

Myasthenia Gravis

ABSTRACTS

- Biron P., 1988. Myasthenia gravis after general anesthesia and hepatitis B vaccine.
- Denny-Brown D., 1947. Neurological conditions resulting from prolonged and severe dietary restriction.
- Josephson EM., 2000. Myasthenia gravis, manganese and the thymus.
- Josephson EM., 1981. Thymus, Manganese, and Myasthenia Gravis.
- Klenner FR., 1973. Response of peripheral and central nerve pathology to mega-doses of the vitamin B-complex and other metabolites.
- McGraw CP., 1975. Effects of thiamine, ascorbic acid and alpha tocopherol on neuronal and muscular function.
- McGraw CP., 1980. Study of megavitamin therapy on experimental myasthenia gravis in Guinea pigs by electromyographic monitoring.
- Peeler RH., 1979. The effect of thiamine, ascorbic acid and alphotocopherol on experimental autoimmune myasthenia gravis (EMG) in rats and rabbits.
- Stout JR., 2001. Effects of resistance exercise and creatine supplementation on myasthenia gravis: a case study.
- Waldbott GL., 1998. The preskeletal phase of chronic fluoride intoxication.

Suggested Reading

- Da-Yuan Z., 1996. Recent studies on traditional Chinese medicinal plants.
- Fernstrom JD., 1981. Dietary precursors and brain neurotransmitter formation.
- Goulon M., 1979. [Myasthenia and pernicious anemia or Biermer's (author's transl)]
- Graham JG., 1982. Neurological complications of pregnancy and anaesthesia.
- Huber A., 1978. [Diagnosis and treatment of eye muscle palsies (author's transl)]
- Martin-Du Pan RC., 1997. [The role of nutrition in the synthesis of neurotransmitters and in cerebral functions: clinical implications]
- Zittoun J., 1979. Humoral and cellular immunity to intrinsic factor in myasthenia gravis.
- Myasthenia gravis after general anesthesia and hepatitis B vaccine.**

Biron P, Montpetit P, Infante-Rivard C, Lery L. Department of Pharmacology, Faculty of Medicine, University of Montreal, Quebec, Canada.

Arch Intern Med. 1988 Dec;148(12):2685.

A 48-year-old man presented with the first symptoms of myasthenia gravis one month after a general anesthesia and a second dose of hepatitis B plasma vaccine. Whether either event may have acted as a nonspecific challenge to the patient's immune system is speculative, but the case is described to discover similar observations, if any.

Neurological conditions resulting from prolonged and severe dietary restriction.

Denny-Brown, D.

Medicine 1947; 26: 41.

No abstract available.

Myasthenia gravis, manganese and the thymus.

Josephson, E.M.

Presented to Section N, American Association for Advancement of Science, December 30, 1946. Boston, MA: Harvard School of Public Health.

Thymus, Manganese, and Myasthenia Gravis 1961.

Josephson, E.M.

Ferndale, MI: A-albionic Research (www.msen.com or www.addall.com).

Response of peripheral and central nerve pathology to mega-doses of the vitamin B-complex and other metabolites.

Klenner, F.R

J. Appl. Nutr. 1973; 25: 16.

No abstract available.

Effects of thiamine, ascorbic acid and alpha tocopherol on neuronal and muscular function

McGraw C.P.; Metcalf D.L. Bowman Gray Sch. Med., Wake Forest Coll., Winston Salem, N.C. United States

Journal of Applied Nutrition (J. APPL. NUTR.) 1975, 27/1 (51-63)

Stimulated by a patient's report that all symptoms of myasthenia gravis disappeared when she took vitamins in large doses, the authors review the literature reporting the relationship between vitamins and neuromuscular function. Of particular interest was Denny Brown's report on outbreaks of myasthenia gravis like symptoms in prisoners of war maintained on low nutritional diets. They found that three vitamins, thiamine (vitamin B1), ascorbic acid (vitamin C) and alpha tocopherol (vitamin E), have been reported to play specific roles in neuronal and muscular function. Those roles and the possible use of those vitamins in the treatment of neuromuscular disorders are the subject of this report. The papers reviewed are those that they believe to be the most relevant to this report.

Study of megavitamin therapy on experimental myasthenia gravis in Guinea pigs by electromyographic monitoring

McGraw C.P.; Paschold E.H.; Currin J.M. Bowman Gray Sch. Med., Wake Forest Univ., Winston-Salem, N.C. United States

Journal of Applied Nutrition (J. APPL. NUTR.) (United States) 1980, 32/1 (37-43)

The underlying defect in myasthenia gravis is not known, but it is manifested as a premature fatigue of the neuromuscular junction upon repeated stimulation. It is generally accepted that the activity at that junction can be compromised by an autoimmune reaction, and it is postulated that the autoantigen of muscle tissue may be released by some pathological occurrence such as loss of cell membrane or chronic autolysis. The resulting autoantibodies may then attack the neuromuscular junction and cause further damage, producing a myasthenia-like syndrome. If this is true, then the goals of therapy should be: restoration of the integrity of the muscle cell membranes with subsequent cessation of the release of autoantigen; facilitation of the neurotransmitter-producing activity of the neuron; and enhancement of the effectiveness of the neurotransmitter after its release from the neuron. Classical therapy achieves only the third goal. Theoretically, on the basis of their physiological activity, thiamine, ascorbic acid and alpha-tocopherol given in megadoses should accomplish the first and second goals as well. In this study, an immunologically induced myasthenia gravis model was used in an attempt to prove that point. The immunological method of Goldstein and Kalden was used in 40 guinea pigs to produce a partial neuromuscular block. Ten healthy nonimmunized guinea pigs served as controls. Half of the animals received megavitamin therapy in whole milk; half received whole milk only. Megavitamin therapy consisted of massive doses of thiamine, ascorbic acid, and alpha-tocopherol. The presence of a neural deficit and the effects of therapy were determined electromyographically. Although the model was produced in 73% of animals, we could show no difference in response of the animals, regardless of therapy. Despite the fact that we were unable to show a favorable effect of megavitamin therapy in this myasthenia gravis model, we believe that the theories that support such an effect are valid and that further study in this area should be pursued with a more satisfactory model.

The effect of thiamine, ascorbic acid and alphatocopherol on experimental autoimmune myasthenia gravis (EMG) in rats and rabbits.

Peeler, R.H., McGraw, C.P., Wagoner, R.O.

J. Appl. Nutr. 1979; 31: 34-44.

No abstract available.

Effects of resistance exercise and creatine supplementation on myasthenia gravis: a case study.

Stout JR, Eckerson JM, May E, Coulter C, Bradley-Popovich GE. Exercise Science Department, Creighton University, Omaha, NE 68178, USA. jstout@nutriciausa.com

Med Sci Sports Exerc 2001 Jun;33(6):869-72

PURPOSE: The purpose of this case study was to determine the effects of 15 wk of resistance exercise and creatine (Cr) supplementation on body composition, training volume, peak strength, and complete blood chemistry in a patient with myasthenia gravis (MG).

METHODS: The patient was a 26-yr-old man who was taking prednisone and azathioprine for his condition. The patient self-administered 5 g of Cr per day in addition to resistance exercise 3 times per week. Fasting blood samples were obtained and body weight (BW) and fat free mass (FFM; via hydrostatic weighing) were measured before and after training and Cr supplementation. In addition, isokinetic (Cybex II) peak strength for leg extension (LE), leg flexion (LF), and volume load (repetition x mass lifted) for the first and last resistance training session were determined.

RESULTS: After Cr supplementation and training, the results demonstrated increases in BW (6.8%), FFM (4.3%), upper body volume load (37.0%), lower body volume load (15.0%), and peak strength for LE (37.0%) and LF (12.5%). Moreover, blood chemistry values remained within normal limits for the duration of the 15-wk study.

CONCLUSION: These data suggest that resistance exercise plus Cr supplementation may promote gains in strength and FFM in patients with MG.

The preskeletal phase of chronic fluoride intoxication.

Waldbott, G.L.

Fluoride 1998; 31(1): 13-20.

No abstract available.

Suggested Reading

Recent studies on traditional Chinese medicinal plants.

Da-Yuan, Z., Dong-Lu, B., Xi-Can, T.

Drug Dev. Res. 1996; 39(2): 147-57.

No abstract available.

Dietary precursors and brain neurotransmitter formation.

Fernstrom JD.

Annu Rev Med. 1981;32:413-25.

The rates of synthesis of serotonin, acetylcholine, and, under certain circumstances, dopamine and norepinephrine by brain neurons depend considerably on the availability to brain of the respective dietary precursors. This precursor dependence seems to be related to the fact that the enzyme catalyzing the rate-limiting step in the synthetic pathway for each transmitter is unsaturated with substrate at normal brain concentrations. Moreover, brain levels of the individual precursors rise following oral or parenteral administration of the pure compound or the ingestion of certain foods. Precursor-induced increases in brain transmitter formation seem to influence a variety of brain functions and behaviors, which suggests that transmitter release has been enhanced. It now appears that these precursors may become useful as therapeutic agents for the treatment of selected disease states, wherein the disease is related to reduced release of transmitter. Examples of Parkinson's disease (tyrosine), myasthenia gravis (choline or phosphatidylcholine), depression (tyrosine), and possibly abnormal appetite (tryptophan). Perhaps the future will bring the identification of still other neurotransmitters, whose rates of synthesis depend on precursor availability. Two potential candidates for which some information is already available are glycine (a spinal cord transmitter) and the prostaglandins (some of which may function as neuromodulators or transmitters) (48, 49). Each time a new precursor-product relationship is described, an opportunity

becomes available for determining whether the precursor might be useful in treating disease states related to reduced transmitter release by neurons. The opportunities are worth exploring, since the use of a natural dietary constituent, even in purified form, is likely to produce fewer unwanted side-effects than are seen following administration of synthetic drugs.

[Myasthenia and pernicious anemia or Biermer's (author's transl)] [Article in French]

Goulon M, Zittoun J, Tulliez M, Estournet B.

Rev Neurol (Paris). 1979 Oct;135(8-9):605-14.

The association of myasthenia and Biermer's anemia is very rarely reported. In a series of 138 cases of myasthenia, this association was found in only one patient, in whom the anemia developed 19 years after the discovery of a calcified thymoma and 13 years after the appearance of the first signs of myasthenia. This led the authors to conduct a prospective study for the presence of intrinsic antifactor antibodies. A total of 81 patients (20 men and 61 women) with myasthenia were studied. The myasthenia had appeared after 35 years of age in 40 patients and 19 had a thymoma. The results of the study for the antibodies was positive in 3 women, as was the test of inhibition of leucocyte migration, but none of them had anemia, vitamin B12 malabsorption, achlorhydria, or gastric atrophy. The discovery of these immunological disorders raises the problem of their significance ; two hypotheses can be discussed : pre-Biermer state or immunological disturbance without pathogenetic significance. The problem can probably only be resolved by studying these antibody levels in a very much larger number of patients with myasthenia.

Neurological complications of pregnancy and anaesthesia.

Graham, J.G.

Clin. Obstet. Gynaecol. 1982; 9(2): 333-50.

No abstract available.

[Diagnosis and treatment of eye muscle palsies (author's transl)] [Article in German]

Huber A.

Klin Monatsbl Augenheilkd. 1978 Feb;172(2):138-40.

After the discussion of the clinical symptomatology of paralytic squint (diplopia, variable angle of squint, compensatory head posture etc.) the importance of modern electromyography for differentiation between myopathy (affection of the eye muscles), myastherias (affection of the neuromuscular transmission) and peripheral neurogenic palsies (affection of the peripheral neuron up to the nuclear region) is discussed. The clinical symptomatology of these affections of different levels is discussed as well as the differential diagnosis. The treatment of eye muscle palsies in principle consists in: 1. medical treatment (local and general), 2. optical treatment (glasses, occlusions, prisms etc.), 3. orthoptic treatment, 4. surgical treatment.

[The role of nutrition in the synthesis of neurotransmitters and in cerebral functions: clinical implications] [Article in French]

Martin-Du Pan RC, Wurtman RJ.

Schweiz Med Wochenschr. 1981 Sep 26;111(39):1422-34.

Much experimental evidence shows that intracerebral synthesis of various neurotransmitters (serotonin, acetylcholine, and catecholamines) may be affected by the amount of their respective precursors (tryptophan, choline and tyrosine) in food. Changes in cerebral functions secondary to the administration of each of these three precursors have been reviewed in physiological and pathological conditions, and in particular in different neuro-psychiatric diseases due to a lack of synthesis or release of neurotransmitters. Possible interactions between digestive and cerebral diseases are considered in relation to lack or excess of precursors.

Humoral and cellular immunity to intrinsic factor in myasthenia gravis.

Zittoun J, Tulliez M, Estournet B, Goulon M.

Scand J Haematol. 1979 Nov;23(5):442-8.

Myasthenia gravis (MG) is an autoimmune disease often associated with other autoimmune disorders. A case history of MG with a coexisting atypical megaloblastic anaemia with vitamin B12 deficiency and anti Intrinsic Factor (IF) antibodies, led to a study of humoral and cellular immunity to IF in 81 MG patients. Within this series, 3 other patients had a disturbed humoral and cellular immunity to IF. These 3 patients presented no other features of pernicious anaemia. The possible origins and significance of the anti IF antibodies in MG patients are discussed.

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