

Epilepsy
Updated: 08/26/2004

ABSTRACTS

The involvement of taurine in the action mechanism of sodium valproate (VPA) in the treatment of epilepsy.

Anyanwu E, Harding GF.

Acta Physiol Pharmacol Ther Latinoam. 1993; 43(1-2):20-7.

Several lines of evidence have shown that sodium valproate (VPA) mechanism of action in the therapy of epilepsy is based on the phenomena of its interaction with neurotransmitters (GABA), receptor sites and ion channels (1). However, there is no conclusive evidence to show the extent of VPA interactions with other neurotransmitters in the brain. Based on this fact, taurine (an amino acid 'neurotransmitter') found distributed in the brain the visual system may probably be involved in the drug action mechanism of VPA. The application of taurine in experimental and human epilepsy started over thirty years ago (2,3) and it has been known to possess some mild anticonvulsant activity in both humans and experimental animal models (4). This review, therefore, will attempt to draw together all the available information on the involvement of taurine in epilepsy and its possible association with the action mechanism of VPA in suppressing epileptic seizures. Structural and physiological distribution of taurine in the brain will be discussed. Its association with the phenomena of VPA action in epilepsy will be cited. Its neurotransmitter candidacy, involvement in ocular pathology, receptor sites and modulatory activity will be dealt with in relation to valproate action in the therapy of epilepsy

Seizure related elevations of extracellular amino acids in human focal epilepsy.

Carlson H, Ronne-Engstrom E, Ungerstedt U, et al.

Neurosci Lett. 1992 Jun 8; 140(1):30-2.

Intracerebral microdialysis combined with electrocorticographic recordings was used in a patient subjected to epilepsy surgery. The patient developed a series of partial seizures during an 8 min period. Marked elevations of aspartate (79-fold), glycine (21-fold), glutamate (16-fold) and serine (8-fold) dialysate concentrations occurred in association with onset of the period with seizures. Recurrent seizures occurred, in spite of normalizing amino acid levels. Other amino acids analyzed (asparagine, threonine, arginine, alanine, taurine, tyrosine, phenylalanine, isoleucine and leucine) showed less pronounced changes (1-5 times the basal levels)

Effect of sustained pyridoxine treatment on seizure susceptibility and regional brain amino acid levels in genetically epilepsy-prone BALB/c mice.

Dolina S, Peeling J, Sutherland G, et al.

Epilepsia. 1993 Jan; 34(1):33-42.

Epilepsy-prone and epilepsy-resistant substrains were selectively bred from a strain of BALB/c mice; audiogenic-sensitive epilepsy-prone animals showed enhanced sensitivity to chemical convulsants. Treatment with pyridoxine (100 mg/L in drinking water) initiated at mating and continued throughout pregnancy and the life of the offspring abolished the enhanced sensitivity to chemical convulsants and reduced the severity of audiogenic seizures. Withdrawal of pyridoxine restored the enhanced seizure sensitivity. [1H] Nuclear magnetic resonance (NMR) spectroscopy of perchloric acid extracts of tissue was used to determine the concentrations of several compounds [N-acetylaspartate (NAA), GABA, glutamate, aspartate, alanine, taurine, creatine, cholines, inositol] in the hippocampus, neocortex, brainstem, and cerebellum of untreated and pyridoxine-treated 6-week-old female animals. The ratios of the concentrations of excitatory to inhibitory putative neurotransmitter amino acids tended to be higher in epilepsy-prone animals, with the most pronounced difference being a significantly elevated glutamate/GABA ratio in every brain region examined. Pyridoxine treatment abolished this imbalance in the hippocampus, brainstem, and cerebellum, but not in the neocortex. Treatment of epilepsy-resistant animals with pyridoxine using the same protocol decreased the glutamate/GABA concentration ratio in the hippocampus, brainstem, and neocortex and resulted in impaired development of the animals. The amino acid imbalance and the accompanying seizure susceptibility in these genetically epilepsy prone mice may

originate from an inborn error in pyridoxine metabolism or in a pyridoxine-dependent enzyme system

Interictal behavioral alterations and cerebrospinal fluid amino acid changes in a chronic seizure model of temporal lobe epilepsy.

Griffith NC, Cunningham AM, Goldsmith R, et al.

Epilepsia. 1991 Nov; 32(6):767-77.

This study extends our previous work in which we described the presence of an interictal behavioral disturbance in a chronic animal model of temporal lobe epilepsy (TLE). In this study, we investigated the cerebrospinal fluid (CSF) neurotransmitter changes underlying the development of chronic recurrent seizures of temporal lobe origin and interictal behavioral disturbance in cats made epileptic after intrahippocampal injection of kainic acid (KA). Using high-performance liquid chromatography, we measured 22 putative neurotransmitter amino acids. After intrahippocampal KA injection, cats developed an initial acute period of intense seizure activity. Cisternal CSF amino acids, which were repeatedly sampled during the acute period through a permanent indwelling cannula, were unchanged apart from a mild elevation in CSF alanine. The high-level seizure activity gradually decreased, and cats entered a chronic epileptic period characterized by recurrent yet intermittent temporal lobe seizures. CSF GABA levels during the chronic epileptic period were significantly decreased. In contrast, CSF levels of other amino acids--alanine, tyrosine, taurine, aspartic acid, and glutamic acid--did not change significantly. Behavioral testing also showed a heightened interictal defensive reactivity during the chronic epileptic period. To the extent that CSF GABA concentration reflects brain GABA concentration, this study suggests that a decrease in brain GABA may contribute both to the epilepsy and interictal emotional lability of animals with a chronic seizure disorder of temporal lobe origin

Protection of the brain by carnitine.

Igisu H, Matsuoka M, Iryo Y.

Sangyo Eiseigaku Zasshi. 1995 Mar; 37(2):75-82.

Carnitine (beta-hydroxy-gamma-trimethylammonium butyrate) is widely distributed in the body including the nervous system. Its physiological function, viz. a carrier of long-chain fatty acids through the inner mitochondrial membrane, has been well established. In this review, mainly based on our experiments, we discuss the possibility that carnitine may have effects other than the "physiological" function and that it may be a potent protector of the brain. When mice were exposed to ammonia (intraperitoneal injection of ammonium acetate), they developed seizures and concentrations of brain energy metabolites were altered; ATP and phosphocreatine decreased while ADP, AMP, pyruvate and lactate increased. The seizures and changes in brain energy metabolites were clearly suppressed when the mice were pre-treated with carnitine. Furthermore, changes in energy metabolites in the brain caused by severe ischemia (decapitation) were also suppressed by carnitine. Since D-carnitine showed similar effects as those of L-carnitine, the effects seem due to function(s) of carnitine yet to be defined. Intrinsic substances including carnitine appear to deserve further studies for possible use in protecting the brain

Increased plasma glutamic acid in a genetic model of epilepsy.

Janjua NA, Kabuto H, Mori A.

Neurochem Res. 1992 Mar; 17(3):293-6.

A significant increase in the plasma levels of glutamic acid and a significant decrease in aspartic acid and taurine in epileptic patients and their first degree relatives was reported more than a decade ago and an underlying genetic basis for these amino acid changes was suggested. The main objective of the present study was to determine the plasma levels of glutamic acid, aspartic acid and taurine in EI mice which are an inbred epileptic mutant mouse strain. The results show a significant increase in plasma glutamic acid but no changes in aspartic acid or taurine in the epileptic mice as compared to controls. The data provide the first evidence of a significant increase in plasma glutamic acid in an animal model of hereditary epilepsy and substantiate the hypothesis that a genetic defect underlies the elevated plasma glutamic acid levels in association with epilepsy. The findings are also compatible with neurochemical and neurophysiological evidence implicating glutamic acid in the mechanism of seizures

Intracerebral human microdialysis. In vivo study of an acute focal ischemic model of the human brain.

Kanthan R, Shuaib A, Griebel R, et al.

Stroke. 1995 May; 26(5):870-3.

BACKGROUND AND PURPOSE: In vivo microdialysis was introduced in 1982 as a technique to study cerebral neurochemistry in awake, freely moving animals. In small animals, bilateral carotid occlusion produces a 7- to 10-fold increase in extracellular glutamate concentrations. This rapidly falls with reperfusion. Increase in extracellular glutamate is currently believed to be a major factor in initiating neuronal injury. Glutamate antagonists are currently undergoing clinical trials in acute stroke. Human data on the extracellular levels of glutamate and other amino acids in the normal or ischemic brain are limited. In this communication we wish to report the extracellular concentrations of glutamate, serine, glutamine, glycine, taurine, alanine, and gamma-aminobutyric acid, as monitored by in vivo microdialysis, in the simulated ischemic model of the temporal lobe of the human brain. **METHODS:** Intracerebral microdialysis was carried out in five patients who underwent resection of the temporal lobe for intractable epilepsy. Surgical excision leads to an acute (from partial to total, ie, from incomplete to complete) ischemic state of the resected brain. This was our model to study the changes in human extracellular fluid during acute focal ischemic conditions. **RESULTS:** Extracellular glutamate concentrations were 15 to 30 $\mu\text{mol/L}$ in the preischemic samples. This increased to $380.69 \pm 42.14 \mu\text{mol/L}$ with partial (incomplete) ischemia and reached a peak of $1781.67 \pm 292.34 \mu\text{mol/L}$ (> 100-fold) with total isolation of the temporal pole (complete ischemia). The levels fell to $394.52 \pm 72.93 \mu\text{mol/L}$ 20 minutes after resection. Similar trends were observed with the onset of ischemia in the dialysate levels of serine, glutamine, glycine, alanine, taurine, and gamma-aminobutyric acid. **CONCLUSIONS:** Our results show that there is a significant increase in extracellular glutamate and other neurotransmitters with ischemia in the temporal lobe model of the human brain. This increase is of a higher magnitude than that in small animals

Differential changes in induced seizures after hippocampal treatment of rats with an antisense oligodeoxynucleotide to the GABA(A) receptor gamma2 subunit.

Karle J, Laudrup P, Sams-Dodd F, et al.

Eur J Pharmacol. 1997 Dec 11; 340(2-3):153-60.

Gamma-aminobutyric acid (GABA) is the principal inhibitory neurotransmitter in the brain. Impairment of GABAergic neurotransmission may be involved in the pathogenesis of epileptic phenomena. We have previously characterized biochemical and histological changes following unilateral intrahippocampal infusion of a phosphorothioate antisense oligodeoxynucleotide to the GABA(A) receptor gamma2 subunit in rats in vivo. The aim of the present study was to investigate the behavioral changes of rats following unilateral hippocampal antisense 'knockdown' of the GABA(A) receptor gamma2 subunit. Antisense, but not mismatch control oligodeoxynucleotide treated rats had a significant weight loss (10%) during 6 d of treatment. Antisense treated rats exhibited no changes in spontaneous behavior, including anxiety-like behavior as measured in the social interaction test, compared to mismatch oligodeoxynucleotide treated rats. However, antisense treated rats developed pronounced changes in induced seizure activity. Seizures induced by subcutaneously injected pentylenetetrazol were markedly accentuated in antisense treated rats compared to treatment naive rats, whereas mismatch treated rats showed a lower seizure score than that of naive rats. Antisense treated rats had a significantly elevated threshold for seizures induced by electrical stimulation in the maximal electroshock seizure threshold test. The results suggest that intrahippocampal infusion of antisense oligodeoxynucleotide to the GABA(A) receptor gamma2 subunit leads to specific alterations in the sensitivity to induced seizures. The results are viewed as consequences of selective down-regulation of GABA(A) receptors and diminished inhibitory neurotransmission in the hippocampus

Practical issues and concepts in vagus nerve stimulation: a nursing review.

Kennedy PA, Schallert G.

J Neurosci Nurs. 2001 Apr; 33(2):105-12.

Estimates of epilepsy incidence among the U.S. population range between 0.5% and 1%. The most common type of seizure in adult patients is partial onset. Approximately 20% of these patients are refractory to antiepileptic drug therapy and experience intolerable side effects such as confusion, dizziness, weight gain, lethargy, and ataxia. The ketogenic diet appears to be beneficial for children but is not considered a standard option for adults. Epilepsy surgery can be an option for many and may offer control or a reduction in seizures. However, many patients are opposed to cranial surgery or may not tolerate the ketogenic diet. Recent advances in biomedical technology and perfection in surgical techniques have shown vagus nerve stimulation (VNS) using the Neuro Cybernetic Prosthesis (NCP) system is an effective new treatment option in reducing seizure frequency. On July 16, 1997, the U.S. Food and Drug Administration (FDA) approved the use of the NCP for vagus nerve stimulation, as an adjunctive treatment for refractory partial onset seizures in adults and adolescents over 12 years of age. Murphy et al. and Wheless have reported similar results in children younger than 12 years. VNS represents the first therapy using a medical device approved by the FDA for the treatment of refractory seizures. An estimated 10,000 patients have been implanted with the device

Disappearance of neonatal seizures and low CSF GABA levels after treatment with vitamin B6.

Kurlemann G, Loscher W, Dominick HC, et al.

Epilepsy Res. 1987 Mar; 1(2):152-4.

In an infant with neonatal seizures, CSF GABA levels were determined before and after treatment with vitamin B6. Before onset of treatment, the level of GABA in CSF was very low (13 pmol/ml). Injection of vitamin B6 blocked the seizures immediately. When GABA level in CSF was again analysed after continued treatment with vitamin B6, a value of 127 pmol/ml was determined, which is within the normal concentration range in children. The data substantiate previous findings in brain tissue from a patient with vitamin B6-dependent seizures, and strongly indicate that impairment of central GABAergic activity was the cause of the seizures

Effects of soman-induced seizures on different extracellular amino acid levels and on glutamate uptake in rat hippocampus.

Lallement G, Carpentier P, Collet A, et al.

Brain Res. 1991 Nov 1; 563(1-2):234-40.

Extracellular amino acid levels in CA3 and CA1 fields of rat hippocampus, an area highly sensitive to seizures, were determined by intracranial microdialysis during seizures induced by systemic administration of soman (o-1,2,2-trimethylpropyl methylphosphonofluoridate), a potent inhibitor of acetylcholinesterase. The glutamate uptake level was determined on another series of animals in hippocampus homogenates. An early and transient increase in the extracellular glutamate level occurred in CA3 within 30 min of seizures, with correlated brief elevations of taurine, glycine and glutamine levels. The glutamate level increased early in CA1, declined and then became more sustained (after 50 min of seizures). Apparent elevations of taurine, glycine and glutamine levels in CA1 accompanied changes in glutamate concentrations. Changes of glutamate level correlated with an increase in the glutamate uptake which rapidly declined after 40 min of seizures. The role of the transient release of glutamate in CA3 and of the sustained release in CA1 in prolonged soman-induced seizures is considered. The correlation between glutamate and other amino acid release is studied

Epilepsy in women: the science of why it is special.

Morrell MJ.

Neurology. 1999; 53(4 Suppl 1):S42-S48.

Epilepsy is a common neurological disorder that may be affected by reproductive hormones and may complicate reproductive health. Many women with epilepsy experience changes in seizure frequency and severity with changes in reproductive cycles, including at puberty, over the menstrual cycle, with pregnancy and at menopause. Ovarian steroids alter neuronal excitability at the membrane and in the genome. Altered protein synthesis as a consequence of changes in RNA mediated gene transcription is one mechanism for steroid mediated effects on excitability. These genomic effects are delayed and sustained. In contrast, membrane effects are immediate and short duration. These effects are mediated at both the GABA-A and NMDA receptors. Estrogen also dynamically alters synaptic connectivity. Estrogen enhances excitability and lowers the seizure threshold, whereas progesterone enhances inhibition and increases the seizure threshold. In experimental models of epilepsy, estrogen is proconvulsant and progesterone is anticonvulsant. The net effect of these steroid actions is to alter neuronal excitability over physiological cycles. Some epilepsy syndromes are expressed or worsened at puberty. One third to one half of women with epilepsy have catamenial seizure patterns, with seizures most likely to occur in the perimenstrual period and at ovulation. More research is needed to understand the effects of menopause on epilepsy. Antiepileptic drugs may exacerbate the risk of reproductive endocrine disorders in women with epilepsy. Fertility rates are lower for women with epilepsy. Women with epilepsy are more likely to have anovulatory menstrual cycles, abnormal pituitary LH release and altered ovarian steroid concentrations. Polycystic ovaries are detected more often in women with epilepsy, particularly those on valproate. Treatment of hormone sensitive seizures relies on standard AEDs. Small trials suggest that adjunctive progesterone therapy is sometimes helpful. The newer AEDs, gabapentin and lamotrigine may have some advantages for women with epilepsy. These drugs do not alter levels of steroid hormones and do not interfere with effectiveness of hormonal contraception. Experience in pregnancy is limited. The dynamic effects of hormones on seizure expression and of seizures on reproductive health complicate the management of epilepsy in women. Newer AEDs may offer advantages for women with epilepsy in the reproductive years

Experience with the ketogenic diet in infants.

Nordli DR, Jr., Kuroda MM, Carroll J, et al.

Pediatrics. 2001 Jul; 108(1):129-33.

OBJECTIVE: To evaluate the effectiveness, tolerability, and adverse effects of the ketogenic diet in infants with refractory epilepsy. **METHODS:** A retrospective review of 32 infants who had been treated with the ketogenic diet at a large metropolitan institution. **RESULTS:** Most infants (71%) were able to maintain strong ketosis. The overall effectiveness of the diet in infants was similar to that reported in the literature for older children; 19.4% became seizure-free, and an additional 35.5% had >50% reduction in seizure frequency. The diet was particularly effective for patients with infantile spasms/myoclonic seizures. There were concomitant reductions in antiepileptic medications. The majority of parents reported improvements in seizure frequency and in their child's behavior and function, particularly with respect to attention/alertness, activity level, and socialization. The diet generally was well-tolerated, and 96.4% maintained appropriate growth parameters. Adverse events, all reversible and occurring in one patient each, included renal stone, gastritis, ulcerative colitis, alteration of mentation, and hyperlipidemia. **CONCLUSION:** The ketogenic diet should be considered safe and effective treatment for infants with intractable seizures

The osmotic/calcium stress theory of brain damage: are free radicals involved?

Pazdernik TL, Layton M, Nelson SR, et al.

Neurochem Res. 1992 Jan; 17(1):11-21.

This overview presents data showing that glucose use increases and that excitatory amino acids (i.e., glutamate, aspartate), taurine and ascorbate increase in the extracellular fluid during seizures. During the cellular hyperactive state taurine appears to serve as an osmoregulator and ascorbate may serve as either an antioxidant or as a pro-oxidant. Finally, a unifying hypothesis is given for seizure-induced brain damage. This unifying hypothesis states that during seizures there is a release of excitatory amino acids which act on glutamatergic receptors, increasing neuronal activity and thereby increasing glucose use. This hyperactivity of cells causes an influx of calcium (i.e., calcium stress) and water movements (i.e., osmotic stress) into the cells that culminate in brain damage mediated by reactive oxygen species

Topiramate increases brain GABA, homocarnosine, and pyrrolidinone in patients with epilepsy.

Petroff OA, Hyder F, Mattson RH, et al.

Neurology. 1999 Feb; 52(3):473-8.

OBJECTIVE: To measure the effects of topiramate on brain gamma-aminobutyric acid (GABA) in patients with epilepsy. **BACKGROUND:** Topiramate is a new antiepileptic medication with multiple putative mechanisms of action. In a recent meta-analysis of the newer antiepileptic drugs, topiramate was the most potent. Homocarnosine and pyrrolidinone are important metabolites of GABA with antiepileptic actions. **METHODS:** In vivo measurements of GABA, homocarnosine, and pyrrolidinone were made of a 14-cm³ volume in the occipital cortex using ¹H spectroscopy with a 2.1-Tesla magnetic resonance spectrometer and an 8-cm surface coil. Twelve patients (eight women) with refractory complex partial seizures were studied while using topiramate. Nine epilepsy-free, drug-free volunteers served as control subjects. **RESULTS:** Topiramate increased mean brain GABA, homocarnosine, and pyrrolidinone concentrations in all patients. In paired measurements, brain GABA increased by 0.7 micromol/g (SD 0.3, n 7, 95% CI 0.4 to 1.0, p < 0.01). Homocarnosine increased by 0.5 micromol/g (SD 0.2, n 7, 95% CI 0.3 to 0.7, p < 0.001). Pyrrolidinone increased by 0.21 micromol/g (SD 0.06, n 7, 95% CI 0.16 to 0.27, p < 0.01). In two additional patients, GABA, homocarnosine, and pyrrolidinone increased after they were switched from vigabatrin to topiramate. **CONCLUSIONS:** Topiramate increased brain GABA, homocarnosine, and pyrrolidinone to levels that could contribute to its potent antiepileptic action in patients with complex partial seizures

Monitoring pregnancy outcomes after prenatal drug exposure through prospective pregnancy registries: a pharmaceutical company commitment.

Reiff-Eldridge R, Heffner CR, Ephross SA, et al.

Am J Obstet Gynecol. 2000 Jan; 182(1 Pt 1):159-63.

OBJECTIVE: Glaxo Wellcome becomes aware of prenatal exposures to its medications as early as the clinical trial phase of development. An international process for monitoring prenatal exposure to all Glaxo Wellcome medicines has been developed. For specific products there are prospective pregnancy registries. **STUDY DESIGN:** The registries are observational, case-registration, and follow-up studies designed to detect evidence of teratogenicity associated with specific medications. After prenatal exposure to the registry medication, pregnancies are registered prospectively, through voluntary reports by health care providers. An advisory committee of independent scientists for each registry reviews data and advises in dissemination of information. Risk of birth defects, as defined by the Centers for Disease Control and Prevention, is compared with published risks both in women in the general population and in women with the underlying condition being treated, if available. **RESULTS:** The following data show results from the prospective first-trimester exposures registered since establishment of each registry. The published risk of birth defects in the general population range is 3% to 5%, and the risk in women with epilepsy is 6% to

9%. The proportions of outcomes with birth defects are as follows: in the Acyclovir (antiviral medication) Pregnancy Registry (1984-1998) (19/581), 3.3% (95% confidence interval, 2.0%-5.2%); in the Lamotrigine (monotherapy and polytherapy antiepileptic medication) Pregnancy Registry (1992-September 1998) (8/123), 6.5% (95% confidence interval, 3.1%-12.8%); in the Sumatriptan (migraine medication) Pregnancy Registry (1996-October 1998) (7/183), 3.8% (95% confidence interval, 1.7%-8.0%). The Valacyclovir, Bupropion, and Naratriptan registries have insufficient data for analysis. CONCLUSION: None of the registries has provided a risk estimate exceeding that expected in the disorder treated, and no pattern of defects has been observed. Whereas information from the larger registries is reassuring regarding risk, these studies cannot rule out possible small excess risks from use of these drugs in pregnancy. Data obtained through these registries are shared with the medical community as a supplement to animal toxicology studies to assist in weighing potential risks and benefits of treatment for individual patients. The success of the registries depends on the continued willingness of the obstetrics and gynecology community to notify the registries of prenatal exposures

Epilepsy: A New Approach.

Richard A.

1995;

Intracerebral microdialysis of extracellular amino acids in the human epileptic focus.

Ronne-Engstrom E, Hillered L, Flink R, et al.

J Cereb Blood Flow Metab. 1992 Sep; 12(5):873-6.

Extracellular levels of aspartate (ASP), glutamate (GLU), serine (SER), asparagine (ASN), glycine (GLY), threonine (THR), arginine (ARG), alanine (ALA), taurine (TAU), tyrosine (TYR), phenylalanine (PHE), isoleucine (ILEU), and leucine (LEU) were monitored by using intracerebral microdialysis in seven patients with medically intractable epilepsy, undergoing epilepsy surgery. In association with focal seizures, dramatic increases of the extracellular ASP, GLU, GLY, and SER concentrations were observed. The other amino acids analyzed, including TAU, showed small changes. The results support the hypothesis that ASP, GLU, GLY, and possibly SER, play an important role in the mechanism of seizure activity and seizure-related brain damage in the human epileptic focus

Excitatory and inhibitory amino acid levels in the cerebrospinal fluids of children with neurological disorders.

Shen EY, Lai YJ, Ho CS, et al.

Acta Paediatr Taiwan. 1999 Mar; 40(2):65-9.

Measurement of amino acid levels in the cerebrospinal fluid (CSF) of children with various neurological disorders was performed with high performance liquid chromatography (HPLC). Glutamate increased in patients with bacterial meningitis, aseptic meningitis and encephalitis. Aspartate increased in bacterial meningitis and seizure disorders. Glycine increased in both bacterial and aseptic meningitis. Taurine increased in bacterial meningitis and encephalitis. GABA, the main inhibitory amino acid, increased in encephalitis. Excitatory and inhibitory amino acids are richly distributed in brain tissue and are related to neuron activity. Changes in amino acid levels in the CSF may reflect the pathologic state and severity of brain insults, and may be useful in monitoring disease processes. Further study is necessary to determine whether CSF amino acid levels have a role in practical clinical application

Vagus nerve stimulation and the ketogenic diet.

Wheless JW, Baumgartner J, Ghanbari C.

Neurol Clin. 2001 May; 19(2):371-407.

Antiepileptic drugs are the primary form of treatment for patients with epilepsy. In the United States, hundreds of thousands of people do not achieve seizure control, or have significant side effects, or both. Only a minority of patients with intractable epilepsy are candidates for traditional epilepsy surgery. Vagus nerve stimulation is now the second most common treatment for epilepsy in the United States. Additionally, the ketogenic diet has established itself as a valid treatment. This article discusses the history, mechanism of action, patient selection, efficacy, initiation, complications, and advantages of vagus nerve stimulation and the ketogenic diet

These statements have not been evaluated by the FDA. These products are not intended to diagnose, treat, cure or prevent any disease. The information provided on this site is for informational purposes only and is not intended as a substitute for advice from your physician or other health care professional or any information contained on or in any product label or packaging. You should not use the information on this site for diagnosis or treatment of any health problem or for prescription of any medication or other treatment. You should consult with a healthcare professional before starting any diet, exercise or supplementation program, before taking any medication, or if you have or suspect you might have a health problem. You should not stop taking any medication without first consulting your physician.