THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

NUMBER 26

FEBRUARY 2003

3rd YEAR



In this issue we report on the trial of the magnesium/bicarbonate (Waller) water and conclude that it may be effective in helping to reduce episode intensity. Whether or not it is likely to reduce episode frequency and duration as well seems to be a highly individual matter and one that needs individual experimentation to ascertain.

We also explore the connection between diet and LAF. Many afibbers have found that dietary changes reduced the frequency and severity of their episodes. Some have found it beneficial to eliminate all dairy and wheat products while others have noticed improvements after eliminating processed foods, trans-fatty acids, aspartame, and MSG (monosodium glutamate)

in all its guises. Yet others have found that avoiding potatoes, coffee, peaches, garlic, chocolate, alcohol, and brewer's yeast helped reduce the number of episodes. Two or perhaps three afibbers have completely vanquished afib episodes by dietary changes and judicious supplementation. I have analyzed the diets of the 3 "lucky ones" and discuss their common factors in this issue.

In addition, the second and final chapter of my own afib journey. You will learn about my initial success and subsequent disappointment with the anti-inflammatory protocol. Also about my battle with IBS, which turned out to be a lot easier to win than the battle with LAF.

If you haven't already done so, don't forget to get your copy of my recent book "Lone Atrial Fibrillation: Towards A Cure" at <u>www.afibbers.org/lafbook.htm</u> - it provides a wealth of information on dealing with LAF.

Enjoy!

Wishing you lots of sinus rhythm in 2003, Hans Larsen

Table of Contents

Magnesium/Bicarbonate (Waller) Water Trial The Diet Connection

- Survey results
- Evaluation of effective diets
- Electrolyte levels
- Proteins, carbohydrates and glycemic load
- Fish oils and fatty acid ratio
- Conclusion

My Afib Journey: Part 2 by Hans Larsen

Magnesium/Bicarbonate (Waller) Water Trial

Magnesium-rich mineral waters are easily absorbed and have many health benefits due not only to their magnesium content, but also because of their content of bicarbonate ions that help neutralize the carbonic acid formed in the body during metabolic processes.

Erling Waller, a former afibber, and Jackie Burgess, both frequent contributors to the Bulletin Board, collaborated to develop a recipe for homemade magnesium/bicarbonate water. The recipe is based on the reaction of magnesium hydroxide (in milk of magnesia) with plain carbonated water according to the formula $Mg(OH)_2 + 2CO_2 --> Mg(HCO_3)_2$. The magnesium/bicarbonate (Waller) water contains approximately 125 mg of elemental magnesium and 625 mg of bicarbonate per liter and has a pH of 8+.

A small trial was undertaken in October 2002 to determine whether consuming the water had a noticeable effect on the frequency or duration of LAF episodes and whether there were any significant side effects from consuming the water.

Fifteen afibbers participated in the trial (13 men and 2 women). The group consisted of 2 adrenergic, 6 mixed, and 7 vagal afibbers. The average age of the group was 59 years with a range of 44 to 73 years. Average age at diagnosis was 51 years with a range of 18 to 68 years. Twenty-one per cent (3 participants) were taking an antiarrhythmic drug and 14% (2 participants) a beta-blocker on a continuous basis. Three respondents (21%) were using an antiarrhythmic (propafenone, flecainide or quinidine) to terminate an episode "on demand". Thus 79% of respondents did not take any drugs on a continuous basis in order to manage their LAF and 58% never took any drugs for LAF management. Sixty-four per cent of all respondents got daily sun exposure (lucky sods!) and 71% either had daily sun exposure or supplemented with vitamin D.

Magnesium Intake

The survey respondents reported drinking an average of 1.4 L/day of magnesium bicarbonate water ranging from 750 mL/day to 2.5 L/day. Sixty per cent made the water as per the original recipe while the remainder had modified the recipe, usually by doubling the amount of concentrate in one liter of drinking water. The average elemental magnesium content in 1 L of drinking water was 238 mg with a range of 125 to 500 mg. Seventy-three per cent of respondents took additional magnesium supplements adding an average of 200 mg/day of elemental Mg to their daily intake. The total average intake of elemental magnesium was 388 mg/day (range of 114 to 1200 mg/day) in addition to that obtained from the diet. The participants had been consuming the water for an average of 11 weeks.

Diet

Nine (64%) of respondents consumed a standard American Diet, while the remaining 5 (36%) consumed a partially vegetarian diet. One respondent did not specify diet type. Ninety-three per cent consumed red meat and grains and cereals on a regular basis. Eighty-five per cent consumed eggs and 100% consumed fruits and vegetables every week (from 7 to 70 servings a week). Only 50% of the group consumed processed or fast foods on a regular basis. All respondents consumed beverages other than the magnesium/bicarbonate water on a daily basis. The intake of other beverages averaged 683 mL/day resulting in a total average fluid intake (excluding food) of 2.1 L/day.

Episode Severity

Three respondents were omitted from the evaluation of episode severity because of interference by atrial flutter, major medication changes or because of the lack of afib episodes during both the baseline and trial periods. The 12 remaining respondents experienced an average of 7 episodes during a typical base period month. The number of episodes experienced during the period when magnesium/bicarbonate water was consumed was 6.5. This difference is not statistically significant. Four participants (1 adrenergic, 3 vagal) experienced fewer episodes, 6 (3 mixed, 3 vagal) experienced more episodes, and 2 saw no change. The decrease in episode frequency ranged from 30 to 70% (average 47%) while the increase ranged from 20 to 200% (average 66%). It is thus clear that the effect of the water on episode frequency is highly individual.

The average duration of episodes was 15.6 hours pre-water and 15.2 hours during the water consumption period. This difference is not statistically significant. Five participants (2 mixed, 3 vagal) experienced a shortening of episodes, 5 (1 adrenergic, 1 mixed, 3 vagal) a lengthening, and 2 saw no change. The decrease in episode duration ranged from 7 to 63% (average 41%) while the increase ranged from 6 to 100% (average 31%). Again it is clear that the effect of the water on episode duration is highly individual.

Eight out of 11 respondents (73%) reported that the intensity (feeling of palpitations) of an episode was less after starting on the magnesium/bicarbonate water. The remaining 27% reported no change. This finding suggests that magnesium or bicarbonate somehow helps make the palpitations less noticeable. It is worth noting that the 2 respondents who had not noticed any change in intensity had quite a low total daily magnesium intake (114 mg and 250 mg/day respectively).

It is possible that magnesium may reduce episode intensity through its action as a natural calcium channel blocker[1-3]. This action would reduce heart rate and might result in a subjective feeling of lower intensity. There is also some evidence that an increase in serum magnesium level improves left ventricular ejection fraction – at least in heart attack patients[4]. An improved ejection fraction could possibly result in a "calmer environment" due to the fact that the heart would need to contract less forcefully if the ejection fraction was greater.

Seven of 12 respondents (58%) felt, on a subjective level, that the magnesium/bicarbonate water had indeed been beneficial in managing their LAF.

Other Benefits

Eight out of 14 (57%) respondents reported other benefits from consuming the water:

- Two reported drinking more water, thus achieving a beneficial increase in daily fluid intake.
- Two reported less heartburn.
- Two reported complete disappearance of nighttime leg cramps.
- One reported fewer ectopic (premature) beats.
- One reported an increased feeling of well-being, less joint pain and stress, and fewer mouth sores.

Side Effects

Only 4 out of 15 respondents (27%) reported side effects from consuming the water (2 respondents reported more than one side effect):

- Three reported looser stools at higher Mg concentrations.
- One reported bloating.
- One reported yeast infection in the corner of the mouth.
- One reported longer afib episodes (at 4 times the recommended Mg concentration).

Most side effects were reversible by reducing Mg concentration or discontinuing the water altogether.

Other Observations

- Users of pharmaceutical drugs did not have fewer or shorter episodes than non-users although "on demand" users did seem to have shorter episodes (not statistically significant).
- No associations were observed between episode severity and any particular food group, but it did seem (not statistically significant) that a more alkaline diet (lots of fruits and vegetables and less meat, grain and eggs) was associated with fewer episodes.
- Afibbers with the least number of episodes were more likely to report other benefits from consuming the water.
- Afibbers who, subjectively, felt that the water was beneficial also reported fewer side effects.
- Afibbers who received daily sun exposure were more likely to report a lessening of episode intensity than were afibbers who did not (vitamin D effect?)

Conclusions

All conclusions drawn from this study must be taken with a large grain of salt due to the small sample size (12-15 respondents) and the relatively short duration of the trial. Nevertheless, the following conclusions seem plausible:

• Magnesium supplementation (bicarbonate water or otherwise) may help reduce episode frequency and duration for some afibbers, but may increase episode severity for others.

- Magnesium supplementation would appear to decrease episode intensity in most afibbers, especially in those with a high daily intake (trend not statistically significant).
- No significant associations were observed between dietary factors and episode severity although there was a trend for a more alkaline diet (greater potassium intake?) to be beneficial.

An adequate daily magnesium intake is of paramount importance to everyone, but especially to afibbers. Drinking magnesium/bicarbonate water is an excellent way of increasing one's intake of highly bioavailable magnesium. However, whether drinking the water will beneficially affect episode severity can only be determined through individual experimentation.

References

- 1. <u>www.barttersite.com/magnesium.htm</u>
- 2. Yamaoka, K, et al. Temperature-sensitive intracellular Mg2+ block of L-type Ca2+ channels in cardiac myocytes. Am J Physiol Heart Circ Physiol, Vol. 282, No. 3, March 2002, pp. H1092-101
- 3. Gourgoulianis, KI, et al. Magnesium as a relaxing factor of airway smooth muscles. J Aerosol Med, Vol. 14, No. 3, Fall 2001, pp. 301-07
- 4. Suzuki, N, et al. Magnesium dynamics and relation to left ventricular function in acute myocardial infarction. Japh Circ J, Vol. 64, No. 5, May 2000, pp. 377-81

The Diet Connection

I believe there is a strong connection between diet and lone atrial fibrillation, but to my knowledge there is, at present, no medical or scientific evidence that such a connection does indeed exist.

Many afibbers have found that dietary changes reduced the frequency and severity of their episodes. Some have found it beneficial to eliminate all dairy and wheat products while others have noticed improvements after eliminating processed foods, trans-fatty acids, aspartame, and MSG (monosodium glutamate) in all its guises. Yet others have found that avoiding potatoes, coffee, peaches, garlic, chocolate, alcohol, and brewer's yeast helped reduce the number of episodes. Caffeine, alcohol and MSG seem to be pretty universal triggers, but it is likely that individual food sensitivities or allergies play a role as far as dairy, wheat, garlic, etc. are concerned.

A diet survey was carried out in October 2001 in an attempt to determine if there were any specific dietary components (meats, fruits, grains, etc.) that seemed to correlate with an increased or decreased episode frequency or severity.

SURVEY RESULTS

A total of 77 afibbers responded to the diet questionnaire. The results were intriguing but raised more questions than they answered. The number of responses simply was not sufficient to draw valid, statistically significant conclusions, especially when it came to looking for diet-related differences between vagal, adrenergic, mixed and permanent afibbers. In retrospect, it is also clear that the questionnaire itself left a lot to be desired (standardized serving sizes, more specific categories, etc.).

The questionnaire also enquired about blood type to test the possibility of a link between blood type and LAF or between blood types and optimum diet. Blood type A was most common among vagal afibbers (63%) while type 0 was most common among adrenergic (50%), mixed (58%), and permanent (71%). Is this of significance? I have no idea, but it is intriguing that vagal afibbers (mostly blood type A) are highly unlikely to turn into permanent afibbers (mainly blood type 0).

It is difficult to give an overall interpretation of the survey results. All the correlations listed below had a statistical significance of less than 0.05; that is, the outcome has a less than 5% probability of being due to chance – so presumably they should be significant.

- Vagal afibbers seemed to have fewer episodes if they avoided pork and omega-6 oils and emphasized fish and shellfish in their diet. Adrenergic afibbers had fewer episodes if they emphasized poultry and olive oil. Mixed afibbers had more episodes on a carbohydrate-rich diet, particularly one rich in whole wheat products, fruits and vegetables.
- Afibbers with blood type A tended to have more episodes if they emphasized whole wheat products, fruits and vegetables, and in general, ate a high carbohydrate diet. Poultry consumption seemed to decrease the number of episodes. There were no obvious dietary triggers for blood type O.
- Vagal afibbers who consumed a lot of vegetables appeared to have longer episodes while adrenergic afibbers who ate a lot of legumes had shorter episodes.
- Diet did not seem to affect episode duration for blood type A, but afibbers with type O tended to have shorter episodes with an increased intake of omega-6 oils.

There was a trend for adrenergic afibbers who emphasized poultry to spend less time in fibrillation while those emphasizing fruits and vegetables spent more. There were no clear dietary effects on total time spent in fibrillation for vagal and mixed afibbers.

Diet did not seem to affect time spent in afib for blood type A, but afibbers with type O spent slightly longer in afib if they also had digestive problems.

It would thus seem that pork, poultry, fish, vegetables, fruits and overall carbohydrate consumption could be important factors, but confounding by other variables such as age and drug use cannot be ruled out. It is conceivable that hormones in poultry could affect the autonomic nervous system, as could carbohydrates through the insulin spike caused by high glycemic index foods in particular. This, however, is pure speculation on my part and can only be confirmed or rejected via a much larger, better designed and professionally interpreted study.

EVALUATION OF EFFECTIVE DIETS

At least three afibbers (2 vagal and 1 mixed) have managed to completely eliminate afib episodes for periods of 6 months or more (maximum of 2 years and counting). They all ascribe at least part of their victory over LAF to major dietary changes. Two believe the diet changes were solely responsible for the improvement while the other credits his improvement to a combination of diet changes and complete amalgam (silver) filling removal and elimination of dissimilar metals in the mouth.

I obtained detailed 2-day dietary records from the three lucky afibbers and analyzed them for over 60 nutrients and nutrient ratios using the US Department of Agriculture Nutritional Database. The three diets had the following common features:

- A lower than officially recommended carbohydrate intake
- A lower overall glycemic load
- A higher than officially recommended protein intake
- Protein with every main meal and most snacks
- A high intake of fish oils
- A favourable ratio between omega-6 and omega-3 fatty acids
- A total water intake of 3.5 to 4 liters a day with at least 1 liter coming from pure drinking water
- A potassium:sodium ratio of 3:1 or higher or about twice the normal ratio

- A daily magnesium intake of more than twice the recommended intake (RDA)
- A calcium:magnesium ratio of about 1.5 or about half the normal ratio.
- A more than adequate intake of dietary fiber, vitamins and minerals with the exception of vitamin D where the dietary intake was seriously deficient in all cases
- Although I was not able to analyze the diets specifically for trans-fatty acids it would appear that they were all very low in this component.

An obvious question now is, is there any scientific evidence supporting that any of these common dietary traits may eliminate LAF?

A relatively high daily intake of water, dietary fiber, vitamins and minerals is clearly a good thing, but none of our LAF survey results have supported the idea that this in itself is likely to affect the frequency or severity of episodes. Could electrolyte (calcium, magnesium, potassium, sodium) levels, glycemic load, protein intake or fish oils influence LAF severity? Let us examine the evidence.

ELECTROLYTE LEVELS

The three effective diets all had a higher than normal content of potassium and magnesium and a higher than normal ratio between potassium and sodium and between magnesium and calcium. That a surplus of the "calming" ions (potassium and magnesium) should be beneficial makes instinctive sense and is supported by the fact that magnesium is known to possess strong antiarrhythmic properties.

Potassium

The average daily intake of potassium in the three diets was 5500 mg/day, substantially higher than the recommended intake of 3500 mg/day. The average potassium:sodium ratio was 3.3:1, which again is substantially higher than the recommended ratio of 1.5:1. A daily intake in excess of 3500 mg has to come from the diet. Standard potassium supplements usually only contain 99 mg per tablet, clearly not enough to make a noticeable difference. Fortunately, potassium is abundant in the food supply, especially in fruits and vegetables.

Magnesium

The average daily intake of magnesium was 670 mg from the diet plus an additional 120 mg from supplements. This is more than twice the recommended daily intake and about 4 times the actual intake of the average North American. The average ratio of calcium to magnesium was 1.5:1, substantially lower than the recommended 3.75:1. So it is clear that the three successful diets were much higher in magnesium than normal.

PROTEINS, CARBOHYDRATES & GLYCEMIC LOAD

The average daily protein content of the three diets was 135 grams or 25% of total energy. This is significantly higher than the officially recommended intake of 45-55 grams/day or about 13% of energy. However, it is probably close to the normal protein level in a typical Western diet. Of more significance may be the fact that all three diets provided some protein at each main meal and most snacks. Having protein with every meal ensures that insulin spikes are kept to a minimum and that an adequate amount of glucagon is produced so as to avoid wide hormone fluctuations. This is especially important for afibbers with a tendency to hypoglycemia and also helps lower the demand for cortisol.

Carbohydrates

The officially recommended intake of carbohydrates is about 60-65% of total dietary energy intake. This would correspond to a daily intake of about 300 grams of carbohydrates on a 2000 kcal/day diet.

The average carbohydrate intake in the three effective diets was 190 grams (adjusted to a 2000 kcal/day diet) or only about 63% of the recommended amount. In other words, the actual energy intake from carbohydrates was only 38% as compared to the recommended value of 60-65% - clearly a major and highly significant difference. Consuming carbohydrates causes the release of insulin and results in an increase in the level of the stress

hormone cortisol[1]. Just like some fats are good and some are bad, so are some carbohydrates good while others are bad. It all depends on the magnitude of the glycemic load they place on the body.

Glycemic Load

The term "glycemic load" was coined by researchers at the Harvard Medical School. The glycemic load of a particular food is the product of its glycemic index and the amount (in grams) of carbohydrate present in a serving of the food divided by 100. For example, a medium-sized apple has a glycemic index of 38 and a carbohydrate content of 21 grams, so its glycemic load would be 8. Proteins and fats contain no carbohydrates so their glycemic index and load are zero. Researchers at the Harvard Medical School reported in June 2000 that women whose food intake produced a high glycemic load were more likely to develop coronary heart disease[2]. More recently, Harvard researchers also reported that women whose glycemic load was high had a 40% higher risk of developing type 2 diabetes than did women who consumed a diet with a low glycemic load[3]. Other researchers believe that high glycemic indices and loads may be a risk factor for colon, breast, and prostate cancers[4].

The term "glycemic index" was first introduced in 1981 as a measure of the rate of absorption and conversion to glucose of various carbohydrate foods. The glycemic index of pure glucose was set to 100, and carbohydrate foods were evaluated against glucose by comparing the rate and extent (area under blood glucose versus time curve) to which 50 grams of the test food was absorbed and converted to glucose with the rate for 50 grams of pure glucose. Recently there has been a trend to use white bread as the standard (glycemic index = 100); under this scheme the glycemic index of pure glucose is 147.

Food with a high glycemic index produces a higher peak in blood glucose levels after a meal, and a greater overall blood glucose response during the first 2 hours after consumption, than do foods with a low glycemic index. This higher response is believed to be detrimental to health[4].

The average daily energy-adjusted (to 1700 kcal) glycemic load in a typical Western diet is probably around 105 while a desirable level would be about 70[5]. The average glycemic load of the three effective diets was 72 or, in other words, well below the typical level and close to the desirable level.

Whether a low carbohydrate and glycemic load diet will help prevent lone atrial fibrillation obviously needs to be determined in a large clinical trial or epidemiologic study. However, until this is done I see no disadvantage and many possible advantages for afibbers in adhering to a diet similar to that consumed by the 3 "lucky" ex-afibbers.

The simplest way to reduce glycemic load is to minimize the intake of high glycemic index foods like potatoes, rice, and white bread.

FISH OILS & FATTY ACID RATIO

The average daily fish oil intake in the three diets during the period when LAF was eliminated was about 2000 mg of EPA (eicosapentaenoic acid) and 2200 mg of DHA (docosahexaenoic acid). This is about 10 times the recommended daily intake, but well within the range of intakes that have proven to be of therapeutic value in diseases and disorders ranging from asthma to schizophrenia.

Fish oils are known to have a direct effect on the central nervous system and can block the entry of sodium and calcium ions into vascular smooth muscle cells. This effect, combined with the anti-inflammatory effect, may explain why fish oils help prevent ventricular arrhythmias and sudden cardiac death[6,7].

Researchers at the Massachusetts General Hospital believe that omega-3 fatty acids (fish oils) inhibit the flow of sodium and calcium into the heart cells by actually modifying the ion channels that control the inflow. As a result, a stronger electrical stimulus is required to initiate the process of contraction and the refractory (rest) period between heart beats is markedly prolonged[8]. Australian researchers have found that the composition of the membranes enclosing individual heart cells (myocytes) changes in animals fed fish oils and that this change is beneficial and may explain the antiarrhythmic properties of fish oils[9]. Finally, Canadian researchers have

found that DHA, a major component of fish oils, inhibits the inflow of calcium ions into myocytes without affecting their ability to contract[10].

All this research is leading edge and the actual effectiveness of fish oils in the prevention of atrial fibrillation in humans still needs to be proven in clinical trials. Nevertheless, I believe the evidence of possible substantial benefits to afibbers is there and am now supplementing with 1500 mg/day of EPA and 1000 mg/day of DHA. A fish oil intake of this magnitude has proven entirely safe in numerous clinical trials and should pose no problems provided the oils are non-rancid and free of mercury. It is quite possible that the optimum intake of fish oils may vary from person to person and that some afibbers may do better if a small amount of GLA (gamma linolenic acid) is periodically included with the fish oil supplementation[6]. Only experiments on a personal basis will resolve this question.

Omega-6 to Omega-3 Fatty Acid Ratio

Omega-6 and omega-3 fatty acids are essential in human nutrition. Seemingly minor differences in their molecular structure make the two EFA families act very differently in the body. While the metabolic products of omega-6 acids promote inflammation, blood clotting, and tumour growth, the omega-3 acids act entirely opposite. Although we do need both omega-3s and omega-6s it is becoming increasingly clear that an excess of omega-6 fatty acids can have dire consequences. Many scientists believe that a major reason for the high incidence of heart disease, hypertension, diabetes, obesity, premature aging, and some forms of cancer is the profound imbalance between our intake of omega-6 and omega-3 fatty acids. Our ancestors evolved on a diet with a ratio of omega-6 to omega-3 of about 1:1. A massive change in dietary habits over the last few centuries has changed this ratio to something closer to 20:1 and this spells trouble[11-13].

The average omega-6 to omega-3 fatty acid ratio in the three diets was 2.7:1. This is well below the "danger level" of 20:1 and even well below the currently recommended ratio of 7:1. Thus it would seem that a low omega-6 to omega-3 fatty acid ratio may well be a prominent feature of an effective afib-preventing diet, but obviously more research is required to prove this. The diets were also very low in trans-fatty acids, but again, more research is needed to see if this is important.

The main sources of omega-6 fatty acids are vegetable oils such as corn and soy oils that contain a high proportion of linoleic acid. Omega-3 fatty acids are found in flaxseed and walnut oils, marine plankton, and fatty fish. The main component of flaxseed and walnut oils is alpha-linolenic acid (LNA) while the predominant fatty acids found in fatty fish (such as salmon, mackerel, sardines) and fish oils are EPA and DHA, which are the most beneficial and active of these fatty acids. Alpha-linolenic acid (from flaxseed or walnut oils) can be converted to EPA and DHA in the body, but the conversion is quite inefficient especially in older people, diabetics, certain ethnic groups, and some children[11,12,14].

So the best approach to achieving a low omega-6 to omega-3 ratio is to limit the intake of vegetable oils and increase the intake of fish oils.

CONCLUSION

I believe dietary changes could well prove to be the most effective natural way of reducing or completely eliminating afib episodes. Not only have two, and perhaps three, afibbers been able to overcome afib by such changes, but also there are also some very good scientific reasons why this approach should work.

I believe all the modifications are important and that they interact to produce the end result. Having protein with every meal, avoiding tyramine-containing foods, reducing carbohydrate intake, and maintaining a low glycemic load would all contribute towards calming the autonomic nervous system and avoiding hypoglycemic episodes. Having an excess of magnesium and potassium in both intra- and extra-cellular fluids would probably help in avoiding the PACs that initiate episodes and a well-hydrated and alkaline environment may help as well.

Meanwhile the fish oils would be working quietly in the background to repair the damage caused to individual myocytes by years of afib. Thus, over time, instead of the situation where "afib begets afib" a situation would develop where "sinus rhythm begets sinus rhythm". Eventually the ion channels would be fully repaired and

functioning normally. The end result would be no more afib and a return to normal life. Utopia? Perhaps, but at this time I believe it is the best hope for those of us determined to find a non-pharmacologic, non-surgical solution to lone atrial fibrillation. We are all, as someone so aptly put it, "an experiment of one". So what may work wonders for some may not have much effect for others.

References

- 1) Gonzalez-Bono, E., et al. Glucose but not protein or fat load amplifies the cortisol response to psychosocial stress. Horm Behav, Vol. 41, No. 3, May 2002, pp. 328-33
- 2) Liu, Simin, et al. A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. American Journal of Clinical Nutrition, Vol. 71, June 2000, pp. 1455-61
- 3) Willett. Walter, et al. Glycemic index, glycemic load, and risk of type 2 diabetes. American Journal of Clinical Nutrition, Vol. 76 (suppl), July 2002, pp. 274S-80S
- 4) Jenkins, David J.A., et al. Glycemic index: overview of implications in health and disease. American Journal of Clinical Nutrition, Vol. 76 (suppl), July 2002, pp. 266S-73S
- 5) Brand-Miller, Jennie. Personal communication, October 3, 2002
- 6) Das, U.N. Beneficial effect(s) of n-3 fatty acids in cardiovascular diseases: but, why and how? Prostaglandins, Leukotrienes and Essential Fatty Acids, Vol. 63, December 2000, pp. 351-62
- 7) Christensen, Jeppe Hagstrup, et al. Heart rate variability and fatty acid content of blood cell membranes: a doseresponse study with n-3 fatty acids. American Journal of Clinical Nutrition, Vol. 70, September 1999, pp. 331-37
- 8) Leaf, A. The electrophysiologic basis for the antiarrhythmic and anticonvulsant effects of n-3 polyunsaturated fatty acids: heart and brain. Lipids, Vol. 36 (suppl), 2001, pp. S107-10
- 9) Honen, B.N. and Saint, D.A. Polyunsaturated dietary fats change the properties of calcium sparks in adult rat atrial myocytes. J Nutr Biochem, Vol. 13, June 2002, pp. 322-29
- 10) Ferrier, G.R., et al. Differential effects of docosahexaenoic acid on contractions and L-type Ca2+ current in adult cardiac myocytes. Cardiovascular Research, Vol. 54, No. 3, June 2002, pp. 601-10
- 11) Simopoulos, Artemis P. Omega-3 fatty acids in health and disease and in growth and development. American Journal of Clinical Nutrition, Vol. 54, 1991, pp. 438-63
- 12) Pepping, Joseph. Omega-3 essential fatty acids. American Journal of Health-System Pharmacy, Vol. 56, April 15, 1999, pp. 719-24
- 13) Uauy-Dagach, Ricardo and Valenzuela, Alfonso. Marine oils: the health benefits of n-3 fatty acids. Nutrition Reviews, Vol. 54, November 1996, pp. S102-08
- 14) Pawlosky, Robert J. Physiological compartmental analysis of alpha-linolenic acid metabolism in adult humans. Journal of Lipid research, Vol. 42, August 2001, pp. 1257-65

My Afib Journey: Part 2 by Hans R. Larsen

The year 2001 did not begin on a good note. By the end of March I had endured six episodes and spent 248 hours in fibrillation. Things were looking pretty grim. My diary entry for January 17th reads, "Life really is a pain!" One thing had become increasingly clear to me though and this was that my episodes were all triggered by emotional, mental or physical stress. It was also clear that most of them began between 3 pm and 5 pm.

During the first half of the year I continued my search for a supplement combination that would work, but did not come up with anything of obvious value. Whether things would have been worse if I had not supplemented is impossible to say. I also tried out some new exercise regimens and underwent Chinese "scraping" and magnet therapy for several months. It certainly made me feel better, but had no effect on the frequency or duration of my afib episodes. I did find that ginkgo biloba (60 mg twice a day) made me less depressed and as ginkgo is also an effective anticoagulant I decided to continue taking it and have done so to this day.

For awhile I took 12.5 mg atenolol at around 2 pm every day in order to protect myself during my vulnerable period between 3 pm and 5 pm. Unfortunately, I was not disciplined enough to take the atenolol every day so it did not have any overall beneficial effect.

In July I finally got the break that I had been searching for. I came across an article written by Dr. Andrea Frustaci, a cardiologist at the Catholic University in Rome[1]. Dr. Frustaci had performed biopsies on 12 patients with LAF and found that 8 of them had signs of a current or past inflammation of the heart lining. The inflammation was active in 3 of the 8 patients; they were treated with prednisone and had no episodes during the following 2 years. I corresponded with Dr. Frustaci and learned that it is quite possible that all the 12 LAF patients actually had signs of inflammation, but that the biopsy missed them in 4 of the cases[2]. Six months later independent teams of American and Greek researchers reported that LAF patients had significantly higher levels of the inflammation marker CRP (C-reactive protein) than did people without LAF[3,4].

After a careful study of Dr. Frustaci's work I decided to assume that I probably had an inflammation of the heart lining and set out to devise a scheme to eradicate it. In retrospect, I probably should have had a CRP test done before I began, but at the time it was not readily available and my physician was not familiar with it.

My approach to eliminating LAF was two-pronged – using dietary modifications and lifestyle changes to avoid "feeding" and constantly aggravating the inflammation and using natural supplements to dampen and heal the inflammation.

I am a type O blood type so I decided to follow Dr. Peter D'Adamo's diet for type O. The main features are total avoidance of all wheat and gluten-containing products, dairy products (except butter), kidney beans, lentils, peanuts, potatoes, eggplant, peppers, and a few other foods. Grains and cereals should be consumed in moderation (cornflakes, corn, oat bran, and shredded wheat should be avoided). The diet emphasizes protein in the form of lean meat, fish, and poultry and avoids pork, bacon, and ham. Other research has shown that a high intake of omega-6 fatty acids promotes inflammation. So I severely cut back on these types of fats (found in margarines and cooking oils) and increased my intake of omega-3 fatty acids (found in fatty fish and flax oil).

I began modifying my lifestyle quite a few years ago and continued with these changes. I avoid alcohol and caffeine and try to control both my emotional and physical stress levels. I took the following supplements daily for the purpose of dampening and eventually eliminating inflammation. These were in addition to my regular supplements.

1 hour before breakfast	Moducare	2 capsules[a]
With breakfast	Vitamin C Quercetin w/bioflavonoids Alpha-lipoic acid Pancreatic enzyme (Cotazym) Fish oil	1000 mg[b] 500 mg 100 mg 1 capsule 1 gram[c]
Mid-morning (on empty stomach)	Bromelain Curcumin (turmeric extract)	300 mg 600 mg[d]
1 hour before lunch	Moducare	2 capsules
With lunch	Quercetin w/bioflavonoids Alpha-lipoic acid Pancreatic enzyme (Cotazym)	500 mg 100 mg 1 capsule
Mid-afternoon (on empty stomach)	Bromelain Curcumin (turmeric extract)	300 mg 600 mg
1 hour before dinner	Moducare	2 capsules

With dinner	Quercetin w/bioflavonoids Vitamin C Pancreatic enzyme (Cotazym) Vitamin E Selenium	500 mg 1000 mg 1 capsule 400 IU 200 mcg
Before bed	Protec probiotic	2 capsules[e]

NOTES:

- a) Moducare should always be taken on an empty stomach 1 hour before or 2-3 hours after a meal. If it is inconvenient to take it first thing in the morning or if you have hypoglycemia you can take it 1 hour before bed.
- b) It is important to use timed-release vitamin C.
- c) The fish oil provided about 350 mg EPA and 230 mg DHA.
- d) If you find that curcumin irritates your stomach, take it with meals or discontinue it.
- e) The probiotic supplement contained the following:
 - 6.4 billion active L.rhamnosus
 - 0.8 billion active L.acidophilus plus
 - 0.4 billion each of B.longum and B.bifidum.

I began my anti-inflammation protocol on August 4th. I found it somewhat hard to completely eliminate all wheat and dairy products (except for butter), but eventually managed to do so once my wife found a great recipe for cookies made with quinoa flour! I now believe this was a very important first step. I also moderated my exercise program and increased my protein intake as per the blood type O diet. I had no problems taking the Moducare capsules on an empty stomach, but found the curcumin/bromelain combination to be quite irritating to the stomach so I switched to taking them with meals. I had originally included the herb Boswellia in my program, but found it gave me a headache so I discontinued it after 2 days.

Throughout the protocol I measured my pulse rate, number of ectopic beats over a 5-minute period, heart rate variability, and autonomic nervous system balance daily using a fingertip pulse monitor and software program (Freeze Framer) developed by the Heart Math Institute in California. On August 3rd I counted 15 ectopic beats (over 5 minutes) and my maximum power spectrum value was 156 milliseconds squared/Hertz – normal is about 8 to 30. So in other words, my heart's performance was rather chaotic.

A month later on September 3rd things had changed quite remarkably. I recorded no ectopic beats and the maximum power spectrum value had decreased to 12. I also felt great, but the balance between the sympathetic (adrenergic) and parasympathetic branches was still a bit unusual.

On September 19th I reduced my Moducare intake to one capsule three times a day instead of the two capsules three times a day I had been on since I began the protocol. Coincidence or not, I don't know, but early in the morning on September 21st I experienced my first LAF episode in 2 months. All veteran afibbers will know what a huge disappointment that was! I carried on with the protocol continuing to take three Moducare capsules a day, but reducing the curcumin/bromelain intake to just once a day (at lunch).

I then went 5 weeks without an episode. During this time I was regularly clocking zero PACs and PVCs, my maximum power spectrum value was around 28 and my autonomic nervous system balance was normal.

On November 29th I decided to discontinue the anti-inflammatory protocol for three reasons:

- I wanted to see what would happen if I went off the protocol.
- I was due to have a series of blood tests on December 3rd and wanted to make sure that the results were not affected by the protocol supplements.
- I wanted to do some more research on the supplements to make sure they had no long-term adverse effects.

Well, on December 4th I got my answer in the form of a 60-hour episode! This was followed by a 25-hour one on December 10th. So I now had the answer to what would happen if I went off the protocol!

In the meantime, I had heard from several afibbers who have found MSM (methyl sulfonyl methane) helpful. MSM has strong anti-inflammatory properties and is a cholinesterase inhibitor meaning that it enhances parasympathetic activity – just the ticket for adrenergic afibbers, but possibly not so great for vagal ones. MSM also crosses the blood/brain barrier and is reputed to bind to mercury and help excrete it. Sounded like a winner, so I decided to add 1000 mg of MSM (taken with breakfast) to the 3 capsules of Moducare (taken ½ hour before main meals).

I had also come across some very interesting information on American ginseng (*Panax quinquefolius*). Apparently American ginseng not only helps keep blood sugar levels under control, it also partially blocks sodium channels. This effect is similar to that of antiarrhythmics such as flecainide (Tambocor) and propafenone (Rythmol). Seemed like a promising candidate for the protocol so I decided to take 500 mg with breakfast. I should point out that only American ginseng has the above effects – Korean, Siberian and Chinese ginseng do not.

My personal anti-inflammatory protocol now consisted of 3 Moducare capsules per day plus 1000 mg of MSM and 500 mg of American ginseng. I want to emphasize that I have the adrenergic form of LAF and MSM may not be good for the vagal kind. I began the new protocol on December 15th and within 4 days it had completely eliminated ectopic beats and produced a very satisfactory HRV graph on the Freeze Framer. I had found no evidence that any of the components of the protocol have any long-term detrimental effects.

The Holiday Season also gave me the opportunity to confirm that I do indeed have a serious reaction to wheatcontaining foods. I normally avoid them, but with all the special cakes and cookies on offer during the holidays I confess I did "slip". Invariably I would end up with numerous ectopic beats about 12 hours later. So wheat in all its many forms is definitely no good for me.

Early in 2002 I received the results of my CRP test. The result was normal indicating that I did not suffer from an inflammation. During the first 2 months of the new year I had 5 afib episodes. The anti-inflammation protocol was obviously not working any longer and since I now knew for certain that I did not have an inflammation I decided to discontinue it altogether.

During the next few months I experimented with an herbal detoxification program, Dr. Andrew Weil's relaxed breathing program, chiropractic and osteopathic manipulation, and healing touch massage. All to no avail. To add insult to injury I was diagnosed with irritable bowel syndrome (IBS) in March. Since this was quite painful and uncomfortable I had to divert my attention from finding a solution to LAF to finding a solution to IBS. Fortunately, this proved to be a lot easier. Avoiding certain foods, taking Metamucil (not the aspartame-containing version) before meals, dealing with the pain by taking enteric-coated peppermint oil capsules and faithfully listening every day to a self-hypnosis tape by Mahoney[5] essentially cured me of the IBS in a little under 4 months. I no longer need the Metamucil or the peppermint oil, but still listen to the tape as it is very relaxing. I wish finding a cure for LAF was this easy!

In July I began investigating the connection between cortisol/DHEA levels and afib episodes. I had my cortisol and DHEA levels measured (in saliva) and while my cortisol level was fairly normal, although low at around 4 pm, my DHEA level was way off the lower end of the scale. Unfortunately, since I live in Canada where DHEA is a banned substance and importation exposes one to the risk of obtaining a criminal record there is not much I can do about this aspect of my research findings. However, for those of you who live in the US the DHEA connection would definitely be worth looking into.

I am now trying to model my diet on that of the three "lucky ones" and hope that this will be the answer. Time will tell. At least this approach has renewed my hope and, as all fibbers know, hope is one of the most important ingredients in coping with afib.

References

- 1) Frustaci, Andrea, et al. Histological substrate of atrial biopsies in patients with lone atrial fibrillation. Circulation, Vol. 96, August 19, 1997, pp. 1180-84
- 2) Frustaci, Andrea. Personal communication to Hans Larsen, July 23, 2001
- 3) Chung, Mina K., et al. C-reactive protein elevation in patients with atrial arrhythmias: inflammatory mechanisms and persistence of atrial fibrillation. Circulation, Vol. 104, December 11, 2001, pp. 2886-91
- 4) Dernellis, J. and Panaretou, M. C-reactive protein and paroxysmal atrial fibrillation: evidence of the implication of an inflammatory process in paroxysmal atrial fibrillation. Acta Cardiol, Vol. 56, No. 6, December 2001, pp. 375-80
- 5) The IBS Audio Program 100 by Michael Mahoney is available on CD or audiocassette at http://www.ibsaudioprogram.com

THE AFIB REPORT is published monthly by: Hans R. Larsen MSc ChE, 1320 Point Street, Victoria, BC, Canada, V8S 1A5 E-mail: editor@afibbers.org World Wide Web: http://www.afibbers.org Copyright 2003 by Hans R. Larsen

THE AFIB REPORT does not provide medical advice. Do not attempt self-diagnosis or self-medication based on our reports. Please consult your healthcare provider if you are interested in following up on the information presented.