Physiological Origins and Functional Correlates of EEG Rhythmic Activities: Implications for Self-Regulation[1]

M. Barry Sterman[2]
Veterans Administration Medical Center Sepulveda, and School of Medicine, University of California, Los Angeles

Recent neurophysiological findings in relation to thalamocortical mechanisms for sensory processing, together with established anatomical and expanding functional evidence, have provided a rational theoretical framework for the interpretation of normal and abnormal EEG rhythmic activities. This perspective is integrated here with earlier animal studies which were the foundation for many current applications of EEG self-regulation as a clinical tool. Basic evidence concerning the origins, frequency modulation, and functional significance of normal EEG rhythmic activities is reviewed here in an effort to provide guiding principles for the interpretation of clinical abnormalities and their remediation with EEG feedback training.

Descriptor Key Words: EEG; thalamic circuits; SMR; event-related responses; epilepsy; ADHD; ADD; substance abuse.

Although much has been learned through basic research about the underlying neural mechanisms and functional correlates of rhythmic EEG activi-

[2]Address all correspondence to M. Barry Sterman, Ph.D., Neuropsychology Research (151-A3), Veterans Administration Medical Center, 16111 Plummer Street, Sepulveda, California 91343.

ties, surprisingly little conceptual integration of this knowledge has been evidenced in recent clinical applications of EEG self-regulation. We now know clearly that relevant EEG rhythmic patterns reflect the unique properties of thalamocortical circuits, that these EEG patterns are topographically localized in relation to nervous system organization, and that the interaction between specific and nonspecific sensory and cortical influences determines their frequency and cortical expression.

The contemporary literature in these areas is so extensive, and the methods so complex, as to preclude an extensive review in this context. Instead, an attempt will be made here to integrate recent neurophysiological findings with earlier animal studies that helped to spawn the rapidly expanding field of clinical EEG self-regulation. Recent human studies will also be evaluated from this updated perspective. Finally, an effort will be made to apply insights derived from this approach to an evaluation of several areas where EEG self-regulation is being used as a clinical tool. It is hoped that this effort will help to bridge the gap between basic and clinical thinking in this new and promising field.

BACKGROUND

Our earliest effort to study the EEG in relation to waking behavior was stimulated by the pioneering experiments of the Russian physiologist Ivan Pavlov. In his classical conditioning studies of salivary and gastric secretion in dogs, Pavlov (1927) encountered what he referred to as psychic influences related to the phenomenon of higher-order neural inhibition. Through the gradual juxtapositioning of conflicting conditioned responses (i.e., food vs. shock) his experimental dogs became progressively lethargic and often fell asleep. He described this response as *"internal inhibition."* According to Pavlov, under these circumstances "the positive conditioned stimulus itself becomes, under definite conditions negative or inhibitory." He proposed further that some instances of natural sleep onset were caused by an identical process.

We became interested in the concept of internal inhibition because of a research focus on the neural mechanisms for sleep onset. EEG criteria for the initiation and staging of sleep were gradually emerging at that time, and provided us with a tool that was not available to Pavlov. If internal inhibition was indeed related to sleep onset, we speculated, then this behavioral state should produce similar EEG changes.

To evaluate this possibility we attempted to elicit internal inhibition using operant conditioning methods instead of the classical conditioning employed by Pavlov (Roth, Sterman, & Clemente, 1967; Sterman & Wyr-
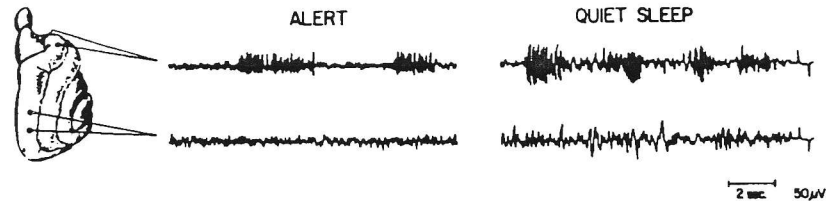
Fig. 1. Bipolar EEG recordings from sensorimotor (coronal) and parietal (marginal) cortex in a cat during alert motionless waking behavior and quiet (non-REM) sleep. Note similarity in topography and frequency of SMR and sleep spindle EEG rhythmic patterns. (From Sterman, Howe, & MacDonald, 1970.)

wicka, 1967; Wyrwicka & Sterman, 1968). We first trained cats to obtain food with a bar-press in an experimental chamber. We next introduced a tone (conditional stimulus) which was associated with a shut-down of the feeder apparatus mechanism. When the tone was sounding a bar press was not rewarded with food. Instead, the tone was prolonged. If the press was made after the tone stopped food was delivered. We hypothesized that a conditioned suppression of the bar-press response during the tone in hungry cats would produce a state approximating internal inhibition.

EEG recordings obtained during the elaboration of this conditioned response disclosed two behaviorally specific EEG patterns. One was related to the learned suppression of the bar-press response during presentation of the tone, and the other to the reward following completion of a correct response. The EEG pattern associated with the suppression of bar-pressing behavior in hungry animals was a 12-20 cycles per second (c/s) rhythm localized to sensorimotor cortex. Because of this localization we labeled this the "Sensorimotor Rhythm," or SMR. This pattern was, indeed, very similar to the characteristic sensorimotor EEG spindle burst seen during sleep (Figure 1). This fact suggested that some aspect of underlying neurophysiology was common to these two behavioral states.

The EEG pattern associated with food reward, on the other hand, was a 4-12 c/s rhythm localized to posterior-dorsal (parietal) cortex, instead. In the well-trained animal it was seen as soon as the feeder mechanism was activated and during food consumption. Because of this consistent association with primary and secondary reward components we labeled it "Post-reinforcement Synchronization," or PRS. Similar activity was seen also during ad lib drinking, grooming, and drowsiness (Figure 2), indicating some common physiological feature among these states as well.

These two patterns were the only clear instances of visually detectable rhythmic EEG activity observed in the alert cat. Further, they were distin-
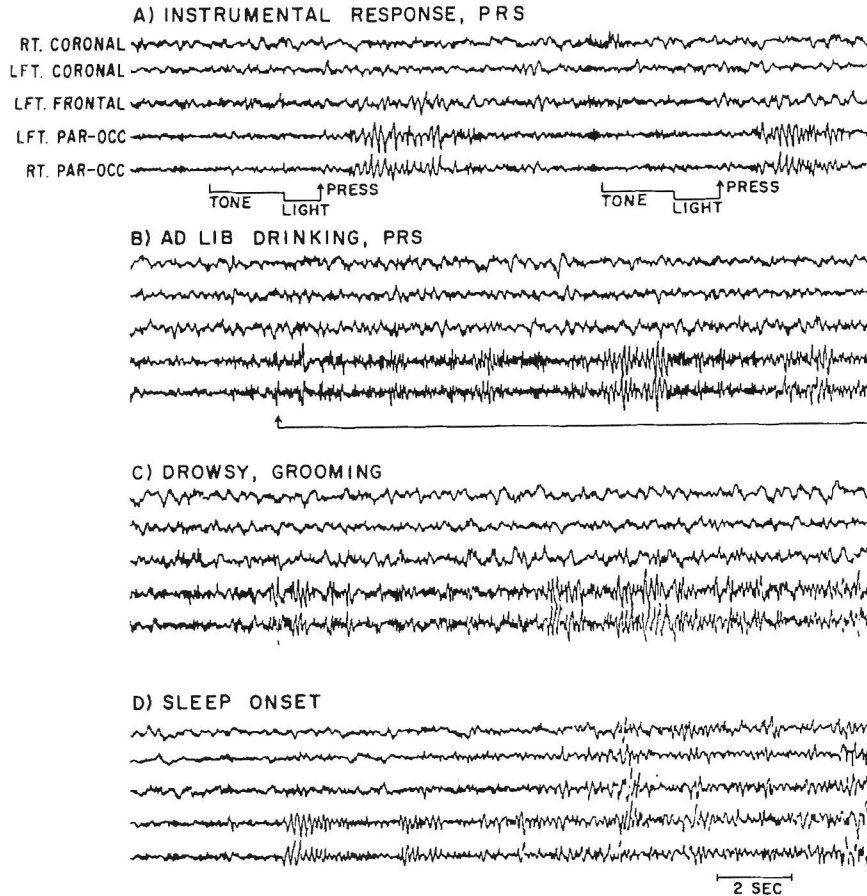
Fig. 2. Sample EEG traces from coronal, frontal, and parieto-occipital cortex in a cat during various behaviors. (A) EEG during bar-press for food. Reward was delivered after a tone-light conditional stimulus. Note rhythmic EEG activity (Post-Reinforcement Synchronization, or PRS) in parieto-occipital traces. Well-trained animals eventually showed PRS during conditional stimulus as well. (B)-(D) spontaneous PRS-like activity during ad lib food consumption, grooming behavior, and just prior to sleep onset. (From Sterman & Wyrwicka, 1969.)

guished by the fact that they emerged clearly from a background of non-rhythmic, low-voltage EEG activity (see Figures 1 and 2). These patterns were so distinct, in fact, that we decided to study them directly using operant conditioning methods in an attempt to train their voluntary elicitation. The resulting EEG feedback studies were among the earliest systematic investigations of self-regulation with this measure.

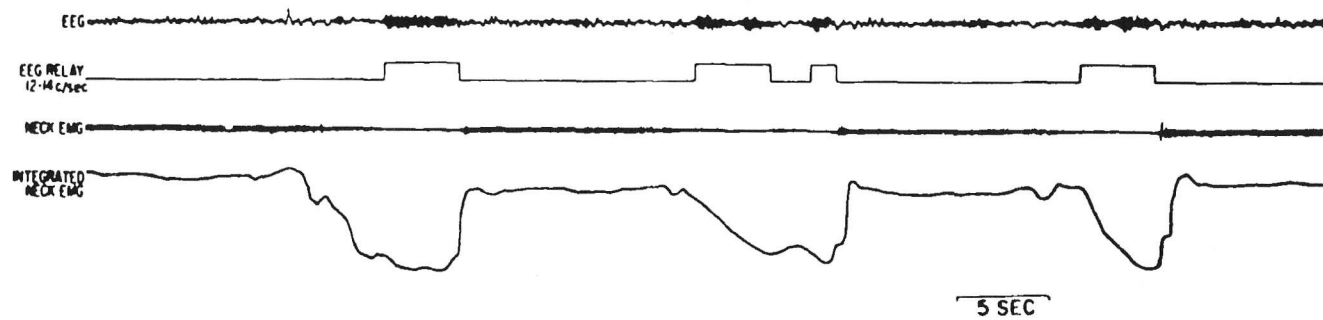Fig. 3. Trained SMR responses are shown here from sensorimotor cortex in a cat together with simultaneously recorded neck EMG activity. Also shown is relay marker for detection of criterion SMR activity and a computer-integrated EMG trace. Note that both direct and integrated EMG traces show characteristic suppression of tonic motor discharge during each SMR response. (From Chase & Harper, 1971.)

THE SENSORIMOTOR RHYTHM

Operant Conditioning

Although frequency analysis of the SMR showed a range of activity between 12 and 20 c/s, peak activity for this rhythm was found at approximately 12-14 c/s. Operant conditioning was thus initiated with an active signal detection filter centered at 13 Hz, and set to activate the feeder device when criterion amplitude and duration triggered a relay (Sterman, Wyrwicka, & Roth, 1969; Wyrwicka & Sterman, 1968). We found that cats could easily be trained to produce this rhythm voluntarily. Between 150 and 200 conditioned responses could be elicited prior to satiety, occurring at approximately 5-10 s intervals during a typical experimental session.

The behavior associated with this conditioned response was again the sustained suppression of motor activity. However, movement suppression was necessary but not sufficient for production of the rhythm. Careful studies of muscle activity changes associated with SMR training showed that each response was immediately preceded by a 4-6 s period of graded muscle tone reduction (Figure 3), at the end of which the SMR would appear (Chase & Harper, 1971; Wyrwicka & Sterman, 1968). These changes in muscle tone occurred within the context of a sustained immobility.

Neurophysiological Considerations

Neurophysiological studies of the SMR in animals disclosed several major facts. First, this rhythmic activity originated in the somatosensory relay nuclei of the thalamus, collectively known as the *Ventrobasal (VB) nuclei* (Howe & Sterman, 1972). This finding was in agreement with a earlier body of evidence from anesthetized preparations, summarized in the popular text by Andersen and Andersson (1968). Thus, for example, when connections between VB thalamus and sensorimotor cortex are severed, rhythmic EEG activity disappears in somatosensory cortex. Second, during conditioned SMR activity VB relay cells changed their firing patterns from fast or random discharge to systematic bursting activity (Harper & Sterman, 1972). This is consistent with contemporary neurophysiological findings, as will be discussed below. Third, the conduction of somatosensory information through the ventrobasal thalamus was suppressed during SMR activity (Howe & Sterman, 1973), a fact that is also consistent with recent neurophysiological findings (Steriade & Llinas, 1988; Warren & Jones, 1994). Finally, as we have already seen, the SMR was associated with a

corresponding suppression of muscle tone and, as later demonstrated, stretch reflex excitability (Babb & Chase, 1974).

The bursting discharge observed in VB thalamic relay neurons during SMR and sleep spindle activity is now understood to result from the intrinsic electrochemical properties of thalamic relay cells (Llinas & Jahnsen, 1982; Lopes da Silva, Vos, Mooibroek, & Van Rotterdam, 1980; McCormick, 1992; Steriade, Gloor, Llinas, Lopes da Silva, & Mesulam, 1990). Communication among neurons is mediated by changes in the membrane that surrounds the cell and its axonal and dendritic processes. When at rest, current gradients maintain a small electrical potential across the cell membrane. Excitatory impulses reaching the cell release transmitter substances that can reduce, or *depolarize*, this potential. If this depolarization reaches the cell's activation threshold, polarity is briefly reversed and the cell discharges, propagating the signal along its axon. Other transmitters or circumstances can increase the resting potential, a process called *hyperpolarization*. Since it is harder to reach activation threshold in this condition, the cell is inhibited.

Signals from sensory pathways are conveyed to the cerebral cortex through relay nuclei in the thalamus, the VB being the relay nucleus for the somatosensory pathway. When relay cells in these thalamic nuclei become hyperpolarized, they show atypical behavior. Rather than remaining in a stable inhibited state, the hyperpolarization gradually decays. A slow calcium influx mediates this depolarization. The resulting "low-threshold calcium spike" causes the membrane to eventually reach its discharge threshold, leading to a subsequent "high-threshold sodium spike" consisting of a unique burst of discharges. This burst is relayed to cortex and, simultaneously, to functionally related cells in an adjacent thalamic nucleus called the *Nucleus Reticularies Thalami* (nRt). This nucleus forms a thin sheath on the outside of all sensory relay nuclei. When excited by these bursts, cells in nRt will respond with a similar burst of activity. The output of these cells is directed back to the same population of relay cells, and releases the inhibitory substance *gamma amino butyric acid*, or GABA, which again hyperpolarizes the relay cells. This hyperpolarization initiates another slow depolarization and burst discharge in the relay cells, and the process starts over again.

The feedback between these thalamic cells produces a recurrent oscillatory discharge which is entrained among many relay cells and can last up to several seconds. Corresponding synchronous volleys of discharge are projected to functionally related pools of cortical cells. These cells respond collectively with summated membrane oscillations resulting in rhythmic field potentials, which can be recorded as EEG rhythms at the cortical surface. Thus, when VB cells become hyperpolarized and initiate bursting
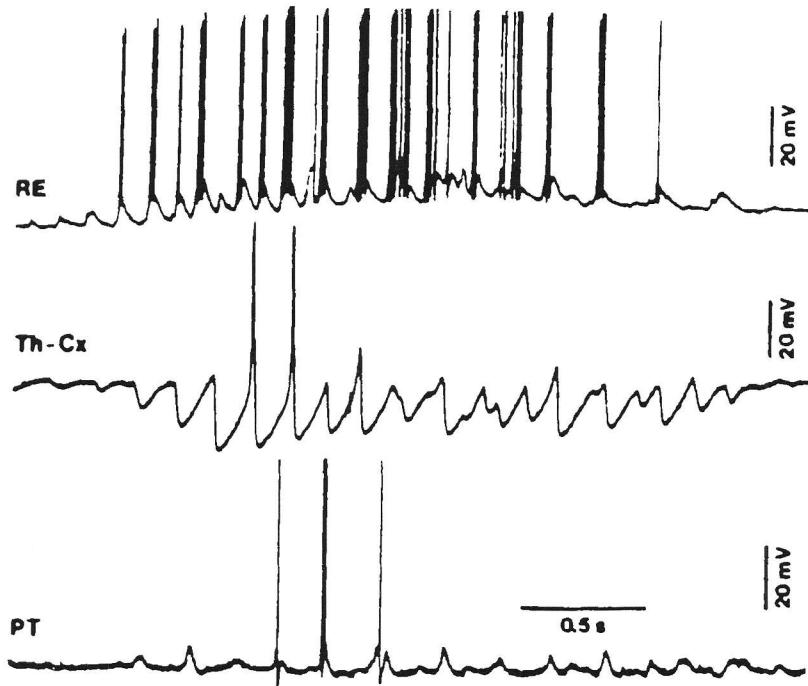
Fig. 4. Intracellular recordings from functionally interacting cells in nucleus reticularis thalami (RE), thalamocortical relay nucleus (Th-Cx), and motor cortex (PT) during a spontaneous EEG spindle in a barbiturate anesthetized cat. Depolarization-induced bursts of RE neurons impose GABA mediated hyperpolarizations on Th-Cx neurons whose subsequent depolarizations again activate RE cells. The resulting oscillation in relay cell activity projects rhythmic volleys to pools of cortical cells whose synchronized field potentials are recorded as EEG rhythms. (From Steriade & Deschenes, 1988.)

discharge EEG rhythms appear on sensorimotor cortex. Similarly, when thalamic relay cells associated with visual functions become hyperpolarized and begin bursting, EEG rhythms appear on parieto-occipital cortex.

The degree of hyperpolarization, the number of thalamic relay cells induced to collective oscillation, and the receptivity of cortical cells to the resulting gated input is determined in large part by additional modulating influences. Among these are the so-called "nonspecific systems," which determine the background excitability of cell populations. For example, anesthetics such as barbiturate act on nonspecific systems to reduce cellular excitability, thereby causing changes in thalamic relay and nRt cells and initiating the EEG rhythm generating process (Figure 4).

Recent neurophysiological studies of this EEG rhythm-generating mechanism have in fact been limited to anesthetized preparations and to *in vitro* studies in extracted tissue slice preparations. They have also focused on input to thalamic relay cells from cortex and nonspecific brain stem systems as the critical modulators of the rhythm-generating process. However, it is abundantly clear that the primary input to sensory relay cells is afferent discharge from appropriate receptor pathways. With regard to the somatosensory system, the work in cats described above has shown that attenuation of this input alone can initiate the oscillatory process in thalamic relay cells.

Other findings support this conclusion. Thus, transection of the spinal somatosensory pathway (dorsal columns) at a high cervical level or lesions of brain stem components of this pathway in cats were both followed by a significant increase in both SMR and sleep spindle activity (Bowersox & Sterman, 1982; Shouse & Sterman, 1979). Further, physical restraint of cats and primates also produced an increase in SMR activity (Bouyer, Debet, Debray, & Rougeul, 1978; Bowersox, Siegel, & Sterman, 1978; Holcomb, Sterman, Goodman, & Fairchild, 1979). Finally, in studies of humans with high spinal cord injuries, where clinical trauma has provided for somatosensory deafferentation, a significant enhancement of SMR and sleep spindle activity has been observed as well (Sterman, 1977).

We may conclude, therefore, that during active behavior a number of excitatory sources can act on ventrobasal thalamic relay neurons to maintain depolarization. These include specific somatosensory, nonspecific brain stem, and various cortical inputs. These excitatory sources suppress cellular bursting (in both VB and nRt cells) and, thus, reduce or eliminate EEG rhythms in sensorimotor cortex. Conversely, during inactive behavior the attenuation of somatosensory inputs alone can hyperpolarize VB cells, promote oscillatory discharge, and initiate SMR rhythms. As we shall see below, if this state is accompanied by drowsiness, hyperpolarization in some thalamic cell populations is increased and slower rhythms emerge as well. From a functional perspective, therefore, the characteristics of the sensorimotor EEG can be viewed as reflecting thalamocortical circuit dynamics related to the presence or absence of movements, the level of general excitation, and the status of motor programming. These same influences also affect intracortical mechanisms which contribute to the magnitude and spread of projected EEG rhythms.

Early studies in cats and primates suggested that the frequency of sensorimotor rhythms varies as a function of activation (Bouyer, Dedet, Konya, & Rougel, 1974; Holcomb et al., 1979; Rougeul, Bouyer, Dedet, & Debray, 1979). Slower frequencies were associated with drowsiness and progressively faster frequencies with increasing excitation. Bouyer et al. (1974) pro-

posed that different thalamocortical generators mediated these different rhythms. We shall call this the "independent frequency generator model." Recent neurophysiological evidence, however, suggests an alternative explanation for these frequency changes. Both Steriade, Dossi, and Nunez (1991) and McCormick and Huguenard (1992) found that the frequency of membrane potential oscillations in thalamic relay cells slows when hyperpolarization is increased. This finding raises the possibility that frequencies may gradually slow with the progressive withdrawal of excitatory inputs to thalamocortical circuits. We shall call this the "graded frequency generator model."

The substrates underlying different EEG frequency patterns are complex. Thalamic sensory nuclei are made up of both relay cells conveying specific afferent information and interneurons which modulate relay cell function (Jones, 1985). Further, the nucleus reticularis appears to be made up of different cell populations that are either tonically active or induced to bursting activity (Pinault & Deschenes, 1992). Additionally, while specific sensory activation of thalamic relay cells is mediated by the excitatory neurotransmitter *glutamate*, all of these cell types receive multiple nonspecific inputs from brain stem cholinergic, adrenergic, and serotinergic pathways as well (McCormick & Huguenard, 1992; Steriade et al., 1990). Finally, the organization of thalamocortical projection and the status of local cortical circuits are clearly significant factors in EEG frequency modulation.

Given these many levels of complexity it is clear that the expression of a given EEG rhythm could easily be multiply determined. It is unfortunate, therefore, that tradition in the EEG field has assigned specific names to specific frequency bands, suggesting that they are discrete, independent entities. Certain frequencies may indeed be associated with specific functional systems. Others, however, can appear either locally or generally, and reflect distinctly different processes. We will have more to say about this issue below.

POSTREINFORCEMENT SYNCHRONIZATION

Operant Conditioning

As described above, the EEG synchronization associated with the reinforcement of a learned operant response in cats was a 4-12 c/s pattern seen over parietal cortex (PRS). Evidence from several sources indicates that this activity is at least partially generated within primary and secondary visual pathways by a thalamocortical gating mechanism similar to that de-

scribed for the SMR but involving lateral geniculate, posterior-dorsal, and adjacent thalamic reticular nuclei (Lopes da Silva, Van Leirop, Schrijer, & Storm van Leeuwen, 1973; McCormick & Huguenard, 1992; Sprague, 1966). Intracortical networks are also involved in its EEG expression. It should be mentioned, also, that a significant output from the visual pathway is relayed to the brain stem reticular core, unlike proprioceptive activity in the somatosensory pathway, which proceeds exclusively to the cerebellum and ventrobasal thalamus (Carpenter, 1991).

One might question how modulation of activity within the visual pathway is related to food reward in the cat. Let us consider the relevant functional circumstances. A conditioned bar press in our cats was always followed by food reward. This signaled the completion of a goal-directed behavior, and was presumably accompanied by reduced activation, a response which itself has been characterized as rewarding (Berlyne, 1971). The animal's attention at this point clearly shifted from an expectant focus on the apparatus to consumption of the reward. This was typically accompanied by a partial closing of the eyes. Given these considerations, the food reward would be associated with a brief period of reduced activity in both specific visual and nonspecific brain stem excitatory pathways. These combined changes would presumably cause a transient hyperpolarization and bursting within relay cells of the lateral geniculate and associated thalamic nuclei of the visual system, leading to a corresponding cortical EEG rhythmic pattern.

To examine these dynamics more systematically we attempted to train PRS activity with the same operant conditioning paradigm used in our SMR studies (Sterman & Wyrwicka, 1967). Conditioning was initiated with active filters set to detect criterion 6-10 Hz activity and to activate a relay for the delivery of a liquid food reward. At first animals merely became quiet in their effort to voluntarily produce PRS activity, often sitting in front of the feeder with eyes partially closed. Within 20-40 trials they began to recline after consuming the food, sometimes failing to respond to the next reward. During this period PRS-like activity emerged at other cortical sites during the operant response phase. Finally, within 30-50 reinforcements, animals disregarded the feeder entirely, and produced sustained and generalized trains of 6-10 c/s EEG activity.

Function and Mechanism

With either simple bar-press training or SMR conditioning for food reward animals generate some 150-200 responses before voluntarily ceasing performance. Further, they remain alert and expectant prior to virtually

every response, standing or sitting in front of the feeder. However, with operant conditioning of the PRS, animals soon reclined after each response, and performance was seldom sustained beyond 50 responses. Behavioral quiescence and generalized slow EEG rhythmic activity accompanied this truncation of performance.

Why did these hungry animals cease working for food? The answer to this question must reside in the underlying neurophysiological changes required for the voluntary production of PRS. Both the SMR and PRS are waking EEG patterns which can occur spontaneously under appropriate conditions. Spontaneous SMR is seen during motor response inhibition and sustained motor quiescence in an otherwise alert animal. Voluntary production of the SMR, therefore, requires the animal to effectively stabilize or suppress somatosensory proprioceptive input while remaining generally attentive. Spontaneous PRS, on the other hand, is seen at the completion of a rewarded response, during intrinsically rewarding behaviors such as feeding and grooming, and when the animal becomes drowsy. Voluntary production of the PRS, therefore, is functionally associated with a transient termination of, or sustained withdrawal from, active engagement.

Support for this conclusion can be seen in Figure 5. Here cortical and VB thalamic EEG recordings are shown during and immediately after an SMR training session. During performance the SMR and PRS were clearly differentiated in relation to behavior, with SMR occurring during movement suppression and PRS following food reward. The VB recording showed activity related to the SMR, as expected, but displayed no activity related to the PRS. However, when the animal became satiated, disengaged, and sat quietly, both faster (SMR) and slower (PRS) frequencies could be seen alternating in sensorimotor cortex *and in VB thalamus*. Sustained motor quiescence can account for the observed SMR activity. However, with the added condition of disengagement, slower activity that was originally limited to posterior cortex was generated in ventrobasal thalamocortical circuits as well.

Since the somatosensory proprioceptive pathway is specific, and goes directly to relay neurons in VB thalamus without sending collaterals into the brain stem excitatory systems, a reduction in afferent activity during operant performance would not simultaneously reduce general activation. As suggested above, withdrawal of this specific input appears to preferentially generate higher-frequency (SMR) activity in cortex. Slower sensorimotor EEG frequencies, therefore, must reflect an independent state change, as was seen with satiety and drowsiness. However, the fact that the visual pathway relays heavily to brain stem excitatory centers suggests that attenuation of this input may directly reduce nonspecific excitation as well. With reference to the "graded frequency generation model," the in-
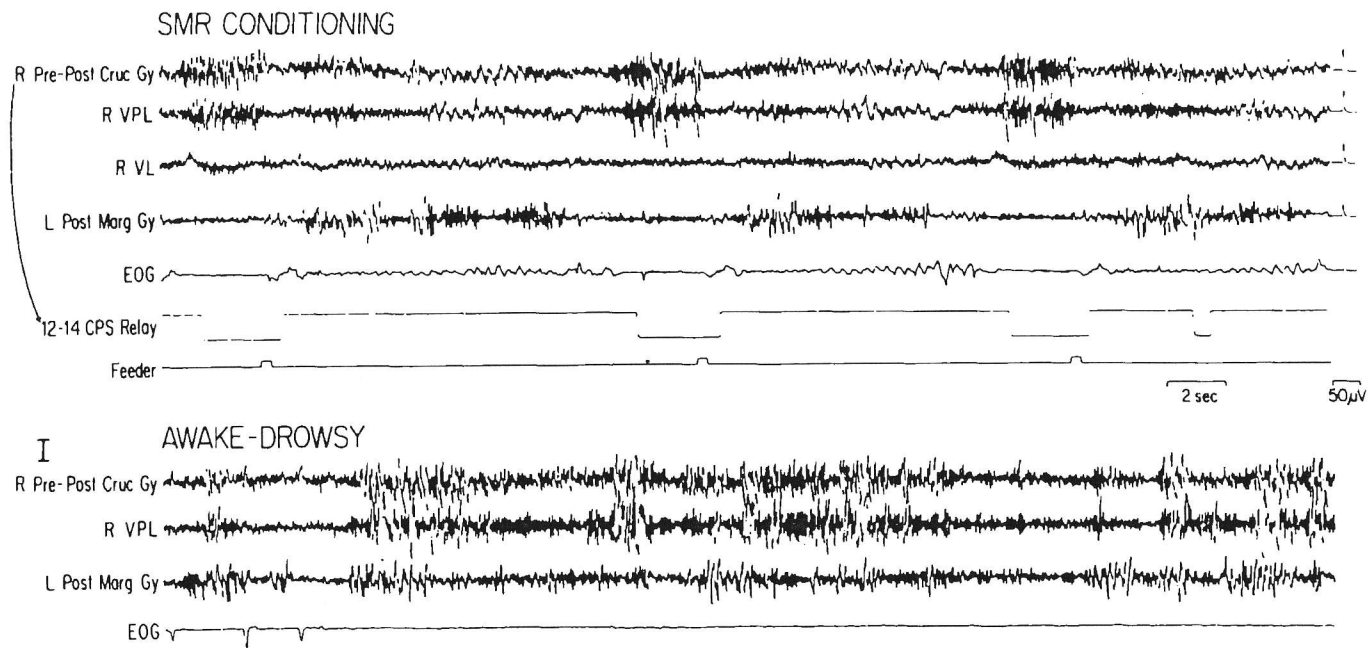
Fig. 5. Sample polygraphic traces during and immediately following an SMR EEG feedback training session in a cat. EEG activity from sensorimotor cortex (pre-post cruciate gyrus), the right nucleus ventralis posterolateralis of thalamus (VPL), and parietal cortex (left posterior marginal gyrus) is shown together with electro-occulographic activity (EOG), a 12-14 Hz criterion SMR response relay marker, and feeder activation. Note systematic SMR-PRS sequencing during performance, with SMR evident in sensorimotor cortex and PRS in parietal cortex. SMR can also be seen at its source in VPL. With satiety and relation both frequency patterns appear in both thalamic and sensorimotor cortical recordings. (From Howe & Sterman, 1972.)

creased thalamic relay cell hyperpolarization resulting from this combined withdrawal of excitatory inputs could explain the prevalence of slower frequency EEG activity from posterior cortex, and the difference between SMR and PRS frequencies. Further, repeated episodes of voluntary PRS production may ultimately lead to a generalized reduction of nonspecific excitation, as indicated by the eventual spread of slower frequencies to other thalamocortical systems. If not otherwise prevented by excitation, this gradual shutdown could, indeed, reduce motivation, preclude the continued processing of external stimuli, and set the stage for sleep. This, presumably, is why the animals showed an early cessation of performance.

HUMAN STUDIES

Bridge to Basics

The feline SMR and PRS have direct analogs in the human EEG. Numerous studies have described faster rhythms in central and precentral cortex that are suppressed with motor activity (see, for example, Jasper & Penfield, 1949; Pfurtscheller, 1981; Sterman, 1977). On the other hand, starting with the pioneering work of Berger (1930), many investigators have confirmed the functional relationship of the parieto-occipital alpha rhythm to visual and attentional processes.

However, several problems make comparisons difficult. The human brain is far more complex than our animal relatives, making it difficult to predict or interpret cognitive responses to environmental circumstances or to experimental instructions. Further, with surface recording, tissues overlying the brain distort field potentials and, in particular, attenuate higher frequencies. This fact, together with the high voltage and relative dominance of slower frequencies in the human EEG, tends to significantly mask faster frequencies. Finally, it is impossible to intentionally alter relevant pathways in humans. While clinical circumstances can be useful, as we have seen from the increased SMR activity that can occur after spinal cord injury, these considerations make it difficult to differentially examine the full range of EEG frequency components in humans.

One solution to this problem is to see what elements of behavior *suppress* rhythmic EEG patterns when sensory and cognitive inputs are manipulated. Different test requirements can be designed to selectively activate different functional pathways. With modern, computer-based EEG analysis methods it is possible to achieve a refined evaluation of both the frequency and topography characteristics of response to such requirements.

We have attempted this approach in a series of several recent studies seeking to differentiate EEG responses associated with simple visual attention, purposeful movements, visual tracking, and visuomotor integration related to vehicle control performance (Mann, Sterman, Suyenobu, & Kaiser, 1995; Sterman, Mann, Kaiser, & Suyenobu, 1994). Using EEG spectral analysis, sequential and overlapping 2 Hz frequency bands were studied between 5 and 17 Hz during multiple 2-min test trials. What we found was both expected and surprising.

When simple visual attention was compared with eyes closed, *all* frequencies between 5 and 15 Hz were significantly suppressed in temporal, parietal, and occipital cortex. The greatest suppression was in the 7-11 Hz range, which included frontal cortex as well. This generalized effect was surprising given the pervasive concept of parieto-occipital "alpha blocking" in the earlier EEG literature. However, the neurophysiological considerations discussed above suggest that it should be expected. Increased activity in specific and nonspecific excitatory pathways should alter all sources of rhythmic discharge in thalamus. This excitation apparently had the greatest effect on the 5-7 and 7-9 Hz bands, since they showed no further reduction with subsequent stimulation. Higher frequencies were clearly suppressed also, but not maximally. Among these, 7-11 Hz activity showed the greatest suppression but was still capable of further reductions with increased stimulation. These findings tend to support the graded frequency generation model (lowest frequencies most affected) but suggest selective effects on independent generators as well.

With body movements 11-15 Hz activity was selectively suppressed in central cortex (Figure 6, central). This was expected, since body movements suppress faster sensorimotor rhythms. With eye movements (visual tracking) 11-15 Hz activity was also selectively suppressed but in temporal-parietal cortex, instead (Figure 6, parietal). This was surprising. However, once again, the underlying neurophysiology provides an explanation. With both body movements and eye movements afferent sensory discharge would be increased in appropriate accending sensory pathways. Proprioceptive discharge from body movements excites pathways related directly and through cerebellum to thalamus and sensorimotor cortex, as discussed above. Proprioceptive discharge from eye movements, however, excites pathways projecting through cerebellum and thalamus to temporal-parietal cortex (Galletti, Battaglini, & Fattori, 1993; Stone & Lisberger, 1990). Thus, both body and eye movements selectively suppressed 11-15 Hz activity in appropriate but different thalamocortical pathways. Conversely, a reduction of this excitation would be expected to selectively *increase* 11-15 Hz activity in these areas. This finding supports the model of independent frequency
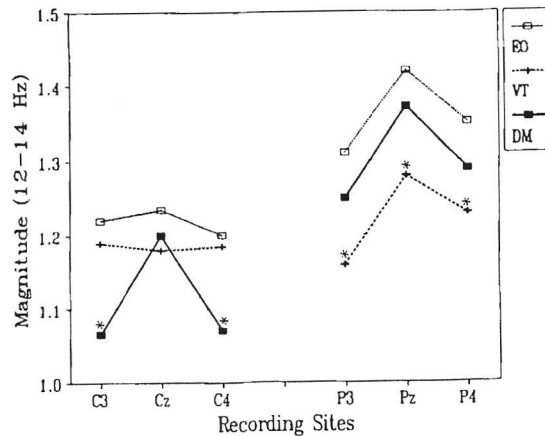
Fig. 6. Mean EEG spectral magnitude changes (n = 21) in the 12-14 Hz band at 3 central and 3 parietal recording sites during various 2-min control conditions in a driving simulation study. Control conditions included: eyes open viewing an out-the-window display (EO), visual tracking as the vehicle was rapidly self-propelled through this display (VT), and instructed driving movements without the display (DM). Asterisks indicate significant suppression of magnitude in comparisons among conditions ($p \leq .01$). Note significant suppression, indicated by asterisks, at central sites during DM and parietal sites during VT. (From Mann et al., 1995.)

generators for proprioceptive pathways. Further, it suggests that there is a VMR (visuomotor rhythm) as well as an SMR.

In both of these studies cognitive engagement suppressed 7-13 Hz activity in cortical regions relevant to the task. This was not surprising, since there is an extensive literature relating modulation in this frequency range to cognitive and attentional factors. In both studies, however, movements producing proprioceptive sensory discharge were involved in the integrative test condition. As discussed above, this would be expected to suppress 11-15 Hz activity in appropriate brain areas. Therefore, in these areas the added cognitive integration factor might have had a more limited effect, involving suppression of 7-11 Hz activity only. This distinction was in fact observed, since areas not related to proprioceptive inputs showed suppression re-

stricted to this lower band. A detailed resolution of this kind is helpful in differentiating specific functional effects. Under these circumstances the findings support the concept of multiple independent EEG frequency generators in all appropriate thalamocortical pathways.

Other laboratories have reported similar findings. For example, Pfurtscheller and Klimesch (1991) have concluded that higher frequencies in the alpha band are attenuated by sensory encoding while lower frequencies are suppressed by internal attentional processing. Additionally, Ray and Cole (1985) reported that high alpha and low beta frequencies were most affected by external stimuli, while the intermediate alpha frequency was most related to attention. It should be remembered, however, that these conclusions relate to circumstances where individuals are relatively alert or task oriented. It is well known that with drowsiness and the onset of sleep, frequencies below 7 Hz are generally increased, while higher frequencies fall off until spindles appear. This fact is consistent with the findings discussed above for these lower-frequency bands, and further supports the graded frequency generation model for lower frequencies, since the general withdrawal of excitatory inputs with drowsiness and sleep onset would increase hyperpolarization in thalamic relay cells.

Both models may therefore be correct, with independent generators functioning as the primary source of rhythmic EEG patterns during alert waking, and a graded process influencing thalamocortical circuits generally as alertness fades. Independent generators related to decreased proprioceptive input may remain active and even be facilitated during sleep, as cerebellar postural functions go off-line, thus accounting for the increased magnitude and spread of 11-15 Hz activity (spindles) during stage 2 sleep. Reality is certainly more complex than this, but these considerations have face validity, and may provide a guide to future insights.

It should be kept in mind, however, that EEG changes related to neural pathology, while consistent in some ways with this perspective, also reflect abnormal circumstances that can alter the substrates for observed EEG frequencies. For example, slower frequencies in the so-called "theta" range normally increase prior to sleep onset and during stage changes within sleep, most likely because of graded membrane changes attending the withdrawal of excitatory inputs, as discussed above. However, with focal cortical lesions or transmitter disturbances associated with seizure disorders, increases in this frequency have been attributed to local circuit dynamics triggered by abnormal cortical excitability (Gloor, Pellegrini, & Kostopoulos, 1979). Additionally, it has been shown that chemically induced changes in the dominant GABA receptor site on thalamic relay neurons can slow and intensify rhythmic oscillations, producing a theta-range EEG pattern resembling that seen with generalized absence seizures
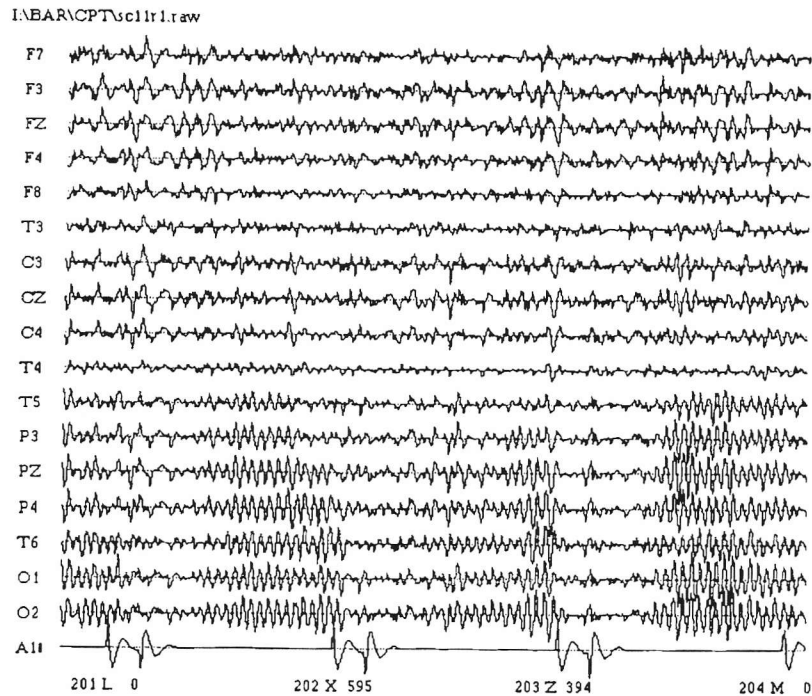
I:\BAR\CPT\sc11r1.raw

Fig. 7. Topographic EEG traces from 17 cortical recording sites in an adult subject during responses to a computer-guided continuous performance task. Subjects were instructed to press the computer space-bar in response to designated target letters displayed on a video screen and embedded in a continuous steam of letters appearing at 2-s intervals. Trace at bottom shows actual keyboard on/off response together with trial number, letter displayed on screen, and reaction time if letter was a designated target. Note transient EEG activations during response (event-related desynchronization or ERD) and trains of rhythmic activity at posterior cortical sites following response (post-response synchronization or PRS).

(Krosigk, Bal, & McCormick, 1993). Thus, at least three separate processes may be capable of producing theta frequency patterns in the EEG.

Event-Related Responses in the EEG

Although there is abundant evidence for the localized suppression of the dominant alpha rhythm with attention and cognitive processing, the

importance of the timing and topographic dynamic of this suppression has only recently been appreciated. In the studies described above timing was not a critical factor since performance was essentially continuous. However, when the sensory stimulus or cognitive task that elicits this suppression is transient, as in most performance and learning contexts, the suppression is also transient. Pfurtscheller and colleagues (Pfurtscheller, 1992; Pfurtscheller & Aranibar, 1977; Pfurtscheller & Klimesch, 1991), who have focused on these responses, have labeled this transient suppression as "event-related desynchronization," or ERD. Under some circumstances the EEG from certain brain areas shows an increase in alpha activity in response to a stimulus event. Pfurtscheller calls this "event-related synchronization," or ERS.

In a recent series of studies evaluating mental effort during a sustained continuous performance task (CPT) we also observed this EEG modulation (Veigel & Sterman, 1993). Subjects were required to make a keyboard response to several preselected letter stimuli appearing on a video screen at 2-s intervals and embedded in a continuous stream of similar nontarget letters. Visual inspection of EEG tracings not only revealed recurrent ERDs to these repeated stimulus events but also showed episodes of EEG rhythmic activity during the intervals between stimuli (Figure 7). Spectral analysis of these bursts showed that their primary expression was in the 8-10 Hz range. These findings suggest that performance was not at all continuous.

Because of their similarity to the PRS in cats, we have preferred to call these events "post-response synchronization" or PRS, to place them in the proper temporal and historical perspective. Like the PRS in cats, this activity was transiently related to the completion of a motivated response. We have seen it also on completion of a more truly continuous task involving the simulated landing of an advanced aircraft (Sterman & Mann, 1995). This PRS was similarly suppressed by excitation and increased task demand, and was most consistent in posterior cortex.

It is possible that the complex nature of human cognitive function produces ERD-PRS sequences continuously during alert waking behavior, perhaps accounting for the inevitable instability of the EEG signal (even after artifact has been removed). As such, the waking EEG reflects the sequential flow of cognitive responses to internal and external events and the variable recovery from these responses. For example, we have evidence from an expansion of the study mentioned above (Sterman, Kaiser, & Veigel, 1995) that rapid ERD and recovery sequences signal accurate recognition of cognitive associations (Figure 8). This may reflect excitation-inhibition sequences in thalamocortical circuits that efficiently encode and integrate information and then reset the system for the next challenge. Conversely,
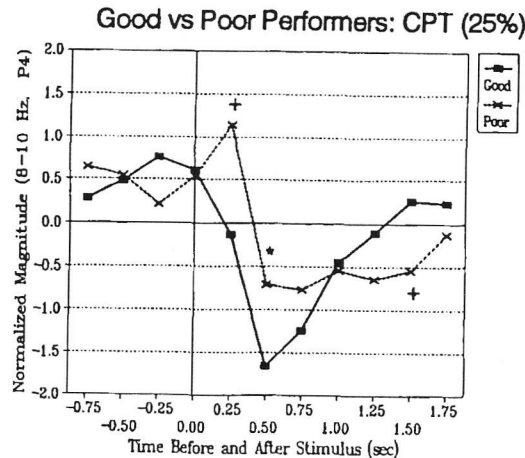
Fig. 8. Averaged event-related EEG response pro-
files at P4 to multiple target stimuli are compared
here in 10 good and 10 poor performers on the CPT
test described in text. EEG spectral magnitude data
at 8-10 Hz were normalized for each subject, aver-
aged across target responses and combined for
groups. Subjects were selected on the basis of accu-
racy scores from a larger test group. Mean magni-
tude values were compared at 0.25-s intervals from
0.75 s prior to stimulus presentation to 1.75 s after-
wards. Stimulus was presented at time 0.00. Poor
performers showed a paradoxical PRS, while good
performers showed a faster and greater ERD re-
sponse and a faster recovery. ($+, p < .05$; $*, p < .01$.)

paradoxical ERSs, followed by delayed and truncated ERDs, and slow re-
covery, were associated with poor recognition or recall of cognitive asso-
ciations. This pattern of response may indicate confusion and inefficient
information processing.

Pfurtscheller (1992) has reported further that ERDs in occipital cortex
during reading were accompanied by a corresponding ERS in central cor-
tex. Conversely, ERDs in central cortex during a strictly motor task were
accompanied by an ERS in occipital cortex. He considers these reciprocal
responses to be indicative of normal physiological regulation during visual
versus motor activities. Another example of the differential regulation of
visual and motor functions was demonstrated also in the vehicle control
studies discussed earlier (Figure 6). Thus, both local signal-processing dy-
namics and reciprocal relationships between functional systems are dis-
closed by a careful evaluation of the topography and timing of event-related
responses. It is clear that these considerations have major implications for

the quantitative study of the EEG, and for applications of EEG self-regulation where attention or other cognitive deficits are of interest. We hope that studies of this type will expand and provide additional assessment and training options for the field of EEG self-regulation.

CLINICAL APPLICATIONS

Seizure Disorders

The animal studies reviewed earlier provided for physiological and functional interpretations of sensorimotor rhythmic EEG patterns. These patterns reflect bursting discharge in VB thalamic relay neurons, which appeared to be initiated by a voluntary suppression of movement and a subsequent reduction in somatosensory afferent activity. Rhythmic activity appeared primarily in the 11-15 Hz frequency range during both wakefulness and sleep but was accompanied by lower frequencies of different origin in nonattentive states. The presence of these rhythms also reflected an increased threshold for concurrent somatosensory stimulation of cortex, since the conduction of afferent signals through the thalamus was attenuated. Once the SMR response was conditioned in animals the voluntary regulation of these neuronal dynamics became routine.

These conclusions are relevant to what was perhaps the most important discovery in our series of cat experiments. At the time we were also engaged in Air Force sponsored research seeking to develop dose-response functions for a highly epileptogenic family of hydrazine compounds used in rocket propulsion systems. Animals from completed studies were eventually entered into this series. Our findings in this regard were disrupted when we encountered cats who were significantly resistant to seizures after exposure to established convulsive doses of hydrazine. We found that these were animals used previously in our SMR operant conditioning research (Sterman, LoPresti, & Fairchild, 1969). Confirmation of the protective effect of SMR training was subsequently obtained in expanded studies of cats and primates (Bowersox & Sterman, 1981; Sterman, Goodman, & Kovalesky, 1978).

Hydrazine, because of its interference with the synthesis of GABA (Wood & Peesker, 1974), acts to profoundly increase both cortical and thalamic excitability. Owing to mechanisms we now understand, EEG rhythmic patterns are essentially abolished. This vulnerable condition allows any form of afferent stimulation, particularly somatosensory feedback from movement, to trigger generalized seizure discharge in cortex (Sterman et al., 1969). SMR trained animals, however, continued to produce rhythmic

activity *after* exposure to this compound, and either showed significantly prolonged latencies to seizures or failed to seize entirely. This could only happen if the training actually altered inhibitory mechanisms in thalamus, perhaps by increasing GABA synthesis, up-regulating GABA receptors, or facilitating some alternate inhibitory process. Whatever the reason, voluntary control of sensorimotor excitability was at least partially spared. Harking back to Pavlov, this may indeed reflect a learned "internal inhibition." Other findings suggest that prolonged training of this response may actually result in a sustained and protective enhancement of intrinsic inhibitory regulation (Sterman, 1977).

As a consequence of these findings clinical trials were initiated in human subjects suffering from epilepsy. Seizure disorders with primary motor symptomatology were deemed as most appropriate. Since humans were unlikely to work for milk or chicken broth, which proved effective in cats, equipment was developed to provide light, tone, and digital count rewards for the production of criterion SMR activity at lateral central cortical sites. Both seizure rates and EEG abnormalities were significantly reduced after as little as 3 months of feedback training in these initial studies (Sterman & Friar, 1972; Sterman, Macdonald, & Stone, 1974). Efforts were expanded in later studies to include larger groups of patients and to incorporate critical controls. These included the monitoring of medications, noncontingent (yoked) control groups, and adding suppression of lower frequencies in crossover designs (Lantz & Sterman, 1988; Sterman & Macdonald, 1978; Sterman & Shouse, 1980). Therapeutic effects limited to contingent training of the SMR with simultaneous theta suppression and uncontaminated by secondary considerations were obtained in all of these studies. Numerous experimental and clinical reports have now documented the therapeutic benefits of SMR training for motor seizure disorders. For the details of this extensive literature one can refer to a number of review articles with diverse perspectives (Kuhlman & Kaplan, 1979; Sterman, 1982, 1986, 1993; Wyler, 1984). It should be pointed out that the addition of low-frequency suppression to the training protocol in human studies was first reported in a study by Lubar and Bahler (1976).

A facilitation of thalamic inhibitory mechanisms provided one explanation for this therapeutic effect, as discussed above. An additional therapeutic effect emerged from studies of SMR training in human epileptics, although one that was also predicted by our animal work: We had found that cats who underwent extended SMR training showed significantly increased EEG spindle densities during sleep and a consolidation of sleep due to prolonged sleep episodes and a related decrease in state transitions (Sterman, Howe, & Macdonald, 1970). We subsequently reported similar effects in humans (Sterman & Shouse, 1980).
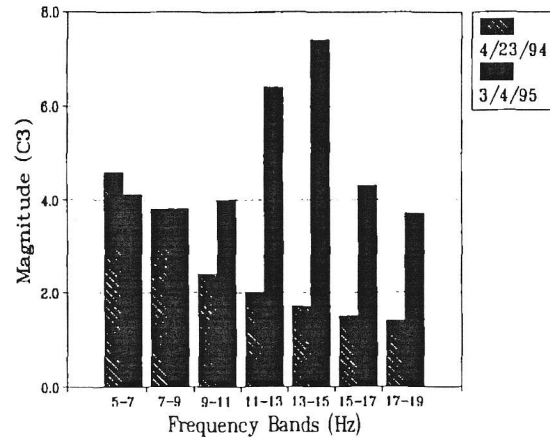
Fig. 9. Comparison of EEG magnitude distributions in 7 narrow-frequency bands at C3 after prolonged EEG feedback training for 12-19 Hz enhancement and 7-9 Hz suppression in an epileptic patient. Data are from standard 4-min feedback tests early in training (hatched bars) and approximately 10 months later (solid bars), and exemplify reliable changes in magnitude distribution. This unmedicated patient suffered from unusual partial-complex epileptic seizures and has accomplished significantly improved seizure control during the course of this training.

These findings are relevant to seizure pathology since it is now well established that patients with generalized and partial complex seizures often show a significant attenuation of EEG sleep spindle activity (Sterman, 1981), and an increased fragmentation of sleep architecture (Baldy-Moulinier, 1982; Hamel & Sterman, 1982; Rosen, Blennow, Risberg, & Ingvar, 1982). Shouse, Siegel, Wu, Szymusiak, and Morrisson (1989) have also found that spontaneous seizures occur most frequently during sleep stage transitions in an experimental animal model of partial complex epilepsy. Thus, the above-noted changes in sleep with SMR feedback training disclose physiological alterations which stabilize sleep and reduce sleep stage transitions, thereby countering some of the pathological consequences of epilepsy and potentially reducing vulnerability.

This protection may be relevant to waking-state transitions as well. Patients are most successful at achieving voluntary control of the SMR by sustaining visual attention while releasing and then stabilizing muscle tension (but not relaxing). In a well-trained patient activity at 11-15 Hz can be dramatically increased while slower and higher frequencies remain relatively suppressed (Figure 9). Suppression of slower frequencies confirms

sustained attention. Evidence for the concurrent suppression of higher frequencies is essential to assure that increases at 11-15 Hz are not mediated by an elevation of higher frequencies related to muscle artifact. Prolonged training to achieve and sustain this state may indeed reduce involuntary bouts of drowsiness, which often presage seizures in epileptics, and even facilitate mental capabilities. Findings from studies of SMR training effects on neuropsychological test performance support this conclusion by showing improved memory functions in successfully trained patients (Lantz & Sterman, 1988).

Attention Disorders

A second major application of EEG self-regulation has been in the area of attention deficit and hyperactivity disorders (ADD and ADHD). Lubar and Shouse (1976) and Shouse and Lubar (1979) first reported that application of an SMR feedback protocol similar to that used in studies of epilepsy resulted in significant behavioral and EEG improvements in boys with ADHD. These studies noted that SMR training primarily affected motor components of the disorder. Since that time Lubar's laboratory and a number of other investigators have offered additional support for this conclusion (Lubar & Lubar, 1984; Tansey, 1985), although robust controls are still lacking in this literature. Further, the Lubars suggest that the suppression of 4-8 Hz activity together with enhancement of frequencies in the 16-23 Hz range is most effective in improving attentional and organizational skills.

In seeking a theoretical explanation for reported therapeutic effects in this area one can certainly apply any of the considerations discussed above in relation to the physiological substrates of sensorimotor EEG patterns and the findings related to SMR training for seizures. In fact, both epileptics with motor seizures and boys with ADD show a significant enhancement of slower EEG frequencies and a deficiency in faster frequencies when compared to matched controls (Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992; Sterman, 1981). One can also draw from the previous discussion to find support for the suggestion that attention is facilitated with the suppression of lower frequencies.

From a theoretical perspective, it would seem that EEG self-regulation strategies might benefit from a consideration of established principles of human perception and performance (Boff, Kaufman, & Thomas, 1986). These principles identify the regulation of sensory input, cognitive integration, and motor output as key element in effective information processing.

Failures or deficiencies in these regulatory components may be etiological factors in attention and learning disorders.

As we have seen, this regulation can be reflected in local as well as topographic modulation of transient EEG responses. Abnormal sensorimotor excitability and associated motor activity may interfere with both perceptual and integrative components of information processing, since motor activity can disengage visual processing areas of the brain (Pfurtscheller, 1992). Moreover, failure to activate the temporo-parietal region was associated with poor attentional and memory performance in our CPT study (Sterman et al., 1995). Learned voluntary control of sensorimotor excitability with SMR training may facilitate processing by decreasing such motor interference, while at the same time maintaining perceptual and memory functions at the ready. It seems clear that the study of event-related EEG responses can contribute to this field by advancing our understanding of these disorders, identifying abnormalities, and, ultimately guiding training strategies.

Substance Abuse

The perspective developed here can be directed also to the recently derived EEG self-regulation strategy for the treatment of substance abuse (Peniston & Kulkosky, 1989, 1990). The so-called "alpha-theta protocol" combines relaxation training and guided imagery with 30-min sessions of eyes-closed feedback reinforcement for both alpha and theta frequency production in the occipital EEG. The alpha frequency band reinforced is 8-13 Hz and the theta 4-8 Hz.

According to our functional model of EEG pattern origins, enhancement of occipital 8-13 Hz implies suspension of both visual encoding (higher component) and integrative processing (lower component). These requirements may be easy at first, since closing the eyes eliminates visual input while relaxation exercises would set the stage for reduced cognitive processing. However, the simultaneous reinforcement of 4-8 Hz and 8-13 Hz frequency bands is problematic.

Physiological considerations indicate that this would, at best, be difficult. Given the eyes-closed relaxed condition of the patient, 8-12 Hz activity should be increased. However, successful enhancement of the slower theta frequency should simultaneously reduce 8-13 Hz activity (graded frequency generation model) and initiate a progression toward drowsiness and sleep. This, as we have seen from studies described above, would prematurely terminate any true voluntary control.

Studies applying the alpha-theta protocol indeed report an initially high and stable alpha level followed eventually by periodic declines in alpha as theta is more or less abruptly elevated. The emergence of theta activity under these conditions is, in fact, a well-established marker of impending sleep. It is likely, therefore, that successful alpha-theta EEG feedback training facilitates a functional transition toward sleep. However, continued reward for the higher-frequency alpha band as well as verbal interrogation by the therapist may prevent progression of the sleep process.

It has been claimed from several therapeutic perspectives that this "edge of sleep" state can be therapeutically useful in divulging suppressed emotional content (Cowan, 1993; Wickramasekera, 1993). It is not likely, however, to represent an ontogenetically more primitive brain state, as Cowan suggests. Evidence indicates, instead, that the withdrawal of brain stem and thalamocortical regulatory influences associated with this state leads to a condition of relative cortical disinhibition, which could indeed facilitate "suppressed" associations. Nevertheless, it should be understood that resulting therapeutic effects are not achieved through a learned control of neural mechanism but rather are facilitated by the favoring of a unique physiological state. While EEG-based feedback may, in fact, be the most efficient method for achieving this unique state, it is hoped that continued, systematic research will provide further clarification of the active process in this interesting area of clinical application.

CONCLUSIONS

Clearly, an appreciation for the neurophysiological and functional bases of EEG characteristics provides a more grounded perspective for interpretation of findings, and a critical guide for the evolution of both methods and concepts. There are many ways in which naivete about the origins and implications of EEG signals can lead to serious errors of conception. The EEG is a powerful physiological tool, which is being investigated by many laboratories around the world. Our understanding of the basic mechanisms underlying its expression and their significance to normal and abnormal brain functions is therefore expanding rapidly. There is no need to use it mindlessly as a clinical "electrophrenology."

Prevailing evidence indicates that EEG rhythmic patterns of interest to the self-regulation community are generated within thalamocortical circuits, which in turn are modulated by influences related to motor behavior, attentional and affective states, and cognitive priorities. We have proposed here that during wakefulness nervous system organization and specific thalamocortical generator mechanisms direct this modulation and account for

distinctive topographic and frequency manifestations. With loss of alertness and progression toward sleep, graded processes may act more generally on these mechanisms and produce topographically diffuse changes. Neuropathology can alter these processes in numerous ways, but these changes can now be more clearly interpreted in terms of underlying disturbances. This, in turn, provides a rationale for remediation with EEG feedback training. This training, however, must be guided by appropriate strategies for EEG normalization, which growing evidence indicates can indeed affect underlying mechanisms.

As our colleagues in the neurosciences continue to clarify EEG substrates and functional implications, the picture will no doubt become clearer and more complex. We must, however, continue to incorporate this knowledge into new research efforts that exploit this clarification. We must also use this knowledge as a guide to our clinical efforts so as to maximize efficacy and avoid unsupported and nonproductive distractions. It is our responsibility, further, to share valid and reliable research and clinical observations with these colleagues in order to help them avoid this same dilemma.

REFERENCES

Andersen, P., & Andersson, S. A. (1968). *Physiological basis of the alpha rhythm*. New York: Appleton-Century-Crofts.

Babb, M. I., & Chase, M. H. (1974). Masseteric and digastric reflex activity during conditioned sensorimotor rhythm. *Electroencephalography and Clinical Neurophysiology, 36*, 357-365.

Baldy-Moulinier, M. (1982). Temporal lobe epilepsy and sleep organization. In M. B. Sterman, M. N. Shouse, & P. Passouant (Eds.), *Sleep and epilepsy* (pp. 347-359). New York: Academic.

Berger, H. (1930). On the electroencephalogram of man, II. *Journal for Psychology and Neurology, 40*, 160-179.

Berlyne, D. E. (1971). *Aesthetics and psychobiology*. New York: Appleton-Century-Crofts.

Boff, K. R., Kaufman, L., & Thomas, J. P. (1986). *Handbook of perception and human performance: Vol. II, Cognitive processes and performance*. New York: Wiley-Interscience.

Bouyer, J. J., Dedet, L., Debray, O., & Rougeul, A. (1978). Restraint in primate chair may cause unusual behavior in baboons; Electrocorticographic correlates and corrective effects of diazepam. *Electroencephalography and Clinical Neurophysiology, 44*, 562-567.

Bouyer, J. J., Dedet, L., Konya, A., & Rougeul, A. (1974). Convergence de trois systemes rhythmiques thalamocorticaux sur l'aire somesthesque du chat et du babouin normaux. *Review EEG Neurophysiologie, 4*, 397-406.

Bowersox, S. S., Siegel, J. M., & Sterman, M. B. (1978). Effects of restraint on EEG variables and monomethylhydrazine seizures in the cat. *Experimental Neurology, 61*, 154-164.

Bowersox, S. S., & Sterman, M. B. (1981). Changes in sensorimotor sleep spindle activity and seizure susceptibility following somatosensory deafferentation. *Experimental Neurology, 74*, 814-828.

Bowersox, S. S., & Sterman, M. B. (1982). Effects of somatosensory deafferentation on spectral characteristics of the sensorimotor EEG in the adult cat. *Experimental Neurology, 77*, 403-418.

Carpenter, M. B. (1991). *Core text of neuroanatomy* (4th ed., Chap. 5). Baltimore: Williams & Wilkins.

Chase, M. H., & Harper, R. M. (1971). Somatomotor and visceromotor correlates of operantly conditioned 12-14 c/s sensorimotor cortical activity. *Electroencephalography and Clinical Neurophysiology, 31*, 85-92.

Cowan, J. D. (1993). Alpha-theta brainwave biofeedback: The many possible theoretical reasons for its success. *Biofeedback, 21*, 11-16.

Galletti, C., Battaglini, P. P., & Fattori, P. (1993). Parietal neurons encoding spatial locations in craniotopic coordinates. *Experimental Brain Research, 96*, 221-229.

Gloor, P., Pellegrini, A., & Kostopoulos, G. K. (1979). Effects of changes in cortical excitability upon the epileptic bursts in generalized penicillin epilepsy of the cat. *Electroencephalography and Clinical Neurophysiology, 46*, 274-289.

Hamel, A. R., & Sterman, M. B. (1982). Sleep and epileptic abnormalities during sleep. In M. B. Sterman, M. N. Shouse, & P. Passouant (Eds.), *Sleep and epilepsy* (pp. 361-377). New York: Academic.

Harper, R. M., & Sterman, M. B. (1972). Subcortical unit activity during a conditioned 12-14 Hz sensorimotor EEG rhythm in the cat. *Federation Proceedings, 31*, 404.

Holcomb, V., Sterman, M. B., Goodman, S. J., & Fairchild, M. D. (1979). The immobilization response in rhesus monkeys: A behavioral and electroencephalographic study. *Experimental Neurology, 63*, 420-435.

Howe, R. C., & Sterman, M. B. (1972). Cortical-subcortical EEG correlates of suppressed motor behavior during sleep and waking in the cat. *Electroencephalography and Clinical Neurophysiology, 32*, 681-695.

Howe, R. C., & Sterman, M. B. (1973). Somatosensory system evoked potentials during waking behavior and sleep in the cat. *Electroencephalography and Clinical Neurophysiology, 34*, 605-618.

Jasper, H. H., & Penfield, W. (1949). Electrocorticograms in man: Effect of the voluntary movement upon the electrical activity of the precentral gyrus. *Archives of Psychiatry and Neurology, 183*, 163-174.

Jones, E. G. (1985). *The thalamus*, New York: Plenum.

Krosigk, M. von, Bal, T., & McCormick, D. A. (1993). Cellular mechanisms of a synchronized oscillation in the thalamus. *Science, 261*, 361-364.

Kuhlman, W. N., & Kaplan, B. J. (1979). Clinical applications of EEG feedback training. In R. J. Gatchel & K. P. Price (Eds.), *Clinical applications of biofeedback: Appraisal and status*. New York: Pergamon.

Lantz, D., & Sterman, M. B. (1988). Neuropsychological assessment of subjects with uncontrolled epilepsy: Effects of EEG feedback training. *Epilepsia, 29*, 163-171.

Llinas, R., & Jahnsen, H. (1982). Electrophysiology of mammalian thalamic neurons. *Nature, 297*, 406-408.

Lopes da Silva, F. H., Van Leirop, T. H. M. T., Schrijer, C. F. M., & Storm van Leeuwen, W. (1973). Organization of thalamic and cortical alpha rhythm: Spectra and coherences. *Electroencephalography and Clinical Neurophysiology, 35*, 627-639.

Lopes da Silva, F. H., Vos, J. E., Mooibroek, J., & Van Rotterdam, A. (1980). Relative contributions of intracortical and thalamocortical processes in the generation of alpha rhythms, revealed by partial coherence analyses. *Electroencephalography and Clinical Neurophysiology, 50*, 449-456.

Lubar, J. F., & Bahler, W. W. (1976). Behavioral management of epileptic seizures following biofeedback training of the sensorimotor rhythm. *Biofeedback and Self-Regulation, 1*, 77-104.

Lubar, J. O., & Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Biofeedback and Self-Regulation, 9*, 1-23.

Lubar, J. F., & Shouse, M. N. (1976). EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): A preliminary report. *Biofeedback and Self-Regulation, 3*, 293-306.

Mann, C. A., Lubar, J. F., Zimmerman, A. W., Miller, C. A., & Muenchen, R. A. (1992). Quantitative analysis of EEG in boys with attention-deficit-hyperactivity disorder: Controlled study with clinical implications. *Pediatric Neurology, 8*, 30-36.26.

Mann, C. A., Sterman, M. B., Suyenobu, B. Y., & Kaiser, D. A. (1995). Suppression of EEG rhythmic frequencies during somato-motor and visuo-motor behavior. *International Journal of Psychophysiology*, in press.

McCormick, D. A. (1992). Neurotransmitter actions in the thalamus and cerebral cortex and their role in neuromodulation of thalamocortical activity. *Progress in Neurobiology, 39*, 337-388.

McCormick, D. A., & Huguenard, J. R. (1992). A model of the electrophysiological properties of thalamocortical relay neurons. *Journal of Neurophysiology, 68*, 1384-1400.

Pavlov, I. P. (1927). *Conditioned reflexes*. Oxford: Oxford U.P.

Peniston, E. G., & Kulkosky, P. J. (1989). Alpha-theta brainwave training and b-endorphin levels in alcoholics. *Alcoholism: Clinical and Experimental Research, 13*, 271-279.

Peniston, E. G., & Kulkosky, P. J. (1990). Alcoholic personality and alpha-theta brainwave training. *Medical Psychotherapy, 3*, 37-55.

Pfurtscheller, G. (1981). Central beta rhythm during sensory motor activities in man. *Electroencephalography and Clinical Neurophysiology, 51*, 253-264.

Pfurtscheller, G. (1992). Event-related synchronization (ERS): An electrophysiological correlate of cortical areas at rest. *Electroencephalography and Clinical Neurophysiology, 83*, 62-69.

Pfurtscheller, G., & Aranibar, A. (1977). Event-related cortical desynchronization detected by power measurements of scale EEG. *Electroencephalography and Clinical Neurophysiology, 42* 817-826.

Pfurtscheller, G., & Klimesch, W. (1991). Event-related desynchronization during motor behavior and visual information processing. In C. H. M. Brunia, G. Mulder, & M. N. Verbaten (Eds.), *Event-related brain research* (pp. 58-65). (*EEG Suppl. 42*).

Pinault, D., & Deschenes, M. (1992). Voltage-dependent 40-Hz oscillations in rat reticular thalamic neurons in vivo. *Neuroscience, 51*, 245-258.

Ray, W., & Cole, H. (1985). EEG alpha activity reflects attentional demands, and beta activity reflects emotional and cognitive processes. *Science, 228*, 750-752.

Rosen, I., Blennow, G., Risberg, A. M., & Ingvar, D. H. (1982). Quantitative evaluation of nocturnal sleep in epileptic children. In M. B. Sterman, M. N. Shouse, & P. Passouant (Eds.), *Sleep and epilepsy*, (pp. 397-409). New York: Academic.

Roth, S. R., Sterman, M. B., & Clemente, C. C. (1967). Comparison of EEG correlates of reinforcement, internal inhibition, and sleep. *Electroencephalography and Clinical Neurophysiology, 23*, 509-520.

Rougeul, A., Bouyer, J. J., Dedet, L., & Debray, O. (1979). Fast somatoparietal rhythms during combined focalized attention and immobility in baboon and squirrel monkey. *Electroencephalography and Clinical Neurophysiology, 46*, 310-319.

Shouse, M. N., & Lubar, J. F. (1979). Operant conditioning of EEG rhythms and ritalin in the treatment of hyperkinesis. *Biofeedback and Self-Regulation, 4*, 299-312.

Shouse, M. N., Siegel, J. M., Wu, M. F., Szymusiak, R., & Morrisson, A. R. (1989). Mechanisms of seizure suppression during rapid eye movement (REM) sleep in cats. *Brain Research, 505*, 271-282.

Shouse, M. N., & Sterman, M. B. (1979). Changes in seizure susceptibility, sleep time and sleep spindles following thalamic and cerebellar lesions. *Electroencephalography and Clinical Neurophysiology, 46*, 1-12.

Sprague, J. M. (1966). Visual, acoustic and somesthetic deficits in the cat after cortical and midbrain lesions. In D. Purpura & M. Yahr (Eds.). *The thalamus* (pp. 391-414). New York: Columbia U.P.

Steriade, M., Curro Dossi, R., & Nunez, A. (1991). Network modulation of a slow intrinsic oscillation of cat thalamocortical neurons implicated in sleep delta waves: cortically induced synchronization and brainstem cholinergic suppression. *The Journal of Neuroscience, 11*, 3200-3217.

Steriade, M., & Deschenes, M. (1988). Intrathalamic and brainstem-thalamic networks involved in resting and alert states. In M. Bentivoglio & R. Spreafico (Eds.). *Cellular thalamic mechanisms* (pp. 37-62). Amsterdam: Elsevier.

Steriade, M., & Llinas, R. R. (1988). The functional states of the thalamus and the associated neuronal interplay. *Physiological Reviews, 68*, 649-742.

Steriade, M., Gloor, P., Llinas, R. R., Lopes da Silva, F. H., & Mesulam, M. M. (1990). Basic mechanisms of cerebral rhythmic activities. *Electroencephalography and Clinical Neurophysiology, 76*, 481-508.

Sterman, M. B. (1977). Effects of sensorimotor EEG feedback training on sleep and clinical manifestations of epilepsy. In J. Beatty and H. Legewie (Eds.), *Biofeedback and behavior* (pp. 167-200). New York: Plenum.

Sterman, M. B. (1981). Power spectral analysis of EEG characteristics during sleep in epileptics. *Epilepsia, 22*, 95-106.

Sterman, M. B. (1982). EEG biofeedback in the treatment of epilepsy: An overview circa 1980. In L. White & B. Tursky (Eds.), *Clinical biofeedback: Efficacy and mechanisms* (pp. 311-330). New York: Guilford.

Sterman, M. B. (1986). Epilepsy and its treatment with EEG feedback therapy. *Annals of Behavioral Medicine, 8*, 21-25.

Sterman, M. B. (1993). Sensorimotor EEG feedback training in the study and treatment of epilepsy. In D. I. Mostofsky & Y. Loyning (Eds.). *Neurobehavioral treatment of epilepsy* (pp. 1-17). New Jersey: Erlbaum.

Sterman, M. B., & Bowersox, S. S. (1981). Sensorimotor electroencephalograph rhythmic activity: A functional gate mechanism. *Sleep, 4*, 408-422.

Sterman, M. B., & Friar, L. (1972). Suppression of seizures in an epileptic following sensorimotor EEG feedback training. *Electroencephalography and Clinical Neurophysiology, 33*, 89-95.

Sterman, M. B., Goodman, S. J., & Kovalesky, R. A. (1978). Effects of sensorimotor EEG feedback training on seizure susceptibility in the rhesus monkey. *Experimental Neurology, 62*, 735-747.

Sterman, M. B., Howe, R. D., & MacDonald, L. R. (1970). Facilitation of spindle-burst sleep by conditioning of electroencephalographic activity while awake. *Science, 167*, 1146-1148.

Sterman, M. B., Kaiser, D. A., & Veigel, B. (1995). Event-related EEG spectra responses during the Continuous Performance Test. *Electroencephalography and Clinical Neurophysiology*, submitted.

Sterman, M. B., LoPresti, R. W., & Fairchild, M. D. (1969). Electroencephalographic and behavioral studies of monomethylhydrazine toxicity in the cat. Technical Report AMRL-TR-69-3, Wright-Patterson Air Force Base, Ohio, Air Systems Command.

Sterman, M. B., & Macdonald, L. R. (1978). Effects of central cortical EEG feedback training on incidence of poorly controlled seizures. *Epilepsia, 19*, 207-222.

Sterman, M. B., MacDonald, L. R., & Stone, R. K. (1974). Biofeedback training of the sensorimotor EEG rhythm in man: Effects on epilepsy. *Epilepsia, 15*, 395-417.

Sterman, M. B., & Mann, C. A. (1995). Concepts and applications of EEG analysis in aviation performance evaluation. *Biological Psychology, 40*, 115-130.

Sterman, M. B., Mann, C. A., Kaiser, D. A., & Suyenobu, B. Y. (1994). Multiband topographic EEG analysis of a simulated visuomotor aviation task. *International Journal of Psychophysiology, 16*, 49-56.

Sterman, M. B., & Shouse, M. N. (1980). Quantitative analysis of training, sleep EEG, and clinical response to EEG operant conditioning in epileptics. *Electroencephalography and Clinical Neurophysiology, 49*, 558-576.

Sterman, M. B., & Wyrwicka, W. (1967). EEG correlates of sleep: Evidence for separate forebrain substrates. *Brain Research, 6*, 143-163.

Sterman, M. B., Wyrwicka, W., & Roth, S. R. (1969). Electrophysiological correlates and neural substrates of alimentary behavior in the cat. *Annals of the New York Academy of Science, 157*, 723-739.

Stone, L. S., & Lisberger, S. G. (1990). Visual responses of purkinje cells in the cerebellar flocculus during smooth-pursuit eye movements in monkeys: II. Complex Spikes. *Journal of Neurophysiology, 63,* 1262-1275.

Tansey, M. A. (1985). Brainwave Signatures. An index reflective of the brain's functional neuroanatomy: Further findings on the effect of EEG sensorimotor rhythm feedback training on the neurologic precursors of learning disabilities. *International Journal of Psychophysiology, 4,* 91-97.

Veigel, B., & Sterman, M. B. (1993). Topographic EEG correlates of good and poor performance in a signal recognition task. *Proceedings of the Human Factors Society 37th Annual Meeting, 1,* 147-151.

Wickramasekera, I. (1993). Observations, speculations and an experimentally testable hypothesis on the mechanism of the presumed efficacy of the Peniston and Kulkosky procedure. *Biofeedback, 21,* 17-20.

Warren, R. A., & Jones, E. G. (1994). Glutamate activation of cat thalamic reticular nucleus: Effects on response properties of ventroposterior neurons. *Experimental Brain Research, 100,* 215-226.

Wood, J. D., & Peesker, S. J. (1974). Development of an expression which relates the excitable state of the brain to the level of GAD activity and GABA content, with particular reference to the action of hydrazine and its derivatives. *Journal of Neurochemistry, 23,* 703-712.

Wyler, A. R. (1984). Operant conditioning of single neurons in monkeys and its theoretical application to EEG operant conditioning in human epilepsy. In Th. Elbert, B. Rockstroh, W. Lutzenberger, & N. Birbaumer (Eds.). *Self-regulation of the brain and behavior* (pp. 85-94). New York: Springer-Verlag.

Wyrwicka, W., & Sterman, M. B. (1968). Instrumental conditioning of sensorimotor cortex EEG spindles in the waking cat. *Physiology and Behavior, 3,* 703-707.