Meta-Analysis of EEG Biofeedback in Treating Epilepsy

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Key Words
EEG Biofeedback
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ABSTRACT

About one third of patients with epilepsy do not benefit from medical treatment. For these patients electroencephalographic (EEG) biofeedback is a viable alternative. EEG biofeedback, or neurofeedback, normalizes or enhances EEG activity by means of operant conditioning. While dozens of scientific reports have been published on neurofeedback for seizure disorder, most have been case series with too few subjects to establish efficacy. The purpose of this paper is to meta-analyze existing research on neurofeedback and epilepsy.

We analyzed every EEG biofeedback study indexed in MedLine, PsychInfo, and PsychLit databases between 1970 and 2005 on epilepsy that provided seizure frequency change in response to feedback. Sixty-three studies have been published, 10 of which provided enough outcome information to be included in a meta-analysis. All studies consisted of patients whose seizures were not controlled by medical therapies, which is a very important factor to keep in mind when interpreting the results. Nine of 10 studies reinforced sensorimotor rhythms (SMR) while 1 study trained slow cortical potentials (SCP). All studies reported an overall mean decreased seizure incidence following treatment and 64 out of 87 patients (74%) reported fewer weekly seizures in response to EEG biofeedback. Treatment effect was mean log (post/pre) where *pre* and *post* represent number of seizures per week prior to treatment and at final evaluation, respectively. Due to prevalence of small groups, Hedges's g was computed for effect size. As sample heterogeneity was possible (Q test, p=.18), random effects were assumed and the effect of intervention was -0.233, SE= 0.057, z = -4.11, p<.001.

Based on this meta-analysis, EEG operant conditioning was found to produce a significant reduction on seizure frequency. This finding is especially noteworthy given the patient group, individuals who had been unable to control their seizures with medical treatment.

INTRODUCTION

Nearly 50 million people currently suffer from epilepsy, 0.8% of the general population, according to the World Health Organization.[1] Medication successfully controls seizures in two-thirds of cases, but potential side effects and health risks associated with long-term usage of antiepileptics remain a concern. When medications fail, neurosurgery is another treatment option, but it has limited success.[2] All told, about one in three epilepsy patients will continue to experience the disability of uncontrollable seizures throughout their lifetime.[3]

The use of neurofeedback to reduce intractable seizures has been under serious investigation for 40 years, beginning with cats,[4,5] monkeys,[6] and humans.[7] Although EEG rhythm training has been associated with clinical improvement as well as electroencephalographic (EEG) normalization of seizure patients (review, see[8]), few neurologists and epileptologists have adopted this approach to help treat seizure disorder.

Research in the United States has emphasized sensorimotor rhythm (SMR) up-training (i.e., increasing 12-15 Hz activity at motor strip)[8-10] with or without simultaneous down-training of slow rhythms (e.g., decreasing 4-7 Hz activity).[11-14] Research in Europe, on the other hand, has focused principally on slow cortical potentials (SCP), which last several hundred milliseconds and reflect the level of excitability of underlying cortex. Negative SCP-shifts are observed before and during seizures and positive shifts appear after their abatement.[15] To prevent seizure onset, patients learn to suppress negativity (excitation) by producing positive shifts (inhibition).

The purpose of the current article is to provide quantitative integration of controlled research on the neurofeedback treatment of uncontrolled epilepsy. Recent review papers already exist, including theoretical considerations on its efficacy.[8,16,17]

The Efficacy Task Force of the Association for Applied Psychophysiology and Biofeedback and the International Society for Neurofeedback and Research propose criteria for evaluating evidence of efficacy. To be considered efficacious, a treatment must be proven superior or equivalent to a control group using a randomized design with sufficient power to detect differences, a population clearly identified through operational definition, involve valid outcome measures, and independent replication of effect.[18] This standard would have to be amended to include studies like those in this meta-analysis which were of necessity limited to small sample sizes and only a single group for which pre- and post-treatment effects are determined.

Despite these limitations, results have been consistent across studies, generally suggesting that neurofeedback leads to reduction in seizures. For example, one study found that over 80% of 83 patients achieved control over seizures when a combination of interventions

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Table 1

Study details

Study	Research Design*	NF Protocol	Length of Treatment	Outcome Measures	Seizure Type	Age Range	Gender	Dropouts
Sterman et al. (1974)	ABA; ABAB	SMR (11-15 Hz)	6 to 18 months (3 sessions/wk)	seizure frequency; EEG	varied	6 to 46	4; not specified	0
Kaplan (1975)	ABA; ABCA	SMR	20 to 25 weeks (3 sessions/wk)	seizures per day; EEG	varied	20 to 30 3 female	1 male;	0
Lubar et al. (1976)	within subject: AB, ABA, ABAB	SMR	80-260 days (3 session/wk)	seizures; SMR criteria met	varied	12 to 29	4 male; 4 female	0
Kuhlman et al., 1978	within subject: AB (AB)	SMR (9-14 Hz)	A:9 sessions sham B:24 sessions feedback (repeated if necessary)	seizures; EEG	4 partial 1 generalized, myoclonic	17 to 42	5 female	0
Sterman et al. (1978)	ABA; ABAB	SMR	12 months? (2 week intervals)	seizures per mo.; EEG	varied	10 to 40	8; not specified	0
Cott et al. (1979)	within subject	SMR, SMR + time out	210 days (2 session/wk)	seizures per mo.	varied	16 to 31	3 male; 4 female	0
Quy et al, (1979)	within subject design	SMR	12 months	seizures per wk; EEG	general, tonic-clonic, psychomotor	23 to 49	2 male; 1 female	0
Lubar et al. (1981)	ABA crossover	SMR	10 months	seizure frequency; EEG; neuropsych. tests	varied	13 to 52	4 male; 4 female	0
Tozzo et al. (1988)	within subject multiple baseline	SMR	5 weeks SMR; 3 weeks "auditory biofeedback"	seizures per phase	absence, atonic, clonic-tonic	18 to 29	4 male; 2 female	0
Kotchoubey et al. (2001)	within subject; between subject; self-selected groups (Med = change of medication regime) (RES = Resp. FB)	SCP	35 SCP sessions; 15 sessions behavior therapy	seizures per wk; psych. battery	simple partial, complex partial, secondarily generalized	14 to 55	SCP: 14 male; 20 female RES: 5 male; 6 female MED: 4 male; 3 female	2

*AB, ABA, ABAB and ABCA designs represent baseline (A), treatment or reinstatement of treatment (B), an optinal additional treatment (C), and withdrawal of treatment (second A).
SMR: sensorimotor rhythm; SCP: slow cortical potentials

was used including identifying precursors and triggers for seizures, diaphragmatic breathing, and SMR biofeedback.[19]

Finally, learned alterations in EEG patterns with neurofeedback are not conscious nor voluntary, as indicated by associated changes and related clinical shifts during the unconscious state of sleep. As SMR increases, nocturnal epileptiform activity decreases.[20-22]

Sterman[8] summarized peer-reviewed neurofeedback epilepsy research from 1972 to 1996 and determined that 4 out of 5 patients enrolled in these various studies improved clinically (142 of 174 patients, or 82%), and most (66% of reported cases) exhibited "contingency-related EEG changes and a shift towards EEG normalization" (p. 52[8]).

Studies utilizing SCP training, though not as numerous, also show positive outcomes. Kotchoubey and colleagues[23] found decreased seizure incidence following SCP training, which was correlated to SCP amplitude. Rockstroh and colleagues[24] reported significant seizure reductions, with six participants having longer seizure-free periods. Finally, Holzapfel, Strehl, Kotchoubey, and Birbaumer[25] also found reductions in seizure rate following SCP training in an individual experiencing generalized clonic-tonic seizures while taking anticonvulsant medications and after an anterior callosotomy.

While results reported in these and other studies are promising, any study alone is insufficient to determine whether neurofeedback is efficacious for treating epilepsy. The goal of the current paper is to combine the appropriate literature into a single evaluation of seizure control (i.e., meta-analysis), which may allow a firm conclusion.

METHOD

Sample

We performed an exhaustive literature search of Medline, Psychinfo and PsychLit databases between years 1970 and 2005 using the words "EEG biofeedback," "neurofeedback," or "neurotherapy" for treatment of "epilepsy" or "seizure disorder." All reports, including abstracts, full publications, review articles and presentations at meetings were carefully screened by two of our authors for assurance that the same or earlier results had not been

Table 2

Post-treatment reduction in seizure frequency and effect size for 10 investigations of neurofeedback training for epilepsy

			Log (post/pre)					
No.	Study	N	Mean	Std. Dev.	t-value	p-value	ES	Variance of ES
1	Sterman (1974)	4	-0.34	0.84	-0.81	—	-0.41	0.27
2	Kaplan (1975)	4	-1.44	2.22	-1.30	—	-0.65	0.30
3	Lubar (1976)	8	-1.56	1.48	-2.97	.02	-1.05	0.19
4	Sterman (1978)	8	-0.96	0.89	-3.04	.02	-1.08	0.20
5	Kuhlman (1978)	5	-0.25	0.18	-3.10	.04	-1.38	0.39
6	Cott (1979)	7	-0.25	1.20	-0.54	—	-0.21	0.15
7	Quy (1979)	3	-0.18	0.44	-0.70	—	-0.41	0.36
8	Lubar (1981)	8	-0.48	0.74	-1.84	.11	-0.65	0.15
9	Tozzo (1988)	6	-1.20	1.20	-2.45	.06	-1.00	0.25
10	Kotchoubey (2001)	34	-0.31	0.88	-2.09	.04	-0.36	0.03

p-values > .20 are indicated by dashes; ES = effect size

reported elsewhere, and any differences in interpretation were resolved. Publications reporting quantitative EEG findings without including seizure data were eliminated from consideration during this initial screen, and all studies had to be prospective.

Exclusion and inclusion of studies

Inclusion criteria

Studies satisfied inclusion criteria only if they consisted of peer-review journal publications, provided full information on patient selection, utilized SMR or SCP, and reported individual pre-and post-treatment seizure rates.

Exclusion criteria

Studies were excluded if they provided group data only,[8,13,20,24] or were preliminary or first in a series of studies.[26] The studies by Lantz and Sterman[13] and Rochstroh et al.,[24] with sample sizes of 24 and 18 respectively, and p values < .005, would have satisfied our inclusion and exclusion requirements if we had selected a more conventional treatment effect (i.e., the mean of post- and pre-change in seizure rates), but seizure rates could not be determined for individual patients.

Ten out of 63 studies met our inclusion criteria. Of these, most were high-quality experimental research (e.g., ABA design, see Table 1) but with relatively small sample size. Sterman et al.[10] was the earliest of the studies meeting our inclusion and exclusion criteria and Kotchoubey et al.[23] the most recent. Kotchoubey et al.[23] is the only SCP study to meet criteria.

DATA ANALYSIS

Most meta-analyses are based on results from studies with one or more active treatment groups and a nontreatment control group in which each treatment group is compared to the control group for significance. However, in the case of patients with seizures, it is often viewed as unethical to have a nontreatment group. The study of Kotchoubey et al.[23] included two control conditions; one group received feedback of respiration rate and the other group had changed its drug regime, combined with extensive psychosocial therapy. We utilized only the group receiving SCP self-regulation. Thus each study included in this meta-analysis contained a single treatment group in which self-reported seizure rates were reported at pretreatment and at the latest treatment or post-treatment period.

All the studies included in this meta-analysis reported change in average number of seizures between pre-treatment and post-treatment or final observation period. Given the large differences in seizure rates among individuals, the treatment effect was defined proportionally, as log (post/pre) or equivalently log (post)-log (pre). We compared performance of log(post/pre) as the test statistic for each study with the more commonly used simple difference (post-pre). The former satisfied conditions for normality based on the Shapiro-Wilk test in 9 of 10 studies while the latter failed in 5 studies. Also the former, using a paired-difference t-test as a test statistic, showed significant improvement on 4 of the studies while the latter was significant for only 1 study. These preliminary results confirmed the advantage of using log (post/pre) as the test statistic in each study. It should also be noted that sample sizes were equal to 4 or less on 3 of the 10 studies so that the power of tests for normality and significance of effect in each study was small.

The software program Comprehensive Meta Analysis (Biostat, Inc., Englewood, NJ) was used to combine the 10 studies. The program computes a standardized difference "d" along with standard error "se" for each study, then calculates a "Hedges" effect size g and standard error from which it determines a study's z-value and p-value, which are combined across studies to obtain overall "Fixed" and "Random" effects for a variable. The Fixed Effect result is computed under the hypothesis that all studies estimate the same effect, and this hypothesis may be tested with a Q-test for heterogeneity. If this test is statistically significant it can be assumed that the different studies are not all estimating the same magnitude of effect and a Random Effects model should be assumed. A funnel plot was also generated to ascertain whether information about individual studies might help to explain any pattern of differences among standardized effect sizes, regardless of Q-test results.

RESULTS

From more than 63 studies identified as evaluating the effects of neurofeedback on epilepsy, only 10 studies involving a total of 87 participants provided the essential information needed for our analysis (see Tables 1 and 2).

Only grouped data were presented in the publication by Kotchoubey et al.[23] but individual data was provided by the authors. Results from each of the 10 studies are shown on Table 2, including a t-test of log (post/pre-seizure frequency) values. In a few instances the number of seizures at post-treatment reduced to 0, in which we substituted the minimum value of log (post/pre)from the other subjects in that particular study.

Standard deviations could not be calculated from Lantz and Sterman,[13] although this study was the most comprehensive of all research carried out in this field, with a relatively large number of

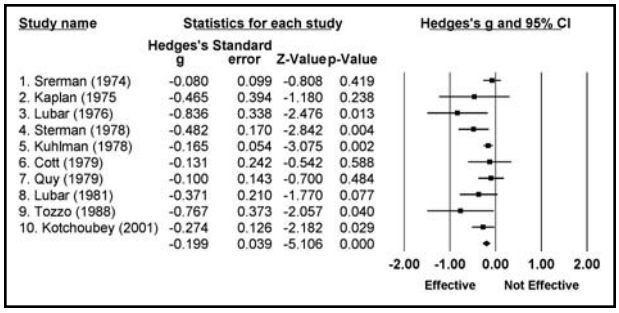

Figure 1.

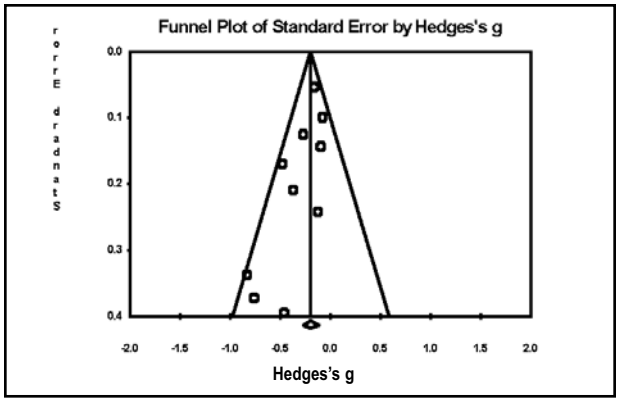

Figure 2.

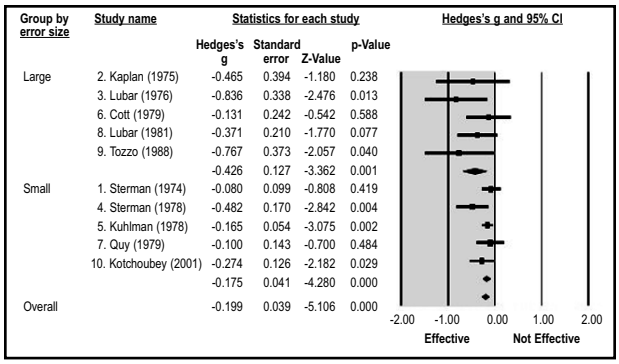

Figure 3.

subjects exposed to both cross-sectional and longitudinal control conditions, with documented drug-refractory seizure histories, and most with previous video, CT-scan, and depth-recording confirmation of seizure and anatomical characteristics, along with drug regimens which were kept constant and monitored through periodic blood analyses. With contingent neurofeedback training the group seizure rate was significantly reduced ($p < 0.005$), resulting in a median seizure reduction of 61 %, which translated to a seizure reduction of at least 13 per month in 12 of the 24 patients.

Results shown on Figure 1 indicate that both Fixed Effect and Random Effect models are significant. The Hedges's fixed effect model gave ES = -0.199, SE = 0.039, z = -5.10, p<.001 and the random effects results were ES = -0.233, SE = 0.057, z = -4.09, p<.001. The Q test for heterogeneity was not significant (Q = 12.54, df = 9, p=.18), but suggested the possibility that not all 10 studies estimated the same treatment effect.

If the largest study by Kotchoubey et al.[23] was not included in the analysis, the results were relatively unchanged: the Hedges's fixed effects results were ES = -0.191, SE = 0.041, Z = -4.66, p<.001 and the corresponding random effects results were ES = -0.237. SE = 0.066, Z = -3.57, p<.001 respectively, with Q = 12.15, df = 8, p = 0.145.

In order to investigate the possibility of publication bias among the 10 studies, we performed a "funnel plot" as shown in Figure 2. Publication bias is typically indicated by greater effect sizes from publications with relatively large standard errors (SE), compared to studies with relatively small SE, as appears to be the case in Figure 2. For example 4 of the 5 studies with the largest SE had larger Hedges's effect sizes (ES) than 4 of the 5 studies with the smallest SE.

To determine the statistical significance of this observation we compared results between the groups of 5 studies with the largest SE to the group of 5 studies with the smallest SE (Figure 3). Hedges's results for the group with larger SE were ES = -0.426, SE = 0.127, Z = -3.36, P = 0.001, while results for the group with smaller SE were ES = -0.175, SE = 0.041, Z = -4.28, P<0.001. The test for difference in effect sizes between the two groups gave Chi square =3.54, df = 1, p =.06. The random effects results were comparable. These results tend to confirm the possibility of publication bias. However, all we can state is the observation that studies with larger SE tended to give results with larger effect sizes.

DISCUSSION

The studies used in this meta analysis had three characteristics in common: epileptic subjects with seizures not controlled by medication alone, utilization of EEG biofeedback training for the purpose of controlling the incidence of seizures, and daily subject records of seizure occurrences from pre-study until the final accumulation of data with family assistance. Nine studies utilized the SMR protocol designed to reduce seizure occurrence by increasing the relative amount of time in an EEG frequency range, initially defined as 12-14 Hz, comparable to spindle occurrence in the sleep EEG, while one study was designed to reduce seizure incidence by controlling SCP. The studies differed in other respects as well. Three studies used an SMR frequency range of 12-15 Hz, one used 12-16 Hz, one used 6-12 Hz along with 12-14 Hz and one used 9-14 Hz. Three studies inhibited theta band activity (e.g., 3-8, 4-7, or 4-8-Hz) with feedback as well. Although the 9 SMR studies achieved mixed success in increasing time spent in the intended frequency band, the results on seizure control were quite successful in controlling seizures. Our meta-analysis revealed a statistical fixed effect of neurofeedback training (SMR or SCP) on seizure frequency (z = -5.10, p<.001). Neurofeedback training (SMR or SCP) reduced seizures significantly. SMR training in particular produced a large effect of seizure frequency, where 42 of the 53 subjects (79%) receiving SMR training had reduced numbers of seizures.

Studies we combined in our analysis included placebo-control by means of random feedback, blind evaluation, and the effect of contingency reversal. Contingency reversal is a very powerful evaluation technique as it controls for adjustment in patient expectation across time and other non-specific effects unmanaged by randomized placebo control research.[27]

Based on these consistent findings, the practical value of neurofeedback should be recognized. Medication, while commonly helpful,

generally provides effective control of seizure for only two-thirds of patients. In addition to side effects, long-term use of many anti-seizure drugs has significant health risks.[28,29] Neurofeedback offers an attractive alternative to neurosurgery or implantation of vagal nerve stimulators. Antiepileptic drugs are also teratogenic: prenatal exposure to anticonvulsant medication puts children at risk for developing autism[30] or fetal anticonvulsant syndrome which manifests as major physical birth defects and infant mortality.[31] Neurofeedback offers women of child-bearing age a possibility of controlling epilepsy without risking the health and well-being of her newborn.

Our review of the literature revealed specific limitations of the body of research for this disorder. With the exception of Kotchoubey et al.,[23] long-term follow-up of neurofeedback treatment has not been formally assessed for epilepsy. Few studies directly compared neurofeedback with other interventions, and only a handful attempted to compare training to a sham control. In order to be accepted by other scientific and medical communities, future research needs to be carefully planned and executed to conform to efficacy requirements.[18] Future studies should involve randomized treatment assignment with a feasible alternative treatment as a control condition. Seizure type and focus should be well documented and a more thorough assessment of functional outcome performed, including neuropsychological and EEG tests. If two such control group studies determine that neurofeedback is superior to traditional medical treatments (e.g., medication or neurosurgery), neurofeedback would qualify as an efficacious and specific treatment for specific epilepsies.[18]

CONCLUSION

Despite certain limitations, results were quite similar across all studies included in our meta-analysis as well as the majority of case reports and case series: SMR or SCP training consistently decreased seizure rate among severe cases of epilepsy which could not otherwise be controlled. In other words, subjects provided their own historical control against which clinical improvement ought to be evaluated. As nearly all patients underwent lengthy unsuccessful medication therapies for epilepsy prior to any neurofeedback trial, and the placebo effect had minimal impact in these previous therapies, its presence in neurofeedback training is just as unlikely and probably negligible.

Given the success of neurofeedback in seizure reduction in such severe cases, one might speculate how much more successful neurofeedback treatment might be in patients whose epilepsy is relatively well-controlled by medication. Might neurofeedback systematically, allow for reduction or elimination of medication in the two-thirds of epilepsy patients currently treated with drugs? While we can clearly state that neurofeedback training is useful for patients with uncontrolled seizures, this may also suggest a promising avenue for future research and treatment for many patients whose seizures do respond to other forms of treatment as well.

APPENDIX: REVIEW OF LITERATURE

Andrews and Schonfeld[19] found that over 80% of 83 patients achieved control over seizures when a combination of interventions was used including identifying precursors and triggers for seizures, diaphragmatic breathing, and SMR biofeedback. Finley[32] reported decrease in seizure activity as SMR increased in a patient with akinetic seizures and in a patient with psychomotor seizures. Tansey[33] reported on a participant who achieved total cessation of absence seizures. Johnson and Meyer[34] reported a drop in seizure rate for a patient who underwent alpha, alpha-theta, as well as theta training. Walker and Kozlowski[17] cited results in 10 consecutive patients. After an average of 34 neurofeedback sessions, 9 patients were seizure-free and 2 were able to terminate anti-seizure medication. Unlike other studies, Walker and Kozlowski[17] provided SMR training along with EEG rhythm training addressed to a patient's specific abnormalities. Forty-two percent of sessions consisted of magnitude training (normalizing amplitudes while reinforcing SMR or beta activity) while the remainder of sessions normalized EEG coherence (connectivity training).

Zhao, Wu, Liang, and Hu[35] reported on 5 patients with intractable epilepsy who underwent approximately 40-50 sessions of SMR uptraining and theta downtraining. Two of the 5 patients reduced seizure frequency from daily (20-30 times/month) to monthly (1-2/month), with significant decrease in seizure intensity and duration. EEG analyses before and after neurofeedback training also revealed significant improvement of the EEG. Finley[32] used non-contingent feedback as a control, and found both clinical improvement and improvement in the EEG in both patients. Similarly, Wyler et al.[36] used non-contingent feedback and EMG as a control condition, and reported clinical improvement in all 4 patients. Kuhlman and Allison[26] likewise used non-contingent feedback as a control, and found 3 of 5 patients showing both clinical improvement and positive changes in the EEG. They suggested that results can be achieved fairly quickly. Random feedback was used by Quy, Hutt, and Forrest[37] as a control condition, and all 3 subjects showed significant clinical improvement. Among ABA cross-over design studies, 79% of 24 patients demonstrated clinical improvement in seizure reduction.[9,38,39]

Employing a between-group design Lantz and Sterman[13] found a decrease in seizure rate in patients receiving SMR training but not in control patients. Finally, neurofeedback training alters EEG patterns and does not necessarily improve conscious skill in self-regulation as evidenced by changes in sleep EEG as a result of training. As SMR increases, nocturnal epileptiform activity decrease.[20-22]

Sterman[8] summarized peer-review neurofeedback epilepsy research from 1972 to 1996 and determined that 4 out of every 5 patients enrolled in these various studies improved clinically (142 of 174 patients, or 82%), and most (66% of reported cases) exhibited "contingency-related EEG changes and a shift towards EEG normalization" (p. 52[8]).

Studies utilizing SCP training, though not as numerous, also show positive outcomes. Kotchoubey and colleagues[23] found decreased seizure incidence following SCP training, which was correlated to SCP amplitude. Rockstroh and colleagues[24] reported significant seizure reductions, with 6 participants having longer seizure-free periods. Finally, Holzapfel, Strehl, Kotchoubey, and Birbaumer[25] also found reductions in seizure rate following SCP training in an individual experiencing generalized clonic-tonic seizure while taking anticonvulsant medications and after an anterior callosotomy.

Studies Included in the Meta-Analysis

A total of 63 studies were located which examined the effect of neurofeedback on epilepsy. However, of these studies, only a few were appropriate for use in a meta-analysis due to limitations in methods and reporting of results. Specific inclusion and exclusion criteria are discussed in detail in the Method section, but more information on the included studies is provided below.

Sterman et al.[10] is the earliest study which meets our inclusion criteria. SMR performance of 4 patients with intractable epilepsy was compared to SMR performance of 4 non-epileptic patients. The epileptic patients often failed to achieve the same consistency of SMR performance as the non-epileptic group, even after extensive training. The authors found decreases in reported and observed seizure rates, particularly for tonic-clonic and myoclonic seizures.[10]

Kaplan[40] initially trained two epileptic patients at 12-14 Hz, which was not associated with significant changes in reported or observed seizure activity. Three additional patients trained at 6-12 Hz followed and although 2 of the 3 patients showed a decreased seizure incidence, she was unable to conclude whether this change in seizure frequency was associated with training.[40]

Lubar and Bahler[38] examined effectiveness of SMR training in patients with a variety of seizure disorders, including grand mal, myoclonic, akinetic, focal, and psychomotor. Three of 8 participants were also diagnosed with mental retardation. While total treatment time varied from 80-260 days, many participants experienced changes in seizure frequency, intensity, and duration and 2 participants remained seizure-free for over 1 month.[38]

Kuhlman and Allison[26] found that reductions in seizure rates for 5 participants were associated only with EEG-contingent feedback and not in response to non-contingent feedback.

Sterman and Macdonald[39] performed biofeedback training with 8 patients utilizing 6-9 Hz band as well as the 12-15 or 18-23 Hz frequency. They found that 6 of the subjects had reductions in seizures which followed successful training at either 12-15 Hz or 18-23 Hz in the absence of the 6-9 Hz training. For the 12-15 Hz band, seizures returned to baseline when the reinforcement contingencies were removed.[39]

Cott et al.[11] studied 3 mentally disabled individuals with seizure disorders and provided them with 8-12 Hz occipital EEG biofeedback training. All 3 participants experienced decreases in some of their seizure activity; however, their inability to increase 8-12 Hz activity brought into question the effectiveness of feedback stimuli used as reinforcers with these individuals. The results of the study indicate that changes in procedures will be necessary for future studies to determine if 8-12 Hz occipital EEG training is effective in reducing epileptic seizures.[11]

Quy, Hutt, and Forrest[37] trained 3 patients with chronic uncontrollable seizures using several different frequencies (e.g., enhancing 8-10 Hz and 12-16 Hz, suppressing high voltage activity) as well as providing random feedback. The authors reported improvements in seizures for all the participants by the end of the study.[37]

Lubar et al.[9] used a double-blind crossover study to evaluate the effect of neurofeedback in 8 subjects with drug-refractory mixed seizures. The study involved multiple phases of training. First, participants were trained to suppress 3-8 Hz, increase 12-15 Hz activity, or perform these together. All subjects were then trained to enhance slow-wave activity. Initial contingencies were reinstated during the final phase. Five participants experienced a reduction in seizure rate by the end of the study.[9]

Similar to many studies using SMR training, Tozzo, Elfner, and May[14] found that all participants (who ranged from mentally disabled to above average IQ) could increase their SMR time, and that this was related to a reduction in seizure activity. Utilizing a multiple baseline design the authors found that while only 2 participants showed improvement during relaxation, 5 of the 6 subjects showed improvement with increased SMR.[14]

Kotchoubey et al.[23] compared SCP training to feedback of respiration rate and a group who received new anticonvulsant medications and psychosocial counseling. All patients were diagnosed with drug-refractory partial epilepsy. Results indicated that participants in the neurofeedback and medications groups reduced seizure frequency. Cortical self-regulation was assessed 6 months after the end of treatment and turned out to be stable; seizure reduction was maintained at 12 months.[23]

DISCLOSURE AND CONFLICT OF INTEREST

G. Tan, J. Thornby, D.C. Hammond, U. Strehl, B. Canady, and K. Arenmann have no conflicts of interest in relation to this article. D. A. Kaiser owns and consults with many companies invovled in manufacturing and distributing quantitative EEG analysis. He asserts that there is no conflict of interest in publishng this specific research and his involvement in any commercialization of quantitative EEG analysis.

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