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Afib and Cardiac Fibrosis

Part 1 – Atrial Fibrosis and the Mechanism of Atrial Fibrillation

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1850572/pdf/nihms19652.pdf

Heart Rhythm. 2007 March ; 4(3 Suppl): S24–S27.

Atrial Fibrosis and the Mechanisms of Atrial Fibrillation
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Abstract

Atrial fibrillation (AF) is commonly associated with congestive heart failure (CHF), and CHF has been shown to be associated with atrial structural remodeling resulting in fibrosis. This atrial interstitial fibrosis has been seen in patients with CHF and animal models of pacing induced heart failure. With atrial fibrosis, conduction abnormalities result in an increase in AF vulnerability. The mechanism of AF that is associated with CHF is still under debate as both focal and reentrant mechanisms have been observed in animal models of CHF. However, recent studies utilizing frequency domain analysis have shown that the AF within this model is characterized by discrete stable, high-frequency areas. The precise signalling processes involved in the development of atrial fibrosis are unknown. Angiotensin appears to play some role, since inhibition of ACE (or ARB) blunt atrial fibrosis in animal models of heart failure and decrease the incidence of AF in patients with heart failure. TGFβ1 also seems to play an important role. Mouse models that overexpress TGFβ1 have profound atrial fibrosis and atrial fibrillation (with normal ventricles). Heart failure in canine models also produces increases in atrial TGFβ1 expression and inhibition of this prevents atrial fibrosis and the development of a substrate for atrial fibrillation. Atrial fibrosis appears to play a role in the development of a vulnerable substrate for AF, especially in the setting of CHF.

Erling

As has been reviewed in various posts, an abundance of science exists supporting the fact that cardiac fibrosis contributes to arrhythmia. If cardiac fibrosis forms in the ventricles, it can be lethal. The formation is probably not exclusively selective for atrial tissue so it's something not to be taken lightly. Science also supports that magnesium deficiency as well as excessive exposure to free-radical damage (ROS) is commonly associated with the genesis of fibrosis.

Fibrosis interference with electrical conduction is examined in numerous reports looking at gap-junction function and the cell-to-cell communication disrupted by the presence of fibrosis. Fibrotic interference in terms of electrical conduction (voltage) throughout the body is observed in various conditions involving the vast system of the fascia and connective tissue. Fibrotic connective tissue proliferation becomes part of the aging process. Tissues in some individuals age faster than in others.

While the etiology of fibrosis and understanding the fibrotic process, itself, is certainly important to understand, it's also important to recognize the need for preventive measures so that fibrosis is less likely form in the first place. Preventive guidelines typically address antioxidant therapy as important but emphasis on magnesium optimization is not common advice.

While not in this review, consideration should also be given to the extensive findings relating fibrotic formations, hypertension and kidney function involvement with aldosterone or angiotension II and the malfunction of the matrix-metallo protein (MMP) system which impacts multiple organ function including the heart. Also not in this review is the impact of oxidative stress injury and resultant cardiac fibrosis in endurance athletes. Many facets of fibrosis to consider.

Recognition that cardiac fibrosis is not something to be ignored is supported in a recent study published in the American Journal of Cardiovascular Disease (2011) *Targeting cardiac fibrosis: a new frontier in antiarrhythmic therapy?* Key-point review follows. This report indicates a need to address fibrosis accumulations as a preventive treatment for arrhythmia and suggests that pharmaceuticals should be developed in addition to those currently available.

While we wait for various new pharmaceutical interventions that inhibit fibrosis formation to be formulated, tested, and receive FDA approval, it makes sense to consider the potential for preventing fibrosis formation in the first place by making sure that our intracellular stores of magnesium are optimal as verified by Exatest, evaluation of existing oxidative stress values and evaluation of critical antioxidants levels that, when missing, allow for inflammation and fibrotic accumulations. Example: deficiency of (antioxidant) glutathione allows for increased lung inflammation and resultant fibrosis.

Additionally, those of us who like to take an active-participation role in personal health management aimed at prevention can consider the use of proteolytic and fibrinolytic enzymes to help break up and dispose of fibrin deposits. Since we have the study showing that Nattokinase degrades Amyloid plaques in brain tissue of Alzheimer patients, those of us using NK and other systemic enzymes such as serrapeptase or the stronger, lumbrokinase, can take comfort in knowing that we are doing good things for our brain, heart, cardiovascular system and undoubtedly, many other organs prone to fibrosis as well if we choose to be our own health advocates and experiment.

What can be more significant than degrading brain plaque? If our hearts happen to benefit as well, it's a win/win all around.

Jackie

Amyloid-Degrading Ability of Nattokinase from Bacillus subtilis Natto
J. Agric. Food Chem., 2009, 57 (2), 503-508

More than 20 unrelated proteins can form amyloid fibrils in vivo which are related to various diseases, such as Alzheimer's disease, prion disease, and systematic amyloidosis. Amyloid fibrils are an ordered protein aggregate with a lamellar cross- structure. Enhancing amyloid clearance is one of the targets of the therapy of these amyloid related diseases. Although there is debate on whether the toxicity is due to amyloids or their precursors, research on the degradation of

amyloids may help prevent or alleviate these diseases. In this study, we explored the amyloid-degrading ability of nattokinase, a fibrinolytic subtilisin-like serine protease, and determined the optimal conditions for amyloid hydrolysis. This ability is shared by proteinase K and subtilisin Carlsberg, but not by trypsin or plasmin.

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Targeting Cardiac Fibrosis: a new frontier in antiarrhythmic therapy?
Hrayr S Karagueuzian, PhD
Am J Cardiovasc Dis. 2011; 1(2): 101–109.

Abstract
Cardiac fibrosis is known to alter cardiac conduction and promote reentry. Recent evidence indicates that fibrosis characterized by increased interstitial collagen accumulation and increased myofibroblast proliferation also promotes enhanced automaticity and early afterdepolarizations (EADs) causing triggered activity. Fibrosis then becomes an effective therapeutic target for the management of lethal cardiac arrhythmias.

While oxidative stress with hydrogen peroxide (H2O2) is shown to readily promote EADs and triggered activity in isolated rat and rabbit ventricular myocytes however, this same stress fails to cause EADs in well-coupled, non-fibrotic hearts due to source-to-sink mismatches arising from cell-to-cell coupling. The triggered activity in the aged fibrotic hearts causes focal ventricular tachycardia (VT) that degenerates within seconds to ventricular fibrillation (VF) after the emergence of spatially discordant action potential duration alternans leading to wavebreak, reentry and VF. Computer simulations in 2D tissue incorporating variable degrees of fibrosis showed that intermediate (but not mild or very severe) fibrosis promoted EADs and TA.

Human studies have shown that myocardial fibrosis was an independent predictor for arrhythmias including sustained VT and VF. A variety of drug classes including, torsemide, a loop diuretic, that inhibits the enzyme involved in the myocardial extracellular generation of collagen type I molecules and the inhibitors of the renin-angiotensin-aldosterone system (RAAS), the mineralocorticoid receptors and endothelin receptors reduce cardiac fibrosis with reduction of myocardial stiffness and improved ventricular function.
It is hoped that in the near future effective antifibrotic drug regimen would be developed to reduce the risk of fibrosis related VT and VF. [www.ncbi.nlm.nih.gov]

Following are selected statements from the full text:

Increased cardiac fibrosis is shown to be associated with cardiac conduction block and reentry in isolated-perfused animal and diseased human cardiac tissues [1-3] as well as in isolated Langendorff-perfused explanted human hearts with dilated cardiac myopathy [4, 5].

While alterations of cardiac conduction [6, 7] and the resulting reentrant wavefront of excitation[8] are uniformly accepted arrhythmic consequences of increased cardiac fibrosis, recent experimental findings in isolated whole heart studies, indicate that fibrosis may also importantly modulate the formation of cardiac after-potentials notably early afterdepolarizations (EADs) that lead to triggered activity causing atrial fibrillation (AF) [9] and ventricular fibrillation (VF) [10-12]

Taken together these findings indicate that increased cardiac fibrosis promotes arrhythmias not only by the mechanism of reentry but also by the mechanism of triggered activity and enhanced automaticity potentially making cardiac fibrosis a highly effective antiarrhythmic target.

We emphasize the importance of fibrosis as (since) non-fibrotic hearts when stressed similarly or at even higher stress levels do no manifest any arrhythmic events.

In 1956, Harmann [25] made his groundbreaking observations on the role of reactive oxygen species (ROS) in the aging process. Thereafter, the concept of ROS became widely accepted in theories of aging [26]. While atrial and ventricular fibrosis may increase with aging, however, fibrosis per se does not promote cardiac arrhythmias [27-30]. Instead, fibrosis as we will see below, provides a substrate that when coupled to a mild form of stress that is of no arrhythmic consequence in non-fibrotic hearts, causes cardiac arrhythmias in the fibrotic heart. In this respect, fibrosis becomes a significant risk factor for increased vulnerability to cardiac arrhythmias.

Why fibrosis is important? The source-to sink mismatch
Our findings highlight the importance of cell-to-cell coupling on the ability of EADs to form and cause arrhythmias in tissue. While isolated ventricular myocytes readily develop EADs and triggered activity in response to oxidative stress, normal non-fibrotic tissue or heart, however, can not generate EADs or triggered when exposed to similar or more intense stressful conditions. This discrepancy supports the supposition that cell-to-cell coupling is a potent mechanism suppressing EAD formation in tissue, by creating a source-to-sink mismatch that prevents local EAD currents generated by a small group of myocytes from reversing repolarization when they are electrotonically coupled to a large group of adjacent normally repolarizing myocytes.

Has the time come for clinical trials to manage cardiac arrhythmias with anti-fibrotic therapy?
The link between ventricular fibrosis and ventricular arrhythmia risk suggests that targeting fibrosis may impart antiarrhythmic benefits. For example, a recent study found that in patients with hypertrophic cardiomyopathy, myocardial fibrosis as measured by the late gadolinium enhancement cardiovascular magnetic resonance (CMR) is an independent predictor of adverse outcome [40]. Interestingly these investigators found that the extent of myocardial fibrosis was an independent predictor for arrhythmias including sustained VT and VF [40]

Collectively, there are a wide range of possible antifibrotic treatments options that target the TGF-β, endothelin-1 (ET-1), connective tissue growth factor (CTGF), angiotensin II, and platelet - derived growth factor (PDGF) networks.[18] The experimental findings are encouraging and suggest that reduction of cardiac fibrosis is possible and may indeed reduce the risk of cardiac arrhythmias [14, 18, 24, 44-47].

For example, studies in rats have shown that pirfenidone mitigates left ventricular fibrosis and dysfunction after myocardial infarction and reduces arrhythmias [48, 49]. Another animal study showed that the agent relaxin-1 reverses cardiac fibrosis and related cardiac dysfunction [50]. Relaxin is a potent antifibrotic peptide hormone that inhibits fibroblast activation (indicated by suppressed expression of α-smooth muscle actin) and collagen synthesis stimulated by angiotensin II or transforming growth factor-β [50].
It is hoped that these basic research findings will be translated to patients at risk of developing cardiac fibrosis-related related arrhythmias. To the extent that such a translation will be successful it is anticipated that a more rational and effective care of patients at risk of VT/VF may be developed.

A method for measuring the presence of myocardial fibrosis in patients with cardiomyopathy is noted to be by the late gadolinium enhancement cardiovascular magnet resonance..[40]
End excerpts.

The entire is worth review for the many key points that are relevant to this ongoing examination. [www.ncbi.nlm.nih.gov]
About the author: Hrayr Karagueuzian Ph.D. [www.med.ucla.edu]

Related Resources:

Atrial Amyloidosis : An Arrhythmogenic Substrate for Persistent Atrial Fibrillation
Circulation. 2002 Oct 15;106(16):2091-7.
Christoph Röcken, Brigitte Peters, Gina Juenemann, Wolfgang Saeger, Helmut U. Klein,
Department of Pathology, Otto-von-Guericke-University, Magdeburg, Germany.
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Structural changes, like atrial fibrosis, may increase the likelihood of atrial fibrillation (AF) occurring in response to triggering events. The influence of isolated atrial amyloidosis (IAA) is largely unknown.

Our study provides evidence that IAA affects atrial conduction and increases the risk of AF. The occurrence of IAA depends on age leading to the formation of an amyloid nidus. The progression and consequences of IAA are then influenced by pathological conditions, such as valve diseases, that increase synthesis and secretion of ANP. The inverse correlation between IAA and atrial fibrosis suggests that these patients may not benefit from treatment with ACE inhibitors to reduce the amount of atrial fibrosis.

Previous studies have shown that atrial fibrosis provides a structural substrate for AF.1 Areas of fibrotic tissue cause conduction inhomogeneities and are included in macro reentry circuits during AF.2 Increased amounts of atrial fibrosis were found in patients with AF, who also showed an activated angiotensin system.3,4 In addition to angiotensin II–related changes, the amount of atrial fibrosis increases with patient age.4
[circ.ahajournals.org]
[circ.ahajournals.org]

Fibrosis in left atrial tissue of patients with atrial fibrillation with and without underlying mitral valve disease
Heart. 2004 April; 90(4): 400–405.
A Boldt,1 U Wetzel,2 J Lauschke,2 J Weigl,2 J Gummert,1 G Hindricks,2 H Kottkamp,2 and S Dhein1

AF is associated with fibrosis. Forms of AF differ from each other in collagen III expression. However, there was no systematic difference in extracellular matrix (ECM) expression between paroxysmal AF and chronic AF. Enhanced concentrations of ECM proteins may have a role in structural remodelling and the pathogenesis of AF as a result of separation of the cells by fibrotic depositions.

Atrial dilatation and fibrosis probably are important factors in the occurrence and maintenance of AF.13 If AF promotes fibrosis, one should expect a progressive increase in fibrosis with increased duration of the rhythm disturbance.

PMCID: PMC1768173
[www.ncbi.nlm.nih.gov]

Gap junction remodeling and cardiac arrhythmogenesis in a murine model of oculodentodigital dysplasia
Proc Natl Acad Sci U S A. 2007 December 18; 104(51): 20512–20516.
Nellie Kalcheva,* Jiaxiang Qu,* Nefthi Sandeep,* Luis Garcia,* Jie Zhang,* Zhiyong Wang,* Paul D. Lampe,† Sylvia O. Suadicani,‡ David C. Spray,‡ and Glenn I. Fishman*§

Abstract
Gap junction channels are required for normal cardiac impulse propagation, and gap junction

remodeling is associated with enhanced arrhythmic risk. Oculodentodigital dysplasia (ODDD) is a multisystem syndrome due to mutations in the connexin43 (Cx43) gap junction channel gene. To determine the effects of a human connexin channelopathy on cardiac electrophysiology and arrhythmogenesis, we generated a murine model of ODDD by introducing the disease-causing I130T mutant allele into the mouse genome. Cx43 abundance was markedly reduced in mutant hearts with preferential loss of phosphorylated forms that interfered with trafficking and assembly of gap junctions in the junctional membrane. Dual whole-cell patch–clamp studies showed significantly lower junctional conductance between neonatal cell pairs from mutant hearts, and optical mapping of isolated-perfused hearts with voltage-sensitive dyes demonstrated significant slowing of conduction velocity. Programmed electrical stimulation revealed a markedly increased susceptibility to spontaneous and inducible ventricular tachyarrhythmias. In summary, our data demonstrate that the I130T mutation interferes with Cx43 posttranslational processing, resulting in diminished cell–cell coupling, slowing of impulse propagation, and a proarrhythmic substrate. [www.ncbi.nlm.nih.gov]
PMCID: PMC2154462 Medical Sciences

Fibrosis in hypertensive heart disease: role of the renin-angiotensin-aldosterone system
Med Clin N Am 88 (2004) 83–97
Arantxa Gonza´ lez, BSca, Begon˜a Lo´ pez, PhDa, Javier Dı´ez, MD, PhDa,b,*
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To make the cardiac fibrosis issue even more complicated, we must consider the findings that genetic mutations are linked to Primary Hypomagnesemia which causes various manifestations of magnesium deficiency.

The following study is just one example. As we know from other discussions on gene flaws, preventive measures to limit the expression of the damaged genes offer a means of managing the result… but undetected, we can appreciate that continual magnesium wasting correlates to arrhythmia and much more. Researcher Dr. Burford-Mason (Canada) noted in her lecture that genetic influences were found in those with arrhythmia and I believe Shannon has mentioned that Dr. Natale has mentioned that as well. That said, keep in mind that it's the gene expression than can be modified and changed. Just because the gene is broken, doesn't mean we have to give into the consequences, according to those who are involved with those treatments and are finding success.

If a familial trend points to arrhythmia in one's family, then all the more reason to be aware that fibrosis can result from magnesium deficiency so heroics with magnesium repletion along with preventive measures for fibrosis are worth pursuing.

Lest one think it's fairly simplistic, the following study illustrates the complexity of genetic disorder "fallout."

Jackie

A missense mutation in the Kv1.1 voltage-gated potassium channel–encoding gene KCNA1 is linked to human autosomal dominant hypomagnesemia
J. Clin. Invest. 119(4): 936-942 (2009).
Bob Glaudemans1, Jenny van der Wijst1, Rosana H. Scola2, Paulo J. Lorenzoni2, Angelien Heister3, AnneMiete W. van der Kemp1, Nine V. Knoers3, Joost G. Hoenderop1 and René J. Bindels1

Primary hypomagnesemia is a heterogeneous group of disorders characterized by renal or intestinal magnesium (Mg2+) wasting, resulting in tetany, cardiac arrhythmias, and seizures. The kidney plays an essential role in maintaining blood Mg2+ levels, with a prominent function for the

Mg2+-transporting channel transient receptor potential cation channel, subfamily M, member 6 (TRPM6) in the distal convoluted tubule (DCT). In the DCT, Mg2+ reabsorption is an active transport process primarily driven by the negative potential across the luminal membrane. Here, we studied a family with isolated autosomal dominant hypomagnesemia and used a positional cloning approach to identify an N255D mutation in KCNA1, a gene encoding the voltage-gated potassium (K+) channel Kv1.1. Kv1.1 was found to be expressed in the kidney, where it colocalized with TRPM6 along the luminal membrane of the DCT. Upon overexpression in a human kidney cell line, patch clamp analysis revealed that the KCNA1 N255D mutation resulted in a nonfunctional channel, with a dominant negative effect on wild-type Kv1.1 channel function. These data suggest that Kv1.1 is a renal K+ channel that establishes a favorable luminal membrane potential in DCT cells to control TRPM6-mediated Mg2+ reabsorption.

Introduction

Occurrence of hypomagnesemia (serum Mg2+ levels below 0.70 mmol/l) in the general population has been estimated to be around 2%, while hospitalized patients are more prone to develop hypomagnesemia (12%) (1). Recent studies of intensive care patients have even estimated frequencies as high as 60% (2). The blood Mg2+ concentration depends on the renal Mg2+ excretion in response to altered uptake by the intestine. Hence, the kidney is essential for the maintenance of the Mg2+ balance (3). The majority of filtered Mg2+ is reabsorbed along the proximal tubule and the thick ascending limb of Henle's loop via a passive paracellular pathway (4). However, fine-tuning of Mg2+ excretion occurs in the distal convoluted tubule (DCT) in an active transcellular fashion initiated by the Mg2+-permeable transient receptor potential cation channel, subfamily M, member 6 (TRPM6) (5, 6). Since the extra- and intracellular Mg2+ concentrations are both in the millimolar range, it has been hypothesized that the membrane potential across the luminal membrane acts as the primary driving force for Mg2+ entry via TRPM6 (6, 7). Previously, genetic studies in families with hereditary renal Mg2+ wasting syndromes revealed several new genes involved in Mg2+ homeostasis, including tight junction proteins claudin 16 and 19 (8, 9), the thiazide-sensitive sodium chloride cotransporter (NCC) (10), the γ-subunit of the Na+/K+-ATPase (FXYD2) (11), TRPM6 (12, 13), and the recently discovered magnesiotropic hormone EGF (14). Despite these discoveries, our knowledge of renal Mg2+ handling remains far from complete.

In this study, we screened a Brazilian family with isolated autosomal dominant hypomagnesemia and identified a missense mutation in KCNA1, resulting in nonfunctionality of the encoded voltage-gated potassium channel Kv1.1.

Results

A heterozygous KCNA1 A763G mutation is causative for hypomagnesemia. [truncated]

Continue: [www.jci.org]
Published in Volume 119, Issue 4 (April 1,2009)

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J. Clin. Invest. 119(4): 936-942 (2009). doi:10.1172/JCI36948.

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Jackie,

"To make the cardiac fibrosis issue even more complicated..." I am not sure why we need to make things more complicated. By now I am sure everybody has understood that we need to ensure that we are replete in magnesium and potassium. That certainly has been repeated *ad nauseum* in numerous postings on this Forum. This posting is largely unintelligble and irrelevant to the vast majority of Forum readers, myself included, so why make it?

Hans

Hans et al, maybe i am mistaken, but i onderstood that remark of Jackie's just to mean that this article would show how complicated the topic of fibrosis is. I am often mistaken and maybe i am now too, but that is how i took it. That article is above my comprehension too, and i just scanned over it.

PeggyM

Peggy's right... that's what I meant. thank you, Peggy. I'll break it down for you, Hans...

I can't see that our readers are so 'unintelligent' that this is utterly confusing... rather it offers explanations as to why some afibbers who are very conscientious with all the protocols suggested are still unable to reverse their afib.

While on the surface, we think we know how to manage afib with the basic protocols. Then comes that interference factor ..fibrosis for instance... ie, the last post noting the gene interference allowing magnesium wasting or inhibiting the optimizing of magnesium (regardless of our heroics) which undoubtedly influences fibrosis production. Vicious cycle.

The *physical* interference from the presence of fibrosis between heart cells means the connections will not be tight enough for continual and proper electrical conduction (and NSR)... and if the fibrosis is formed because a gene flaw prevents optimizing magnesium, then until we get rid of the fibrosis, progress is absent.

Ablations don't remove fibrosis and fibrosis isn't just going to form exclusively in atrial tissue. In the ventricles, fibrosis can cause lethal problems... (the focus of that recent report *Targeting Cardiac Fibrosis*).

This current information is news-worthy. That's why I posted it. It shows official concern about fibrosis and arrhythmia.

If anyone doubts the impact of cardiac fibrosis, please request that I email you the photo of the fibrotic heart.
Remember that this is microscopic until it overtakes the whole heart.

The encouraging news for those that must have proof that enzymes break down fibrotic tissue is that study showing Nattokinase breaks down amyloid plaques in Alzheimer patients.

Jackie

I follow you, Jackie, and I definitely got the overall information from your post, even if some of the technical stuff was over my head. I saw your post as supportive of the use of enzymes as part of an overall nutritional supportive program. Thanks for posting.

Louise

Jackie,

That's good news about Natto and brain plaque.

So to demonstrate the complexity of afib, you post information so complex that no one can understand it? I think most readers understand that it is complicated. Most readers that have been reading closely understand that myocardial fibrosis can be a factor in afib.

If you find any independent studies related to your enzymes, that would noteworthy. None of the studies you post mention anything about your enzyme therapy. And regarding Natto, this website has lots of posters who take Natto every day for it's blood thinning properties and they are still afibbing.

I agree with Hans.

EB

Hello Jackie,

The primary purpose of afibbers.org and the LAF Forum specifically is to provide a “meeting place” for people with **lone** atrial fibrillation where they can share experiences and obtain information which is useful in dealing with their condition. Your posting, although possibly of interest to some readers, does not align with this purpose. For an afibber, especially a newbie, to be confronted with sentences like

Kv1.1 was found to be expressed in the kidney, where it colocalized with TRPM6 along the luminal membrane of the DCT.

And

Upon overexpression in a human kidney cell line, patch clamp analysis revealed that the KCNA1 N255D mutation resulted in a nonfunctional channel, with a dominant negative effect on wild-type Kv1.1 channel function.

This is not the slightest bit helpful and is highly likely to discourage new visitors from ever visiting the Bulletin Board again.

I am not disputing that atrial fibrillation and fibrosis are associated (see my first book published 10 years ago pp. 131-135); however, I would like to see evidence of clinical trials proving that reversing cardiac fibrosis, if that is indeed possible, will eliminate **lone** atrial fibrillation. Most patients with paroxysmal, **lone** atrial fibrillation can be “cured” simply by electrically isolating the pulmonary veins from the left atrium. In other words, afib can be eliminated without reversing fibrosis present in the atrial walls. Thus it is still a big “if” if reversing fibrosis in the left atrial wall will eliminate paroxysmal episodes originating in the pulmonary veins.

In any case it is time to consolidate the many postings on this subject so I have moved them to Conference Room Session 74 under the title **Afib and Cardiac Fibrosis**.

Hans

Thanks so much for posting this Jackie, I appreciate your hard work in getting this information out to us, especially for those of us who are looking beyond what conventional medicine currently has to offer those with Atrial Fibrillation. I acknowledge that some of conventional medicine is helpful, but it is limited (hopefully that will change).

Personally, so many of yours and Erling's posts do me good, as they offer hope for the future in dealing with this condition. The fibrosis issue is one worth writing about. I hope you continue to do so.

Warmest to you,
Po

Po,

Thank you for paying attention to this and taking advantage of Jackie's invaluable information -- not likely available from conventional medicine. I am fully convinced of my 7 year debilitating AF history having been by atrial fibrosis caused by deepening magnesium deficiency over the previous 10 years, and my AF cure ten years ago being from reversing the deficiency, .thus restoring Mg-dependent fibrosis-dissolving enzymes. I arrived on this forum just after curing myself nutritionally, my sole intention being helping others overcome their AF dis-ease.

A case in point is the article Jackie provided, **A missense mutation in the Kv1.1 voltage-gated potassium channel-encoding gene KCNA1 is linked to human autosomal dominant hypomagnesemia** [www.jci.org] Far from being information posted *ad nauseum* (demeaning choice of words?) this article sheds light on what I have posted since first arriving here ten years ago, namely resistance to magnesium repletion, aka "refractory magnesium deficiency". Now we know that one of the reasons may be an inborn error (mutation) in a nuclear gene (KCNA1) encoding a potassium channel protein (Kv1.1) in the kidney's distal convoluted tubules (DCT). Here are some helpful definitions of terms in the text:

-- missense mutation [en.wikipedia.org]
-- Kv1.1 [en.wikipedia.org]
-- voltage-gated ion channel [en.wikipedia.org]
-- KCNA1 [www.ncbi.nlm.nih.gov]
-- autosomal [en.wikipedia.org]
-- TRPM6 [www.ncbi.nlm.nih.gov]
-- DCT [en.wikipedia.org]
-- HEK cells [en.wikipedia.org]
-- episodic ataxia [en.wikipedia.org]

Dr. Alan Gaby, MD, MS biochemistry, wrote the following in his 1994 booklet MAGNESIUM, just one year before my AF woes began in earnest. I have posted it again and again (ad nauseam say you?) ever since arriving here in '02, in hopes it would help others understand their AF condition, and possibly what to do about it. For those who have ears, please listen up: **the root cause of lone AF is atrial fibrosis, and the root cause of atrial fibrosis is magnesium deficiency.**

"As disease progresses, cells lose their ability to function properly. Most of the cells of the body maintain a very high magnesium concentration relative to that in the blood serum. For example, there is about ten times as much magnesium inside the cells of a healthy heart as there is in the serum. This high concentration of magnesium is necessary for cells to perform their various biochemical tasks. However, maintaining this steep concentration gradient between cells and blood requires a great deal of energy. The laws of random motion cause magnesium ions to leak continually out of the cells and into the bloodstream. Each time a magnesium ion leaks out, another one must be pulled back in by special pumps that reside on the cell membrane. Pulling against a concentration gradient is analogous to swimming upstream or to carrying bowling balls up a hill, only to see them roll right back down. As inefficient as that sounds, that is how the body works. Indeed, a substantial proportion of the calories you burn each day are used to maintain higher concentrations of some nutrients inside cells than in the bloodstream.

When you become ill, some of the cells in your body may become less efficient in holding on to

magnesium. The cell membranes may break down, allowing more magnesium to leak out. In addition, the cell membrane pumps that pull magnesium back in may also be weakened by disease. The end result is that disease itself can be a cause of magnesium deficiency. Since magnesium deficiency may have been one of the original causes of the disease, a vicious cycle of greater deficiency and increasingly severe disease may result. A substantial minority of patients fail to improve after taking oral magnesium for months or even years. In these cases, administering magnesium by injection is necessary to overcome their medical problems."

Erling

Thanks Po for your kind comments.

Alan Gaby's wisdom prevails and all afibbers, former and active, should take note.

As this relates to this ongoing investigation of cardiac fibrosis as an interference factor for reversing atrial fibrillation, consider these facts:
*It's well documented that magnesium deficiency facilitates formation of cardiac fibrosis
*The prevalence of myocardial fibrotic formations in heart pathology is abundant.
*If cardiac fibrosis is present, this is no longer considered Lone Atrial Fibrillation
*Regardless of the cause of cardiac fibrosis, addressing it is (or should be) of paramount importance.
*Preventive measures are well-known and available.

Following is a collection of reports examining the connection between myocardial fibrosis and arrhythmia. Since we have so many endurance athletes posting about their arrhythmia experiences, it's worth noting that link to fibrosis as well and the many online publications regarding exercise and cardiac fibrosis.

The following clips from a 2008 study may not have received the emphasis needed when we reviewed it back when first published, but it serves as a documented reminder that when all the routine nutritional protocols we typically recommend fail to work to reverse AF in some, then myocardial fibrosis should be considered as the interfering factor.

1. Title: New risk factors for atrial fibrillation: causes of 'not-so-lone atrial fibrillation'
Bas A. Schoonderwoerd, Marcelle D. Smit, Lucas Pen and Isabelle C. Van Gelder*
Department of Cariology, Thoraxcenter, University Medical Center, Groningen, The Netherlands
Manuscript accepted 18 April 2008

The concluding statement observes, it's better to address underlying factors preventively – specifically fibrosis because once it forms, you are out of the Lone Afib category.

Conclusion
In most patients, AF develops from a substrate that is the common final pathway of different underlying cardiovascular disorders. The process of atrial remodelling leading to this substrate already commences a long time before the first episode of AF occurs. Therefore, treating the underlying disease is the first step in trying to prevent AF and reduce AF burden once the first paroxysms appear in these patients.

Direct quotes from the text:

There is increasing evidence that, from a pathophysiological point of view, the underlying mechanism of lone AF is different than that of AF in the setting of underlying disease. The latter is 'substrate related', i.e. due to diseased and dilated atria with stretch and fibrosis. In contrast, lone AF is probably more related to electrophysiological phenomena (triggers) in structurally normal

atria. This explains why patients with real lone AF have a normal life expectancy when compared with individuals without arrhythmia (Figure 1), a low risk for stroke, and why paroxysmal lone AF does not often progress to persistent or permanent AF.3,4

There are several mechanisms by which sports may induce AF. First, sporting results in enlargement of the cardiac chambers and an increase in the left ventricular mass and left atrial diameter as an adaptation mechanism. One could speculate whether these adaptations are associated with the development of fibrosis or electrophysiological remodelling in the atria. Pelliccia et al.44 determined the left atrial dimension in 1777 competitive athletes and found a dimension of ≥40 mm in 20% of these individuals. Nevertheless, the prevalence of AF was only 0.3% in this group

In most patients, AF develops from a substrate that is the common final pathway of different underlying cardiovascular disorders. The process of atrial remodelling leading to this substrate already commences a long time before the first episode of AF occurs. Therefore, treating the underlying disease is the first step in trying to prevent AF and reduce AF burden once the first paroxysms appear in these patients.

…….clinicians should ask themselves when AF is truly lone? Underlying hypertension is often not recognized after institution of rate-control therapy by β-blockers or calcium channel blocking agents. Long-term data from the Mayo Clinic revealed that only 2% of the total population of patients with AF really have lone AF.3 Apart from the (cardiovascular) conditions traditionally known to be related to AF, other factors may also be involved in the pathogenesis of arrhythmia, i.e. being risk factors for AF. In what follows, we discuss some of these risk factors and the underlying mechanisms by which these conditions may contribute to the development of, apparently lone, AF. These underlying factors also may alter the prognosis. Adequate treatment or reduction of these risk factors possibly may reduce the prevalence of AF and improve prognosis.

Although regular exercise is well known to reduce cardiovascular morbidity,36 excessive (endurance) sport practice is associated with a higher prevalence of AF.

The link between myocardial fibrosis in endurance athletes and/or excessive exposure to free radical generation damage (Reactive Oxygen Species) has also been documented in numerous studies.

An unexpectedly high prevalence of myocardial fibrosis (50%) was observed in healthy, asymptomatic, lifelong veteran male athletes, compared with zero cases in age-matched veteran controls and young athletes. These data suggest a link between lifelong endurance exercise and myocardial fibrosis that requires further investigation.

In a reference to alcohol consumption, the observation that during both drinking and withdrawal of alcohol, a hyperadrenergic state is achieved and alcohol causes an impaired vagal tone. Furthermore, alcohol causes an increase in intra-atrial conduction time which is also reflected by a prolongation of the P-wave.

The entire study is worth reading as it addresses contributors to AF such as sleep apnea, alcohol, exercise, genetics, and inflammation.
[europace.oxfordjournals.org]

2. Diverse patterns of myocardial fibrosis in lifelong, veteran endurance athletes.
J Appl Physiol. 2011 Jun;110(6):1622-6. Epub 2011 Feb 17.
In six (50%) of the veteran athletes, LGE of CMR indicated the presence of myocardial fibrosis (4 veteran athletes with LGE of nonspecific cause, 1 probable previous myocarditis, and 1 probable previous silent myocardial infarction).

An unexpectedly high prevalence of myocardial fibrosis (50%) was observed in healthy, asymptomatic, lifelong veteran male athletes, compared with zero cases in age-matched veteran controls and young athletes. These data suggest a link between lifelong endurance exercise and myocardial fibrosis that requires further investigation.
[www.ncbi.nlm.nih.gov]

3. Post-mortem evidence of idiopathic left ventricular hypertrophy and idiopathic interstitial myocardial fibrosis: is exercise the cause?
Br J Sports Med 2008;42:304-305 doi:10.1136/bjsm.2007.038158
Abstract
A growing body of evidence reporting altered cardiac function and myocardial damage after arduous exercise, together with the increased prevalence of arrhythmias observed in highly trained athletes, suggests that repetitive bouts of prolonged, arduous exercise may be deleterious to long-term cardiac health. We report the case of an experienced, highly trained marathon runner who died suddenly while running.

On post-mortem examination, left ventricle hypertrophy and idiopathic interstitial myocardial fibrosis was found. We believe that life-long, repetitive bouts of arduous physical activity resulted in fibrous replacement of the myocardium, causing a pathological substrate for the propagation of fatal arrhythmias. [bjsm.bmj.com]

4. Is life-long exercise damaging to the heart?
Br J Sports Med 2012;46:9 623-624 Published Online First: 16 February 2012
[bjsm.bmj.com]

5. Increased Incidence of Myocardial Fibrosis with Reduced Cardiac Function in Elite High-Endurance Athletes: A Cardiovascular Magnetic Resonance (CMR) Study
Cardiac MRI: Prognostic Value
(Circulation. 2008;118  _840.)2008 American Heart Association, Inc.
Myra S Cocker1; Oliver Strohm1; David J Smith2; Craig Butler3; Israel Belenkie4; Willem Meeuwisse5; Matthias G Friedrich6

Background: Athletes possess greater relative risk of sudden death due to cardiovascular causes, where autopsies reveal diffuse myocardial fibrosis. Indirect biochemical evidence suggests that athletes have increased collagen degradation and fibrosis. Utilizing CMR based late enhancement (LE) imaging to assess myocardial fibrosis, we hypothesized that fibrosis is a feature of the athlete's heart and is associated with reduced cardiac function.

Conclusion: This is the first investigation to provide evidence for the increased incidence of myocardial fibrosis in elite high-endurance athletes, which is associated with depressed cardiac function. [circ.ahajournals.org]

6. Myocardial fibrosis in a veteran endurance athlete
Reminder of important clinical lesson
BMJ Case Reports 2009; doi:10.1136/bcr.12.2008.1345

Summary
This study reports the cardiac structure and function of a lifelong male endurance athlete, who has run over 148 000 miles, who presented with symptoms of chest discomfort, dyspnoea and loss of competitive running performance. Importantly, the athlete documented several periods of regular intensive endurance activity while suffering with flu-like symptoms. Cardiovascular MRI demonstrated a pattern of late gadolinium enhancement, which indicated myocardial scarring as a result of previous myocarditis.
Myocarditis is a non-ischaemic inflammatory disease of the myocardium associated with cardiac dysfunction and arrhythmogenic substrate. The clinical course of viral myocarditis is mostly insidious with limited cardiac inflammation and dysfunction. However, as in the present case,

overwhelming inflammation may occur in a subset of patients leading to myocardial fibrosis due to recurrent inflammation.

Myocarditis should be suspected in athletes with unexplained cardiac arrhythmias and dysfunction especially if preceded by a flu-like syndrome. An early diagnosis is desirable in order to avoid the risk of fatal consequences since physical activity can enhance the inflammatory process.3 In patients with acute or chronic myocarditis, arrhythmia may be the only clinical symptom in the natural course of the disease. Factors responsible for the increased incidence of cardiac arrhythmias include structural changes, ventricular haemodynamics and vascular changes. The potentially malignant tachyarrhythmias and bradyarrhythmias caused by myocarditis are of particular concern. Acutely, inflammatory processes in the cardiac myocytes and interstitium can lead directly to fluctuations in membrane potential hence arrythmogenesis.3[www.ncbi.nlm.nih.gov]

7. Strenuous endurance exercise: is more better for everyone? Our genes won't tell us
Br J Sports Med doi:10.1136/bjsm.2010. Editorial
André La Gerche David L Prior Hein Heidbüchel

There is accumulating evidence that strenuous endurance exercise may be associated with an increase in some cardiac arrhythmias and speculation that some of the structural changes associated with the 'athlete's heart' may not always be benign. Thus, there is a clear need to expand our understanding of the effects of greater doses of exercise.
[bjsm.bmj.com]
[bjsm.bmj.com]

8. From Dr. John M's blog … he writes:

Cardiac Fibrosis and endurance exercise:
Here, I refer you first to Gretchen Reynolds, the famous NYTimes exercise writer. Her column is where I first learned the story of the marathon rat. I know, humans aren't rodents, but the data from the highly respected lab of Stanley Nattel (Canada) are sobering. Yes, you can guess the results: rats that were run excessively (if given the chance, most rats will indeed run too much) developed areas of scarring (fibrosis) in the heart.

And this very troubling finding doesn't just happen in rats. Here are two studies that suggest the possibility of this irreversible phenomenon happening in humans: (there's many more)

Whyte G, Sheppard M, George K, et al. Post-mortem evidence of idiopathic left ventricular hypertrophy and idiopathic interstitial myocardial fibrosis: is exercise the cause? Br J Sports Med 2008;42:304-305.

Cocker MS, Strohn O, Smith DJ, et al. Increased incidence of myocardial fibrosis with reduced cardiac function in elite high-endurance athletes: a cardiovascular magnetic resonance (CMR) study (abstr) Circulation 2008;118S840–b.

On the association of atrial fibrillation and endurance exercise:
There are hundreds of citations documenting the risk of atrial fibrillation in long-term endurance athletes. This is a start.…

Karjalainen J, Kujala UM, Kaprio J, Sarna S, Viitasalo M. Lone atrial fibrillation in vigorously exercising middle aged men: case-control study. BMJ. 1998; 316: 1784–1785.

Furlanello F, Bertoldi A, Dallago M, Galassi A, Fernando F, Biffi A, Mazzone P, Pappone C, Chierchia S. Atrial fibrillation in elite athletes. J Cardiovasc Electrophysiol. 1998; 9: S63–S68.

Mont L, Sambola A, Brugada J, Vacca M, Marrugat J, Elosua R, Pare C, Azqueta M, Sanz G.

Long-lasting sport practice and lone atrial fibrillation. Eur Heart J. 2002; 23: 477–482.

Hoogsteen J, Schep G, Van Hemel NM, Van Der Wall EE. Paroxysmal atrial fibrillation in male endurance athletes: a 9-year follow up. Europace. 2004; 6: 222–228.

Heidbuchel H, Anne W, Willems R, Adriaenssens B, Van de Werf F, Ector H. Endurance sports is a risk factor for atrial fibrillation after ablation for atrial flutter. Int J Cardiol. 2006; 107: 67–72.

Elosua R, Arquer A, Mont L, Sambola A, Molina L, Garcia-Moran E, Brugada J, Marrugat J. Sport practice and the risk of lone atrial fibrillation: a case-control study. Int J Cardiol. 2006; 108: 332–337.

[www.drjohnm.org]

Jackie

Part 2 – AF Cure via Enzymes?

At this point we know for certain:
-- AF will be caused by atrial fibrosis and/ or necrosis -- either/ both will derange signal conduction pathways [spo.escardio.org]
-- atrial fibrosis/ necrosis will be caused by intracellular magnesium deficiency (M. Seelig, MD, MPH. K. Shivakumar, MD et al. [www.afibbers.net]
-- atrial fibrosis (excessive collagen, fibrin, amyloid) results from insufficient proteolytic/ fibrinolytic enzymes.
-- synthesis of internal (endogenous) proteolytic/ fibrinolytic enzymes requires magnesium-dependent enzymes and cofactors.
-- **ex**ogenous oral enzymes - e.g. nattokinase, serrapeptase, etc. - may remove atrial fibrosis.

What we don't yet know:
-- which oral enzymes/ combinations will be effective/ most effective in removing atrial fibrosis?
-- what dosage will be effective?.
-- what length of treatment for results to be experienced?

Erling

Erling,

You are not posting correct information.

Just keeping it real here, regarding your first statement "we know for certain that AF will be caused by atrial fibrosis..."

This is not correct. You don't know how many people are walking around in NSR with myocardial fibrosis. There are plenty of people with fibrotic hearts that don't go arrhythmic. Some do, but we don't know what percentage. And an important related question; why is it that some fibrotic hearts manifest with afib, why do some not? It would be good to have the answer to this question-

Second, it would be a more correct statement to say that myocardial fibrosis MIGHT BE a cause of AF. This is a correct statement but very different from what you said. There are lots of causes of AF NOT RELATED to fibrosis (such as heart valve surgery, hypokalemia, hyperkalemia, etc).

This is pretty basic stuff Erling. How many times on this website have you seen it posted "we are all an experiment of one?" There are lots of different causes.

I noticed in a post the other day that you yourself do not take enzymes, yet you proclaim yourself to be cured. How can this be, if fibrosis is the only cause, as you stated.

Since you are gearing up to go down the magical enzyme cure for afib road. you should preface that with Jackie's experience. She had an ablation in France due to increased afib episodes. The ablation was very effective, however later, after Jackie started taking enzymes, she experienced afib. That causes a problem for the case you are getting ready to try to build-

Stay accurate my friend!

EB

E.B. - CR 24 examines the connection between AF, fibrosis and remodelling and the supportive statements are from research by cardiology researcher David Van Wagoner, PhD and his team at the Cleveland Clinic. This was offered back in 2004 so it's not something new. Dr. Van Wagoner describes the basic mechanisms of atrial fibrillation...

Just one segment:
Structural changes
•In addition the space between individual myocytes typically becomes more fibrotic, with fatty infiltration, and the atria is less able to contract. These changes make it more likely that blood will remain longer in the atrial appendage, increasing the possibility of clot formation that can cause strokes. (Learn more:Atrial Structural Remodeling)

•Electrical changes:
Fibrillating atria tend to have more complicated patterns of electrical activity. This is due both to the increased fibrosis, and to intrinsic changes in the electrical activity in the atrial myocytes. Research at the Cleveland Clinic Foundation has helped to characterize the electrical remodeling process associated with long-standing AF. (Learn more: Atrial Electrical Remodeling)

The current focus on fibrosis (revisited) would be for those who are unable to make substantial progress by repleting magnesium to the optimal levels and providing other core essential nutrients required for proper energy production indicating that a structural interference should be considered. Addressing fibrosis through enzyme therapy is worth consideration since no heart is healthy when loaded with fibrotic tissue. The photo of a fibrotic heart referenced in CR 24 seems to no longer have an active link. I'm trying to find a way to provide an active link so afibbers can fully appreciate the appalling appearance.

Most important is the need to consider why fibrosis occurs in the first place and take preventive steps. Since it appears that specific systemic enzymes offer both a protective measure as well as remedial to breakdown and eliminate fibrosis, it would seem a most valuable, prudent and very inexpensive preventive approach. Certainly less costly than and less invasive. There is a great deal of important success in treating other ailments with systemic enzymes so it makes sense to apply that to heart tissue as well.

Now, for accuracy....You should know that I had a Natale ablation at the Cleveland Clinic (2003) and my enzyme experience with fibrotic vein tissue is reported in CR 24 was before the ablation but by then, I had actually gone from afib every day to zero events so quite possibly, the enzymes were already helping. I do attribute my ablation success to Dr. Natale's skill and the fact that I had done my own extensive heroics prior to the procedure.

My breakthrough experiences relate to electrolyte imbalances which can be attributed, in part, to a mild kidney function impairment (age), excessive stress, and my (former) lack of diligence when eating restaurant food....all of which have been more carefully monitored. No breakthroughs for

2+ years. I continue to take Nattokinase for managing fibrinogen, but not the stronger systemic enzymes that manage systemic fibrosis. After currently reviewing the literature and all my archived papers from CR 24, I will be taking a separate regimen of enzymes specific for fibrosis... which will most likely help aging kidneys.

Enzyme therapy (for a variety of conditions including fibrosis) is valid and actually quite unique in that it cleans up other problems that may not have yet have surfaced as ailments or symptoms. As Dr. Wong points out..." Fibrosis is the Enemy of Life."

The MgD/Fibrosis/Enzyme connection is an important consideration for all afibbers. Since you don't have arrhythmia, it may be easy for you to dismiss, but for those struggling, it bears consideration as it certainly isn't harmful.

Stay healthy,

Jackie

Jackie,

You think that because I don't have arrhythmia anymore that I am unsympathetic? Your complex is showing itself through your accusation; "physician heal thyself."

I spent 2 1/2 years (between my ablation and my five box procedure) searching for a solution. I know how frustrating it is to try diets and supplements and to seemingly be successful; then here comes afib again. I would not wish this disease on anyone.

I don't dispute the fact that myocardial fibrosis is a factor for some afibbers. I have a surgical report that states that it was a factor for me.

I do dispute a number of other things that are being presented here.

Erling presents fibrosis as the overall cause and the enzymes as the cure-all. That is very far from the truth.

Your assertion in earlier posts, where you claim that these enzymes will successfully treat pulmonary fibrosis, fibroid uterine tumors, and cancer of the spine (in addition to myocardial fibrosis). I want to repeat what I said then: if this were true, then the Nobel Prize for health would be awarded. These are terrible diseases and your enzymes represent a false hope to the sad victims of these maladies.

To sum up, your enzymes are an expensive ($114.00 per month at iHerb), unproven therapy that might benefit a few people. The placebo effect is a very real thing. The Steve Jobs effect is also very real. If people delay until they become persistent, it doesn't make the treatment easier.

EB

Well, here's the thing, EB… a lot of people in this country do not have medical insurance …or insurance that will fully cover all expenses that involve pre-testing, the procedure, the rental of the equipment, the room, etc… and many who do, are still wanting to avoid an ablation procedure because of the invasiveness or the risks.

Since enzyme therapy is working for the practitioners using it for fibrosis prevention and elimination, then why not disseminate the information and let afibbers decide?

When the AF and Fibrosis topic for CR 24 was launched (2004)…. I sent it to Hans for approval.

If he thought it was a bunch of bunk, he would have said, Thanks but No Thanks…Instead, he published it with his congratulations on a good job.

This fibrosis connection and Afib is an important one that seems to be continually ignored, yet it's been known and discussed in the literature for over 10 years. This doesn't mean we should sit by and ignore the connection.

Dr. Van Wagoner wrote the following observations

Atrial Structural Remodeling
"The fibrillating atria are subjected to continuous, high rate electrical activity (with rates up to 500 per minute). This results in impaired atrial contractility, and the initiation of structural changes. At the macroscopic level, structural remodeling is frequently characterized by increased atrial fibrosis and fatty infiltration, both on the endocardial surface, and between muscle bundles.

The figure to the right shows an extreme example of AF-induced fibrosis. The normal left atrial appendage is very compliant and collapses when not filled with blood. This appendage had become a rigid structure, with the fibrosis keeping the appendage open even when empty. Thus, while muscular tissue surrounds the fibrous layer, the contractility of the tissue was significantly impaired by the mechanical restraints imposed by the fibrosis. This can promote blood stasis in the appendage and thus clot formation.

At the microscopic level, fibrosis can isolate muscle bundles. The sections in the figure below are stained to show fibrosis in blue and muscle bundles in red. The panel on the left was from a normal right atrial appendage with little fibrosis apparent. The panel on the right was from the atria of a heart transplant recipient with end stage heart failure and atrial fibrillation due to ischemic cardiomyopathy. It is evident that fibrosis can isolate muscle bundles, and that this can alter the pathway of electrical activation, creating a substrate that can promote the persistence of atrial fibrillation.

In addition to the impact of AF on atrial interstitial fibrosis, numerous subcellular changes are evident in the fibrillating atria. These include altered mitochondrial size and function, increased glycogen storage, loss of contractile elements, and myocyte hypertrophy. The AF-induced cellular changes have been compared to those changes that follow a myocardial infarction in the ventricle, in which the poorly contractile muscle is said to be in a "hibernating" state. This may represent an attempt of the atria to preserve myocyte viability, at the cost of decreased contractility. Studies are ongoing to evaluate the mechanisms underlying structural remodeling in the fibrillating human atria." (end quote)

My thought is that the use of both magnesium repletion and proteolytic/systemic enzymes have the potential to either prevent fibrosis in the first place or if fibrosis formation is stimulated, it will help reduce or eliminate the potential for something more serious than arrhythmia. Since we don't hear much about preventive or restorative therapy from our doctors, why not give these two natural therapies consideration?

Michael Murray, ND, (Director of Product Development and Education for Natural Factors) writes that proteolytic enzymes are helpful in recovery from surgery, fibrocystic breast disease, acute and chronic sinusitis and bronchitis, and chronic obstructive pulmonary disease and asthma. Dr. Wong writes of his experiences ridding various fibrotic collections in the body. Dr. Garry Gordon relies on systemic enzymes and Nattokinase and Lumbrokinase in his practice. Nicholas Gonzales, MD, the NYC oncologist successfully treats cancer patients with enzymes. It's not as if enzymes were something new or experimental.

Dr. Murray references the work of Dr. Hans Nieper…who calls serrapeptase.. The Miracle Enzyme.
Dr. Hans Nieper, a legendary medical doctor known for his extensive use of proteolytic enzymes,

called serrapeptase the "Miracle Enzyme." Dr. Nieper used the enzyme primarily to open up clogged arteries supplying the brain. This enzyme is more powerful than the pancreatic enzymes chymotrypsin and trypsin. It has been used in Europe and Japan for over 25 years. As evident in Table 1, good clinical results have been demonstrated in clinical trials. In addition to its general anti-inflammatory effects, it is particularly beneficial in fibrocystic breast disease as well as upper respiratory tract conditions like sinusitis, bronchitis, asthma, and chronic obstructive pulmonary disease due to its ability to improve the structure and function of the mucus lining.

Dr. Murray: Are proteolytic enzymes preparations safe?
Proteolytic enzymes are generally well-tolerated and are not associated with any significant side effects. Even in people with presumably normal pancreatic function, taking proteolytic enzymes produced no untoward side effects nor did it reduce the capacity for these subjects to produce their own pancreatic enzymes. However, my recommendation is to utilize these preparations only when there is apparent need.
Although no significant side effects have been noted with any of the proteolytic enzymes, allergic reactions may occur (as with most therapeutic agents). Pancreatic enzymes should not be used by anyone allergic to pork; bromelain should not be used in anyone allergic to pineapple; and papain should not be used in anyone sensitive to papaya.

The discussion for positive results in controlling the formation of fibrosis is an important one to everyone and not just because of afib. We need to understand what stimulates the production of fibrotic tissue and take steps to either prevent it in the first place or remediate, once formed. Since fibrosis formation is linked to magnesium deficiency and since fibrosis links to arrhythmia, it is extremely important to consider the potential. As stated initially, for those who have done a variety of heroics and still have ongoing afib, it makes sense to consider that fibrosis may be the cause. Even with ablation, fibrosis will still exist and unless or until the potentiator is managed, the subsequent consequences can be significant.

I'm all for prevention.
Jackie

The link referenced to the photo of the fibrotic heart is no longer an active weblink. I'll be happy to send the photo by email to any afibber who would like to see what the inside of a fibrotic heart looks like.

One step at a time-

Well, here's the thing, EB… a lot of people in this country do not have medical insurance …or insurance that will fully cover all expenses that involve pre-testing, the procedure, the rental of the equipment, the room, etc… and many who do, are still wanting to avoid an ablation procedure because of the invasiveness or the risks.

Whether or not a patient has insurance is a moot point since insurance rarely covers supplements such as these enzymes. At $114.00 for a month's supply, that is a significant expense for anybody. More importantly, your claim that they will avoid an ablation is unproven.

Since enzyme therapy is working for the practitioners using it for fibrosis prevention and elimination, then why not disseminate the information and let afibbers decide?

Working for what percentage of practitioners? Define "working" Do you have studies other than testimonials from people selling the product?

When the AF and Fibrosis topic for CR 24 was launched (2004)…. I sent it to Hans for approval. If he thought it was a bunch of bunk, he would have said, Thanks but No

Thanks…Instead, he published it with his congratulations on a good job.
This fibrosis connection and Afib is an important one that seems to be continually ignored, yet it's been known and discussed in the literature for over 10 years. This doesn't mean we should sit by and ignore the connection.

As I have previously stated, I have no doubt that fibrosis is a factor in some afibbers. What percentage of afibbers, I don't know (and you don't either. Erling thinks everybody has it). I do have lots of doubt about your presented treatments.

Regarding your quotation of Michael Murray, ND, the Director of Product Development and Education for Natural Factors: here is a fellow who stands to directly benefit in the sale of these enzymes. What did you expect him to say? "This stuff is better than snake oil.?" He's not objective, not even close.

And again I have to come back to your earlier claim about these enzymes curing pulmonary fibrosis and cancer etc. It is really an outrageous claim and I don't doubt that you believe it, however you should not be surprised that most people will not believe it because it's just not true.

In the past, you have given some good advice on this website. You are off the mark on this one.

EB

Pitting facts against erroneous opinion sure gets tiresome, but important so readers won't be misled. The statements in the lead post are all facts, e.g. *"AF will be caused by atrial fibrosis and/ or necrosis (cell death) -- either/ both will derange signal conduction pathways"*

Readers: please study carefully all of the referenced **Proarrhythmic Potential of Fibrosis** [spo.escardio.org]

Erling.

PS - More about Dr. Michael Murray, ND. [doctormurray.com]
Quote: *"He is a graduate, former faculty member, and serves on the Board of Regents of Bastyr University in Seattle, Washington."*

Erling,

Dr. Murray has a great resume. He also gets paid for every bottle he sells. When you come up with some independent studies, organized and performed in an objective manner, please enlighten the readership. Until then, all you have are opinions.

Do you really doubt the intelligence of the readership of this website that much?

EB

Sorry EB - you and I will always disagree about the intention and integrity of these well-known leaders in the field of nutritional therapy and functional medicine aimed at health and wellness.

Doctors who push drugs on patients for every symptom get paid whether or not they are effective or the patient feels better or worse or has adverse side effects and ends up in the ER.....either way, they get paid. You go in for surgery… they get paid, whether or not it works. If not, you might get another surgery and they get still paid. Not many people are working for free. Everyone gets paid.

Not that it matters, but I've taken seminars by Dr. Murray and spoken with him at length on two occasions. He is a brilliant, highly-respected naturopathic physician who is a genuine professional interested in sharing ways to maintain and/or restore health in individuals looking for the actual causes of their medical problems. There is a choice.

Many of us writing here prefer to use restorative and functional medical science which identifies and corrects the source of the problem. Just because we do, doesn't mean we are wrong or uninformed or unintelligent or rude to those who wish to remain in the care of a physician who practices strictly conventional medicine.

Actually, that brings up something that is worth sharing.

In 2005, Mark Hyman, MD, who is a well-known leader in the field of Functional Medicine, wrote a an article *The Real Alternative Medicine: Reconsidering Conventional Medicine.* Dr. Hyman points out that rather than just removing the “pathology” of a medical condition, a better approach is to identify and correct functional, genetic and molecular imbalances.

It’s worth reading.
[drhyman.com]

Mark Hyman, M.D. is a practicing physician and an internationally recognized authority in the field of Functional Medicine -- a revolution in 21st century medicine that provides a new road map for navigating the territory of health and illness. He is founder of The UltraWellness Center where he treats patients using this new model in his medical practice.
[www.huffingtonpost.com]

My mission is and always has been to create awareness by examining all options for solutions to medical issues.

Knowledge is power. Knowledge is health,

Jackie

Jackie,

We do disagree on a number of issues but that doesn't make you a bad person (nor does it make me a bad person). Your distrust of traditional medicine is clear- other than Dr. Natale, you don't seem to think too highly of most traditional doctors. Your comment about doctors getting paid applies to functional medicine doctors as well as traditional. Just because Dr. Murray endorses a product, that doesn't challenge his integrity; unfortunately neither does it guarantee the success of the product.

The missing piece is the independent trialing, testing, and analysis that traditional medicines and procedures are subjected to. For example, there are bunches of studies about Catheter ablation, looking at success rates, mortality rates, complications, all sorts of things. Any patient considering an ablation needs only to access the internet to get a world of information. Obviously we do not have access to similar independent information for products like these enzymes. What we have are anecdotal recommendations from people who stand to benefit from the sale of the product.

And we have statements like yours, where you claim that these enzymes will cure pulmonary fibrosis. That really hurts the case you are trying to make.

The readers of this website are a pretty intelligent group. For one thing, they are seeking out information about their disease and that is a smart move. It is their afib and their decision what to do about it. I don't doubt your sincerity in wanting to help and you shouldn't doubt mine.
EB

EB,

If you know the answers, then what should a person who can't afford an ablation do? You seem to be suggesting that's the only cure.

Colin

Colin,

Others may claim to know all the answers, but I don't. When it comes to afib, no one has all the answers.

For me, an ablation did not help, but surgery fixed it. That might apply for you and might not. I would never assume that your afib is like mine.

The issue that I have is that the claim has been made that if you take these enzymes, you will avoid an ablation. That is not proven by any independent study or trial.

Regardless, it is a hard situation for anyone without health insurance, no doubt about that.

EB

It seems to me that I have been dealing since getting here, with an order of preference. i.e. I would prefer that supplementation fixes my problems rather than invasive surgery or other procedures (it has without any doubt in my mind helped my aFib and continues to do so.... cause and effect... I am a physics grad; a scientist. I believe what I see and feel).

I am not a candidate for ablation as my left atrium has enlarged due to being mistreated by an MD. Poor me, but I have moved along to one of the best rated MD's at a top rated facility and began dofetilide in December; I have been in NSR since and when aFib makes an attempt to rear its ugly head I am amazed at what a whack of Potassium does to rid me of it quickly.

I am prepared for invasive intervention (whatever it takes) but will stick with dofetilide as the art progresses (and it does progress rapidly).

I supplement religiously and as a physicist (not practicing Physics) can see cause and effect plain as day.

I cannot comment on this specific argument but want to say that keeping an open mind is critical to the advancement of the cause of finding a cure or at least relief from aFib.

Something as simple as attitude and a daily solid belly laugh has kept my first cousin alive... a survivor of malignant melanoma... for some 20 years. I believe he may make the Guinness Book of World Records. Not that he has not tried everything under the sun including supplements, acupuncture, surgeries galore, etc. He keeps an open mind. No argument presented either way by an MD sways him too far these days. He is his own advocate and tries the "whatever it takes" route.

I think with an affliction like aFib, if we get ten experts in a room around a table, we are bound to come out of that room with 11 opinions and conclusions. That, to me, seems to be the nature of the beast.

Murray L

According to FiveBox EB, "the claim has been made that if you take these enzymes, you will avoid an ablation ..."

How does FiveBox EB come up with these fantastic distortions?

Erling

Erling,

Here is an excerpt from Jackie's post on July 26, 2012: The thread was entitled " Fibrinolytic nattokinase, fibrosis, AF? " You started the thread.

"EB - compared to the cost of an ablation and/or even a second procedure, the cost of systemic enzymes is really quite small and certainly worth trying. Further, if the systemic/proteolytic enzymes eliminate fibrosis and prevent MI, then that seems like a trivial expense compared to those consequences."

There are other examples where Jackie talks about people who don't have health insurance and the point she is making is that if you spend the $114.00 per month, you will will avoid more costly traditional procedures.

My point is, that is not proven by any study or trial.

If I misstated Jackie's point I imagine she would have pointed that out by now. Our debate has been going on for some time.

I hope you won't resort to name calling again Erling. That was disappointing and sad to see.

EB

EB - my comment was not limited to spending $100 or more on enzymes...it's probably totals more than that overall. Rather, it was offered to point out that since science supports the causes of fibrosis and fibrosis is well known to occur in atrial fibrillation... and for people looking for alternatives to an ablation procedure or who are just marking time until they are ready to submit to ablation...or who are not covered by insurance, it is a useful suggestion to connect the dots for eliminating the electrical interference from cardiac fibrosis with proteolytic enzymes since they have worked wonders for other areas of fibrotic involvement. It seems obvious why so few classical studies have been conducted to "prove" that enzymes work. For those who want to experiment, it makes sense to connect the dots. Why not consider the option? If they don't have fibrosis, then the enzymes will just work on other areas of interest and be flushed out of the body. Enzymes offer a healthy approach to living longer and healthier.

Since magnesium deficiency allows for or facilitates fibrotic accumulations and because magnesium deficiency is prevalent in most everyone but especially so in afibbers, common sense would say... replete the magnesium. Then, if an individual has tried the other essential nutrients and lifestyle changes but still has not controlled the arrhythmia, the proteolytic enzymes offer a way to unblock the electrical interference from fibrosis.

Most of the success stories in CR 61, which are only a few published and many more testimonials remain unpublished, are the results of innovative thinking by afibbers who wanted to try alternatives to drugs and surgery for their own reasons. Since various alternatives have been the focus of this forum and Hans' book from the outset, it makes no sense for you to so vigorously interrupt every thread that is focused on helping others find alternative solutions.

Many people prefer to try other options before giving in to ablation. If we help just one afibber who

is unable or unwilling to proceed with an ablation procedure, we have done a remarkable humanitarian service to that person and all who follow. Going back to the original core group of early participants here who told of reversing their afib, the classic example is Fran Ross who reverse hers after 20 years of debilitating afib. Her story is legendary...as is Erling's. There are many, many more who stay in touch and are eternally grateful to have found this forum and reversed their afib without ablations.

Question to readers:
Is there a diagnostic that can assess the degree of fibrotic accumulation in the heart? Wouldn't this show up as lowered cellular energy? Erling? Dr. Tennant probably covers that in *Healing is Voltage*? Can you steer us to that information?

Jackie

There are so many broad assumptions made on this board...that is why studies of large groups of persons (not animals) are so important, IMO.

How do you know that supplements and diet are the only reason for afib remission for persons such as described above? It is not unthinkable that other factors (glandular or other organ changes while aging, for example) could have played a large part in such seemingly rare situations.

Magnesium is not the answer for all of us, as I have said before, magnesium supplements trigger my afib, and when testing magnesium supplements using an EKG, the errant P waves became very pronounced (i.e. stronger) the QRS height was greatly reduced...not a good thing at all. I doubt that I am a unique case in that regard.

My own take is that it is important to seek alternatives to surgeries (including ablations), but not to assume such alternatives are the answer until things improve over the long term. I gave myself over one year to try diet solutions, which included virtually all the touted supplements (tested individually), and that year has netted 6 months of remission followed by over 6 months of heightened sensitivity and now finally persistent afib.

Tom B.

Jackie,

Erling stated that I distorted your claim about taking enzymes to avoid an ablation, I thank you for confirming that I did not.

Jackie, I agree with a lot that you say in this post (that probably surprises you). Regular readers of this website who are still searching for their cure do typically try different supplements and diets (I know I did). There is a fortunate few that find their cure.

But for the majority, the disease progresses to the point of needing either an ablation or a surgical intervention. That's just the way this disease works. Tom B's situation is a classic example.

The debate you and I are having is mostly centered around the enzyme treatment that you are recommending for myocardial fibrosis.

Erling presents myocardial fibrosis as the be all, end all problem for everybody. You know as well as I do that is not the case. It is a contributory factor to some, but what percentage we don't know. Then the issue of the enzymes' effectiveness: for argument's sake, they might help some people but again, we don't know what percentage. So you are recommending a supplemental regimen not knowing if the patient has fibrosis and also not knowing if if the enzymes will help.

The likelihood of success is pretty low with those kind of parameters.

I understand your comment about helping one afibber and that is well and good. I am concerned about the 99 other afibbers who spend their money, tried the treatment and their afib progressed.

If you presented the enzyme regimen with the disclosure that these products have not been proven to cure afib, then you and I would have much less to debate about.

Your question about a diagnostic to assess the degree of fibrosis is certainly a relevant question. Is there a credible source with the answer?

EB

Part 3 – Magnesium Deficiency and Fibrosis in AF

From **Magnesium Deficiency in The Pathogenesis of Disease** (1980) by Milded S. Seelig, MD, MPH. [mgwater.com]

Here are a few relevant quotes (my emphases). References are linked alphabetically at the bottom of the book's page:

8. **Clinical Cardiac Abnormalities and Magnesium** [mgwater.com]

8.1. **Cardiomyopathies Not Secondary to Disease of the Major Coronary Arteries or to Infection**

"Hudson (1970) suggests four features that characterize cardiomyopathies: cardiomegaly, endocardial thickening, mural endocardial thrombus, and myocardial scars. He considers the **fibrotic cardiomyopathies** (sometimes accompanied by foci of myocardial calcification) to be likely related to hyperreactivity to vitamin D in infancy. He cites **myocardial fibrosis** in adults as the commonest form of idiopathic cardiomyopathy; it can be familial or occur as isolated cases."

9. **Magnesium Deficiency and Cardiac Dysrhythmia** [mgwater.com]

9.1. **Electrocardiographic Changes of Experimental Magnesium Deficiency**

"Bajpai et al. (1978) have correlated the ECG changes produced by hypomagnesemia in rats with abnormalities in mitochondrial oxidative phosphorylation. They confirmed the significant reduction of the P-, QRS-, and T-wave voltages of magnesium deficiency, and attributed the changes to decreased energy production associated with the decreased oxidative phosphorylation. They propose that magnesium deficiency reduces the amount of current transmitted from cell to cell, as a result of increased resistance in the intercellular connections (desmosomes) as these membrane structures swell [similarly to the swelling of the plasma membranes of magnesium deficient erythrocytes (Elin, 1978)]."

"When magnesium (0.5 mEq/kg) was given intramuscularly, clinical improvement was noted within six hours. The precordial impulse improved, the cardiac sounds became louder and of better quality, and a stable normal sinus rhythm developed (Caddell, 1969b). Caddell made an interesting observation that seems worth exploring. She commented that survivors of PCM [protein caloric malnutrition] often have persistent PR interval and T-wave abnormalities, that **endomyocardial fibrosis** is found in the same geographic regions as PCM, and that the morphology of the cardiac lesions resemble those that Selye (1958f) reported to be protected

against by magnesium and potassium. She speculated, thus, that the ECG abnormalities of PCM might reflect mineral imbalance, and that persistent deficiencies of magnesium and potassium might be contributory to the development of **endomyocardial fibrosis** (Caddell, 1965)."

"ECG tracings from a 39-year-old man, who died 2 years later of familial **myocardial fibrosis**, disclosed auricular [atrial] flutter and occasional ventricular ectopic beats with flat and negative T waves in several leads. (Nieveen and Huber, 1970)."

9.3.8. **Infantile Arrhythmias and Cardiomyopathies**

"An infant born with A-V block, who soon developed congestive heart failure and died at two months of age, had degenerative changes and calcification in the central body of the A-V bundle, **fibrosis** in the left bundle branch, and subendocardial calcification adjacent to the right bundle branch (R. A. Miller et al., 1972). Kariv et al. (1964, 1971) observed that the familial form of cardiomyopathy, which begins very early in life, is characterized by arrhythmia, and that this suggests very early development of histopathologic changes. Arteriosclerosis of large and small coronaries, and **focal myocardial necrosis and fibrosis**, have been found in infants who died suddenly and in others who had been ill with clinically manifest heart disease, many of whose first cardiac manifestations developed at about two to four months of age, the age of peak incidence of SIDS." [sudden infant death syndrome]

9.3.9.1. **"Benign" Arrhythmias**

"Many cardiac electrical disturbances occur without detectable heart disease (Kastor, 1973). Their cause is unknown. Among the abnormalities reported in hearts diagnosed as "healthy" are paroxysmal atrial fibrillation (Peter et al., 1968), supraventricular tachycardia (Cass, 1967), bundle-branch blocks (Beach et al., 1969; R. F. Smith et al., 1970), "benign" premature ventricular beats and parasystoles (Myburgh and Lewis, 1971), and even some cases of paroxysmal ventricular tachycardia (Lesch et al., 1967). Kastor (1973), who presented this complex of "benign" electrical disturbances, commented that people with such abnormal ECGs cannot be distinguished from normal subjects by any specific pathological abnormality. He suggested that reentrant arrhythmias with preexcitation [Wolff-Parkinson-White (W-P-W) syndrome] might represent a form of congenital heart disease, as might cases of sinus node disorders (Spellburg, 1971). He points out that **fibrosis** of the peripheral bundle blocks might be responsible, and that since it occurs in the absence of evidence of coronary artery, myocardial, or infectious disease it, like the other cited "benign" arrhythmias, can be categorized only by the functional disorder, or the presence of **fibrosis of unknown cause.** The tachyarrhythmias of the W-P-W syndrome are predominantly paroxysmal supraventricular tachycardia (in 70-80% of the cases) and atrial flutter / fibrillation (Tonkin et al, 1976)."

9.3.9.2. **Similarity to ECGs of Magnesium Deficiency**

"The similarity of the "benign" arrhythmias of unknown cause (Kastor, 1973) and that seen in the patient with familial **myocardial fibrosis** with those of experimental and clinical magnesium deficiency is provocative. Comparison of the tracings obtained from Kellaway's and Ewen's (1962) patient during her acute magnesium depletion and that of Nieveen and Huber (1970) show remarkable similarities. Very-thick-walled arterioles were seen in a muscle biopsy taken from the patient with familial **myocardial fibrosis** (Nieveen and Huber, 1970)."
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From **The magnesium Factor** (2003) by Mildred S. Seelig, MD, MPH, and Andrea Rosanoff, PhD.
Page 11:

"Among the enzymes that have been studied intensively, over 350 need magnesium, directly, to

do their jobs properly. ... In addition to the more than 350 enzymes for which magnesium is directly necessary, it is indirectly needed for thousands of others."

"...magnesium is directly necessary to the enzymes that... make proteins (all enzymes are proteins)..." [such as the internally synthesized fibrin-forming and fibrin-dissolving (fibrinolytic) enzymes.]
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From **K. Shivakumar, MD, et al:**

Kumar BP, Shivakumar K, Kartha CC.
Magnesium deficiency-related changes in lipid peroxidation and collagen metabolism in vivo in rat heart.
Int J Biochem Cell Biol. 1997 Jan; 29(1):129-34.

"Magnesium deficiency is known to produce a cardiomyopathy, characterised by myocardial necrosis and fibrosis."

[www.ncbi.nlm.nih.gov]
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Shivakumar K.
Model of cardiovascular injury in magnesium deficiency.
Med Hypotheses. 2001 Jan; 56(1):110-3.

"Magnesium deficiency is known to produce cardiovascular lesions."

[www.ncbi.nlm.nih.gov]
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C. Kumaran and K. Shivakumar
Superoxide-mediated activation of cardiac fibroblasts by serum factors in hypomagnesemia
Free Radical Biology and Medicine, Vol. 31(7) (2001) pp. 882-886

"Magnesium deficiency is known to produce myocardial fibrosis in different animal models..."

[www.ncbi.nlm.nih.gov]
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Shivakumar K.
Pro-fibrogenic effects of magnesium deficiency in the cardiovascular system.
Magnes Res. 2002 Dec; 15(3-4):307-15.

"Magnesium deficiency is known to produce cardiovascular injury."

[www.ncbi.nlm.nih.gov]
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Chaturvedi Kalawati Jagmohan Das Memorial Award

2001 Citation.

Dr. K. Shivakumar

The Chaturvedi Kalawati Jagmohan Das Memorial Award was instituted in 1975 by Dr. S.P.

Chaturvedi in the memory of his parents. This prize is awarded once in three years to an eminent scientist, preferably a medical person, for his/her work in the field of Cardiovascular Diseases.

The Prize for 2001 is being presented to Dr. K. Shiva Kumar, Scientist-E, Division of Cellular & Molecular Cardiology, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Thiruvananthapuram, ***for his research work on myocardial fibrosis in response to cerium and magnesium deficiency.***

The research work carried out by Dr. K. Shivakumar has led to the postulation of a neural pathway of activation of cardiac fibroblasts and delineation of a novel protective mechanism that may limit fibrogenesis in the aging myocardium and aetiopathogenesis of tropical endomyocardial fibrosis.

Dr. Shivakumar has more than 25 publications in reputed international and national scientific journals.

Erling

Special thanks to Paul Mason for making this priceless information available online at his website and Thank you, Erling, for taking the time to sift out these very relevant nuggets from that very complete masterpiece. Most of all, THANK YOU, angel Mildred Seelig, for compiling the extremely valuable documentation on the importance of magnesium - all in one place as an historical account and most valuable reference. It's unfortunate to have so much relevant information basically ignored by the medical establishment.

This fits in nicely with the new report that is in the works on this topic. The studies supporting the connection are abundant.

Jackie

Thanks Jackie, and thank you, dear Mildred Seelig, from the bottom of my heart for this most extraordinary work published in 1980 **Magnesium Deficiency in The Pathogenesis of Disease**. One day in '96 I happened to see it at the medical school library -- the information in Chapter 9 started me on the path leading to cure of my debilitating AF by 2002

And sincere thanks to you, Paul Mason, for putting this entire book on your fabulous website -- all 400-plus pages!! [mgwater.com]

It is to the medical establisment's shame that this hugely important 1980 work has rarely "seen the light of day" in the "mainstream" or in the so-called "alternative". For instance, **Frustaci et al (1997)* shows that fibrosis correlates with LAF but doesn't mention the long-proven connection with magnesium deficiency.** My personal 12 year experience ('90 to '02) with arrhythmias and its doctors was that not one - no matter how "board certified" - could tell me the slightest thing about the heart's - the entire body's - profound need for magnesium.

Erling.

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* Circulation. 1997 Aug 19;96(4):1180-4.

Frustaci A, Chimenti C, Bellocci F, Morgante E, Russo MA, Maseri A.
Histological substrate of atrial biopsies in patients with lone atrial fibrillation.
[www.ncbi.nlm.nih.gov]

BACKGROUND

Lone atrial fibrillation (LAF) is a common clinical syndrome, but its origin remains unknown.

CONCLUSIONS:
Abnormal atrial histology was uniformly found in multiple biopsy specimens in all patients with LAF. It was compatible with a diagnosis of myocarditis in 66% of patients (active in 25%) and of noninflammatory localized cardiomyopathy in 17% and was represented by patchy fibrosis in 17%. **The cause of the pathological changes**, which were found only in atrial septal biopsies but not in biventricular biopsies, in 75% of patients **remains unknown.**

[ajpheart.physiology.org]

Am J Physiol Heart Circ Physiol 291: H436–H440, 2006.
First published February 10, 2006.

Cardiac fibrogenesis in magnesium deficiency: a role for circulating angiotensin II and aldosterone

S. Sapna,* S. K. Ranjith,* and K. Shivakumar
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[Introductory paragraphs:]

MAGNESIUM PLAYS an important role in maintaining the structural and functional integrity of the cardiovascular system (1, 2,19). It has been known for a long time that magnesium deficiency produces a cardiomyopathy characterized by focal myocardial necrosis and fibrosis (3, 9). Many theories have been advanced to explain the molecular basis of myocardial damage associated with magnesium deficiency (22). A large body of experimental evidence suggests that increased oxidative stress in the setting of an immunoinflammatory reaction may produce cardiovascular injury in magnesium deficiency (30). Furthermore, alterations in collagen metabolism and fibroblast proliferation rates, reported earlier from this laboratory (12), point to the activation of cardiac fibroblasts in a rodent model of acute magnesium deficiency. Whereas the pro-oxidant agents that may contribute to increased oxidative stress in magnesium deficiency have been investigated (30), factors that modulate cardiac fibroblast activity and promote myocardial fibrosis in magnesium deficiency have received little attention.

We had demonstrated earlier that serum from magnesium deficient rats (MgD) has a more pronounced stimulatory effect on cell proliferation, net collagen production, and superoxide generation in adult rat cardiac fibroblasts than serum from magnesium-sufficient rats (MgS). Superoxide was found to mediate the enhanced proliferative response of the cells (14). The pro-fibrotic serum factors were, however, not identified. The present study was undertaken to identify the circulating factors that were earlier shown to activate cardiac fibroblasts in hypomagnesemic rats.

ANG II [angiotensin II] is an important regulator of cardiac fibroblast function and collagen turnover (6, 17, 28). Moreover, ANG II can trigger most of the changes reported to be associated with magnesium deficiency, including induction of pro-oxidant (24, 32) and pro-inflammatory conditions (8, 16). It stimulates the expression of intercellular adhesion molecule-1 by human vascular endothelial cells (16) and that of E-selectin by human coronary endothelial cells to promote leukocyte adhesion to these cells (8). Furthermore, it is a very potent inducer of vasospasm (26) and an important etiological factor in hypertension (10, 18). ANG II also promotes hypertrophy and hyperplasia of vascular cells (25). Based on these observations, Shivakumar (20) published a model of cardiovascular injury in magnesium deficiency that postulated a role for the renin-angiotensin system in the pathobiology of magnesium deficiency.

Against this backdrop, the major goals of the present study were 1) to test whether circulating ANG II and aldosterone activate cardiac fibroblasts and 2) to examine levels of expression of the

components of the renin-angiotensin-aldosterone system (RAAS) in magnesium deficiency. Furthermore, preliminary experiments on the effect of orally administered enalapril on lipid peroxidation in the heart were also performed to confirm the involvement of ANG II in vivo in magnesium deficiency. This communication presents evidence for the first time that circulating ANG II and aldosterone may play a role in activating cardiac fibroblasts in magnesium deficiency. The findings provide useful insights into mechanisms of cardiac fibrogenesis in magnesium deficiency that remain unclear.

9.1. **Electrocardiographic Changes of Experimental Magnesium Deficiency** [mgwater.com]

"Bajpai et al. (1978) have correlated the ECG changes produced by hypomagnesemia in rats with abnormalities in mitochondrial oxidative phosphorylation. They confirmed the **significant reduction of the P-, QRS-, and T-wave voltages of magnesium deficiency, and attributed the changes to decreased energy production associated with the decreased oxidative phosphorylation. They propose that magnesium deficiency reduces the amount of current transmitted from cell to cell, as a result of increased resistance in the intercellular connections (desmosomes) as these membrane structures swell [similarly to the swelling of the plasma membranes of magnesium deficient erythrocytes** (Elin, 1978)]."

[Note: the significance of those facts to AF was addressed on page 2 of CR 72 *The Potassium / Sodium Ratio in Atrial Fibrillation* (2011) [www.afibbers.org]

ATP is synthesized from oxygen and food molecules in a process requiring Coenzyme Q10, carnitine, magnesium, ribose, phosphate, and many co-factors. In body cells the continuous pumping of K and Na consumes about 25% of the ATP produced, while in high energy-demand heart, brain, and neurons the consumption is as much as 70%.[4] Therefore, if ATP and magnesium are deficient, and if the intracellular ratio of K to Na is low, the cells' voltage will be low. Low cell voltage may express as abnormalities in cells and systems throughout, as in blood pressure, kidney function, electrically excitable tissues of the heart and brain.]

Erling

Erling and others,

I am trying to get a handle on the electrochemical processes but in the meantime would like to ask your advice about my situation.

My energy has been low for over 5 years. 2 years ago even if I was in rhythm, I often couldn't even maintain body temperature. I remember sitting on top of a floor furnace 45 minutes before I could stop shivering and get back to bed even though the apartment wasn't below 65 degrees. I subsequently took ribose 3x day. If I skipped a dose I noticed it. Finally after 3 months the boost was not noticeable and I quit. On several occasions at night, I felt I didn't have enough energy for my essential organs.... scary feeling.

My VA cardios reported that my heart was normal except for slight thickening of the endocardium(?). They always take a lot of blood work and it is normal. Since that time I have used supplements coq10, magnesium and K sporadically. Lately- religiously except for potassium. Plus carnitine and more recently fish oil and systemic enzymes. Lately, my arrhythmia has included a very debilitating high rate rhythm that fortunately reverts to slower arrhythmia after a few minutes.

I read Dr. Sinatra's book on the 3 essentials for hearts. He indicated that even after an event the heart would return to normal levels of atp if supplemented with ribose for a period of 2 weeks.

I have a rare appt with the cardiologist tomorrow. Any suggestions you or anyone else may have

are appreciated. They are normally cooperative in the area of testing. I also had a radiologic stress test which showed normal blood flow.

Milleniaman

Part 4 – Fibrinolytic Nattokinase, Fibrosis and AF?

Please read this informative article: [cnmwellness.com]

Natto and Its Active Ingredient Nattokinase
Martin Milner N.D. and Kouhei Makise M.D. -
Alternative & Complementary Therapies -- June 2002

Does its description indicate that nattokinease would be effective in removing atrial fibrosis? If so, what would be the effective dose? For how long? At what cost?

Quotes:

-- Nattokinase is the most potent fibrinolytic enzyme found among the approximately 200 foods that have been investigated for oral fibrinolytic therapy.

-- It is noteworthy that fibrinolysis (nattokinase's main physiologic effect) is a natural physiologic process. Fibrin, a naturally
occurring blood protein is broken up into fibrin-degradation products during fibrinolysis. There are several naturally occurring fibrin-degradation processes, all of which are well-documented in conventional literature. Nattokinase, therefore,
undeniably promotes mechanisms of action that occur naturally.

-- In summary, it is noteworthy that nattokinase lyses fibrin directly, changes prourokinase to urokinase, and increases tissue plasminogen activator (t-PA).

-- Natural Products:

Bromelain has been well documented for its ability to activate fibrinolysis via stimulating plasmin production. Because nattokinase inhibits fibrin more effectively in vitro than plasmin, nattokinase is a much more potent fibrinolytic agent than bromelain.

Garlic. Both fried and raw garlic increase serum fibrinolytic activity significantly and reduce fibrinogen and fibrinopeptide by 10 percent. Streptokinase activated plasminogen and fibrinopeptide Bβ 15–42 are also increased by approximately 10 percent.

Chinese ginseng. Panax ginseng has inhibited fibrinogen conversion to fibrin. The herb's mechanism of action appears to be via promoting urokinase's fibrinolytic activity.

Erling

Erling - This information came up during my examination of various enzymes for blood viscosity management for CR 39 and 40. It was thought back then and still is, that the fibrinolytic enzymes such as NK are very effective for managing fibrinogen and blood viscosity as preventive for thrombolytic activity and in combination with the proteolytic enzymes, may be also useful in fibrosis but are not as potent as the serrapeptase or lumbrokinase typically used for the tougher, more massive fibrotic lesions.

It's important for afibbers to measure fibrinogen so they can keep tabs on their blood viscosity and therefore clotting tendencies during an event if they are not using an anticoagulant.

If I had a known fibrosis or if I were struggling with ongoing AF with a history of heavy oxidative stress exposure and resulting inflammation; i.e., runners, heavy aerobic exercisers, etc, I'd use the higher dosing recommendations for serrapeptase and obviously using NK as a thrombolytic just in case of prolonged events.

The important fact to remember here with NK and viscosity is that NK increases PAI-1 levels that fall during the early morning hours when clot formation is likely to occur. (Plasminogen Activator Inhibitor - 1) as mentioned in CR 39 and 40. Therefore, the bedtime dose of NK is obviously a very important preventive measure.

As you know, I'm a huge fan of NK since it spared me from that clot in the LAA from my post-ablation cardioversion.

Jackie

Hi all - The following is my personal experience of eliminating fibrosis with an orally administered fibrinolytic enzyme, namely nattokinase.

About half of my sixty-some working years were spent at aerospace design engineering, the others at residential design and contracting / hands-on construction with dusty environments and physical hazards. In addition to decades of exposure to airborne dust from wood, cement, fiberglass, solvents, paint fumes etc., a severe fall damaged my shoulder resulting in a number of dislocations.

At some point I began experiencing subtle random chest pains which I thought might be caused by a degree of "pulmonary fibrosis" from decades of inhaled dusts and fumes. More worrisome, increasingly frequent chest-centered discomfort on awakening seemed likely due to a narrowed artery. A few years earlier an imaging stress test had revealed possible ischemia at the heart apex, so I looked for a natural way to reduce arterial blockage. The nattolinase article by Drs. Milner and Makise (above) encouraged me to start taking nattokinase to see what it might do for the chest pains, if anything.

The first result came after just three weeks of nattokinase use -- unintended and unexpected. The constant shoulder pain that had become increasingly severe was gone!!. In earlier consultation with a surgeon for relief, an MRI and arthrogram had clearly revealed the extent of damage and scar tissue from the long-ago fall. I was told that the physical damage could be repaired surgically, but with no assurance that the pain would be relieved, so I delayed. When the pain vanished spontaneously it seemed clear that internal scarring (fibrosis) had been taken away by the fibrinolytic potency of the nattokinase. This complete relief from pain (and surgery) came about even before finishing the first $18.00 bottle from iHerb!

With another month of nattokinase use all random chest pains had stopped, and the (presumed) anginal pains had also stopped. I'm quite optimistic that nattokinase, or some other fibrinolytic enzyme, can be successfully used to remove cardiac fibrosis and in time restore normal conduction and NSR.

Erling

The role of fibrosis in pathogenesis and of lone (idiopathic) atrial fibrillation is an interesting and complex issue. The main complexity lies in the fact that we do not posses standardized, easy and accessible means to quantify the extent of fibrosis in people afflicted with LAF. MRI scans can be used to assess the degree of fibrosis, but most LAF patients have normal MRIs. Does it mean

they do not have any fibrosis or alternatively that even latest MR imaging is not sensitive enough to detect the presence of atrial microfibrosis. Interatrial voltage mapping can be used to evaluet for scar tissue, but studies demonstrate that it is notably absent in majority of patients with LAF. I've recently read publication based on small study done in Russia which took atrial tissue biopsy samples from patients with LAF and concluded that most of them had viral genome present in their myocardium. Despite the fact that patients in the study had normal MRIs, their biopsy samples revealed the presence of sub clinical cell necrosis and fibrosis on a cellular level. The authors of the study conclude that atrial myocarditis, of predominantly viral genesis, is a fairly common cause of idiopathic atrial arrhythmias. I am going to find that study and post a link here for those who would be interested.

I am not suggesting the viral myocarditis is the sole cause of lone AF. Numerous other genetic and non-genetic factors might influence the development of this disaese. However, I think that viral subclinical myocarditis fairly common aetiology of idiopathic AF and for that matter other idiopathic rhythm disturbances and acquired conduction abnormalities. This begs a question, how one should deal with the issue? I highly doubt that electrolytes and anti-fibroitc enzymes could have a substantial benefit on people afflicted with a chronic viral heart infection. The cardiac inflammation, remodeling and fibrogenic processes will continue as long as the viral genome is present and active in the myocardium. Ablation might slow down the electrical remodeling of the heart, but it will not cure the underlying condition, which is more likely than not is chronic in nature.

Namor

Jackie,

"I'd use the higher dosing recommendations for serrapeptase"

And what are these?

Thanks!

George

Erling,
I've had good luck with nattokinase regarding chest pain and circulation in general. However, I don't associate that success with fibrosis, rather, I am convinced nattokinase simply allows for better blood circulation and the healing that comes with that. The chest pains I used to feel during the exercise transition from non-aerobic to aerobic states is now gone, and my hands now also have better circulation.

Tom B.

Namor - In reply to your good comments:

> *"The main complexity lies in the fact that we do not posses standardized, easy and accessible means to quantify the extent of fibrosis in people afflicted with LAF."*

Based on such studies as reported in ***Proarrhythmic potential of fibrosis*** [spo.escardio.org] one might reasonably conclude the majority of idiopathic (lone) AF is caused by fibrosis, and one should do an inexpensive and safe course of oral fibrinolytic enzymes. There's the funny play on Woody Allen's words: **There are 2 important things in arrhythmology: The first one is fibrosis, and ... I do not remember 'de second.**

> *"Interatrial voltage mapping can be used to evaluate for scar tissue, but studies demonstrate*

that it is notably absent in majority of patients with LAF."

This seems at odds with the studies and (the play on) Woody Allen's words above --- perhaps it means that voltage mapping does not reliably identify fibrosis.

> *"I've recently read publication based on small study done in Russia which took atrial tissue biopsy samples from patients with LAF and concluded that most of them had viral genome present in their myocardium. Despite the fact that patients in the study had normal MRIs, their biopsy samples revealed the presence of sub clinical cell necrosis and fibrosis on a cellular level. The authors of the study conclude that atrial myocarditis, of predominantly viral genesis, is a fairly common cause of idiopathic atrial arrhythmias. I am going to find that study and post a link here for those who would be interested."*

This is very interesting – please post the link if possible so we may read the study. Apparently there is also viral involvement in arterial plaque formation.

> *"I think that viral subclinical myocarditis is fairly common aetiology of idiopathic AF and for that matter other idiopathic rhythm disturbances and acquired conduction abnormalities. This begs a question, how one should deal with the issue? I highly doubt that electrolytes and anti-fibroitic enzymes could have a substantial benefit on people afflicted with a chronic viral heart infection. The cardiac inflammation, remodeling and fibrogenic processes will continue as long as the viral genome is present and active in the myocardium."*

Systemic viral infections can be eradicated with oral nano-particle solid colloidal silver (*not ionic* colloidal silver) See for example [www.jnanobiotechnology.com] (for overall info on "true" colloidal silver see [www.afibbers.net])

> *"Ablation might slow down the electrical remodeling of the heart, but it will not cure the underlying condition, which more likely than not is chronic in nature."*

Fully agreed. Also, if "chronic in nature" then other body cells, tissues, organs are likely affected, making atrial ablation a very partial health remedy..

Erling

Namor – Thanks for your excellent contribution. I will be very interested in reading the link when you post it.

It makes sense that addressing microbial infections that also cause inflammation (a link to arrhythmia) are an important consideration. Also extremely important is the neurodegenerative factor involved with many of these stealth microbial infections… viral or otherwise or in the case of Lyme, the spirochete, Borellia burgdorferi.

As relating to arrhythmia, Lyme disease infections have been discussed here in other posts as Erling mentions about treating with the "true colloidal silver" as a safe and effective alternative to trying various antibiotics which often don't work and cause other problems, long term.

In the news for the past year has been the growing concern about the endemic prevalence of Lyme disease throughout the US and other similar microbial infections that are going undiagnosed and causing serous problems. I'm digging out those sources and will post here as relevant to this thread. It's an important consideration to consider the Lyme disease connection to AF as well as any other type of viral infection.

Here's one to start with as it is an excellent assessment of the conundrum patients face right now on the Lyme diagnosis problem in this country.

Lyme Disease: Misdiagnosed, Underreported—and Epidemic
Feb 15, 2011
This is yet another example of the US medical-industrial complex run amok. Lyme is one of the most serious epidemics of our time. Yet the opinions of 2% of the medical community are dominating the beliefs and practices of the mass majority of practicing Lyme physicians!

The number of Lyme disease cases in the United States has doubled since 1991. The Centers for Disease Control and Prevention estimate that there are nearly 325,000 new cases each year—making Lyme disease an epidemic larger than AIDS, West Nile Virus, and Avian Flu combined. Yet, only a fraction of these cases are being treated, due to inaccurate tests and underreporting. Each year, hundreds of thousands go undiagnosed or misdiagnosed, often told that their symptoms are all in their head.

You may well ask, “If it’s such a huge epidemic, why are we not hearing anything about it?” The media is silent because doctors and insurance companies alike dismiss it as being a hypochondriacal illness, just as they’ve done for years to sufferers of fibromyalgia and chronic fatigue syndrome. It can be expensive to treat—untold numbers have it, and there is no protocol that is completely effective for all patients, no sure-fire cure. And at the root of it all, one Lyme disease organization, in its desire for power and control, is pitting doctors against doctors, prompting health insurance companies to deny medical claims at an alarming rate, and leaving suffering patients stuck in the middle.
Continue: [www.anh-usa.org]

Lastly, I agree with your assessment that Ablation might slow down the electrical remodeling of the heart, but it will not cure the underlying condition, which is more likely than not is chronic in nature.

Jackie

Past posts on Lyme and AF

Flu, Afib, Lyme Disease, ‘True’ Colloidal Silver
[www.afibbers.net]

MesoSilver: Lyme Disease Treatment Protocol and Case Studies
[www.afibbers.net]

Lyme Disease and heart irregularities
[www.afibbers.net]

George - from my archived files… these are quotes from the literature at the time at Dr. William Wong’s website. I've pulled this together rather hastily....I hope it's not confusing.

First is the Warning. As a precaution, it is recommended that those on blood thinners/anticoagulant therapy should not use these enzymes. Also not to be used with Ginkgo, aspirin, antioxidants, Omega 3 fish oil, vitamin E or garlic… anything that adds to further blood thinning… as a precaution and it’s advisable to have periodic fibrin levels checked (fibrinogen), PT and PTT times.

Two issues - My extensive stack of reports on the properties of Nattokinase as a fibrinolytic enzyme for managing blood viscosity and adverse clotting, does not go into detail about reducing fibrosis. I certainly can’t argue with Erling’s success, or mine for that matter, when we initially experimented with the Naturally Vitamins NK product. Enzymes are naturally anti-inflammatory so eliminating one of the causes of fibrosis formation (inflammation) is certainly worthwhile.

However, I have an equally extensive collection of reports on systemic or proteolytic enzymes that specifically target fibrotic tissue and various other proteins such as viral coatings and biofilms that support pathogens. Serrapeptase-based proteolytic enzymes would seem to have greater potential for fibrosis elimination or perhaps a combination of both NK-based and the proteolytic enzymes would be most effective. William Wong has his own formulation, Zymessence. Vitalzym offers a serrapeptase-based product and the Cardio version. Michael Murray has a serrapeptase product by Natural Factors Zymactive and it comes in double strength as well. There are many others. I trust these and if I were treating for cardiac fibrosis, I'd probably go for Zymessence….. just compare the ingredients.

Naturally Vitamins Nattokinase contains both nattokinase and rutin (anti-inflammatory)
2 tablets provide 1500 FUs(75 mg) NK
And 100mg Rutin (bioiflavonoid)

Enteric Coating
Clarification on the need for enteric coating. Serrapeptase-based products must always indicate enteric coating either incorporated into the manufactured powder or as an enteric coated tablet/capsule Check the labels. Dr. Wong's latest formulation (Zymessence) uses the latest technology for ensuring that the entire product is enteric-protected. [www.zymessence.com]

Nattokinase enzymes are typically manufactured by culturing enzymes to be heat- and acid-resistant so the enteric coating caveat is not typically specified or labeled as such.

Dosing recommendations:
My files indicated the following from one of Dr. Wong's web pages at the time. His new product directions are listed at the Zymessence link and are different than the original.

He states that an adequate dosage in one enteric coated tablet would be:
Pancreatin (8X) 200 mg.
Papain (30,000USP/mg 120 mg
Peptizyme SP (200,000 SPU/g) 52 mg of Serrapeptase – silk worm enzyme
Bromelain (1.200 MCU/g) 50 mg.

Typical dosage of this formula on an empty stomach when non-digestive effects are desired.

Vitalzym Dosing
(this is from Dr. Wong's original instructions and before he developed his own version.)

For people with Fibrosis and inflammation symptoms need to find the dose at which they begin to feel better. They can then maintain that dose or after 3 months, try to reduce the dose and see if the benefits are still felt.

Start by taking 3 capsules, 3 times a day between meals. If in 3 days no benefits are felt, increase the dose to 4 capsules, 3 times a day for 3 days. Continue increasing the number of capsules taken until a benefit is felt. Most will feel the Vitalzym kick in somewhere between 3 and 5, 3 times a day.

There is no Toxicity from enzymes (no LD 50). Some customers who have suffered accidents have been placed on 10 Vitalzym capsules 3 times a day by their doctors for a few weeks until the bulk of the inflammation and resulting pain is gone. Then they lowered the dose for maintenance to prevent the formation of scar tissue.

Although he doesn't specifically call out cardiac fibrosis, note the Organ Fibrosis directions… When we were corresponding, he was also helping a woman eliminate uterine fibroids and recommended

3 to 5 capsules of Vitalzym 3 times a day which worked for her.

Chronic Fatigue/Fibromyalgia
3 capsules, 3 or 4 times a day
5 capsules, 2 times a day for 2 months; then to prevent recurrence – 3 capsules 2 times a day.

Traumatic Inflammation
Severe: 5 capsules, 3 times a day
Moderate 3 capsules, 3 times a day
Mild: 3 capsules, 2 times a day.

Post-Surgical Recovery.
3 capsules, 3 times a day.

Note: The FDA has not evaluated the above information. This product or the listed information is not intended to diagnose, treat, cure or prevent disease. For all conditions or illnesses, see a health professional for a full evaluation, diagnosis and treatment plan.

See the update at this link with his own Zymessence formulation [www.totalityofbeing.com]

While this link to an article by Michael Murray on The Healing Properties of Proteolytic Enzymes is for cancer treatment, the enzyme info is worth noting. [doctormurray.com]

I hope this is useful, George. If I can dig out more, let me know.

Best to you,
Jackie

Healing and Rejuvenation with Enzyme Therapy [www.sunherb.com]
Terri L. Saunders
Certified Natural Health Professional

Quote:

"Dr. Wong tells the remarkable story of his wife who had an emergency C-section with the delivery of their son. As is common after abdominal surgery, a tremendous amount of scar tissue formed throughout her abdominal cavity, threatening to strangle her intestines. He knew that more surgery would just perpetuate the problem. Dr. Wong gave his wife serrapeptase to attempt to reduce the scarring, however it wasn't until he discovered Vitalzym, a unique vegetarian blend of systemic enzymes including serrapeptase, proteolytic enzymes and other enzymes and nutrients, that his wife got results. Within eight weeks after taking Vitalzym, all of the scar tissue had dissolved and surgery was no longer needed. A more potent, enhanced therapeutic formula called VitalzymX has since been produced and is available from conventional and holistic health professionals. VitalzymX is also an effective anti-inflammatory and immune system modulator and is rejuvenating to the entire body. I have experienced the extraordinary healing power of this enzyme formula both personally and with clients, and highly recommend it."

Terri Saunders is an Herbalist, Nutritionist, and Certified Natural Health Professional in Charlottesville, Virginia where she does consultations, telephone consultations and teaches classes on natural healing. She can be reached at 434-984-2665 or email at sunherb@mindspring.com. Check website at [www.sunriseherbshop.com] for more information on products and consultations.

Erling

Jackie,

Thank you for posting the FDA disclaimer and the contraindications. Erling should take a lesson, it would add a little value to his Infomercial.

In an earlier post, Erling notes the low cost of these enzymes. On iHerb, a bottle of 450 caps of Vitalzym is $114.00. For Severe Traumatic Inflamation, the dosage is 15 caps per day, so the 450 bottle is a 1 month supply.

I am thinking that many readers of this forum would not consider $114.00 per month for one supplement to be a minor cost. We probably have a few one- percenters, but not many.

Many of these folks are on fixed incomes, and they should buy this false hope? Caveat emptor.

EB

EB - compared to the cost of an ablation and/or even a second procedure, the cost of systemic enzymes is really quite small and certainly worth trying. Further, if the systemic/proteolytic enzymes eliminate fibrosis and prevent MI, then that seems like a trivial expense compared to those consequences. [www.theheart.org]

If you were to survey those afibbers from CR 61 who have reversed their afib as a result of lifestyle and dietary changes plus adding supplements, I'm sure they'd confirm the cost was worth it. I have never regretted any of the considerable dollars I spend on supplements that have reversed many of my other health issues after spending...No, actually wasting.... $$ on office visits and Rx drugs.

It's always good to know about options and based on my own negative medical experiences, I'm happy to be sharing potential aids that progressive practitioners offer as alternatives to drugs or invasive surgical procedures that may have adverse results in addition to expense.

People are responsible for their own health and the choices they make should be educated choices. I am an advocate of examining all the potentials with an open mind because I have been led down the wrong path in the past too many times in the past. As a result, I've become a Health Awareness Advocate to help direct those who are interested in learning about healthy options.

Systemic enzymes do work.

Jackie

Jackie,

Your distrust of the traditional medical community is obvious from your posts. We could probably debate all day about homeopathic remedies vs generally accepted medical practice, or we could just bang our heads against the wall. It has been my experience that some homeopathic remedies have positive results and some do nothing but increase the bank account of the guy that sold it. It would be very interesting information to know how many people have tried these enzymes and the percentage who benefitted. If it was a high percentage, I am sure you and Erling would be waving that flag; the fact is, you don't know and in all probability it is a low percentage.

Regarding cost, it is important to remember that supplements are rarely, if ever, covered by insurance and if they cure you, then indeed they would allow you to avoid the copayment part of an ablation/ surgical option. The problem is, they are unlikely to cure you. While we are on that

subject, you yourself had an ablation; how is it that these enzymes didn't prevent the need for that? As I recall, you had a recent break through; have you stopped taking the enzymes?

As I said in an earlier post, it seems that you don't respect people who resort to surgical options, be it ablation or five box mini-maze. You have a complex about traditional medical practice and it colors your advocacy of alternate medicine. We do agree on one thing; people are responsible for their own health and should make educated choices. I expect you will continue to post information, some of it good but some very questionable (at least you don't post pure Infomercials like Erling). I will continue the rebuttal; I am just calling a spade, a spade.

It has been almost a year since the untimely death of Steve Jobs, the Apple computer founder. When Jobs discovered that he had pancreatic cancer, his first reaction was to eschew traditional treatment and try various dietary and alternative treatments; after about nine months he turned back to the traditional medical community but his delay had allowed the cancer to spread. There is no question that this tragic decision shortened his life. Read the entire article here: [www.forbes.com]

Systemic enzymes might work, but if something sounds too good to be true, then it probably isn't.

EB

EB - I don't know where you get the idea that I'm recommending or use homeopathic remedies. I never used that term in this thread. It doesn't even fit in with what I've offered. No relevance.

I'm talking about Functional Medicine or Systemic Medicine analysis and then treatment to restore organ systems back to optimal function. These fields of advanced medicine are well accepted and are saving lives (and money) every day. Those of us who are fortunate enough to understand and use them, are the beneficiaries of true 'health' care.

Continue if you like to disparage but the facts are out there as evidenced by the many organizations that support this advanced type of nutritional intervention. Even the Cleveland Clinic opened up a whole new "Integrative Medicine" department just to keep up.

The tragic story of Steve Jobs hardly compares with this topic. I've followed it and it makes me very sad.

Generalities can be misleading. Specifics as recommended here in various posts and in the Vitamin Shop nutritional recommendations for health support are well known to be effective and without adverse effects. It takes time to become conversant with the field of nutritional healing.

Having had abundant personal successes, I'm committed to helping other avoid medical pitfalls that are out there for the unsuspecting.

Jackie

Oh - PS... my breakthrough was a couple years ago... I did not stop taking enzymes, but became a bit complacent about optimizing magnesium and potassium. At my age, my FM MD suspects I may be experiencing a bit of kidney dysfunction that allows for slight wasting so I need to always be diligent with my intake from food and supplement sources.

Part 5 – Cardiac Fibrosis and AF

Consider this a prelude to the coming update on CR 24 – Cardiac Fibrosis
Following are a few key points from my ongoing observations found in the literature. .
Specific details will be included in the final report.

Jackie

Consideration should be given to the use of systemic enzymes to eliminate fibrosis as it is a valid and effective treatment.

In CR 24 on cardiac fibrosis, reference was made to the explanation of the fibrosis connection as a substrate for atrial fibrillation by David Van Wagoner, PhD, researcher in Molecular Cardiology at the Cleveland Clinic.

Fibrosis is a long-term response to injury, oxidative stress damage, inflammation and other influences as well. There is abundant support in the literature linking cardiac fibrosis and atrial fibrillation since fibrosis interferes with proper cardiac electrical conduction. A report as recent as June 2011, suggests that "targeting cardiac fibrosis has potential as a new frontier in anti-arrhythmic therapy"… indicating that cardiac fibrosis management is a logical consideration. Yet, instead of developing drugs for this purpose, a safe, natural remedy already exists. Systemic enzymes.

The recommendation to reduce various types of fibrosis with systemic or proteolytic enzymes is not new. Practitioners of functional, regenerative or restorative and anti-aging medicine have been using these various enzyme combinations for many years safely and with great success; most importantly, without resorting to invasive surgical procedures.

When I researched the use of the fibrinolytic enzyme, nattokinase, I spent considerable time learning about managing fibrinogen as it relates to blood viscosity. Also learning about systemic enzymes was unavoidable as often they are combined in treatment plans. Audio and video presentations explaining various applications to eliminate fibrosis by Dr.Wiliam Wong and Dr. Garry Gordon added to the material supplied by enzyme manufacturers along personal interviews with marketers of the Wobenzyme and Vitalzyme people. In one audio, Dr. Wong tells of his wife who had abdominal adhesions from surgery that began to contract so severely she was unable to stand completely upright. To avoid another surgery to remove the fibrotic scar tissue, he used the enzymes which allowed her to regain her full stature and functionality. He tells of many other patients who were severely impaired by various forms of fibrosis and who were restored to health with systemic enzymes.

I felt comfortable following using the guidance of Dr. Wong. As covered in CR 24, and as it turned out, my choice of initially using Vitalzym for almost a year prior to my ablation, most likely helped improve my ablation success, although I was primarily concerned with blood viscosity issues and less aware of the fibrosis factor at the time. In hindsight, the enzymes along with my other heroics undoubtedly were very instrumental in enabling me to progress from having afib every day or every other day, often for 24 – 27 hours, down to zero events for several months prior to ablation.

Wobenzyme was introduced in 1960 in Europe and eventually, reached US practitioners. Wobenzyme Mucos Pharma of Germany provided some of the original data for my initial research as endorsed by Dr. Gordon. Wobenzym N is proven effective by over 160 clinical studies. [www.mucos.cz] For many years, Dr. Gordon (now in his mid 70's) has successfully incorporated systemic enzymes into his own protocols and that of his patients.

Dr. Wong worked with World Nutrition (Vitalzyme) for a number of years and then formulated his own version of what he considers to be a highly effective product based on his hands-on treatment experience.

Dr. William Wong's credentials:
Texas Complimentary and Alternative Medical Association professional member and World Sports Medicine Hall of Fame member, Dr. William Wong is a Classical Naturopath, a PhD. Exercise Physiologist, Certified Athletic Trainer (AATA), Certified Sports Medicine Trainer (ASMA), Old Rite Catholic Priest and Health/Fitness Consultant.

Dr. Wong has more than 27 years of professional experience in natural health as applied to sports medicine and rehabilitation. The last 8 years of which have been devoted to the application and teaching of Systemic Enzyme Therapy (SET). Studying SET techniques in both the US and Germany, Dr. Wong is widely acknowledged as one of the foremost experts in the field. In the 1990's, he taught Physical Medicine at the South West College of Naturopathic Medicine. In 1993, Dr. Wong was inducted into the Martial Arts Hall of Fame as Wing Chun Kung Fu Instructor of the Year and in 2004, after 37 years in the Martial Arts, he achieved the rank of Grand Master from the Soke (Grand Master's) Council and was awarded a Doctor of Philosophy in Martial Arts.

Dr. Wong has authored books on natural healing and on sports medicine. His shorter writings have appeared in such diverse magazines as G.Q., Black Belt, Survival Guide, The Townsend Letter for Doctors, Well Being Journal, BeautyWalk.com e-zine, and Healthy Options magazine in New Zealand. He has been a guest on over 1000 national and local radio programs, and has appeared on the nationally acclaimed PBS series Healthy Living hosted by Jane Seymour and Heartbeat of America hosted by William Shatner.

Continuing to lecture across the USA, Dr. Wong traveled to India in the Fall of 2005, where he conducted seven different lectures before hundreds of physicians teaching OBGYN's, Plactic Surgeons, Thoracic and General Surgeons, Cardiologists, General Practicioners and Dentists on how to integrate Systemic Enzyme use into Orthodox Medicine.

Currently, Dr. Wong writes books and lectures on anti-aging and pro-sexual topics. He also consults with individuals, specializing in the development of personalized programs for longevity and virility to help people overcome the effects of aging and the after effects of chronic debilitating conditions.

Dr. Gordon is a legend in his own right. One can get lost for days in his collection of audios and YouTube presentations.
This article on cardiovascular use of enzymes is one of many.
[gordonresearch.com]

Garry F. Gordon, MD, DO, MD(H)
President, Gordon Research Institute
www.gordonresearch.com

Dr. Gordon is an internationally recognized expert on chelation therapy and antiaging medicine. He is also a consultant for various supplement companies and the coauthor of The Chelation Answer. He lectures extensively on the topic, The End of Bypass Surgery is in Sight. He is on the board of the Homeopathic Medical Examiners for Arizona, is cofounder of ACAM (American College for Advancement in Medicine) and a board member of the International Oxidative Medicine Association. He received is Doctor of Osteopathy in 1958 from the Chicago College of Osteopathy in Illinois and completed his radiology residency at Mt Zion in San Francisco in 1964. He was the medical director of Mineral Lab in Hayward, CA, a leading laboratory for trace mineral analysis worldwide. He does telephone consultations for patients from around the world offering second opinions on any type of health issue from his offices in Arizona. Dr. Gordon is dedicated and passionate about educating doctors and patients about the harmful and devastating effects of environmental pollution and he provides documented alternatives for any health condition. He wants everyone to feel as good as he does at age seventy-six, having restored himself to optimal health in spite of suffering from serious illnesses for most of the first thirty years of his life

including genetic heart disease.

About Vitalzym:

Systemic Enzymes Help You Feel Better, Look Better, and Live Longer
Vitalzym contains potent proteolytic enzymes designed to support health and promote healing and repair. It is an extremely effective systemic enzyme blend with a high Serrapeptase content.

Vitalzym works to break down fibrin in the body. Fibrin is a hard, sticky protein that has been associated with scar tissue, inflammation and pain, among other symptoms and conditions. Additionally, Vitalzym can help reduce viral load and regulate the immune system, reduce toxins and impurities in the blood, promote cellular detoxification, reduce internal inflammation, and promote overall better health.

Enzymes are said to be the “sparks of life.” They are considered keys that can unlock the door to a healthier you because they not only help improve digestion and nutrient absorption; they are also responsible for millions of bodily functions.

Vitalzym works synergistically to provide total system support. It contains protease, serrapeptase, papain, bromelain, amylase, lipase, rutin and amla. According to Dr. Peter Streichhan, a world-renowned enzyme researcher from Germany, "enzyme mixtures have a wider range of therapeutic advantages than do individual enzymes." 1
Continue: [www.energeticnutrition.com]

My thoughts are:
In light of the role cardiac fibrosis plays in causing and sustaining arrhythmia, for those afibbers who have supplemented and tested for electrolytes, methylation issues, kidney, adrenal, thyroid, pH etc involvement and still seem unable to achieve NSR, treating with the systemic enzymes seems an important inclusion. Emphasis on using systemic enzymes has not been emphasized regularly or consistently but should be a definite consideration. This will be covered in detail in the fibrosis update report.

As mentioned in another thread, the research by Dr. Shivakumar indicates that fibrosis formation is rooted in magnesium deficiency. Those afibbers who are refractory to magnesium repletion (who seem unable to achieve optimal or sustained intracellular magnesium levels in spite of heroics) are likely caught in the MgD/fibrosis-generation web. Eliminating the fibrosis may enable magnesium repletion and/or help facilitate proper conduction without fibrosis interference.
Read more: [www.afibbers.net]

Conference Room 24 - Cardiac Fibrotic Remodelling [www.afibbers.org]

Thank you, Jackie, for continuing and enlarging upon this hugely important topic, left incomplete 8 years ago.

For clear, visual understanding of cardiac fibrosis / AF, here is an informative "slide-show" with attribution to various authors / journal articles. [spo.escardio.org] (might be slow to load.)

Proarrhythmic potential of fibrosis
Clinic Barcelona Hospital Universitari
Disclosure of conflicts of interest:
Research funds and consulting for St. Jude, Boston, Medtronic, Biosense, Biotronic.

A few quotes:

There are 2 important things in arrhythmology:
The first one is fibrosis, and I do not remember 'de second.
Modified from Woody Allen

What do we call fibrosis?
Excessive collagen deposition.

The collagen network of the normal heart:
-- provides support.
-- maintains myocardial structure.
-- sustains the transmission of force.

Etiology of fibrosis:
-- Reactive fibrosis:
Secondary to overload, stretching. (No myocyte necrosis? Reversability?)
-- Reparative fibrosis
Induced by myocyte necrosis, apoptosis (no reverseability)

Etiology of fibrosis:
-- Multiple etiologies create different substrates and produce different pictures at the myocardium, and may co-exist in the same heart.
-- Permanent arrhythmia creates fibrosis by itself, as in permanent AF.

Collagen fibers in AF
[page 12, descriptive pictures comparing normal collagen (NSR) and fibrosis (AF)]

Arrhythmogenicity of fibrosis
1. Slowing of conduction velocity.
2. Dispersion of refractoryness.
3. Decrease in safety factor for conduction. Current to load mismatch.
4. Unexcitable scars and barriers for conduction.
5. Triggered activity.

Erling

Hello Jackie and Erling,

Thank you for bringing the issue of the role of fibrosis in atrial fibrillation to the fore again. Although the subject was thoroughly discussed in Conference Room session 24 in February 2004 [www.afibbers.org] I certainly agree that additional research reported in the last eight years ought to be included in the proceedings of session 24. Once this discussion is finished I will do so.

I have a few comments:

While in no way belittling the trail-blazing work of Dr. Van Wagoner and his colleagues at the Cleveland Clinic I believe that credit should be given where credit is due. As far as I know Dr. Andrea Frustaci at the Catholic University in Rome was the first to point out the connection between **lone** atrial fibrillation and fibrosis. His findings are discussed in my first book *Lone Atrial Fibrillation: Towards a Cure* (pages 130-134). Here is an excerpt:

Inflammation and LAF
Could an inflammation be involved in lone atrial fibrillation (LAF)? Indeed it could. In 1997 Dr. Andrea Frustaci, MD and colleagues at the Catholic University of Rome made a fascinating discovery. They performed biopsies of the right atrium in 12 patients with LAF and found that 8 (67%) of them had evidence of a current or past inflammation in the heart tissue (myocarditis).

They also checked 11 control subjects and found that none of their biopsy samples showed any signs of inflammation. The Italian researchers conclude that inflammation and its aftermath (fibrotic tissue) is a likely cause of LAF [www.ncbi.nlm.nih.gov].

I think it is very important to always clearly distinguish between common AF (atrial fibrillation with underlying heart disease) and lone AF (atrial fibrillation without underlying heart disease). Unfortunately, most research papers do not make this distinction even though the article by Patrick Chambers, MD (PC) *Lone Atrial Fibrillation: Pathologic or not?* [www.ncbi.nlm.nih.gov] makes it quite clear that the two disorders have very different etiologies indeed. Nevertheless, it would seem that the two forms of afib do indeed share a connection with inflammation and fibrosis. Inflammation in turn, has been associated with mental and physical stress, vigorous exercise, alcohol consumption, mercury poisoning, bacteria, viral, and fungal infections and oxidative stress.

In a personal communication with Dr. Frustaci (2001) he pointed out that, in his opinion, individual heart cells, which have been exposed to inflammation, can revert to normal cell structure – provided that the DNA of the cell has not been damaged beyond repair. This is indeed encouraging as it may mean that LAF could be permanently eliminated if the inflammation and fibrosis are vanquished – assuming of course that inflammation is the underlying cause.

I found the Barcelona slide show most interesting. Particularly the part that discusses the effect on fibrosis of detraining rats that had been exposed to vigorous, sustained exercise. Professor Philippe Coumel also proposed detraining as an effective approach to managing afib in endurance athletes [www.ncbi.nlm.nih.gov].

In conclusion, I agree that elimination of inflammation and fibrosis could be an important step in the management of LAF and look forward to conclusive evidence that therapy with systemic enzymes is effective in doing so. It may also be worth considering the possibility that a combined regimen of systemic enzymes and natural anti-inflammatories may do a better job than systemic enzymes on their own.

Hans

Here is a nice anecdotal testimonial for fibrinolytic enzyme therapy. Change the words "uterine fibroids" to "atrial fibrosis" and "hysterectomy" to "ablation" and it gets interesting: [www.energeticnutrition.com]

My Uterine Fibroid Tumor Story
Using Natural Supplements to Shrink my Fibroids
By Lorraine McGorry
Owner, Energetic Nutrition
August 25, 2004
==========

Full disclosure ('tis the season):
The undersigned has no affiliation with Lorraine McGorry, Energetic Nutrition, Wobenzyme, or with anything that earns $$ for anyone, including me.

Erling

As I mentioned, initially, about a year prior to ablation, I did use Vitalzym which contains Bromelain, Papain, Rutin, Amylase, Protease, Lipase, Serrapeptase, Alma Extract and I combined that with Nattokinase at the recommended preventive dosing (6,000 FUs daily in divided doses).

Post-ablation, I continued on with just the Nattokinase since, at that time, I was more concerned with managing blood viscosity and lowering the risk of adverse clotting. The ablation was in 2003 and I have continued with daily Nattokinase and added combo NK/Serrapeptase product. The digestive enzyme I use contains many of the other Vitalzyme components and I've taken digestive enzymes for years as well.

Initially, about 12 years ago, I used Zyflamend by New Chapter as it is an excellent formulation for inflammation although I prefer a higher daily intake of the curcumin component and a few other herbals.

Later, I found the product I use today (in addition to the NK and Serrazyme) called Inflammatone ™ which is a combination of herbs, nutrients and proteolytic enzymes that modulate the inflammatory response and herbals that protect from free radical damage. This product breaks down inflammatory proteins (kinins and fibrin) and helps heal damaged tissue.

Of significance is the fact that my HS-CRP levels remain below 1.0 so I know my systemic and cardiac inflammation levels are under control. I also attribute some of that success to the higher dosing of vitamin D3 that I've been using for the past five years. Additionally, I've taken high dose Omega 3 essential fatty acids since the year 2000 - also known to be anti-inflammatory.

Lowering the inflammatory response is an important step in eliminating or reducing significantly, the fibrotic response.

Jackie

Thanks, Hans for your comments. Finding the source of any and all influences that may be instrumental in causing and sustaining LAF is definitely an important goal. Obviously, Van Wagoner is not the only resource for examining the influence of fibrosis. Even more concerning is a lack of recommendations for safe and natural remedies to control inflammation other than using statin drugs. My personal experiences with non-cardiac fibroses are many and as a result of my travails and research, I've used numerous combinations of various natural remedies including enzymes and natural anti-inflammatories and found them to be highly effective.

It may take some time to organize and condense this update and there there is much current information worth reporting.

Your comments on fibrosis as a result of overexercising/overtraining and detraining definitely show up repeatedly in the literature.

Jackie

Thanks, Erling for these important contributions.

Years ago, I had uterine fibroids, fibrocystic breast disease and fibromyalgia. My regret is that I didn't have access to this type of information back then..... if it was even known.

At least going forward, we can direct afibbers to the literature which will enable them to become aware of the consequences of cardiac fibrosis and the other related manifestations.

I am truly appreciative of the hands-on experiences Dr. Wong shares as a result of his focus on fibrosis.

Jackie

The article which the author said that she was able to shrink her Ovary tumor with Vitalzym, would that apply to all tumors? She said her tumor was composed of fibrin, tumors are composed of cells and blood, so are there differences in composition of tumors.

My brother has tumors that have metastasized to his spine, he has a large tumor which has rendered him paralyzed from his waist down, he is getting radiation to try and shrink the tumor, but there isn't much hope. So, I guess you know where I am going with my question, would Vitalzym do anything for his tumor?

Liz

Jackie - Would your research and conclusions on fibrosis (which I must read several more times to comprehend) translate to other fibrotic diseases such as Pulmonary Fibrosis?

Stevie

Hi Liz- – I am so very sorry to read about your brother. He's been through so much. Very sad and certainly a huge stress burden for you.

I did a Google search on Vitalzym and cancerous tumors. A number of hits surfaced which tend to indicate that it could be worth trying. Here's a link to an article on "Enzymes and Cancer" by Dr. Wong. At the end of that piece, there are several references that may provide useful information. [www.globallight.net]

Taking it to the next level, though, there are references to systemic enzymes for cancer treatment in the book, *Outsmart Your Cancer*, *Alternative Non-Toxic Treatments that Work* (Tanya Harter Pierce). The chapter about cancer-treatment pioneer, William Donald Kelley DDS who is legendary, reviews the work of New York physician, Nicholas Gonzalez who followed Dr. Kelly's research and has become well known for helping those with metastasized cancer and who are thriving 10 – 15 years after their diagnosis. The typical treatment includes a protocol that uses specifically-developed high-dose pancreatic enzymes.

Dr. Mercola has online a 2011 report on Dr. Gonzalez … in the summary statement he offers:

For more information about Dr. Gonzalez and his practice, see www.dr-gonzalez.com. He's also working on a series of books, two of which have already been published and received five-star reviews: The Trophoblast and the Origins of Cancer, and One Man Alone: An Investigation of Nutrition, Cancer, and William Donald Kelley , which is the original monograph of Dr. Kelley's work that he couldn't get published 23 years ago.

This written summary is only a small glimpse of the insights that were shared in our interview. If you or anyone you know struggles with cancer I would strongly encourage you to listen to the entire interview.

Thankfully Dr. Gonzalez is still on the front lines and actively engaged in helping people by helping coach them with natural alternatives to toxic drugs and radiation. His office is in Manhattan and he can be reached at 212-213-3337.
[articles.mercola.com]

Take care.

Jackie

Stevie - yes for the pulmonary fibrosis. Enzymes lyse or dissolve the fibrous tissue accumulation.

Start with this article and continue with a Google for the words, "systemic enzymes pulmonary fibrosis."
[www.totalityofbeing.com]

If you need help in deciphering, send me an email and I'll try.

Keep in mind that the emphasis here is the source or cause of the fibrosis... it's a response to something... such as inflammation, irritation, etc. but key to anyone with fibrosis and especially afibbers is the link to the fundamental causative which is magnesium deficiency.

When the body is magnesium deficient, then various pathologies can manifest and it obviously can be one adverse condition for some, such as asthma and migraines, or cardiac or pulmonary fibrosis, for others... cardiac leading to arrhythmias in some cases (and obviously, much more) because critical functional mechanisms are not able to work efficiently or completely. Details in the new report.

As Erling has continually reminded us, magnesium researcher, Mildred S. Seelig, MD, MPH, compiled her original 1980 book *Magnesium Deficiency in the Pathogenesis of Disease* from scientific literature dating back to the 30's. The problem has been and currently is, that in the rush to "cure" various ailments, including Afib, mainstream medicine seldom considers a nutritional deficiency as a core cause and prescribed drugs don't typically replete the deficiency.

Jackie

Hans/Jackie,

As I posted a short time back, in my surgical report following my Five Box procedure last January, Dr. Sirak noted myocardial fibrosis in my left atrium, which necessitated multiple ablation passes to achieve electrical isolation. Dr. Sirak told me after the surgery that he see fibrosis in some patients, not all, but in particular it was not usual to see it in runners / people who have done lots of cardio exercise in their lifetimes.

So the connection between AF and fibrosis is fact (and Han's point about LAF being different is certainly arguable). The question is, what do you do about it? Hans notes a study about detraining rats; Jackie promotes enzymes that dissolve everything from fibroid tumors, spine cancer, and pulmonary fibrosis (plus of course myocardial fibrosis).

This is where I tend to question the validity of some that is written here. I can Google pulmonary fibrosis and find a study by the National Institute of Health that states that there is no cure for Pulmonary Fibrosis (if anyone doesn't believe me, please Google away). I Googled myocardial fibrosis treatment and found lots of references to drugs like digoxin and surgery, but no references to magical enzymes.

If anyone could prove that a particular therapy would cure Pulmonary Fibrosis, Fibrous cancer, and myocardial fibrosis, I am quite sure that the Nobel Prize for Health would be one of many awards that would follow.

It is possible that these enzymes might help some of these maladies; but what percentage and how much, we don't know. Compared to placebo? We don't know that either.

You should tell your readers that fact.

EB

EB - Thanks for shooting the messenger! And thanks as well for opening up another opportunity to help direct interested readers to various reliable sources that discuss success in treating patients with systemic enzymes. The fibrosis successes by Dr. Wong are all worth reading. The history of suppressing this type of treatment is extensive but fortunately the topic is still alive and well and the truth is…it does work!

Also, welcome to the world of disparity between natural therapies that work and conventional medicine based on drugs and surgery that doesn't address the core causes but rather just mask symptoms in many cases. You probably weren't reading when I posted my various experiences being treated by conventional drugs and surgery only to learn later that not only did some not work, but that I could have avoided the surgery in the first place. Additional and relatively minor conditions I treated for with various expensive Rx drugs and office visits that were not resolved, were reversed by nutritional interventions. That's fodder for another thread on another forum.

The successes of the doctors treating with enzyme therapy should not be ignored as they are valid. However, because of the relative difference in cost for treatment (read income for the medical establishment and Big Pharma), they continually are disparaged. Yes, you can Google to your heart's content to find pros and cons on just about any issue you care to refute. Knowing who is enjoying success becomes important and that's what I briefly addressed.

Thank you for reporting that Dr. Sirak found fibrosis during your procedure. It confirms once again the evidence that will be reviewed in the new Cardiac Fibrosis examination as cardiac fibrosis relates to oxidative stress damage, inflammation, cellular malfunction and for some people, atrial fibrillation... and of course…. the core issue: Ta da….. magnesium deficiency! The upcoming new Cardiac Fibrosis report will be based on the scientific literature.

The fact that your EP did detect fibrosis is very telling and if I were in your shoes, I'd be taking systemic enzymes and plenty of magnesium to be sure that the fibrosis was eliminated and would not cause problems in the ventricles.

Here's a comment by Dr. Gonzales the physician treating with enzymes that I mentioned in the post to Liz…

(quote) Dr. John Beard, the brilliant English scientist, first proposed in 1902 that pancreatic enzymes represent the body's main defense against cancer and would be useful as a cancer treatment. At the time, physiologists had already identified the many classes of pancreatic enzymes, which they acknowledged served as a major digestive function, breaking down complex proteins, fats and carbohydrates into simpler, easily absorbable molecules.

But Beard claimed that above and beyond this digestive activity, the protein-digesting enzymes kill cancer cells. Subsequently, in both laboratory and human tests, he proved his point, reporting the successes of his treatments in the mainstream medical literature. He published his wonderful book, *The Enzyme Treatment of Cancer*, in 1911 but by the time he died in 1924, his work was forgotten. Fortunately, my mentor, Dr. Kelley, revived Beard's thesis in the 1960's with great success in his practice treating patients with advanced cancer. We've been employing high-dose enzyme therapy since we opened our practice in 2987, again with gratifying success. (end quote) pp270-271

There is much more to read about enzyme treatment in his chapter in the book *Bombshell* by Suzanne Somers which is about anti-aging nutraceuticals but includes many nuggets that relate to her cancer story and other cancers as well.

I won't get into the Garry Gordon references as that would just tie up way too much time and space. He's used enzymes successfully on himself and patients for years.

Best to you.
Jackie

EB,

In regard to your comments about detraining of rats being associated with a decrease in cardiac fibrosis. Detraining also works for vagal afibbers who continue strenuous exercise despite the fact that such exercise probably is the cause of their afib in the first place. A quote from an article by Professor Philippe Coumel - one of the world's foremost authorities on cardiac arrhythmias prior to his untimely death in 2004:

To conclude on the influence of sport, it is known that in well-trained people suffering from vagal AF, the first step of therapy should be deconditioning by discontinuing high-level training. It may be sufficient to bring about an improvement in the patient and is often a necessary adjuvant to facilitate pharmacological therapy [eurheartj.oxfordjournals.org].

Hans

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