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Publisher: Hans R. Larsen MSc ChE

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VIRTUAL LAF CONFERENCE

Proceedings of 53rd Session
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SUBJECT: Magnesium Supplementation: Friend or Foe?

Recent posts on the Bulletin Board have indicated that magnesium supplementation may worsen ectopics and increase episode frequency in some afibbers. It would clearly be important to establish the reason(s) for this.

PC brought to my attention a 1987 article by RA Farkas of the Dallas Veterans Administration Medical Center. Dr. Farkas found that laboratory rats fed a diet rich in magnesium sulfate (Epsom salt) actually increased their potassium excretion. He also noted that rats fed a magnesium-deficient diet experienced both magnesium and potassium depletion (1). I know people don't usually behave like rats :-), but it is possible that a similar mechanism could operate in afibbers. In other words, relying on getting Mg from supplementing with a poorly absorbed magnesium compound (like magnesium oxide) could lead to magnesium deficiency followed by potassium depletion resulting in increased ectopics.

It is also possible that overdosing with magnesium could be a problem in that it would result in diarrhea accompanied by electrolyte (potassium) depletion. I am sure there are several other possible explanations as to why magnesium supplementation could be detrimental in some cases. Your input and discussion participation would be much appreciated.

On a slightly different topic, but still related to magnesium and potassium. Do afibbers who have undergone a successful ablation still need to supplement with potassium and magnesium in order to help keep the PACs and PVCs at bay? A recent study by Japanese researchers concluded that ANP and/or BNP levels are elevated in afibbers, but decrease significantly after a PVI (2). Since both ANP (atrial natriuretic peptide) and BNP (brain natriuretic peptide) are diuretics and thus would reduce blood levels of both magnesium and potassium when elevated is it possible that ablated afibbers no longer need to supplement with K and Mg? Again, your input and discussion as well as your personal experience with Mg and K supplementation after ablation would be appreciated.

Hans

(1) Farkas RA et al., Effect of magnesium salt anions on potassium in normal and magnesium-depleted rats. J Lab Clin Med. 1987 Oct; 110 (4) : 412-7

(2) Yamada, T, et al. Plasma atrial natriuretic peptide and brain natriuretic peptide levels after radiofrequency catheter ablation of atrial fibrillation. American Journal of Cardiology, Vol. 97, June 15, 2006, pp. 1741-44.

I take a very small dose of magnesium 2 or 3 times a day and it definitely helps me avoid any heart blips.

I use the true food magnesium from www.highernature.co.uk 50mg which I split into 3 pieces.

On the bottle it says:

"As a guide one 50mg tablet is comparable in effect to 200mg of standard isolated magnesium supplements"

Joyce

I take 500mg Magnesium twice day, having slowly increased to this amount over about 6 months with no problems. I don't intend going any higher with it. As I posted to Bill on the main BB, the only supplement that I thought was giving me a problem with ectopics was L-carnitine so I stopped it and the problem stopped too.

Sue

"It is also possible that overdosing with magnesium could be a problem in that it would result in diarrhea accompanied by electrolyte (potassium) depletion. I am sure there are several other possible explanations as to why magnesium supplementation could be detrimental in some cases. Your input and discussion participation would be much appreciated."

I did experience electrolyte depletion from taking too much and the wrong kind of magnesium. Several years ago, I started taking a certain magnesium as part of my afib protocol (can't remember what kind, but it wasn't gluconate) and after about six months began waking up with loose bowels every morning. If I remember right, I was taking one 500 mg tablet with every meal, along with my other supplements, including potassium. I didn't worry about it because I was grateful for the clean-out everyday and it wasn't hitting me at odd times, just first thing in the morning. Well, after having experienced that for nearly a year, my afib started getting worse (that may not have been the entire reason for it increasing, but now I'm sure it contributed). I always felt the loose bowels were not natural to have everyday but thought it contributed to controlling my afib and felt that was the trade off. But since my afib started picking up again, my husband and I finally concluded on our own (after learning how important electrolyte balance is from your books) that having the watery, loose bowels everyday had messed with my electrolytes. I stopped all magnesium for a while and then switched to magnesium gluconate. I have found that I utilize the Doctor's Best High Absorption Magnesium (I just started taking that brand of gluconate about three month's ago) so well that I only need to take one 500 mg tablet a day, rather than the two I was taking, because I did start experiencing a bit of diarrhea recently. Thanks to the Bulletin Board, I learned that magnesium does build up in the body. I believe I have found my saturation point with the magnesium and have it under control. As I posted on the Bulletin Board recently, I feel my supplementation and the dosages I take now are keeping me in NSR.

Claire Q

After nearly two years on 400mg of Mg per day (as citrate) I tried experimenting with the dose starting in January 2006. Before this my episode frequency has remained pretty constant at approx. 1 episode every 4 weeks. Sometimes I went for nearly 3 months with no episodes, but generally once per 4 weeks was the norm.

Of course, one needs always be wary of post hoc propter hoc inferences, but I noticed a definite increase in my episode frequency with the increased magnesium. Once I got the dose up to 1000mg (with Doctor's Choice mg) I started to have episodes every two weeks! This happened three times and had never happened before. I also noticed an increase in atrial tachycardia in the days before an episode- something I have had before but never this often and I also hadn't noticed it in over a year.

So I at the beginning of May I stopped all magnesium- sadly I didn't have exatest labs done for all this- so I could let the mg levels drop. In May I had an episode after 3 weeks and had none of the atrial tach (which I particularly hate, even more than afib). After that episode I resumed supplementing with 400mg QD of citrate and right now I am at about 23 days and counting.

I am convinced that it was the increased magnesium. I feel like I am back to my baseline. It just feels the same.

I have two theories as to what might have happened: 1. I noticed increased gut motility but not diarrhea on the

increased mg- maybe this increased my vagal tone at night (I am vagal) 2. Maybe the increased mg caused atrial tachycardia which degenerated into afib. Overall, I did not notice increased ectopics.

One strange and interesting thing is that the afib episodes were shorter than usual so that my overall afib burden was actually lower on the increased mg. While this may be a benefit- and that is not certain- I have to say I prefer longer intervals between episodes.

I kept my potassium at approx. 5gms per day from all sources during this experiment.

Thanks Hans for all your work!

Thomas

This is good timing, Hans, I was just gathering information to post on the importance of magnesium as a review for new readers and also to review the latest findings on the implications of magnesium deficiency and inflammation. I'll respond separately with my personal experiences a bit later.

Jackie

With my awareness focus on natural therapies and nutritional interventions for healthy aging, my response is that no one should neglect magnesium intake or forget the value or importance of it. I claim it as "friend" – and a very important one, at that!

As you might expect, my position is that we need to be sure that magnesium stores are continually optimal since the majority of people (including afibbers) probably remain magnesium deficient or are at least borderline, whether or not they've had an ablation.

For those who are intolerant of supplements, this is still a significant concern because of the well- recognized facts on the role of magnesium in the body. The heart and arrhythmia are not the only concerns, although they certainly are a major category. The fact remains magnesium deficiency still is rampant in the population and because magnesium is so very important to overall health, we must look to optimal stores as measured by red blood cell magnesium analysis or the Exatest in order to be defensive toward all adverse health conditions brought on by deficiency.

We need to be aware of magnesium deficiency and the relationships to other disease conditions including vascular tone, inflammation, thrombosis risk, oxidative stress, immune system stress, depression, osteoporosis, hypertension and especially regulating insulin-mediated glucose uptake so critical in metabolic syndrome and diabetes. It would be difficult to rank benefits of optimal magnesium by importance, but the recently published findings linking magnesium deficiency (MgD) and inflammation surely ranks very high on any list.

Everyone concerned with healthy aging, especially afibbers, need to be aware of the importance of keeping inflammation low or hopefully, absent. To remain healthy, we must control inflammation for numerous reasons not the least being the role of inflammation in both atrial fibrillation but also cardiovascular disease and risk of myocardial infarction, thrombosis, and cardiomyopathy. In fact, in other pathologies involving MgD and inflammation, consideration should be given to the influence of MgD and pain, post-trauma and neurodegenerative diseases.

As posted in Conference Room Session 40 (the second section), we know the connection between elevated blood viscosity and inflammation, strokes and heart attacks. Silent inflammation is of serious concern in that it is a factor in so many disease conditions.

There is also research supporting that inflammation as measured by C-reactive protein (CRP) levels is found elevated in afibbers – making the connection between inflammation and atrial fibrillation. [Chung MK, et al C-reactive protein elevation in patients with atrial arrhythmias: inflammatory mechanisms and persistence of atrial fibrillation. *Circulation*. 2001 Dec 11;104(24):2886-91].

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=11739301&query_hl=2

Numerous publications indicate the role of silent inflammation in heart disease and stroke including that by Barry Sears, PhD – The Anti-Inflammation Zone, and cardiologist Steven Sinatra, MD, has written extensively on silent inflammation (see my posts from the Anti-Aging Congress). Life Extension Foundation has an excellent online story about silent inflammation I also posted previously Dr. Sinatra's excellent article, "Fire in the Heart."

A recent study (April 2006) indicates that a magnesium deficiency plays a role in inflammation.

Summary:

This study on magnesium deficiency in rats found a clinical inflammatory syndrome characterized by leukocyte and macrophage activation, release of inflammatory cytokines, and excessive production of free radicals. Increase in extracellular magnesium concentration decreases inflammatory response, while reduction in the extracellular magnesium results in cell activation. Because magnesium acts as a natural calcium antagonist, the molecular basis for inflammatory response is probably the result of modulation of intracellular calcium concentration. Magnesium deficiency contributes to an exaggerated response to immune stress and oxidative stress as the consequence of the inflammatory cascade.

Further studies are needed to assess the role of magnesium in, but these experimental findings in animal models suggest that inflammation is the missing link to explain the role of magnesium in many pathological conditions.

While rats are not humans, it was noted in the study that magnesium deficiency can be easily produced in rodents by dietary depletion. Very rapid decline in plasma magnesium concentration was observed. Observations of clinical symptoms of inflammation from magnesium deficiency have been published since the 1930s.

The authors state that the underlying mechanisms for inflammatory response in magnesium deficiency are not clearly defined but list the following triggering events for consideration:

- cellular entry of calcium and priming of phagocytic cells
- opening of calcium channels and activation of NMDA receptors
- release of neurotransmitters such as substance P
- membrane oxidation and activation of nuclear factor kappa B (NF- κ B).

Most notable is the finding that the inflammatory response to the magnesium deficiency is most likely mainly related to a modification in the extracellular magnesium concentration because there is a decline in plasma magnesium, while intracellular magnesium concentration does not fall at all in phagocytic cells following 2 days of magnesium deficiency.

Source:

"Magnesium and the inflammatory response: Potential physiopathological implications"

Andrzej Mazur a, Jeanette A.M. Maier b, Edmond Rock a, Elyett Gueux a, Wojciech Nowacki c, Yves Rayssiguier
Archives of Biochemistry and Biophysics - April 2006

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=16712775

While we know many of the benefits of maintaining optimal intracellular magnesium, following are some quoted/paraphrased points of interest that serve as reminders that we need not neglect magnesium intake.

Source:

William Makel, DC teleconference (1)

Patients who have high levels of magnesium in brain or heart tissue have very little ischemic tissue damage if they do suffer from stroke or MI because the magnesium acts as an antioxidant.

Magnesium acts like a magnet for calcium and pulls it into bone matrix. Deficient magnesium; less calcium in bones

People with chronic pain and who are non-responsive to therapeutic interventions probably are magnesium deficient.

All carbonated drinks deplete magnesium. Sugar is another huge depletor as is stress and stress can be from the

condition or conditions the patient endures.

People who are unable to tune out background noise or interference in a noisy restaurant are found to be magnesium deficient. Goes along with the magnified startle reflex.

Magnesium and potassium are both needed (cofactors) to convert cysteine into gamma-glutamylcysteine and then needed again to convert gamma-glutamylcysteine into glutathione. Glutathione, an antioxidant, protects cells from toxins such as free radicals. Without the protection from oxidative injury afforded by glutathione, cells may be damaged or killed. Free radicals help create inflammation.

In Finland, they had the dubious distinction of being #1 in heart diseased. They added magnesium to their salt and went from 1st to #7 on the list.

Normal Shealy, MD, neurosurgeon and inventor of the TENS unit, said that “a magnesium deficiency, coupled with a bad attitude – namely fear – was the leading cause of death.”

Russell Blaylock, MD, neurosurgeon (2)

Extreme athletes like marathon runners need magnesium as it is highly effective in preventing stroke or heart attack. Based on studies, we learn that marathon runners require at least six months to replace magnesium lost in a single marathon race.

Magnesium is vital as it prevents inflammation, improves blood flow through blood vessels, thins the blood, acts as an antioxidant, and prevents coronary artery spasm. In case of a heart attack, magnesium has been shown to significantly reduce the severity of damage to the heart muscle.

Many medications severely deplete certain nutrients including many of the oral diabetic and blood pressure medications, birth control pills, steroids, statins (for lowering cholesterol) and heart drugs exhaust magnesium, CoQ10, thiamine, riboflavin and other nutrients. This is in addition to the nutrient depletion caused by the stress of your disease or surgery.

While magnesium deficiency is common in the general population, diabetics are especially at risk as low magnesium accelerates dramatically atherosclerosis, increase the risk of heart arrhythmias, stroke and heart attacks and make the brain vulnerable to a number of diseases which includes substantial proliferation of free radical generation, lipid peroxidation and inflammation. Magnesium supplementation has been proven to dramatically reduce the risk of all disorders associated with diabetes and the metabolic syndrome.

Most doctors make the mistake of thinking that normal magnesium readings on a blood test mean you have adequate stores of the substance in your body. But magnesium is an intracellular ion, meaning that 90% of it is stored in cells and tissues — not in the blood. You can have severely low magnesium levels in your tissue and simultaneously maintain normal blood levels.

Precaution with magnesium supplementation:

If you have the heart problem known as a conduction defect, you need to consult with your cardiologist before taking supplemental magnesium. While there is no evidence that oral magnesium in these doses will harm you, it is best to let your doctor know you're taking it. Those with kidney disease should also consult their doctor, since significant impairment of kidney function can spur the development of very high serum levels of magnesium. Interestingly, magnesium protects the kidney by reducing free radical damage and improving blood flow through the kidney.

Pain Control Since pain control in hospitalized patients is a major problem and dependence on pain medication can lead to significant immune suppression (increasing pneumonia risk and other infections), increasing magnesium by IV also acts as a pain medication in addition to all its other benefits. Oral supplementation, though can take months to be effective.

Adverse affects of medications:

Many anti-hypertensives are known to reduce vital nutrients: B1, K, B6 and ascorbate along with well as magnesium,

zinc, calcium and CoQ10. In particular, the body's supply of CoQ10, a critical anticancer nutrient, is siphoned off through use of hypertensive and antidiabetic medications, statins (cholesterol-lowering drugs) and certain antidepressants (phenothiazines and tricyclics). Statins pilfer vitamins A, B12, D, E and K, beta-carotene and folate, as well as calcium, zinc, phosphorus and magnesium.

Chemotherapy drugs and magnesium depletion is seen with cisplatin. Magnesium levels can fall so low that the result is heart damage and brain injury. Birth control pills are also among prescription drugs that sap magnesium, and women taking these over many years are not only more prone to developing cancer, but they are at greater risk during subsequent surgery and chemotherapy.

Recent studies have shown that Alzheimer's dementia and other neurodegenerative diseases are associated with chronic inflammation and that most people with these diseases have elevated C-reactive protein levels.

Drugs and nutrients that reduce inflammation have been shown to lower the risk of getting Alzheimer's disease six to twelve times.

Aged garlic is an excellent way to lower CRP, cholesterol and eliminate chronic inflammation along with help from quercetin, curcumin, gamma tocopherol vitamin E, magnesium, and vitamin D.

As a preventive measure for post-operative blood clots or heart failure during surgery, magnesium added to the patients' IV fluid in Dr. Blaylock's practice.

Exatest (3)

Conclusions: Sublingual epithelial cell (Mg)_i correlates well with atrial (Mg)_i but not with serum magnesium. (Mg)_i levels are low in patients undergoing cardiac surgery and those with AMI. Intravenous magnesium sulfate correlates low (Mg)_i levels in AMI patients. Energy-dispersive x-ray analysis determination of sublingual cell (Mg)_i may expedite the investigation of the role of magnesium deficiency in heart disease.

Noninvasive measurement of Tissue Magnesium and Correlation with Cardiac levels Circulation: 1995;92:2190-2197)
American Heart Association
Marc CP Haigney, MD, Burton Silver, PhD, et al.

More studies of interest:

Heart Fail Rev. 2006 Mar;11(1):35-44.

The nerve-heart connection in the pro-oxidant response to Mg-deficiency.

Tejero-Taldo MI, Kramer JH, Mak IuT, Komarov AM, Weglicki WB.
Div. of Experimental Medicine, Dept. of Biochemistry & Molecular Biology, Washington.

Magnesium is a micronutrient essential for the normal functioning of the cardiovascular system, and Mg deficiency (MgD) is frequently associated in the clinical setting with chronic pathologies such as CHF, diabetes, hypertension, and other pathologies.

Animal models of MgD have demonstrated a systemic pro-inflammatory/pro-oxidant state, involving multiple tissues/organs including neuronal, hematopoietic, cardiovascular, and gastrointestinal systems; during later stages of MgD, a cardiomyopathy develops which may result from a cascade of inflammatory events.

In rodent models of dietary MgD, a significant rise in circulating levels of proinflammatory neuropeptides such as substance P (SP) and calcitonin gene-related peptide among others, was observed within days (1-7) of initiating the Mg-restricted diet, and implicated a neurogenic trigger for the subsequent inflammatory events; this early "neurogenic inflammation" phase may be mediated in part, by the Mg-gated N-methyl-D-aspartate (NMDA) receptor/channel complex. Deregulation of the NMDA receptor may trigger the abrupt release of neuronal SP from the sensory-motor C-fibers to promote the subsequent pro-inflammatory changes: elevations in circulating inflammatory cells, inflammatory

cytokines, histamine, and PGE(2) levels, as well as formation of nitric oxide, reactive oxygen species, lipid peroxidation products, and depletion of key endogenous antioxidants. Concurrent elevations of tissue CD14, a high affinity receptor for lipopolysaccharide, suggest that intestinal permeability may be compromised leading to endotoxemia. If exposure to these early (1-3 weeks MgD) inflammatory/pro-oxidant events becomes prolonged, this might lead to impaired cardiac function, and when co-existing with other pathologies, may enhance the risk of developing chronic heart failure.

PMID: 16819576 [PubMed - in process]

Free Radic Biol Med. 2006 Jul 15;41(2):277-84. Epub 2006 Apr 21.

Long-term moderate magnesium-deficient diet shows relationships between blood pressure, inflammation and oxidant stress defense in aging rats.

Blache D, Devaux S, Joubert O, Loreau N, Schneider M, Durand P, Prost M, Gaume V, Adrian M, Laurant P, Berthelot A. INSERM U 498, Biochimie des Lipoproteines, Dijon, F-21079, France; Faculte de Medecine, Universite de Bourgogne, Dijon, F-21079, France.

Epidemiological and experimental studies have indicated a relationship among aging, dietary Mg, inflammatory stress, and cardiovascular disease. Our aim in the present study was to investigate possible links between dietary Mg, oxidant stress parameters, and inflammatory status with aging in rats. We designed a long-term study in which rats were fed for 22 months with moderately deficient (150 mg/kg), standard (800 mg/kg), or supplemented (3200 mg/kg) Mg diets. Comparisons were made with young rats fed with the same diets for 1 month. Compared to the standard and supplemented diets, the Mg-deficient diet significantly increased blood pressure, plasma interleukin-6, fibrinogen, and erythrocyte lysophosphatidylcholine, particularly in aging rats, it decreased plasma albumin. The impairment of redox status was indicated by increases in plasma thiobarbituric acid reactive substances and oxysterols and an increased blood susceptibility to in vitro free-radical-induced hemolysis.

We concluded that Mg deficiency induced a chronic impairment of redox status associated with inflammation which could significantly contribute to increased oxidized lipids and promote hypertension and vascular disorders with aging. Extrapolating to the human situation and given that Mg deficiency has been reported to be surprisingly common, particularly in the elderly, Mg supplementation might be useful as an adjuvant therapy in preventing cardiovascular disease.

Diabetes Metab Res Rev. 2006 Apr 5; [Epub ahead of print]

Hypomagnesemia, oxidative stress, inflammation, and metabolic syndrome.

Guerrero-Romero F, Rodriguez-Moran M. Medical Research Unit in Clinical Epidemiology, Mexican Social Security Institute, Research Group on Diabetes and Chronic Illnesses, Durango, Mexico.

BACKGROUND: Although hypomagnesemia, oxidative stress, and inflammation are involved in the pathogenesis of cardiovascular diseases, there is not a previous description concerning their potential interaction; thus, the aim of this study was to examine the relationship between metabolic syndrome (MetS), hypomagnesemia, inflammation, and oxidative stress.

CONCLUSIONS: The interaction of inflammation and oxidative stress is related and increases the risk for MetS, whereas serum magnesium levels and MetS are independently associated.

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Magnesium deficiency is prevalent in diabetes mellitus and may result in increased risk to cardiac arrhythmias, hypertension, myocardial infarction and altered glucose metabolism. The association between hypomagnesemia and diabetes has been documented as early as 1946.

Magnesium Deficiency and Diabetes Mellitus: Causes and Effects", Rude, Robert K.,M.D., Postgraduate Medicine, October 1992;92(5):217-223.

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Magnesium deficiency may cause neuromuscular, psychiatric, cardiac, gastrointestinal and metabolic changes.

Magnesium deficiency can be caused by diarrhea, malabsorption, diuretics, renal loss, diabetes, poor nutrition, drugs, alcohol, pregnancy and delivery, organ decompensation and burns
Magnesium and Diabetes", Practical Diabetology, March/April 1991;10(2):1-5.

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The results of this study involving a small number of subjects suggest that patients with diabetes may have a high occurrence of magnesium deficiency.
Magnesium deficiency and insulin resistance in patients with type 2 diabetes mellitus],” Lima Mde L, Pousada J, et al, Arq Bras Endocrinol Metabol, 2005; 49(6): 959-63.

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In this study, decreased levels of magnesium were found to exist in obese children, and an association was seen between magnesium deficiency and insulin resistance. This study shows that a relationship between magnesium deficiency and insulin resistance is present as early as childhood. It is possible that a decreased dietary intake of magnesium may be causing the deficiency of serum magnesium. The study suggests that supplementing with magnesium and/or increasing intake of magnesium-rich foods, may play an important role in helping to prevent the development of Type II diabetes in obese children.
Magnesium deficiency is associated with insulin resistance in obese children," Huerta MG, Roemmich JN, et al, Diabetes Care, 2005; 28(5): 1175-81.

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Magnesium is an essential cofactor for more than 300 different enzymes. Causes of magnesium deficiency include alcoholism, chronic diarrhea/dieting, chronic stress, cytotoxic agents, diabetes and loop diuretics. Frequent findings of magnesium deficiency include hypertension, hypokalemia, migraine headaches, neuromuscular irritability, premenstrual syndrome and urticaria. Complications of long term magnesium deficiency include possibly Alzheimer's disease, asthma, cardiovascular disease, fibromyalgia, postmenopausal osteoporosis and premature delivery. Diseases associated with magnesium deficiency include allergy, Alzheimer's disease, apnea, asthma, diabetes, exercise, hypertension, migraine, mitral valve prolapse, multiple sclerosis, prematurity, PMS, SIDS and the uses of epinephrine and theophylline.
Magnesium is also important in the cytochrome P450 enzyme.
Consider Magnesium Homeostasis: II: Staging of Magnesium Deficiencies", Mansmann, Herbert C., Jr., M.D., Pediatric Asthma, Allergy and Immunology, 1993;7(4):211-215.

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Signs and symptoms of acute magnesium deficiency include arrhythmias, hypokalemia, hypocalcemia and neuromuscular hyperexcitability. Chronic magnesium deficiency can result in bone loss, high blood pressure, vascular disease and abnormal blood sugar metabolism.
Clinical Manifestations of Magnesium Deficiency", Abbott, Lisa G. and Rude, Robert K., Mineral and Electrolyte Metabolism, 1993;19:314-322.

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Magnesium deficiency reduced vitamin E, which is a major antioxidant protecting against free radicals. Magnesium deficiency is a risk factor for pathological effects that may work via free radicals in lipid peroxidation.
Role of Lipid Peroxidation in Vitamin E and Magnesium Deficiency", Gunther, T., et al, Magnesium-Bulletin, 1992;14(2):57-63.

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Magnesium deficiency is associated with cardiac arrhythmias, ischemic heart disease and hypertension. This study concludes magnesium deficiency is a common condition in high risk cardiovascular disease patients such as hypertensives.
Magnesium Deficiency in Two Hypertensive Patients", Seelig, Charles B., M.D., Southern Medical Journal, July 1990;83(7):739-742.

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Magnesium deficiency is associated with atherosclerotic coronary artery disease, coronary artery spasm, coronary artery thrombosis, hypertension, obesity and insulin resistance.
Magnesium Disorders and Cardiovascular Diseases," Purvis JR and Movahed A, Clin Cardiol, August, 1992;15:556-568.

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Magnesium deficiency may be an important risk factor for the development of cardiovascular disease. These researchers have previously reported that magnesium stimulates prostacyclin synthesis. The authors conclude that this magnesium infusion which totaled 600 mgs over 3 hours in normal subjects inhibits prostacyclin and thromboxane B2 release, thrombin- induced platelet aggregation, eicosanoid synthesis and release reaction. The authors further conclude that magnesium administration may provide a novel therapeutic approach to reducing thrombotic disease in man.

Magnesium Plays a Key Role in Inhibiting Platelet Aggregation and Release Reaction", Nadler, Jerry, et al, Arteriosclerosis and Thrombosis, September/October 1991;11(5):1546a.

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Just an aside on the absorbability of magnesium forms:

One of the leading mineral manufacturers, Albion, and patent holder of the chelated magnesium glycinate process notes from their research department (Courtesy Dr. D. Graff, Weber State University) indicates that this form – magnesium bis-glycinate - has an 87% absorption rate compared to magnesium oxide which has an absorption rate of 16%. Magnesium sulfate has an absorption rate of 29%.

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J Am Coll Nutr. 1990 Apr;9(2):155-63.

Cardiology. 1988;75(2):81-9 Hemodynamic and metabolic effects of hypomagnesemia in spontaneously hypertensive rats. Chrysant SG, Ganousis L, Chrysant C. Department of Medicine, University of Kansas, Kansas City, Mo.

We conclude: (1) dietary-induced hypomagnesemia aggravated the hypertension of SHR; (2) it caused widespread tissue calcification; (3) the adverse effects of hypomagnesemia on arterial pressure were possibly produced through calcium-mediated systemic vasoconstriction and increase in peripheral vascular resistance.

References/Resources

(1) DFH teleconference, William Makel, DC presentation on Neurodegenerative Conditions

(2) Russell Blaylock, MD, neurosurgeon

(3) Intracellular Diagnostics, Inc. Foster City, CA (650) 349-5233 (Exatest)

Dr. Russell Blaylock edits NewsMax.com's Blaylock Wellness Report. He is a nationally recognized board-certified neurosurgeon, health practitioner, author and lecturer. He attended the Louisiana State University School of Medicine in New Orleans and completed his internship and neurosurgical residency at the Medical University of South Carolina in Charleston, S.C. For the past 26 years, he has practiced neurosurgery in addition to having a nutritional practice. He recently retired from his neurosurgical duties to devote his full attention to nutritional studies and research. Dr. Blaylock has authored three books on nutrition and wellness, including Excitotoxins: The Taste That Kills, Health and Nutrition Secrets That Can Save Your Life and his most recent work, Natural Strategies for The Cancer Patient. An in demand guest for radio and television programs, he lectures extensively to both lay and professional medical audiences on a variety of nutrition-related subjects. Dr. Blaylock is a member of the international board of the World Natural Health Organization. He is the 2004 recipient of the Integrity in Science Award granted by the Weston A. Price Foundation.

Dr. Blaylock serves on the editorial staff of the Journal of the American Nutraceutical Association and is the associate editor of the Journal of American Physicians and Surgeons, official publication of the Association of American Physicians and Surgeons.

He previously served as Clinical Assistant Professor of Neurosurgery at the University of Mississippi Medical Center in Jackson, Miss., and is currently a visiting professor of biology at the Belhaven College, also in Jackson. \$48/year

www.newsmax.com

Hi Hans,

This is a very good topic. I am one of the unfortunate people that have not figured out how to incorporate supplements into my life without increased ectopics or other side effects.

I took magnesium in the recommended dose and very shortly experienced ectopics and that evening restless legs. I attributed this to the fact that magnesium affects the muscles. Taurine, even in a very small dose, after the first day makes me very hyper and cranky. It does seem to help the ectopics, but is a trade off with how I feel. L-theanine increases ectopics. I tolerate lsv8 and try to eat magnesium rich foods. Fish oil capsules also seem to cause ectopics.

I have done much research and understand how important all these supplements are to the body and really would like to incorporate them into my daily diet (especially taurine), but it's hard to believe something that can make you feel so bad could be good for you. Right now, ectopics are few and far between and all I take is lsv8. The added potassium definitely helps keep the heart calm, but all else seems to aggravate things. I'll be interested in future postings and will follow all.

Barb

Five years ago, shortly after I suddenly went straight from nsr into permanent a-fib, I started supplementing with magnesium. I have no written records from back then, but I think I was ingesting about 600 or 800 mg of elemental magnesium per day.

Since then, over the years, I have had to steadily keep reducing the supplementary dose until now I can't supplement with magnesium at all without inducing noticeable softening of my bowel movement.

One conclusion which could be reached from this data is that my system is now saturated with magnesium.

But, if that is the case it would tend to suggest that a magnesium deficiency was probably not a major factor in the appearance of my a-fib.

This conclusion is also somewhat unsatisfactory because I have (and had) migraines and restless legs, both of which, along with a-fib, are conventionally attributed to magnesium deficiency. While both the migraines and restless legs have improved, they have not disappeared. A successful ablation eliminated the a-fib.

At no time have I noticed any adverse heart related behavior attributable to magnesium supplementation (as glycinate, gluconate, citrate, sulfate, ionic (ww), and other forms of magnesium that I've tried and have forgotten the names of).

Wil S.

I would like to approach this topic from a totally different direction.

Probably most afibbers take Mg thinking it acts in an intercellular way and that is correct of course but Mg also acts in a second way and that is to reduce the strength of the stomach acid. Most of you are familiar with my success in eliminating af by using PPI's (Mg omeprazole) to lower stomach acid. I am certainly not the only one to find lower stomach acid beneficial in reducing or elimination af. A standard off the shelf antacid tablet, "Mylanta", contains 200mg of Mg Hydroxide. To really reduce stomach acid, a double strength Mylanta contains 400mg of Mg. So, if you relate these figures to the amount of Mg Wil Schumann supplements in the above post, and that is 600 to 800mg of Mg elemental a day, then would not this level of Mg supplementation be reducing Will's stomach acid in a big way? Will probably takes his 800mg Mg in divided doses throughout the day (with meals?) so would be maximising the effect of Mg reducing the stomach acid.

So I say Mg is a definite "friend" but not because of the reasons most people imagine. It reduces the stomach acid.

I have tried to find information, like a table etc, about what strength of Mg is needed to reduce stomach acid by a certain amount. Maybe the pharma companies have this info? Please post if you can find this info.

Dean

Part 2 –

Continuing from my original post about the importance of magnesium to maintain health and help prevent inflammation, following are more clips from my files. Note the implications of stress in a system that is attempting to function under magnesium deficiency. Here is that oxidative stress relationship – confirmed once again.

I like so much, this introductory statement: (quote) “ A cursory review of published clinical research, as well as the textbooks devoted to nutritional biochemistry that examine or elaborate the roles of magnesium in the maintenance of the human body should convince even the most avid sceptic of the importance of magnesium to health.”

Back in 2003, this information in a magnesium update reported numerous studies linking magnesium deficiency (MgD) to chronic disease conditions and clinical disorders.

- Reduced intracellular magnesium – a link between Type 2 diabetes and hypertension. Hypertension, Diabetes Mellitus and Insulin Resistance (The role of intracellular magnesium), Paolisso G and Barbagallo M, Am J Hypertens 1997; 10:346-355.
- Magnesium is responsible for the maintenance of the transmembrane gradients of sodium and potassium.
- Patients with refractory hypokalemia often do not respond to potassium supplements until magnesium deficiency is corrected. Magnesium: Its proven and potential clinical significances, Fox D, Ramsoomair D, and Carter C, South Med J 94(12):1195-1201, 2001.
- In the presence of a severe potassium deficiency, magnesium deficiency should be considered.

The Fox et al article, covering 67 published studies, emphasized that MgD is implicated in diabetes, hypertension, cardiac arrhythmias, acute myocardial infarction and atherosclerosis.

The biological mechanisms resulting from MgD connecting these disorders include:

- Dysregulation of the Na-Mg exchanger, resulting in higher intracellular sodium and higher blood pressure.
- A relatively low magnesium level creates an intracellular imbalance between calcium and magnesium, leading to increased vascular tone in arterial smooth muscle and increased blood pressure.
- MgD causes insulin resistance, which causes hyperinsulemia, leading to hypertension, diabetes and hyperlipidemia.
- Testing for intracellular magnesium can be from muscle biopsy, lymphocytes and red blood cells done with NMR spectroscopy and ion specific electrode measures. These tests are expensive so serum magnesium is what is used.

When serum magnesium levels are low, intracellular magnesium is also low. But, many patients with low intracellular magnesium have normal serum magnesium.

- Low levels of magnesium accelerate atherosclerosis by increasing LDL and the oxidative modifications along with the promotion of inflammation. In vitro studies show that low magnesium results in endothelial dysfunction, the first event in the formation of plaque. In addition, oral magnesium therapy has been demonstrated to improve endothelial function in patients with coronary artery disease. Low Magnesium and atherosclerosis: an evidence-based link. Maier JA Mol Aspects Med 2003 Feb 6;24(103):137-46.

- Magnesium deficiency enhances electrical excitability, and arrhythmic changes are typically presumed to be due to a

disturbance in potassium balance. The heretofore poor ability to detect clinical magnesium deficiency has led to its neglect by medical practitioners.

- Magnesium deficiency with stress may be of clinical significance, leading to arrhythmic, hemodynamic, and ischemic changes in the heart.

- Chronic MgD is accompanied by increased free radical generation, which can impact myocardial excitability and contractility. Stress of all kinds will promote free radical generation, and the additive effect of the free radical generation from a magnesium deficiency and stress could potentially be the reason for enhanced sensitivity to stress in the presence of magnesium deficiency.

Alteration of Myocardial Mechanics in Marginal Magnesium Deficiency. Nair RR and Nair P. Magnesium Res 2002 Dec;15(3-4):287-306.

Additional notes indicate the success of treating children and adolescents suffering from asthma with magnesium. And, in another study, a specific magnesium Bis-glycinate form known as Chelazome ® which is pharmaceutically pure and anion free was found to reduce or improve abdominal menstrual pain. Primary dysmenorrhea, Abraham GE, Clin Ob Gyn, 21:139-145, 1978.

Source:

Excerpts from Research Notes October 2003 by Albion Advanced Nutrition, the maker of patented chelated minerals, and of special note, magnesium glycinate.

Title: "Magnesium – Clinical and Health Benefits Still Without Limits"

Jackie

Wil - Thanks for posting your observations.

Initially when loading with magnesium, I was able to get up to 1200 mg daily with no bowel intolerance, but settled on about 800 daily before ablation as a comfortable level.

Although my current intracellular testing has shown my stores are good, I decided to challenge my system to see what would happen. I typically take about 400 - 600 mg. magnesium glycinate daily as a matter of course. This seems to be a good maintenance level for me since I notice symptoms if I try to back off so my assumption is that I use a lot of magnesium daily and need the replenishment.

I ramped my dose up to the former 800 mg and experienced several days of very uncomfortable bowel intolerance. Experiment of one. I need for maintenance, at least 400 mg. magnesium glycinate daily.

Maintaining intracellular saturation may be easier for some than others. Apparently, I was very depleted and I do attribute at least some of that deficiency to the onset of AF. Had I found this site early enough in my AF career, I might have spared myself the ablation.

It's a tragedy that when one first consults a physician for atrial fibrillation that the correct intracellular assessments of the critical electrolytes aren't ordered. It's simply a case that mainstream medicine prefers to give drugs rather than find the cause of the problem, so typical of all our ailments. I had a holistic doctor early in my afib and even he wasn't as knowledgeable as we are on this forum about the importance of electrolytes - especially magnesium and potassium. Perhaps it's not that simplistic or easy, but in retrospect, I wish I had known earlier to try all the suggestions newcomers have available to them to this site currently.

Thanks Hans for making this possible.

Jackie

Ever since I started taking flec and warfarin I have had constipation, which med causes it I don't know. However a few months ago I started taking 125mg chelated mag and 500 mg Taurine everyday, at first it was wonderful, full NSR for 3 weeks, I had been having episodes almost daily until then, ranging from a few minutes to 18 hours. However like Claire Q I found I was having loose bowels each morning, welcomed at first after the constipation, but the Afib has returned although I don't think for as long or as severe, also I am sure I have more energy and less tiredness than before. Do I continue with the supplements or cut them down, 125mg is not a lot compared to some, or do I change brands? Info welcomed.

Jackie OJ

I noticed that one recent member of the forum, Blanche, wrote that she was prescribed Mg by her physician. Is that common? If it is well known that the peeing of AF depletes Mg and K, why isn't prescription of these minerals mandatory for Afibbers after each attack?

Last time I was at the hospital for AF the physician remarked that my K level was a little high. I take both and ACE and an ARB for hypertension is said to influence accumulation of K. Nobody has ever commented on my Mg level. Is test for Mg level included in the standard blood test which is taken each time before cardioversion? In the beginning of my AF career, I had rather long and violent bouts of AF until I was cardioverted, about 30 hours. Will the body replenish such wasting?

Gunnar v/61/na

Hi all,

Further to my post above, I would like to explore further the relationship between Mg supplementation and reduced stomach acid.

If double strength Mylanta antacid tablets contain 400mg Mg as one of the major ingredients then supplementing with 800-1000 mg Mg a day would be the equivalent of taking two and a half double strength Mylanta antacid tablets daily!

This would then lead to vastly reduced strength of the stomach acid and consequently less breakdown of food and more importantly, reduced uptake of nutrients in the gut. In other words the more Mg you take the less effective your stomach acid and the LESS UPTAKE OF MAGNESIUM due to inefficient nutrient absorption. A bit of a paradox really. The MORE Mg ingested the LESS mg actually absorbed into the cells. This suggests heavy Mg supplementation is totally ineffective.

So what constitutes an ideal level of Mg supplementation that is low enough to prevent weakening stomach acid but high enough to be absorbed into the body? Are Mg injections or infusion the only way to elevate Mg in the cells?

These posts below seem to support my theory. The MORE Mg ingested the LESS is actually absorbed.

What do you think? Is there any substance to my theory?

Author: Lynn

Date: 07-15-06 15:38

Hi All,

I have been wondering why I cannot raise my intracellular magnesium much despite over 1 year of supplementation with magnesium (600-800 mg of magnesium glycinate per day and no processed foods). In fact, for the first time, my intracellular levels dropped below normal, to 32.8, during the last year despite the continued adequacy of the blood levels.

Author:PC

Date: 07-15-06 21:33

Hi Lynn,

I too had the same problem. After one year of 800-1000 mg per day of elemental aqueous magnesium (most bioavailable form) my intracellular level went from 34.3 to 34.6. Since I had no trouble with loose stools, I could draw no other conclusion than that I was absorbing it. But I also must have been experiencing simultaneous urinary magnesium wasting.

This could have been due to elevated ANP, which causes not only urinary sodium wasting but Mg wasting as well. Or it could have been due to something else. Now that my AF episodes have come to an end post successful ablation Hans and I have been speculating on what effect this might have on intracellular Mg. AF => increased ANP => decreased Mg. Although ANP is good for K+, it doesn't help much if you can't keep it inside your cells, which requires Mg.

Author: Alex Encel
Date: 07-15-06 15:56

I had the same problem for years. Then I read Dr Myhill and Dr Stoll about magnesium injections or intravenous as being the only way as you can lose the ability to absorb magnesium by oral means also if you have been very athletic which I am even at my age of 71.

I had injections which were more practical than infusions and my magnesium level moved from below the bottom of the reference range more towards the middle [exatest and red cell tests]. My afib is rare and not easy to notice now.

Dean

It is the hydroxide part, OH-, of the Mylanta that is reducing your acidity. Not the Mg.

Gunnar

About the time I started supplementing with Mg, K & taurine, a friend had two afib episodes in fairly short order. Both came on in the early morning. The 1st resolved itself fairly quickly. The 2nd caused a lot of discomfort, high HR and a trip to the ER, although it too resolved itself in 36 hours.

I suggested supplements to my friend. He acted on this suggestion and has not had another episode of afib. He recently had a 24 hour Holter test - with 20 SVE's (PAC's), 80 PVC's and 4 PVC couplets in 24 hours. These are very low ectopic levels. Here is what he has to say:

=====
My regimen of supplements include:
Mg glycinate 360mg per day
Mg citrate 360mg per day
PLUS plenty (approx 3/4 cup per day) of raw nuts (all varieties)

I was taking Potassium as well, but dropped that in lieu of adding Celtic sea salt to most foods. My latest sodium blood serum was normal.

This is included with full compliments of other supplements: Bs, C, 400iu of D, EFAs(OE3) and probiotics.

It has taken 12-15 months on this routine to get where if I miss a few days of the Mg, I don't have muscle twitches. So far so good and I don't plan on seeing what it takes to get the "twitches" again!

=====

Now, back to my own experience: initially, I was concerned about what Dean said - raising of stomach pH by magnesium. I took a lot of Betaine Hydrochloride to counteract this. Now, I usually take my Mg several hours after meals so it (or the K, which is also very basic) do not raise the pH during digestion. So in my case, I don't think the pH issue is the driver for supplement effectiveness. I've not had any afib in 15 months. My most recent holter (within the last month) showed 8 SVE's (PAC's) and 220 PVC's in 24 hours. More recent tests with my Polar monitor, show readings of readings of 0 PAC's and 0-5 PVC's per hour. So I would say that the supplements, including magnesium

are effective and that it is at the cellular level, not in the gut.

I started out supplementing with 1.2 grams of Mg Glycinate/day for several months and was concerned about raising pH, so I backed off to 0.8 grams/day and have continued at this level for a total of 21 months (including two months at 1.2 grams of Mg/day). I've yet to experience bowel issues. Early on in afib, I noticed an eye twitch. This resolved three months into the supplement program. I had an Exatest intracellular Mg test prior to the start of supplementation. The results were at the very low end of normal. I've not had a retest.

George N

Hi George,

K and Mg do not change pH.

There seems to be a confusion about Celtic salt. Bob K had an interesting post about Celtic salt:
http://www.afibbers.net/forum/read.php?f=4&i=12350&t=12331#reply_12350

I use Seltin salt which is:

NaCl: 50%

KCl 40%

MgSO4 10%

and iodine 5 mg

Gunnar

George - if you take the chelated form, magnesium glycinate, it does not break down in the stomach and in fact, does not need or depend on stomach acid to break down but rather, is ready to go into the blood stream with no further chemical reactions needed once it reaches the alkaline condition of the small intestine.

Other magnesium forms do need the presence of stomach acid to break down the compound and should be taken with meals. The chelated form can be taken either with or away from meals since it does not break down in the stomach.

Magnesium is, as you say, an alkalinizing mineral, as is potassium and calcium. They are the body's buffers to be sure the system doesn't become too acidic.

Jackie

Jackie & Gunnar,

I do take the glycinate form of magnesium.

As to the pH, as I'm always one for 1st hand data, I took a pill of each (Mg & K citrate) and ground them up & put in a bit of water, each in their own glass. Then I stuck in some litmus paper & watched it turn black. As I recall (this was 18 months ago), the pH for the K solution was 8- 8.5 and for the Mg glycinate, it was off the scale on the basic side (very black paper). Obviously the quantity of water would make a difference, but in any case the pH was basic.

I did this as I was trying to determine the cause of poor digestion. What I finally figured out, 11 months later, is that natto (food) was the problem. I had thought it was raised stomach pH.

George N v/51/na

Hi George,

now you are talking about the solutions. You cannot say that K or Mg is alkaline or basic. It depends on the whole molecule and how soluble it is.

pH is the negative value of 10th logarithm of the proton concentration: $\text{pH} = -\log\{\text{H}^+\}$

It might be that a solution of potassium citrate is more acid than magnesium citrate, but then say that K is more acid than Mg is wrong especially if it is used in a context where other molecules of the same substance are mentioned like Mg glycinate or Mg hydroxide.

Gunnar

George - testing with pH paper isn't as obvious as it would appear on the surface. It's the property imparted once in the body. True, magnesium is alkalinizing. But, just dipping a pH strip into a solution doesn't always tell you how it's going to react in the body once metabolized. For instance, a lemon dipped will test highly acidic, but in the body lemon and citrus in general metabolize to an alkaline ash and therefore are alkalinizing.

Almonds, high in magnesium, are alkalinizing when metabolized. Soy is rated 4.5 pH (with neutral being 4.0) but I don't see natto on the chart I have. Could be the fermentation changes the nature of it.... or it could be that it, like soy, is just enough alkaline to interfere when consumed regularly. I'm not sure I've read much about digestive problems but I do remember your posts on your experiences with natto food.

Jackie

I went to Dr. Halbert today and got the results from my Intracellular Test. Here are the results does anyone have comments on these would appreciate.

Magnesium 31.9 Range 33.9--41.9
Calcium 4.6 Range 3.2--5.0
Potassium 167.4 Range 80.---240
Sodium 5.7 Range 3.8--5.8
Chloride 6.0 Range 3.4--6.0
Phosphorus 14.8 Range 14.2---17.0

Intercellular Elemental Ratios

Phosphorus/Calcium 3.2 Range 3.5--4.3
Magnesium/Calcium 6.9 range 6.1--12.2
Magnesium/Phosphorus 2.2 Range 1.8--3.0
Potassium/Calcium 36.4 Range 19.1--38.
Potassium/Magnesium 5.2 Range 2.4--4.8
Potassium/Sodium 29.4 Range 19.4---38.9

I was put on a multiple vitamin, Magnesium supplement and CoQ10

What does every one think about these. Dr. Halpert is also going to do a study on COUMADIN AND NATTO.

Steve

Steve - It's good that your doctor has recommended supplemental magnesium as obviously, you are on the low side and with the calcium on the high side (which would be predictable), it could be the reason for some of your afib. Calcium (excitatory) occupying more of the heart cells than magnesium (relaxing).

Again - while your potassium and sodium are within the normal ranges, your potassium is on the low side and the sodium on the high side. This means that inside the heart cells, the potassium (again stabilizing or relaxing to heart cells) is less than the sodium which is excitatory. Work at getting this ratio weighted higher in potassium and lower in sodium. This could be just as simple as eliminating high sodium foods like packaged or commercially prepared foods from your diet. Taking supplements of potassium will offer a sure method.

Again - your chloride is on the high side and others have reported this as well.... but we have yet to find a suitable explanation as to why this happens.

There is a science to analyzing the other ratios and while I've heard it explained, I'm not conversant enough to repeat it here or advise you further. Your doctor should contact the company doing the testing and have the ratio values explained and the nutritional remedies for any imbalances.

Jackie

Thanks Gunnar, George and Jackie for the detailed replies.

I'll toss my theory into the dustbin and start working on another one.
Mg is alkalizing but not in the strength I suspected it would be.
Looks like my understanding of chemistry is a bit lacking too.....

Dean

Jackie,

I think the natto issue was not related to pH or acidizing/alkalinizing. I think I reacted to the natto and this created the digestive issues - not in my stomach but in my intestines - terminal gas. Your posts on leaky gut put me on to this. My guess is that many others may not react in the same way.

It does point out you have to pay attention to what YOUR body is saying.

George

Jackie, I was interested in your comments about soy. My husband is a vegetarian and for the past 3 years had uncontrollable afib and PV ablation in Feb. He still has triggering but no afib due to the ablation. During those 3 years, he really increased his soy intake, now eating soy (burgers and tofu) breakfast, lunch and several dinner's /week. He just started taking another beta-blocker (Toprol) but we've been looking at starting to take magnesium, which he has never done before. What is the affect of the soy in his diet? What affect will it have on the mg supplements?

Elaine

Elaine,

I've been a veg. for 16 years. I've found that my digestion improved dramatically when I got rid of soy. I wouldn't say I never ever eat it now, but it is not a regular part of my diet.

I did this after controlling afib with supplements (K, Mg and taurine). Therefore I can't say that this change did anything one way or the other for afib.

George

My husband doesn't have very poor digestion. He is eating soy because he feels he has little protein in his diet other than soy.

Elaine

George, my husband was interested in your 'getting rid of soy'. He's been adding and adding soy for 3 years, with worsening afib (now better for the ablation but he's stilling triggering). What is your diet? What is your source of protein without soy?

Elaine

Hi Elaine,

Getting rid of soy did not do anything to promote or discourage afib. I basically keep afib at bay with Mg glycinate, potassium and taurine. As I mentioned, getting rid of soy did help my digestion (not my cause of afib).

I eat a mostly vegan diet. Mostly, because I do tend to have some yoghurt for breakfast. The yoghurt helps complement the grains for protein. Other than that, protein comes from grains. I personally feel that if you eat sufficient calories of a varied veg. diet, protein is not a problem. Protein can be an issue with a calorie deficient, limited veg. diet. I also combine legumes with grains (standard veg approach). Nuts, avocado & etc will also complement grain protein.

I try to eat a varied diet of lots of veggies, some fruits, grains. legumes and nuts.

I've been eating this way for 17 years without a problem. As I mentioned, I do supplement with 3 grams potassium, 0.8 grams magnesium as glycinate, and 4 grams taurine/day (doses divided and taken 2x per day).

I monitor ectopic counts, PAC and PVC rates/hour 2x per day while meditating (see the previous conference room for details). I've been on this program for 22 months and these rates continue to drop. I had a 24-hour Holter about a month ago showing 8 PAC's in 24 hours- basically 0/hour.

I've not had afib in 16 months and only 3 episodes since I started the supplements. It did take months to build up my mg stores & eliminate an eye twitch.

I'm an active guy - I'm racing up Pikes Peak (13.3 miles, 7850' elevation gain ending at 14,110') in two weeks. I'm not especially thin - 6' and 200 lbs. I just got home from a 3 day backpacking trip, climbing several thousand feet with a pack and camping at 10,500'. I really don't see protein as a big issue.

I ate a lot of soy for a long time, but I'm now not convinced that it is that healthful, especially the unfermented type.

Hope this helps.

George

Hello George,

I was interested in your comment " It did take months to build up my mg stores and eliminate an eye twitch". Are you saying it was low magnesium that was causing your eye twitch? For the past 6 months I've had an irritating twitch occurring just under my right eyelash line or in my right eyelid. I've been told it is a sign off stress (I don't feel stressed).

I take CardioX - it contains, among other things, magnesium amino acid chelate 3 gms (equiv. elemental magnesium 300mg) per 9 gm daily dose. Perhaps I should increase the dose?

I was glad to read what you said about soy too. I agree.

Best wishes,

Emmi

Emmi,

Yes, from what I understand, twitches, spasms, and cramps are a sign of low mg.

From memory, it took 3 to 4 months for the twitch to completely go away.

An afibber friend, who took my advice to increase electrolytes after his first two afib episodes, had a similar experience. He has not had afib since he started the mg replacement. He also had twitching that took months to resolve.

You can probably find more info on this if you Google eye twitch and magnesium.

George

Thanks for the info. My husband did the medicine route for 3 yrs and had ablation in Feb. He's had no afib but is triggering every day. Instead of pills, we thought to try the supplements since a) he's not in afib so it seems like a good time to try it and b) this website really sells the virtue of them. The comments about soy and high blood sugar had me concerned about his diet. He's not vegan (he drinks milk, but no eggs), but has been a vegetarian for 34 years and was healthy until the afib started. Meds did little to control it, but the ablation was good. His EP gave him Toprol but he had a bad reaction. EP says he should stay on a beta-blocker, but we'd like to see if he can get off of meds. We'll give the supplements until Feb and if no improvement the do another ablation. I really appreciate your info. Thanks.

Elaine

Elaine wrote:

".....but has been a vegetarian for 34 years and was healthy until the afib started."

I believe lack of taurine in the human diet will catch up with most people sooner or later. Although it is considered a non-essential amino acid I believe our ability to make it declines with age, and it is found naturally in animal products, mainly flesh, only.

Joyce

I was searching for something else and came across this "vibrant" exchange about side effects and toxicity of Mg between myself, Erling, Jackie and others in Aug 2004. It contains a wealth of info on Mg and particularly Erling's views on Mg. As most of you know Erling and Jackie are the doyen of Mg followers and I had just recovered from a very bad experience of 9 months of Mg supplementation eventually causing pre hypertensive blood pressure.

In 2004, to say anything bad about the God Magnesium in afibbers was to be branded a heretic but in the above discussion a few people came out with bad Mg experiences too.

I couldn't imagine Hans putting up this current conference room topic back in 2004. He would have been burned at the stake.

It's a pity Erling doesn't post anymore. I really admire his knowledge about LAF.

http://www.afibbers.net/forum/read.php?f=3&i=16990&t=16990#reply_16990

Dean

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Hans R. Larsen MSc ChE, 1320 Point Street, Victoria, BC, Canada, V8S 1A5
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