

## Neurofeedback treatment of epilepsy

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With electroencephalographic (EEG) biofeedback (or neurofeedback), it is possible to train the brain to de-emphasize rhythms that lead to generation and propagation of seizure and emphasize rhythms that make seizures less likely to occur. With recent improvements in quantitative EEG (qEEG) measurement and improved neurofeedback protocols, it has become possible in clinical practice to eliminate seizures or reduce the amount of medication required to control them.

The use of neurofeedback to treat human epilepsy dates back to the early 1970s [1]. Subsequent studies (without qEEG guidance) showed that seizure frequency could be reduced in most patients who were trained to decrease slow frequencies in the 3- to 8-Hz range and reward frequencies in the 9- to 18-Hz range. Complete abolition of seizures was rare, however.

With the development of sophisticated qEEG databases it has become possible to more precisely characterize power and coherence abnormalities associated with drug-resistant epilepsy. If there is focal excessive power in a frequency band, it may be downtrained. If there is a focal deficiency in power, it may be uptrained. Similarly, significantly decreased coherence between brain areas may be uptrained and significantly increased coherences may be downtrained. This approach has been found in clinical experience to decrease or abolish seizures in all patients appropriately trained. Many even become medication free.

In this article, the history of neurofeedback for epilepsy is presented, followed by discussions of the relevant neurophysiology of epilepsy. A model of how

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Neither author has a relationship with a commercial company that has a direct financial interest in the subject matter or materials discussed in this article or with a company that has a competing product.

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neurofeedback might raise the seizure threshold is then presented. Clinical experience using a qEEG-guided approach is described, including a representative case study.

### **Critical terms and the process of encephalographic biofeedback**

The EEG signal is characterized based on the number of times the waveform seen on the oscilloscope goes from one peak to the next in a second. The entire range of frequencies, measured in cycles per second or hertz (Hz), is conventionally subdivided into four standard bands. Although the exact cut-off points differ somewhat from one laboratory to the next, the four commonly used EEG frequency bands used include (1) delta, 1.5 to 3.5 Hz, (2) theta, 3.5 to 7.5 Hz, (3) alpha, 7.5 to 12.5 Hz, and (4) beta, 12.5 to 25 Hz. Total power represents the frequency range of 1.5 to 25 Hz. The range from 12 to 15 Hz also is sometimes referred to as sensorimotor rhythm (SMR). Some important technical terms in the discussion below are defined as follows:

- **Magnitude:** the average strength in absolute microvolts of the signal of a band during an epoch
- **Power:** the square of the microvolts of a frequency during an epoch
- **Relative power:** the microvolts of the particular band divided by the total microvolts generated by all bands at a location
- **Peak amplitude:** the peak value in microvolts of a frequency band during an epoch
- **Peak frequency:** the highest frequency obtained during an epoch within a frequency range
- **Coherence:** the average amplitude similarity between the waveforms of a particular band in two locations over an epoch

The process of neurofeedback can be conceived as the operant conditioning of the EEG signal. Electrodes are applied to measure EEG activity. The signal is then computer analyzed based on the particular variable or variables being trained and then is “fed back” in real time to the trainee in a form that is more easily understood, along with a series of hints or signals (often called “rewards”) that are provided as the signal changes in the direction being shaped. For example, in more traditional amplitude training, the visual feedback might take the form of a race among three spaceships. Each spaceship is “powered” by a band of EEG activity selected by the clinician. If the goal of the training is to reduce theta and increase SMR activity, real-time variation in the amplitude of theta is reflected in changes in the “speed” and “power meter” of one of the spaceships, whereas real-time variation in the SMR range is reflected by another ship. Beeps are given and points accrued for every interval in which the theta amplitudes remain below and SMR amplitudes remain above thresholds set by the clinician. The beeps and

points are seen to “reward” the emission of the wanted EEG response and are used to shape the ongoing EEG activity within gradually modified parameters, much as emergent overt behavior is continuously shaped over time. Even young children are able to understand the goal of making the designated spaceship win the race and experience the beeps and points as exciting and rewarding. The same sort of process can be used to train other EEG variables, such as coherence, phase, or symmetry.

### **Efficacy research on neurofeedback for epilepsy**

The value of neurofeedback for human epilepsy was first established by Stermann and his colleagues in 1974 [1]. Twelve to 16 Hz activity (SMR) at 20 to 25  $\mu$ V was rewarded over central and frontal areas in four medically refractory patients during three sessions per week for 6 to 18 months. All four patients exhibited a reduction in seizure frequency during SMR training over 0.5 to 1.5 years. A rebound in seizure frequency occurred after cessation of training in three patients. Finley et al [2] found that training to increase SMR was associated with reduced seizure frequency and normalization of the EEG in a patient with severely epilepsy. In an important paper, Lubar et al [3] performed a within-subjects ABA condition-reversal study of neurofeedback for uncontrolled epilepsy. They found that training to inhibit 3- to 8-Hz activity and increase 11- to 19- or 12- to 15-Hz activity was associated with a decrease in seizure frequency. During the condition reversal phase, subjects were trained to increase 3- to 8-Hz activity and decrease SMR. This training occasioned the predicted increase in seizure activity, which indicated the specificity of the training effect with regard to seizure suppression. Seizure frequency was reduced again once the theta inhibit/SMR reward condition was reinstated.

Two other studies with a similar ABA design obtained similar results [4,5]. Wyler et al [6] rewarded desynchronization (increase in fast activity, decrease in slow activity) that was effective in reducing seizure frequency in several medically intractable patients. Ayers [7] reported successful treatment of absence epilepsy by training patients to inhibit 4- to 7-Hz activity and reward 15- to 18-Hz activity using bipolar training at T3/C3 and T4/C4. Ten patients became and remained seizure free for 10 years. In another study, noncontingent training had no effect on seizures, but seizure reduction was observed with 9- to 14-Hz or 14- to 26-Hz training (Kuhlman [8]). Later, Lantz and Stermann [9] showed that contingent training was superior to noncontingent training in 24 patients using a yoked, wait-list control design.

Stermann reviewed all of the literature up to 2000 and found that every study of neurofeedback for epilepsy reported positive results [1]. In his meta-analysis, 82% of patients demonstrated more than 30% reduction in seizures, with an average more than 50% reduction. Approximately 5% remained seizure free for up to a year. There has been no systematic study to determine which precise

frequencies should be trained to maximize the reduction in seizures. Several other studies not reviewed by Sterman in 2000 also support the effectiveness of neurofeedback in reducing seizure frequency in poorly controlled epilepsy [10–14].

In recent years, a different approach to normalize the EEG of persons with epilepsy by using qEEG to guide neurofeedback training has been used in clinical work by Walker and colleagues [15]. The general approach is to determine the most significant abnormalities and train those areas to normal. Power training alone resulted in significant seizure reductions but rarely resulted in cessation of seizures. Based on the successful use of coherence training in closed head injury [15], these protocols were used to normalize coherence in patients with medically refractory epilepsy. The combination of power and coherence training to normalize the EEG has proved dramatically effective in several cases [16]. Recent advances in qEEG, notably reliable databases that include single-Hertz bins [17,18] and broadly based coherence determinations, have improved diagnostic specificity.

Several studies have examined qEEG abnormalities in patients with epilepsy [19–21]. Traditional broad-band spectral analyses generally find focal increases in relative power of theta, with decreased values in the alpha and delta bands when compared with normative data. Diaz et al [22] found a positive correlation between the magnitude of the quantitative abnormalities and the amount of paroxysmal activity. There was a slowing of mean frequency of alpha in the group with epilepsy. They found that focal epilepsy may be associated with widespread changes in the frequency spectra. In clinical experience, widespread abnormalities in coherence and phase, often remote from the spike focus, have been observed. No published studies of coherence abnormalities in patients with epilepsy exist.

Another important issue relates to the transition from interictal to ictal EEG [23]. Recent clinical experience has suggested that it may be an important goal of neurofeedback to prevent that transition by downtraining the rhythms that are found to initiate the seizure. This proved to be the key to eliminating frequent episodes of status epilepticus in one patient, whose seizures invariably began with rhythmic theta activity in one or the other temporal lobe [16]. Long-term monitoring may be required to determine the rhythms responsible for the transition from interictal to ictal activity.

### **The neurophysiology of epilepsy and possible mechanisms for reducing seizures in epilepsy**

The human brain must be stable and flexible to operate efficiently [24]. By changing the amounts of excitation and stimulation in simulated neural networks, one can induce synchronous bursting behavior of large neural networks (the hallmark of epilepsy) [25]. In an animal model of epilepsy (low magnesium), Nyikos et al [26] have shown that recurrent seizure-like events in brain slices are

initially characterized by high amplitude electrical triplets (paroxysmal spikes [6]) with a single rhythm that starts at 200 Hz and continuously declines to below 1 Hz at termination of the seizure-like event. Other *in vitro* models demonstrate abnormalities, such as high-frequency bursts [27] and rapid oscillations (100–300 Hz) [28], which lead to seizure activity. These abnormalities are usually undetectable in conventional EEG recordings because of muscle artifact and remoteness from surface electrodes.

Medvedev [29] has observed that intense gamma activity (40–60 Hz) often precedes epileptic discharges in patients and in some animal models. He analyzed power, coherence, and phase in the kainic acid model in the rat hippocampus and neocortex. At onset of discharges, highly coherent, intense gamma rhythms were followed by a slow rhythm of epileptiform spikes and sharp waves. During spike activity and immediately afterwards, the gamma power and coherence were significantly decreased. He hypothesized that epileptiform spike activity may result from extreme activation of the “anti-binding” mechanism that controls “temporal binding” at high frequencies (ie, the “epileptiform” discharge develops as a protective mechanism to suppress fast activity). “Temporal binding” is a theoretical model for how the brain encodes multiple facets of the same event or object in time in contrast to classic neural network models of “connectivity.” “Anti-binding” is a necessary activity to prevent “global synchrony” of these events that would lead to seizure activity. Fisher et al [30] found an increase in 40-Hz activity just before epileptiform discharges in patients with complex partial seizures. Medvedev [29] suggests that postictal depression of the EEG may represent a selective decrease in coherence at high frequencies rather than a nonspecific suppression of all frequencies. It may be that fast activity is desynchronized and suppressed by spike activity. Previously, Engel et al [31] suggested that interictal spikes may inhibit seizure activity in kindling models of epilepsy. Other frequency-specific abnormalities may include a decrease in beta frequencies over well-defined epileptic spike foci [19].

Another way to view the pathophysiology of epilepsy is to consider seizures as an example of a disturbance in excitatory and inhibitory input to the seizure focus [32,33]. The simplest models propose that the generation of seizures results from the disruption of the normal balance of inhibitory and excitatory input. An excess of excitatory input (eg, glutamergic) connections or a deficiency in inhibitory connections (eg, GABA-ergic) may be present. Drugs that increase glutamergic activity typically produce seizures, whereas drugs that enhance GABA-ergic activity typically are antiepileptic. It is not clear, however, whether human epilepsy is produced in this way. Slices of brain taken from seizure foci in children who underwent surgical treatment for epilepsy appeared to be qualitatively normal in terms of synaptic transmission and local neurologic circuits [34]. Often, ictal recordings show that seizure onset is remote from the spike focus [35].

Stevens [36] has suggested that the interictal spikes must have survival value to have persisted through evolutionary history. Spikes are normal when confined to certain axial nuclei of the brain during sleep (K-complexes) or during photic or

Table 1  
Epilepsy patients

Patient	Age	Type seizure	Pre-neurofeedback seizure frequency	On medication	Protocols	# sessions	Result
#1 – M	62	PCS, 2 <sup>o</sup> gen R anterior temporal Simple partial L parietal	3/wk	Dilantin, 300 mg/d neurontin, 3600 mg/d	1. Downtrain 1–8 Hz R frontal 2. Downtrain 27–30 Hz R frontal 3. Uptrain coherence alpha L midtemporal/L frontal	5 5 10	Class II
#2 – F	43	PCS (brief) (no focus)	5/d (average)	Dilantin, 300 mg/d	1. Downtrain 2–7 Hz/uptrain 15–18 Hz L anterior temporal	20	Class I
#3 – F	19	PCS (no focus)	3/wk	Lamictal, 50 mg twice daily tegretol, 400 mg twice daily	1. Uptrain coherence theta L frontal/L occipital 2. Downtrain coherence theta L prefrontal/L frontal 3. Downtrain coherence beta R central/R parietal 4. Downtrain coherence alpha L prefrontal/L frontal 5. Uptrain coherence beta L occipital/R occipital	5 5 5 5 10	Class II Class II Class II Class II Class II
#4 – M	31	PCS, status epilepticus (no focus)	6/d 4/mo		1. Uptrain coherence alpha L occipital/R occipital 2. Uptrain coherence theta posterior temporal/R posterior temporal 3. Downtrain 4–7 Hz/uptrain 12–15 Hz R central 4. Downtrain 4–7 Hz/uptrain 15–18 Hz midcentral 5. Downtrain 4–7 Hz/uptrain 15–18 Hz L central 6. Downtrain coherence 12–15 Hz R central/midparietal	9 10 5 10 10 10	Class II

#5 – F	PCS (No focus)	1/d	Tegretol, 400 mg twice daily neurotin, 300 mg three times daily	<ol style="list-style-type: none"> <li>1. Uptrain coherence theta L occipital/R occipital</li> <li>2. Uptrain coherence theta left anterior temporal/R anterior</li> <li>3. Downtrain 2–7 Hz/uptrain 15–18 Hz L temporal central</li> <li>4. Downtrain 22–30 Hz L central</li> <li>5. Downtrain 4–7 Hz midfrontal</li> </ol>	5 5 5 10 2 10	Class III No seizures but “drop attack” 1/wk Class II
#6 – F	5 PCS: multifocal spikes	1/wk	Lamictal 25 mg three times daily	<ol style="list-style-type: none"> <li>1. Downtrain 4–8/ uptrain 15–18 R anterior temporal</li> <li>2. Downtrain 4–8/uptrain 15–18 R parietal</li> <li>3. Uptrain coherence delta L parietal/L occipital</li> <li>4. Downtrain coherence theta L prefrontal/L midtemporal</li> </ol>	6 9 3 T = 28 10 7 T = 17	Class I Class II
#7 – F	6 PCS: Right occipital spike focus	5/d	Primidone 125 mg/d	<ol style="list-style-type: none"> <li>1. Downtrain 27/uptrain 15–18 Hz L occipital</li> <li>2. Downtrain 27/uptrain 15–18 Hz R occipital</li> </ol>	7 T = 17	Class II
#8 – M	19 PCS with 2° generalization sharp waves: L occipital/L prefrontal biparietal	5/mo	Lamictal 350 mg/d, topamax 200 mg/d	<ol style="list-style-type: none"> <li>1. Downtrain 1–7 Hz R occipital</li> <li>2. Uptrain coherence delta L frontal/ R central</li> <li>3. Uptrain coherence theta L anterior temporal/midcentral</li> <li>4. Uptrain coherence alpha L prefrontal/L frontal</li> <li>5. Downtrain coherence beta R central/R posterior temporal</li> <li>6. Downtrain 2–7 Hz/uptrain 15–18 Hz L posterior temporal</li> <li>7. Downtrain 1–6 Hz/uptrain 15–18 Hz L prefrontal</li> <li>8. Uptrain coherence delta L anterior temporal/R prefrontal</li> </ol>	5 5 5 5 5 5 5 5	

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Table 1 (continued)

Patient	Age	Type seizure	Pre-neurofeedback seizure frequency	On medication	Protocols	# sessions	Result							
#9 – M	35	PCS with 2° gen: no spike foci	1/wk	Tegretol, 300 mg three times daily	9. Uptrain coherence beta L frontal/ L prefrontal	T = 82	Class II							
					10. Downtrain 1–4 Hz L prefrontal									
					11. Uptrain coherence delta L central/L anterior temporal									
					12. Uptrain coherence alpha R anterior temporal/R occipital									
					13. Downtrain coherence theta R anterior temporal/R occipital									
					14. Uptrain coherence alpha L frontal/midfrontal									
					15. Downtrain 2–7 Hz L prefrontal									
					16. Downtrain 2–7 Hz R posterior temporal									
					1. Uptrain alpha coherence L frontal/ L occipital									
					2. Uptrain alpha coherence R frontal/R occipital									
					3. Uptrain alpha coherence R frontal/R occipital									
					4. Uptrain beta coherence L midtemporal/ L posterior temporal									
					5. Uptrain 12–15 Hz midcentral									
					#10 – M			9	PCS with 2° gen: no spike foci	1/wk	Depakote, 500 mg twice daily	1. Downtrain 2–7 Hz L prefrontal/L frontal	T = 41	Class II
												2. Downtrain 2–7 Hz R prefrontal/R frontal		
												3. Downtrain 2–7 Hz L prefrontal/L frontal		
4. Downtrain 2–7 Hz R anterior temporal														
							T = 25							

Abbreviations: Class I, seizure free off medications for 3 months; Class II, seizure free on medications for 3 months; Class III, occasional seizure (<1/mo) for 3 months; Class IV, more than one seizure per month (failure) (none seen); L, left; PCS, partial complex seizures; 2° gen, secondary generalization; R, right.



sexual excitation and may be compensatory after deafferentation or hypoxia. In epilepsy surgery, it is usually assumed that the structural and biochemical pathology is confined to the area in and around the spike focus, based on the fact that seizures often stop when the focus is removed. There is evidence from animal models and humans that in many cases the behavioral abnormalities responsible for the epileptic activity are remote from the focus. When a freezing lesion is made in the right posterior cortex of the rat, the spike focus is likely to be in the anterior cortex on the same or the other side. Slowing is seen over the site of the biochemical lesion. Application of tetanus toxin to one hemisphere is associated with seizure development in the contralateral hemisphere [37].

Lieb et al [38] examined propagation of ictal discharge in patients with complex partial seizures of mesial temporal origin using depth electrodes in mesial temporal, lateral temporal, and frontal lobes. During seizure onset there was a marked increase in intrahemispheric coherence, which indicated that areas remote from the focus participate in the initiation of focal seizures. Steriade et al [39] have reviewed a body of evidence that supports the importance of thalamocortical synchronization in the genesis of epileptic rhythms. Bartolomei et al [40] implanted temporal lobe depth electrodes in patients with drug-resistant epilepsy and described four different seizure types. One type begins in the medial cortex with phasic discharges spreading to neocortex and high coherence between medial temporal cortex and neocortex. A second group had seizures that started in medial cortex and spread to lateral cortex with a fast low-voltage discharge, which spread rapidly to neocortex and high coherence between medial and lateral structures. A third group exhibits fast low-voltage discharge that starts in lateral neocortex and spreads rapidly to hippocampus and amygdale, with high coherence between these structures. Interhemispheric connections probably play a less important role than intrahemispheric connections, even when the seizures spread to the opposite hemisphere [41].

Collectively, these studies suggest that a technique, such as neurofeedback, which can normalize coherence, could likely reduce propagation of seizures from the site of onset. Chavez et al [42] recently described decreased synchrony at 10 to 25 Hz in the seizure onset zone 30 minutes before seizures in two patients with neocortical partial epilepsy. Neurofeedback training of coherence may prevent transient synchrony changes that result in seizures.

### **Thoughts on how neurofeedback might lower seizure threshold in epileptic patients**

There have been a few efforts to develop a model of how neurofeedback works. Lubar has proposed an excellent model for how neurofeedback enhances attentional capabilities [43]. This is easily adapted for explaining how neurofeedback might work for epilepsy. A more detailed model, which could be adapted to include neurotransmitter influences, may be found in the Hughes and John paper [44]. For our purposes, it is sufficient to say that our goal is to produce

optimal coherence and phase synchronization, in order to raise seizure threshold and reduce the likelihood of having a seizure. Lubar et al [3] have pointed out that neurofeedback training produces very long-lasting and perhaps permanent, learning. Thus, one literally learns not to have seizures.

Obviously, one would not want to diffusely decrease gamma or delta/theta activity, but rather train the epileptic foci or connections relevant to the seizure, since delta, theta and gamma are important for normal sleep and learning.

### **Results of single case trials of quantitative electroencephalographic-guided power and coherence training**

The approach taken by Walker and colleagues in clinical trials is to “train away” abnormalities of power and coherence in an attempt to decrease and hopefully abolish seizure likelihood. Generally, the most statistically abnormal power or coherence abnormality is treated first, followed by successively less severe abnormalities, targeted in five to ten sessions for each. Details of this methodology may be found in a paper on the use of this intervention for closed head injury [15]. Table 1 indicates details for each patient, including the spike focus (if one was found), training protocols, and degree of success. These results seem to be superior to previously published results regarding neurofeedback for partial complex seizures, because all patients became seizure free and many were able to stop their anticonvulsant treatment.

### **Case examples**

A 6-year-old girl was sitting in a chair, tipped it back, and fell on the back of her head. She did not lose consciousness but had a headache for a day. Approximately 1 month later she began to “zone out” at home and at school. Her breathing slowed and she stared into space. Most spells lasted 5 to 10 minutes. She was unresponsive during the spells. She did not blink or have other facial movements. She was taken to the emergency room on two occasions when she did pass out. When she came to in the emergency room, she gasped for air. When she came to our clinic, she was having 20 to 40 spells per day. She had been a straight-A student before the spells began but was unable to attend school or do her work because of the frequent, prolonged spells. An EEG revealed occasional slow sharp wave discharges that originated in the right occipital region. A qEEG revealed an increase in absolute theta over most of the left hemisphere and in the right occipital region. There was an increase in the relative power of delta over the entire scalp. There was a decrease in the relative power of alpha at the O2 qEEG lead position. Interhemispheric coherence theta was increased at FP1/FP2 and C3/C4 leads. Intrahemispheric coherence of theta was increased at T3/T5, F3/O1, and F4/O2. She was placed on primidone, 250 mg, at 6 PM every night. She stopped zoning out and was able to do her school work and was more

attentive, but she became depressed. The primidone was decreased to 125 mg once a day, and she began to have occasional spells again. She began neurofeedback at that point. She had ten sessions to downtrain 2 to 7 Hz and uptrain 15 to 18 Hz at O1, followed by seven sessions to downtrain 2 to 7 Hz and uptrain 15 to 18 Hz at O2. The spells stopped and the primidone was discontinued. She has had no further seizures and is doing well in school (9-month follow-up so far).

A 31-year-old man had recurrent partial complex and secondarily generalized seizures and frequent episodes of status epilepticus. Treatment with nine different anticonvulsants singly and in combination did not control his seizures or prevent status episodes. He was not a surgical candidate because depth electrode monitoring revealed independent foci of onset in the left and right temporal lobes. A vagal nerve stimulator was implanted and did not reduce his seizures or prevent status episodes.

A qEEG revealed an increase in absolute theta at F7 and T4 and increased relative theta at FP1, FP2, F7, and F8. Decreased coherence of theta was found at T3/T5, O1/O2, and C3/C4. Decreased beta coherence was found at O1/O2.

Neurofeedback training was performed three times per week. First, theta coherence was downtrained at T3/T5 (ten sessions), then O1/O2 theta coherence was downtrained (five sessions). Next, beta coherence was downtrained at O1/O2. Then theta coherence was downtrained at C3/C4. Finally, theta was downtrained and SMR (12–15 Hz) was uptrained at T3.

After completion of the first protocol, there were no further episodes of status epilepticus. Generalized seizures decreased to one per week after the first protocol, and none occurred after the second protocol was complete. Partial complex seizures decreased to ten per week after the first protocol, five per week after the second protocol, and one per week after the third protocol. When an increase in seizure frequency was noted with the fourth protocol, it was discontinued and the fifth protocol was completed. No more seizures occurred. The patient returned to work full time and began driving again. Shortly afterward, he discontinued phenytoin. His speech and memory improved. He chose to continue his other medication, not wanting to risk a seizure while driving. He completed graduate school and became an independent businessman.

These cases illustrate the clinical potential of qEEG-guided neurofeedback in the management of severe epilepsy. The training is noninvasive and effective in a relatively short time. The cost is low in comparison with epilepsy surgery or vagal nerve stimulation. It should prove to be particularly important for treating patients with drug-resistant epilepsy. Finally, they suggest that the combination of coherence training with traditional power training may be more effective than either approach alone.

### **Possible future directions**

Recent work suggests that feedback of slow direct current (DC) potentials could be effective in reducing seizure frequency. Future magnetoencephalo-

graphic machines will be able to record simultaneously from both hemispheres to better localize deep epileptogenic zones and determine where training is most likely to be effective [45]. LORETA neurofeedback, real-time feedback based on the low-resolution electromagnetic tomography (LORETA) EEG source localization technique, may be used to train some deep foci [46]. EEG-linked functional MRI has been used to image single interictal discharges and could be used to image sites of seizure onset. LORETA training might be used to train at the focus rather than at the surface. Modern spectral analysis techniques allow high resolution in the higher beta range (>18 Hz) [47]. There have been few studies of higher beta in epilepsy or neurofeedback, and it seems that higher beta may be important in both fields [48]. The availability of implantable subdural electrode arrays has made systematic studies of electrocorticographic coherence possible [49]. They could enable more precise coherence training, which might spare the need for brain resection. Nonlinear dynamic approaches to studying epilepsy recently have come to the fore (eg, chaos theory) [50]. Newer feedback systems that use nonlinear approaches have been developed [51]. Finally, downtraining of gamma activity over the spike focus may be an effective way of inhibiting seizure activity with the newer biofeedback systems [52].

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