



THE STRATEGY WHAT METABOLIC CARDIOLOGY MEANS TO AFIBBERS

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ACKNOWLEDGEMENTS

The fundamentals of basic cellular biochemistry and physiology of energy production relative to metabolic cardiology provide the links to identifying and understanding the initiators of atrial fibrillation. Several key resource books provide the facts:

The Sinatra Solution: Metabolic Cardiology

Integrative Cardiologists Stephen T. Sinatra MD, FACC, CNS, CBT
James C. Roberts MD, FACC

The High Blood Pressure Solution with The K Factor

Richard D Moore, MD, PhD biophysics

The Magnesium Factor

Magnesium researchers, Mildred S. Seelig MD. MPH.
Andrea Rosanoff, PhD.

Lone Atrial Fibrillation – Toward a Cure

Hans R. Larsen, MSc ChE
The Foreword by Prof. Philippe Coumel .
Preface by Patrick Chambers, MD

If you deal with afib, these books are requisite reading.

References from books, research papers and articles provide important support. Using these resources to connect the dots is our former afibber, Erling M. Waller, one of the first to cure his afib after a 10-year journey and to share his success story with us years ago. With Erling's coaching, I have attempted to condense an extensive amount of information into a long, but hopefully understandable and useful reference and guide that will serve to assist afibbers for many years to come.

Most importantly, because of the website (afibbers.org) made possible by Hans Larsen, MSc, ChE (former afibber), we have repeatedly discussed all of the issues involved through an abundance of remarkable contributions including Hans' countless reports and book series. At one point, former afibber, Patrick Chambers, MD, devoted an extraordinary amount of time to enlighten us almost from the beginning, and many of his contributions live on in the Conference Room Proceedings. Countless other dedicated and respected contributors helped with an enormous amount of input. The goal, of course, has always been finding a way to sustain NSR.

INTRODUCTION

For clarification, this report addresses specifically Lone Atrial Fibrillation (LAF).

Numerous influences contribute to the onset of lone atrial fibrillation. One underlying factor definitely supports either normal sinus rhythm (NSR) or arrhythmia. Confirmed in published scientific observations, this factor is the ability to maintain a specific state or condition of heart tissue (cells) that sustains or supports a prolonged refractory period (where the heart is unable to be stimulated into arrhythmia).

Many former afibbers(1) have managed to accomplish that goal through an optimal state of what I'm calling "tissue compliance" brought about by dietary and lifestyle modifications which have enabled them to be relatively afib free. In many studies, the absence of this tissue compliance is termed "pro-arrhythmic substrate." The substrate designation is sometimes ambiguous, so for simplicity, I like to think of it as compliant tissue...meaning it meets all requirements to maintain a heart in NSR.

If the optimal tissue compliance state becomes unbalanced, afib re-appears; but for the most part, these former afibbers are maintaining NSR without drugs or ablation procedures. Reference (1) is a partial accumulation of testimonials/success stories describing their methods. As time goes on, we've had many more join the ranks of "The List" once they achieve the compliant-tissue state. It should be noted that this doesn't work automatically or universally for every LAFer; although it has and does, frequently. Many influencing factors can interfere with reaching the goal.

Ablation blocks the errant signals without correcting the defective substrate or myocardial tissue of the atria. The refractory period issue remains. This relates directly to the compliant tissue discussion. For those on The List who did succeed, it is because they lengthened the effective refractory period (ERP). This is a dynamic tissue condition or status and one that can change to pro-arrhythmic when compliance becomes unbalanced. Virtually everyone on The List as well as many of us who are successfully ablated can have AF breakthrough when tissue compliance is not optimal. When ATP energy produced in the mitochondria is reduced, thereby shortening the ERP, afib can occur.

In my ablated heart, if I fail to consistently optimize my own specific requirements for cellular nutrition, then I'll have afib events because that requisite state of tissue compliance is breached or unbalanced.

While it's important to determine whether your LAF is adrenergic or vagally mediated, tissue compliance applies to all hearts and is first line defense for maintaining NSR.

Hopefully, this report will serve to help clarify the complexity of the magnesium/potassium/taurine/coQ10/carnitine/and ribose/Omega 3 supplementation protocol which is much more than about one specific nutrient. Rather, it's about synergistic effects, cellular function, energy production and metabolic cardiology. Metabolic cardiology involves numerous components and synergy. Cellular functions are dependent upon synergy and that effect can exponentially increase the results or benefits.

Synergy: The interaction of two or more agents or forces so that their combined effect is greater than the sum of their individual effects.

And, it's complicated because it's deeply rooted in biochemical reactions and cellular physiology. This summary report barely skims the surface. I've tried to make the technical details understandable by interpretation, and I have included the science as backup. Some reiterations may seem redundant; however, they serve to emphasize important facts that need to be understood.

Quotes and/or paraphrases from well-known highly-credentialed and respected experts are included. A reference section included at the end lists links for further research along with other suggested reading resources.

There is a section for each essential component so, unfortunately, this is a lengthy read. One of the final sections covers dosing recommendations for each nutrient. I suggest printing the report for ease of study and quick reference. It should be worth the effort.

Remember.... Knowledge is power. So, stick with it, understand it and empower yourself!

Jackie Burgess, RDH (ret)
8-year LAF journey (vagal)
14+ years ongoing AF research
Onset afib age 59
PVI ablation age 67 (Natale 2003)

Caveat/Caution: Anyone planning to use supplemental magnesium or potassium should have healthy kidney function confirmed by testing ordered by their physician. If they are using medications that are potassium sparing, then supplementation must proceed under medical supervision. Adverse effects can result from using too much potassium. Be aware.

Be aware also that unfortunately, a great number of physicians are not well versed in recommending or supervising nutritional support and quite often, will dismiss your inquiries about nutritional successes. In *Metabolic Cardiology* by Stephen Sinatra, the introduction by Integrative Cardiologist James C. Roberts, MD, comments about their educational mission directed at physicians...

..... “Our agenda is to improve the nation’s cardiac health—why should our patients be the only ones to get better? That’s why we spend so much time away from home educating doctors and patients about the fundamental relationship between nutrition and health and wellness.

So why don’t classically trained cardiologists recognize this? Why don’t they flock to this message? They are obviously intelligent, well-educated people who have the best interest of their patients at heart. The answer is really quite simple: politics, money and training. Major medical research is funded by drug companies; they also fund our meetings and their advertising funds our professional journals. Nutritional therapies do not move the revenue needle for hospitals, doctors, research institutions or the drug companies.

And because traditionally, doctors have not been well trained in biochemistry, there is a lot of misunderstanding about the fundamental physiological relationships between basic cellular bioenergetics and cardiac functions.

Because of this lack of understanding, doctors don’t want to be known as ‘vitamin doctors’. They don’t want their local peers to see them as kooks and don’t want referrals from family doctors to dry up. They stay mainstream, and use the “lack of science” argument when discussing nutritional therapies.” The studies are there but the doctors just don’t know about them (or don’t) want to know about them. The orthodox medical community is ten years behind in this area of research. “(Roberts) (2)

THE FUNCTION OF ELECTROLYTES

To clarify the importance of electrolytes (minerals) specifically as it relates to this report, the following explanatory excerpt from a teleconference by Paula Rochelle, ND, (4) Board Certified/Licensed Naturopathy/Naturopathic Endocrinology, is relevant.

Dr. Rochelle says: The body doesn’t function at all without electrolytes. Without them, we’re dead. Electrolytes are ionized molecules found throughout the body – blood, tissue, cells and are either positive cations or negative anions that conduct an electrical current that helps keep pH balance (the acid/base level of our body) in check. They aid in passage of fluid between and within the cells by osmosis and regulate the neuromuscular, neuroendocrine and excretory system. Most people we see are dehydrated and we should actually be composed of about 60% body weight in fluid and the electrolytes would be in that fluid to regulate body function and fluid balance.

Basically, the GI tract, kidneys endocrine system, pituitary, thyroid, adrenals regulate fluid electrolyte balance. Forty per cent of body weight is intracellular and 20% is extracellular

KEY ELECTROLYTES: potassium, magnesium, calcium, sodium, chloride, hydrogen phosphate, hydrogen carbonate

PREPARATORY INFORMATION

As a preamble and to help clarify “tissue compliance,” following is an inquiry response from researcher, David Van Wagoner, PhD researcher at the Cleveland Clinic wherein he describes the electrical activity involved. This guides the way to identifying the associated metabolic cardiology components that are the topic of this report.

“ I can provide some scientific information that is relevant to your question, but does not constitute medical advice. I am a basic scientist in the department of Cardiology here at CCF, and my work is focused on cellular studies of human atrial fibrillation, as well as in experimental models.

The excitability of cardiac cells is influenced by their resting electrical potential, and by the activity of specific protein molecules (ion channels) that are present in the surface membrane of the cardiac cells. There are ion channels with selectivity for different ions including potassium, sodium and calcium. Potassium channels are open at rest in cardiac myocytes, and control the electrical potential during periods of relaxation. During cardiac contraction (action potential), sodium and calcium channels both open and then close. A different set of potassium channel openings help to re-establish the quiet electrical period between heart beats.

At normal plasma (blood) potassium levels (4-5 mmol/liter), there is a healthy balance between potassium channels open at rest and channels that open to end a period of electrical activity (the action potential). When the potassium in the plasma is elevated (for example, 10 mmol/liter) the cardiac cell becomes depolarized and unexcitable. When the potassium concentration is low, the cells become unusually excitable, and the channels that normally open to terminate an action potential are less effective. The combined effect is that spontaneous activity tends to increase in areas of the heart that do not normally have spontaneous activity, and this can be noted as an increase in either premature atrial or ventricular contractions. These can potentially trigger more sustained arrhythmias, such as atrial fibrillation. Thus, it is very desirable to keep the blood potassium in the normal range.” (3)

In his paper, *Pharmacologist Relevance of K⁺ Channel Remodeling in Atrial Fibrillation* (2000), Dr. Van Wagoner states:

...”Experimental studies have demonstrated that AF requires both a tissue substrate that can sustain the arrhythmia and a trigger to evoke it. Much evidence now supports the notion that AF is largely a re-entrant arrhythmia, with multiple circulating wavelets of electrical activity. Tissue area, conduction velocity, and refractory period thus determine whether re-entrant rhythms are spontaneously terminated or can be maintained. Given the absence of changes in other parameters, the refractory period is perhaps the most dynamic parameter. Antiarrhythmic drug strategies based on ion channel blockage either low conduction velocity (class I drugs such as flecainide) or prolong the refractory period (class III drugs such as sotalol or dofetilide).

The refractory period largely reflects the duration of the atrial action potential. Thus, the determinants of this refractory period are the inward and outward ionic currents that summate to form the action potential. Both the inward sodium current (I_{Na}) and transient outward current (I_{to}) are activated and inactivate rapidly during the action potential.” (3)

In the reference section, following the Van Wagoner reference(3) is a collection of web links on articles on the function of the ion channels and the sodium/potassium pump function for those who want more detail.

Based on that science, and from the success testimonials from former afibbers who have 'cured' themselves, it's a reasonable assumption that the goal of tissue compliance was met (by various means). Be aware that many factors can interfere with or inhibit optimizing the critical nutrients so while it may be easy to say supplemental nutrients don't work, the problem may lie with the fact that the nutrients are unavailable (absorption issues) to the inside of cells where they function, or that they are wasted so quickly cells can't function as they should, or that a genetic mutation is causing interference with availability, absorption, utilization or the stabilizing effect... and, there is always the compliance issue to consider.

The following sections are overviews of each essential nutrient and how they function synergistically to maintain NSR.

Definition: Mitochondria, ATP

Mitochondria are rod-shaped organelles that can be considered the power generators of the cell, converting oxygen and nutrients into adenosine triphosphate (ATP). ATP is the chemical energy "currency" of the cell that powers the cell's metabolic activities. This process is called aerobic respiration and is the reason animals breathe oxygen. Without mitochondria (singular, mitochondrion), higher animals would likely not exist because their cells would only be able to obtain energy from anaerobic respiration (in the absence of oxygen), a process much less efficient than aerobic respiration. In fact, mitochondria enable cells to produce 15 times more ATP than they could otherwise, and complex animals, like humans, need large amounts of energy in order to survive."

<http://micro.magnet.fsu.edu/cells/mitochondria/mitochondria.html>

Images of mitochondria: <http://tinyurl.com/2ew65v2>

KEY NUTRIENT #1 MAGNESIUM

When new afibbers visit the website, typical introductory instructions include a focus on the Essential Trio (magnesium, potassium, taurine) as a place to begin. We assume that the majority of afibbers are deficient in magnesium.

Magnesium (Mg) lies at the core of metabolic cardiology because it is so intricately involved in cellular function. When magnesium deficiency exists, various systemic symptoms or dysfunctions occur. Some are quite obvious such as constipation, muscle cramps and fasciculations. Others, such as malfunction of cellular mechanisms, not as obvious until the 'strategy' is understood. From the literature and personal experiences, we (afibbers) have learned that magnesium is critically important; and, specifically, it's the magnesium that is inside each and every cell in our body that counts... intracellular magnesium.

Numerous influencing factors contribute to insufficient cellular nutrition that can set the stage for afib as well as other chronic disease conditions and dysfunctions. These include the obvious but are not limited to:

- inadequate dietary intake,
- drinking water sources,
- depleted or missing food-nutrient values as a whole,
- GI - gut mal-absorption or interference issues,
- wasting syndromes and genetic flaws,
- known depleters of magnesium or too much calcium intake,
- high carbohydrate intake,
- Rx drugs,
- stress,
- alcohol,

exercise , name a few.. And, it's not uncommon for afibbers to harbor several influencing factors at the same time. Magnesium deficiency is common and depending on who's quoting, the deficiencies range from 65% to 80% in general populations in the U.S. and globally.

This begs the question: "If magnesium is so critical and most everyone is deficient, why doesn't everyone have or eventually get afib?" As we know, over the past 20 years, the incidence of afib is increasing. What's changed? The answer should eventually become obvious.

All articles or reports on magnesium mention it is a required cofactor for over 300 enzymatic reactions throughout the body that are known; there could be more.

Key point and most relevant to this metabolic cardiology discussion, the cellular energy molecule, ATP, (adenosine triphosphate) must be complexed to magnesium to be metabolically available. So starting with this fact, magnesium becomes the lynchpin for cellular energy production, heart tissue compliance and rhythm stabilization. Without energy, cells can't function well or survive.

Aileen-Burford Mason, PhD,(5) Canadian immunologist, cell biologist, lecturer and clinician offers some observations about magnesium which are helpful when understanding the facets of The Strategy.

...." Magnesium's regulatory role in energy production, in the biosynthesis of catecholamines and other neurotransmitters needed for neuromuscular activity, as well as neurological excitability, muscle relaxation after contraction, and bone metabolism, gives magnesium a key role in musculoskeletal function and health.

Stress and magnesium depletion Dr. Burford-Mason says "Stress is involved in magnesium homeostasis. The biggest issue we are facing with regard to our needs for magnesium is stress and the medical problems that ensue from magnesium deficiency go right back to excessive stress. A review of the literature finds many studies looking at the physical, medical, environmental, mental, psychological factors that affect magnesium status. Things that we know that are good for us like exercise also depletes magnesium particularly if we sweat a lot; heat and cold; sleep deprivation, you can't sleep because you are short on magnesium so it's a downward spiral because you are depleted by the stress of not sleeping. Childbirth, pregnancy---it is no surprise that as a pregnancy goes on women get leg and foot cramps and can't sleep because of magnesium deficiencies. When the immune system is activated whether through allergy or infection, magnesium is used up big time.

Even things like noise. Very interesting studies done on noise-induced hearing loss and blocking the damage that's done whether you are a pop musician or work in a factory with a lot of noise by using magnesium and even reversing hearing loss with magnesium supplementation. Where there is stress and more cortisol, catecholamine production, you will urinate out more magnesium.

The emphasis is on getting magnesium inside the cell so insulin is necessary to do that. Therefore, in insulin-resistant people, there is difficulty achieving that. Insulin may be the most important hormone in the body because it is our storage hormone...it doesn't just store excess sugar for later use, it stores everything. So if you become insulin resistant, by definition, you cannot store magnesium in red cells, amino acids in neurons and all the other minerals as well. She says if there is a disease of diseases, it is metabolic syndrome or insulin resistance because it leads to dementias, heart disease and stroke, diabetes, osteoporosis because of the failure to store nutrients.

And vice versa...we know that by supplementing with magnesium, we improve insulin sensitivity because insulin requires magnesium for binding and for synthesis. Some people think that magnesium deficiency is a big part of diabetes. I think it is a big part of everything, she says.

It's often said magnesium is nature's calcium blocker. I like to change that to say "calcium channel blockers are the pharmaceutical arms to magnesium deficiency." Magnesium should be intracellular most of the time and it keeps calcium outside the cell.

Women who choose not to do any hormonal therapy may be more magnesium deficient because estrogen helps get magnesium inside the cell. In magnesium deficiency, there will be a lot of sweating --- particularly in men... night sweats in men are always

magnesium deficiency. In women with hot flashes, they sweat out the magnesium and they sweat because they are short in magnesium. In these women, once you get the magnesium up to bowel tolerance, they sweat less and that's because the sweat glands are worked with little smooth muscle cells.(5)

Sherry Rogers, MD, author of *Is Your Cardiologist Killing You* (Prestige Publishing), comments "If you have arrhythmias, guess what? Magnesium and essential fatty acids can help you." Dr. Rogers cites studies have found that magnesium deficiency induces arrhythmias. And that several studies have found magnesium to be more effective than any prescription drug at treating arrhythmias. Dr. Rogers says studies have found that "environmental chemicals damage the autonomic nervous system and create any arrhythmia imaginable."

"As well, all drugs use up or deplete nutrients from the body in the work of detoxifying them. So by a second mechanism, drugs also create new symptoms and diseases that seem unrelated and are often unsuspected as being caused by medications.

The exciting part is that we now know so much molecular biochemistry of the body, and how it is orchestrated to harmonize with foods and nutrients that we have enormous power to actually cure conditions that are currently relegated only to drug therapies."

More strong support for lone afibbers being magnesium deficient comes from The Afibbers report, thanks to Hans Larsen's publication:

Afibbers are magnesium-deficient

HARTFORD, CONNECTICUT. Magnesium (Mg) is an enormously important mineral being a cofactor in over 300 enzymatic reactions continuously taking place in the body. Magnesium is also a vital component of the skeletal structure and about 65% of the body's magnesium stores are found in bone, another 34% is found in transcellular fluids, and the remaining 1% is found in extracellular fluids such as blood. It is thus clear that measuring magnesium in blood serum is not likely to be a very accurate measure of the body's overall magnesium status.

There is increasing evidence that magnesium plays a crucial role in preventing and terminating cardiac arrhythmias. A group of cardiologists and pharmacologists at the Hartford Hospital reasoned that a pre-procedure infusion of magnesium might help prevent the acute development of atrial fibrillation following a radiofrequency ablation for this disorder. As a first step in proving or disproving this hypothesis, they decided to do a trial in which half the participants would have saline solution (0.9% sodium chloride) with 4 grams of magnesium sulfate (800 mg elemental magnesium) infused over a 15-minute period just prior to accessing the left atrium in a standard PVI procedure, while the other half would just have a saline solution infusion.

The trial involved 22 patients with paroxysmal or persistent afib. Samples of venous blood (for determination of extracellular Mg concentration) and buccal scrapings (scrapings from inside the cheek) were collected before the start of the procedure, 15 minutes after the completion of the infusion, at the end of the ablation procedure, and at 6 hours after the infusion. The blood samples (serum) were analyzed for extracellular magnesium concentration and the buccal scrapings were analyzed (using the *EXAtest*) for intracellular magnesium concentration as well as for concentrations of calcium, potassium, sodium, chloride, and phosphate. At least one study has shown that there is an excellent correlation between the magnesium (intracellular) content of buccal scrapings and that of myocytes (heart cells). The major findings are as follows:

- None of the study participants were deficient in Mg at baseline when considering blood serum values only. The average serum Mg concentration was 2.08 mg/dL versus the normal lower limit of 1.6 mg/dL.

The majority (89%) of participants were magnesium-deficient at baseline when considering intracellular (*EXAtest*) values only. The average intracellular Mg concentration was 32.2 mEq/IU versus a normal lower limit of 33.9 mEq/IU. NOTE: The unit is defined

as x-ray intensity (peak divided by background) divided by unit cell volume.

There was no correlation whatsoever between serum magnesium and intracellular magnesium concentrations.

· Serum levels of Mg rose rapidly in the magnesium infusion group 15 minutes post-infusion and, although declining over the 6-hour observation period, remained considerably higher than the level in the placebo group (saline infusion only).

Intracellular level of Mg increased rapidly in the magnesium infusion group 15 minutes post infusion and continued to rise throughout the 6-hour observation period. Somewhat surprisingly, the intracellular Mg level also increased somewhat (over baseline) in the placebo group over the 6-hour period. The Hartford researchers speculate that the ablation procedure itself, most likely the anaesthesia, facilitates the transfer of magnesium from serum to intracellular space.

The intracellular calcium concentration increased significantly in the Mg infusion group post infusion, but gradually reverted to baseline over the 6-hour period.

The intracellular potassium concentration increased by about 50% from baseline to the end of the PVI procedure and then began to drop off at the 6-hour mark.

The authors of the report conclude that future studies are needed to evaluate the electrophysiologic benefits of magnesium repletion and the effects of routine procedures and anaesthesia on intracellular electrolytes.

Shah, SA, et al. The impact of magnesium sulfate on serum magnesium concentrations and intracellular electrolyte concentrations among patients undergoing radio frequency catheter ablation. Connecticut Medicine, Vol. 72, May 2008, pp. 261-65

Editor's comment: A 2006 LAF Survey (LAFS-11) found that, among a small sample of 7 afibbers who had EXAtest results, all 7 were either below or very close to the lower normal limit. The Hartford report provides important additional evidence to support the conclusion that afibbers are likely low in intracellular magnesium even though their blood serum levels may be normal. It is also of interest that replenishing magnesium via an infusion not only increases intracellular Mg concentration, but also increases intracellular potassium levels. This is all good support for our long-held conviction that lone afibbers with normal kidney function are likely to benefit from supplementing with magnesium, potassium, and taurine (facilitates the uptake of Mg and K).

Key point to remember:

The critical role of magnesium: Influences function of the sodium/potassium pump mechanism.

While magnesium is critically important, potassium is equally critical. They both have to be in optimal supply.

KEY NUTRIENT #2 POTASSIUM

(Seelig/Rosanoff – The Magnesium Factor)(6)

“If the level of magnesium within a cell becomes too low, there are three dire results:

- 1) There is not enough ATP available for the cell's necessary energy reactions to maintain the “enzymatic” pump that moves potassium into and sodium out of the cells.
- 2) Potassium leaves the cell and cannot re-enter, which creates a risk of arrhythmia.
- 3) Calcium rushes into the cell, where it does not belong, and creates its excitatory and hardening havoc.”

Drs. Seelig and Rosanoff explain that this low magnesium state allows cells to malfunction in predictable ways:

- The secretion of adrenaline increases abnormally and cells over-respond to the adrenaline stimulation
- All muscle cells, including those in the heart and blood vessels tend to contract and become unable to relax
- Glucose is not properly processed as a result of insulin resistance,
- Blood tends to clot more easily – risk for MI and stroke

The supporting biochemistry:

Magnesium is required by adenosine triphosphatase (ATPase), which maintains the sodium-potassium gradient across all membranes and regulates intracellular calcium levels, myocytes function and calcium reuptake by the sarcoplasmic reticulum. Magnesium is also important for maintenance of cardiac rhythm, vasomotor tone, neuromuscular function and parathyroid hormone metabolism.(7)

Magnesium is important in regulating the intracellular potassium content. Intracellular magnesium activates membrane-bound magnesium-dependent sodium-potassium ATPase, which pumps sodium out of the cell in exchange for potassium. In addition, extracellular magnesium retards cell efflux of potassium on a biophysical basis. Thus, magnesium deficiency impairs the sodium-potassium pump and allows potassium to escape from the cell, to be lost in the urine. Hence, magnesium depletion can lead to potassium depletion. In fact, hypokalemia occurs in 46% of hypomagnesemic patients; total body potassium depletion maybe profound, and massive supplementation of potassium may fail to correct it until the magnesium deficit is repaired.(7)

THE SODIUM-POTASSIUM PUMP – as it relates to afibbers

Richard D. Moore, MD, PhD (8) goes into great detail about the function of the sodium/potassium pump (Na/K pump) and what he calls the K Factor. (K for potassium; Na for sodium). While he is specifically addressing hypertension in his book, *The K Factor*, the science fits for afib as well because “tissue compliance” is dependent upon a dietary intake of potassium that provides a favorable ratio of K to Na in the body and ultimately, cells... every cell in all tissue of your whole body.

Early man’s consumption of sodium and potassium worked out to about 11,000 mg daily of potassium and only 690 of sodium. A ratio of about 16:1. (Moore) Other sources say the potassium was around 6,000 mg with 600 mg sodium. Salt was not a big factor in early man’s diet.

Dr. Moore says: “The low ratio of potassium to sodium in the typical American diet is one of the biggest - perhaps even *the* biggest -cause of bad health in our country.” So, let’s examine what this might mean to afibbers.

Cellular biophysics tells us that one of several dozen energy-consuming mechanisms in living cells uses about 25% of the total energy of that cell and therefore is of vital importance to that cell. This mechanism is the Na-K pump and it’s the key to survival for every cell in our body. Not only does this specialized pump keep potassium levels in the cell high and sodium levels low, but it makes an electrical current which is carried by positive sodium ions that the cell uses for itself, but also serves to regulate acid and calcium levels inside the cell. Dr. Moore says: “When a living cell is exposed to a substance that specifically inhibits the Na-K pump and without affecting any other mechanism...that cells dies. So this pump is of vital importance to everyone – afibbers especially.

Must view: video simulation of *How the Sodium-Potassium Pump Works*.
<http://tinyurl.com/yutaw4>

Quote from *Principles of Biochemistry*, 1992:

The activity of this Na/K-ATPase in extruding Na⁺ and accumulating K⁺ is an essential cell function. About 25% of the energy-yielding metabolism of a human at rest goes to support the Na/K-ATPase.

25%!! Think about it! If we don't have these pumps working, imagine how this affects heart tissue, especially.

Dr. Moore says the ideal ratio of dietary intake will be between 2 and 4 parts potassium to one part sodium. In his book, he emphasizes 4:1.

His message: "The sum of intracellular K⁺ Na is a constant... meaning you can't raise IC potassium unless you lower IC sodium."

Moore) (8) Chapter 4, p. 78: The Key Problem: An Imbalance in the Ratio of Potassium to Sodium (The K-factor)

"For purely physical reasons (connected with the law of osmotic equilibrium), inside the cell the sum of sodium plus potassium *must* be constant. This means that sodium can go up *only* if potassium goes down. Likewise, if potassium goes up, sodium *must* go down. So potassium and sodium are unalterably linked together like two children on a teeter-totter. You can't change one without changing the other."

POTASSIUM AND THE REFRACTORY PERIOD.

Potassium prolongs the refractory period...or the time when the heart is resting between beats. At this time, heart cells can't be stimulated to contract.

Supporting biochemistry:

In the resting state, cardiac muscle cells are polarized due to gradients established by the active inward transport of potassium ions and the outward transport of sodium ions. Various stimuli—including drug-induced effects—can cause shifts in these gradients, producing a decrease in the internal negative membrane potential. This process is known as depolarization.(9)

Adverse effects or clinical consequences of potassium depletion predominantly affect the cardiovascular and neuromuscular systems. Both respond to the associated hyperpolarization of electrical tissue.

The presence of hypokalemia (potassium depletion), decreased membrane permeability to potassium (which prolongs action potentials), shortens refractory periods and increases the incidence of spontaneous and early depolarizations.

For cardiac cells, the result of these alterations is a propensity for arrhythmias, particularly in persons who are taking digitalis (Rutecki & Whittier) (10)

Caution: "A note of caution about beta blockers. Beta blockers diminish the regulation of serum K during potassium loading. In the presence of beta blockers, plasma potassium can spike during a potassium load."

Dr. Moore (8) Chapter 19, Information for the Physician, p. 323,

Drugs That May Make the K Factor Dangerous

It's not simply the amount of potassium that's important, it's the dietary and IC potassium-to-sodium ratio, which needs to be at least 4:1

Insufficient dietary intake leads to potassium deficiency (hypokalemia)

A 'tea and toast diet' typical in the diminished appetites of the elderly is conducive to hypokalemia but easily applies to the typical Standard American Diet (SAD) devoid of potassium-containing fresh vegetables and fruit. Therefore, while arrhythmia is easily explained in an aging population, it also can just as easily be associated with poor dietary choices—foods with high salt content (sodium chloride) or other potassium depleters. Even something as routinely basic as heavy

exercise producing abundant perspiration sets the stage, especially if potassium stores are already low or marginally low.

About 90 percent of the sodium consumed in the average diet is in excess of body needs and must be eliminated in the urine. Therefore, urine levels reflect dietary intake. Aldosterone, a hormone made and secreted by the adrenal cortex, acts on the kidneys to regulate sodium metabolism.

The Senate Select Committee on Nutrition and Human Needs suggests about 5 grams of salt, which provides about 2 grams of sodium, per day. We really need only about 0.5 gram to maintain the body's salt concentration and probably 1-2 grams to be safe, unless we perspire a great deal or are active exercisers.

Most people consume excess sodium. The average American diet contains about 3-6 grams of sodium, or about 7-15 grams of salt, per day.(11)
True culprit in over-doing salt intake is not the salt shaker, but rather processed, packaged foods...the hidden salt.(12)

Systemic organ dysfunction Brief mention that additional consideration to the Na/K balancing dilemma includes systemic dysfunction such as -- hyperaldosteronism, adrenal tumor, kidney impairment with sodium/potassium retention/excretion dysfunctions are suspect when the normal levels are not maintained, and the appropriate testing to rule out organ dysfunction is definitely important. Some instances, judicious of using the potassium-sparing diuretic aldosterone antagonist, Spironolactone or similar drugs, are useful in preventing the body from absorbing too much sodium and keeps potassium from getting too low. Another observation (7) indicates potassium-sparing diuretics such as amiloride, triamterene and, to a lesser extent, spironolactone have magnesium sparing properties and may be useful in the prevention of magnesium deficiency secondary to long-term diuretic therapy..

Importance of Potassium in Cardiovascular Disease(13) Mg^{++} and/or K^+ depletion can play a pivotal role in the genesis of cardiac arrhythmias, particularly in patients with underlying ischemic heart disease. Mg^{++} has important electrophysiologic effects; it plays a critical role in the regulation of energy sources and in the control of ion pumps, and it has a direct membrane stabilizing effect on excitable myocardial membranes without adversely affecting repolarization time.

Cardiac Implications of Potassium K^+ is critical to the maintenance of CV health and the normokalemic state is vital to the prevention of potentially serious sequelae, especially in the at-risk CV patient. In such patients, many factors, such as endogenous and exogenous catecholamine activity, activation of the RAAS, and/or the use of potent K^+ -wasting diuretics, may lead to hypokalemia. K^+ -wasting diuretics are noteworthy in this regard in that their use can lead to significant K^+ or Mg^{++} deficiency, or both.

Electrophysiologic Effects of Potassium Because K^+ serves as the primary ion mediating cardiac repolarization, the hypokalemic state is highly arrhythmogenic, particularly in the presence of digoxin or antiarrhythmic drug therapy. Hypokalemic states produce complex effects on myocardial refractory periods and the potential for triggered arrhythmias. In contrast, hyperkalemia causes slowed conduction and conduction block which, if sufficiently progressive, can result in asystole. Hyperkalemia may also attenuate the effects of antiarrhythmic agents and repolarizing K^+ currents.

Potassium and Arrhythmias Clinically, arrhythmias associated with hypokalemia include atrial fibrillation and multifocal atrial tachycardias. The most concerning and life-threatening arrhythmias associated with K^+ deficiency states are ventricular tachyarrhythmias, which range from an increase in the frequency of premature ventricular contractions, linearly related to the fall in serum K^+ concentrations, to nonsustained ventricular tachycardias and triggering of monomorphic and polymorphic ventricular tachycardias, including torsade de pointes and ventricular fibrillation. Low extracellular K^+ concentrations also alter the effects of antiarrhythmic drugs.(13)

CAVEAT ON CALCIUM INTAKE – SOURCE OF TROUBLE FOR AFIBBERS

Before we move away from magnesium and potassium, it's important to discuss the role of calcium in arrhythmias. While calcium is an important electrolyte, a high level of intracellular calcium-- especially when intracellular magnesium levels are deficient-- can be a primary cause of arrhythmia. Calcium inside heart cells is excitatory.

Most recommendations for calcium-to-magnesium ratios indicate a preference 2:1 but many afibbers find that a 1:1 ratio is better or even less (Ca) to avoid arrhythmias triggered by calcium dominance inside heart cells. Some people are unable to take more than 600 or 800 mg of magnesium a day and avoid the bowel issue, so keeping track of calcium intake from food sources and then closely controlling supplements, if taken at all, is definitely something worth monitoring until the prevalence of afib events is diminished greatly or absent entirely. [I can't tell you how many AF people I've helped just by having them stop or lower significantly, their calcium supplements and increase magnesium.]

Dr. Burford-Mason(5) states:

If magnesium intake is low, a high calcium intake can make people more vulnerable to heart disease than people who do not have a high calcium intake. The current promotion of calcium-rich foods and supplements to protect our bones encourages the consumption. This is fine as long as magnesium nutrition is adequate. But calcium intakes that are unduly high relative to magnesium can intensify the problem caused by the low magnesium contents of most modern diets.

Calcium is an important essential nutrient, but it must be guarded and controlled and balanced by adequate magnesium if it is not to cause damage to the cells and the body as a whole.

We are overdoing calcium, big time, since only about 7% of calcium we take can get into bones without adequate levels of vitamin D - Our hunter-gatherer forbearers had a very low intake of calcium, yet according to archeologists and anthropologists, they had strong bones and they were tall. They didn't have degenerative bone disease until we became farmers and that became obvious there were more bone problems then. They did well on 300mg of calcium a day but they had a lot more exposure to sunshine than we do.

Modern acidic diets (based on urine pH evaluations) utilize calcium to buffer acidity instead of being used for bone building. We do know that calcium is regularly being laid down regularly in soft tissues where it should not be. For instance, we are unwittingly monitoring this in one organ and not for calcium deposits per se but in women getting regular mammograms. In North America in all the recalls for mammograms only 1.5% of them have to do with cancer; the rest to calcification. We have overdone calcium and we didn't realize the role that vitamin D plays. Certainly we have not paid interest to magnesium that we should have been paying to it. In fact, magnesium alone has been shown in studies to reverse osteoporosis as well. And along with bone health, vitamin K is shown to reverse arterial calcification due to the involvement with the matrix GLA proteins... and we are probably all short on vitamin K as well...although not a deficiency. In the case of vitamin K, most people have enough for clotting but not enough for bones. (end quote)

Just to reemphasize this calcium point one more time, as it relates to afibbers, a clip from a 1999 newsletter on Calcium Overload.

Calcium overload is a killer. Indeed, calcium overload is the only killer. It occurs when the ion-regulating apparatus in your cells breaks down and can no longer maintain normal intracellular concentrations. You can say that Mr. Jones died of heart failure, or drowned or had a Harley accident. He really left this life because of calcium overload secondary to heart failure or drowning or trauma.

Maintenance of the intracellular ionic milieu is the fundamental definition of life. We drink water, breathe oxygen and eat food for no other reason than to sustain the ability of our cells to exert tight control over the ionic levels within them.

The concentration of Ca²⁺ on the outside of our cells is 10,000 times higher than it is on the inside. The life-process of every cell is nothing more than a perpetual struggle to make use of this enormous electrochemical gradient without being smothered by it. Aging is simply the slow but inexorable failure of the ability of an organism's cells to hold back this towering tide of calcium.

When a cell malfunctions, because of that high extracellular calcium level, the result is a spike in intracellular calcium. The tripwire that signals a cell to destroy itself (apoptosis) is a sustained rise in intracellular calcium that the cell cannot handle via its various defense mechanisms.(14)

Intracellular Calcium Overload and Atrial Fibrillation(15)

VanWagoner et al studied atrial ICal in patients with and without chronic AF. Whereas ICal was significantly reduced in the myocytes of patients with chronic AF (11 patients), half of the patients in the control group (19/38) with the greatest ICal may be more easily subject to atrial calcium overload. Thus Ca overload may be an important factor in the initiation of AF and the reduction of functional intercellular calcium density in myocytes from the atrial of chronic AF patients may be an adaptive response to the arrhythmia-induced Ca overload.

.....the high rate in AF can induce Ca(i) overload, contributing to further increase of spontaneous SR Ca²⁺ release. With respect to the chronology of the major electrophysiological changes, Ca(i) overload is likely to be a primary factor mediating both the short-term and chronic electrophysiological remodeling associated with AF.

.....We conclude that Ca(i) dynamics are important in the generation and maintenance of AF.

(15) Atrial Fibrillation: From Bench to Bedside by Andrea Natale, José Jalife

KEY NUTRIENT #3 TAURINE

Taurine has been shown to:

- Modulate heart-muscle calcium

- Act as an antioxidant

- Lower blood pressure

- Prevent arrhythmias and

- Improve muscle strength.

By regulating cellular calcium, taurine not only improves heart muscle contraction but also prevents it from becoming overly irritable which can lead to arrhythmias.(Blaylock)(16)

Taurine is a sulfur-containing amino acid that has been shown to protect against heart attack damage. Taurine plays a myriad of roles in promoting health- especially for afibbers. Arrhythmia may be a sign of taurine deficiency. Taurine strengthens the heart muscle and plays a major role in regulating the heart's contractility.(17)

Taurine also acts as a natural diuretic by keeping potassium and magnesium inside cells and keeping excess sodium out but unlike prescription diuretics, taurine does not have adverse effects on the kidneys. By eliminating excess fluid, taurine helps alleviate pressure on blood vessels. It increases circulation and stabilizes blood pressure by dampening the sympathetic nervous system which when overactive constricts blood vessels.

Food additives such as monosodium glutamate (MSG) and aspartame lower the body's concentration of taurine. Afibbers are affected by these excitotoxins as are those affected with seizure activity.

Using taurine may be especially helpful for those who experience LAF as a result of reactive hypoglycemia as it is known that taurine helps stabilize blood sugar in both Type I and Type II diabetes. Taurine appears to do this by potentiating the activity of the insulin receptor.

Few adverse reactions are associated with taurine supplementation. Most people tolerate between 1 and 4 grams per day well. However, those with ulcers should use taurine carefully because taurine may increase the secretion of stomach acid. If you have a medical condition, please take

taurine with the guidance of your health care practitioner, as taurine may change or reduce your need for certain medications. (18)

For more information on taurine – refer to this post at afibber.org, title:

What About Taurine?

http://www.afibbers.net/forum/read.php?f=8&i=18914&t=18885#reply_18914

Also see Ref. 19, 20. –(the article by Dr. Smayda)

Taurine and neural cell damage. 2000;19(3-4):509-26

Saransaari P, Oja SS. Brain Research Center, Medical School, University of Tampere, Finland.
blpisa@uta.fi

The inhibitory amino acid taurine is an osmoregulator and neuromodulator, also exerting neuroprotective actions in neural tissue. We review now the involvement of taurine in neuron-damaging conditions, including hypoxia, hypoglycemia, ischemia, oxidative stress, and the presence of free radicals, metabolic poisons and an excess of ammonia. The brain concentration of taurine is increased in several models of ischemic injury in vivo. Cell-damaging conditions which perturb the oxidative metabolism needed for active transport across cell membranes generally reduce taurine uptake in vitro, immature brain tissue being more tolerant to the lack of oxygen. In ischemia nonsaturable diffusion increases considerably. Both basal and K⁺-stimulated release of taurine in the hippocampus in vitro is markedly enhanced under cell-damaging conditions, ischemia, free radicals and metabolic poisons being the most potent. Hypoxia, hypoglycemia, ischemia, free radicals and oxidative stress also increase the initial basal release of taurine in cerebellar granule neurons, while the release is only moderately enhanced in hypoxia and ischemia in cerebral cortical astrocytes. The taurine release induced by ischemia is for the most part Ca²⁺-independent, a Ca²⁺-dependent mechanism being discernible only in hippocampal slices from developing mice. Moreover, a considerable portion of hippocampal taurine release in ischemia is mediated by the reversal of Na⁺-dependent transporters. The enhanced release in adults may comprise a swelling-induced component through Cl⁻ channels, which is not discernible in developing mice. Excitotoxic concentrations of glutamate also potentiate taurine release in mouse hippocampal slices. The ability of ionotropic glutamate receptor agonists to evoke taurine release varies under different cell-damaging conditions, the N-methyl-D-aspartate-evoked release being clearly receptor-mediated in ischemia. Neurotoxic ammonia has been shown to provoke taurine release from different brain preparations, indicating that the ammonia-induced release may modify neuronal excitability in hyperammonic conditions. Taurine released simultaneously with an excess of excitatory amino acids in the hippocampus under ischemic and other neuron-damaging conditions may constitute an important protective mechanism against excitotoxicity, counteracting the harmful effects which lead to neuronal death. The release of taurine may prevent excitation from reaching neurotoxic levels.

<http://www.ncbi.nlm.nih.gov/pubmed/11140356>

KEY NUTRIENTS #4 , #5, #6 - COENZYME Q10, CARNITINE, RIBOSE

Many books have been devoted specifically to each of these key nutrients. A couple of explanatory paragraphs can hardly do the topic justice. Chapters in Dr. Sinatra's book(2) are much more complete. See reading suggestions at the end for other recommendations. Meanwhile, these are very basic descriptions of how these nutrients function synergistically to help afibbers.

CoQ10, the carnitines and ribose all work to enhance the production of the energy molecule, adenosine-triphosphate (ATP). Backtracking to Page 9 and the statement from the Magnesium Factor for emphasis:

"If cellular magnesium becomes too low, then there is not enough ATP available for the cell's energy reactions to maintain the sodium/potassium pump. "

Coenzyme Q10 is called "The Spark of Life" in Dr. Sinatra's book (2) Chapter 4. Without CoQ10, we can't survive. As cellular levels of CoQ10 fall, so does general health. Not only is CoQ a powerful antioxidant providing protection from free radical (oxidative stress damage), but CoQ's role in cellular energy production is of paramount important. CoQ transports electrons to the

mitochondria where energy is used to generate ATP thereby making fuel for every cellular function. (Heart cells have the highest concentration of mitochondria-about 5000 in each heart cell compared to an average of about 70 in a biceps muscle for instance).

In the mitochondria of each cell, CoQ provides the spark that initiates the energy process. When there is a CoQ deficiency, the cellular "engine" misfires and may fail or die...leading to a failing heart, or immune system and the inevitable weakening of defense against disease and aging... [and relevant to this discussion – the energy for the ATP to maintain the Na/K pump and the requisite tissue compliance factor that supports the prolonged refractory period and therefore NSR.]

Dr. Sinatra calls CoQ10 "fertilizer" for heart." He says, "with a fertilizer like CoQ to fortify the mitochondria, it makes sense to treat or fortify an energy-starved heart composed of thousands of mitochondria. This makes the cardiac cell pulsate and you get more mileage out of the cell, especially in an ischemic cell."

CoQ also acts to reduce artery damage and plaque formation via its antioxidant action. It also has a remarkable affinity to protect heart cells that are deficient in oxygen. This is of major importance in the tissue compliance factor. As a powerful antioxidant, CoQ helps diminish the detrimental effects of free-radical damage or oxidative stress. Certainly, an influencing factor for AFib.

CoQ10 prolongs the action potential and we know that is what helps maintain NSR. Dr. Sinatra notes studies using CoQ10 (60 mg/day) reduced incidence of premature ventricular contractions (PVCs). Other studies indicate CoQ shortened the QT interval reflecting membrane stabilization in heart attack patients.

CoQ10 researcher William V. Judy, PhD, (21) said this in his teleconference on Coenzyme Q10: Co Q is responsible for conduction of the impulse down that nerve into the AV node, the SA node and is important for membrane stabilization. Remember when the heart contracts, sodium rushes in, calcium rushes in – there is a long plateau of the action potential, then potassium rushes out and so forth, but that final stabilization where the membrane becomes stable – requires the sodium/potassium ATP pump which is energy driven and if you have CoQ deficiency in a small locus then you have unstable membranes which lead to heart dysfunctions.(21)

Q. The body makes CoQ. How large of an impact is endogenous CoQ?

A. We have looked for years for an endocrine mechanism- we know that thyroid controls metabolism – maybe some control over thyroxine over metabolism and there, over CoQ, but we know the major stimulus for CoQ synthesis is the demand for energy and oxygen at the cellular level.

People who exercise produce more CoQ than those who are sedentary. Athletes produce a tremendous amount of CoQ where non-athletes have maybe half that. So we really think that exercise is the promoter of CoQ synthesis. When people stop on a daily basis their CoQ levels decrease rapidly.

CoQ is peak in the body at age 21. They decrease gradually and at the age of 65 most people are 45-50% low. Fortunately there are people who are above that – maybe 12% of the population have high levels and they age very slowly, never sick and live well in their tenth decades. Then we have a group that has early deficiencies of CoQ in early 40's and mid 50's and have early aging and have early age –related degenerated diseases – have high medical bills

Pharmaceutical Issues We know about the CoQ10 and statin drugs; many other interactions interfere with pharmaceuticals. The book by Ross Pelton "Pharmaceuticals that Interfere with Energy Synthesis" says: – we know of a hundred pharmaceuticals, especially the beta blockers, the diabetic drugs, antidepressants, cardiac drugs, statins, that interfere either with CoQ synthesis or the CoQ in the electron transport system to produce energy. This is why the most common clinical symptom complaint among people who are on a lot of drugs is, "Doctor where is my energy? I have no energy." Often it is the

drugs and the amount of drugs interfering with nutrition. And CoQ – by all means-- is nutrition

On the other hand, we know very little interaction between CoQ and drugs except for one thing -- CoQ raises metabolism and therefore may increase metabolism of certain drugs in the body.

Note: AF patients on warfarin/Coumadin are often told to avoid Coenzyme Q10 as there is a warfarin interference. Dr. Judy says:

It's been shown in England (St. Mary's Hospital) that neither Co10 nor vitamin E interferes with the clotting processes. We have hundreds of patients on Coumadin who have taken CoQ for 20 years and have no blood clotting problems

Professor Gian Littarru(22) is recognized as the foremost Coenzyme Q10 researcher today. He observes that CoQ10 levels decline with age, peaking in heart tissue at ages 19-21 and declining continually. In the brain, tissue levels peak at age 45 and then decline.

He also notes that because of the high antioxidant properties, CoQ concentrations in LDL is prophylactic in the formation of atherosclerosis.

Important Aside: Statins block Coenzyme Q10 production.

Not for this discussion, but serious consideration should continue along the lines of questioning the wisdom of prescribing statins for the majority of the population as a "preventive" drug....given the preceding information about the importance of CoQ.

KEY NUTRIENTS #3 AND #4 CoQ10 AND CARNITINE

Dr. Sinatra says Coenzyme Q10 and carnitine work together and in his book, calls them the "twin pillars of heart health." He found that 15% of people given CoQ10 did not respond to it (were refractory to it), until he added L-carnitine and then he got a greater response.

Co Q10 ignites the spark that generates ATP, but L-carnitine is the energy shuttle that transports long-chain fatty acids to the inner membrane of the mitochondria where they are burned as fuel. The heart needs a lot of energy and the best source of energy-producing fuel is fat. Dr. Sinatra says, "What a lot of people don't understand, including researchers and cardiologists, is that more than 60% of the energy of the heart is generated from the burning of fat, not carbohydrates. The brain requires carbohydrates."

ATP is transported to various parts of the cell to supply energy. This energy is the vital force of life. Enzymes use ATP as chemical energy for enzymatic reactions.

Carnitines are created by the body to transport fatty acids to the mitochondria and remove toxins. L- Carnitine is not used in the body as a building block for proteins. It is made in the body using lysine as a precursor but is not classified as a vitamin. While L-carnitine is often classified as an amino acid, it isn't. It is closely related to amino acids, but technically it is a nitrogen-containing, short-chain carboxylic acid. The manufacture or synthesis is complicated and just as we are less efficient in producing our own Coenzyme Q10, we are also less efficient producing our own carnitine. Food sources of carnitine – red meats, especially lamb, are often avoided and blood levels of carnitine can become too low.

Carnitine is so important to the heart that heart cells have a special mechanism that concentrates L-carnitine from the blood, making heart levels significantly higher by using a special transporter called the OCTN2 carnitine transporter.(23)

Dr. Sinatra in his book and then a more recent (2006) interview with biochemist, Robert Passwater, PhD, (23) discusses the various forms of supplemental carnitine and the benefit of each. It's worth reading. He likes a combination of all. The newest member of the carnitine, propionyl- L-carnitine (PLC) targets heart tissue specifically.

In his chapter on Carnitine, Dr. Sinatra(2) says increased levels of carnitine accelerate energy metabolism and low levels impair it. Other critical functions of carnitine include the metabolism of

branched-chain amino acids, ammonia detoxification and lactic acid clearance from tissue. Strenuous exercise can result in high levels lactic acid which makes the blood and tissues too acidic. The toxic byproduct of protein and ATP catabolism, ammonia, is another factor in exercise-induced fatigue. Carnitine helps to combat ammonia poisoning by converting ammonia to urea which is excreted in urine.

He says there is a limit on dosing for carnitine and research suggests no significant advantage of using doses that exceed 2 grams at any one time because saturation of intestinal mucosa occurs at this dose. Maximum blood concentrations are reached in about 3.5 hours. The half-life in the blood is approximately 15 hours.

The propionyl-L-Carnitine form is a powerful vasodilator and helps open blood vessels which increases blood supply to heart, muscles and other tissues at times additional blood flow or oxygen are needed. A study showed that this form of carnitine is rapidly taken up in the heart, muscle, kidney and other tissues and when saturated, is excreted in urine. The PLC form of carnitine has a half life of about 1.5 hours indicating how quickly it is taken up and utilized by tissue.

Oral carnitine isn't fully absorbed so taking it three times a day is preferred. That not used in tissue will be excreted in urine.

More carnitine (at once) is not typically better since bioavailability actually decreases the more the dose increases.

Several studies using the PLC form of carnitine have found it improved exercise capacity and preserved heart function heart failure patients. Beside the effect on heart energy, it has antioxidant effects and prevents oxidation of heart-muscle proteins which is a major mechanism of heart damage. In these studies, the L-carnitine was used as they would use a drug; that is, alone. (Blaylock)(16)

Dr. Sinatra comments about the various forms of carnitine:

L-carnitine fumarate works well for the heart, because fumarate is a Krebs cycle component. He says you can probably get more mileage out of the fumarate over the tartrate because fumarate is a Krebs cycle component and is more easily assimilated or metabolized in cells that are struggling for oxygen.

Acetyl L-carnitine may be better for the brain, as it crosses the blood brain barrier.

Dosage varies with the patient and the ailment. I generally recommend doses of 250 to 500 mg of L-carnitine fumarate three to four times a day.

In Dr. Sinatra's book, he discusses a new form of Amino Carnitines which is a powerful combination formulated in a specific way...

Start reading here in his interview with Dr. Passwater about this form and why he thinks it's highly effective. (too lengthy to review in detail here)

http://www.drpasswater.com/nutrition_library/Part%20OnePLC.html

It's important to note what he says about checking thyroid when giving the higher doses of carnitine:

Sinatra: I have patients who go up to two to three grams of L-carnitine a day. When at this level, I check thyroid function, as high-dose carnitine could possibly interfere with iodination of the thyroid hormone and I have seen patients come down with a little hypothyroidism. Hypothyroidism is rampant in our culture today. It is, in fact, at epidemic levels, so I do watch thyroid levels when I have people on more than two grams of carnitine a day. (from the Passwater interview).

KEY NUTRIENT #5 RIBOSE

The mitochondria within each cell convert fuel into energy and it requires a steady supply of d-ribose to do so but they first have to manufacture it. D-ribose is very different from glucose or any other sugar. It's found in red meat yet red meat consumption is discouraged. It only lasts for 30 minutes in the bloodstream so a continuous supply is needed. Damaged hearts or oxygen-deprived hearts can't make enough. So, the ATP supply suffers.

In 1973, German physiologist Heinz-Gerd Zimmer of Munich found damaged hearts recovered faster when ribose was given immediately following a heart attack (24) so it's obviously not a new discovery. There are over 100 clinical and scientific studies on the benefits of ribose, yet very few afibbers learn it about from their physicians or cardiologists.

There is a good deal of information on the use of ribose and fibromyalgia/ chronic fatigue syndrome. A Google search on ribose produced 174,000 hits. In the archived bulletin board posts, several threads on the use of ribose can be found and are listed in the reference section.(26) I've found ribose to be extremely helpful when added to my supplement protocols. Some afibbers have found it is too stimulating so once again, we are all experiments of one so proceed with small doses and first and increase gradually.

Ribose is a five-carbon sugar that is a regulator in the production of ATP and is a carbohydrate that is the backbone of genetic material such as RNA and is needed in the production of many metabolic compounds.

Note that ribose does not raise blood glucose levels, rather, it may lower it so it's advisable to take with food.

The heart's ability to maintain energy is limited by one thing-- the availability of ribose.

Ribose increases tolerance to cardiac stress.

Ribose improves exercise tolerance and physical function.

Ribose provides cardiac energy needed to maintain normal heart function.

Ribose increases cardiac efficiency and lowers stress during exercise.

Ribose maintains healthy energy levels in heart and muscle.

My initial ribose research was from published data by Bioenergy Life Sciences (25) and their Corvalen patented ribose product. At the time Clarence Johnson was a key researcher. Following is an important observation by Dr. Johnson.

What will ribose do for someone who exercises on a regular basis?

Scientific research shows that three or four workouts per week may not allow enough rest time between sessions for heart and muscle energy pools to return to normal levels.

Taking Ribose shortens the time needed by heart and muscle tissue to replace energy that is lost through vigorous exercise. Keeping energy pools full helps to keep heart and muscles in good physiological condition, increase power and endurance, and reduce fatigue. Recent research has also shown that ribose supplementation during exercise reduces free radical formation and lowers cardiac stress associated with hypoxia.

Dr. Johnson says: "In normal healthy hearts that have been stressed with ischemia or hypoxic insult, research shows that it takes more than 10 days to fully recover the energy charge. That's in a normal healthy heart. In muscle, we know it takes more than 3 days because we've done all the studies to show the ATP recovery rate in skeletal muscle following exercise and we know it to be greater than 3 days.

So, with a chronic case of oxygen deprivation, like in heart disease or what I call chronic exercise – when people exercise every day or when high intensity exercise every day or every other day, and the oxygen deprivation is repeated, the tissue simply can't make ribose fast enough to ever recover. [note – emphasizing "ever"]. That's why for example in congestive heart failure, the energy level of the heart continues to go down and down until the patient finally dies.

The referenced previous forum post links offer a good review on the function of ribose and how it is of benefit to afibbers, especially after a prolonged afib event. (26)

(Roberts) (2) .."while coenzyme Q10 and L-carnitine improve ATP recycling in the mitochondria, it is D-ribose that accelerates energy synthesis and refills depleted energy pools."

Dr. Sinatra uses ribose in his metabolic cardiology protocol along with the long-chain omega-3 fats and, L-carnitine to transport them to the inner membrane of mitochondria, coenzyme Q-10 to spark the production of ATP and D-ribose to help produce ATP. This improves the efficiency of the tissue as well as the heart as a whole.

[With the afibbers group, we have found it is often very helpful to also include supplemental potassium as an essential nutrient as well when the focus is specifically managing AF.]

Stress is a major player influencing AF. It's rare today not to have some degree of stress. A complete B complex supplement is especially important in the afibbers regimen as well.]

Essential Nutrient #7 Omega 3 Essential Fatty Acids or fish oil

No discussion on heart health and arrhythmia would be complete without mentioning that the Omega 3 essential fatty acids should be included on a daily basis along with a Gamma E to prevent oxidation of the oil once it reaches the tissue.

Choose a reputable brand that assures a pharmaceutical grade, molecularly distilled product that is assayed for purity and is free of heavy metals, PCBs, dioxins and other toxins that fish accumulate. Some of the brands known for purity and reliability include Coromega, Metagenics, Designs for Health, Natural Factors, Nordic Naturals, Carlson's. Check the label to choose the product that delivers the maximum dose of EPA content to help cut down on the number of doses to reach the recommended daily intake. The Omega 3's are known to help prevent arrhythmias. A consideration as to one way this may occur is by improving the quality or health of the phospholipids layer – the outer membrane layer of each cell. When this outer layer becomes stiff or distorted due to damage from trans-fats, the nutrient receptor sites are damaged or crimped and critical nutrients can't access the interior of the cell to function. In this case, we think of magnesium or potassium and what that would mean in terms of fostering LAF.

Keep in mind that when AF does not respond to nutrients, it could be the damaged receptor sites so 3 – 4 months of Omega 3 supplementation could be of benefit.

People who are insulin resistant are found to be deficient in magnesium. Omega 3's play an important role in reversing insulin resistance by restoring magnesium's intracellular access. In my case, I was insulin resistant as a result of beta blockers first prescribed for afib and my FM MD directed me to take 4 – 6 grams N-3 daily...which I continue to do with no adverse effects.

Omega 3's are known to improve hypertension and it's likely because of the cell membrane and damaged receptor sites... magnesium especially...when the cell envelope is repaired, then the Na/K pumps can function properly to lower sodium.

The US FDA has set an upper limit of safe EPA + DHA intake at 3000 mg/day. The US prescription version of Omega 3's Lovaza is labeled for lowering high triglycerides at a dosage of 4 g/day.

Barry Sears, PhD – Omega 3 expert and author of the Anti-inflammatory Zone (about Omega 3's) indicates that 10 grams daily of N-3's in studies have reversed bipolar disorders. He says Eskimos regularly consume 7-10 grams daily and have no depression compared to New Zealanders who have 50 times more depression.

A good collection of study reviews on the benefits of N3's and arrhythmia is this link:

<http://www.oilofpisces.com/arrhythmias.html>

<http://www.oilofpisces.com/> - index

<http://www.theheart.org/article/1013007.do>

http://www.cbn.com/health/NaturalHealth/drsears_depression.aspx

Omega 3 Fatty Acids are found to alter genetic actions.(27)

This can be of great significance with afibbers as N-3's are known to prevent arrhythmias. Current research indicates it is not DNA itself that determines how or what genes will be expressed. The real story is what we expose to our DNA through diet and lifestyle. In a nutshell, we are what we eat and what we are exposed to in our environment that directly affects our DNA and its expression. The growing field of epigenetics studies how diet, lifestyle, environment and even thoughts determine our health, rather than DNA alone. Nutrigenomics studies how food and nutrients in particular modulate (turn on and off) gene function. Together, they are among the most interesting areas of research into how EPA and DHA, the Omega 3 essential fatty acids work in the body.

Recent studies (27) have shown that EPA and DHA are genetically associated with cardio-vascular health outcomes and that supplementing with fish oil altered the gene expression profiles in cells to a more anti-inflammatory and anti-atherogenic status.

GENETIC MUTATIONS - LINK TO AFIB

There are afibbers who will not have success no matter how diligent they are with the protocols described here. That may be linked to a genetic mutation. Dr. Burford-Mason mentioned in her teleconference the following observations. I won't take additional space to discuss further

A study published April 2009, found a small segment of the population have genetic mutations that cause them to have very low levels of magnesium (Mg²⁺), which can cause altered heart beats, seizures, and involuntary muscle contraction.

A heterozygous KCNA1 A763G mutation is causative for hypomagnesemia

Title: *A missense mutation in the Kv1.1 voltage-gated potassium channel encoding gene KCNA1 is linked to human autosomal dominant hypomagnesemia .*

Published in Volume 119, Issue 4 (April 1,2009) *J. Clin. Invest.* 119(4): 936-942 (2009).

<http://www.jci.org/articles/view/36948>

While this is a small study segment and the magnesium deficiency stems from a kidney problem due to the gene mutation, Dr. Burford-Mason indicated there are six other genetic mutations that should be considered. For those who don't respond to The Strategy, it's certainly reasonable to consider the genetic connection for afib regarding magnesium deficiency or inability to retain a proper balance of electrolytes.

She says: The various wasting syndromes such as Gitelman's and Barter's can certainly be linked to arrhythmias, given the problem with retaining magnesium and potassium. Apart from GS, an autosomal recessive disorder, several other genetic magnesium-wasting syndromes have been identified.⁶¹ Intestinal magnesium wasting is associated with the *TRPM6* gene, which encodes an apical epithelial magnesium-conducting channel expressed in the intestine and in the kidney. Intra- and extracellular magnesium levels are associated with the major histocompatibility complex (HLA). Individuals possessing HLA B35 genes have higher red cell and plasma magnesium, and HLA-B38 positive individuals have lower levels compared to noncarriers of either gene.⁶² In GS, multiple mutations in the *SLC12A3* gene, which encodes the thiazide-sensitive sodium chloride cotransporter, have been identified.⁶¹

61. Knoers, N.V., deJong, J.C., Meij, I.C., Van Den Heuvel, L.P., and Bindels, R.J., Genetic renal disorders with hypomagnesemia and hypocalcuria, *J. Nephrol.*, 16, 293-296, 2003.

62. Henrotte, J.G., Pla, M., and Dausset, J., HLA- and H-2-associated variations of intracellular and extracellular magnesium content, *Proc. Natl. Acad. Sci. USA*, 87, 1894-1898, 1990.

A few of many studies on the genetic wasting syndromes:

Hypokalemic metabolic alkalosis: apropos of a case of Gitelman's syndrome.

PubMed ID: 15219074

Gitelman's syndrome revisited: an evaluation of symptoms and health-related quality of life.
11168953 [PubMed]

Inherited forms of Hypomagnesia – an update:
<http://www.springerlink.com/content/d448v7p83263m5q2/fulltext.pdf>

New molecular players facilitating Mg²⁺ reabsorption in the distal convoluted tubule
Kidney International (2010) 77, 17–22; doi:10.1038/ki.2009.358; published online 7 October 2009
<http://www.nature.com/ki/journal/v77/n1/full/ki2009358a.html>

Inherited Disorders of Renal Magnesium Handling
<http://jasn.asnjournals.org/cgi/content/full/11/10/1937>

MAGNESIUM TESTING

On Magnesium testing, Carolyn Dean, MD author of *The Magnesium Miracle* says:

“A serum test for magnesium is actually worse than ineffective, because a test result that is within normal limits lends a false sense of security about the status of the mineral in the body. It also explains why doctors don't recognize magnesium deficiency; they assume serum magnesium levels are an accurate measure of all the magnesium in the body.”

Monitoring magnesium levels. Burford-Mason(5)

She doesn't think it is worth doing blood tests or Red Blood Cell magnesium testing. She says: The Mg doesn't stay put in a red cell once the red cells is formed so it isn't a long-standing indicator of Mg status – Mg is only 1% of blood; the rest is divided between bone and other tissue and about 30% in bone can be quickly released and dumped into blood if needed for balance if that 1% goes down.

The balance of magnesium and calcium for cardiac function is so vital.

No studies have shown that red blood cell magnesium or serum Mg either one are good reflectors of whole body magnesium status. In fact if you talk to biochemists in hospital laboratories, they'll tell you the only people who ever have a low serum magnesium are those who are very, very ill or are in bed all the time. Physical activity recycles magnesium out of bones as well.

If extremely low – be concerned but if high or mid-range, it doesn't tell much because 5 minutes later, it can be completely different. So it's much easier – no waiting on lab results -- to just question patients and you can determine from physical signs – all related to full function or mal-function of skeletal, smooth or cardiac muscle. If they have constipation, muscle cramps, fasciculations, eye lid twitches, you just know they need more magnesium.

She likes to start patients on low doses (100 mg) of magnesium glycinate before bed and titrate up gradually over time in divided doses throughout the day so they have 2 – 3 soft bowel movements a day...sometimes those with none of the outward symptoms may be because vitamin D is still too low and masks the symptoms. It's important to treat for low D levels along with magnesium.

How high? – in cases of long-standing constipation, be sure that there is no structural or functional bowel disorder. Some people will tolerate 500 mg of magnesium glycinate twice a day and some use 3 + 3 doses.

People become aware of dosing and knowing how much they need they are absolutely adamant about how much they need and one thing they will never give up is their magnesium... even 5 years later, she observes.

EXATEST

The gold-standard and only known reliable measurement of intracellular electrolytes is by Exatest. Visit the website: www.exatest.com – check the studies and information so you can get your

physician to order. It's covered (for now) by Medicare and some insurances. It's best to phone or email for information. Knowing the ratio of the various minerals, Sodium to Potassium, Magnesium to Calcium is especially useful when managing stubborn cases of LAF.

GETTING STARTED WITH SUPPLEMENTS

(Reminder: this assumes healthy kidney function when taking Mg and K+)

Magnesium

Choose the amino acid chelated form of magnesium glycinate identified on the label as an Albion patented product. Check label to be sure it is not a combo product including calcium or other minerals. This amino acid glycinate has an 87% absorption rate. Typically, these capsules will be 100 or 150 mg.

The protocol is a ramping up or titrating up the dose from a small amount – one capsule a day...increasing every 4 – 5 days --- very slowly to allow for tissue saturation without causing bowel intolerance. The chelated amino acid form is specifically formulated not to cause bowel intolerance until dosing levels are quite high. The goal dose is 600 – 800 mg daily in divided doses. This form of magnesium does not depend on stomach acid for metabolizing so it can be taken with or without meals. It's sometimes easier to remember if taken with meals.

Start with a low dose and titrate up very slowly so the tissues acclimate to the magnesium and eventually become saturated... this will be signaled by two soft bowel movements a day. Obviously, back down to a lower dose if diarrhea occurs. Diarrhea is counterproductive because electrolytes are lost.

Example - start one 100 mg capsule at bedtime...for 4 – 5 days....then:

Increase to another capsule at breakfast - continue 4 – 5 days,

Add another 100 dose at lunch; continue 4 – 5 days;

Add another 100 at dinner making a total of 400 divided over the day and evening.

Then increase again starting with 200 at the nightly dose - wait 4 – 5 days etc.

Always be very aware of loose stools and cut back immediately. You may have to coast a bit on a lower dose before trying to increase again... but eventually, you'll find a dose that works without causing bowel intolerance. Everyone is in different stages of magnesium depletion and usage, so it's simply a matter of trying and keeping track.

The very worst thing to do is take a large amount of magnesium all at once. That doesn't accomplish the goal. It takes about 120 days to recycle old blood cells with new so results many not happen quickly.

This dosing and ramping up process may take a very long time – in some people as long as 6 months or more. Mg wasters never reach optimal repletion.... It's like trying to fill a bucket that has holes in it...you can never fill to overflowing. This wasting does happen in some afibbers and they find they need to take very large doses of magnesium supplements in order to achieve the NSR goal. Remember that if there are bowel absorption issues or interference... such as Candida overgrowth or the damaged magnesium receptor sites, optimizing magnesium may not be possible until the physical interferences are resolved first. If there is a gene mutation interference, that complicates progress.

Other forms of magnesium are more apt to cause loose stool much faster than the glycinate form...however, some afibbers like to add magnesium citrate which is very highly absorbable, but it just doesn't stay in tissue very long and it is used commonly as a laxative, so be aware.

Dr. Burford-Mason's comments about the citrate form of magnesium:

My observation of that was that magnesium citrate is well absorbed but not well-retained and that's when I changed to magnesium glycinate and found a very different clinical response.

Food sources of magnesium such as raw almonds offer additional intake, but for the most part, it's difficult to get therapeutic amounts of magnesium needed by afibbers from food sources....not impossible, but difficult.

For a convenient, economical and easy way to continually take in small amounts of magnesium every day, many of us use the homemade version of the patented bottled magnesium water sold in Australia called Unique Water. Erling Waller tinkered with the formula and developed a recipe for making this ionized magnesium water which we dubbed "Waller Water" or WW. It's easy to make and add to all your drinking and cooking water. That way, you get small amounts on a continuing basis. It's mildly alkalizing and it makes my well water taste wonderfully smooth.

Waller Water Recipe: http://www.afibbers.net/forum/read.php?f=3&i=7760&t=7752#reply_7760

Transdermal Magnesium Oil... These topical gels also offer a convenient and fast way to get magnesium into the body. For sore muscles, they are amazingly effective. I love using the gel on my neck or shoulders after a long session at the computer.

This is magnesium chloride and it has an oily, slippery feeling when applied but really isn't an oil. It works very quickly...Dr. Burford-Mason observes in as little as 5 minutes. When she has tense patients, she has them add a quarter size drop to the inner arm fold opposite the elbow. The patients are always amazed as how quickly they are relieved of tension and anxiety. The gel can be washed off in about 20 minutes. There is also a liquid, oral form (MagChlor) that can be used effectively.

She says: Magnesium chloride compounded in the liquid, oral form helps with 'super anxious' people. Once they use it, it's impossible to take them off. They just want it. It seems to affect neurotransmitters and brain health very quickly. So in my office experiences with the topical magnesium chloride on their arms or legs, I see people relax quickly if they are very uptight. It's quite remarkable.

Epsom Salt Baths are another form of transdermal or topical delivery. This is the magnesium sulfate form. Many people who don't tolerate oral supplements well find that either the topical oil or the Epsom salts baths offer a solution to adding magnesium. The baths, especially, at night are very relaxing. Directions say to soak for about 20 minute in warm water to which 2 cups of the magnesium sulfate salts have been added. This is very inexpensive and almost always readily available at the local pharmacy. A quick version would be a foot soak – warm water and half cup of the salts. Be aware that the bowel intolerance issue can surface with the baths, so it's best not to over do.

Potassium

It's very important to remember that in order for potassium to be effective in lengthening the refractory period, the intracellular stores of magnesium need to be very good if not optimal--first. Adding potassium too soon to the protocol can make afib worse instead of better. So don't rush adding potassium.

By law potassium supplements are limited to 99 mg/tablet or capsule. Professional grade products can supply higher doses. There is an option we find useful and that is the bulk powdered version – potassium gluconate – NOW brands, available through the web vitamin link at afibbers.org.

One teaspoon of the powdered potassium gluconate equals 540 mg. Typically, we use 1 to 3 or 4 teaspoons a day... added to water. Some of us put the powder in an empty salt shaker and add it to food for a very mildly salty addition. Hans developed a PAC- Tamer recipe using this gluconate powder.

<http://www.afibbers.org/conference/session38.pdf>

The gluconate form is the least likely to cause gastric distress, but be aware that some afibbers report it causes some problems. It's suggested that it be taken along with meals to help avoid that.

The Rx version is Potassium chloride with higher doses and sustained release forms is especially irritating to stomach tissue.

We often recommend using Low Sodium V8 or Campbell's Low Sodium Tomato juice as a convenient source of potassium. This has low sodium but added potassium chloride. Because of the stomach irritation issue, one should be aware of using too much at one time, but it does deliver

a significant amount of potassium. It's a good tip to remember if you need potassium in a hurry as in traveling. (Check the label for potassium content of each serving).

As a precautionary measure, I always carry potassium supplement tablets with me – typically potassium citrate... for when I eat in restaurants. I always eat as plainly as possible to avoid overly salted sauces or seasoning, but sometimes, I get surprised... so I'll take a preventive dose of 5 or 6 tablets while I'm eating just to be sure.

The daily requirement for potassium is around 5 grams. It's suggested most of this come from food, but often we don't eat regularly or well enough to get to that level. Probably the most significant influence in afib... at least for me... is slacking off on optimizing potassium intake.... or there is a slight kidney dysfunction that allows wasting.

Each afibber will need to experiment with potassium supplementation and find the dosing combination in addition to potassium-containing foods to maintain NSR. Go back and review the importance of the K factor and keeping dietary potassium at a high ratio compared to sodium.

Just remember that if you have a significant amount of PACs or can feel your heart beating when you lie down at night, it's probably lack of potassium.

Taurine

Bulk powder or capsules, most afibber find benefit of taking from 1 – 4 grams a day (1,000 – 4,000 mg) in divided doses. See previous text about potential stomach irritation.

There has been discussion about not taking with aspirin although Dr. Braverman indicates that a study found taurine powder protects the stomach and liver from aspirin-induced irritation in some circumstances. Experimenting and taking the aspirin far apart from the taurine would seem reasonable.

The bulk powder is somewhat difficult to dissolve. I just place ½ teaspoon of the powder in my mouth, chew on it a bit to mix with saliva and wash down with water. Tasteless and easy to do.

Eric Braverman says in his book (20)--effective doses range from 1 mg to 5 grams orally without significant documented risk. Intravenous doses up to 20 grams have been used.

The maximum safe dose has not been established.

Coenzyme Q10

There are food sources of CoQ10 but most people don't eat them regularly or enough... The highest is sheep mutton; lamb and beef offer modest amounts.

The newer forms of CoQ supplements are made more absorbable. The ubiquinol form is recommended as being the most absorbable. I use a combination.

It's suggested that you take CoQ in divided doses throughout the day and that they be taken with a fatty food. I usually take some with my Omega 3's.

Dosing as suggested by Dr. Sinatra in his book (p 75)

90-150 mg daily preventive for cardiovascular or periodontal disease

180-360 mg daily for arrhythmia, angina, hypertension, and those taking statins

300-600 mg daily for mild to moderate congestive heart failure

....360-600 mg daily for severe congestive heart failure and dilated cardiomyopathy

....166 – 1200 mg daily for improvement in quality of life in Parkinson's disease

For severely impaired immune systems, as in cancer, even higher doses may be required.

Note that the newer forms of the highly absorbable CoQ enable us to cut down somewhat on the dosing. Again, this requires some experimenting. Some hearts are going to need more than the standard dosing. I am taking 400 mg of the nano-particle version and it is working well for my overall energy deficit and mitochondrial dysfunction problem.

Carnitine – refer to the previous section on Carnitine.

Ribose

The suggested dosage for ribose is 5 grams twice a day, before and after exercise.

In cases of severe muscle fatigue as in Chronic Fatigue Syndrome and Fibromyalgia, the regimen begins with 15 grams a day in divided doses for about 6 weeks and then adjusting down to the 10 grams.

Some heavy exercisers and runners like to add ribose to their water bottle.

As mentioned in the summary about ribose, some afibbers find it is very stimulating so as always, start with a low dose... perhaps just half a teaspoon or less as a start to see how you react to it.

I have found that ribose has made *the* difference in stabilizing my heart after ablation. (That may be biochemical individuality including my age at work here.)

Always check the product label to ascertain that the product you are buying is made according to the patented BioEnergy/Corvalen standards. Because ribose is a mildly sweet powder, it would be easy to add filler to it so you'd be cheated on value.

One teaspoon of the powder should yield 5 grams. Some products provide a scoop and indicate the dose is one or two scoops. Make sure it equals the 1 tsp. = 5 grams measurement. There are a couple of brands that are not meeting that standard.

Omega 3 Essential Fatty Acids – refer to the previous section on this nutrient

Some of the other nutrients found important for afibbers

- B complex
- Vitamin D3
- VITAMIN K2 MK7
- VITAMIN C
- VITAMIN E
- Nattokinase
- L-theanine for anxiety and stress.

See Hans' recommendations at the web vitamin link – intro page.

<http://www.afibbers.org/vitamins.htm>

BEGINNING INSTRUCTIONS FOR NEW READERS

1. Eat Paleo

(by eating Paleo, you avoid starchy carbs, gluten, alcohol and sugar – all known to be detrimental plus all packaged foods with chemicals, additives, preservatives & hidden salt etc. Eat only fresh foods. As much raw as possible.) Limit fruit (due to the fruit sugar) <http://paleodiet.com/>

2. Identify triggers and avoid (some are food sensitivities)

3. Avoid MSG and other chemicals, food additives and packaged food

4. Avoid "diet" anything – foods, beverages – artificial sweeteners are excitatory

5. Add key nutrients – Essential Trio Plus.

6. Hydrate well

7. Limit sodium

8. Limit calcium – at least initially until magnesium stores are optimal

9. Be aware that stomach irritation, gas, bloating and digestive issues are often triggers for afib. Typically, it's the lack of adequate stomach acid (HCl) and adding digestive enzymes, betaine HCl, or using DGL is found to be very helpful.. It's important to rule out Candida overgrowth or H.pylori infection. Both can be triggers.

10. It's always smart to know your levels of

a. High Sensitive or Cardiac C-reactive protein (inflammation)

b. Ideally, intracellular electrolytes via Exatest (www.exatest.com)

- c. Fibrinogen (indicator for blood viscosity)
- d. Ferritin
- e. Homocysteine
- f. Fasting glucose & insulin levels
- g. Hemoglobin A1-C
- h. Thyroid function – complete thyroid panel including antibodies, Free T3 and Free T4, Reverse T3. Hypo or hyper thyroid can trigger afib.
- i. Intestinal dysbiosis tests including Candida Albicans & H.pylori
- j. Adrenal Stress Profile
- k. Vitamin D levels

RECOMMENDED READING AND RESOURCE LINKS

The Sinatra Solution – Metabolic Cardiology
 Stephen T. Sinatra, MD, FACC, CNS, CBT
 Introduction by James C. Roberts, MD, FACC...
 Basic Health Publications North Bergen, NJ ©2005

The High Blood Pressure Solution with The K Factor
 Richard D Moore, MD, PhD (biophysics)
 Healing Arts Press. 1993, 2001

The Magnesium Factor
 Mildred S. Seelig, MD, MPH
 Andrea Rosanoff, PhD
 Avery Publishing – Penguin Group
 New York, NY © 2003

Energy and Defense
 Gian Paolo Littarru, PhD (biochemistry)
 Rome, Italy, 1995

The Coenzyme Q10 Phenomenon
 Steven T. Sinatra, MD, FACC
 Keats Publishing 1998

The Carnitine Miracle
 Robert Crayon, MS
 Evans & Company 2000

The Anti-Inflammatory Zone –
 Reversing the Silent Epidemic that's Destroying Our Health
 Barry Sears, PhD
 Harper Collins Publisher ©2005

Lone Atrial Fibrillation – Towards a Cure - Volume I
 By Hans R. Larsen MSc ChE
<http://www.afibbers.org/lafbook.htm>

Thrombosis and Stroke Prevention
 Hans R. Larsen MSc ChE
<http://www.afibbers.org/strokebook.htm>

All Conference Room Session Links offer important information. Try to read them all.
<http://afibbers.org/conference/index.htm>

Start with #14
<http://www.afibbers.org/conference/PCMagnesium.pdf>

<http://www.afibbers.org/conference/magnesium.htm>

Success of Paleo diet..

<http://www.afibbers.org/conference/session54.pdf>

Visit this afibbers support group website and lurk a while. <http://www.afibbers.org/>

Sign up for the subscription afibbers report – get the latest research summaries

<http://www.afibbers.org/subscriptions.htm>

REPORT RESOURCE REFERENCES

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(Erling Waller's story, CR Session #61, page 2)

(2) The Sinatra Solution: Metabolic Cardiology
Integrative Cardiologists Stephen T. Sinatra MD, FACC, CNS, CBT and
James C. Roberts MD, FACC

(3) David R. Van Wagoner, Ph.D., Director, Basic Cardiac Electrophysiology Laboratories
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[http://www.ncbi.nlm.nih.gov/pubmed/10888261?ordinalpos=&itool=EntrezSystem2.PEntrez.Pubmed.ResultsPanel.SmartSearch&log\\$citationsensor](http://www.ncbi.nlm.nih.gov/pubmed/10888261?ordinalpos=&itool=EntrezSystem2.PEntrez.Pubmed.ResultsPanel.SmartSearch&log$citationsensor)
J Mol Cell Cardiol 32, 1763–1766 (2000) doi:10.1006/jmcc.2000.1224, available online at
<http://www.idealibrary.com>

Ion Channel – Sodium/Potassium Pump web links

The Na⁺-K⁺-ATPase (Sodium Pump)

http://www.vivo.colostate.edu/hbooks/molecules/sodium_pump.htm

Pharmacological evidence for a role of ATP-dependent potassium channels in myocardial stunning
<http://circ.ahajournals.org/cgi/content/abstract/86/1/311>

The voltage-gated potassium channels and their relatives.

[Nature](#). 2002 Sep 5;419(6902):35-42.

PMID: 12214225 PubMed

Computer simulation of synchronization of Na/K pump molecules

[J Bioenerg Biomembr](#). 2008 Aug;40(4):337-45. Epub 2008 Aug 5.

PMID: 18679778 PubMed

(4) Function of Electrolytes

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(5) Aileen Burford-Mason, PhD, is an immunologist, cell biologist and lecturer with a deep interest in the evidence base for complementary and alternative medicine (CAM), especially as it applies to the use of diet and nutritional supplements in health maintenance and disease prevention. Designs for Health Teleconference http://www.holistichealthresearch.ca/AileenHB_bio.gk

(6) The Magnesium Factor (pp 15 – 19)

Mildred S. Seelig, MD, MPH

Andrea Rosanoff, PhD

Avery Publishing – Penguin Group

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 Eric R. Braverman, MD
 Basic Health Publications 1987, 2003
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 Richard Smayda, D.O. <http://www.mgwater.com/taurine.shtml>
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<http://www.bioenergy.com/store/Main.aspx?html=br>

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http://www.afibbers.net/forum/read.php?f=6&i=11441&t=11441#reply_11441 update

(original ribose post) Energizing Heart & Muscle Cells with D-Ribose

http://www.afibbers.net/forum/read.php?f=4&i=17456&t=17456#reply_17456
http://www.afibbers.net/forum/read.php?f=6&i=11441&t=11441#reply_11441
http://www.afibbers.net/forum/read.php?f=6&i=17607&t=17199#reply_17607
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The complete resource for Magnesium Research

<http://www.mgwater.com/>

This is Paul Mason's website and is a virtual collection of relevant magnesium data.

Nutrition in Foods Calculator

<http://www.nutritiondata.com/>

Keep current with the latest in Electrophysiology.

<http://www.eplabdigest.com/>

http://www.jafib.com/index.php?cont=current_issue

...

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