30 Conditioning Mechanisms, Behavior Technology, and Contextual Behavior Therapy

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Along with the earliest documentation of epileptic seizures, behavioral conditioning mechanisms were noted as a viable means to control seizures. Galen described seizures as a predictable chain of behaviors that could be interrupted by stimulating different parts of the body (1). In 1881, Gowers published a series of case studies showing that behavioral techniques could interrupt seizure development (2). He described specific forms of stimulation, such as putting pressure on and massaging the hand where the patient felt a seizure begin, as well as more general techniques, such as the application of a strong aroma at the beginning of a seizure. In 1857, Brown-Sequard presented case studies in which seizures were successfully aborted using a variety of stimulations, also contingent upon seizure onset (3). In 1931, Jackson wrote that the motor seizure now called "Jacksonian march" (4) could be stopped by vigorous rubbing of the affected limb (5).

These early reports showed an understanding of the value of behavioral techniques in controlling seizures and were conceptualized under the principle of competitive recruitment. This principle holds that seizures arising from hyperexcitable groups of neurons in the brain, when localizable by a specific and identifiable behavioral correlate, can be blocked from progressing by behaviors that competitively recruit relatively normal brain cells, thereby increasing their normal activity and reducing hypersynchronization of the seizure focus.

During the first half of the twentieth century, experimental behaviorists demonstrated that seizures in animals could be elicited and interrupted using conditioning techniques (6–10), consistent with the principle of competitive recruitment. Different forms of behavioral stimulation were found to interrupt seizure progression when applied at the start of the seizure.

Conditioning techniques were subsequently applied to patients. In 1956, Efron reported the case of a jazz singer who consistently experienced seizures as she was about to perform on stage (11). Her seizures could be arrested using second-order conditioning of an olfactory stimulus to a bracelet. Efron first introduced the smell of jasmine, which the patient associated with a calming effect, presumably associated with a decrease in cortical activity. The patient inhaled the jasmine under situations that placed her at high risk of having seizures (specifically, performing on stage) as well as when seizures actually began. When the singer had learned to successfully interrupt seizures using the jasmine, Efron then conditioned the smell of jasmine to a bracelet, and finally to the thought of the bracelet. This study demonstrated that the singer was able to counteract the increase in cortical activity associated with her seizure onset through conditioning, consistent with the principle of competitive recruitment,

and it marked the beginning of conditioning as seizure therapy for patients with epilepsy.

Electroencephalographic (EEG) technology further contributed to understanding the effects of conditioning mechanisms on seizures. Forster, for example, used video-EEG over a 30-year period to study conditioning mechanisms involved in reflex epilepsy in children (12). In 1964, he and his coworkers showed that seizure thresholds could be altered by habituation (13). They determined the threshold frequency and intensity by which specific stimuli could trigger seizures in patients with reflex epilepsy. Then, by exposing patients to stimuli at these thresholds over prolonged periods, seizures were habituated, and the patients were no longer sensitive to the seizure-triggering stimuli, resulting in fewer seizures.

Forster further used video-EEG to show that seizures could be extinguished by combining desensitization with competing responses (14). Seizure behaviors were analyzed for patients with reflex epilepsy, who were then exposed to their identified seizure-triggering stimuli and, at the same time, instructed to perform a distracting ritual. These procedures resulted in nearly complete seizure control, along with significant reductions of epileptiform abnormalities on continuous EEG recordings. By 1977, Forster presented similar data from over 30 patients with various forms of reflex epilepsy, demonstrating nearly complete seizure control and reduced epileptiform abnormalities (12).

This chapter reviews several specific forms of behavioral therapy for patients with epilepsy, including their underlying principles, methods, and outcomes.

EEG BIOFEEDBACK

Using EEG biofeedback techniques, operant conditioning mechanisms were shown by two research groups to produce brain rhythms that protected against seizures. These investigators used EEG biofeedback to train patients to normalize their brain wave activity and thereby elevate seizure threshold. Sterman and colleagues published studies from 1970 to 1981 showing the sensory motor rhythm (SMR) was anticonvulsant in cats and patients with epilepsy. After training patients to produce the SMR, they found seizure frequency reductions from 35 to 50% at one-year follow-up (15). Behavioral correlates to the SMR are active inhibition of peripheral motor activity and the mental state described as concentrated alertness.

Birbaumer and colleagues (16, 17) showed that instrumental conditioning of slow cortical potentials (SCP) reduced epileptiform activity. By observing that all organisms have a tendency to seize as the brain fluctuates in cortical activity, they redefined epilepsy as the inability to control cortical excitability. Whereas normal feedback mechanisms within the brain control these transitions of excitability, these authors reasoned that patients with epilepsy have hyperexcitability of cortical tissue because of a failure of these down-regulating mechanisms, leading to an explosive chain reaction of excitation among neuronal networks, manifesting as a seizure. The specific seizure symptoms depend on where and how much of the network is involved. Treatment with SCP biofeedback is thus designed to train the person with epilepsy to downshift his or her cortical excitability and thereby reduce the risk of a seizure.

SCP treatment is therefore based on a functional analysis of behavior as compared to other biofeedback techniques. Whereas SMR therapy trains the patient to produce a specific antiepileptic brain rhythm to protect the patient in much the same way as drug therapy, SCP training is built on an awareness of the chain of seizure behavior, requiring the patient to generate up- or down-going responses of cortical activity contingent on its baseline level.

THEORY OF CONDITIONING

On the basis of animal research (18), Fenwick theorized a conditioning mechanism underlying seizures and suggested using this mechanism as a basis for the behavioral treatment of epilepsy (19). He suggested that focal seizures occur when restricted populations of neurons surrounding the focus are sufficiently excited, whereas generalized seizures occur when the level of cortical excitability reaches the point at which thalamic recruiting volleys generalize and spread. He further proposed that these epileptogenic activities are not random and do not occur in a vacuum, but rather act in a predictable manner and are influenced by behavior.

Fenwick referred to the animal research of Lockard and Ward (18), which conceptualized the conditioning mechanism underlying the seizure process in terms of what they called group one and group two neurons. Group one neurons were defined as the dysfunctional pacemaker group that consistently fire epileptogenic activity, whereas group two neurons were the partially affected neurons surrounding the group one neurons. Group two neurons mostly behave in a normal fashion but occasionally get recruited by the group one dysfunctional neurons, resulting in epileptogenic activity spreading out and away from the focus (group one neurons). The patient would typically sense the seizure onset when dysfunctional signals passed from group one to group two neurons. Consistent with this model, Lockard and Ward observed that not all monkeys developed seizures after injury to the exact same brain site, though all showed abnormal EEG activity from group one neurons. They speculated that monkeys who reacted with increased activity of group two neurons at the first signs of spreading of

the dysfunctional signals counteracted and stopped the spread of the "would be" seizure.

Such observations in the context of this model suggested that damaged neurons predisposed to epilepsy but that the behavioral response to the dysfunction was critical to whether epilepsy actually develops.

BEHAVIOR MEDICINE MODEL OF EPILEPSY

In the behavior medicine model, epilepsy consists of an organic predisposition to seizures, and intrinsic and extrinsic factors that influence the probability of seizure occurrence. Wolf summarized this new paradigm (20):

> Epileptic seizures can be triggered by both nonspecific facilitating factors such as sleep withdrawal, fever, or excessive alcohol intake, and specific reflex epileptic mechanisms. These consist of sensory or cognitive inputs activating circumscribed cortical areas or functional anatomic systems that, due to some functional instability, respond with an epileptic discharge. Interruption of seizure activity at the stage of the aura (i.e., locally restricted discharge) also can be achieved by nonspecific (e.g., relaxation or concentration techniques or vagal nerve stimulation) or by specific focus-targeted sensory or cognitive inputs. The latter, again, activate circumscribed cortical areas. Intriguingly, in some patients, the same stimulus can either precipitate or abort a seizure. The response depends on the state of cortical activation: seizure precipitation occurs in the resting condition, and seizure interruption occurs when the epileptic discharge has begun close to the activated area. These relations can be understood on the background of experimental data showing that an intermediate state of neuronal activation is a precondition for the generation of paroxysmal depolarization shifts, whereas a hyperpolarized neuron will remain subthreshold, and a depolarized neuron that already produces action potentials is not recruitable for other activity. Sensory input meeting an intermediately activated pool of potentially epileptic neurons is adequate to produce a seizure. In another condition, the same stimulus can depolarize a neuron pool in the same area sufficiently to block the further propagation of nearby epileptic activity. Understanding these interactions facilitates the development of successful nonpharmaceutical therapeutic interventions for epilepsy.

APPLIED BEHAVIOR ANALYSIS IN THE TREATMENT OF EPILEPSY

The behavior medicine model of epilepsy is based both on the underlying pathophysiology and principles of conditioning. Epilepsy is defined as a predisposition that can be both triggered and inhibited by certain interactions, behaviors, and cognitions. Depending on the baseline "state of cortical excitation," a stimulus can either trigger or inhibit a seizure. Treatment strategies are based on functional analysis and are individualized to help the patient with epilepsy do the following:

- *Predict* the seizure response by discrimination of intrinsic and extrinsic factors associated with seizure onset
- *Prevent* seizure occurrence by applying exposure procedures to "high"-risk situations or activities associated with seizure occurrence
- *Interrupt* or *counteract* an ongoing seizure response by initiating an appropriate "correcting" or competing response
- *Reinforce* himself or herself for doing so

Methods for functional analyses and individualized treatment procedures are presented in a handbook (21).

Prediction of Epileptic Seizures

Discriminating "high risk" factors and early symptoms of seizures allows patients to *predict* the occurrence of seizures. Tools to enhance identification of these factors include seizure diaries, seizure behavior observations, and, if possible, video-EEG recordings. The most reliable way to confirm the identities of the seizure-inducing factors is to confirm how the patient actually responds to what he or she believes are the seizure triggers.

Spector and co-workers found that 88% of patients were reliably able to identify factors that precipitated their seizures (22). Antecedents included specific triggers that enhanced cortical excitation and synchronization in discrete areas of the brain that were uniquely receptive to these particular influences, such as certain light and sound frequencies or visual patterns. On the other hand, triggers could be nonspecific factors that also generally enhanced shifts in neuronal excitation; examples included anticipation, stress, conflict, fear, and physical exertion. Another type of trigger is the physical sensation at the start of the seizure.

Not all seizures begin with signs that are identifiable to the patient. Patients are usually able to recognize the initial symptoms of simple partial and complex partial seizures. The seizure response itself may have been observed by others a number of times and may be on video. In the case of generalized seizures, antecedents may include emotional, physical, or environmental factors.

Several studies show that fear and stress increase the risk for seizures and, consequently, *preventive* treatment studies have targeted these antecedents (22). Several studies have evaluated the effects of relaxation techniques (23, 24) and yoga (25), performed interictally, to reduce stress levels.

Interrupting Ongoing Epileptic Seizures

Interrupting an ongoing seizure has been attempted by most patients with epilepsy, either consciously or by accident (21), resulting in aborting or postponing seizure onset, moving to a safe place, and shortening seizure length. Figures vary, but between 23% and 53% of patients have aborted a seizure (22). Most commonly, patients either increase or decrease cortical activity depending on the upward or downward shift in cortical activity induced by the seizure trigger (21).

The functional analysis helps the patient determine, in each situation, which direction the countermeasure should aim for. If the trigger is characterized by a high cortical excitation, the countermeasure would be a slow transition downward, whereas if the trigger is a drowsy state, the countermeasure would warrant an increase in cortical excitement. A variety of techniques for changing cortical activity are available so that patients can choose an appropriate countermeasure depending on their specific situation (21, 26, 27). Examples of up-going (that is, cortically exciting) countermeasures are whistles, strong smells (for example, a raw onion), strong tastes like fresh ginger, singing, shouting, tactile stimulation with massage or pinching, and jumping up and down. Examples of down-going countermeasures are breathing exercises, muscle relaxation, or focused concentration on a song, a mathematical problem, or a calming picture. Betts and co-workers (28, 29) used aromas to stimulate a general arousal when applied at the time of seizure onset, resulting in an immediate halt to seizure activity. In the case of developmentally challenged individuals, countermeasures can be employed by caretakers to help interrupt seizures.

Seizure triggers and seizure responses constantly change along with the infinite variations of contingencies. Under the seizure behavioral analysis approach, patients are taught the underlying principles and to swiftly apply the appropriate countermeasure at the start of each seizure. They learn that this approach is different conceptually from standard drug therapy. For the latter, epilepsy is seen as an uncontrollable illness with unpredictable seizures, where medication is the only alternative, in contrast to the seizure behavioral analysis approach, which conceptualizes epilepsy as a tendency to seize in which seizures are predictable and controllable.

Treating the Function of Seizures

One of the most difficult parts of the behavioral analysis approach is to understand the *function* of the actual seizure in the life of the patient. At first glance, an epileptic seizure would probably not be viewed as having a function. In fact, when medical professionals use the word "functional" to refer to seizures, they mean psychogenic nonepileptic seizures. In the operant conceptualization, all epileptic seizures would be called functional since they all "function" or operate on the environment in some way. For example, seizure behavior looks scary or painful to observers, or elicits a range of behaviors towards the person with epilepsy.

Is it possible that seizures could be reinforced by the effects they have on the environment? In a long-term study, a majority of children with refractory seizures did not want to become seizure-free, counter to the wishes of parents, caretakers, teachers, and physicians (30). Children and young people report positive reinforcement from others based on their seizures, such as special privileges or attention, physical contact, being someone special; further, seizures themselves may be stimulating or may induce euphoria. Dostoyevsky describes his seizure experience as follows "the air was filled with a big noise and I tried to move. I felt the heaven was going down upon the earth and that it had engulfed me. I have really touched God. He came into me myself. Yes, God exists. I cried, and I don't remember anything else. You all, healthy people . . . can't imagine the happiness we epileptics feel during the second before our fit . . . I don't know if this felicity lasts for seconds, hours, or months but believe me, for all the joys that life may bring, I would not exchange this one" (31). This positive description shows the complexity of the function of the seizure experience.

Young people and adults also report that seizures may be a means to avoid undesirable situations or to reduce anxiety and tension. Not surprisingly, therefore, the *function* of seizures correlates with social skills. In a long-term study of children with drug-refractory seizures, social skill competency was inversely related to the frequency of seizures occurring in social situations (30). The more developed the social skills, the fewer seizures that occurred in social situations; whereas the less developed the social skills, the more often that children's seizures occurred in specific social settings. Children with poorly developed social skills reported that seizures prompted desirable social consequences such as being held, being seen, and being the center of attention. Seizures that were reported as stimulating were often maintained while nonstimulating seizures were controlled, and seizures that were anxiety-reducing were less likely to respond to conventional anticonvulsant drugs.

This overview of the possible functions of a seizure shows how complicated and sophisticated the functional analysis must be. If the patient's seizures function in a desirable direction or effectively reduces something undesirable, conventional treatment is not likely to work. Still, the issue of function is a difficult one for all treating professionals. Do people choose to have seizures? In our experience, people can influence the probability of a seizure occurring, and once it starts, they can choose

to some degree the course it will take. The treating professional should look carefully at what functions the seizures may serve and help the client to find more attractive alternatives.

EVALUATION OF BEHAVIOR TREATMENT OF EPILEPSY

Studies evaluating behavior therapy as a treatment for epilepsy have been reviewed a number of times over the past 30 years. Mostofsky and Balaschak reviewed 60 studies in 1977 (32), Kraft and Poling (1982) added 11 studies to their review of studies from 1960 to 1980 (33), Goldstein reviewed studies published from 1980 to 1990 (34), and, most recently, Cochrane reviewed studies of the psychologic treatment of epilepsy (35). Methodological inadequacies discussed in the early reviews were low numbers of subjects, inadequate randomization, absence of serum anticonvulsant concentrations to confirm medication compliance during treatment phases, lack of objective physiological measures, and sole reliance on self-rating.

Goldstein reviewed seven studies that used EEG testing, anticonvulsant serum concentration measurements, sufficient numbers of subjects, one-year follow-ups, and appropriate statistical methods (23, 36–40). These studies included measures relevant to cognitive behavior therapy, including seizure diaries to document seizure frequency, duration, situation, response, and consequence. Social skills and coping skills were rated as well. Most dependent measures were tracked over a 10-week baseline, thereby providing the information needed for the seizure behavioral analysis. Treatments were individualized based on the behavioral analysis but also included preventive exercises, discrimination of seizure triggers, training in seizure countermeasures, and contingency management.

The Cochrane review assessed randomized or quasi-randomized studies of one or more types of psychologic or behavior modification techniques (35). Using strict criteria comparable to medical studies, the meta-analysis determined that all the trials were too small and their methodologies inadequate, leading to the conclusion that there was no reliable evidence to support the use of behavior treatments for patients with epilepsy and that further trials were needed.

ACCEPTANCE AND COMMITMENT THERAPY

Acceptance and Commitment Therapy (ACT) is a behavior therapy based on a new theory of language and cognition (41). ACT has been previously evaluated for a number of disorders such as depression, pain (42), and diabetes (43), generally with good outcomes.

ACT and other behavioral approaches for seizure management were studied in patients with refractory seizures in South Africa and India (44). In developing countries such as South Africa and India, the majority of people with epilepsy do not have access to modern anticonvulsant drugs; therefore, inexpensive and assessable alternative treatments are essential. Both studies were randomized, controlled trials and compared ACT and a control condition in South Africa, and ACT and yoga in India. Essentially the same ACT treatment protocol was used in both studies. Participants included adults with frequent, EEG-verified refractory seizures. Treatment consisted of 11 hours of therapy divided between two individual and two group sessions, and additional booster sessions at 6 months and one year. The South African study, conducted at an institution for persons with severe epilepsy, showed a significant reduction in seizure frequency and an increase in quality of life at the one-year follow-up (44).

ACT consists of acceptance, defusion, values, contact with the present moment, committed action, and self as context (45). Behavior management of seizures is typically taught toward the end of the ACT protocol (21). The aim of ACT treatment is to help the client create psychologic flexibility around "stuck places" and establish contact with direct and natural contingencies of one's valued directions.

Acceptance refers to accepting aspects of epilepsy that the patient cannot change. Clients learn to accept their predisposition to seize and the associated fears and negative thoughts and emotions, thereby avoiding "going to war" with their seizures, since this is a war the patient cannot win by fighting. Accepting the risk of having seizures and nonetheless living a full life is a skill that must be learned and practiced. Acceptance can improve seizure frequency, as exemplified by some patients who undergo video-EEG monitoring. In this situation, patients "try" to have seizures, and it is exactly this "trying" or acceptance that usually leads to no seizures, even among patients with very frequent seizures.

Defusion involves creating a distance between the client and his or her thoughts, associations, and rules related to epilepsy. Epilepsy-related thoughts, rules, and feelings of stigmatization are looked at objectively rather than from within. The client learns to use the thinking process more effectively.

Contact with the *present moment* means helping clients to be present with positive reinforcement or the vitality of the "here and now" rather than being engaged in problems of the past or fears about the future. Patients struggling with epilepsy commonly believe that they need to get somewhere else other than where they actually are to begin to live, and that they must be seizure-free before they would be acceptable as a partner or in the work force. Learning to make contact with the here and now

gives patients a way to let go of the struggle with their private events and to start creating the life they want.

Self as context is one of the fundamental principles of ACT. Through this process, the client creates a place from which fears, thoughts, and feelings can be looked at. The client *has* epilepsy, but he or she *is not* epilepsy. The client *has* fears but *is not* those fears. Self as context entails a perspective that the contents of life are products of human history. Self as context promotes a skill whereby the client learns that "I" is the constant context that has always been there, observing all experiences in life. From this perspective, the daily life experiences that constantly change appear less threatening. If there is no difference between the client and his or her epilepsy, there will be a struggle and suffering. By contrast, distinguishing self from life experiences allows the patient to put the epilepsy in perspective; epilepsy and seizures will simply be one of the many experiences in the content of a person's life, but not the basis for defining the person.

Values are an important process in ACT, since values provide the motivation necessary to commit to the work of getting back into life. "Values illness" is a term describing the process of giving up important, valued life directions in order to control symptoms. When symptom control becomes a person's main preoccupation, important sources of positive reinforcement are neglected and vitality is lost. The more the patient struggles and organizes his or her life around prevention, avoidance, and control of the seizures, the less time that he or she has to become involved in valued life activities. As the avoidance agenda grows, quality of life diminishes. Contacting constant, valued directions provides a way toward a meaningful and vital life and shows patients how far off course they have veered because of their emphasis on avoiding seizures.

Committed Action is the final process in ACT and entails a public commitment regarding actual steps the client is willing to take here and now to overcome obstacles and reclaim or create the chosen valued life. A committed action in ACT is a step taken toward a valued life, fully present and mindful, regardless of the resistance of private events and fears.

The Treatment Protocol

The six processes in ACT create psychologic flexibility and a space for valued living. The following is an outline of the objectives for the four-session ACT treatment protocol.

Session one focuses on establishing the "life compass." The therapist helps the client experience the discrepancy between how the participant wants to live (valued directions) and how he or she is actually living. Barriers to this desired life and how the patient has previously handled those barriers are examined. Associations and "rules" about the seizures held by the client are mapped out. Typical such rules are "I would like to study, but I have epilepsy," or "I would like to have a partner and a family, but no one wants someone with seizures." These rules are then targeted using the process described above.

Sessions two and three each last three hours and typically consist of six to eight people. The goals are to help participants understand, experience, and practice the concepts of taking steps in valued directions despite the obstacles, seeing thoughts as thoughts and not truths, learning and practicing seizure interruption methods, and making public commitments toward creating the vital life of their choosing. Role-playing allows participants to understand with their intellect but also to experience through practice. The group conducts functional analyses of each participant's seizures and brainstorms possible seizure interruption methods.

The aim of the fourth and final session is to summarize and evaluate how the participant is applying the treatment components, taking steps in the valued direction, defusing less useful behaviors, and using seizure control techniques. New commitments to taking steps in the valued life directions are made.

Outcome Measurements

Dependent variables that can be used to measure the effects of this treatment model are seizure frequency and duration, quality of life, vitality, and experiential avoidance with regard to epilepsy. Seizure frequency and duration are combined in the seizure index, used previously in several behavioral studies (27, 36). Two standardized quality-of-life measures that are used are the World Health Organization Quality Of Life (WHOQOL-bref) (46) and the Satisfaction With Life Scale (SWLS) (47). New instruments developed for this purpose include a variation of the Acceptance and Action Questionnaire (AAQ), called here the AAQEP, and the Bulls-Eye, a measure of vitality. The AAQ has shown good reliability and validity, with test-retest reliability values of 0.89 and 0.92 (Cronbach's alpha) (48). The Bulls-Eye shows a test-retest reliability of 0.86 (Pearson's correlation) (49).

The Bulls-Eye measures participants' functioning level, along with persistence and vitality in actions, and consists of four dartboards. For the first three dartboards, the client is asked to choose a valued direction that he or she would like to improve or move toward (or is not satisfied with). The bull's eye of the dartboard represents 100% vitality of a defined valued direction. Using the first three dartboards, subjects are asked to mark how close they are living in the direction of vitality in that chosen value. The last dartboard asks the clients to rate how often they generally try to follow valued life directions despite difficulties and barriers. The distance between the

bull's-eye and the edge of the dartboard is 4.5 centimeters. On the first three dartboards, the measurement is figured by taking the distance between the X placed by the subject and the center of the bull's-eye, and an average distance is calculated. The last dartboard is measured the same way as the first three but is presented as a single measure.

Results

Overall the ACT intervention produced >90% reduction in total amount of time spent seizing from baseline to the one-year follow-up (44). Quality of life also significantly improved for the ACT participants as measured by the WHO measure, which includes psychologic health, physical health, environmental health, and quality of social relationships. Interestingly, improvement was not immediately apparent but was significant at the one-year follow-up, with a very large effect size (Cohen's $d = 1.57$). Not only was the outcome positive, but the model itself was supported. For example, the post-treatment score on the AAQEP and the Bull's-Eye both showed a very large effect in favor of the ACT condition, with effect sizes between 1.95 and 3.23. These processes fully mediated the effect seen one year later both in seizure reduction and—more impressively, because these changes emerged only over time—quality of life. For example, the three process scores at post-treatment accounted for 43 to 53% of the variance in quality-of-life outcomes seen a year later, depending on the specific process examined (50).

SUMMARY

In summary, behavior treatments of epilepsy include both specific techniques that manipulate the seizure process and contextual methods of helping the client relate to his or her epilepsy and seizures in ways that allow for living in desired vital life directions. At the very least, behavioral interventions should be used to complement conventional medical therapies. For patients who prefer a nondrug approach or for others who do not have access to anticonvulsant drugs, behavior treatment, which has the most evidence based support of any of the available alternatives, should be considered.

References

1. Temkin O. *The falling illness*. Baltimore: The Johns Hopkins Press, 1945:36–196.
2. Gowers W. *Epilepsy and other chronic convulsive diseases*. London: Churchill., 1881.
3. Brown-Sequard C. *Researches on epilepsy: its artificial production in animals, and its etiology, nature and treatment in man*. Boston: Clapp, 1857.
4. Eriksson T. Jacksonian march. *Arch Neurol Psychiatry* 1940; 43:429.
5. Jackson JH. *Selected writings on epilepsy and epileptiform convulsions*. Taylor J, ed. London: Hodder and Stoughton; 1931.
6. Gelhorn E. Effects of afferent impulses on cortical suppression areas. *J Neurophysiol* 1947; 10:125–138.
7. Leao A. Spreading depression of activity in the cerebral cortex. *J Neurophysiol* 1944; 7:359–390.
8. Leao A. Pial circulation and spreading depression of activity in cerebral cortex. *J Neurophysiol* 1944; 10:409–414.
9. Leao A. Further observations on spreading depression of activity in cerebral cortex. *J Neurophysiol* 1947; 10:409–414.
10. McCulloch W. Mechanism for the spread of epileptic activation of the brain. *Electroencephalogr Clin Neurophysiol* 1949; 1:19–27.
11. Efron R. Effect of olfactory stimuli in uncinate fits. *Brain* 1956; 79:267–281.
12. Forster F. *Reflex epilepsy: behavior therapy and conditional reflexes*. Springfield, IL: Charles C. Thomas; 1977.
13. Forster F, Ptacek L, Peterson W, Chun R, et al. Stroboscopic-induced seizures altered by extinction techniques. *Trans Am Neurol Assoc* 1964; 89:136.
14. Forster F, Campos G. Conditioning factors in stroboscopic induced seizures. *Epilepsia* 1964; 5:156.
15. Lantz D, Sterman M. Neuropsychological assessment of subjects with uncontrolled epilepsy: effects of EEG feedback training. *Epilepsia* 1988; 29:163–171.
16. Birbaumer N, Lutzenberg W, Rockstroh B. Area specific self-regulation of slow cortical potential on the sagittal midline and its effects on behavior. *Electroencephalogr Clin Neurophysiol* 1992; 84:353–361.
17. Rockstroh B, Birbaumer N, Elbert T, Lutsenberger W. Operant control of EEG, event related and slow potentials. *Biofeedback & Self Regulation* 1984; 9:139–160.
18. Lockard J, Ward JA. Epilepsy: a window to brain mechanisms. New York: Raven, 1980.
19. Fenwick P. The behavioral treatment of epilepsy generation and inhibition of seizures. *Neurol Clinic* 1994; 12:175–202.
20. Wolf P. From precipitation to inhibition of seizures: rationale of a therapeutic paradigm. *Epilepsia* 2005; 46:Suppl 1.15–16.
21. Dahl J. *Epilepsy: a behavior medicine approach to assessment and treatment in children*. Göttingen: Hogref & Huber, 1992.
22. Spector S, Goldstein L, Cull C, Fenwick P. Precipitating and inhibiting epileptic seizures: a survey of adults with poorly controlled epilepsy. London: International League Against Epilepsy, 1994.
23. Tan S, Bruni J. Cognitive-behavior therapy with adult patients with epilepsy: a controlled outcome study. *Epilepsia* 1986; 27:255–263.
24. Pushkarich C, Whitman S, Dell J, Hughes J, et al. Controlled examination of effects of progressive relaxation training on seizure reduction. *Epilepsia* 1992; 33:675–680.
25. Ramaratnam S, Sridharan K. Yoga for epilepsy. In: The Cochrane Library 1. Oxford: Update Software, 1999.
26. Dahl J, Brorson L-O, Melin L. Effects of a broad-spectrum behavioral medicine treatment program on children with refractory epileptic seizures: an 8-year follow-up. *Epilepsia* 1992; 33(1):98–102.
27. Dahl J, Melin L, Brorson LO, Schollin J. Effects of a broad-spectrum behavior modification treatment program on children with refractory epileptic seizures. *Epilepsia* 1985; 26(4):303–309.
28. Betts T. An olfactory countermeasures treatment for epileptic seizures using a conditioned arousal response to specific aromatherapy oils. *Epilepsia* 1995; 36(Suppl 3): 130–131.
29. Betts T, Fox C, MacCallum R. Assessment of countermeasures used by people to attempt to control their own seizures. *Epilepsia* 1995; 36(Suppl 3):130.
30. Dahl J, Melin L, Leissner P. Effects of a behavioral intervention on epileptic seizure behavior and paroxysmal activity: a systematic replication of three cases of children with intractable epilepsy. *Epilepsia* 1988; 29(2):172–183.
31. Alajouanine F. Dostoyevski's epilepsy. *Brain* 1963:86; 214–221.
32. Mostofsky D, Balaschak B. Psychobiological control of seizures. *Psychol Bull* 1977; 84:723–750.
33. Kraft K, Poling A. Behavioral treatments of epilepsy, methodological characteristics and problems of published studies. *Appl Res Ment Retard* 1982; 3:151–162.
34. Goldstein L. Behavioral and cognitive behavioral treatments for epilepsy: a progress review. *Br J Clin Psychol* 1990; 29:257–269.
35. Ramaratnam S, Baker GA, Goldstein LH. Psychological treatments for epilepsy. *Cochrane Database Syst Rev* 2003; 4.
36. Dahl J, Melin L, Lund L. Effects of a contingent relaxation treatment program on adults with refractory epileptic seizures. *Epilepsia* 1987; 28(2):125–132.
37. Fried R. Rubin S, Carton R, Fox M. Behavioral control of intractable seizures; self regulation of end tidal carbon dioxide. *Psychosom Med* 1984; 46:315–331.
38. Lindsay W, Baty F. Behavioral relaxation training: exploration with adults who are mentally handicapped. *Ment Handicap* 1986; 15:159–162.
39. Montgomery J, Epsie C. Behavioral management of hysterical pseudo seizures. *Behav Psychother* 1986; 143:34–40.
40. Rosseau A, Hermann B, Whitman S. Effects of progressive relaxation on epilepsy: analysis of a series of cases. *Psychol Rep* 1985; 57:1203–1212.
41. Hayes SC, Barnes-Holmes D, Roche B. *Relational frame theory: a post-Skinner account of human language and cognition*. New York: Plenum Press, 2001.

42. Dahl J, Wilson KG, Nilsson A. Acceptance and Commitment Therapy and the treatment of persons at risk for long-term disability resulting from stress and pain symptoms: a preliminary randomized trial. *Behav Ther* 2004; 35:785–802.
43. Gregg JA. A randomized control effectiveness trial comparing patient education with and without Acceptance and Commitment Therapy. Ph.D. diss., University of Nevada, Reno, 2004.
44. Lundgren T, Dahl J, Melin L, Kies B. Evaluation of Acceptance and Commitment Therapy for drug refractory epilepsy: a randomized trial in South Africa: a pilot study. *Epilepsia* 2006; 47:2173–2179.
45. Lundgren T. A development and evaluation of an integrative health model in the treatment of epilepsy. Master's thesis, University of Uppsala, Sweden, 2004.
46. WHO. WHOQOL-BREF: introduction, administration and generic version of the assessment. Geneva: World Health Organization, Program on Mental Health, 1996.
47. Diener E, Emmons RA, Larsen RJ, Griffin S. The Satisfaction With Life Scale. *J Pers Soc Psychol* 1985; 49:71–75.
48. Hayes SC, Strosahl KD, Wilson KG, Bissett RT, et al. Measuring experiential avoidance: A preliminary test of a working model. *Psychol Rec* 2004; 54:553–578.
49. Lundgren T, ed. Validation of the Bulls-Eye. Presented at the 2nd World Congress of Acceptance and Commitment Therapy and Relational Frame Theory; July 23–28; 2006, London.
50. Dahl J, Lundgren T. Behavior analysis of epilepsy: conditioning mechanisms, behavior technology and the contribution of ACT. *The Behavior Analyst Today* 2005; 6(3):191–202.