

THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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Editorial

In this issue we continue with the evaluation of the LAF survey results. Thanks to the contribution of almost 100 afibbers we now have some meaningful data to work with. Part VI of the results deals with the benefit, or otherwise, of taking antiarrhythmic drugs. We also cover some fascinating new research concerning the association between inflammation and LAF. Read on!

*Yours in health,
Hans Larsen*

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SURVEY RESULTS – PART VI

Effect of Drugs (Antiarrhythmics)

Data was available for 79 lone afibbers with the paroxysmal (intermittent) variety and for 20 with the chronic variety. There were 35 vagal, 24 adrenergic, and 20 mixed variety in the paroxysmal group. Overall the 29 paroxysmal afibbers not taking any drugs spent an average of 107 hours in fibrillation over the 6-month survey period. The 50 respondents taking drugs spent an average of 125 hours in fibrillation. This difference, although not statistically significant ($p=0.6967$), does not support the contention that antiarrhythmic drugs are uniformly beneficial for LAF patients. Afibbers on drugs had more episodes (over 6 months) than afibbers not on drugs - an average of 22 versus 12. Conversely, afibbers on drugs had shorter episodes (average duration of 11 hours) than did afibbers not on drugs (average duration of 17 hours). These differences were not statistically significant.

My explanation of this finding, not substantiated by any other evidence that I am aware of, is that some antiarrhythmic drugs are slightly proarrhythmic at normal heart rates, thus more episodes, but do become antiarrhythmic at rapid heart rates, thus shorter episodes. The facts that antiarrhythmics can convert atrial fibrillation to atrial flutter, increase the frequency and duration of paroxysmal AF, and convert paroxysmal AF to chronic AF are well-documented[1].

There were no differences between drug users and non-drug users as far as average age, gender distribution or total years of LAF. The finding that overall, afibbers who take antiarrhythmics are no better off than afibbers who do not is indeed surprising and obviously needs further scrutiny. First of all it should be kept firmly in mind that none of the drugs prescribed for LAF have been specifically developed to deal with this condition and, as a matter of fact, several of them are not even approved for the treatment of atrial fibrillation as such. So essentially whenever a LAF patient is prescribed an antiarrhythmic it is a trial and error procedure – there is no guarantee of success. This is compounded by the fact that many afibbers are clearly receiving the wrong drugs for their particular condition. This is particularly pronounced among vagal afibbers.

Drugs in Vagal LAF

Twenty-six of the 35 vagal afibbers (74%) were taking antiarrhythmics or other drugs to prevent further episodes. There is ample evidence that vagal afibbers should not take digoxin (Lanoxin), beta-blockers or antiarrhythmics with beta-blocking properties as these drugs will markedly worsen their condition[2,3]. Yet of the 26 vagal afibbers on drugs 14 (54%) were on a drug contraindicated for their condition. These people spent an average of 105 hours in fibrillation (over 6 months) as compared to 40 hours for the people on the drugs best suited for vagal LAF flecainide (Tambocor) and disopyramide (Norpace, Rythmodan). Even vagal afibbers taking no drugs at all spent less time (90 hours) in fibrillation than did the people who were on the wrong drugs. Vagal afibbers on flecainide did the best and spent only 23 hours in fibrillation and had an average of 6 episodes (average duration of 3 hours) over the 6 months. This compares to 6 episodes (average duration of 24 hours) for non-drug users and 24 episodes (average duration of 13 hours) for people on contraindicated drugs. There was no significant difference in age or time since diagnosis between the drug and non-drug groups.

It was, unfortunately, not possible to establish the statistical significance of the above-mentioned differences because the individual sub-groups were too small and quite heterogeneous. Nevertheless, it seems clear that flecainide and disopyramide may be of benefit for vagal afibbers while other antiarrhythmics are not. Flecainide or disopyramide for that matter are not for the faint of heart though. They are highly dangerous drugs that should only be used by people with an absolutely sound heart. Side effects can be serious and potentially fatal.

Drugs in Adrenergic LAF

Afibbers with the adrenergic variety were somewhat older on average (53 years) than vagal afibbers (49 years). Of the 24 adrenergic afibbers 13 took no drugs and 11 (46%) were primarily on beta-blockers with atenolol (Tenormin) being the most popular (used by 55%). There was no significant difference in the time spent in fibrillation in the drug group (146 hours) and the non-drug group (155 hours). The non-drug group did, however, have more episodes than the drug group (14 versus 8 for the 6-month period). There was no significant difference in age or time since diagnosis between the drug and non-drug groups.

Drugs in Mixed LAF

Afibbers with the mixed variety were again older than the vagal group with an average age of 54 years. The 13 respondents of the drug group (65%) spent an average of 197 hours in fibrillation over the 6-month survey period and had an average of 39 episodes lasting an average of 11 hours. In contrast, the 7 non-drug users spent only 40 hours in fibrillation with 14 episodes lasting an average of 9 hours. Thus it would appear that mixed afibbers on drugs are substantially worse off than those not on drugs. This is really not surprising as most of the drug group were taking drugs (including 3 on digoxin) that would aggravate the vagal component of their condition.

The results and conclusions for the mixed group are somewhat confounded by the fact that the average age of the non-drug group was 48 years as compared to 58 years for the drug group. Looking closer at the regression analysis results it would appear that the age difference could account for about 25 extra hours of fibrillation in the older drug group. So even taking age into account it is still clear that drug users spent about 4 times longer in fibrillation and had almost 3 times as many episodes as did non-drug users.

Drugs and Chronic LAF

Afibbers with chronic LAF tended to be older than paroxysmal afibbers (average age of 59 years versus 51 years). Women were also somewhat over-represented in the chronic group at 30% versus 15% in the paroxysmal group. Six of the 20 respondents with chronic LAF did not take any drugs to control their heart rates. Four took diltiazem (Cardizem, Tiazac). Four took atenolol either alone or in combination with diltiazem, two took propafenone (Rythmol), and one each took sotalol (Betapace), digoxin (Lanoxin) or amiodarone (Cordarone). One chronic afibber was on a mixture of diltiazem and propafenone. Diltiazem seemed to be the most helpful of the lot as far as keeping the heart rate under control.

It is not immediately obvious why some chronic afibbers are on antiarrhythmics as there is no evidence that this will help them convert to sinus rhythm unless they are being prepared for cardioversion – none of the

respondents were. Certainly being on digoxin can only make things worse and amiodarone has some very serious long-term side effects.

In conclusion the data collected in the LAF survey does not support the assumption that treatment with antiarrhythmics is beneficial to people with lone atrial fibrillation. There are clearly cases where afibbers have been helped by these drugs, e.g. flecainide for vagal afibbers, but in general terms they do not seem to be helpful and, in many cases, are clearly detrimental. It would appear to be up to each individual, in cooperation with his or her physician, to find the right drug or to forego antiarrhythmics altogether. Remember that LAF is not life-threatening, but antiarrhythmics can be. The best and safest approach for many afibbers may well be to just take verapamil during an episode to keep the heart rate under control.

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That is it for this edition of the LAF survey results. In the next issue we will take a look at the correlation with other variables such as aspirin usage, fish oil and magnesium supplementation, and the presence of amalgam dental fillings or dissimilar metals in the mouth. Stay tuned!

THE INFLAMMATION CONNECTION

Inflammation is the body's immediate response to an injury, infection or other type of stress. It is usually time limited and ceases when healing is completed. However, in some cases, the inflammatory response continues unchecked and this can lead to the development of inflammatory diseases such as asthma, rheumatoid arthritis, and irritable bowel syndrome. An elevated erythrocyte (red blood cell) sedimentation rate (ESR) and high blood levels of interleukin-6 (IL-6) and C-reactive protein (CRP) are prominent features of inflammation.

There is mounting evidence that a systemic inflammation of the blood vessel lining is heavily involved in the initiation and progression of atherosclerosis. Austrian researchers have found that chronic dental infections, urinary tract infections, and chronic respiratory infections all substantially increase the risk of atherosclerosis[1]. Italian researchers have found elevated blood levels of IL-6 and CRP in patients with unstable angina and have associated such higher levels with an increased risk of heart attacks[2]. Very recently researchers at the Harvard Medical School found that high CRP levels are a potent risk factor for peripheral arterial disease (intermittent claudication)[3].

Just recently diabetes, depression and most common cancers were also added to the list of inflammatory diseases[4,5,6]. It is probably not an overstatement to conclude that over 90% of all that ails us is caused by an underlying inflammation.

So why are we so inflamed? There are several possible explanations:

- Our lifestyle often emphasizes factors that are known to initiate inflammation – mental, emotional and physical stress, vigorous exercise, alcohol consumption, mercury poisoning (mostly from dental amalgams), and oxidative stress. Inflammation can also be initiated by a bacterial, viral or fungal infection.

- Many common foods are inflammatory given the right conditions. The excessively high ratio of omega-6 polyunsaturated fatty acids to omega-3 fatty acids found in our modern diet favours the production of inflammatory prostaglandins, which certainly does not help matters[7].
- Childhood exposure to bacteria and viruses has been sharply curtailed through vaccinations and an excessive preoccupation with cleanliness. According to the “hygiene hypothesis” this has created an imbalance in the body’s T-cells (key immune system defenders) so that the ones that promote inflammation have become dominant[8].

Whatever the reason, there is no doubt that inflammation and the many diseases resulting from it are rampant today.

Inflammation and LAF

Could an inflammation be involved in lone atrial fibrillation (LAF)? Indeed it could. In 1997 Dr. Andrea Frustaci, MD and colleagues at the Catholic University of Rome made a fascinating discovery. They performed biopsies of the right atrium in 12 patients with LAF and found that 8 (67%) of them had evidence of a current or past inflammation in the heart tissue (myocarditis). They also checked 11 control subjects and found that none of their biopsy samples showed any signs of inflammation. The Italian researchers conclude that inflammation and its aftermath (fibrotic tissue) is a likely cause of LAF[9].

The inflammation was found to be active in 3 of the 8 patients. These patients were treated with the anti-inflammatory medication prednisone. They had no further LAF episodes over a 2-year follow-up. The remaining patients were treated with propafenone, sotalol, flecainide or amiodarone and had numerous LAF episodes over the next 2 years.

Through recent correspondence with Dr. Frustaci I learned that 2 more patients had later shown signs of active inflammation and had been successfully treated with prednisone[10]. Dr. Frustaci concurred that a relapse of atrial inflammation could result in new episodes of LAF and that it is quite possible that all the 12 LAF patients actually had signs of inflammation, but that the biopsy missed them in four of the cases. Dr. Frustaci also agreed that a high concentration of mercury or antimony in the heart tissue could produce electrical instability perhaps leading to LAF. Dr. Frustaci has earlier reported that some patients with congestive heart failure have levels of mercury and antimony in their heart tissue that are 22,000 and 12,000 times higher respectively than those found in healthy people[11]. Canadian researchers at the University of Calgary have pointed out that dental amalgams (silver fillings) would be the most likely source of the mercury[12].

More recently Dr. Frustaci and colleagues reported a link between ventricular arrhythmias (tachyarrhythmias) and the presence of inflammation in the left ventricle. The inflammation in turn was linked to the presence of hepatitis C virus, enterovirus or influenza virus in the inflamed tissue[13].

Just last month a team of American and Greek researchers reported that many patients with congestive heart failure also have an active inflammation of the heart lining which, in some cases, can be treated successfully with prednisone. They observed that about one third of the patients with active inflammation had elevated erythrocyte sedimentation rates[14,15]. It is intriguing to speculate about a possible link between Dr. Frustaci’s findings of grossly elevated levels of mercury and antimony in the heart tissue of patients with congestive heart failure and this new finding.

The Source of LAF

It is profoundly interesting and revealing that most of the triggers for LAF identified in our survey are associated with an inflammatory response. Mental and physical stress, vigorous exercise, alcohol consumption, mercury poisoning, bacterial, viral and fungal infections and oxidative stress have all been identified as potential initiators of inflammation[1,16-20]. There is also a distinct association between autonomic nervous system dysfunction and the inflammatory response[21,22]. Animal experiments have shown that an excessive release of norepinephrine (noradrenaline) can cause an inflammation and conversely that an inflammation can damage the nerve endings that release norepinephrine[23,24].

The connection between the autonomic nervous system and inflammation is indeed an intriguing one and may well hold the key to the origin of LAF. Please note that the following is pure speculation on my part and not supported by any clinical evidence that I am aware of.

It would seem that LAF requires the presence of both an inflammation of the heart lining (myocarditis) and an imbalance in the autonomic nervous system. Is it possible that this combination of inflammation and autonomic nervous system dysfunction could trigger the damage to nerve endings (vagal or adrenergic) in the myocardium? Is it also possible that the body would compensate for this damage by increasing the output of norepinephrine (the adrenergic transmitter) and acetylcholine (the parasympathetic (vagal) transmitter) at adjacent nerve endings? If this were indeed the case it would explain the creation of highly sensitive *foci* on the surface of the heart. These *foci* in turn would initiate an LAF episode whenever the dominant (unbalanced) branch (adrenergic or vagal) of the autonomic nervous system became overloaded through physical or mental stress, etc. The highly sensitive *foci* would be discernible during an electrophysiology study and would be the ones destroyed during ablation therapy. Again I want to emphasize that this hypothesis is pure speculation on my part; however, it does seem to make sense and could be a plausible explanation for the initiation and continuation of LAF episodes.

Elimination of LAF

Dr. Frustaci believes that individual heart cells, which have been exposed to inflammation, can revert to normal cell structure – assuming that the DNA of the cell has not been damaged beyond repair. This is indeed encouraging as it may mean that LAF could be permanently eliminated if the inflammation is vanquished[10].

So how can the inflammation be eliminated? Clearly a two-pronged approach is required:

- The causes (triggers), which bring on flare-ups of inflammation, must be avoided.
- The immune system must be rebalanced to prevent an excessive inflammatory response.

Cutting out alcohol, caffeine, cold drinks, MSG, aspartame etc. is the easy part. The most difficult part for many afibbers, especially those with the vagal variety, will be to refrain from vigorous exercise and workouts until the inflammation has subsided. This is absolutely essential though. Exercise will fan the inflammation; as a matter of fact Harrison's "Principles of Internal Medicine" suggests that bed rest may be necessary in more severe cases of myocarditis[25]. Swedish sports medicine experts are adamant that exercise should be avoided whenever myocarditis is suspected[26]. So the message is clear. No vigorous exercise while working on getting rid of the inflammation. A couple of leisurely walks each day is probably OK and should be enough to ensure adequate bowel movement for the 4-6 weeks it will take to overcome the inflammation. Avoidance of excessive emotional or work-related stress is also mandatory during the recovery period.

Bacterial, viral and fungal infections are potent triggers of inflammation. Both myocarditis (inflammation in the heart associated with LAF) and atherosclerosis have been linked to an infection with the *Chlamydia pneumoniae* bacterium[27-29]. Some researchers have reported the presence of *Helicobacter pylori* bacteria in atherosclerotic lesions, but the evidence of a causative link is not as convincing as for *C. pneumoniae*[30]. There is also some evidence that a systemic *Candida albicans* infection can promote inflammation of the heart tissue (myocarditis) in severely immuno-compromised AIDS patients[31]. Just recently American researchers reported that mice infected with the coxsackievirus (associated with the common cold, meningitis and encephalitis) or the cytomegalovirus (associated with mononucleosis, hepatitis and colitis) developed myocarditis within 2 weeks of becoming infected[32]. Adding this evidence to Dr. Frustaci's findings[9,13] it is clear that there is an association between bacterial, viral and fungal infections and the development of myocarditis and, furthermore, that there is an association between myocarditis and heart arrhythmias (both atrial and ventricular). So if you have LAF it would seem prudent to undergo testing for possible infections and follow-up with medical treatment to eradicate them as necessary.

Diet can also be a potent source of inflammation. In some people wheat, dairy products and certain foods of the nightshade family (potatoes, peppers, eggplant, tomatoes) can cause a chronic inflammation[33]. I have

found Dr. Peter D'Adamo's book "Eat Right for Your Type" particularly helpful in sorting out what to avoid and what to emphasize in the diet[33].

Elimination of Persistent Inflammation

If the inflammation persists after eliminating the causes as discussed in the previous section it is likely that it is due to an immune system dysfunction. There are several ways of correcting this.

It is evident from Dr. Frustaci's work that the inflammation (myocarditis) associated with LAF can be eliminated by treatment with prednisone[9]. Unfortunately, rather high dosages are required, at least initially. Prednisone has the potential for serious adverse reactions and its use is generally not recommended for extended periods of time. Dr. Frustaci used 1 mg per kg bodyweight per day for 4 weeks tapered to 0.33 mg for 4 months. So while prednisone may do the job, at least if the inflammation is active, the overall benefit/risk ratio is not encouraging although probably no worse than that of long-term amiodarone (Cordarone) treatment.

An unfavourable benefit/risk ratio also applies to the use of aspirin and other NSAIDs to combat inflammation. They do not get at the root cause of the inflammation and can cause serious bleeding complications.

The cholesterol-lowering drug pravastatin (Pravachol) is effective in reducing the level of the inflammation marker C-reactive protein (CRP)[34]. This could benefit patients with atherosclerosis or rheumatoid arthritis, but as far as I know no work has been done to investigate the use of pravastatin in lone atrial fibrillation. Unfortunately, pravastatin has many potentially serious side effects including liver dysfunction, myopathy, rhabdomyolysis, and possibly cancer. Pravastatin has also been found to lower coenzyme Q10 levels possibly leading to impaired cardiac function and congestive heart failure[35].

Human growth hormone replacement is another possible route for combating inflammation. Researchers at the Harvard Medical School recently reported that levels of IL-6 (interleukin-6) and CRP were both significantly reduced by the administration of recombinant human growth hormone in men with adult-onset growth hormone deficiency. The reduction in CRP level (30%) was similar to that obtained with pravastatin. IL-6 levels decreased by almost 40% as compared to the placebo group. The researchers conclude that, "long-term growth hormone replacement in men reduces levels of inflammatory cardiovascular risk markers" (IL-6 and CRP)[36].

It is interesting that one member of our group of afibbers has found growth hormone (HGH) therapy to be useful. Chuck, a vagal afibber, started using an oral sublingual HGH spray in early May (Sol RX available at www.atlantis.to/Products/gh-atlantis-home.htm). He used to have daily episodes lasting 4 to 5 hours. Since using HGH both his episode frequency and duration have decreased by about 50%[37].

If anybody decides to try HGH replacement please let me know how it goes. As always, please check with your physician first to make sure you have no specific contraindications to using it. Also please remember that Chuck has the vagal variety of LAF. HGH replacement may not work for adrenergic or mixed afibbers.

Balancing the Immune System

Lymphocytes, a specialized kind of white blood cells, are important components of the immune system. They can be subdivided into B-lymphocytes, which produce antibodies, and T-lymphocytes (helper T-cells), which help identify foreign cells and antigens so that killer cells can dispose of them. T-cells come in two varieties, TH1 cells and TH2 cells. TH1 cells produce lymphokines that enhance the ability of the immune system to kill viruses, bacteria, fungi, and parasites. TH2 cells are involved in allergic reactions and release interleukin-6, a powerful marker of inflammation. A healthy immune system has an optimum balance of TH1 and TH2 cells. The results of too many TH2 cells are autoimmune diseases, allergies, inflammation, and pain while not enough TH1 cells can lead to cancer and infectious diseases[38].

Extensive research carried out at the University of Stellenbosch in South Africa has shown that a proprietary mixture of plant sterols and sterolins (Moducare) is very effective in increasing TH1 cell production (the "good" T cells) and decreasing TH2 cell production (the "bad" T cells). Moducare also normalizes the ratio

between DHEA and cortisol[38]. Moducare has strong anti-inflammatory effects and sharply reduces IL-6 production. It has been found useful in the treatment of chronic viral infections, tuberculosis, and HIV infection[39]. Also it has been found to reduce the inflammatory response associated with excessive physical exertion[40]. The recommended dosage of Moducare is two capsules one hour before the main meals for the first month and then one capsule one hour before breakfast, lunch and dinner.

I believe Moducare could be a very valuable ally in eliminating LAF and am taking it myself. Please let me know the results if you try it out. You should be able to obtain Moducare in your health food store, but if not you can order it on-line at www.moducare.com. Please note that people who have had an organ transplant should not take Moducare as it may interfere with immunosuppressive drugs.

Alternative Approaches

Besides Moducare there are several other natural remedies that may be beneficial in reducing excessive inflammation. None of these remedies have been evaluated specifically for the inflammation involved in LAF or even atherosclerosis, but they have been found useful in the treatment of other inflammatory diseases such as rheumatoid arthritis and inflammatory bowel disease.

- **Boswellia** (*Boswellia serrata*, Frankincense) - This resin obtained from the *Boswellia serrata* tree has been used as an anti-inflammatory in ayurvedic medicine for centuries. Recent research has found it to be highly effective in the treatment of ulcerative colitis, Crohn's disease and asthma[41,42,43].
- **Curcumin** – The yellow pigment of turmeric is as effective as cortisone in combating acute inflammation[44,45]. The recommended dosage is 400 mg three times daily preferably on an empty stomach[44].
- **Bromelain** – A mixture of enzymes found in pineapple has been found effective in the treatment of rheumatoid arthritis[44,46]. The recommended dosage is 250-750 mg/day[44].
- **Ginger** (*Zingiber officinalis*) – It is a strong antioxidant that inhibits the formation of inflammatory compounds. It has been found highly useful in the treatment of rheumatoid arthritis[44,47]. The recommended dosage (fresh ginger root) is 8-10 grams/day[44].
- **Sarsaparilla** (*Smilax sarsaparilla*) – This herb contains natural steroids and has been used since the Middle Ages in the treatment of rheumatism. It is also said to be useful in the treatment of mercury poisoning[48]. More recent animal experiments have shown it to be highly effective in the treatment of inflammation[49]. I have not found a recommendation for dosage so an herbalist or a practitioner in Chinese Medicine should be consulted before using.
- **Omega-3 fatty acids** – Fish oils have been found beneficial in reducing rheumatoid arthritis symptoms[44,50,51]. The recommended daily dosage is 1.8 grams of eicosapentaenoic acid (EPA) from fish oil[44].
- **Pancreatic enzymes** – These have been found to be beneficial in the treatment of chronic inflammatory conditions such as rheumatoid arthritis[52]. They should be taken before meals.
- **Probiotics** – A recent review of the benefits of probiotics (*Lactobacillus* and *Bifidobacterium*) concluded that the modification of gut microflora by probiotic therapy might help alleviate inflammatory diseases such as arthritis and inflammatory bowel disease[53].
- **Antioxidants** – Last, but certainly not least, it is very important to ensure an adequate daily intake of the major antioxidants (vitamin C, vitamin E, selenium, beta-carotene, proanthocyanidins and alpha-lipoic acid). They all help to combat oxidative stress, a potent source of inflammation.

I now firmly believe that the key to permanently overcoming LAF is to eliminate the dormant or active underlying inflammation of the heart lining. I have personally made some significant changes to my diet, lifestyle, and supplements in order to achieve this and will keep you informed of my progress. You can find the detailed "Larsen Protocol" for eliminating inflammation and LAF after the references. If you try it please let me know the results.

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The Larsen Protocol

My approach to eliminating LAF is 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. The "Larsen Protocol" does not replace whatever other measures you are now taking to control your LAF (e.g. Dr. Lam's protocol) - it is solely designed to eliminate inflammation.

I should point out that I have experienced the adrenergic type of LAF for the past 11 years. I am not on any drugs and have had 8 episodes over the last six months with an average duration of 54 hours. Since beginning the protocol I have had zero episodes, my number of ectopic beats has dropped from 4-5/minute to 0-1/minute, and my autonomic nervous system is now in far better balance than it was prior to starting on the protocol. I have now gone 40 days without an episode – this is a bit of a record!!

This protocol has certainly made a great difference to me. Whether it will do the same for vagal, mixed and perhaps chronic afibbers remains to be seen.

If you decide to try the protocol please make sure you discuss it with your physician first. Although the components of the protocol are generally well tolerated and I am not aware of any side effects from the supplements there may be factors in your medical history or in any medicines you are taking that should be taken into account before you start. It may also be advisable to ease into the protocol over a one to two week period rather than start taking all the supplements all at once. I began by just taking Moducare, vitamin C, pancreatic enzyme and curcumin/bromelain for the first week.

I am not aware of any potential interactions between the recommended supplements and warfarin, but if you are on warfarin you may wish to check your INR a bit more frequently when you first start the protocol.

Dietary Modifications

I have been very much impressed by Dr. Peter D'Adamo's work relating optimum diet to blood type. I am a type 0, as I suspect most afibbers are, and have been following the Type 0 Diet for a couple of weeks now. 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 it is essential to cut back on these types of fats (found in margarines and cooking oils) and increase the intake of omega-3 fatty acids (found in fatty fish and flax oil).

I would urge you to obtain one or both of Dr. D'Adamo's books "Eat Right for Your Type" and "Live Right for Your Type" and then follow the diet appropriate for your blood type. Eating the wrong foods sets up an internal war that aggravates inflammation. You can purchase the books in your local book or health food store or you can order them by mail (Amazon.com) through the LAF Forum (www.yourhealthbase.com/lafforum.html).

Lifestyle Changes

I began modifying my lifestyle quite a few years ago so I don't believe that these changes can explain my recent improvements. I avoid alcohol and caffeine and try to control both my emotional and physical stress levels. I had my amalgam (silver) fillings replaced a couple of years ago. Unfortunately, the procedure was not done under optimum conditions and my detoxification was also a bit haphazard so I am not sure that I am totally mercury-free as yet. I do not exercise vigorously. Vigorous exercise can cause and exacerbate inflammation in the muscles being exercised including the heart muscle. So for the month or so it takes to test the protocol I strongly recommend replacing vigorous exercise with a leisurely walk or two every day.

Supplements

I take the following supplements daily for the purpose of dampening and eventually eliminating inflammation. These are in addition to my regular supplements (multivitamin, magnesium, taurine, saw palmetto, etc.)

DAILY SUPPLEMENT PROTOCOL

Upon arising (1 hour before breakfast): 2 Moducare capsules [1]

At breakfast: 1000 mg time-release vitamin C [2]
500 mg quercetin (with bioflavonoids)

100 mg alpha-lipoic acid
1 pancreatic enzyme capsule (Cotazym)
1 g fish oil [3]

Mid-morning (on an empty stomach)
600 mg curcumin (turmeric extract) [4]
300 mg bromelain

One hour before lunch: 2 Moducare capsules

At lunch: 1 pancreatic enzyme capsule (Cotazym)
500 mg quercetin
100 mg alpha-lipoic acid

Mid-afternoon (on an empty stomach)
600 mg curcumin
300 mg bromelain

One hour before dinner: 2 Moducare capsules

At dinner: 1 pancreatic enzyme capsule (Cotazym)
1000 mg time-release vitamin C
400 IU natural vitamin E
500 mg quercetin
200 microgram selenium

Before bed: 2 Protec Probiotic capsules [5]

NOTES

[1] Moducare should always be taken on an empty stomach (1 hour before or 2-3 hours after a meal). If you find it inconvenient to take it first thing in the morning or if you have hypoglycemia you can take it one hour before bed.

[2] It is important to use timed-release vitamin C.

[3] The fish oil should provide about 350 mg EPA and 230 mg DHA.

[4] You may find that curcumin irritates your stomach. If so, take it with meals or discontinue it periodically.

[5] The probiotic supplement should contain about

6.4 billion active *L.rhamnosus*
0.8 billion active *L.acidophilus* plus
0.4 billion each of *B.longum* and *B.bifidum*.

I noticed a significant improvement in my heart stability and indeed in my general health and well-being within two weeks of beginning the protocol. I am very hopeful that four to six weeks on the protocol will eliminate my LAF episodes altogether – although individual results may depend on systemic mercury status.

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