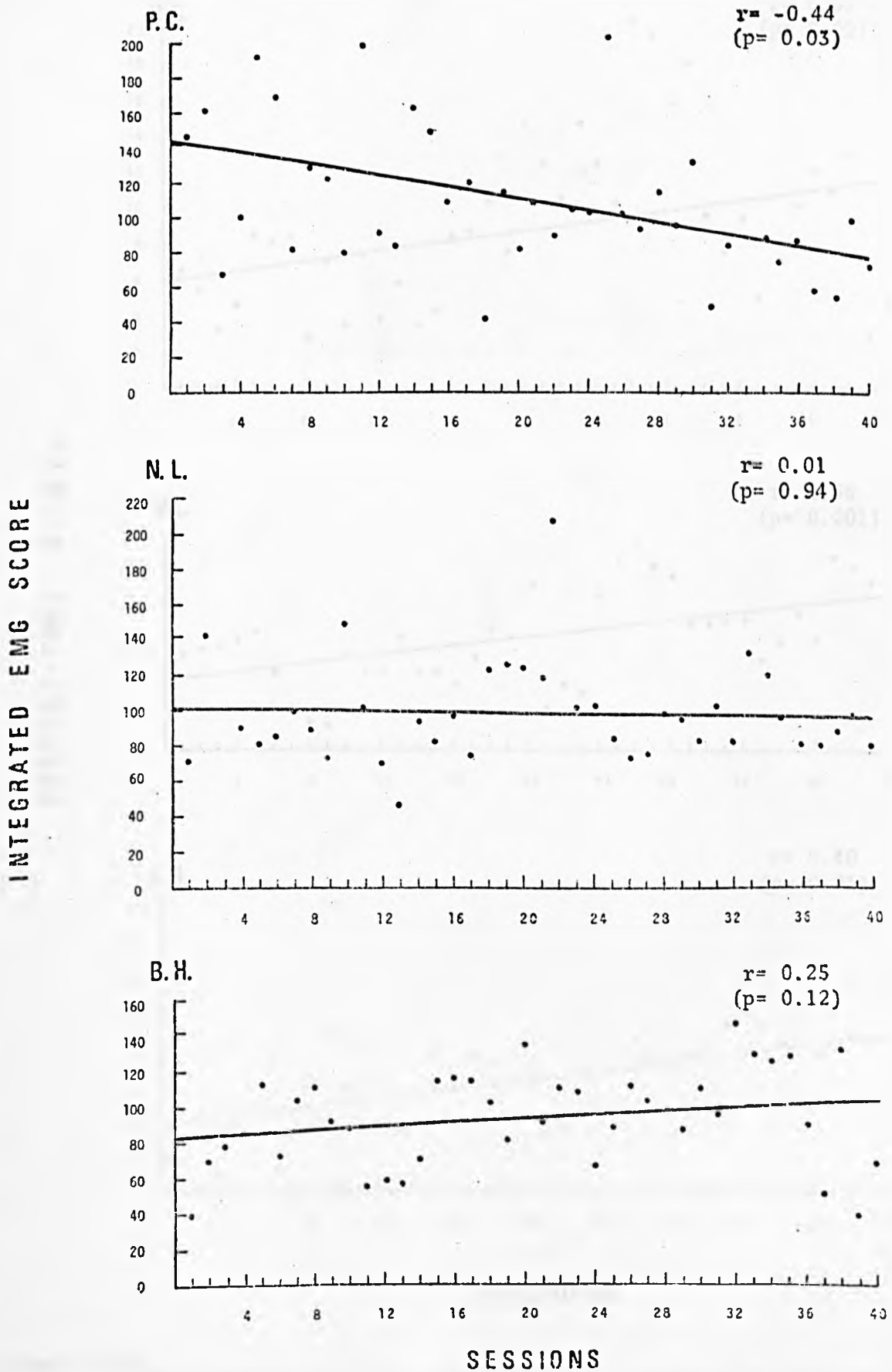


Figure 6.7 Integrated frontalis EMG scores (on an arbitrary but linear scale) against training sessions for the three subjects.

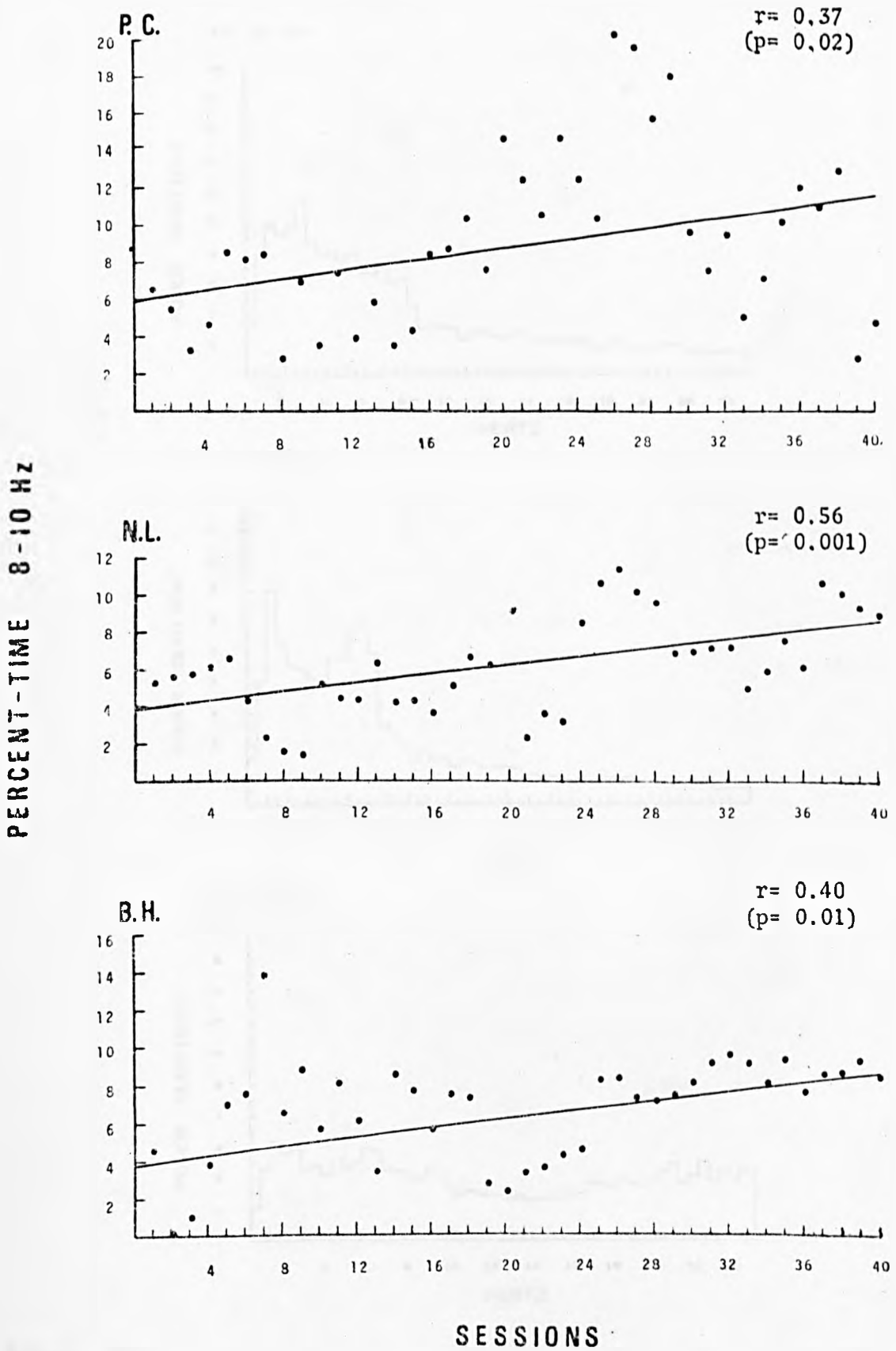


Figure 6.8
Percent-time 8-10 Hz against training sessions for the three subjects.

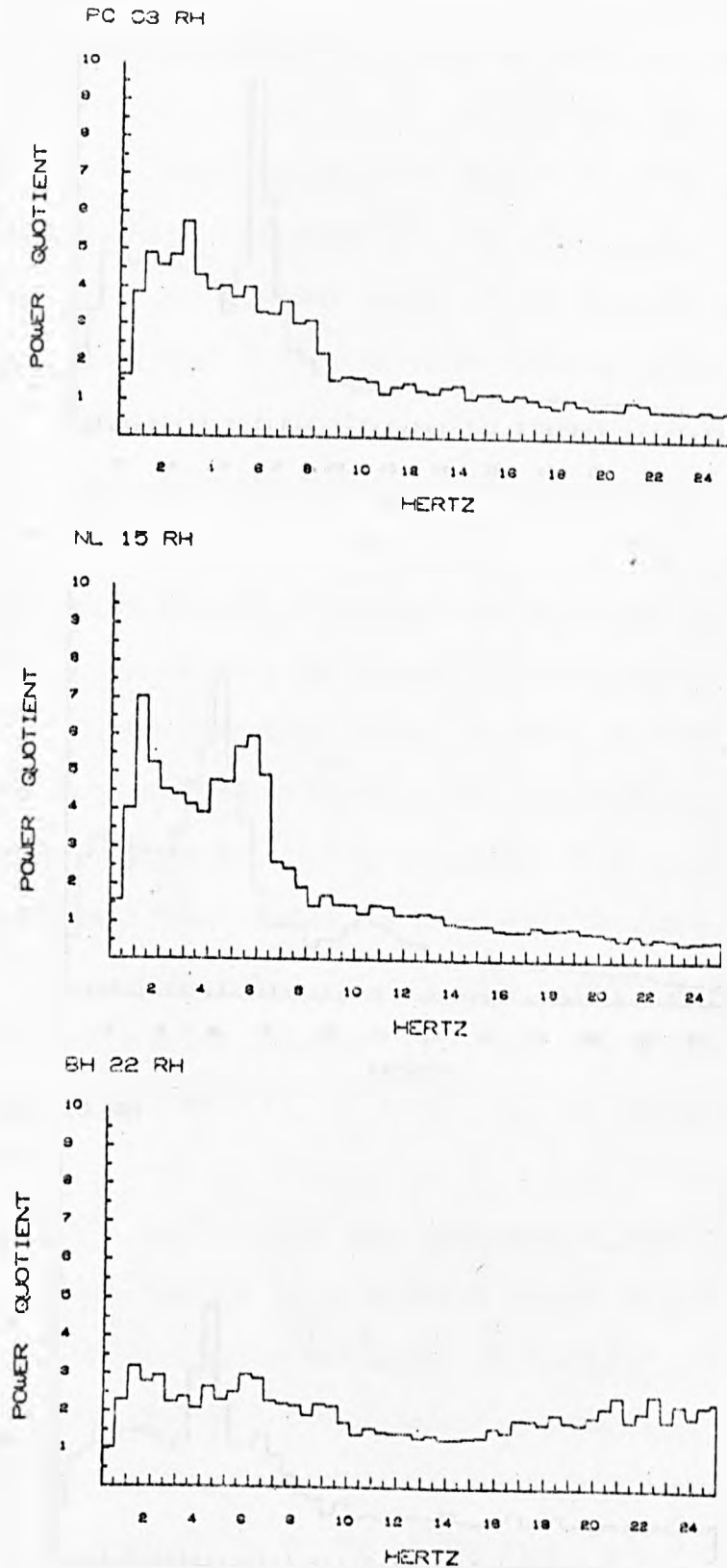


Figure 6.9

Normalised EEG power spectra illustrating the typical predominance of slow wave activity for subjects P.C. and N.L., but not for subject B.H. The mean spectral frequency is 8.47 Hz for P.C., 7.63 Hz for N.L., and 12.21 Hz for B.H.

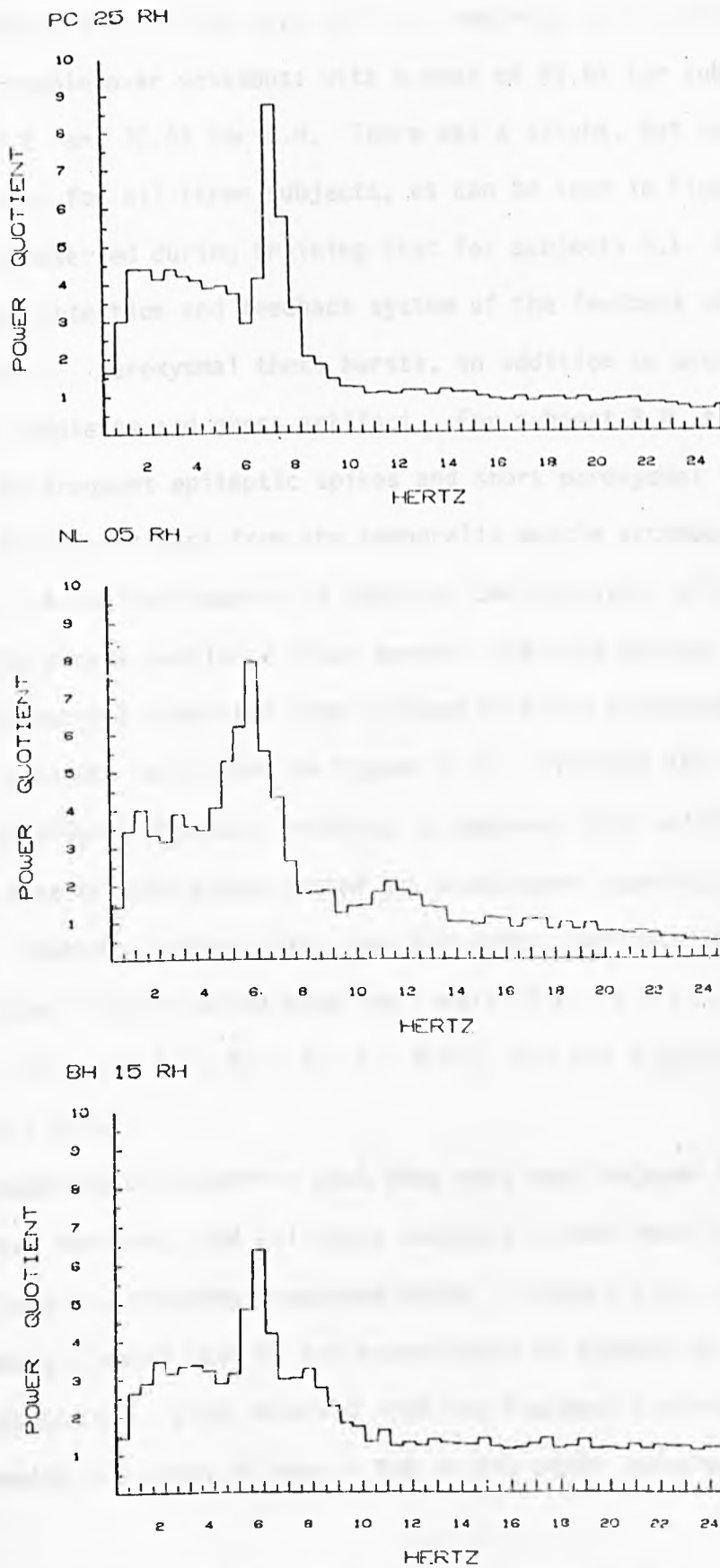


Figure 6.10

Examples of a strong 6-7 Hz spectral peak in the EEG of all three subjects.

The proportion of slow wave (2-7 Hz) activity in the EEG was relatively stable over sessions: with a mean of 48.8% for subject N.L., 46.3% for P.C. and 32.4% for B.H. There was a slight, but non significant, downward trend for all three subjects, as can be seen in Figure 6.11.

It was observed during training that for subjects N.L. and P.C. the high voltage detection and feedback system of the feedback unit mainly responded to paroxysmal theta bursts, in addition to occasional poly spike-wave complexes and gross artifact. For subject B.H. the system was activated by frequent epileptic spikes and short paroxysmal theta spindles, and often by EMG artifact from the temporalis muscle accompanying jaw clenching¹. A digital counter to register the operation of the inhibit circuit only became available after several training sessions had been given. The percent-time that high voltage activity occurred across the remaining sessions is plotted in Figure 6.12. Although the subjects received contingent feedback training to suppress this activity, it can be seen that none of them demonstrated any consistent learning curve across sessions. However, subjects N.L. and B.H. both suppressed high voltage activity significantly below baseline levels (N.L.: $t = 2.1$, $df = 23$, $p < 0.05$; B.H.: $t = 2.1$, $df = 23$, $p < 0.05$), but not subject P.C. ($t = 1.48$, $df = 21$, $p > 0.1$).

The subjects all reported that they very much enjoyed coming along to the feedback sessions, and all three subjects stated that they had "felt better" since the training programme began. Subject P.C., in particular, was extremely pleased that he had experienced an absence of attacks for six weeks. Subject N.L. also reported that the feedback training had enabled him to prevent the onset of one or two of his minor seizures (which were

¹It should be remembered that the amplitude threshold of the inhibit circuit was set to 50 μV for subjects N.L. and P.C., and 40 μV for subject B.H., so as to prevent the spurious operation of the frequency detection and feedback systems. The term high voltage is thus defined relative to the low thresholds of these systems.

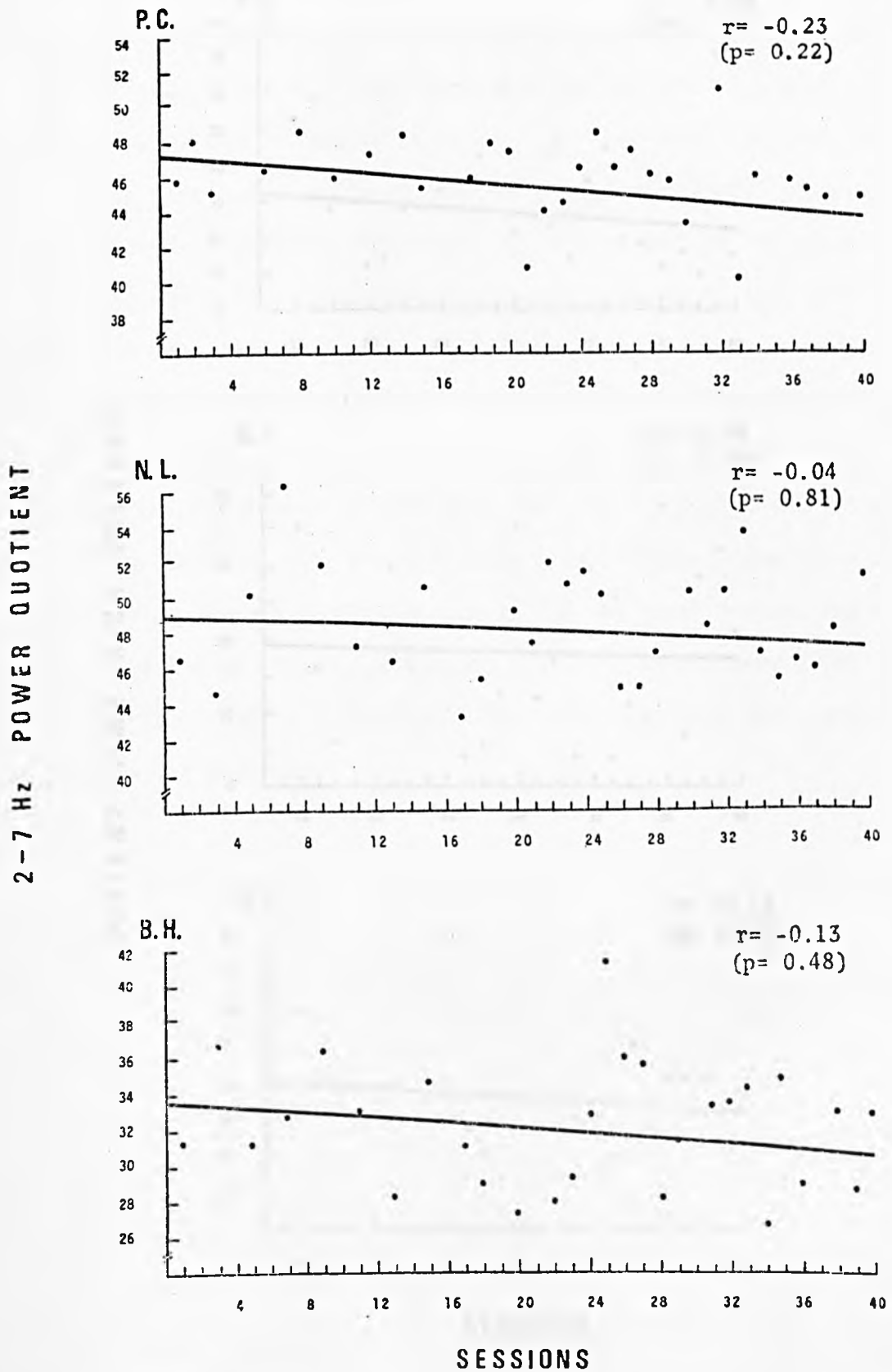


Figure 6.11 *Relative power of 2-7 Hz activity against training sessions for the three subjects. (Data was recorded on alternate sessions during the initial part of the training programme.)*

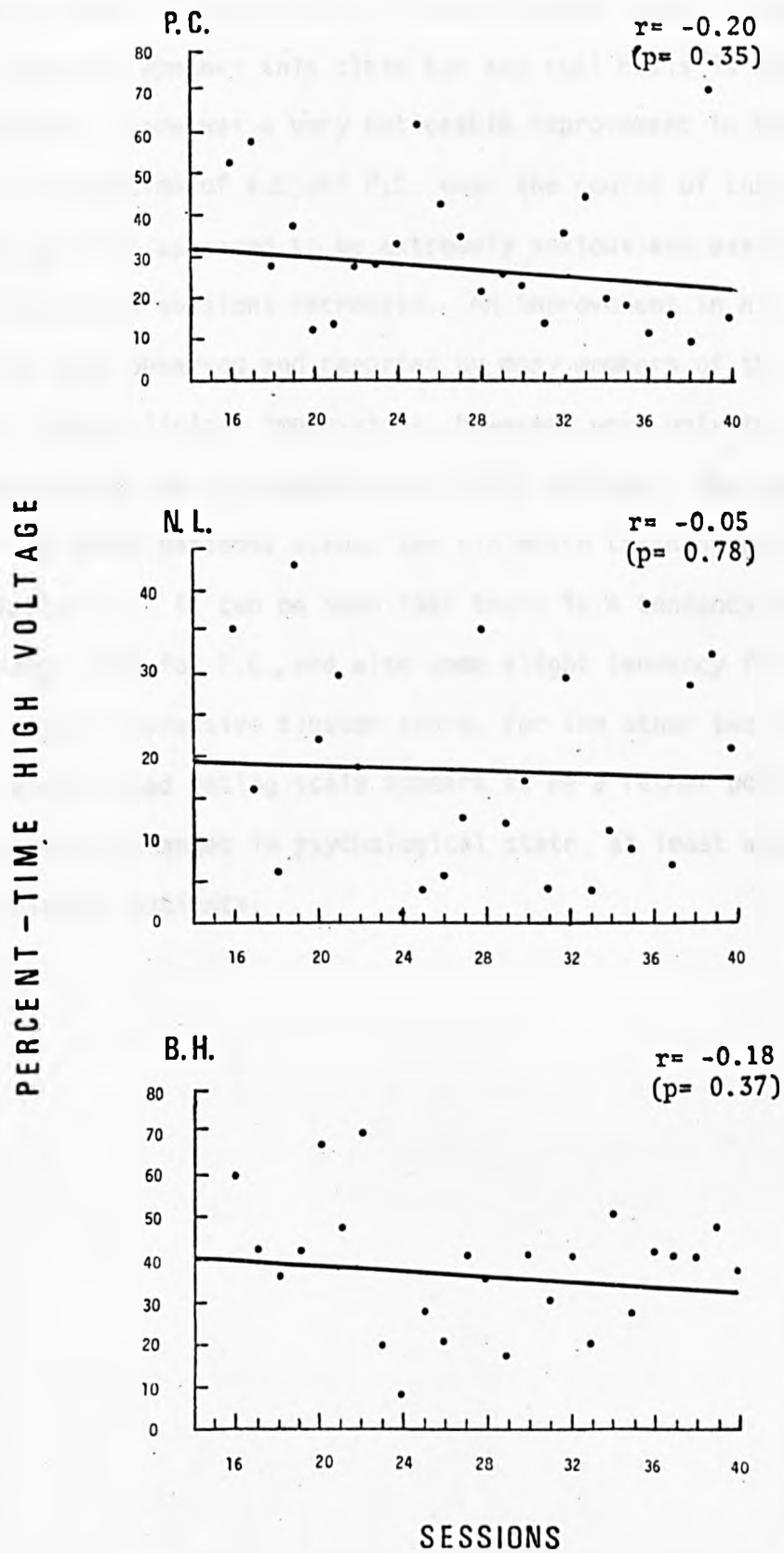


Figure 6.12 Percent-time high voltage activity across training sessions for the three subjects. A separate register to quantify this data was not available during the earlier sessions.

preceded by an aura in the form of a "buzzing sound" and a "muzzy" feeling). However, whether this claim had any real basis is not known.

In addition, there was a very noticeable improvement in the general psychological condition of subject P.C. over the course of the training period. He at first appeared to be extremely anxious and easily startled, but this lessened as sessions increased. An improvement in his general behaviour was also observed and reported by many members of the staff of the Centre. These clinical impressions, however, were only to a slight extent reflected by the *Wittenborn* psychiatric ratings. The standardised scores for the three patients across the six month training period are listed in Table 6.2. It can be seen that there is a tendency for a decline in the anxiety score for P.C., and also some slight tendency for a lessening of the anxiety or depressive cluster scores for the other two patients. Thus, this widely used rating scale appears to be a rather poor index of clearly observable changes in psychological state, at least when applied to non-psychiatric patients.

Table 6.2. Standard score profiles on the Wittenborn Psychiatric Rating Scale for the three patients across the period of 12-16 Hz feedback training.

SYMPTOM CLUSTER	JULY			AUGUST			SEPTEMBER			OCTOBER			NOVEMBER			DECEMBER		
	PC	NL	BH	PC	NL	BH	PC	NL	BH	PC	NL	BH	PC	NL	BH	PC	NL	BH
Anxiety	5	4	2	4	3	2	3	1	2	4	1	2	3	3	2	4	3	2
Hysterical Conversion	2	3	3	1	2	3	2	1	3	2	1	3	1	1	3	1	1	1
Manic State	4	6	5	5	6	5	5	5	5	5	7	5	5	5	7	5	3	1
Depressive Total	6	4	3	6	3	3	5	1	1	6	2	1	4	2	2	5	4	1
Obstructive	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Apathy	5	4	1	4	5	1	4	1	1	5	1	1	4	1	1	4	2	1
Withdrawal	7	3	2	7	2	2	7	1	1	7	1	1	7	3	1	7	6	1
Affective Flatness	6	4	4	5	1	3	4	1	1	5	3	1	1	1	2	2	2	1
Schizophrenic Total	4	1	1	2	1	1	3	1	1	2	1	1	1	1	2	1	1	2
Verbal	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2
Motoric	4	1	1	2	1	1	3	1	1	2	1	1	1	1	2	1	1	2
Psychotic Total	5	6	4	4	4	1	4	5	3	3	5	4	3	4	4	4	3	2
Attention Demanding	2	5	1	2	4	1	1	5	2	1	6	2	1	4	2	2	2	2
Assaultive	5	4	3	4	2	1	4	2	2	3	2	3	3	2	3	4	2	2
Paranoia	2	1	1	2	1	1	2	1	2	2	1	2	2	1	1	2	1	1
Hebephrenic Total	4	3	2	3	1	2	2	1	2	2	1	2	3	1	2	2	1	2
Incontinence	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Silliness	3	1	2	2	1	2	1	1	2	1	1	2	1	1	2	1	1	2
Resistance	3	3	1	3	1	1	2	1	1	2	1	1	3	1	1	2	1	1
Compulsive Obsessive	3	3	1	2	1	1	2	1	1	1	1	1	2	1	2	3	1	2
Intellectual Impairment	4	4	1	1	1	1	1	1	1	2	1	1	2	1	1	1	1	1
Homosexual Dominance	3	1	1	1	3	1	1	1	1	1	3	1	3	2	1	3	3	3
Ideas of Grandeur	2	1	1	1	2	1	2	1	1	1	1	1	1	1	1	1	1	1

DISCUSSION

The results confirm that an impressive reduction in epileptic seizure manifestation can be induced through feedback training of central 12-16 Hz EEG. However, the data clearly do not support an association between clinical benefit and the selective enhancement of this frequency band. Kaplan (1974; 1975) and Kuhlman (Kuhlman, 1976; Kuhlman and Alison, 1977) were similarly unable to find any evidence of an increase in the abundance of 12-14 Hz rolandic EEG in those of their patients who exhibited a reduction in seizure frequency. Moreover, Sterman (1976c), who in his current study of EEG feedback training for the treatment of epilepsy has begun to quantify his data, reports that following various training procedures over a twelve month period, three patients showed a pronounced reduction in seizure incidence, combined with a generalised increase in high frequency EEG activity and a decrease in lower frequency activity. However, he notes that, "Despite these changes, and the fact that reward was provided for 12-15 Hz activity, the incidence of SMR often did not change with training".

According to Sterman, the training of SMR could produce a central neuronal reorganization, possibly through a morphological process by which repeated "exercise" of specific neural circuits leads to decreased excitability (Sterman and Friar, 1972; Sterman, 1973a, 1974). He further proposes that such changes result in a raised threshold for CNS seizure discharge, perhaps due to the facilitation of a central motor inhibitory process (Sterman et al., 1974, *see* 2:5:2), and that epileptics as a group are deficient in SMR (Sterman, 1974; 1976c; 1977a).

Seifert and Lubar (1975) similarly argue that, "SMR represents a type of cortical idling associated with inhibition of motor activity; hence increasing this idling through operant conditioning of the SMR could block overt epileptic activity". Finley also (1976) comments, "Assuming an

epileptic seizure represents a massive neuronal discharge, it appears that 12 Hz feedback alters the balance of excitation-inhibition within the epileptic brain".

Sterman also advances the hypothesis that SMR and sleep spindles might participate in a common inhibitory mechanism (Sterman, 1974; *see* 2:4:3), and claims that epileptics are deficient in sleep spindles as well as in SMR (Sterman, 1976c; 1977a). Thus he found that a heterogeneous group of thirteen epileptic patients demonstrated significantly less 11-15 Hz activity in their rolandic EEG obtained from sleep recordings than thirteen non-epileptic subjects (Sterman, 1976c). He proposes that spindling at this frequency range might therefore be refractory to seizure activity, and that the feedback training of SMR might enhance this refractory phenomenon (Sterman, 1974; 1976c). However, this theory is rebutted by the observation of Wyler (1974) that sleep spindles in the epileptic monkey are associated with an enhancement of pathological discharges and seizure activity. Moreover, Wyler et al. (1976) found that 12-14 Hz spindles augmented abnormal neuronal burst discharges in the epileptogenic monkey cortex to the point that in many instances a secondary generalised seizure was precipitated. Several other workers have also demonstrated that lesions of the rostral thalamic region both suppress sleep spindles and greatly reduce generalised seizures (Feeney and Gullotta, 1972; Kusske et al., 1972).

The hypothetical involvement of SMR in the inhibition of epileptic activity is based on the finding that operant conditioning of SMR in the cat led to an increased latency of seizure onset to the administration of monomethylhydrazine (Sterman et al., 1969; Sterman, 1976a). This compound is thought to elicit tonic-clonic seizure activity by disrupting the metabolism of inhibitory interneurons in much the same manner as strychnine (Sterman, 1973a). Hence, the reduction in seizure manifestation of

Sterman's SMR trained cats during experimentally induced neuronal hyperexcitability can reasonably be supposed to stem from a reduction in afferent stimulation, as a result of the state of behavioural quiescence that is the corollary of feline SMR. It is much less likely that any such effect should apply in general to epilepsy.

Since the evidence clearly demonstrates that the clinical effects of SMR feedback training are not dependent on any enhancement of SMR activity, it appears to be very difficult to continue to maintain the SMR hypothesis. Similarly, Wyler et al. (1976) recently reached the conclusion that, "There is not a particular rhythm exclusive to the rolandic cortex which if reinforced will provide adjunctive therapy in all types of epileptics."

The significant reduction in seizure rate demonstrated by subject P.C. in this study was very likely associated with the changes in his psychological state that were also observed. This may have been in the form of a placebo effect as a consequence of the close involvement with the patients that was necessarily a part of the study (*see* 6:7:1). Alternatively, Kaplan (1975) has proposed that the effects of EEG feedback training on epilepsy stem from the relaxation therapy which is a corollary of the feedback setting. A reduction in anxiety over the course of training for subject P.C. is suggested by the general clinical impression and the *Wittenborn* ratings. The decline in frontalis EMG levels for both training and baseline periods might also reflect decreased anxiety, since a positive relationship between manifest anxiety and frontalis EMG has been reported (Malmo and Smith, 1955). Thus it is possible that the clinical improvement for subject P.C. resulted from an amelioration of the seizure precipitating effects of emotional excitement or stress, as will be further discussed below (*see* 7:2:2).

6:5 CONTROL PROCEDURE: FEEDBACK TRAINING OF CENTRAL 8-10 Hz EEG

6:5:1 Introduction

As discussed previously (*see* 2:4:2), there is considerable confusion in the literature concerning the relationship of feline SMR to human mu rhythm. Some authors regard SMR as an homologue of mu rhythm, whereas others hold that the SMR is a discrete EEG pattern in both man and cat. Thus, in investigating SMR feedback training for the treatment of epilepsy, it would seem logical to compare the effects of training mu frequencies and SMR frequencies. Moreover, the therapeutic effects of SMR training appear to be related to motor inhibition and the human mu rhythm is known to be closely associated with motor inhibition. Hence, although pure mu rhythm is infrequently observed in the EEG, there is a stronger theoretical basis for investigating whether the enhancement of rolandic EEG in the mu frequency range has any therapeutic effects on epilepsy, than for simply extrapolating SMR from the cat to man.

The EEG feedback unit was designed so that the clinical effects of training EEG in the SMR and in the mu frequency ranges respectively could be compared. The data obtained during the 12-16 Hz feedback training condition indicated that all three subjects exhibited an increase in the 8-10 Hz mu frequency band. Therefore, as a control procedure, contingent feedback of 8-10 Hz activity was provided in order to determine whether it too might result in any clinical benefit.

6:5:2 Method

The same apparatus was used as before, but with the feedback contingency switched so that the red lamp signalled the occurrence of 8-10 Hz activity in the EEG. Feedback of high voltage activity was again given by the green lamp, and similarly coins or *Smarties* were provided as a

reward for responses exceeding criterion duration.

The alteration in procedure was made after a four week break for the Christmas period. The subjects were not informed, and none of them gave any indication during the subsequent training sessions that they noticed any change in the procedure. The density of feedback was approximately equivalent to that in the 12-16 Hz training condition.

The electrode placements for subjects P.C. and N.L. remained unchanged. For subject B.H., however, it was thought that the anterior-posterior derivation used for the detection of 12-16 Hz would also register some of her 8 Hz occipital alpha rhythm. Hence the electrode sites were shifted to a transverse derivation identical to that used for the other two subjects. The EMG, ECG and respirogram recordings were discontinued.

Apart from the above changes, the general procedures remained as described previously for the 12-16 Hz feedback training condition. The 8-10 Hz training was maintained for fourteen weeks.

6:5:3 Results

Seizure frequency per week is shown for the three subjects in Figure 6.13. Subject P.C. exhibited a 19% increase in mean seizure rate in comparison to the 12-16 Hz training condition, but this was not statistically significant ($\chi^2 = 0.17$, $p > 0.5$). A further, nonsignificant, increase of 8% was also exhibited by subject N.L. ($\chi^2 = 0.003$, $p > 0.5$). Conversely, subject B.H. experienced only two double seizures during this period, hence her mean seizure rate was 33% below that for the previous condition ($\chi^2 = 29.4$, $p < 0.001$). However, since the seizure rate for this subject is relatively low, it is rather an insensitive index of clinical change.

The reduction of anticonvulsant medication for subjects N.L. and P.C. was prescribed during this period. It appeared to have no obvious clinical

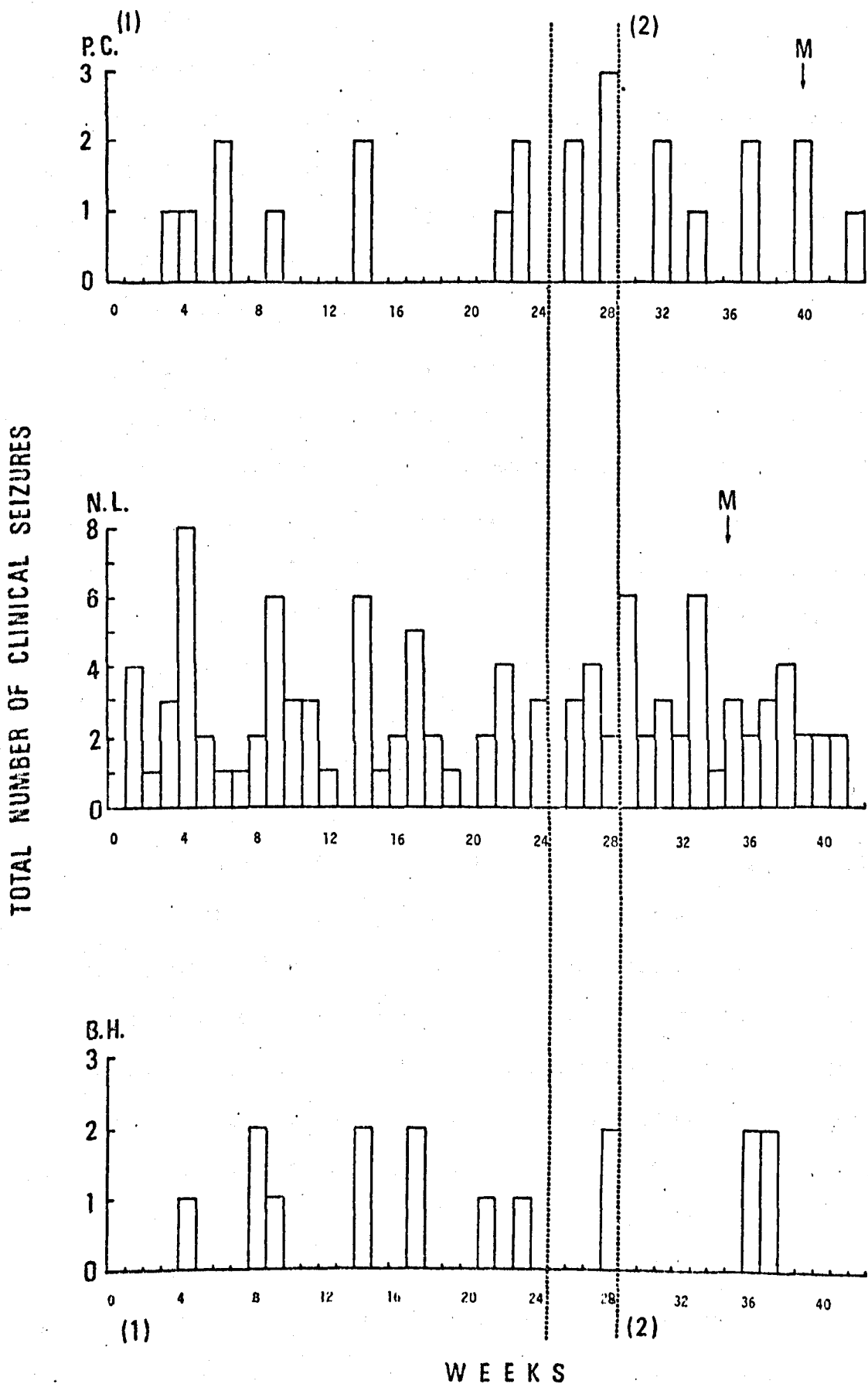


Figure 6.13

Weekly seizure incidence for three subjects across (1) the 12-16 Hz and (2) the 8-10 Hz training conditions. The dashed lines indicate a break for the Christmas vacation. M denotes medication changes.

consequences for either subject. Moreover, the drug serum level assays that were carried out over the course of the clinical study indicated that the change in medication had very little effect on the amount of anticonvulsants that actually entered the blood stream of the subjects, as demonstrated in Figure 6.14. It can be seen that most of the drug serum levels exhibit considerable lability, but there were no salient increases which could account for therapeutic effects. The only consistent change appears to be some decline in the serum level of phenobarbitone for subject N.L., which was not much influenced by the 30% reduction in drug intake. Wyler et al. (1976) similarly observed that drug serum levels varied considerably over several months, even though their patients were maintained on the same anticonvulsant regimen. In contrast, Sterman (1973a, 1974; Sterman et al., 1974) and Kuhlman (1976; Kuhlman and Alison, 1977) state that the blood serum levels for their subjects remained "stable".

The percentage of 8-10 Hz activity in the EEG over the twenty-four training sessions is plotted for the three subjects in Figure 6.15. B.H. was the only subject to exhibit a significant upward trend. Furthermore, she demonstrated a very significant enhancement of 8-10 Hz activity above baseline levels ($t = 6.79$, $df = 23$, $p < 0.0001$), indicating that the feedback training enabled her to exert some influence over her EEG. However, as Figure 6.16 illustrates, there was no progressive learning effect across sessions. This subject could not specify how she increased the occurrence of the appropriate feedback; but, from observations of her behaviour, it appeared to largely involve a conscious effort to relax, thus, she was often noticeably more tense during baseline periods. Neither of the other two subjects exhibited any significant difference between training and mean baseline levels of 8-10 Hz activity (P.C.: $t = 0.008$, $df = 23$, $p > 0.9$; N.L.: $t = 0.25$, $df = 23$, $p > 0.8$).

There was a very slight, but nonsignificant, increase in 12-16 Hz

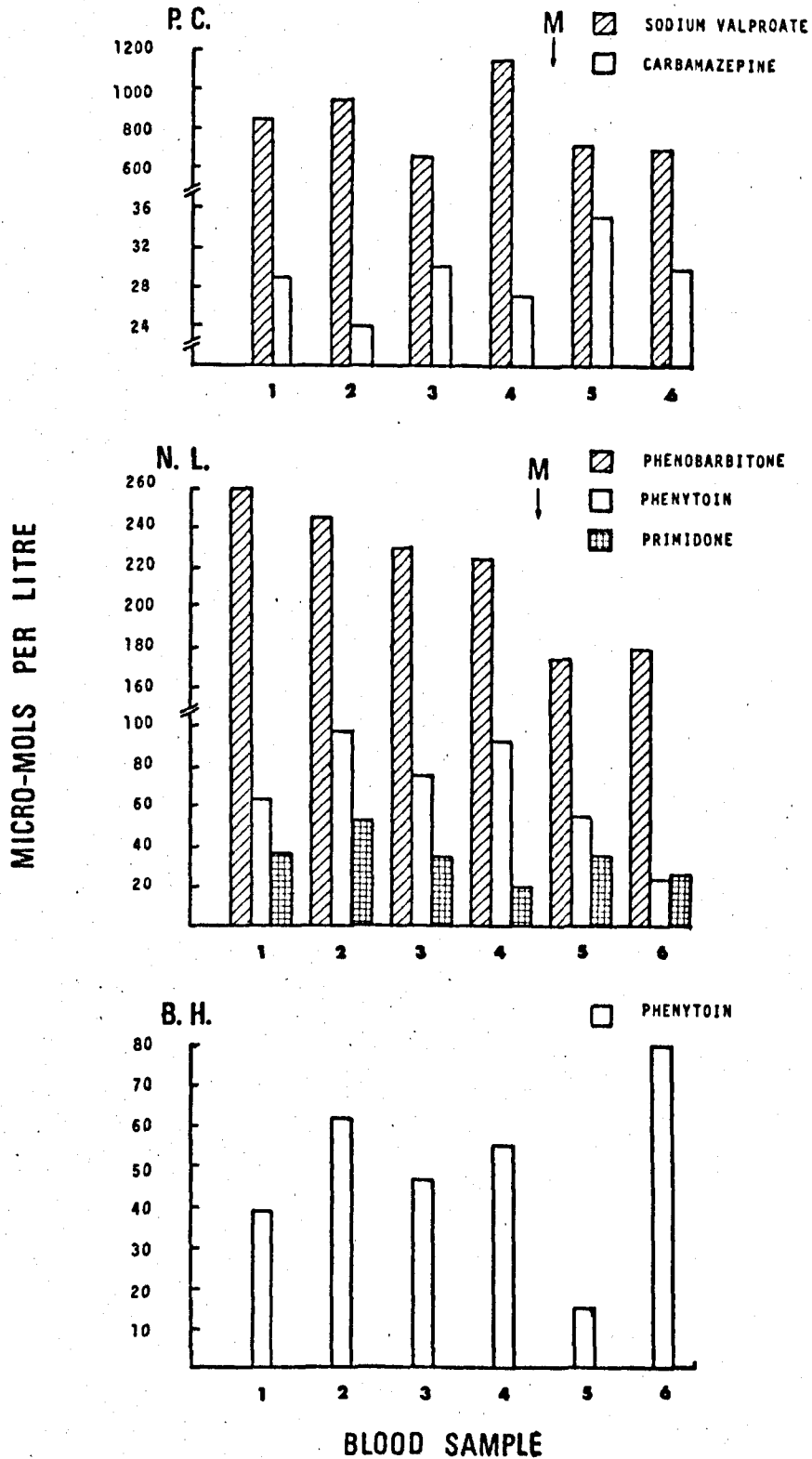


Figure 6.14

Serum levels of anticonvulsants in blood plasma, as determined by gas-liquid chromatography, for the three patients. The blood samples were taken at irregular intervals during the clinical study by the staff of the David Lewis Centre. M denotes the point at which the phenobarbitone dosage was reduced from 90 mg to 60 mg, and phenytoin dosage from 400 to 375 mg, for N.L.; and sodium valproate dosage from 3,000 mg to 2,400 mg for P.C.

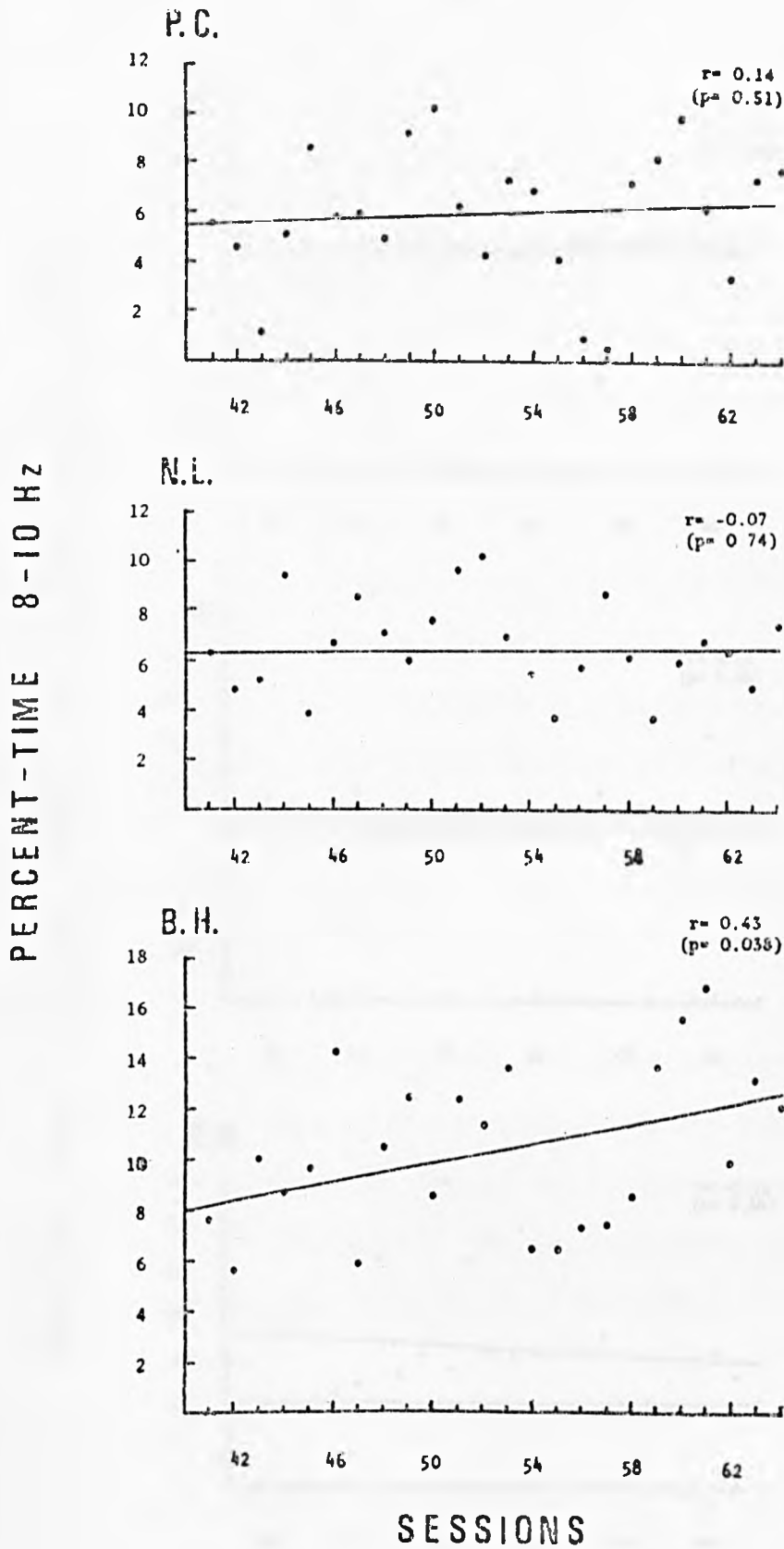


Figure 6.15
Percent-time 8-10 Hz against training sessions for the three subjects.

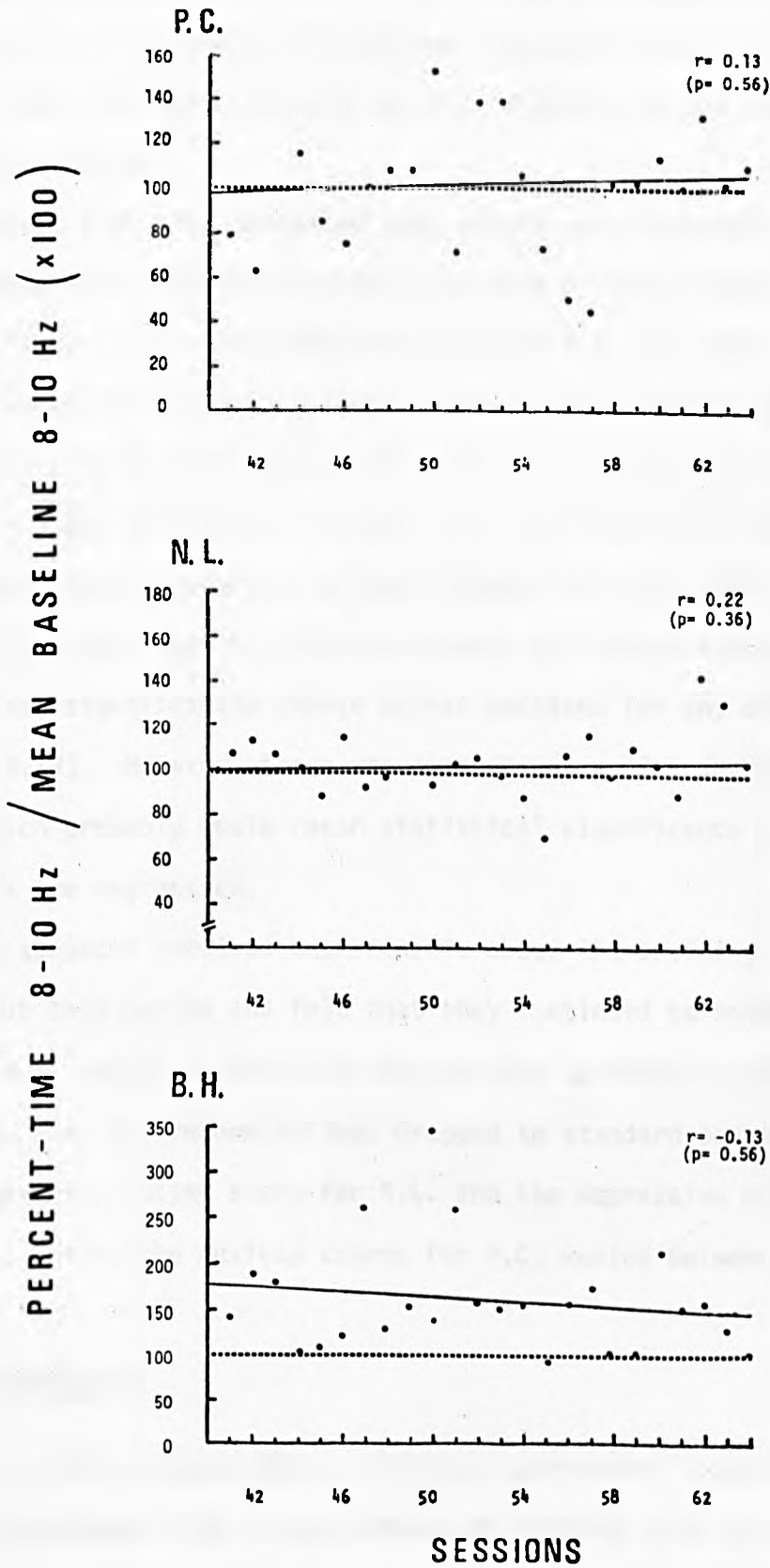


Figure 6.16

Ratio of training to mean baseline levels of 8-10 Hz activity (expressed as a percentage) against training sessions for the three subjects.

activity across sessions for all three subjects (Figure 6.17). This contrasts with the pronounced combined increases in 8-10 Hz and 12-16 Hz rolandic EEG that were observed for N.L. and B.H. during the 12-16 Hz training condition.

Subject B.H. also exhibited some slight, but nonsignificant, tendency to decrease high voltage EEG activity across training sessions, as can be seen in Figure 6.18. Both subjects N.L. and B.H., but not P.C., suppressed high voltage activity significantly below baseline levels during training, as in the 12-16 Hz training condition (N.L.: $t = 2.3$, $df = 23$, $p < 0.05$; B.H.: $t = 3.93$, $df = 23$, $p < 0.001$; P.C.: $t = 1.14$, $df = 23$, $p > 0.2$).

Power spectral analysis of the EEG data from the final baseline periods indicated that the relative amount of slow wave activity in the EEG did not significantly change across sessions for any of the subjects (Figure 6.19). However, there was some evidence of a downward trend for P.C., which probably would reach statistical significance with more data points in the regression.

The subjects remained enthusiastic about the training programme throughout this period and felt that they continued to benefit from it. Subject N.L. again reported that he was able to inhibit some of his minor seizures. The *Wittenborn* ratings dropped to standard scores of around 1 on the anxiety cluster score for N.L. and the depressive cluster score for B.H., whilst the anxiety scores for P.C. varied between 2 and 3.

6:5:4 Discussion

The results suggest that a clinical improvement in epilepsy is more closely associated with an enhancement of rolandic 8-10 Hz activity than with 12-16 Hz, since in both cases where a significant reduction in seizure rate was obtained - subject P.C. in the previous training condition and B.H. in this - the percentage of 8-10 Hz increased across sessions,

PERCENT-TIME 12-16 Hz

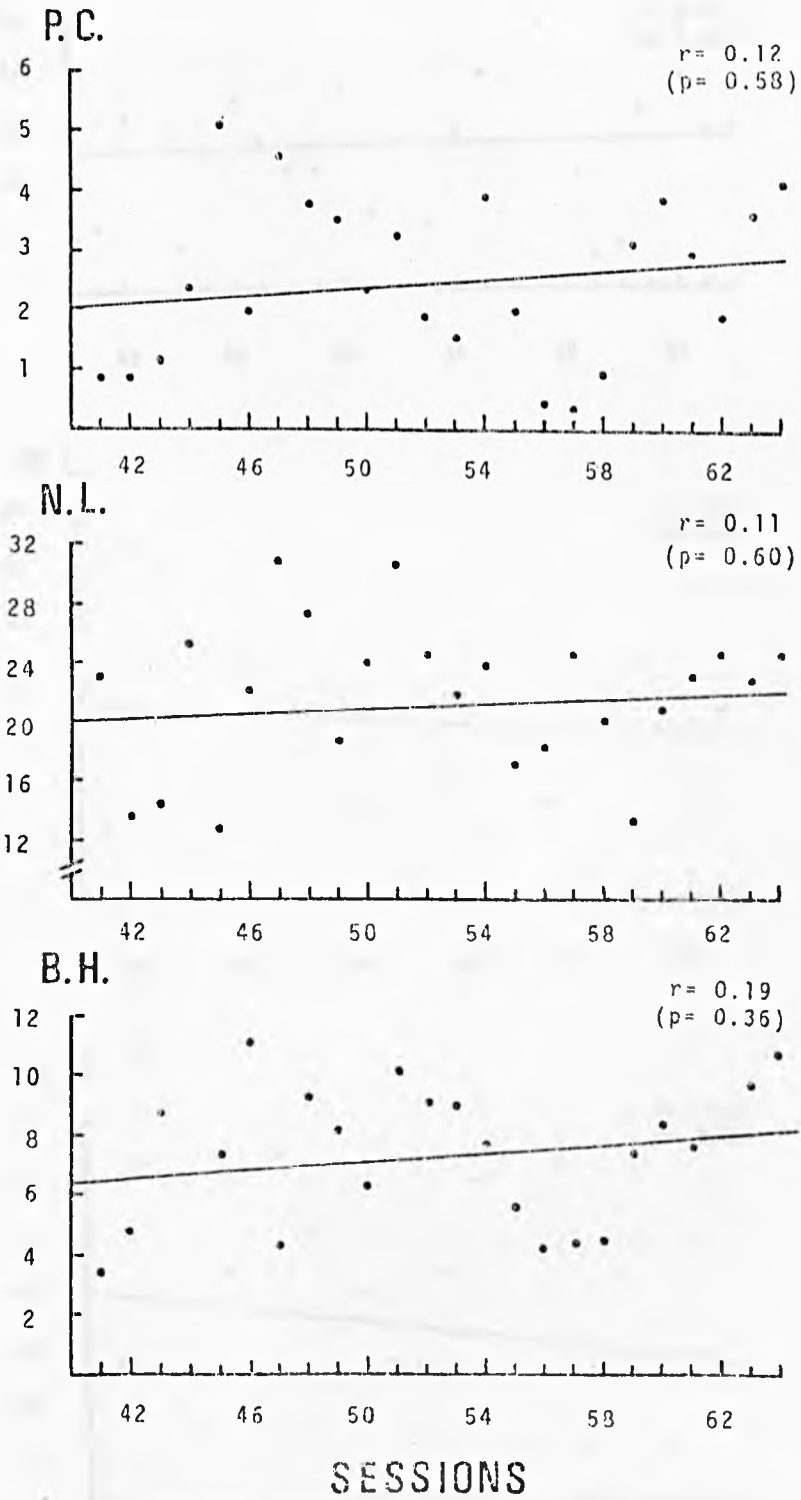


Figure 6.17
 Percent-time 12-16 Hz against training sessions for the three subjects.

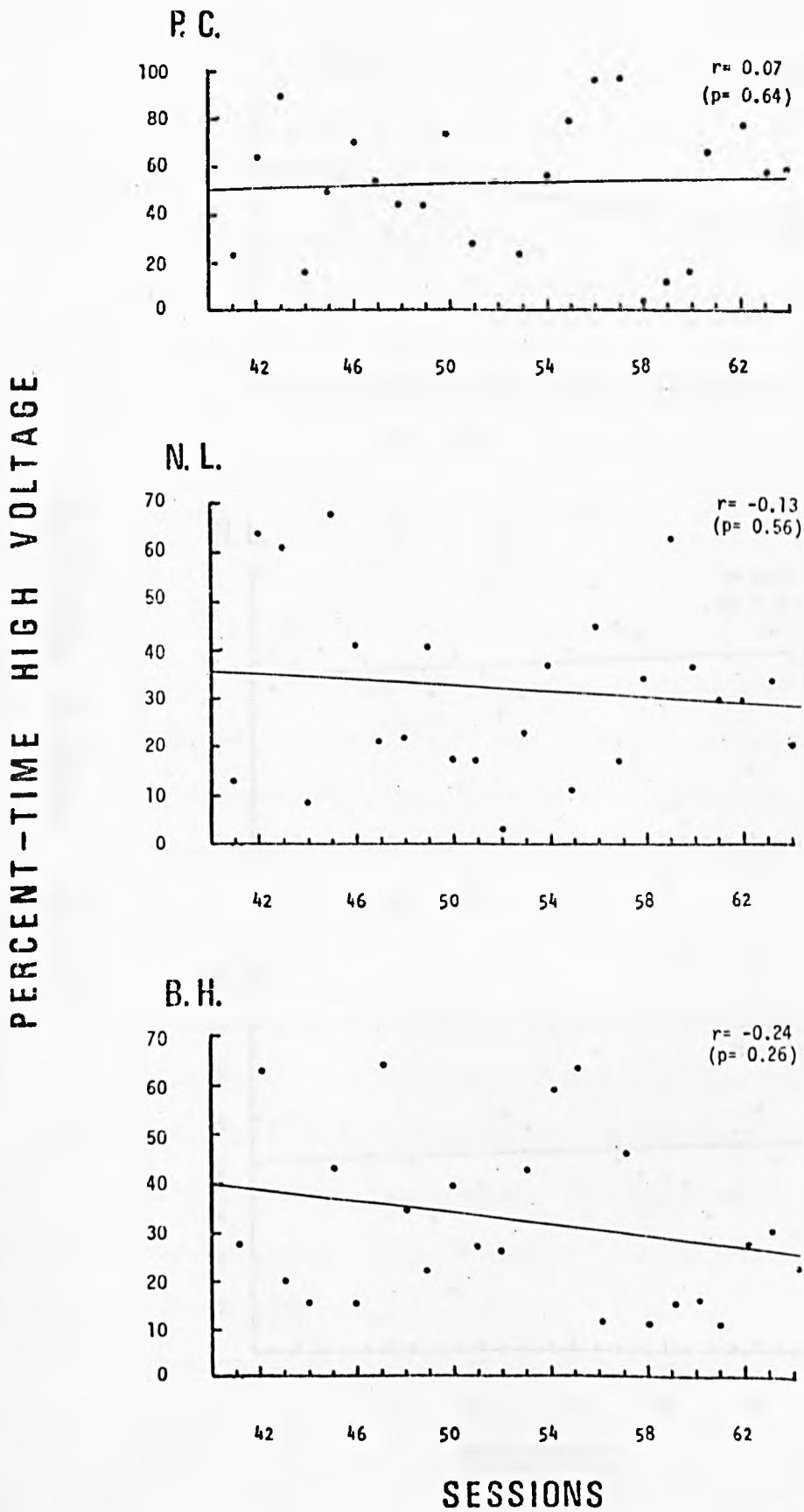
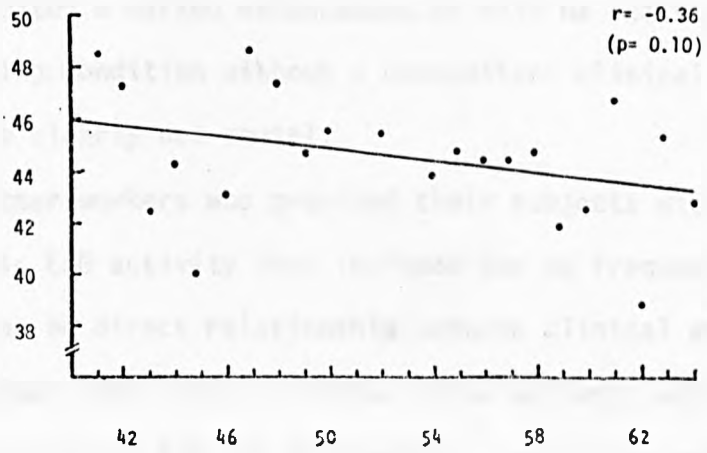


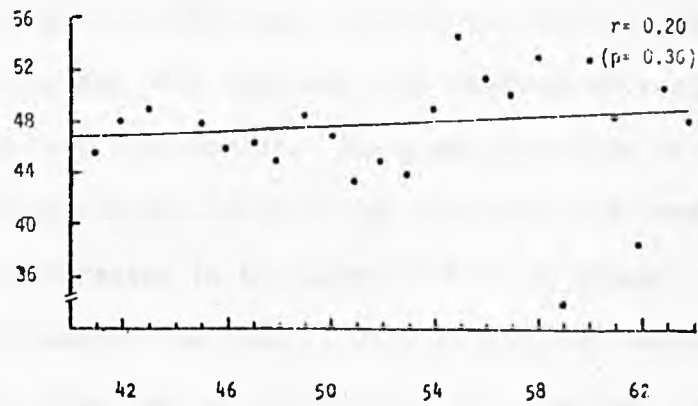
Figure 6.18
Percent-time high voltage activity against training sessions
for the three subjects.

2-7 Hz POWER QUOTIENT

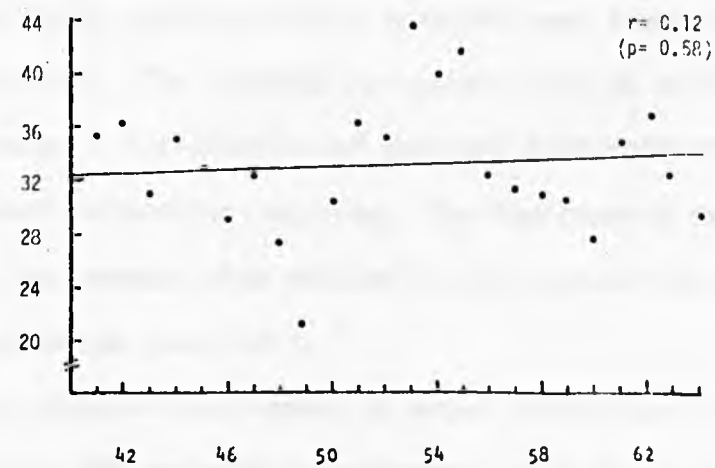
P. C.



N. L.



B. H.



SESSIONS

Figure 6.19

Relative power of 2-7 Hz activity against training sessions for the three subjects.

whereas the percentage of 12-16 Hz did not. However, since both subjects N.L. and B.H. exhibited a marked enhancement of 8-10 Hz activity during the 12-16 Hz training condition without a concomitant clinical change, the relationship is clearly not causal.

A number of other workers who provided their subjects with feedback training of rolandic EEG activity that included the mu frequency band also found that there was no direct relationship between clinical and EEG changes. Thus Kaplan (1974; 1975) provided three patients with feedback training of central 6-12 Hz EEG for 4-5 months. Two of the patients experienced a reduction in seizure rate, but this was not correlated with any increases in the 6-12 Hz EEG band. Similarly, Kuhlman (1976; Kuhlman and Alison, 1977) provided five patients with feedback of central 9-14 Hz for periods ranging from 4-10 months. There was a decline in the incidence of seizures, averaging 70% for three of the patients, but none of them showed any reliable increases in the power of 9-14 Hz present in the EEG.

It is doubtful whether the central 8-10 Hz activity recorded from the subjects in this study was representative of mu rhythm, since the characteristic mu waveform (pointed negative and rounded positive components) was not seen, and the 8-10 Hz spindles were always of less than one second duration. The increase in central 8-10 Hz activity for B.H. probably reflected a facilitation of cortical synchronisation as part of a more general relaxation response. The improvement in her clinical condition was perhaps also related to this relaxation response, as will be discussed below (*see* 7:2:2).

In view of the proposed involvement of motor inhibition in the therapeutic effects of SMR training on epilepsy (*see* 6:4), it should be noted that the human mu rhythm has been associated with a number of pathological states by some workers (Beek, 1958; Babb and Chase, 1974), and has been observed in close proximity to epileptic focal lesions

(Ciganek, 1959). If this association with pathology is correct, then clearly the enhancement training of mu activity would be an inappropriate technique for the treatment of epilepsy.

6:6 CONTROL PROCEDURE: HIGH VOLTAGE SUPPRESSION TRAINING

6:6:1 Introduction

The feedback training procedure in the clinical study involved the suppression of high voltage paroxysmal or artifactual activity in addition to the enhancement of 12-16 Hz or 8-10 Hz EEG. A similar dual contingency was involved in all of the studies where therapeutic effects were attributed to SMR training (Stermann, 1973a, 1977a; Stermann et al., 1974; Finley et al., 1975; Seifert and Lubar, 1975; Finley, 1976; Lubar and Bahler, 1976). Hence the suppression training procedures alone might be responsible for the clinical effects. This possibility is further suggested by the finding of Finley et al. (1975) that the addition of feedback of epileptiform (4-7 Hz) activity to the feedback of SMR (12 Hz) appeared to bring about a reduction in the variability of epileptiform and seizure activity. Hence a necessary control procedure is to determine the effects of providing subjects with suppression training of high voltage activity only. The EEG feedback unit was therefore modified so that feedback training to suppress the operation of the inhibit circuit alone could be given. However, since the time remaining for the clinical study was very limited, the high voltage suppression training was only given to subjects N.L. and B.H.; whilst subject P.C. was used for another important control procedure, which will be described in the next section.

6:6:2 Method

For subjects N.L. and B.H. the red feedback lamp was disconnected during the training sessions so that only feedback of high voltage activity (i.e. the green lamp) was available. In addition, the digital display of the EEG feedback unit was modified so that, for these subjects, it no longer indicated the occurrence of a criterion response of 12-16 Hz or

8-10 Hz activity. A free-running clock was incorporated, which was gated by the inhibit circuit, so that in the absence of high voltage activity in the EEG, the digits incremented at a rate of one per second, but when high voltage activity was present the count stopped. In order to minimise error in the score, a clock rate of 100 Hz was used and then divided down to 1 Hz after the clock pulses had been gated by the inhibit circuit.

The subjects were instructed to attempt to turn the green light off and to increase the reward score, as before. They were informed that the score now indicated how well they were able to keep the green light off, rather than the red one on. Because the number of rewards with this procedure was much higher than before, the *Smarties* were discontinued. However, both N.L. and B.H. were now well motivated to perform successfully in the feedback training sessions, and said that the *Smarties* were unimportant.

This training condition began after a two week break for the Easter vacation and was continued for ten weeks. The EEG recording, training protocol, and other general procedures, remained as before.

6:6:3 Results

The weekly seizure incidence for subjects N.L. and B.H. during the high voltage suppression training is indicated in Figure 6.20.

Subject N.L. demonstrated a dramatic, 63% reduction in seizure rate during this period, in contrast to the rise in seizure activity seen in the previous training conditions ($\chi^2 = 60.3$, $p < 0.0001$). Subject B.H. experienced only one double seizure, which represents a further, nonsignificant, reduction in seizure frequency of 28% ($\chi^2 = 0.09$, $p > 0.5$).

It can be seen in Figure 6.21 that subject N.L. exhibited a quite strong tendency to decrease high voltage activity across the sixteen training sessions, but this trend failed to reach statistical significance

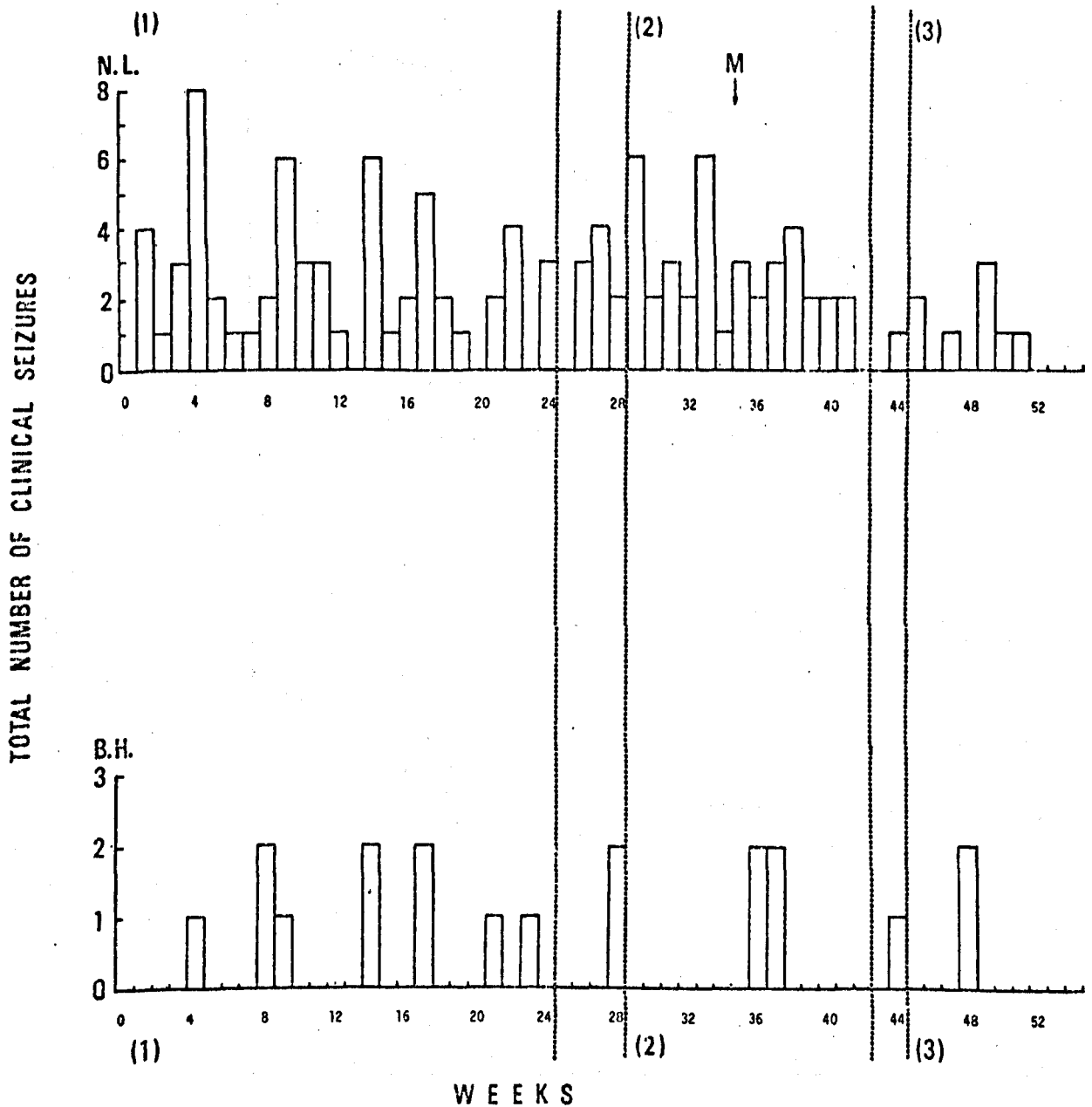


Figure 6.20 Weekly seizure incidence for subjects N.L. and B.H. across (1) the 12-16 Hz, (2) the 8-10 Hz, and (3) the high voltage suppression training conditions. The dashed lines indicate breaks for Christmas and Easter vacations.

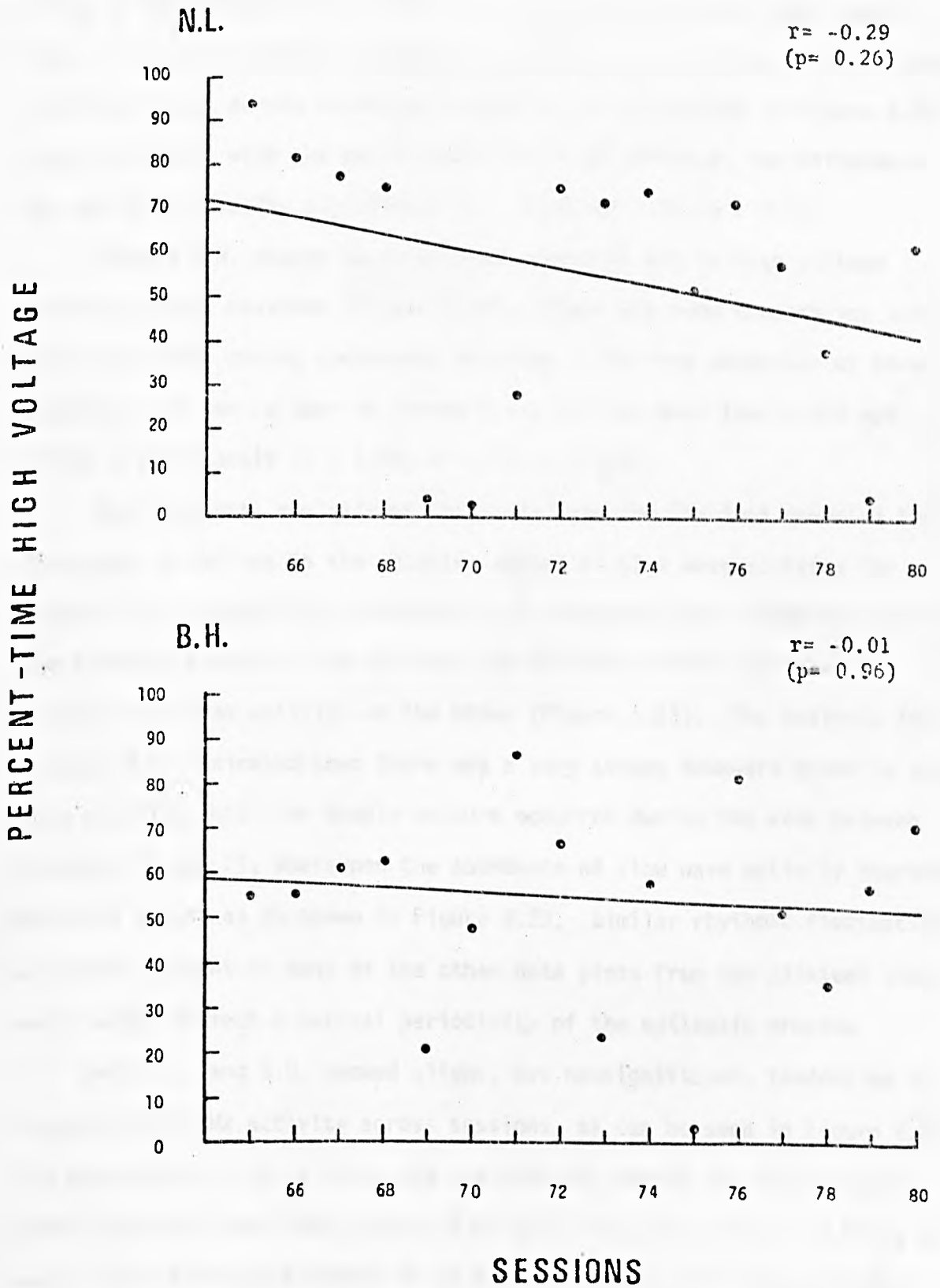


Figure 6.21
Percent-time high voltage activity against training sessions
for subjects N.L. and B.H.

owing to the presence of several very low scores, and the small sample size. N.L. also showed a tendency to suppress high voltage activity below baseline levels during contingent training, as is evident in Figure 6.22. Again, however, with the small sample and high variance, the difference was not statistically significant ($t = 1.68$, $df = 15$, $p > 0.1$).

Subject B.H. showed no systematic change at all in high voltage activity across sessions (Figure 6.21). There was some suppression below baseline levels during contingent training - with the exception of three sessions - as can be seen in Figure 6.22, but the mean levels did not differ significantly ($t = 1.45$, $df = 15$, $p > 0.1$).

Power spectral analysis of the final baseline EEG data revealed that there was no decline in the relative amount of slow wave activity for subject N.L. to parallel the tendency to decrease high voltage activity in the training period on the one hand, or the very marked reduction of clinical seizures activity on the other (Figure 6.23). The analysis for subject B.H. indicated that there was a very strong downward trend in slow wave activity until the double seizure occurred during the week between sessions 70 and 71, whereupon the abundance of slow wave activity increased markedly again, as is shown in Figure 6.23. Similar rhythmic fluctuations are often evident in many of the other data plots from the clinical study, which might reflect a natural periodicity of the epileptic process.

Both N.L. and B.H. showed slight, but nonsignificant, tendencies to increase 12-16 Hz activity across sessions, as can be seen in Figure 6.24. The percentage of 12-16 Hz during the training period was significantly below baseline levels for subject B.H. ($t = 3.13$, $df = 14$, $p < 0.001$), but very little difference was shown by N.L. ($t = 0.37$, $df = 13$, $p > 0.7$). There was a similar slight, nonsignificant increase in 8-10 Hz activity across sessions for both subjects (Figure 6.25), but the mean training level did not differ significantly from baseline for either B.H. ($t = 0.18$,

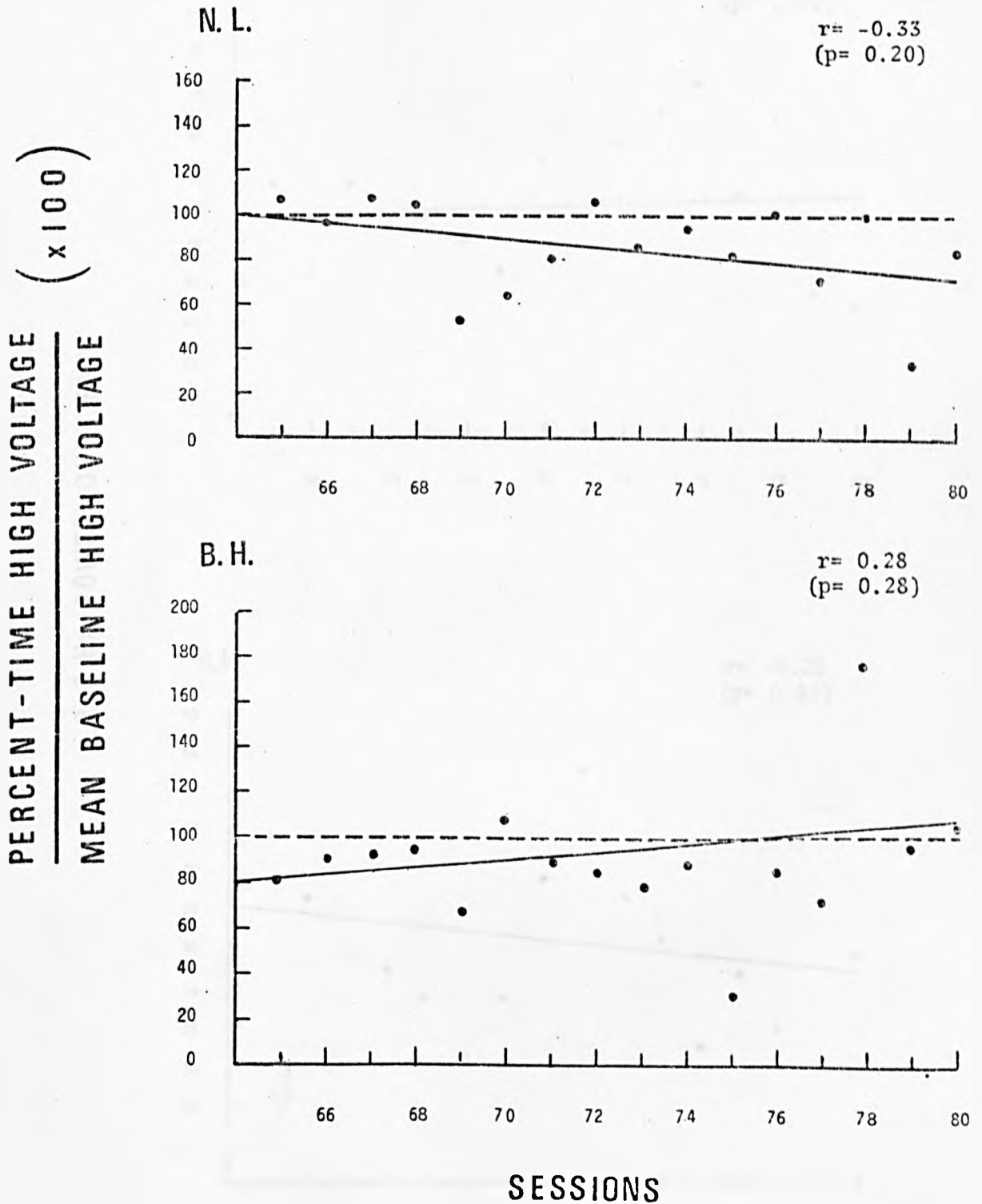


Figure 6.22

Ratio of training to mean baseline levels of high voltage activity (expressed as a percentage) against training sessions for subjects N.L. and B.H.

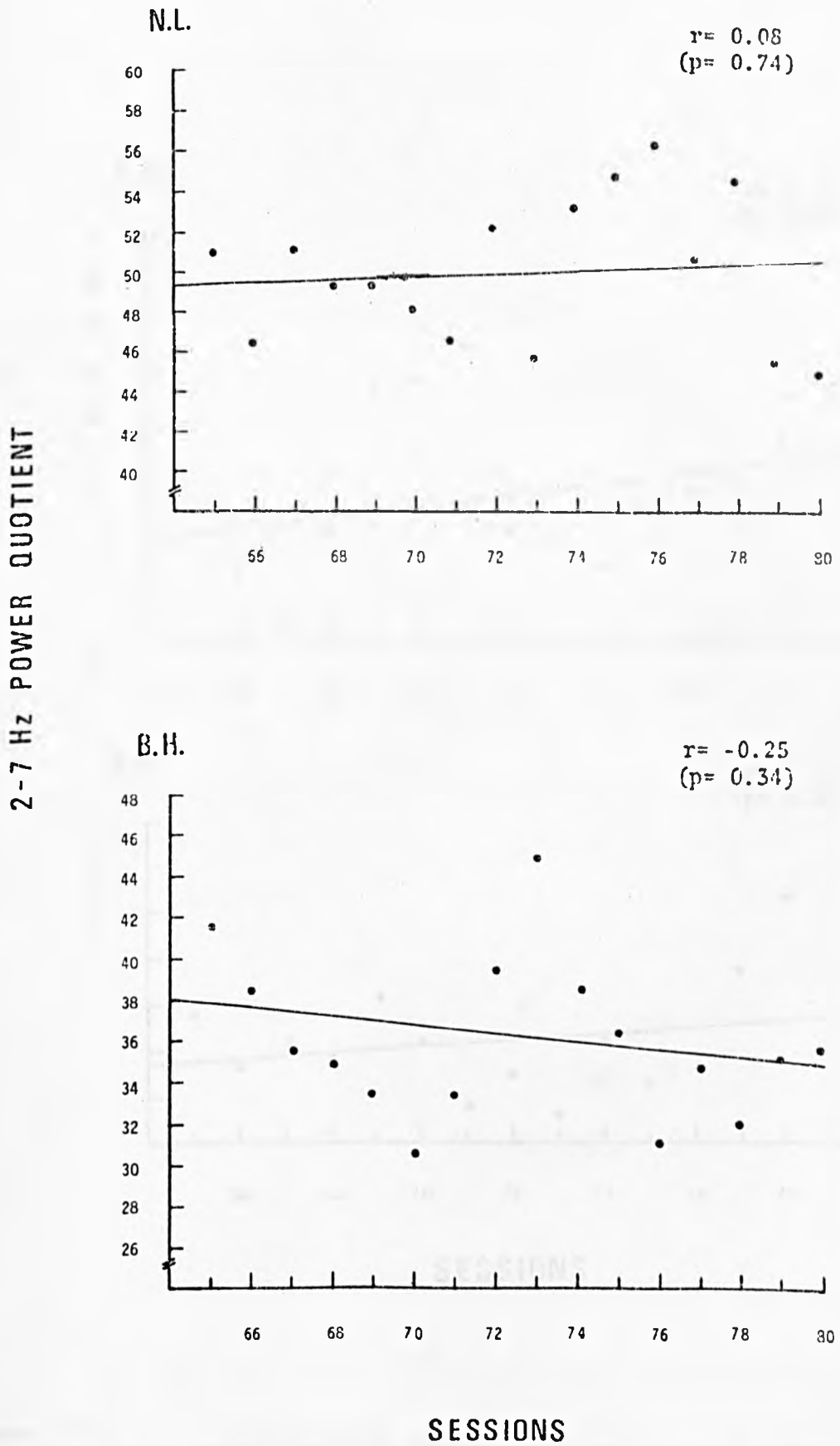


Figure 6.23
Relative power of 2-7 Hz activity against training sessions
for subjects N.L. and B.H.

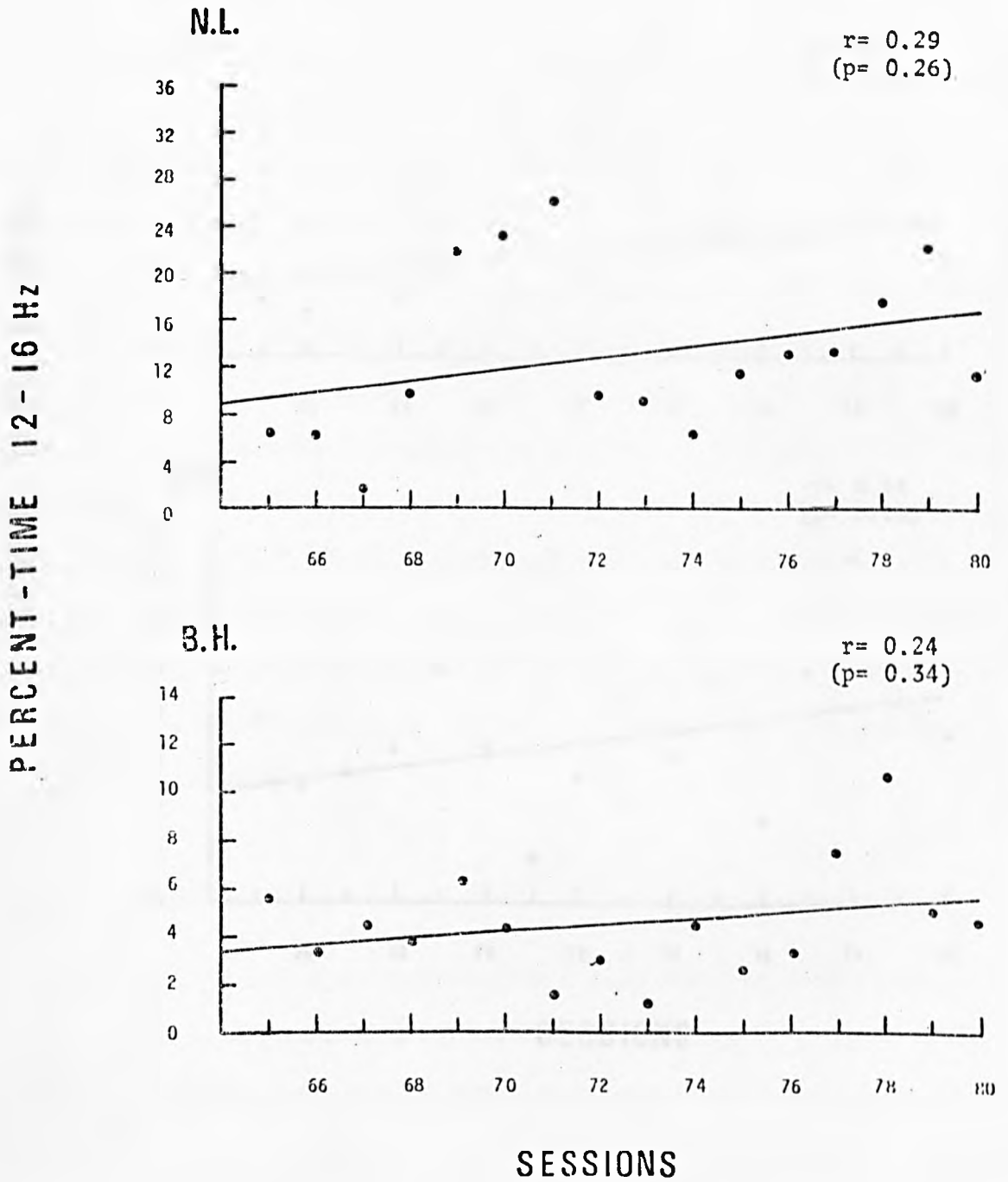


Figure 6.24
Percent-time 12-16 Hz activity against training sessions for subjects N.L. and B.H.

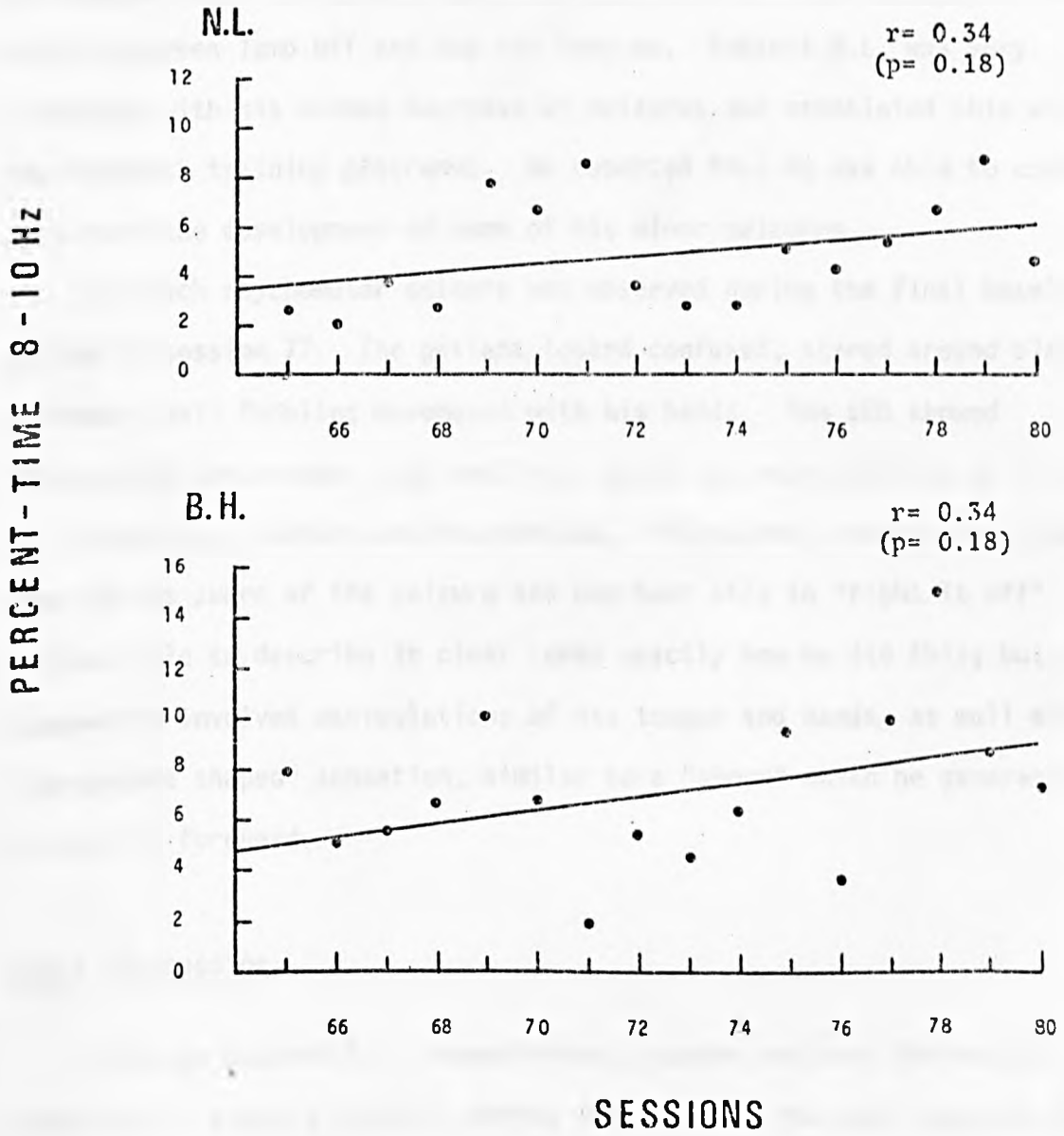


Figure 6.25

Percent-time 8-10 Hz activity against training sessions for subjects N.L. and B.H.

$df = 14, p > 0.8$) or N.L. ($t = 0.32, df = 14, p > 0.7$).

Both subjects N.L. and B.H. stated that they preferred this feedback training procedure to the previous one, since it was much simpler for them to concentrate on keeping the green lamp off, rather than trying to keep both the green lamp off and the red lamp on. Subject N.L. was very impressed with his sudden decrease of seizures, and associated this with the feedback training programme. He reported that he was able to continue to arrest the development of some of his minor seizures.

One such psychomotor seizure was observed during the final baseline period of session 77. The patient looked confused, stared around blankly, and made small fumbling movements with his hands. The EEG showed bilaterally synchronous high amplitude spike and wave activity at 1.5-2.0 Hz. The attack lasted some two minutes. Afterwards, the patient claimed that he was aware of the seizure and had been able to "fight it off". He was not able to describe in clear terms exactly how he did this; but it apparently involved manipulations of his tongue and hands, as well as a "horse-shoe shaped" sensation, similar to a "shock" which he generated across his forehead.

6:6:4 Discussion

Although subject N.L. demonstrated a sudden and very impressive reduction in seizure activity during this period, the data indicate that this was not closely associated with the suppression of high voltage activity in the EEG. Nevertheless, there was some indication of a decline in high voltage activity and, as only a small number of data points were obtained in this final training condition, it would be difficult to obtain a significant correlation.

It is evident from Figure 6.20 that the reversal in clinical trend for this subject was not, in fact, time-locked to this present training

condition, but began toward the end of the 8-10 Hz training period. This improvement in clinical condition might merely represent spontaneous remission. Alternatively, it is possible that it is related to the subject's claim that he was sometimes able to inhibit the intensification of his minor seizures, since it is a well known clinical observation that some epileptics are able to block the onset of seizure activity by the concentration of attention on certain behaviour (see 7:2:1). Thus the concentration involved in the performance of the various manipulations that N.L. described might have had some inhibitory effect. Several subjects in other EEG feedback training studies have similarly reported that they developed the ability to inhibit seizure activity, as a corollary of the feedback training programme (see 8:2).

There was also no association between the small reductions in seizure rate shown by subject B.H. during this training condition and the level of high voltage activity in her EEG. The data therefore indicate that suppression training of high voltage paroxysmal and artifactual activity does not of itself appear to be responsible for the treatment effects.

6:7:1 Introduction

The clinical improvements demonstrated by subject P.C., and later also by B.H., might reflect 'secondary gain' associated with the training programme. Thus the charge most often raised against EEG biofeedback for the treatment of epilepsy is that the clinical results can be attributed to placebo or other non-specific effects. It is a well known fact that some epileptics show clinical improvements associated merely with admission to hospital; hence the extensive close attention to the subjects involved in the research programme might perhaps produce similar effects.

One way to control for this possibility is simply to substitute random feedback for the contingent EEG feedback, whilst maintaining all other factors constant. This control procedure has, in fact, been carried out by several workers. Thus, Finley (1976) recently reports that noncontingent feedback was introduced for a seven week period after thirteen months of SMR feedback training, without the knowledge of the patient, his family, or some of the research staff. It was found that there was no significant change in seizure rate, although the incidence of seizures accompanied by urine loss did increase significantly, which Finley interprets as suggesting that the seizures experienced during the noncontingent period were more severe. In addition, it is reported that there was a significant 4% increase in epileptiform activity (as indicated by the slow wave inhibit circuitry), and a significant 8% decrease in SMR (12 Hz). However, Finley notes, "All three variables seemed to recover to pre-sham levels toward the latter half of the sham period".

Kuhlman (1976; Kuhlman and Alison, 1977) provided his five patients with noncontingent feedback for the first twelve training sessions over 4-5 weeks. No sustained increase in seizure rate was observed, in contrast to

the average decline in seizure rate of 70% that was observed when contingent feedback of 9-14 Hz central EEG was given. However, in a second period of one month during which two patients received noncontingent feedback the seizure rate showed very little change. Nevertheless, Kuhlman (1976) concludes that, "The clinical effects were specific to the feedback training and were not due to placebo effects, attention, or habituation to to the feedback setting".

A successful reduction in seizure rate was also obtained for four epileptics by Wyler et al. (1976) following feedback training of EEG activity in the 14-30 Hz frequency range. However, the seizure rate did not change significantly when two patients were given noncontingent feedback. Thus these workers concluded that, "Involving a patient in the operant protocol *per se* was not an important variable in the modification of seizures". Curiously, in the abstract of this report they state that, "Pseudo-conditioning and control periods ruled out placebo effects".

The effects of the substitution of random for contingent EEG feedback were therefore investigated in this study. Subject P.C. was used for this control procedure since, at this stage, he had experienced the greatest reduction in seizure frequency.

6:7:2 Method

It was found that a very effective way of providing random feedback was to feed white noise into the headbox of the electroencephalogram instead of the patient's EEG. This is a much more convenient method than the replaying of tape-recordings of previous sessions as other workers have done (Finley, 1976; Kuhlman, 1976), since it does not require two tape decks; and it has the further advantage that it enables truly random, rather than just delayed, feedback to be presented.

A small, battery-operated white noise generator was constructed. The

base-emitter junction of a silicon transistor (2N3702) was used as the noise source, followed by a variable gain amplifier to produce an output level of 0-10 mV (RMS). By suitable adjustment of the pre-amplifier response, and the amplitude thresholds of the EEG feedback unit, it was possible to perfectly simulate contingent EEG feedback as illustrated in Figure 6.26.

Random feedback was presented to subject P.C. for ten weeks after a two week break for the Easter vacation. The Director of the Centre and myself were the only persons aware of the change in procedure. Rolandic EEG was recorded on another channel during each session and stored on magnetic tape. It was then later replayed into the EEG feedback unit in order to determine the subject's true scores.

6:7:3 Results

The incidence of seizures for subject P.C. during the random feedback

White noise (Low-pass filtered at 50 Hz)

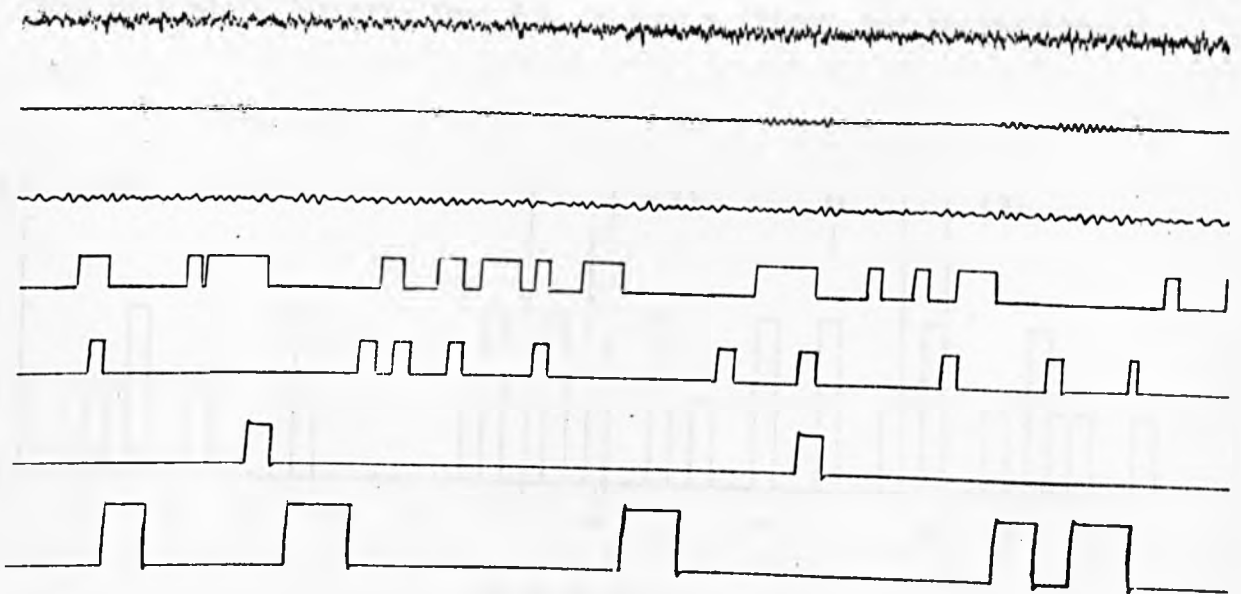


Figure 6.26

Illustration of random feedback generated by feeding white noise into the EEG headbox. The various filter and detector outputs are as indicated in Figure 6.2.

period is indicated in Figure 6.27. The mean seizure rate was 20% greater than that for the 8-10 Hz training condition, but this increase was not statistically significant ($\chi^2 = 0.46$, $p > 0.5$). During this period he also suddenly began to show a very pronounced increase (130%) in the number of myoclonic episodes that necessitated the intravenous administration of valium. At first it was thought that this might be a consequence of the random feedback procedure; but one of the nursing staff suspected that these episodes were hysterical in nature, and P.C. then admitted that he was "putting them on" because he "liked the injections". When further questioned about this matter, it soon became clear that it was not the actual injections that he enjoyed, but the fact that it was customary for him to be excused from work for the rest of the day after the injection! This incident illustrates some of the problems in obtaining reliable objective data that can be met in an assessment of antiepileptic treatments, where varagies of drug compliance, hysterical attacks, or unexpected behaviour patterns associated with secondary motivation can easily confound the clinical picture.

The data plots indicate that P.C. showed a slight, but nonsignificant,

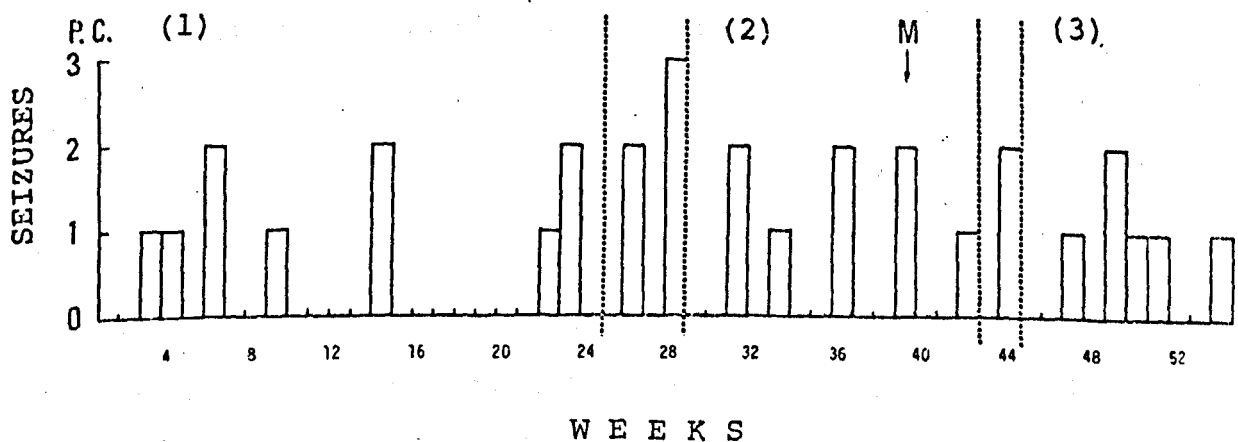


Figure 6.27

Weekly seizure incidence for subject P.C. across (1) the 12-16 Hz, (2) the 8-10 Hz and (3) the random feedback training conditions.

decrease in high voltage activity across the fourteen random feedback sessions (Figure 6.28a), together with a slight, but nonsignificant, increase in the 12-16 Hz band (Figure 6.28b), and the 8-10 Hz band (Figure 6.28c). The mean percentage of high voltage activity was significantly greater during the baseline periods ($t = 4.71$, $df = 15$, $p < 0.001$). Conversely, the percentage of 12-16 Hz was significantly reduced below mean baseline levels during the random feedback period ($t = 5.09$, $df = 15$, $p < 0.001$), and similarly the percentage of 8-10 Hz ($t = 4.85$, $df = 14$, $p < 0.001$). Generally, the abundance of high voltage activity in the EEG was greater than in either of the previous training conditions, and the abundance of 12-16 Hz and 8-10 Hz activity less - which would logically follow from the operation of the inhibit circuit. The high voltage activity mainly consisted of paroxysmal delta-theta activity and epileptic spikes, as before; but, in addition, the absence of contingent feedback resulted in an increase in the presence of artifact from head movement or jaw clenching. Power spectral analysis indicated that there was no progressive change in the relative amount of slow wave activity in the final baseline EEG across sessions (Figure 6.28d).

Subject P.C. gave no indication that he was aware that the feedback signals were noncontingent during this part of the training programme, and happily continued to attempt to control the randomly flashing lamps as before.

6:7:4 Discussion

The substitution of random for contingent feedback for subject P.C. did not result in any significant aggravation of seizure rate. The small increase that did occur probably represents a continued upward shift toward a more stable level following the marked decrease seen in the first four months of the study. The data do suggest, however, that the noncontingent

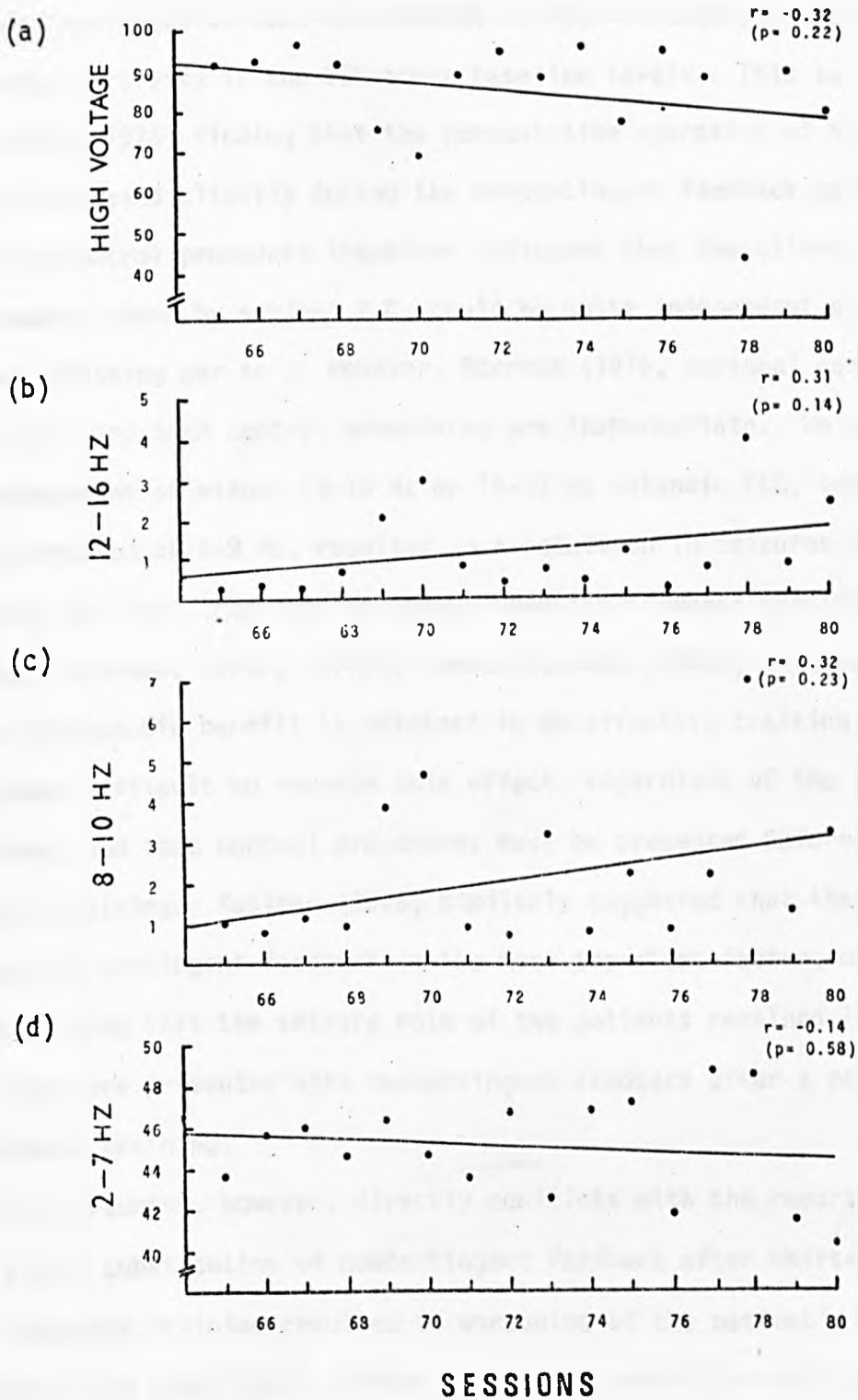


Figure 6.28

- (a) Percent-time high voltage activity across training sessions for subject P.C. in the random feedback condition.
- (b) Percent-time 12-16 Hz against training sessions for P.C.
- (c) Percent-time 8-10 Hz against training sessions for P.C.
- (d) Relative power of 2-7 Hz against training sessions for P.C.

feedback itself led to some enhancement of high voltage paroxysmal and artifactual activity in the EEG above baseline levels. This is similar to Finley's (1976) finding that the percent-time operation of his inhibit circuit increased slightly during the noncontingent feedback period.

This control procedure therefore indicates that the clinical improvements shown by subject P.C. could be quite independent of any feedback training per se. However, Sterman (1976, personal communication) argues that any such control procedures are inappropriate. He found that the enhancement of either 12-15 Hz or 18-23 Hz rolandic EEG, together with the suppression of 6-9 Hz, resulted in a reduction in seizures for two patients, but that there was no change when the feedback contingencies were reversed (Sterman, 1976c; 1977a). Hence Sterman (1976c) maintains that once a therapeutic benefit is obtained in an effective training condition it becomes difficult to reverse this effect regardless of the training procedure, and thus control procedures must be presented before contingent feedback training. Kuhlman (1976) similarly suggested that the initial exposure to contingent feedback is the most important factor, on the basis of his finding that the seizure rate of two patients remained low and stable when they were presented with noncontingent feedback after a period of 9-14 Hz feedback training.

This argument, however, directly conflicts with the report of Finley (1976) that substitution of noncontingent feedback after thirteen months of 12 Hz feedback training resulted in worsening of the patient's clinical condition and significant changes in the EEG, which he concludes "seem to argue strongly against any placebo effect".

Placebo effects are said to typically occur when treatments have been altered or when new treatments are introduced (Shapiro, 1960). Thus the initial marked reduction in seizure rate followed by a slow increase for subject P.C., together with the salient changes in his psychological

condition, strongly implicate the involvement of a placebo factor.

However, this is much less likely in the cases of subjects B.H. and N.L., where clinical improvements were not observed until more than six months after the beginning of the treatment. The motivation and expectation of the patients were probably highest at the beginning of training, and it is reasonable to assume that a placebo effect would be maximal at that time. Hence the latency of the clinical changes for these subjects suggests that placebo effects were not the primary cause.

A number of workers have also argued that placebo effects tend to be transitory and that their importance is thus likely to be diminished by the length of the feedback training studies (Stermann, 1973a; 1974; Seifert and Lubar, 1975; Lubar and Bahler, 1976). Some of Sterman's patients have been investigated for more than four years (Stermann, 1976b), and, similarly, some of Lubar's patients received feedback training for up to three years (Lubar and Bahler, 1976). Hence, as Sterman (1973a) concludes, "One might expect that secondary gain related to attention and novelty would be dissipated after so long a period of involvement". Moreover, a number of follow-up studies have demonstrated that the therapeutic effects can persist for more than a year after the patients have been completely withdrawn from training (*see* 8:3). Therefore, if a placebo effect is responsible for the successful treatment of epilepsy by EEG feedback, then, as Miller (1974) has pointed out, it is a powerful phenomenon which is significant in its own right, and one which merits much more investigation than it has received.

A clinical study was set up to investigate the reported relationship between the enhancement of SMR and therapeutic effects on epilepsy. Three patients at the David Lewis Centre for Epilepsy were selected for this study. Two patients: P.C. (male, 23 years of age) and N.L. (male, 39 years of age) had the diagnosis of partial epilepsy with secondary generalisation; whilst a third patient, B.H. (female, 49 years of age), had the diagnosis of primary generalised epilepsy. All three patients suffered from frequent clinical seizures that had proved resistant to conventional chemotherapy; N.L. had also undergone an unsuccessful right temporal lobectomy.

The subjects were provided with feedback training to enhance activity in the 12-16 Hz frequency band, and to suppress high voltage activity in the rolandic EEG. Training sessions were run twice weekly for six months. Each session consisted of thirty minutes of feedback training, together with a three minute pre- and post-feedback baseline period. Occipital EEG, eye movements, frontalis EMG, ECG and respiration were also recorded for each session. Clinical seizures were logged by the staff of the Centre, and anticonvulsant serum levels routinely monitored. The *Wittenborn Psychiatric Rating Scale* was regularly completed for the patients by the Director of the Centre.

A significant reduction in mean seizure rate, compared to the six months immediately preceding the study, was obtained for subject P.C., but not for the other two subjects. However, whereas both subjects N.L. and B.H. experienced a very significant progressive increase in rolandic 12-16 Hz activity across training sessions, subject P.C. did not. In addition, there was no evidence that the abundance of 12-16 Hz was related to behavioural quiescence. Hence, the data provide no support for any association between the enhancement of an SMR and the treatment effects; or for the hypothetical involvement of a motor inhibitory mechanism.

Rolandic activity in the 8-10 Hz mu range (Gastaut, 1952) was also monitored. It was found that all three subjects exhibited a very significant increase across sessions. Hence the feedback contingency was switched to 8-10 Hz for the next fourteen weeks as a control procedure. A significant reduction in mean seizure rate was shown by subject B.H. She also demonstrated a significant progressive increase in 8-10 Hz activity across sessions. There were no significant changes in the seizure rate or the EEG frequency bands for the other two subjects.

Random feedback from a white noise source was presented to subject P.C., without his knowledge, for a further control period of ten weeks. This did not lead to any significant change in seizure rate, but there was some indication of an enhancement of high voltage activity in the EEG. Subjects N.L. and B.H. were provided with training to suppress high voltage activity only during this period, as an additional control procedure. A very significant reduction in seizure rate was obtained for subject N.L. There were no significant changes in the EEG data, however, for any of the subjects.

7:1 OVERALL CLINICAL AND ELECTROENCEPHALOGRAPHIC EFFECTS

7:1:1 Clinical Changes

An apercu of the clinical data and the results of the three feedback training procedures is given in Table 7.1. As can be seen, all three patients showed a significant decrease in seizure rate by the end of the study compared with the six month period preceding it. However, the data clearly demonstrates that the therapeutic effects are not related to any one particular training condition. Thus, while 12-16 Hz feedback training may be sufficient for seizure reduction in some patients, it is not necessary in others.

It is unlikely that the clinical improvements were due to changes in drug compliance or absorption, since there were no consistent variations in the level of anticonvulsant serum in the blood plasma other than a slight decrease in phenobarbitone for subject N.L. (Figure 6.14). It is also unlikely that, except perhaps in the case of the marked initial reduction in seizure manifestation shown by subject P.C., the clinical benefit is simply due to a placebo factor, because of the latency of the effects. Finally, although it is possible that some spontaneous remission might have occurred, it is most improbable that the results for all three subjects can be attributed to this factor.

7:1:2 Changes in EEG Spectra

In order to determine whether there were any overall changes in the EEG that might account for the clinical effects, a regression analysis of

Table 7.1. Summary of data from the clinical study.

	SUBJECT P.C.	SUBJECT N.L.	SUBJECT B.H.
Age and Sex	23 years, male	39 years, male	49 years, female
Diagnostic Classification	Partial epilepsy with multiple foci, secondary generalization	Partial epilepsy with multiple foci, secondary generalization	Primary generalized epilepsy
Seizure Pattern	Tonic-clonic seizures and myoclonus	Psychomotor and tonic-clonic seizures	Tonic-clonic seizures
EEG	Dominant 3-7 Hz. Frequent random spikes	Dominant 2-7 Hz. Some spike-wave episodes at 2.0-2.5 Hz	Low-voltage fast activity. Isolated random spikes
Pathological History	21 years	31 years	33 years
Medication (daily)	Epilim : 3000 mg Tegretol : 800 mg (since 17/3/75)	Mysoline : 750 mg Phenobarbital : 90 mg Epanutin : 400 mg (since 28/1/75)	Epanutin : 250 mg (since 12/5/75)

SEIZURE FREQUENCY

	Average/ week	Change from Pre-test	Relative change	Average/ week	Change from Pre-test	Relative change	Average/ week	Change from Pre-test	Relative change
Six-months Pre-test	0.88		-	2.40		-	0.44		-
Condition 1	0.42	-52.3%	-50.0%	2.50	+ 4.2%	+ 4.2%	0.42	- 4.5%	- 4.5%
Condition 2	0.50	-43.2%	+19.0%	2.71	+12.9%	+ 8.4%	0.28	-36.4%	-33.3%
Condition 3	0.60	-31.8%	+20.0%	1.00	-58.3%	-63.0%	0.20	-54.5%	-28.6%

CORRELATION COEFFICIENTS FROM REGRESSION ON SESSIONS OF TRAINING

	12-16 Hz	8-10 Hz	Inhibit	12-16 Hz	8-10 Hz	Inhibit	12-16 Hz	8-10 Hz	Inhibit
Condition 1	-0.07	0.37*	-0.20	0.46*	0.56*	-0.05	0.53*	0.40*	-0.18
Condition 2	0.12	0.14	0.07	0.11	-0.07	-0.13	0.19	0.43*	-0.24
Condition 3	0.37	0.32	-0.32	0.29	0.35	-0.29	0.24	0.34	-0.01

(* significant at 5% level or less)

Condition 1 = Feedback training of 12-16 Hz rolandic EEG (24 weeks)
Condition 2 = Feedback training of 8-10 Hz rolandic EEG (14 weeks)
Condition 3 = Inhibit (high voltage) suppression training for N.L. and B.H.;
random feedback for P.C. (10 weeks)

the EEG power spectra obtained from all of the sessions in the clinical study was carried out. The results are summarised in Table 7.2

Table 7.2. Product-moment coefficients and significance levels for regression of 2-7 Hz power quotient and EEG mean frequency on training sessions. (With missing data $df = 65$.)

PARAMETER	SUBJECT		
	<u>P.C.</u>	<u>N.L.</u>	<u>B.H.</u>
Slow Wave Activity (2-7 Hz)	$r = -0.33$ ($p = 0.006$)	$r = 0.10$ ($p = 0.410$)	$r = 0.25$ ($p = 0.038$)
EEG Mean Frequency	$r = 0.25$ ($p = 0.038$)	$r = 0.06$ ($p = 0.640$)	$r = -0.22$ ($p = 0.069$)

It can be seen that subject P.C. exhibited a small, but significant, negative trend in the relative amount of slow wave activity in the EEG across training sessions, combined with a significant increase in the mean frequency of the EEG. This suggests that there was some tendency toward a reduction of pathological delta-theta activity over the period of the clinical study.

Conversely, subject B.H. showed a small, but significant, progressive increase in slow wave activity across sessions, and a tendency for a negative relationship between mean EEG frequency and training sessions that just failed to reach statistical significance. The feedback training procedures thus appear in her case to have led to a tendency for a downward shift in the frequency spectrum of the EEG. It should be remembered that this subject did not present a dominant slow wave peak in her EEG, as did the other two subjects (Figure 6.9).

There was no significant relationship between the percentage of slow wave activity, or EEG mean frequency, and training sessions for subject N.L.

This probably results from the fact that, although by the end of the study he had achieved the greatest reduction in seizure rate, he experienced a slight increase for the first eight months or so.

Since the power spectral analyses were performed on the EEG data recorded during the final baseline periods, the trends for subjects P.C. and B.H. provide evidence that there was a carry-over effect from the feedback training period. However, no data on the extinction characteristics of this effect is available from the clinical study.

A number of routine clinical EEG recordings were carried out on these three patients during the course of the clinical study by the staff of the David Lewis Centre. These were not used as a primary data source since a professional electroencephalographer was not available to provide an impartial interpretation. Moreover, it was evident during the twice weekly feedback sessions over the twelve-month period of the study, that all three patients could exhibit considerable variability in the morphology of their EEG from day-to-day (as is reflected by the scatter of the data plots). Thus, whether the EEG appears as grossly, or only mildly, pathological on any one particular clinical recording must very largely be a matter of chance.

In order to test the hypothesis that feedback training might lead to unilateral changes in the EEG, such as Sterman (1973b; 1974; 1976c; Sterman et al., 1974) has claimed, power spectra were computed for the final baseline EEG that was recorded from the left rolandic region. Regression lines were then fitted to the 2-7 Hz power quotient and EEG mean frequency scores for each subject, as before. A comparison of the regression lines for the data from the right and left hemispheres was carried out in order to determine whether they differed in slope. There was very little difference for subject P.C. for either slow wave activity

($t = 0.004$, $df = 97$, $p > 0.9$), or for EEG mean frequency ($t = 0.008$, $df = 97$, $p > 0.9$). Similarly, the regression lines for both slow wave activity ($t = 0.45$, $df = 97$, $p > 0.5$) and EEG mean frequency ($t = 0.004$, $df = 97$, $p > 0.9$) were almost parallel for subject B.H. The corresponding correlations for subject N.L. were both nonsignificant for the left hemisphere, as with the right, and so the regression lines were not compared. Sterman's reports of unilateral changes in the EEG, favouring the side that was used for feedback training, were therefore not replicated by this study in which the EEG data were actually quantified.

The power spectra were further examined to determine whether any enhancement of the 12-16 Hz or the 8-10 Hz frequency bands in the EEG occurred over the course of the clinical study. Since the normalization procedure necessarily results in a strong inverse correlation between the power quotient of these frequency bands and that of slow wave activity, partial correlation coefficients were computed holding the effects of slow wave activity constant. The results are listed in Table 7.3. It can be seen that there were no significant trends.

Table 7.3. Coefficients and significance levels for correlation between 12-16 Hz power quotient, or 8-10 Hz power quotient and sessions of training, with the effects of 2-7 Hz power quotient partialled out ($df = 64$).

PARAMETER	SUBJECT		
	<u>P.C.</u>	<u>N.L.</u>	<u>B.H.</u>
12-16 Hz	$r = 0.04$ ($p = 0.7$)	$r = 0.04$ ($p = 0.7$)	$r = 0.18$ ($p = 0.1$)
8-10 Hz	$r = 0.10$ ($p = 0.4$)	$r = 0.17$ ($p = 0.2$)	$r = 0.18$ ($p = 0.1$)

A progressive reduction in slow wave activity similar to that observed for subject P.C., has been reported in many other studies (Sterman et al.,

1974; Finley et al., 1975; Seifert and Lubar, 1976; Sterman, 1976b; 1976c; Lubar and Bahler, 1976), although this claim has not usually been substantiated by any quantitative analysis. Kuhlman (1976) also reports that two subjects exhibited a shift in dominant frequency of the EEG spectra from lower to higher bands. Generally, it is proposed that such changes represent "normalization" of the EEG (Sterman et al., 1974; Finley et al., 1975; Kuhlman and Alison, 1977; Lubar and Bahler, 1976; Wyler et al., 1976), since slow wave activity is closely associated with the combination of the epileptic process and anticonvulsant medication. Sterman (1976c), for example, found that a group of thirteen epileptic patients exhibited significantly more 4-7 Hz activity in the spectra of their sleep EEG than thirteen normal subjects; although Sterman notes that individual differences were very apparent. Hutt (1966) similarly reports that background EEG activity was significantly slower for a group of epileptic children than for normal children.

7:2 MECHANISMS FOR THE EFFECTS OF EEG FEEDBACK TRAINING ON EPILEPSY

7:2:1 Arousal Hypotheses

It has been suggested that the effects of SMR feedback training might result from a generalised enhancement of EEG desynchronization, so as to bring about an attenuation of pathological slow wave activity (Wyler et al., 1976; Kuhlman, 1976). Thus, Wyler et al. (1976) argue: "One would predict that reinforcement of any rhythm conducive to alerting would benefit the patient". Sterman (1976c) similarly recently conceded that, "The mere stabilization of the EEG and enhancement of higher frequencies, in general, could account also for the results obtained in both cats and humans".

As an investigation of the desynchronization hypothesis, Wyler et al. (1975b; 1976) provided five epileptics with training to enhance low voltage fast activity (14-30 Hz), and simultaneously suppress slow activity (below 10 Hz) in the EEG recorded from the vicinity of the epileptic focus. After some three months of training, four of the patients presented a clinical improvement. There was also some indication that they were able to selectively increase the proportion of high frequencies in the EEG, although the parameters on which the quantification of the data were based were not kept constant.

Upton et al. (1975) similarly carried out a study with thirteen epileptics in which feedback training to desynchronize focal spike discharges was given, using various auditory and somatosensory stimuli. The number of feedback sessions per patient varied from one to eleven. A marked reduction of epileptiform discharges was obtained in eight patients - including a reduction of greater than 50% in four, although there was considerable variability between sessions. Six of the subjects showed a reduction in clinical seizures.

A number of earlier workers employed a variety of activating stimuli to desynchronize epileptiform activity, with some successful results (Efron, 1956; 1957; Stevens, 1961; Guerrero-Figueroa et al., 1963; Ounsted et al., 1966; Stevens et al., 1967; Tassinari, 1968; Korein et al., 1971). Direct brain stimulation has also been used to produce EEG desynchronization in an attempt to suppress epileptic activity (Cooper et al., 1974). In addition, some epileptics are themselves able to inhibit seizure activity by forced mental or physical activity (Symonds, 1961; Paulson, 1963). Thus in a survey of some nine hundred epileptics, Servit et al. (1962) found that 31.2% reported factors which could sometimes arrest the development of seizures: the most frequently noted being concentration of attention on certain mental or physical events.

Thus it has been well documented that arousal and conditioned desynchronization is beneficial for some epileptic patients. On the other hand, relaxation techniques have also been successfully employed to treat other epileptics (Lennox, 1960; Cautela and Flannery, 1973). Cabral and Scott (1974; 1976) provided three patients, whose seizures were closely associated with anxiety and phobic symptoms, with a combination of alpha feedback and conventional relaxation therapy so as to reduce the response to anxiety evoking stimuli. A clinical improvement was obtained during both procedures and, after six months, all three patients demonstrated a very marked seizure reduction - with one becoming seizure-free. Kaplan (1975) similarly reports that her two subjects who showed a clinical improvement following feedback training of rolandic 6-12 Hz EEG both experienced more seizures during stress; and that they both spontaneously reported that the feedback situation helped them to relax. Hence she argues that the reason for the improved clinical condition of the patients is that they learned to function at a lower level of arousal, as a consequence of the biofeedback setting which enabled them to learn new techniques of relaxation.

The desynchronization and relaxation hypothesis for the effects of EEG feedback training on epilepsy at first sight appear to conflict (Wyler et al., 1966). However, as we will see here, they can both be subsumed under a more general conception.

For many epileptic patients there is an increased likelihood of seizure activity during fatigue, relaxation, drowsiness or sleep (Weinberg, 1945; Gibbs and Gibbs, 1946; Livingstone, 1956; Servit et al., 1962; Pompeiano, 1969). Conversely, excitement and stress are also well known contributing, and sometimes precipitating, events in the occurrence of epileptic seizures (Allen, 1956; Stevens, 1959; Servit et al., 1962; Small et al., 1964; Feldman and Paul, 1976). The precipitation of seizures by startle, photostimulation, or specific visual or auditory patterned stimuli has similarly been well documented in the literature (Daube, 1966; Gastaut and Tassinari, 1966; Bickford and Klass, 1969).

Hutt (1972), from an analysis of clinical data, found that the most frequently reported conditions associated with epileptic seizures (apart from those seizures related to particular reflex mechanisms) were states of tiredness and drowsiness on the one hand, and states of intense emotion, anger, frustration, anxiety or fear, on the other. Hutt (1972) therefore proposed that seizures are more likely to occur at low and high levels of arousal, and he obtained some experimental evidence to demonstrate that the tendency toward hypersynchronous epileptiform discharge in the EEG is least at intermediate levels of arousal. Atzev (1962) also obtained similar clinical and experimental evidence that the effect of non-specific afferent stimuli upon epileptic activity is related to the tonic level of arousal in the CNS.

Some recent neurophysiological data supports the concept of a bi-modal relationship between epileptic manifestation and the degree of electrocortical arousal. Wyler and associates experimentally induced

epileptogenic foci in the motor cortex of the monkey by subpial infusion of aluminium hydroxide gel. Microelectrode recording from single neurons in the foci revealed that pathological cells were characterised by a proclivity to fire in high frequency bursts (Wyler et al., 1973; Wyler, 1974; Wyler et al., 1975a). This pathological discharge pattern has also been observed for neurons in human epileptic foci (Calvin et al., 1973). Wyler and Fetz (1974) arbitrarily classified the pathological neurons into two groups on the basis of the variability of the percentage of discharges occurring in bursts ("burst index"). *Group I* epileptic neurons were those that exhibited almost continuous structured and stereotyped high frequency discharges, thus indicating extreme pathology. *Group II* neurons, on the other hand, could sometimes appear to be normal and at other times show similar epileptic activity to the *Group I* neurons. It is suggested that the degree of pathology is perhaps associated with gross dendritic deafferentation (Wyler et al., 1974), although, since the membrane properties of these cells appear to be normal between focal epileptic discharges, it has also been suggested that abnormal synaptic input might be responsible for their hyperexcitability (Ayala et al., 1970; Prince and Futamachi, 1970). The *Group II* neurons, because of their inherent lability of burst discharge, are seen as representing a potential "critical mass" for the rapid enlargement of an epileptogenic focus and the subsequent propagation of the pathological cellular activity, so as to bring about a clinical seizure (Wyler et al., 1974b; 1975a).

It was further found that the *Group II* neurons exhibited periodic waxing and waning of burst episodes (Wyler et al., 1973; Wyler, 1974). Moreover, the burst index of these cells is closely related to the general level of activation in the CNS, such that the tendency for pathological discharge during EEG synchronization is much greater than during desynchronization (Wyler, 1974; Wyler and Fetz, 1974). Thus Wyler (1974)

reports that alerting during drowsiness or sleep results in a dramatic and immediate reduction of the burst index for all *Group II* neurons. Similarly, Wagner et al. (1975) found a strong correlation between EEG synchronization and focal epileptiform discharges, and that localised or generalised EEG desynchronization would markedly attenuate the pathological activity. It is possible that this restriction of firing patterns into synchronized pathological bursting could result from the release of recurrent inhibitory loops in the thalamus and cortex following the reduction of the level of excitatory drive (*see* 2:2:3).

Conversely, it was also found that the orthodromical or antidromical activation of the epileptic foci, by stimulation of the medial thalamus or the pyramidal tract, evoked pathological burst discharges from the epileptic neurons, and that normal cells surrounding the epileptic focus were sometimes recruited to fire in synchrony with the pathological discharge (Wyler et al., 1974). Furthermore, the probability of evoking a burst discharge from a *Group II* neuron was observed to be directly proportional to the burst index of the cell at the time of stimulation. Thus the greater the burst index, the more consistently was a burst discharge elicited by antidromic or orthodromic stimulation (Wyler et al., 1975a). Hence, the tendency for increased pathological discharge would be greater at high as well as at low levels of cortical activation.

It is conceivable, therefore, that a synchronous synaptic barrage associated with a strong stimulus might lead to the amplification and propagation of epileptogenic bursting during conditions of activation, in contrast to the ameliorative affect that an alerting stimulus has during EEG synchrony. Thus, as Penfield and Jasper (1954) point out, the effect of stimuli on epileptic discharges can be facilitative as well as inhibitory since there is a delicate balance between the desynchronizing action of afferent impulses and their excitatory effects. Similarly, during high

tonic levels of activation, such as in conditions of emotional excitement or stress, pathological cellular discharges could become aggravated by nonspecific afferent stimuli. It is also plausible that there might be a progressive increase of epileptogenic activity, perhaps assisted by metabolic and biochemical pathology (Ward et al., 1966), so that stimuli which are otherwise without effect can lead to the precipitation of a clinical seizure.

7:2:2 Interpretation of Clinical Study

In view of the above discussion, it is suggested that the therapeutic effects of EEG feedback training might therefore reflect a decline in epileptogenic states of activation. Some support for this hypothesis is provided by the indication of a decrease in hypersynchronous slow wave activity for P.C. in this study, and similar findings reported by many other workers. Thus the EEG desynchronization model would, at the neurophysiological level, represent a decreased tendency for weakly epileptic neurons to be recruited into epileptic foci. In addition, the improvement in the psychological condition of subject P.C. might also have had therapeutic action, since it was noticeable that he would often experience myoclonic episodes when he became emotionally excited or distressed.

Similarly, the shift toward slower frequencies in the highly activated EEG of subject B.H. could reflect a move toward a less epileptogenic state. It is interesting that a progressive *decrease* in slow wave activity up to the occurrence of her double seizure was observed during the final training condition, which suggests that there was a build up in CNS excitation. This patient also reported that she felt more relaxed since she had been coming to the feedback training sessions, and was "sleeping better". Thus relaxation therapy, as an adjunct of EEG feedback training, might be

responsible for the clinical effects in patients of her type with high arousal tonus.

The data does not support any general modulation of the level of activation for subject N.L., although this might have become apparent if a sufficient number of measures had been available from the time that he began to experience clinical benefit. However, if his claim to be able to block some of his minor seizures is genuine, then this would suggest that his reduction in recorded seizure rate was associated with a specific desynchronization of the EEG, and hence the inhibition of neuronal epileptiform discharges. EEG desynchronization can be engendered by many stimuli via the reticular activating system (Moruzzi and Magoun, 1949; Tissot and Monnier, 1959) and this system itself can be strongly influenced by corticofugal projections (French, 1959; Schlag, 1974).

There is also some evidence of active mechanisms for the desynchronization and inhibition of epileptic activity (Gastaut and Fischer-Williams, 1959; Kreindler, 1962). In addition, various brain structures have been demonstrated to have anticonvulsant effects when stimulated (Wagner et al., 1975). This inhibitory action might be associated with the synaptic modulation and gating of weakly pathological neurons so as to prevent their recruitment into epileptic foci. Thus Wyler and colleagues (Wyler and Fetz, 1974; Wyler et al., 1974b) found that the discharge patterns of *Group II* epileptic neurons in the monkey sensorimotor cortex revert to normal when they participate in genesis of motor activity, or are brought under direct operant control. It is possible that these inhibitory mechanisms are facilitated by the EEG feedback training procedures.

SUMMARY

By the end of the clinical study, all three subjects demonstrated a significant reduction of seizure frequency. However, the data demonstrate that the therapeutic effects are not specifically dependent on any one particular training procedure. Changes in drug compliance are not involved; and the latency of the effects for subjects B.H. and N.L. argue against a placebo factor being solely responsible.

Power spectral analysis of the baseline EEG revealed that there was a significant decline in abnormal slow wave activity for subject P.C. across the period of the clinical study. Conversely, there was a shift toward lower frequencies in the EEG of subject B.H., which was characterised by low voltage fast activity. There was no evidence of any progressive increase in SMR or mu frequencies in the EEG for any of the subjects. Previous claims for a unilateral normalization of the EEG were also not replicated.

Some workers argue that the therapeutic action of EEG feedback training on epilepsy reflects a facilitation of arousal and EEG desynchronization; whereas others propose that an increased ability to relax and withstand stress is involved. Since there is evidence that the *proclivity for seizure activity* is greater during both low and high states of arousal, it is suggested here that the clinical effects might be associated with a modulation of the balance of synchronization and activation in the CNS to a less epileptogenic state. It has been demonstrated that individual neurons in experimental epileptic foci exhibit varying degrees of pathology as a function of the state of cortical activation, such that abnormal burst discharges are augmented both by thalamo-cortical synchronization and by afferent stimulation. Techniques which lead to a reduction of hypersynchronous slow wave activity, or of anxiety states, are thus interpreted to be beneficial. Both effects were

evident in this study.

In addition, subject N.L. reported that he developed the ability to arrest the onset of some of his psychomotor seizures, which were usually preceded by a specific aura. This phenomenon might possibly be associated with the learned facilitation of active mechanisms in the desynchronization of neuronal epileptiform activity.

8:1

METHODOLOGY

This work has confirmed that individuals are able to learn to modify selectively their EEG in a relatively short period of time through the provision of sensory feedback. However, it has also been demonstrated that there is a great variation in the response to feedback training between different subjects. Some subjects have been able to achieve a considerable enhancement of occipital alpha or central 12-16 Hz EEG above baseline levels; whereas others have consistently shown a response opposite to the one sought by training.

This problem of the individuality of the response to feedback training, as yet, has received very little attention in the literature. However, since it may easily confound the interpretation of the data, it would seem that an experimental methodology is required that would take such individual differences into account. One approach might be to screen a large number of subjects so as to select them according to their ability to modify the desired EEG response. This group of subjects could then be used to investigate the validity of feedback training phenomena without the inclusion of subjects who merely serve to increase the error variance.

In addition, the reasons underlying the variation in the response to feedback training need to be investigated, since we cannot expect to obtain predictable results across subjects until these powerful individual differences are understood. It is possible that psychometric techniques may provide some illumination of this problem. For example, Travis et al. (1975c) found that the subjects who were the most successful at controlling their alpha activity scored highly on the neuroticism scale of the *Eysenck Personality Inventory*. Similarly, Ancoli (1975) reports that such subjects had low authoritarianism scores according to the *California F-*

Scale, and high trust scores according to the *Comrey Personality Scale*. However, similar reports of correlations between baseline alpha and various personality measures (Saul et al., 1949; Mundy-Castle, 1955; Werre, 1957; Savage, 1964) have not proved to be very robust.

Another factor that this present work has demonstrated to be of crucial importance, but again one which has received very little attention in the literature, is that of the techniques used for signal detection and feedback training. Since there are many technical pitfalls to trap the unwary in this field of research, a thorough understanding of the operation of the apparatus used is essential in order for the results to be properly interpreted. For it is the detection of specific components in the EEG that provides the criterion for contingent feedback and, thereby, the basis for learned control. Accordingly, the accuracy of detection as well as the functional relevance of the signal component selected is of prime importance. The variety of artifacts and transients which can invade the highly amplified and filtered EEG signals are such as to require close attention. However, relatively few investigators have seriously considered the problems involved in EEG signal detection. Many workers employ commercial biofeedback devices, which are in general designed without much regard to the complexity of the EEG signal. Thus in a recent evaluation of twenty-six such devices Rugh and Schwitzgebel (1975) found that there was little uniformity or precision with respect to many critical characteristics. For example, the bandwidths of alpha filters varied from 0.9 Hz to 9.7 Hz! Such differences make the results of laboratories using different devices extremely difficult to compare. Furthermore, commercial biofeedback devices are often employed without the aid of concurrent polygraphic recording. Since the results are highly susceptible to contamination by artifact, this practice can only lead to a further conflict of data. Although these issues may appear to be highly technical, it is

essential that they should be dealt with scientifically since, clearly, any claims for the training of the EEG or for the implications that this work may have are meaningless if the data on which they are based are fundamentally unsound.

Many parameters of EEG feedback training remain to be investigated. For example, there has recently been a debate concerning the use of analogue feedback or binary feedback signals. Analogue feedback is reported to be the most effective for enhancing alpha activity in the eyes-closed condition; and binary feedback the most effective for enhancing alpha in the eyes-open condition (Travis et al. (1974b)). Similarly, it has been argued that integrated measures are more sensitive to changes in the EEG than binary measures, since all information about signal amplitudes above the criterion threshold is lost by the binary technique (Travis et al., 1974b; Hardt and Kamiya, 1976b).

A major disadvantage of both the analogue feedback and the integral quantification techniques that has been overlooked in the literature, however, is that they normally rely upon a band-pass filter to select the appropriate EEG signal; hence out-band activity, especially high-amplitude artifact, can contribute significantly to the data. In order to minimise this problem in this present work a binary detector was used in conjunction with an integrator (*see* 3:5:2).

Other factors whose influence on the EEG feedback training phenomenon needs to be more thoroughly investigated include the instructional set given to the subjects, and the general experimental conditions under which the feedback training is presented. Thus it was claimed in a number of early studies that the enhancement of EEG alpha rhythm often resulted in feelings of great tranquility and well-being (Brown, 1970, 1971, 1974; Kamiya, 1967, 1968, 1969; Maslow, 1969). However, subsequent workers have demonstrated that such experiences may almost certainly be attributed to expectation and experimenter effects (Walsh, 1974; Travis et al., 1975b;

Plotkin, 1976a, 1976b; Plotkin and Cohen, 1976).

The main emphasis of EEG feedback training has remained on the alpha rhythm, although there have been some reports of the modification of other EEG components.

The claims for enhancement of 12-16 Hz SMR have been discussed in detail in this thesis. It was found that, although some subjects were able to enhance 12-16 Hz activity in their central EEG, there was little support for the assumption that this activity corresponds to feline SMR. Rather it would appear that the nearest human analogue to the feline SMR is the classic mu rhythm, which is normally of a lower frequency although mu waveforms within the SMR frequency band have been observed (*see* 2:4:2). There was, however, some evidence of 12-16 Hz activity localised to the rolandic cortex in one subject studied in this work. Hence further investigation is necessary to determine whether 12-16 Hz central EEG components are functionally related to motor inhibition, or whether they merely represent the filtered 'noise' of the background EEG. More sensitive measures are needed in order to investigate the possibility of time-locked changes in other physiological variables during the presence of this activity, similar to those accompanying SMR in the cat (*see* 2:5:2), before it can be concluded that SMR does, or does not, exist in man.

There have been a few attempts to train the 3-7 Hz theta rhythm of the EEG, but the results have been somewhat inconsistent. In general, a significant suppression of theta activity has been obtained, but there is less evidence for its enhancement (Beatty et al., 1974; Klug and Brown, 1974; Lutzenberger et al., 1975; O'Hanlon et al., 1976); although Sittenfelt et al. (1976) report that by modifying the training procedure to suit the individual response pattern of each subject, most subjects were able to demonstrate some enhancement of theta. Theta activity associated with the onset of drowsiness is, of course, a potent confounding

factor in any such study.

Beatty (1971) reported the training of beta activity. However, the beta activity was simply defined as desynchronized occipital activity of 13 Hz or above. Hence the beta training was essentially a corollary of alpha enhancement and suppression training, and did not involve the selective modification of any functionally distinct EEG pattern.

Recently, there have been claims for the training of a specific 40 Hz component in the EEG (Newton et al., 1975; 1976; Sheer 1975a; 1975b). The enhancement of this activity is said to be associated with high arousal and concentration. However, the isolation of 40 Hz EEG activity from EMG artifact is a formidable technical exercise. Moreover, there has been no evidence presented to demonstrate that the findings did not result merely from a non-specific increase in EEG desynchronization.

Much more rigorous research is needed on the methodology of training EEG patterns other than the alpha rhythm in order that the EEG feedback training work may become more broadly based. In addition, since the various components of the EEG may not be independent, it would be enlightening to determine the effect that the selective modification of one particular EEG pattern has on other significant EEG waveforms. Hence there is a good case for arguing that more sophisticated data processing techniques, such as spectral analysis, are needed in order that the overall effect of feedback training on the EEG might be assessed.

The evidence, at present, suggests that EEG synchronization patterns reflect a localized balance of inhibition and excitation within the CNS, particularly in thalamo-cortical subsystems (*see* 2:2:1). From the results of this work, and that of other investigators, it appears that the experimental modification of EEG activity is primarily effected through the selective enhancement of the behavioural state that produces the appropriate balance of neuronal inhibition and excitation for EEG synchrony at a particular frequency and cortical localization.

For example, it is well known that the appearance of the alpha rhythm in the EEG may be readily influenced by changes in the level of sensory stimulation, that is opening or closing of the eyes. However, since in the feedback training context the subject is invariably instructed either to keep the eyes open or to keep them closed, the afferent sensory drive is maintained at a relatively constant level. Hence a number of workers have suggested that alpha enhancement and suppression is mainly mediated by the regulation of efferent oculomotor processes (*see* 2:2:3); although changes in sensory input are also likely to be involved to some extent as, for example, in the defocusing strategy used for alpha enhancement in the eyes-open condition. The hypothesis that oculomotor activity is closely related to alpha abundance, however, remains to be experimentally verified. It is essential that oculomotor activity should be much more carefully monitored than in the previous alpha feedback studies. In order to do this, methods for the continuous quantification of ocular tracking, accommodation, and convergence need to be developed.

In the case of feline SMR, the cessation of phasic motor activity, adoption of fixed posture, and specific somatomotor and visceral changes strongly implicate somatic mediation for its enhancement (*see* 2:5:2). No evidence has yet been found, however, for a similar involvement of motor

inhibition in the enhancement of rolandic 12-16 Hz in man. On the basis of results of this work, it was suggested that subjects might enhance 12-16 Hz components in the rolandic EEG by subtle modulation of the level of psychophysiological arousal (*see* 4:2:4).

It would seem probable that a number of general factors are important for the appearance of specific EEG patterns. For example, because of the strong involvement of non-specific excitatory drive in the resetting and gating of thalamic rhythmic discharges (*see* 2:2:2), the individual must learn to attain the appropriate state of arousal. Thus, for many subjects, an important factor contributing to the alpha increases observed during feedback training is the habituation of the influences associated with sensory and cognitive stimuli that normally serve to block alpha activity. The degree to which these factors can be voluntarily excluded is likely to vary greatly from subject to subject. In addition, gross bodily functions such as cerebral blood flow, oxygen tension, and free hydrogen ion concentration (pH) have significant and widespread influences on the EEG (Penfield and Jasper, 1954) and so must be maintained within the appropriate limits. However, there is not necessarily any close relationship between autonomic activity and EEG patterns. Nowlis and Kamiya (1970) were unable to find any consistent relationship between heart rate, blood pressure, respiration rate, or GSR and the abundance of alpha in five subjects who were successful at modifying their alpha levels. Similarly, Beatty and Kornfeld (1972) found no correlation between heart rate or respiration rate and alpha abundance.

Since peripheral factors appear to be closely involved in feedback training of the EEG, it has been argued that the direct control of EEG activity has not yet been demonstrated (Johnson, 1976). Similarly, a major theoretical controversy surrounding the early work on the experimental modification of autonomic and visceral responses was whether or not somatic or cognitive mediators were responsible for the effects

observed (Katkin and Murray, 1968). However, since the morphology of the EEG is itself a complex expression of many influences, it is doubtful whether any such distinction between mediated and unmediated learning has any theoretical or practical relevance.

Nevertheless, the possibility of the direct facilitation of synchronous EEG activity within the CNS itself exists by virtue of strong corticofugal innervation of the specific and non-specific thalamo-cortical projection systems. For example, it has been demonstrated that thalamic synchrony may be induced from projections arising in the frontal lobes (Penfield and Jasper, 1954; Sterman and Wyrwicka, 1967) or the motor cortex (Massion, 1975). In addition, studies of electrocortical classical conditioning have provided some evidence for the training of EEG activity at the central level. Thus the study of Doty and Giurgea (1961) indicated that cortico-cortical coupling could be established as a simple and direct consequence of a large number of stimulus and response repetitions. The operant conditioning of single units, both in the cortex (Fetz (1969) and in sub-cortical structures (Olds, 1965), also illustrates the extent to which a very fine degree of control over electrical activity in the CNS may be obtained.

The EEG training work has often been interpreted as an operant conditioning paradigm, with the feedback signal providing a continuous schedule of reinforcement (Beatty, 1971). However, it is unclear whether the traditional operant model is appropriate for the human biofeedback learning situation, since many of the characteristic features of operant learning, such as extinction phenomena, have not been reported (Peper and Mulholland, 1970).

An alternative interpretation is that the feedback signal functions as a discriminative cue so as to enable the subjects to differentiate the appropriate behavioural and subjective conditions which are required for a particular EEG event, although they may not necessarily be aware of

this process. For example, Brener (1974) argues that the extrinsic feedback stimuli may serve to calibrate the connection between a particular response pattern and the internal state, as defined by interoceptive feedback, that is associated with it. In general, this viewpoint emphasises the informational rather than the motivational character of the feedback signal, which is in accordance with the results from this work (*see* 3:4:3).

Although there has been very little investigation of the processes by which individuals acquire control over normally involuntary responses, there is some evidence to suggest that this is accomplished through the aid of responses that are already under voluntary control. The desired event appears to be elicited initially as part of a global response and then becomes progressively selected (Bair, 1901; Kimble and Perlmutter, 1970; Johnston, 1976). In a number of studies of electrocortical conditioning it was thus found that there was a consecutive alteration of events, at first diffuse, and subsequently focal (Magoun, 1964; Wyler and Fetz, 1974). In this present work, the strategies described by the subjects who were successful at modifying their EEG were mostly very general, and the subjects could only guess at the specific actions involved (*see* 4:2:4). It is reasonable to expect, however, that with extensive practice they would be able to more accurately distinguish the relationship between the occurrence of feedback and the accompanying internal state.

As suggested above, it is desirable that there should be closer attention to the monitoring of subtle changes in behavioural factors during EEG feedback training. This might then provide some insight into how the selective modification of EEG activity is accomplished; and, in turn, might also afford us with a deeper understanding of the relationship between brain activity and behaviour.

Much has been written about the potential clinical application of EEG feedback training, but in practice very little has been achieved. Many workers have been eager to apply this new approach and have claimed successful results. For example, the enhancement of alpha has been reported to be an effective treatment for tension headaches (McKenzie et al., 1974), phobias (Benjamins, 1976), intractable pain (Gannon and Sternbach, 1971), dyslexia (O'Mallery and Conners, 1972), and behaviour disturbances (Childers, 1975). SMR training has been reported to relieve both insomnia (Hauri et al., 1976) and hyperkinesis (Shouse and Lubar, 1976), in addition to epileptic seizures. Theta training is also said to be a successful treatment for insomnia (Sittenfeld et al., 1976). Finally, the enhancement of 40 Hz EEG has been claimed as a therapy for attentional and learning disabilities (Sheer, 1975b).

None of these findings can be regarded as reliable, however, since the studies are characterised by a confounding of variables and a lack of control procedures. An objective evaluation of the results is made almost impossible because insufficient attention has been paid to important conceptual and methodological issues. A much more rigorous approach is needed than has been evident in much of the biofeedback literature, since uncritical claims and wishful thinking can ultimately lead only to disappointment.

At present, the treatment of epileptic seizure disorders is the only clinical application of EEG feedback training to be well documented. Nine independent laboratories have presented results, to date, from studies involving a total of forty-eight patients of diverse seizure types, including those in the present work. Thirty-eight (79%) of these patients responded successfully to the various EEG training procedures. Moreover, it is important to remember that the patients chosen for the feedback

training studies had typically experienced severe and frequent seizures for many years, and that these seizures had failed to respond to prolonged trials of anticonvulsant medication, which in some cases reached toxic levels. It has been estimated that about 25% of epileptic patients have seizures that are inadequately controlled by conventional chemotherapy (Seifert and Lubar, 1975). In the United Kingdom this figure would represent some seventy-five thousand patients (Pryse-Phillips, 1969). Considering the alternatives that are presently available for the treatment of the drug-refractory epileptic, the results of the feedback training studies must therefore be regarded as most promising.

EEG feedback training might also be an effective adjunct to chemotherapy for patients whose seizures are controlled by sufficient anticonvulsant medication so as to enable it to be reduced, since the medication itself may produce severe and debilitating side effects. Some of these toxic effects that have been identified are hypersomnia, ataxia, hirsutism, skin disorders, and various metabolic disturbances (Woodbury et al., 1972). It has also been demonstrated that phenobarbitone, which is often the drug of first choice for epilepsy, can produce an impairment of perceptual motor performance even at dosages within the normal therapeutic range (Hutt et al., 1968).

Epilepsy is often associated with strong psychological dependencies and depression. The feedback training paradigm emphasises the participation of the patient in the control of the malady, and might thus encourage self-esteem and confidence - factors which in themselves may well have secondary beneficial effects on the disease process.

It has been demonstrated that the therapeutic effects of EEG training persist after the training has been terminated, provided that the withdrawal of training is made gradually. Sterman (1977b) reports that the clinical improvement of one of his patients was maintained over a

twelve-month follow-up period after the final withdrawal of SMR training. Lubar (1976) also reports that when two patients were gradually weaned from feedback training a transitory increase in seizure activity was observed, but they eventually became seizure free. Finley (1976) similarly reports that only a transitory increase in seizures occurred for his patient during a six-month follow-up period after the termination of training. Cabral and Scott (1976) also found in their study of the use of alpha feedback and relaxation training for the treatment of stress-related seizures that, for all three patients, the therapeutic effects obtained persisted over a fifteen-month follow-up period. It was not possible to carry out a similar follow-up study to the present clinical study because of insufficient data and changes in the treatment of the patients. However, the three patients all firmly expressed the wish to continue the feedback training, and so arrangements were made for training sessions to be given by staff at the Centre on an informal basis.

Thus, although EEG feedback training cannot be claimed to be a cure for epilepsy, in the light of these findings it does appear to be a potentially powerful therapeutic tool. However, the specific aspect of the feedback training paradigm that is responsible for the clinical effects is still open to question. It is not possible to infer causality in the relationship between changes in the EEG of the epileptic and clinical improvements, since this relationship may be circular. In addition, since very few controls have been carried out, it has not been adequately demonstrated that placebo factors are not involved. It is still possible that merely being a subject in these studies would itself have been as effective as the feedback training.

One significant facet of the EEG feedback training and epilepsy studies is that there are several examples of subjects who report that they developed an ability to directly inhibit seizure activity, as with

subject N.L. in the present study (*see* 7:2:2). In the study by Wyler et al. (1976) one patient stated that the feedback helped her to learn to "fight off" attacks. Lubar and Bahler (1976) also report that feedback training resulted in some of their patients experiencing an aura for the first time in association with epileptic activity, and that they were able to block many of their seizures successfully. Upton et al. (1975) similarly found that four patients became aware of auras during feedback training. For one of them this happened in the first training session, and thereafter they remained free of minor seizures for eighteen months. Other examples of individual subjects who acquired the ability to detect the onset of a seizure for the first time, and to inhibit its development following EEG training procedures are provided by Kaplan (1975) and Stevens (1962).

According to Lennox and Cobb (1933), approximately one half of all epileptic patients experience some form of aura; up to about one third may be able to inhibit their seizures after the aura has begun (Servit et al., 1962; Paulson, 1963). It is thus possible that feedback training might benefit some patients by teaching them to recognise an internal state associated with epileptic disturbances. Once this recognition has occurred the patient can then experiment with inhibitory manoeuvres such as fist clenching or fixed concentration. A successful suppression of epileptic seizures achieved in this way, of course, rules out any question of a placebo effect.

Clearly, a thorough and properly controlled investigation of EEG feedback training and its effects on epilepsy is needed. It is intended to carry out such an investigation as a follow-up study to this project, using a much larger group of patients. A new EEG feedback training unit has been developed for this purpose, on the basis of the results of the clinical study. An eclectic approach was taken in the design of this new

unit in that it incorporates all the EEG feedback training paradigms that have been demonstrated to be of therapeutic benefit (*see* 7:2:1). Thus training is given for the enhancement of a broad 9-20 Hz band in the EEG, combined with the suppression of slow wave activity below 7 Hz. Visual feedback is presented by means of a panel meter that is preset to give maximal deflection for normal EEG activity, but to recede as the proportion of pathological slow wave activity increases. Auditory feedback may also be presented in the form of a white noise signal which increases in intensity according to the predominance of low to high frequency activity in the EEG. The visual and auditory feedback signals are thus independent of absolute changes in the amplitude of the EEG, either within or between subjects. In addition, a spike detection and feedback circuit described by Upton et al. (1975) is included for the benefit of patients, such as subject B.H. in the present study, who exhibit very few abnormal slow wave discharges. Feedback of the occurrence of any epileptic spikes, sharp waves, and muscle artifact in the EEG is given by a short tone signal that varies in pitch according to the amplitude of the event.

The training unit has been designed to be portable and simple to operate. Detachable saline pad electrodes are positioned on a headband to record inter-hemispherically from approximately the same sites on different subjects. The EEG amplifier itself is also mounted on the headband so as to minimise artifact. The complete unit is pictured in Plate III.

It is proposed to compare the effects of contingent and random feedback using matched groups of patients from the David Lewis Centre. One group will receive feedback training to enhance broad-band 'normal' EEG activity and to suppress pathological slow wave and transient activity; the other group will receive similar feedback, but derived from a white

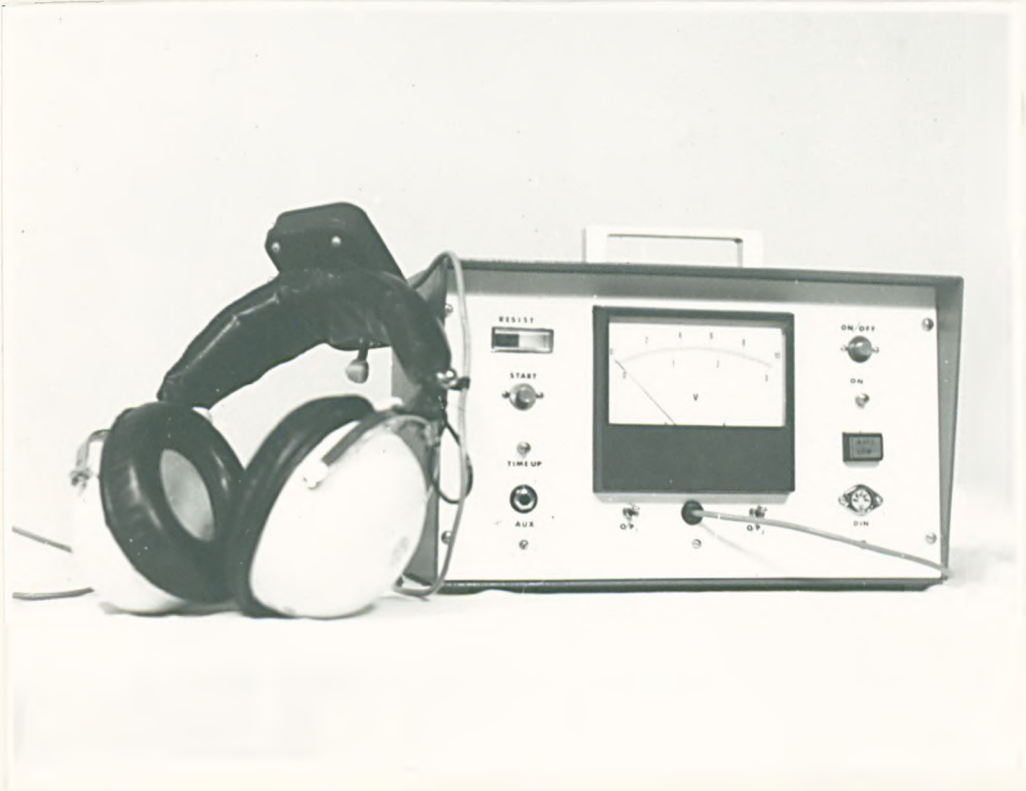


Plate III.

EEG feedback training unit developed for the proposed follow-up study to this work. The EEG is recorded by the headband mounted electrodes and pre-amplifier. Feedback of the balance between slow wave activity and EEG of a broad mid-frequency band (9-20 Hz) is provided either by the large meter or by amplitude-modulated white noise. Auditory feedback of the occurrence of epileptic spikes and muscle artifact in the EEG may also be obtained. Outputs are available for the external monitoring and quantification of the data.

noise source. If possible, this procedure will be carried out 'double-blind'. In addition, since a much larger group of patients will be involved than in previous studies, it is possible that some insight may be gained into the basis of individual differences in the response of epileptics to EEG feedback training. Such differences may be shown to be related to seizure types, or to various physiological and psychological factors.

If EEG feedback training does prove to be a practical treatment for epileptic seizure disorders, then because the new feedback unit has been specifically designed so that it may be operated easily by the patients themselves, there is the hope that therapy in the long-term might be secured through the provision of such a device for use in the home.

An overview is presented of the many methodological, technological, and theoretical issues that remain to be resolved in the area of EEG feedback training. Of particular importance are individual differences in the response of subjects to feedback training; the limitations of the training and data processing techniques that are used, and the mechanisms by which subjects are able to learn to modify their EEG.

Some possible directions for future research are indicated. In particular, it is proposed to carry out a comparative, follow-up study of the effects of random and contingent EEG feedback on epileptic seizures, using a much larger group of patients.

This research began in the hope that, in addition to illuminating some of the issues involved in EEG feedback training, an effective adjunct to conventional anticonvulsant therapy might be developed. This hope, at least in part, has been fulfilled.

APPENDIX

A SYNOPSIS OF THE SENSORIMOTOR RHYTHM DETECTION AND FEEDBACK SYSTEMS
USED BY OTHER WORKERS IN THE TREATMENT OF PATIENTS WITH EPILEPSY

The equipment used by the other groups who have reported the effects of SMR feedback training on epilepsy to date is outlined below from the published specifications. The groups are presented alphabetically according to the principal worker. Brief comments on each system are made in the light of the considerations to be taken into account in the design of such equipment, that were discussed in Chapter Five.

Finley (Finley, 1974; 1975; Finley et al., 1975; Finley, 1976).

EEG ($C_3F_3 - A_2$) was fed to a high-Q resonant (twin-tee) filter, sharply tuned to 12 Hz and with steep skirts ranging from 76 to 188 dB/octave. SMR was detected by a level-sensitive relay circuit which apparently turned on to 3-4 waveforms of 12 Hz equal or exceeding 5 μ V (Finley, 1976) and off when the 12 Hz activity was absent for at least 0.5 sec (Finley et al., 1975). The input to the filter was limited to 60 μ V by a clipping amplifier. The EEG was also passed to another resonant filter tuned to "5.5 \pm 1.5 Hz" with skirts of 20 dB/octave. A relay operated by this filter at an unspecified voltage inhibited the 12 Hz relay. The percentage of time that the 12 Hz and 5.5 Hz relays were enabled during 100 sec intervals was measured by timer counters. Feedback of 12 Hz activity was presented by a blue light and a 1500 Hz tone. A red lamp was also operated by the 5.5 Hz relay. In addition, monetary reward was given for every 5 sec of 12 Hz activity accumulated.

An example of the output of this system is provided by Finley et al. (1975). The 12 Hz SMR relay is seen to be registering SMR almost continuously over the epoch of some 30 sec shown. Similarly, Finley (1976) reports that the SMR for his subject increased from 10% to an average of 70%

following training. However, these results are extremely difficult to reconcile with those of Serman et al. (1975), who found that in all their subjects there was a paucity of narrow band EEG activity, and concluded that, "The narrow-band requirement of the detection system utilized clearly was testing the physiological limits of voluntary EEG manipulation": the more so since the 12 Hz filter used by Finley was, in fact, even more narrowly selective than that employed in the Serman study (*see* 5:1:2).

It is possible, of course, that Finley's patient was highly unusual in this respect. Alternatively, much of the output of the high-Q SMR filter might have been due to the effects of transients and sharp waveforms in the EEG; or perhaps simply reflect in-band 'noise' components of the high voltage background EEG.

Kaplan (Kaplan, 1974; 1975).

EEG (1 cm posterior to C_4 referred to A_1) was fed through a band-pass filter of 24 dB/octave to a PDP/12 computer which was programmed to function as a digital filter based on peak and trough detection. Both filters were set to a passband of 12-14 Hz, which, in practice, gave a maximal response between approximately 13-14.5 Hz, falling to zero at 12-15 Hz (according to the response curve provided by Kaplan, 1975). When the appropriate EEG frequency occurred the computer operated a relay which controlled various feedback devices such as tone signals, music, coloured lights, television, tape recorder, or a slide projector. Separate power spectral analysis (averaging 4 sec epochs sampled across each session) was employed to determine the relative power of the 12-14 Hz activity.

Although Kaplan used very precise digital filtering to isolate 12-14 Hz activity in the EEG, no inhibit circuits of any sort were provided. Furthermore, it appears that no minimum threshold level was specified for the 12-16 Hz activity. Clearly any harmonic components of epileptiform

and artifactual waveforms in this frequency range would be registered by the computer as genuine 12-14 Hz EEG, in addition to any spurious signals generated by the effects of these high voltage waves on the analogue filter. Thus the subjects were likely to receive a considerable amount of feedback during the occurrence of undesirable and pathological activity, which is possibly why Kaplan failed to obtain much clinical improvement in her study of 12-16 Hz feedback training.

Kuhlman (Kuhlman, 1976; Kuhlman and Allison, 1977).

Kuhlman provided his epileptic patients with feedback training of rolandic EEG (the exact electrode sites are not specified) in the 9-14 Hz frequency range. Hence this work cannot strictly be considered to be a study of SMR training. However, the amount of activity in the 12-14 Hz SMR frequency range for each session was quantified by off-line power spectral analysis of the EEG. The 9-14 Hz activity was detected by band-pass filtering and zero-crossing analysis; a lamp and counter were used to provide feedback. No inhibit circuitry was included, and hence "the patients were not required to concomitantly suppress muscle activity or low frequency abnormal EEG activity in order to receive feedback" (Kuhlman, 1976).

Lubar (Lubar and Seifert, 1975; Seifert and Lubar, 1975; Lubar, 1976; Lubar and Bahler, 1976).

EEG was initially recorded from electrodes placed at 10% and 30% down from the vertex on either side of the head. The recording leads were then shifted to $C_3 - T_3$ and $C_4 - T_4$ after three months of training (Lubar and Bahler, 1976). A relay system was arranged so that each hemisphere was alternately used for feedback training. The EEG was fed to a band-pass filter of greater than 80 dB roll-off during the first octave, which was tuned to approximately 12-14 Hz. If the amplitude of the filter output

exceeded a threshold level in the order of 5 μ V, further analysis was carried out by an instantaneous frequency to amplitude converter, and a voltage comparator set to generate a train of pulses during the presence of 12-14 Hz. The raw EEG was simultaneously passed through a 4-7 Hz band-pass filter of 24 dB/octave. A voltage discriminator on the output provided an inhibit pulse which was gated with the pulses from the SMR detector. The threshold amplitude of the 4-7 Hz inhibit circuit is not given. In the absence of an inhibit pulse, the SMR output pulses were accumulated by a digital rate-meter over one-minute epochs. In addition, a burst detector was incorporated which required the subject to produce six cycles of 12-14 Hz activity in 0.5 sec in order to register a criterion response. Feedback of the criterion responses was given by a row of 10 coloured lights and a tone signal. A digital display indicated the cumulative number of criterion bursts per minute. A slide projector was also sometimes used. Feedback of high voltage slow wave activity was provided by a green lamp controlled by the inhibit circuitry.

This equipment, with a combination of analogue and digital processing plus interacting amplitude and duration analysis, is very similar to the system that was used for the clinical study. One difference is that the inhibit circuit in Lubar's system was only tuned to slow wave activity; hence epileptic spikes or EMG artifact might engender both false feedback and erroneous data (*see* 4:1:3).

Sterman (Sterman and Friar, 1972; Sterman, 1973a; 1973b; 1974; Sterman et al., 1974).

EEG (from varying derivations but principally $C_zC_3-C_3T_3$) was filtered by a 13 Hz resonant filter which (from the response characteristics given by Sterman, 1976b) had an attenuation slope of some 40-60 dB/octave. A level sensitive relay circuit was used to indicate activity above a threshold amplitude of 3-6 μ V (Sterman, 1973a). An amplitude discriminator

detected the presence of activity exceeding 40 μ V (Stermán, 1976b) in the raw EEG and operated a relay to inhibit feedback (Stermán et al., 1974). A duration logic circuit was included so that a burst was required to exceed a criterion duration in order for the subject to receive feedback. The criterion duration was set in the region of 0.5 sec (Stermán, 1973b) to 0.75 sec (Stermán, 1974); but, because of the analogue level detection technique used, a total burst of some 1.0 sec was needed to produce a response (Stermán, personal communication, 1974). Feedback was given by two rows of ten lamps. Each criterion response advanced illumination of the top row of lamps and was accompanied by the sounding of a single chime. Each ten criterion responses successively activated the lamps in the bottom row, accompanied by a double chime. The criterion responses were also used to operate a slide projector. A single large lamp was included that glowed in proportion to the output of the 13 Hz filter. A digital counter was used to record the incidence of criterion responses.

The response curve of the SMR filter provided by Stermán (1976b) indicates that there would be very little response to out-band activity when used in conjunction with amplitude discrimination above 40 μ V. However, it also indicates that this selectivity is achieved because the filter does not, in fact, respond equally to 12-14 Hz activity but is tuned to only a narrow peak at 13 Hz (see 5:1:2). Hence it is unlikely that the output of this system would provide an accurate indication of the presence of SMR activity in the EEG.

Stermán (Stermán 1976c; 1977a)

Stermán's more recent work involved a portable home-training unit (Neurofeedback Instruments, Neuroanalyser 4000). This provided for the reinforcement of one frequency band contingent upon the suppression of activity in a second frequency band. One test band was fixed at 12-15 Hz, another at 6-9 Hz, and a third at 18-23 Hz. The permutations of these

frequency bands were varied according to the experimental design. Criterion amplitude and duration circuits were provided, and an inhibit circuit used to block the reward system whenever there was a transient above $50 \mu\text{V}$ in the raw EEG signal. The portable unit displayed a bright continuous amber lamp as the desired frequency occurred, plus a flashing discrete green lamp, a tone, and an increment of a digital display when criterion amplitude and duration were reached. Red lamps were turned on both for criterion activity in the undesired frequency band and for the occurrence of high voltage activity. The unit contained a four-channel strip chart recorder which registered each of the above events on very slowly moving paper. Off-line power spectral analyses of the EEG ($C_2C_3-C_3T_4$) were made from sleep EEG recordings at three-month intervals and the percentage of 12-15 Hz contained within the 9-20 Hz band; and the percentage of 4-7 activity within the 0-9 Hz band, was quantified.

This portable unit provided for the feedback of broader frequency bands than the earlier equipment used by Serman, although information on the actual frequency response of the analogue filters in the unit has not yet been given. This unit also had the advantage of inhibition and feedback circuits for both slow wave activity and for high voltage transients. However, the extent to which this system would be able to efficiently discriminate against spurious responses cannot be assessed without more detailed specifications.

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