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Alternative and Complementary Strategies for Epilepsy

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WHAT IS EPILEPSY?

Epilepsy affects 1 in 2000 people.⁸ It is the second most common neurological disorder after stroke but is still little understood. It is not a simple disorder, but a group of approximately 30 central nervous system (CNS) disorders. (The term "epilepsy" may be thought therefore as an umbrella term.)

The disorder has a lifetime cumulative incidence of 3%, and is characterised by certain clinical and electroencephalographic (EEG) abnormalities. Seizures can develop as a symptom in consequence of several CNS disorders - for example, brain anoxia (lack of oxygen to the brain), and assume a wide variety of forms ranging from a loss of consciousness to convulsive moments. Largely genetic, the disorder can be precipitated by various insults to the brain. The feature common to all seizures is an abnormal synchronous discharge from millions of neurons in the brain, perhaps resulting from abnormal reverberating circuits.

Given sufficient circumstances, any person will have a seizure. The amount of stimulation required to cause a seizure is called the *seizure threshold*.

Many people with epilepsy are considered to have a low seizure threshold.

The discharges stimulate many of the neurons to send nerve impulses over their conduction pathways. As a result, the person having the attack may contract skeletal muscles involuntarily, and certain portions such as the reticular activating system (RAS), may shut down during the attack. Medical treatment therefore, generally involves the prevention of this abnormal discharge or reducing its spread.

Stedman's Medical Dictionary cites epilepsy as being "a chronic disorder characterised by paroxysmal brain dysfunction due to neuronal discharge, and usually associated with some alteration of consciousness. The clinical manifestations of the attack may vary from complex abnormalities of behaviour including generalised or focal convulsions to momentary spells of impaired consciousness. These clinical states have been subjected to a variety of classifications, none universally accepted to date, and accordingly, the terminologies used to describe the different types of attacks remain purely descriptive and non-standardised; they are variously based upon:

1. The clinical manifestations of the seizure (motor, sensory, reflex, psychic or vegetative)
2. The pathologic substrate (heredity, inflammatory, degenerative, neoplastic, traumatic, or cryptogenetic)
3. The location of the epileptogenic lesion (rolandic, temporal, diencephallic regions of the brain)
4. The time at which the attacks occur (nocturnal, diurnal, menstrual, etc.)"

CLASSIFICATIONS OF EPILEPSY

Stedman's goes on to describe a multitude of 'classifications' according to the specific nature of the attack, but generally, epilepsy can be categorised into the following main groups as summarised from a variety of sources:

ABSENCE (previously known as *petit mal*)

This type of seizure is most common in children and teenagers.

It is characterised by a blank stare lasting about half a minute or more, and the person appears to be day dreaming. Very young or elderly suffers may drool. Sometimes, there is little outward sign that anything is wrong. During this type of seizure, the individual is unaware of their surroundings. Usually, there is a rapid return to normal after two or three minutes. Staring or daydreaming in children should not be confused with absence seizures. If a child is daydreaming, he or she can be aroused by simple touch or by someone talking to them. A child having an absence seizure can not.

ATONIC (drop attack)

A childhood seizure in which the child loses consciousness for about ten seconds and usually falls to the ground due to a complete loss of muscle tone.

COMPLEX PARTIAL (temporal lobe, or psychomotor)

A blank stare, automatic or random activity, and a chewing motion are characteristic of this type of seizure. The person may be dazed and unaware of their surroundings, and may act oddly. There is no memory of this seizure. A person may experience a distinct warning sign called an aura in this type of seizure. The aura is itself a form of partial seizure, but one in which the person retains awareness. The *aura* may be experienced as a peculiar odour, "butterflies" in the stomach, or a distorted sound.

GENERALISED TONIC-CLONIC (previously known as *grand mal*)

This type of seizure is characterised by a sudden cry (as the diaphragm often spasms, forcing air from the lungs), a fall, momentary rigidity and then jerking of the muscles, uncontrolled spasmodic movement of the head limbs and body, shallow breathing, and bluish skin (cyanosis). Loss of bladder control is also possible. It usually lasts two to five minutes, and is followed by confusion, drowsiness and fatigue, and/or memory loss. It can be frightening to witness, especially for the first time observer.

MYCLONIC

Brief, but massive muscle jerks occur.

SIMPLE PARTIAL (Jacksonian)

Jerking begins in the fingers and toes and progresses up through the body. It produces a sudden shock-like jolt to one or more muscles which increases muscle tone and causes the sudden jerky movements (similar to those that sometimes occur in healthy people as they go to sleep). The person remains conscious.

SIMPLE PARTIAL (sensory)

The person may see, hear, or sense things that do not exist. This may also occur as the preliminary symptom (aura) of a generalised seizure.

FEBRILE CONVULSIONS

When a child has an abnormally high fever from an infection, the body temperature rises to the point where the child's immature temperature regulation mechanism cannot cope. At this critical point, the child convulses in what appears to be seizure, or 'fit'. The seizure only lasts for a few minutes, and generally there appears to be no lasting effect on the child. Febrile convulsions are common, and approximately 3% of children aged 6 months to six years may have a convulsion when they have a high temperature.

Usually, there is a previous history of infection, the child will normally be quiet, and appear sick, skin is flushed and hot to the touch, eyes 'roll back', may become stiff or floppy, becomes stiff and prostrate, and begins convulsing, salivary drool, becomes cyanosed (turns bluish) due to absent breathing (loss of control of the diaphragm). After one to three minutes, the child usually starts to breathe again, recovers and commences crying. You should seek medical advice.

PSYCHOGENIC NONEPILEPTIC SEIZURES

Psychogenic nonepileptic seizures (PNES), or pseudoseizures are paroxysmal episodes that resemble and often misdiagnosed as epileptic seizures; however, PNES are psychological in origin, though nonteleless real to those suffering them. Paroxysmal nonepileptic episodes can be either organic or psychogenic.

PNES are common at epilepsy centres, where they are seen in 20-30% of patients referred for refractory seizures.

Misdiagnosis of epilepsy is common. Misdiagnosis occurs in approximately 25% of patients with a previous diagnosis of epilepsy that does not respond to drugs. Most cases of misdiagnosed epilepsy are eventually shown to be PNES. Other paroxysmal conditions are occasionally misdiagnosed as epilepsy, but PNES is by far the most commonly misdiagnosed condition, accounting for >90% of misdiagnoses at epilepsy centres. EEGs interpreted as providing evidence for epilepsy often contribute to this misdiagnosis (Benbadis, 2003).

Reversing a misdiagnosis of epilepsy can be difficult, as it is with other chronic conditions. Unfortunately, after the diagnosis of seizures is made, it is easily perpetuated without being questioned, which explains the usual diagnostic delay and cost associated with PNES. Despite the ability to diagnose PNES with near certainty by using EEG video monitoring, the time to diagnosis is long, about 7-10 years. This delay indicates that neurologists may have an insufficiently high enough index of suspicion for PNES.

Resistance to antiepileptic drugs (AEDs) is usually the first clue and the reason for referral to the epilepsy center, though intractable epilepsy is a common cause of resistance to AEDs.

Stress is the most common trigger of a psychogenic seizure.

WHAT CAUSES EPILEPSY?

Seizures are the most common neurological problem affecting children. One third of all people with seizure disorder are children.²

Idiopathic epilepsy (seizures of unknown cause) or febrile seizures (non-epileptic seizures induced by a fever) affect about 3% of children. Seizures in very young children often stem from brain injury before or during birth, damage to the central nervous system, or metabolic inconsistencies. In older children, onset of epilepsy is more likely to result from genetic factors, exogenic (environmental) factors, infections of the central nervous system or head injury.

Not everyone who has a seizure has epilepsy.

The actual causes are still relatively unknown. The spontaneous discharges characteristic of epilepsy may occur for *no apparent reason*, or may be triggered by a wide range of things, including exposure to an allergen; congenital defects; lead poisoning or injury during childbirth; use of or intoxication from alcohol or drugs, drug or alcohol withdrawal; fever; neurocysticercosis (encystment of cysticercus larvae of some tapeworms [eg. *Taenia solium*, or *T. saginata*] in brain tissue)²¹; flashing lights; hunger; hypoglycemia (low blood sugar level); hypocalcemia (low levels of calcium in the blood); uremia (excessive amounts of urea and other nitrogenous wastes in the blood); hypoxia (lack of oxygen in the blood); infection; vascular disturbances (hemorrhage, hypotension - low blood pressure); lack of sleep; metabolic or nutritional imbalances (diabetes mellitus complications; electrolyte imbalances, kidney failure, uremia, phenylketonuria, uremia (toxic accumulation of wastes); physical or emotional trauma.

Brain injury is also a cause and may affect any age, with highest incidence in young adults, and is most likely if the brain membranes are damaged. Seizures usually begin within 2 years after the injury. Early seizures [within 2 weeks of injury] do not necessarily indicate that chronic seizures [epilepsy] will develop.

Tumours and abscesses of the brain that occupy space (such as haematomas), may affect any age, more common after age 30 - partial (focal) seizures are the most common type initially, and may progress to generalized tonic-clonic seizures.

Disorders affecting the blood vessels such as stroke, transient ischemic attack (TIA), are the most common cause of seizures after age 60.

Infections may be a reversible cause of seizures and may affect all ages. (Brain infections [meningitis, encephalitis]; brain abscesses; acute severe infections of any part of the body; chronic infections (such as neurosyphilis); complications of AIDS or other immune disorders.

Genetic factors are thought to contribute to the aetiology in up to 60% of cases. Various molecular and cellular mechanisms give rise to epilepsy, and epilepsy genes fall into several distinct categories. They include genes in which mutations cause abnormal ion-channel function, disordered brain development, progressive neurodegeneration and disturbances of cerebral energy metabolism.²³

As mentioned, seizures occur when there is an excess of excitatory processes in the brain compared with inhibitory processes. Changes in afferent excitation, disinhibition, shifts in extracellular ion concentrations, voltage-gated ion-channel opening and enhanced neuronal synchrony are all important in the initiation and propagation of seizure activity. Neuronal activity is regulated by the concentration of ions in the extracellular and intracellular spaces, and the selective flux of these ions across the neuronal membrane. Voltage-gated or ligand-gated ion-channel genes are, therefore, attractive candidate genes for the epilepsies. Mutations in such genes could lead to channel dysfunction, which could alter ion concentrations across the cell membrane, resulting in reduced or increased neuronal excitability.²³

The vast majority (70%) of seizures are in fact idiopathic - the cause is simply unknown.

Some common pesticides such as dieldrin, lindane, and the pyrethroids, inhibit gamma aminobutyric acid (GABA) receptors and promote convulsions in susceptible individuals by binding with benzodiazepine receptors.⁸

Recent reports suggest that adult onset of idiopathic epilepsy (of no apparent cause) may be due to minor strokes or to be a forewarning of one.

After a first attack, a full medical workup, including an electroencephalogram (EEG) and a [QEEG](#) needs to be conducted. The QEEG is best done on the 'unmedicated brain' as this will enable the clinician, along with EEG practitioners to target training sites for [neurofeedback](#). Further investigation including functional Magnetic Resonance Imaging (fMRI) may need to be done.

IF YOU ARE PRESENT WHEN SOMEONE IS HAVING A SEIZURE

When witnessing a seizure, try to remember exactly what happened, such as:

- Did limbs twitch? If so, which ones and on which side.
- Did the head twist, neck go rigid, eyes turn or roll?
- Was there drooling or foaming at the mouth?
- Was there a chewing motion or smacking of the lips?
- Was there any change in consciousness?
- Did the victim bite the inside of the cheek, tongue or lips?
- Was there loss of bladder or bowel control?

If there was no twitching at all, just a sudden blanking out, as in a faint, then it may have been a faint if the victim recovered promptly after lying down. In an epileptic seizure, it usually takes minutes or hours to recover. Noting these things will be of use when medical advice is sought.

- Do not try to restrain the person.
- Do not try to put anything into the person's mouth. A person who is having a seizure may bite their tongue - but this is not life threatening. (The story about swallowing the tongue is irrelevant - control of the diaphragm is lost during the seizure, so the person cannot breathe anyway. Upon cessation of the seizure, reflex usually takes over and the tongue will move forward and the person will breathe normally).
- If possible try to ease a person's fall so that the person does not hit anything as they collapse. Sometimes people will let you know that they are about to have a seizure, so you can ask them to sit or lie on the floor before a fall.
- Leave the person lying flat on a safe surface, move any furniture or other objects out of the way so that the person who is having the fit will not injure themselves, and if on a hard surface like tiles or concrete, carefully so as not to injure the person or yourself, place padding beneath the head.
- **DO NOT PANIC!** Loosen any tight clothing and stay with the person until the seizure has stopped. The person will be confused and tired after the seizure.
- Allow the seizure to run its course without interference.
- In case of loss of bladder control, loosely cover the person so as to protect their privacy and dignity.
- Immediately upon cessation of the seizure, check their airway is clear and that they are breathing, turn the person onto their side in a stable position and allow them to recover quietly. Allow them to sleep if they need. When they come round, gently ask what assistance may be required and assist if necessary.

- If the person has repeated seizures, one after another, seek medical assistance, as this may be the onset of status *epilepticus* and will have serious consequences if left untreated. With a baby or young child, or an elderly person, medical help should be sought immediately.
- If it is the first time the person has had an epileptic attack, they should see their doctor for a full medical workup and referral for brain scan / EEG to check there is no predisposing cause.

STRATEGIES IN THE TREATMENT OF EPILEPSY

Modern medical practice treats epilepsy with anticonvulsant drugs which include:

- Carbamazepine (Tegretol, Teril, Carbium)
- Sodium valporate (Epilim, Valpro)
- Phenytoin sodium (Dilantin)
- Phenobarbitone
- Acetazolamide (Diamox)
- Tiagabine (Gabitril)
- Primidone (Mysoline)
- Gabapentin (Neurontin)
- Sulthiame (Ospolot)
- Clonazepam (Rivotril)
- Topiramate (Topamax)
- Ethosuximide (Zarontin)
- Lamotrigine (Lamictal)

Some of the more common side effects of these drugs include:

- Irritability, aggression, agitation
- Haematological abnormalities
- Hyperkinesia in children
- Anorexia
- Gastrointestinal disorders
- Sleep disturbances
- Headaches
- Vertigo and in coordination

- Mental confusion
- Ataxia
- Depression
- Respiratory Depression

If one consults the MIMS (An annual publication of approved pharmaceuticals utilised by health care professionals in Australia), for the majority of drugs quoted as being effective in the management of epilepsy, the actual action of the drug in prevention of the seizures is unknown. There are many more, less common but nevertheless published potential side effects of the drugs listed, and there is clear admission that the drugs are not a cure - they purely mask the symptoms.

The majority of the anticonvulsant drugs are competitive inhibitors of biotin transport in the human intestine. They also tend to reduce tissue levels of vitamin E, as well as reducing levels of B12 and folate within the body. Depending upon the chemical composition the particular anticonvulsant being ingested, other essential nutrients are depleted as well. In essence, anticonvulsant drug therapy blocks the sodium ion (Na⁺) potentials, thus inhibiting transmission of the synaptic impulse along the nerves. It is interesting to note that this is exactly what tetrodotoxin - the poison of the puffer fish does, effectively shutting down all autonomic body processes.

Further reading suggestions:

- [Synopsis of findings of studies in the treatment of epilepsy with medication](#)

A page of concise reports of findings - essential reading!

[Epileptic women pregnancy danger](#) Sourced from the BBC News

The health of pregnant women and their unborn children is being threatened by a lack of information about treatments, a survey has found. The British Epilepsy Association (BEA) quizzed 2,000 women with epilepsy.

[Babies of epileptic women at risk](#) Sourced from the BBC News

Babies born to women taking epilepsy medication through pregnancy are at treble the risk of learning disorders and birth defects, a study has shown.

It is VITALLY IMPORTANT that persons taking these drugs do NOT discontinue their use without consulting a health care professional!

Coming off too quickly can produce a rebound effect and severe withdrawal symptoms compounding the initial problem even further in some instances.

SEVERE CASES OF EPILEPSY

Occasionally, epileptics have described a period of intense emotion, and uncontrollable, sometimes violent behaviour just prior to, and in onset of the seizure. Whilst this is relatively uncommon, it was reported in a case study by neurologists Vernon Mark and Frank Ervin (1970), and has since become known as loss-of-control-syndrome. In other cases, the sensations of onset of the seizure (often referred to as the aura), have been interpreted by the individual as ecstasy of the highest order.

In severe cases of intractable (not adequately controlled by medication) temporal tonic-clonic seizure, neuroscientists/surgeons have performed bilateral stereotactic amygdalotomies, the lesions so produced blocking or interrupting discharge from the amygdala to hypothalamus which is reported to be 'successful' in 50%-80% of cases.⁷ This begs the question about the other 20%-50%.

Further reading suggestion:

[Epilepsy 'master gene' found](#)

The Australian team behind the research said their discovery was likely to impact most on families and isolated cases with "non-specific" learning difficulties.

THE ALTERNATIVES

There are alternatives and complementary strategies to medication (or surgery) for the amelioration of epilepsy.

In one school of thought, it is considered that epilepsy may be a self-defense mechanism of the brain to avoid anoxia.

Evaluation by a qualified osteopath, or craniosacral therapist for 'structural' deficiencies is therefore prudent since the musculoskeletal system may be causing pinched nerves or blood vessels, as a result of 'locking' or 'armouring' mechanisms, or through a genetic/inherited predisposition to malformations etc. These conditions may reduce fluid flow in the body and subsequently, in the brain.

Evaluation for chemical deficiencies and/or toxicity by a qualified naturopath or nutritionist is also desirable. This should be followed through with a dietary and exercise regime within the limits of the individual, that is, tailored to the individual, as it will be beneficial to enhance cardiac fitness and hence blood supply and oxygenation of the tissues.⁸

HYPERBARIC OXYGEN THERAPY

Hyperbaric Oxygen Therapy is reported to have had some good results in assisting people with epilepsy.²

NUTRITIONAL

Common nutritional deficiencies quoted with epilepsy are manganese, zinc, and magnesium, so that nutritional analysis and supplementation where necessary is vital to successful management.⁸

Vitamin E supplementation alone has been quoted as reducing seizure rates by as much as 50%. Selenium supplementation may also be of benefit.⁸

It is essential that anyone taking anticonvulsant medications take adequate supplementation of nutrients that are likely to be depleted by both the condition and the interventional drug under the supervision of a qualified health care professional. Equally, it will be important to continually monitor liver and other bodily functions through regular medical checkups as these drugs play havoc with the body's homeostatic mechanisms. Unfortunately, a large number of medical practitioners demonstrate remarkable incompetence when it comes to clinical nutrition.⁵

Functional / Nutritional Considerations to the management of epilepsy might include:

1. Avoidance of all gluten containing grains (wheat, rye, barley oats, triticale)
2. Avoidance of monosodium glutamate spiced foods (ie. Sausage, salami, Chinese takeaways etc.).
3. Avoidance of stimulants (tea, coffee, alcohol).
4. Reduce stress through a relaxation technique and adequate exercise.
5. Check for food, chemical sensitivity and / or toxicity - especially pesticide exposure.
6. Breast feed infants if possible. (Cows milk has less taurine, therefore infants fed with this may have a lower threshold to febrile convulsions)
7. Check for blood pressure or arteriosclerosis and minister to appropriately.
8. Avoid pesticides.
9. Increase magnesium rich foods and/or supplement with magnesium.
10. Viral infection can mobilise the release of dieldrin and lindane (pesticides) that may be stored in the body's (fat) lipid reserves. This then increases the probability of convulsions in susceptible individuals during the period of infection.
11. A detoxification diet may be of benefit to reduce the body's stores of xenobiotics.
12. Improving brain oxygenation by improving fitness and lung function.

OTHER CONSIDERATIONS MAY INCLUDE

- Specifically working toward self-care and working toward becoming as free from drugs and seizures as possible. Make it a point to learn about your condition and the alternatives to management. Being aware of the drug you are taking. Know its potential side-effects and interaction with foods, herbal preparations, or other drugs you may be taking. How does this drug interact with your body and mind?
- Increasing intake of beet greens, chard, eggs, green leafy vegetables, raw nuts, seeds.
- Drinking "live", fresh juices made from beets, carrots, green beans, green leafy vegetables, peas, red grapes, and sea-weed for concentrated nutrients.
- Eating smaller and more frequent meals, not drinking large quantities of liquids all at once, and taking two tablespoons of olive oil daily. Supplementation of the vitamins A, B, C, & E, and other vitamins/minerals etc. that may be depleted by the condition or drug. Investigation of metabolics will be necessary to arrive at individualised requirements for supplementation.

Further reading suggestion:

- [Nutritional Depletion as a Side Effect of Anticonvulsant Medications](#)

An informative mini-article about some of the nutrients depleted by the more commonly prescribed medications

HERBAL REMEDIES

Herbal remedies have a long history in alleviating some forms of epilepsy. It is recommended that you seek the advice of a qualified, experienced and specialist herbalist who understands both the manifest forms of epilepsy and the potential for drug/herb interactions.

QEEG BIOFEEDBACK (NEUROFEEDBACK)

[QEEG biofeedback](#) had its origins in seizure with the work of Professor M. Barry Stermán of UCLA. He was the first researcher to isolate the sensorimotor rhythm (SMR) of 12-15 Hz in his early experimentation with 10 cats who were trained to produce this brainwave frequency at will. The SMR state is exhibited clinically as relaxed, but alert attention. At the time, his findings were considered interesting, but what was the clinical value? As with all breakthrough discoveries, only time and serendipity would tell.

He was later called in to assist in a project with the US army to research monomethylhydrazine, or rocket fuel. This substance is highly neurotoxic and workers who breathed in its fumes suffered nausea, severe epileptic seizures, and eventually death.

In this new study Stermán brought in 50 cats. The cats were exposed to the rocket fuel and minutes after, all did the same thing: they vomited, made noises, salivated, and panted. Most went into grand mal epileptic seizure after one hour - most, but not all. The 10 cats from Stermán's original work were included in the 50 cats. Seven of these SMR trained cats displayed significant delay in the onset of seizure, and three did not fit at all. The SMR training had raised their seizure threshold.

In epileptics, a portion of the brain is unstable, or hyperexcited and cannot resist as slow theta waves, in the 4-8 Hz range start to creep in. This in turn recruits other brain regions to produce the abnormally low frequency. During an epileptic seizure, it is as if all the musicians of an orchestra are playing at once, but *without* a score or conductor. Normal motor function is disrupted.

Everyone passes through the SMR state all the time, a split second here and perhaps a few seconds there. What biofeedback or more specifically, EEG Biofeedback / Neurofeedback training does is to help the individual dwell in that state for longer periods of time. Dwelling there is what teaches the cortex to maintain stability thus raising the seizure threshold.

The results of clinical use of EEG biofeedback (Neurofeedback) in the management of epilepsy are truly remarkable, Stermán's first biofeedback/epilepsy case was deemed by doctors to be intractable. The SMR training was so successful that the person was medication and seizure free for three months afterward - this was totally unprecedented for this particular type of epilepsy¹¹. Surprisingly, there were added rewards as a result of the SMR training by way of increased cognitive function; enhanced sense of self and personal confidence. Stermán's paper on the case was published in the prestigious journal of the *EEG and Clinical Neuroscience Society* (ECNS).

The patients participating in subsequent studies said Sterman, "represented a most difficult subset of epileptics. Typically, individuals with severe seizure disorders of relatively long duration, often with significant comorbidities, and uniformly refractory to anticonvulsant medications."¹³ Further, most remained on medications, a condition that not only distorts the EEG but also impacts considerably on their ability to acquire new information.¹⁷

In the paper, Sterman goes further to say "82% of the subjects of the study demonstrated significant (>30%) seizure reduction, with an average value exceeding 50%. There was also significant reduction in seizure severity. Approximately 5% of patients experienced complete control for over 1 year even when anticonvulsants were subsequently reduced or entirely withdrawn".

The consensus arising from the findings was that "most epileptic patients who show clinical improvement with EEG biofeedback also show contingency related EEG changes and a shift towards EEG normalisation. However, not all clients who respond to this training show expected EEG changes, and a few clients who show EEG changes experience little clinical improvement. One is reminded of the fact that a similar percentage of people undergoing anterior temporal lobectomy for surgical treatment of complex-partial seizures failed to show the expected hippocampal sclerosis or other lesions in microscopic studies of the tissue removed. Further, 27% of those with documented lesions showed little clinical improvement."

Both EEG biofeedback (Neurofeedback) and anterior temporal lobectomy treatments are confounded by our relatively primitive comprehension of neural regulation and seizure pathology, and by the limitations of current analytic methodology.

Estimates of adequate control with medications in the largest subset of epileptics, namely those with localisation-related partial seizures, have been as low as 30%.¹⁸ With nocturnal primary generalised myclonic epilepsy, control has also been estimated at 30%.¹⁹ Since available medications have not been effective, further titration of the dosage will likely not be productive. Turning to new "experimental" drugs is no certain panacea, and can introduce certain risk factors.¹³

In real terms, EEG biofeedback (Neurofeedback) can reduce seizure rates by as much as 60% in severe cases¹¹.

Frank Duffy, neurologist at the Children's Hospital, Boston, states in his editorial of the journal *Clinical EEG*, Vol.31, No.1, January 2000, ***"The literature, which lacks any negative study of substance, suggests that EEG Biofeedback should play a major therapeutic role in many difficult areas. In my opinion, if any medication had demonstrated such a wide spectrum of efficacy it would be universally accepted and widely used"***.²²

Given the 30 years of peer reviewed research demonstrating impressive EEG and clinical results with the most refractory subset of seizure patients it is no longer acceptable to view EEG operant conditioning as "experimental" as this view is neither rational, objective or in the best interest of this population.¹³

It is interesting to note that [QEEG \(Quantitative Electroencephalography\) and Neurofeedback](#) training have been approved in the state of Texas in September 2001. The mandated bill makes it mandatory for American health insurance companies to pay for QEEG assessment and Neurofeedback training in cases of traumatic brain injury. Hopefully, with further demonstration of it's efficacy this will also be the case with epilepsy - where Neurofeedback has it's roots.

HAEMOENCEPHALOGRAPHY (HEG)

The relatively new field of haemoencephalography (HEG), studies cortical haemodynamics: vascularity, blood volume, oxygenation, metabolism or temperature in real time.

Through the use of a near infrared spectrophotometer (NIRS) and a feedback unit, a person can easily learn to direct blood flow in the brain at will. These vascular variables are in fact voluntarily controllable, hence HEG's usefulness as a therapeutic exercise agent in biofeedback.

Given the issue of anoxia being implicated in epilepsy, one can easily see why this training will be useful to the epileptic person, provided of course that aetiology is compatible.

HOMEOPATHY

There are homeopathic remedies available addressing many forms of epileptic attack. A specialist homeopath should be consulted for an all-embracing remedy that will include the rest of the person's constitution as well as their epilepsy.

FURTHER READING SUGGESTIONS

- Quantitative Electroencephalography - (QEEG)
- Neurofeedback - EEG Biofeedback - a Drug-Free Strategy for ADHD, Learning Disorders and Other Conditions
- Synopsis of findings of studies in the treatment of epilepsy with medication
- Nutritional Depletion as a Side Effect of Anticonvulsant Medications
- QEEG and Neurofeedback - diagnostic and training modalities for the enhancement of CNS functioning in ADHD and other disorders

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LINKS

PLEASE NOTE :

Learning Discoveries offers the links below as a convenience to our clients and the users of this website. However, we do not control third party websites and we are not responsible for the websites content.

- Epilepsy Action of Australia
(Previously known as the Epilepsy Association of Australia)

<http://www.epilepsy.org.au/index.asp>

The Epilepsy Action website has a wealth of information concerning epilepsy in all its forms. The site also includes complementary strategies such as biofeedback and other 'alternative' interventions into epilepsy. It should be noted though, that Neurofeedback (EEG biofeedback) is the only recognised form of biofeedback that is supported by more than thirty years of research into amelioration of epilepsy.

- Epilepsy Research Centre
(Previously known as the Epilepsy Research Institute)

<http://www.epilepsyresearch.org.au/>

This is Australia's leading clinical epilepsy research group.

- Epilepsy Society of Australia

<http://www.epinet.org.au/>

The Epilepsy Society of Australia is a professional organisation for clinicians, scientists and technologists involved in the diagnosis, treatment and research of epilepsy in Australia.

- Epilepsy Queensland Inc

<http://www.eqj.org.au/about/index.htm>

Since 1969, Epilepsy Queensland Inc has been dedicated to improving the quality of life of people with epilepsy and their carers / families through advocacy, research, support and information. Considerable effort is put into increasing public awareness and raising community understanding of epilepsy.

- Epilepsy Foundation of Victoria (A member of Epilepsy Australia)

<http://www.epinet.org.au/>

The Epilepsy Foundation of Victoria is dedicated to providing high quality information, advice, support, training, counselling and practical assistance, as well as undertaking research and advocacy on behalf of people with epilepsy. They work to raise awareness of epilepsy in the community to reduce the stigma and create a more welcoming and inclusive society.

- Children's Neurobiological Solutions, Santa Barbara, California, USA

<http://www.cnsfoundation.org/site/PageServer>

CNS is a non profit research foundation improving the lives of children disabled by neurological disorders through research focused on brain repair and regeneration.

REFERENCES

1. Andreassi, J.L., Psychophysiology - Human Behaviour & Physiological Response, 1995, Lawrence Erlbaum & Associates, Hillsdale, New Jersey.
2. Balch, J.F., (MD) & Balch, P.A. (CNC), Nutritional Healing, 2000, Penguin, Putnam In., New York, NY.
3. Bland, J.S., (Ph.d) et al, Clinical Nutrition: A Functional Approach, 1999, The institute for Functional Medicine, Gig Harbour, Washington.
4. Evans, R.E, & Abarbanel, A., Quantitative EEG and Neurofeedback, 1999, Academic Press, San Diego, CA.
5. Galland, L. (MD), The Four Pillars of Healing, 1997, Random House, New York, NY.
6. Kimble, D.P., Biological Psychology, 1998, Harcourt Brace Publishers, Orlando, FL.
7. Miller, B.L. & Cummings, J.L., The Human Frontal Lobes - Functions & Disorders, 1999, The Guilford Press, New York, NY.
8. Osiecki, H. (B.Sc.(hons) Grad.Dip. Nutrition & Dietetics), The Physician's Handbook of Clinical Nutrition, 1998, Bioconcepts Publishing, Kelvin Grove, Queensland, Australia.
9. Durlach, J. et al, 1987, The Control of Central Neural Hyperexcitability in Magnesium Deficiency, Nutrients and brain Function, Karger Publishers.
10. Pugh, M.B. (Senior Editor), Stedman's Medical Dictionary, 2000, Lippincott, Williams & Wilkins, Baltimore, Maryland.
11. Robbins, J., A Symphony in the Brain, 2000, Atlantic Press, New York, NY.
12. Sernberg, R.J., In Search of The Human Mind, 1994, Harcourt Brace Publishers, Orlando, FL.
13. Serman, B., Basic Concepts and Clinical Findings in the Treatment of Seizure Disorders with EEG Operant Conditioning, 2000, Journal of EEG and Clinical Neuroscience Society, Vol. 31 No.1, January, Wheaton ILL.
14. Tortora & Grabowski, Principles of Anatomy & Physiology, 1993, Harper Collins Publishers, New York, NY.

15. Upledger, J.E. (DO, OMM), *A Brain is Born*, 1996, North Atlantic Books, Berkley, CA.
16. Webb, P., (Dr), *Encyclopaedia of Homeopathic Remedies*, 1997, The Book Company International Pty Ltd, Sydney, Australia
17. Thompson, P.J. & Trimble, M.R., *Anticonvulsant serum levels; relationship to impairments and cognitive functioning.*, 1983, *Journal Neurology, Neurosurgery & Psychiatry*, Vol 46 p.p.227-233
18. DeVivio, D.C., 1983, *How to use other drugs (steroids) and the ketogenic diet* In *Anti-epileptic Drug Therapy in Paediatrics*, Morselli, Pippenger & Penry editors, Raven Press, New York, NY, pp 239-248
19. Touchon, J., 1982, *Effect of awakening on epileptic activity in primary generalised myoclonic epilepsy.* In: Serman MB, Shouse, M.N, Passouant, P. (editors) *Sleep and Epilepsy*, Academic Press, New York, NY.
20. 2000 MIMS Annual, 2000, Havas medimedia Australia, St. Leonards, NSW, Australia.
21. Carpio A, Escobar A, Hauser WA. *Cysticercosis and epilepsy: a critical review.* *Epilepsia.* 1998;39:1025-1040.
22. Duffy, Frank *Clinical Electroencephalography.* Vol.31, No.1, January 2000, p v-viii.
23. Fisher, R.S. (1995) *Cellular mechanisms of the epilepsies.* In *Epilepsy.* (2nd edn) (Hopkins, A., Shorvon, S.D. and Cascino, G., eds), pp. 35-58, Chapman and Hall Medical, London, UK