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Your Gateway to Better Health!

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Editorial

Medical researchers at the Brigham and Women's Hospital in Boston have just announced the discovery of a strong association between inflammation and the development of type 2 diabetes. This adds diabetes to a long list of other common diseases that are initiated and sustained by a chronic inflammation. Among them, atherosclerosis, angina, heart attack, congestive heart failure, intermittent claudication, asthma, rheumatoid arthritis, irritable bowel syndrome, hepatitis, gastritis, pancreatitis, prostatitis, and on and on it goes. Just recently depression and most common cancers were also added to the list[1,2]. 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 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[3].*

- *Childhood exposure to bacteria and viruses has been sharply curtailed through obsessive cleanliness and vaccinations. 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.*

Whatever the reason, there is no doubt that inflammation and the many diseases resulting from it are rampant today. In this issue of The AFIB Report I provide convincing evidence that lone atrial fibrillation is also caused by an inflammation (of the heart lining).

So what can be done? Conventional medicine, unfortunately, does not have a very good answer. Prednisone will certainly dampen inflammation in the short-term, but it has so many serious side effects that its long-term use is strongly discouraged.

Fortunately, it seems like alternative medicine may have the answer. After extensive research I have come up with a protocol, the "Larsen Protocol" for lack of a better name, which will, I believe, safely eliminate any systemic inflammation in 4 to 6 weeks. You can find the details of the protocol at the end of this month's AFIB Report. I have been on the protocol now for 3 weeks and feel great. Incidentally, it will also make you lose weight if you need to. Let me know if you try it – I would be most interested in your feedback.

Yours in health,
Hans

1. *Brown, Phyllida. A mind under siege. New Scientist, June 16, 2001, pp. 34-37*
2. *O'Byrne, K.J. and Dalgleish, A.G. Chronic immune activation and inflammation as the cause of malignancy. British Journal of Cancer, Vol. 85, No. 4, August 2001, pp. 473-83*
3. *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*

September Highlights

ABSTRACTS

Helicobacter pylori implicated in pancreatic cancer

BETHESDA, MARYLAND. The presence of *Helicobacter pylori* bacteria has been strongly linked to stomach ulcers and stomach cancer. Researchers at the National Cancer Institute and the Finnish National Public Health Institute now report that a *H. pylori* infection may also be a strong risk factor for pancreatic cancer (exocrine). The study involved 29,133 male Finnish smokers aged 50 to 69 years at baseline. During 10 years of follow-up 121 of the men developed cancer of the pancreas. The case subjects were matched with 226 cancer-free subjects. The researchers noted a higher incidence of *H. pylori* infection among the cancer patients than among the controls (82 versus 73 per cent) and concluded that pancreatic cancer was twice as common among participants who tested positive for *H. pylori* as among those who tested negative. This association became stronger after adjusting for years of smoking. The researchers also noted that cancer cases had significantly lower blood serum levels of folic acid, vitamin B6 (pyridoxine), and vitamin E (alpha-tocopherol).

Stolzenberg-Solomon, Rachael Z., et al. Helicobacter pylori seropositivity as a risk factor for pancreatic cancer. **Journal of the National Cancer Institute**, *Vol. 93, June 20, 2001, pp. 937-41*

Homocysteine and longevity

BERGEN, NORWAY. It has been known for about 20 years that a high blood level of homocysteine is a potent risk factor for heart disease. Researchers at the University of Bergen now report that a high homocysteine level is a risk factor for premature death overall, whether it be from heart disease, stroke, cancer, lung disease or whatever.

The study involved 2127 men and 2639 women aged 65 to 67 years when they entered the study between 1992 and 1993. By February 1997 162 men and 97 women had died; 121 from cardiovascular causes (including stroke), 103 from cancer, and 33 from other causes. Using a baseline homocysteine level of 9.0 micromol/L the researchers found that for every 5.0 micromol/L increment in homocysteine levels all-cause mortality increased by 49 per cent, cardiovascular mortality by 50 per cent, cancer mortality by 26 per cent, and deaths from other causes (respiratory, gastrointestinal and central nervous system diseases) by 104 per cent. These percentages refer to values obtained after adjusting for cholesterol level, blood pressure, smoking, body mass index, physical activity, cardiovascular disease risk status at baseline, age and gender. About 78 per cent of the study group had homocysteine levels at or above 9.0 micromol/L and 12 per cent had levels exceeding 15 micromol/L.

Smoking and drinking coffee were associated with higher homocysteine levels while taking vitamins

and exercising were associated with lower levels. The researchers conclude that homocysteine levels have a pervasive influence on longevity and recommend that future research on homocysteine and health should focus on diseases other than cardiovascular ones. **Editor's Note**: Homocysteine levels can be safely and effectively lowered by daily supplementation with folic acid (800 micrograms/day), vitamin B6 (50-100 mg/day) and a vitamin B12 (1000 micrograms/day) sublingual tablet.

Vollset, Stein Emil, et al. Plasma total homocysteine and cardiovascular and noncardiovascular mortality: the Hordaland Homocysteine Study. **American Journal of Clinical Nutrition**, *Vol. 74, July 2001, pp. 130-36*

Malinow, M. Rene. Plasma concentrations of total homocysteine predict mortality risk. **American Journal of Clinical Nutrition**, *Vol. 74, July 2001, p. 3 (editorial)*

Fats and breast cancer

MILAN, ITALY. The association between the intake of various fats and oils and the risk of breast cancer has been the subject of several studies. There is still no real consensus as to which fats are beneficial and which are not. The evidence for a protective role of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), the main components of fish oils, is probably the strongest.

Researchers at the Italian National Cancer Institute have just completed an investigation aimed at clarifying the association between fat intake and breast cancer risk. A total of 4052 postmenopausal women were followed for an average of 5.5 years. During this time 71 cases of invasive breast cancer were diagnosed. The cancer patients were matched with 141 controls. All study participants had blood samples drawn and red blood cell (erythrocyte) membranes were analyzed for their fatty acid content. The researchers point out that erythrocyte membranes are good biomarkers for not only dietary fat intake, but also for other dietary and hormonal factors.

Women with DHA concentrations in the highest tertile had less than half the risk of breast cancer than did women in the lowest tertile. Polyunsaturated fatty acids overall were also protective with omega-3 acids being somewhat more protective than omega-6 acids. Saturated fatty acid concentrations were not significantly related to breast cancer risk. A higher concentration of monounsaturated fats, especially oleic acid, was associated with a significantly increased risk. The researchers point out that most oleic acid in mammalian tissue is derived from saturated stearic acid through a process involving the enzyme delta 9-desaturase. Saturated fatty acids, cholesterol, carbohydrates, insulin, testosterone, and estrogen all activate this enzyme whereas dietary polyunsaturated fatty acids and fasting deactivate it. The researchers conclude that the delta 9-desaturase enzyme may be an important link between breast cancer risk and dietary fat consumption and urge further research in the field.

Pala, Valeria, et al. Erythrocyte membrane fatty acids and subsequent breast cancer: a prospective Italian study. **Journal of the National Cancer Institute**, *Vol. 93, July 18, 2001, pp. 1088-95*

How about pickled cucumber for breakfast?

LUND, SWEDEN. Carbohydrates, notably white bread, cause blood sugar and insulin levels to rise (postprandial glycemia). The glycemic index was developed in order to quantify the extent of the blood sugar increase. White bread is the reference point with a glycemic index of 100. Similarly, white bread with a value of 100 is also the reference point for the insulinemic index that quantifies the rise in insulin after consumption of carbohydrates. Research has shown that consuming a diet with a low glycemic index improves blood sugar control and cholesterol levels and reduces the risk of heart attacks and type 2 diabetes.

It has generally been assumed that foods with a low glycemic index also have a low insulinemic index. Researchers at the University of Lund now challenge this assumption at least when it comes to milk products. After carrying out several laboratory experiments using human volunteers the researchers conclude that even though regular milk has a glycemic index of only 30 its insulinemic index is 90. Fermented milk products, which have very low glycemic indexes of 15, were

found to have insulinemic indexes of 98. All the milk products induced postprandial hypoglycemia within 50 minutes of consumption. In other words, milk products play havoc with blood sugar control.

The researchers speculated that organic acids such as vinegar might reduce the insulinemic index of the standard milk and carbohydrate breakfast. They compared a breakfast of white bread plus regular milk (250 grams) plus fresh cucumber (50 grams) [meal 1] with one of white bread plus yogurt (250 grams) plus pickled cucumber (50 grams) [meal 2]. They found that the glycemic and insulinemic index for meal 1 was 79 and 117 respectively as compared to meal 2 where the values were 55 and 79 respectively.

The researchers conclude that the vinegar in the pickled cucumber is responsible for the highly significant and beneficial reduction in insulinemic index (they had previously found that yogurt did not reduce it). They conclude that milk, despite its relatively low glycemic index, increases the insulinemic index quite considerably while organic acids such as vinegar decrease it.

Ostman, Elin M., et al. Inconsistency between glycemic and insulinemic responses to regular and fermented milk products. **American Journal of Clinical Nutrition**, *Vol. 74, July 2001, pp. 96-100*

Diabetes linked to inflammation

BOSTON, MASSACHUSETTS. A systemic inflammation is characterized by high blood plasma levels of interleukin 6 (IL-6) and C-reactive protein (CRP). Medical researchers at the Brigham and Women's Hospital now report that they have discovered a strong association between the risk of developing type 2 diabetes and high blood levels of IL-6 and CRP.

Their study involved 27,628 female health professionals who were free of diabetes at enrollment in 1992. Four years later 188 of the women had been diagnosed with diabetes. They were paired with 362 healthy, age-matched controls and blood levels of IL-6 and CRP (at baseline) were compared between the two groups. The researchers found that women with a high CRP level had a 15.7 times higher risk of diabetes than did women with a low level (highest quartile versus lowest quartile). Similarly, the risk among women with a high IL-6 level was 7.5 times higher than among women with a low level. Adjusting for body mass index, family history of diabetes, smoking, exercise, use of alcohol, and hormone replacement therapy reduced the excess risk related to high IL-6 levels to 2.3 and that for CRP to 4.2.

The researchers conclude that elevated levels of CRP and IL-6 predict the development of type 2 diabetes and that their data supports a role for inflammation in the development of diabetes.

Pradhan, Aruna D., et al. C-reactive protein, interleukin 6, and risk of developing type 2 diabetes mellitus. **Journal of the American Medical Association**, *Vol. 286, July 18, 2001, pp. 327-34*

COX-2 inhibitors may not be heart friendly

CLEVELAND, OHIO. COX-2 inhibitors have had a rapid rise to fame. They were introduced in 1999 and by October 2000 annual sales exceeded $3 billion in the United States corresponding to about 100 million individual prescriptions. The COX-2 class drugs, celecoxib (Celebrex) and rofecoxib (Vioxx), are mainly used in the treatment of rheumatoid arthritis, but have also been prescribed for general pain relief. They are less likely to promote internal bleeding and stomach ulcers than are aspirin and other NSAIDs (nonsteroidal anti-inflammatory drugs).

Researchers at the Cleveland Clinic now warn that the COX-2 inhibitors may not be as benign as originally thought. An extensive literature review turned up the finding that the risk of having a heart attack while on rofecoxib is 42 per cent greater than if taking a placebo (0.74 per cent versus 0.52 per cent annual rate). The same applied to celecoxib where the risk is 54 per cent greater (0.80 per cent versus 0.52 per cent annual rate). The data was extracted from trials involving 23,407 patients. Another trial found that people taking rofecoxib had twice as many cardiovascular events (heart attacks, strokes, angina, etc.) than did patients on the NSAID naproxen. The Cleveland researchers call for large-scale clinical trials to verify or refute their findings, but in the meantime urge caution in prescribing COX-2 inhibitors to people at risk for heart disease.

Mukherjee, Debabrata, et al. Risk of cardiovascular events associated with selective COX-2 inhibitors. **Journal of the American Medical Association**, *Vol. 286, August 22/29, 2001, pp. 954-59*

Glucarate and breast cancer prevention

LAWRENCEVILLE, NEW JERSEY. So far the "war on cancer" initiated by President Nixon in 1971 has had a fairly limited success. In 1971 a total of 635,000 new cases of cancer was diagnosed in the USA; by the year 2000 this number had risen to 1,225,000 – an increase of 93 per cent. The number of new cases of breast cancer has increased by about 61 per cent since 1973 and now stands at 184,000 annually. A 1991 report issued by the US Government Accounting Office concluded that there had been no progress in the prevention of breast cancer or in reducing mortality from breast cancer.

Researchers at the Simone Protective Cancer Institute point out that perhaps 90 per cent of all cancers are caused by dietary or nutritional factors. It is also clear that inadequate removal or detoxification of carcinogenic substances in the body plays a significant role in cancer initiation and progression. Evidence is mounting that a simple natural compound, glucarate, found in many vegetables and fruits is very effective in boosting the immune system and detoxifying the body. Calcium glucarate converts to D-glucaric acid in the stomach and is the precursor of the enzyme glucuronyl transferase. This enzyme binds to carcinogens like polycyclic aromatic hydrocarbons, nitrosamines, and steroids and safely excretes them. Animal experiments have found glucarate effective in inhibiting cancers of the colon, skin, lung, and breast and it alone or in combination with vitamin A has been found to inhibit the growth of human breast cancer cells. Glucarate is effective in doses from 1 gram/kg of body weight to 27 grams/kg of body weight.

Simone, Charles B., et al. Cancer, lifestyle modification and glucarate. **Journal of Orthomolecular Medicine**, *Vol. 16, No. 2, 2nd Quarter 2001, pp. 83-90 [86 references]*

Webb, T.E., et al. Mechanism of growth inhibition of mammary carcinomas by glucarate and the glucarate/retinoid combination. **Anticancer Research**, *Vol. 13, No. 6A, November-December 1993, pp. 2095-99*

Soy-based formulas found safe

PHILADELPHIA, PENNYSYLVANIA. Many mothers who cannot breast feed their infants prefer to use soy-based infant formulas rather than conventional formulas based on cow's milk. A team of researchers from the universities of Iowa and Pennsylvania has just released the results of a major study of soy-based formulas and concludes that they are safe.

The study involved 563 infants who were fed cow's milk formula during infancy (starting before the age of 9 days) and 248 who were fed soy formula. The infants were enrolled during the period 1965 to 1978 and their medical and reproductive history examined in 1999 when they were between the ages of 20 and 34 years. The researchers looked at such factors as height, weight, body mass index, pubertal maturation, pregnancy outcomes, menstrual difficulties, sexual orientation, testicular cancer, drug usage, smoking, and education level attained (as a measure of IQ). Most of these factors could conceivably be influenced by the phytoestrogen content of soy formula. The researchers conclude that the exposure to soy formula in infancy does not lead to different health or reproductive outcomes than does exposure to cow's milk formula. Thus "the findings of the current study are reassuring about the safety of soy infant formula".

EDITOR's NOTE: For a rather different view concerning the safety of soy products see http://www.nexusmagazine.com/soydangers.html and http://www.westonaprice.org/infant.htm.

Strom, Brian L., et al. Exposure to soy-based formula in infancy and endocrinological and reproductive outcomes in young adulthood. **Journal of the American Medical Association**, *Vol. 286, August 15, 2001, pp. 807-14*

Comparison of prostate cancer treatments

BOSTON, MASSACHUSETTS. At present there are three major conventional treatment options for non-metastatic prostate cancer. Expectant management essentially means to watch and see if the cancer gets worse. Prostate tumours grow very slowly (doubling every four years) so for many men, especially older ones, this approach is quite viable. Expectant management often includes androgen-deprivation therapy (castration or estrogen). External beam radiotherapy involves the shrinking or destruction of the tumour by radiation. Radical prostatectomy involves surgical removal of the entire prostate gland. This procedure carries a substantial risk of subsequent impotence (60 per cent incidence rate) and incontinence (39 per cent incidence rate).

Researchers at several major American hospitals and universities have just released a major study of the long-term outcome of the three standard therapies. The study involved 2311 men aged 55 to 74 years at time of diagnosis during 1971 to 1984. By 1994 584 men had died from prostate cancer and 828 from other causes. The 10-year survival rate in the case of prostate cancer was 75 per cent for expectant management, 67 per cent for radiotherapy, and 86 per cent for radical prostatectomy. The researchers emphasize that these percentages are not directly comparable. For example, the members of the expectant management group were considerably older (45 per cent between the ages of 70 and 74 years) than the members of the other two groups (17 per cent between the ages of 70 and 74 years). In all cases men who underwent androgen-deprivation therapy had poorer survival than men who did not.

The results of this study differ somewhat from the results of a similar study carried out at the Johns Hopkins School of Hygiene and Public Health. This study concluded that patients who underwent radiation therapy had an 81 per cent higher risk of dying from prostate cancer than did men who received no treatment (watchful waiting). Patients who received hormones or were castrated increased their risk of dying by 85 per cent (compared with no treatment) while men who just underwent surgery with no additional treatment had a 23 per cent lower death rate (from prostate cancer) than did men who had not received any treatment. It is not clear whether the poorer survival rate among men who had androgen-deprivation therapy is due to the fact that this therapy is actually detrimental or due to the fact that it is preferentially prescribed for men with more advanced cancer.

Barry, Michael J., et al. Outcomes for men with clinically nonmetastatic prostate carcinoma managed with radical prostatectomy, external beam radiotherapy, or expectant management: a retrospective analysis. **Cancer**, *Vol. 91, June 15, 2001, pp. 2302-14*

Newschaffer, Craig J., et al. Causes of death in elderly prostate cancer patients and in a comparison nonprostate cancer cohort. **Journal of the National Cancer Institute**, *Vol. 92, April 19, 2000, pp. 613-21*

Vitamin C prevents cataracts

BOSTON, MASSACHUSETTS. Researchers at Tufts University and the Harvard Medical School have confirmed that a high intake of antioxidants (from diet or supplements) helps prevent the development of cataracts (age-related nuclear lens opacities). Their recent study included 478 female nurses who had completed food frequency questionnaires every second year since 1980. The questionnaires included details of vitamin and mineral supplement use. During the period 1993 to 1994 the study participants all underwent a detailed eye examination and had blood samples taken for analysis of plasma concentrations of vitamins C and E and the carotenoids.

The researchers conclude that vitamins C and E, riboflavin (vitamin B), folic acid, beta-carotene, and lutein/zeaxanthin all protect against cataract development. However, after adjustment for other nutrients only the association with vitamin C remained statistically significant. Women whose daily vitamin C intake (from diet and supplements) was between 140 and 180 mg/day had a 48 per cent lower risk of cataracts than did women whose intake was below 140 mg/day. Women with an intake between 240 and 360 mg/day had a 66 per cent lower risk than did women with an intake of less than 140 mg/day. Intakes above 360 mg/day did not lower the risk beyond the reduction obtained at intakes between 240 and 360 mg/day. This finding is consistent with prior observations that human eye tissues become saturated with vitamin C at intakes between 200 mg and 300 mg/day. The new findings make it glaringly obvious that the officially recommended

daily vitamin C intake for women of 75 mg/day (RDA) is inadequate to provide any meaningful protection against cataracts. The researchers also found that women who had used vitamin C supplements for 10 years or longer had a 64 per cent reduced risk of cataracts while those having supplemented with multivitamins had a 43 per cent lower risk.

Jacques, Paul F., et al. Long-term nutrient intake and early age-related nuclear lens opacities. **Archives of Ophthalmology**, *Vol. 119, July 2001, pp. 1009-19*

Black tea is good for heart patients

BOSTON, MASSACHUSETTS. Endothelial dysfunction is a disorder of the lining of the blood vessel and manifests itself by reduced arterial blood flow and greater platelet adhesiveness. It is believed to be a precursor of atherosclerosis and is a common feature of cardiovascular disease.

Researchers at the Boston University School of Medicine and the Linus Pauling Institute now report that drinking black tea reverses endothelial dysfunction in patients with coronary artery disease. Their experiment involved 50 patients who consumed either 450 ml of tea or 450 ml of water two hours before having their brachial blood flow measured. The blood flow increased by 57 per cent in the tea group, but no significant increase was seen in the water group. At another time the patients were assigned to drink either 900 ml of tea or 900 ml of water daily for four weeks. The blood flow increased by 58 per cent in the tea group, but no significant increase was seen in the water group. An equivalent dose of caffeine (200 mg) also had no effect on endothelial function. The researchers conclude that short- and long-term black tea consumption reverses endothelial dysfunction in coronary heart disease patients. They believe the effect is attributable to the flavonoids found in tea.

Duffy, Stephen J., et al. Short- and long-term black tea consumption reverses endothelial dysfunction in patients with coronary artery disease. **Circulation**, *Vol. 104, July 10, 2001, pp. 151-56*

NEWSBRIEFS

Cancer linked to inflammation. Researchers at the University of Leicester in the UK have reached the conclusion that chronic inflammation is the underlying cause of most common cancers. They believe that chronic inflammation leads to excessive oxidative stress that, in turn, induces DNA damage and harmful mutations. They conclude that therapeutic intervention aimed at inhibiting inflammation and stimulating immune responses may have a major role in preventing most cancers.

British Journal of Cancer, Vol. 85, No. 4, August 2001, pp. 473-83

Warning concerning statin drugs. The cholesterol-lowering drug Baycol (cerivastatin) was recently withdrawn from the market in Italy, Japan, Germany, the United States, and many other countries following reports of numerous deaths related to its use. Baycol has been linked to rhabdomyolysis, an often fatal muscle disease. The FDA is now considering the requirement for "black box" warning labels on all statin drugs including pravastatin (Pravachol), atorvastin (Lipitor), fluvastatin (Lescol), lovastatin (Mevacor) and simvastatin (Zocor). Several of these drugs have also been linked to rhabdomyolysis. Says Sidney Smith, MD of the American Heart Association, "Doctors should warn their patients to watch for dark urine and muscle aches and pains if they are taking statins."

Medscape News, August 24, 2001

Asthma linked to diet. Australian researchers have discovered that children whose diet includes a high level of polyunsaturated margarines and cooking oils are more than twice as likely to have asthma than children who consume less of these polyunsaturated fats. The researchers believe that omega-6 fatty acids, which metabolize into inflammation-causing prostaglandins, are the culprits. They estimate that as many as 17 per cent of all childhood asthma cases may be related to a high intake of omega-6 fatty acids.

New Scientist, July 28, 2001, p. 19

Second thoughts on folic acid. Swedish researchers have found that women who take

folic acid before and during pregnancy are more likely to have twins. Of 2569 women who used folic acid supplements 2.8 per cent gave birth to twins. This compares to a twin birth rate in the general Swedish population of 1.5 per cent. Bengt Kallen of the Tornblad Institute in Lund has calculated that if 30 per cent of 100,000 women in Sweden took folic acid there would be 225 extra pairs of twins. These twins would have a lower birth weight and an increased risk of cerebral palsy. This should be weighed against the benefit of avoiding 4 or 5 neural tube defect (spina bifida) cases. Not all researchers are convinced that the link between twins and folic acid is real though and further research is urged.
New Scientist, July 28, 2001, p. 7

Second-hand smoke is a killer. Researchers at the University of California in San Francisco warn that even a little bit of second-hand smoke can put you on the road to atherosclerosis and heart disease. A recent clinical trial found that just 30 minutes of exposure to second-hand smoke damaged the endothelial function in coronary arteries of non-smokers to the same extent as observed in habitual smokers. Endothelial dysfunction is an early manifestation of atherosclerosis. Other research has shown that passive smoking increases the risk of heart disease and death by 30 per cent. The researchers point out that all workplaces in California, including restaurants and bars, are now totally smoke-free.
Journal of the American Medical Association, Vol. 286, July 25, 2001, pp. 462-63

Goodbye root canals. It is estimated that dentists in the USA perform about 20 million root canal treatments every year. These treatments usually become necessary because the nerve inside the tooth has been damaged by earlier dental work. Dr. Richard Hansen of the Center for Advanced Dentistry in Fullerton, California has developed a new treatment for damaged nerves, which makes root canal work obsolete. The new procedure makes use of an erbium laser that destroys diseased nerve tissue, but leaves healthy tissue alone. So far Dr. Hansen has treated over 600 patients who would otherwise have required root canal work. His success rate is almost 100 per cent. To fellow dentists who claim the new procedure is too slow Dr. Hansen has this answer, “If you want to do things quickly use a hammer and chisel.”
New Scientist, July 21, 2001, p. 16

THE AFIB REPORT

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!

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.

References

1. Atrial Fibrillation: Mechanisms and Management, 2nd ed., edited by R.H. Falk and P.J. Podrid. Lippincott-Raven Publishers, Philadelphia, 1997, p. 371
2. Coumel, Philippe. Paroxysmal atrial fibrillation: a disorder of autonomic tone? European Heart Journal, Vol. 15, Suppl A, April 1994, pp. 9-16
3. Prystowsky, Eric N. Management of patients with atrial fibrillation: a statement for healthcare professionals from the subcommittee on electrocardiography and electrophysiology, American Heart Association. Circulation, Vol. 93, March 15, 1996, pp. 1262-77

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 work-outs 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 pneumonaie* 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. pneumonaie*[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 is 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, rhabdomylosis, 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.

References

1. Kiechl, Stefan, et al. Chronic infections and the risk of carotid atherosclerosis. Circulation, Vol. 103, February 27, 2001, pp. 1064-70
2. Biasucci, L.M., et al. Inflammation and acute coronary syndromes. Herz, Vol. 25, March 2000, pp. 108-12
3. Ridker, Paul M., et al. Novel risk factors for systemic atherosclerosis, Journal of the American Medical Association, Vol. 285, May 16, 2001, pp. 2481-85
4. Pradhan, Aruna D., et al. C-reactive protein, interleukin 6, and risk of developing type 2 diabetes mellitus. Journal of the American Medical Association, Vol. 286, July 18, 2001, pp. 327-34
5. Brown, Phyllida. A mind under siege. New Scientist, June 16, 2001, pp. 34-37
6. O'Byrne, K.J. and Dalgleish, A.G. Chronic immune activation and inflammation as the cause of malignancy. British Journal of Cancer, Vol. 85, No. 4, August 2001, pp. 473-83
7. 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
8. Helm, R.M. and Burks, A.W. Mechanisms of food allergy. Curr Opin Immunol, Vol. 12, No. 6, December 2000, pp. 647-53
9. Frustaci, Andrea, et al. Histological substrate of atrial biopsies in patients with lone atrial fibrillation. Circulation, Vol. 96, August 19, 1997, pp. 1180-84
10. Frustaci, Andrea. Personal communication to Hans Larsen, July 23, 2001
11. Frustaci, Andrea, et al. Marked elevation of myocardial trace elements in idiopathic dilated cardiomyopathy compared with secondary cardiac dysfunction. Journal of the American College of Cardiology, Vol. 33, May 1999, pp. 1578-83
12. Lorscheider, Fritz and Vimy, Murray. Mercury and idiopathic dilated cardiomyopathy. Journal of the American College of Cardiology, Vol. 35, March 1, 2000, p. 819
13. Chimenti, C., et al. Inflammatory left ventricular microaneurysms as a cause of apparently idiopathic ventricular tachyarrhythmias. Circulation, Vol. 104, July 10, 2001, pp. 168-73
14. Wojnicz, R., et al. Randomized, placebo-controlled study for immunosuppressive treatment of inflammatory dilated cardiomyopathy. Circulation, Vol. 103, July 3, 2001, pp. 39-45
15. Parillo, Joseph. Inflammatory cardiomyopathy (myocarditis). Circulation, Vol. 103, July 3, 2001, pp. 4-5 (Editorial)
16. Chrousos, G.P. Stress, chronic inflammation, and emotional and physical well-being: concurrent effects and chronic sequelae. Journal of Allergy and Clinical Immunology, Vol. 106 (suppl 5), November 2000, pp. S275-91
17. Lutgendorf, S., et al. Effects of relaxation and stress on the capsaicin-induced local inflammatory response. Psychosomatic Medicine, Vol. 62, July-August 2000, pp. 524-34
18. MacIntyre, D.L., et al. Markers of inflammation and myofibrillar proteins following eccentric exercise in humans. European Journal of Applied Physiology, Vol. 84, March 2001, pp. 180-86
19. Nadarajah, V., et al. Localized cellular inflammatory responses to subcutaneously implanted dental mercury. Journal of Toxicology and Environmental Health, Vol. 49, October 11, 1996, pp. 113-25
20. Pignatelli, B., et al. Helicobacter pylori eradication attenuates oxidative stress in human gastric mucosa. American Journal of Gastroenterology, Vol. 96, June 2001, pp. 1758-66
21. Jartti, T. Asthma, asthma medication and autonomic nervous system dysfunction. Clinical Physiology, Vol. 21, March 2001, pp. 260-69

22. Roupe van der Voort, C., et al. Stress induces increases in IL-6 production by leucocytes of patients with the chronic inflammatory disease juvenile rheumatoid arthritis: a putative role of alpha(1)-adrenergic receptors. Journal of Neuroimmunology, Vol. 110 October 2, 2000, pp. 223-29
23. Allman, F.D., et al. Time-dependent changes in norepinephrine-induced left ventricular dysfunction and histopathologic condition. Journal of Heart Lung Transplant, Vol. 17, October 1998, pp. 991-97
24. Miller, L.E., et al. The loss of sympathetic nerve fibers in the synovial tissue of patients with rheumatoid arthritis is accompanied by increased norepinephrine release from synovial macrophages. FASEB Journal, Vol. 14, October 2000, pp. 2097-107
25. Harrison's Principles of Internal Medicine, 12th edition, 1991, McGraw-Hill, p. 980
26. Friman, G. and Wesslen, L. Special feature for the Olympics: effects of exercise on the immune system: infections and exercise in high-performance athletes. Immunol Cel Biol, Vol. 78, No. 5, October 2000, pp. 510-22
27. Gnarpe, H., et al. Chlamydia pneumoniae and myocarditis. Scand J Infect Dis Suppl, Vol. 104, 1997, pp. 50-2
28. Phillips, J.I. and Shor, A. Association between Chlamydia pneumoniae and atherosclerotic lesions. Cardiovascular Journal of South Africa, Vol. 12, February-March 2001, pp. 42-46
29. Blessing, Erwin, et al. Chlamydia pneumoniae induces inflammatory changes in the heart and aorta of normocholesterolemic C57BL/6L mice. Infection and Immunity, Vol. 68, August 2000, pp. 4765-68
30. Ameriso, S.F., et al. Detection of Helicobacter pylori in human carotid atherosclerotic plaques. Stroke, Vol. 32, February 2001, pp. 385-91
31. Horman, P., et al. Fungal myocarditis in acquired immunodeficiency syndrome. Arch Mal Coeur Vaiss, Vol. 85, February 1992, pp. 203-08 [article in French]
32. Fairweather, D., et al. From infection to autoimmunity. Journal of Autoimmunology, Vol. 16, May 2001, pp. 175-86
33. D'Adamo, Peter J. Eat Right 4 Your Type, 1996, G.P. Putnam's Sons, NY 10014
34. Ridker, P.M., et al. Long-term effects of pravastatin on plasma concentration of C-reactive protein. The Cholesterol and Recurrent Events (CARE) Investigators. Circulation, Vol. 100, 1999, p. 230
35. Compendium of Pharmaceuticals and Specialties, 35th Edition, Canadian Pharmacists Association, 2000, pp. 1258-60
36. Sesmilo, Gemma, et al. Effects of growth hormone administration on inflammatory and other cardiovascular risk markers in men with growth hormone deficiency. Annals of Internal Medicine, Vol. 133, July 18, 2000, pp. 111-22
37. Miller, Chuck. Personal communication, August 1, 2001
38. Vanderhaeghe, Lorna R. And Bouic, Patrick J.D. The Immune System Cure, 1999, Prentice Hall Canada, Don Mills, ON
39. Bouic, P.J. and Lamprecht, L.H. Plant sterols and sterolins: a review of their immune-modulating properties. Alternative Medicine Review, Vol. 4, June 1999, pp. 170-77
40. Bouic, P.J., et al. The effects of B-sitosterol (BSS) and B-sitosterol glucoside (BSSG) mixture on selected immune parameters of marathon runners: inhibition of post marathon immune suppression and inflammation. International Journal of Sports Medicine, Vol. 20, May 1999, pp. 258-62
41. Gupta, I., et al. Effects of Boswellia serrata gum resin in patients with ulcerative colitis. European J Med Res, Vol. 2, January 1997, pp. 37-43
42. Gerhardt, H., et al. Therapy of active Crohn's disease with Boswellia serrata extract H 15. Z Gastroenterol, Vol. 39, January 2001, pp. 11-17 [article in German]
43. Gupta, I., et al. Effects of Boswellia serrata gum resin in patients with bronchial asthma: results of a double-blind, placebo-controlled, 6-week clinical study. European J Med Res, Vol. 3, November 17, 1998, pp. 511-14
44. Murray, Michael and Pizzorno, Joseph. Encyclopedia of Natural Medicine, revised 2nd edition, 1998, Prima Publishing, Rocklin, CA 95677, pp. 770-89
45. Srimal, R. and Dhawan, B. Pharmacology of diferuloyl methane (curcumin), a non-steroidal anti-inflammatory agent. J Pharm Pharmac, Vol. 25, 1973, pp. 447-52
46. Cohen, A. and Goldman, J. Bromelain therapy in rheumatoid arthritis. Pennsylvania Medical Journal, Vol. 67, 1964, pp. 27-30
47. Srivastava, K.C. and Mustafa, T. Ginger (Zingiber officinale) and rheumatic disorders. Medical Hypothesis, Vol. 29, 1989, pp. 25-28
48. Mabey, Richard, editor, et al. The New Age Herbalist, 1988, Collier Books, NY 10022, p. 84
49. Ageel, A.M., et al. Experimental studies on antirheumatic crude drugs used in Saudi traditional medicine. Drugs Exp Clin Res, Vol. 15, No. 8, 1989, pp. 369-72
50. Kremer, J., et al. Fish oil supplementation in active rheumatoid arthritis: a double-blinded, controlled cross-over study. Annals of Internal Medicine, Vol. 106, 1987, pp. 497-502
51. Kelley, D.S. Alpha-linolenic acid and immune response. Nutrition, Vol. 8, 1992, pp. 215-17
52. Murray, Michael T. Encyclopedia of Nutritional Supplements, 1996, Prima Publishing, Rocklin, CA 95677, p. 397

53. Isolauri, Erika. Probiotics in human disease. American Journal of Clinical Nutrition, Vol. 73 (suppl), June 2001, pp. 1142S-46S

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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