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Editorial

Dietary advice can be a bit of a minefield. Not long ago fast-food outlets were chastised for using lard in the preparation of French fries. They switched to partially hydrogenated vegetable oils and touted the health benefits of doing so. It now turns out that this may not have been such a good move after all as partially hydrogenated vegetable oils are high in trans-fatty acids which are now known to be very unhealthy.

In a similar vein, government agencies and heart associations have long advocated that people replace saturated (animal) fats in their diet with polyunsaturated fats – preferably vegetable oils. In this issue we report that researchers have found that people with a high blood level of free fatty acids are at increased risk for sudden cardiac death. Dr. Alexander Leaf of the Harvard Medical School believes that it is the omega-6 fatty acids found in vegetable oils such as corn, safflower, sunflower, and peanut oils that are responsible for initiating the arrhythmias leading to sudden death. Fish oils, on the other hand, exert a protective effect in amounts as low as 600-1000 mg/day (EPA+DHA). It is indeed unfortunate that the organizations advocating an increase in the intake of polyunsaturated fatty acids did not distinguish between omega-6 fatty acids that appear to promote sudden death and omega-3 fatty acids (EPA and DHA) which prevent it.

More evidence that the wrong foods can indeed harm or even kill you comes from a recent study of a newly discovered disorder, syndrome X. Syndrome X is sweeping the civilized world and is a major cause of diabetes and heart disease. There is substantial evidence that syndrome X is related to today's excessive consumption of sugar, particularly from soft drinks and processed foods.

The bottom line is avoid processed foods, reduce your intake of sugar, and eat more fish or take a fish oil supplement.

*Yours in health,
Hans Larsen*

October Highlights

Cataracts and multivitamins	p. 2
Prostate cancer and osteoporosis	p. 3
Crohn's disease and lack of antioxidants	p. 3
Vitamin E and homocysteine	p. 4
Carnitine and thyroid hormone	p. 4
Low cholesterol levels in older people	p. 5
New risk factor for sudden death	p. 5
Beware of syndrome X	p. 6
Angioplasty and inflammation	p. 7
Newsbriefs	p. 8
The AFIB Report	p. 9

LETTERS TO THE EDITOR

I have polymyalgia and have been on 5 mg of prednisone daily for the past 18 months. What do you suggest for relief of muscle pain and stiffness especially in the morning. I cannot tolerate NSAIDs.

MS, USA

Editor: *Polymyalgia Rheumatica is an inflammatory disease and as such may respond*

well to the anti-inflammation protocol described in the September 2001 issue of *The AFIB Report*

(<http://www.yourhealthbase.com/afibreport.html>). I would try this for 4 to 6 weeks. The dietary considerations are likely just as important as the supplements. However, to get things rolling I would suggest you start the time-release vitamin C and the Moducare. Please note that Moducare might interfere with prednisone so you should not take both at the same time.

Could you tell me more about the treatment of root canals with erbium laser?

JG, USA

Editor: The erbium laser treatment is done in California at the Center for Advanced Dentistry. You can find out more about them at <http://www.advancedental.com>.

Read your excellent article on fish oils. I have always been a believer in using whole foods

rather than fragmented foods and I view any of the oils as fragmented foods. Couldn't the benefits you identify from fish oils be obtained from fish? If so, which fish do you recommend and in what amounts? Also, why couldn't soaked flaxseeds be ingested rather than flaxseed oil for a plant source of omega-3 fatty acids?

RK, USA

Editor: The benefits obtained from fish oils can equally well be obtained from fatty fish such as salmon, herring, sardines, menhaden, halibut, tuna, and mackerel. One or two servings of these fish per week will provide very significant benefits. Unfortunately, some of these fish notably tuna and king mackerel can be quite high in mercury somewhat cancelling out the benefits. This is why I usually prefer molecular distilled fish oil as a supplement.

Flaxseed, provided it has not been stored in conditions that would make it go rancid, is an equally good source of plant-based omega-3. However, it is usually easier to ensure non-rancid oil than it is to ensure non-rancid seeds.

ABSTRACTS

Cataracts and multivitamins

SYDNEY, AUSTRALIA. Several studies have found that vitamin C and vitamin E delay the development of cataracts. Now researchers at the University of Sydney report that supplementing with multivitamins especially vitamin A and vitamin B complex also have a protective effect. Their study involved 2873 men and women between the ages of 49 and 97 years who had completed food frequency questionnaires including type, dose, and duration of supplement usage. The researchers checked each participant for the presence of cataracts (cortical, nuclear or posterior subcapsular) using lens photography and a standard grading procedure.

After adjusting for age, gender, hypertension, diabetes, smoking, sun exposure, education, and use of steroids the researchers conclude that vitamin A and certain B vitamins lower the risk of nuclear and cortical cataract development substantially. Thiamin (vitamin B1) was found to

lower the risk of nuclear cataract by 40 per cent and the risk of cortical cataract by 30 per cent in doses greater than 4.4 mg/day. Folic acid and vitamin B12 were found to be strongly protective against both cortical and nuclear cataracts (40-70 per cent risk reduction). Vitamin A was found to be highly protective against nuclear cataract particularly if taken in doses of 3000 micrograms/day (15,000 IU) or greater (risk reduction of 90 per cent). Riboflavin (vitamin B2) and, to a lesser extent, niacin (vitamin B3) were also found to be somewhat protective against cortical cataract.

The researchers conclude that dietary vitamin supplement use is associated with a reduced incidence of both nuclear and cortical cataract and could delay their onset.

Kuzniarz, Maciek, et al. Use of vitamin supplements and cataract: the Blue Mountains Eye Study. American Journal of Ophthalmology, Vol. 132, July 2001, pp. 19-26

Prostate cancer and osteoporosis linked

BOSTON, MASSACHUSETTS. Men with advanced prostate cancer undergoing hormone treatment or castration (androgen-deprivation therapy) experience rapid bone loss markedly exceeding that associated with menopause in women. This bone loss, in turn, leads to a two- to six-fold increase in bone fractures especially those involving the hip. Medical doctors at the Massachusetts General Hospital now warn that the osteoporosis and fracture risk is magnified by the fact that many men with advanced prostate cancer have osteoporosis or low bone density before they even start androgen-deprivation therapy.

The study involved 41 men with locally advanced, lymph node positive or recurrent prostate carcinoma with no evidence of bone metastases. The men underwent dual-energy x-ray absorptiometry (DXA) and quantitative computed tomography (QCT) to determine their bone density. The prostate cancer patients

tended to have significantly lower bone density in the hip and lumbar spine area than did age-matched controls and young adult men. The QCT values for trabecular bone mineral density of the lumbar spine indicated that 63 per cent of the 41 men had osteoporosis. Seventeen per cent of the men had a vitamin D deficiency and 59 per cent had a calcium intake below the recommended daily intake of 800 mg/day. The researchers point out that a vitamin D deficiency is a risk factor for both prostate cancer and osteoporosis. They also emphasize that there is no established causal relation between prostate cancer risk and calcium intake above 800 mg/day. They conclude that prostate cancer patients should be routinely screened for bone mineral density before androgen-deprivation therapy is initiated.

Smith, Matthew R., et al. Low bone mineral density in hormone-naive men with prostate carcinoma. **Cancer**, Vol. 91, June 15, 2001, pp. 2238-45

Crohn's disease patients lack antioxidants

TORONTO, CANADA. Researchers at the Toronto General Hospital report that patients with Crohn's disease (CD) suffer from oxidative stress and have significantly lower blood levels of antioxidants than do healthy controls. Their study involved 37 non-smoking CD patients (22 women and 15 men) and 37 healthy controls matched for age, sex, and body mass index. Thirty per cent of the CD patients had active disease and 78 per cent were taking one or more medications to treat CD. All participants underwent breath analysis tests (pentane and ethane) to determine the degree of lipid peroxidation (fat oxidation) in their system. A high degree of lipid peroxidation is an indication that the body's antioxidant defenses are overwhelmed and that the body is suffering oxidative stress. Blood plasma levels of the common antioxidants, vitamin C and vitamin E,

carotenes, glutathione peroxidase and selenium, were also measured.

The researchers conclude that CD patients have a significantly higher level of lipid peroxidation than do healthy people. This applies even to patients whose disease is in remission. CD patients also have significantly lower levels of vitamin C (ascorbic acid), alpha-carotene, beta-carotene, lycopene, and beta-cryptoxanthin. There were no significant differences in vitamin E (alpha-tocopherol), selenium, and glutathione levels between patients and controls. The researchers recommend that a study be made of the effects of antioxidant supplementation in CD patients.

Wendland, Barbara E., et al. Lipid peroxidation and plasma antioxidant micronutrients in Crohn disease. **American Journal of Clinical Nutrition**, Vol. 74, August 2001, pp. 259-64

Sun exposure prevents prostate cancer

STAFFORDSHIRE, UNITED KINGDOM. British researchers have confirmed that exposure to sunlight helps prevent prostate cancer. Their study involved 210 men diagnosed with prostate cancer and 155 men with an enlarged prostate,

but no prostate cancer (controls). The men were interviewed in order to estimate their lifetime sun exposure. Men with the lowest exposure were found to have a three times greater incidence of prostate cancer than did

men with a high lifetime exposure. Sunburns in childhood were found to be particularly protective with men having had one or more childhood sunburns being six times less likely to develop prostate cancer than men who had not experienced childhood sunburns.

A history of regular foreign holidays, presumably in sunnier climes, also had a protective effect with men having had such holidays having a 60 per cent lower risk of prostate cancer. Regular sun bathing was also found to be protective. The risk of prostate cancer was not associated with skin type, hair colour or eye colour, and the associations with sun exposure were not

affected by including occupation, vasectomy or dietary factors in the analysis.

The researchers are not sure why sun exposure is protective, but speculate that vitamin D and parathyroid hormone may somehow be involved.

Editor's Note: Excessive sun exposure has been linked to an increased risk of certain non-melanoma skin cancers. These cancers, however, are rarely fatal whereas prostate cancer often is. So on balance, cultivating a healthy suntan is still a good idea.

Luscombe, Christopher J., et al. Exposure to ultraviolet radiation: association with susceptibility and age at presentation with prostate cancer. The Lancet, Vol. 358, August 25, 2001, pp. 641-42 (research letter)

Vitamin E and homocysteine

IOWA CITY, IOWA. There is substantial evidence that homocysteine (a sulfur-containing amino acid) promotes atherosclerosis and hypertension through increased oxidative stress and by causing a dysfunction of the lining of the arteries (endothelial dysfunction) that results in reduced arterial blood flow. Earlier research has shown that vitamin C can reverse endothelial dysfunction.

Researchers at the University of Iowa now report that vitamin E has a similar beneficial effect. Their randomized, double-blind, placebo-controlled clinical trial involved 10 healthy volunteers who each went through the four phases of the trial separated by at least a one-week washout period. The four experimental protocols were:

- oral placebo at 7 a.m.
- L-methionine, 100 mg/kg bodyweight at 7 a.m.
- vitamin E, 1200 IU at midnight followed by placebo at 7 a.m.

- vitamin E, 1200 IU at midnight followed by methionine at 7 a.m.

L-methionine is known to sharply increase blood levels of homocysteine. Blood level of homocysteine and blood flow through the forearm was measured six to eight hours after placebo or methionine administration. The forearm blood flow was measured after an infusion of acetylcholine or bradykinin to simulate the body's normal way of ensuring adequate blood flow. The researchers found that the high homocysteine levels caused by the methionine administration very significantly lowered blood flow. The effect was completely eliminated when vitamin E had been previously administered. They conclude that vitamin E, like vitamin C effectively reverses the endothelial dysfunction caused by high homocysteine levels.

Raghuveer, Geetha, et al. Effect of vitamin E on resistance vessel endothelial dysfunction induced by methionine. American Journal of Cardiology, Vol. 88, August 1, 2001, pp. 285-90

Carnitine counteracts adverse effects of thyroid hormone

MESSINA, ITALY. It is estimated that goiter (enlarged thyroid gland) affects about 200 million people worldwide. It is far more common among women and is almost always the result of an iodine deficiency. It can often be corrected by increasing iodine intake. In some cases it is necessary to dampen the activity of the thyroid-stimulating hormone (TSH) in order to treat the goiter. This is usually done by prescribing

thyroid hormone (levothyroxine). Levothyroxine, however, has many adverse effects and also needs constant monitoring to ensure that blood levels are adequate.

Researchers at the University of Messina now report that supplementation with L-carnitine, a quaternary amine which occurs naturally in the body, can eliminate the side effects of thyroxine treatment and reduce the need for constant

monitoring. Their randomized, double-blind, placebo-controlled, crossover trial involved 50 women who were being treated for benign nodular goiter with levothyroxine. The women were allocated to a placebo group, group A that received 2 or 4 grams of carnitine daily for two months out of the six-month trial period or group B which received 2 or 4 grams carnitine daily for four months.

The researchers measured a number of blood parameters and symptoms in the placebo and treatment groups. They conclude that L-carnitine supplementation significantly reduces thyroxine therapy induced weakness, breathlessness, palpitations, nervousness, insomnia, tremors, and elevated heart rate. Carnitine also increases bone mass (bone

mineral density) by about two per cent after just four months of supplementation. In contrast, placebo group members lost bone mass during the trial. The researchers point out that L-carnitine is non-toxic and does not interact with any drugs. They suggest that L-carnitine supplementation (perhaps as little as 1 gram/day) can be of clinical use in goiter therapy and in the treatment of other thyrotoxicosis-related disorders such as amiodarone-related thyrotoxicosis.

Benvenga, Salvatore, et al. Usefulness of L-carnitine, a naturally occurring peripheral antagonist of thyroid hormone action, in iatrogenic hyperthyroidism: a randomized, double-blind, placebo-controlled clinical trial. Journal of Clinical Endocrinology & Metabolism, Vol. 86, August 2001, pp. 3579-94

Low cholesterol levels may not benefit older people

HONOLULU, HAWAII. There is little doubt that high cholesterol levels are detrimental to people aged 65 years or younger. Whether high cholesterol levels also affect older people negatively is a lot less clear. Some studies have shown a similar correlation between cholesterol levels and mortality as found for younger people while others have concluded that high cholesterol levels are actually beneficial to people over 65 years of age.

Researchers at the University of Hawaii now weigh in with a large study designed to settle the controversy. The study involved 3572 Japanese/American men between the ages of 71 and 93 years. The men had their cholesterol levels and numerous other parameters measured in 1991-93. By December 1996 there had been 727 deaths in the group. The researchers found that men with an average total cholesterol level of 5.99 mmol/L (235 mg/dL) had a 35 per cent lower mortality rate (adjusted for age) than did men with an average

cholesterol level of 3.85 mmol/L (150 mg/dL). The correlation between mortality and cholesterol levels was not changed after adjusting for other variables that could affect mortality. The researchers also found that cholesterol levels tended to decline with age – from an average of 5.0 mmol/L (195 mg/dL) in those aged 71 to 74 years to an average of 4.61 mmol/L (180 mg/dL) in those older than 85 years. They also observed that study participants who had maintained low cholesterol levels for 20 years or more had the highest mortality of all.

The researchers cannot explain why high cholesterol levels should equate to longer life in older people, but caution against prescribing cholesterol-lowering drugs to older people.

Schatz, Irwin J., et al. Cholesterol and all-cause mortality in elderly people from the Honolulu Heart Program: a cohort study. The Lancet, Vol. 358, August 4, 2001, pp. 351-55

New risk factor for sudden death

PARIS, FRANCE. Sudden cardiac death is a common occurrence in industrialized countries. There is evidence that a high level of free fatty acids in the blood plasma is an independent risk factor for ventricular arrhythmias and sudden death in people who have suffered a heart attack. Medical researchers at the University of Paris now report that a high level of circulating

free fatty acids (non-esterified) is also a potent risk factor for sudden death in men without cardiovascular disease.

The study involved 5250 men, aged 42 to 53 years at the start of the study in 1967-72. All participants were free of cardiovascular disease at the time of entry. The men were followed for an average of 22 years during which 1601

deaths occurred – 91 of them were classified as sudden cardiac deaths and 145 as fatal heart attacks. Analysis of test data showed that the level of free fatty acids circulating in the blood plasma is a potent risk factor for sudden death. Men with a high level had a 70 per cent higher risk than did men with a low level. Surprisingly, high fatty acid levels were not a risk factor for fatal heart attack. Other prominent risk factors for sudden death were parental sudden death, parental heart attack, smoking, high systolic blood pressure, and high body mass index (obesity). High cholesterol levels increased the risk of sudden death by a relatively modest 18 per cent.

The researchers and Dr. Alexander Leaf, MD of the Harvard Medical School point out that not all fatty acids are detrimental. There is ample evidence that the omega-3 fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), the main components of fish oil are actually highly protective against sudden cardiac death. Dr.

Leaf believes that it is the omega-6 fatty acids found in vegetable oils (corn, safflower, sunflower, and peanut) that are responsible for initiating the arrhythmias leading to sudden death. Fish oils, on the other hand, exert a protective effect in amounts as low as 600-1000 mg/day (EPA+DHA). Dr. Leaf points out that government agencies and heart associations have long been advocating an increased intake of polyunsaturated fatty acids without making any distinction between omega-6 essential fatty acids which appear to promote sudden death and omega-3 fatty acids (EPA and DHA) which prevent it.

*Jouven, Xavier, et al. Circulating nonesterified fatty acid level as a predictive risk factor for sudden death in the population. **Circulation**, Vol. 104, August 14, 2001, pp. 756-61*

*Leaf, Alexander. Plasma nonesterified fatty acid concentration as a risk factor for sudden cardiac death: the Paris prospective study. **Circulation**, Vol. 104, August 14, 2001, pp. 744-45 (editorial)*

Beware of syndrome X

LONDON, UNITED KINGDOM. Syndrome X is the name of a recently discovered disorder which is sweeping the civilized world and if unchecked leads to diabetes and heart disease. A diet high in sugar and saturated fats and frequent high-energy snacks are the main culprits behind the syndrome. The frequent release of insulin resulting from a frequent intake of sugar eventually turns off the liver's control mechanism for dealing with triglycerides and these dangerous fats then flood the blood stream especially after a meal and promote atherosclerosis. They may even start killing insulin-secreting pancreatic cells, a sure road to diabetes. In short, syndrome X is a very bad actor and is on a seemingly inexorable rise. Why?

Dr. Victor Zammit of the Hannah Research Institute in Scotland has no doubt about the reason. Foods high in fructose – and this includes ordinary sugar – are major instigators for syndrome X and from a health standpoint are just as bad or worse than saturated fats. Processed foods are particularly high in fructose and Dr. Zammit suspects that soft drinks, now consumed in vast quantities, could be a very

major cause of syndrome X. A can of cola contains 15 grams of fructose and a 100-gram chocolate bar contains 25 grams. Fructose is also present in fruit and vegetables, but in extremely small amounts and, furthermore, is bound up with complex plant fiber and other healthy nutrients. Dr. Len Storlien of AstraZeneca points out that fish oils suppress the liver's release of harmful triglycerides and thereby help combat syndrome X.

To add insult to injury, food manufacturers in the late 1960s began using corn (maize) syrup as an inexpensive sweetener in most breakfast cereals and many processed foods. Corn syrup is almost pure fructose. At least one prominent scientist believes the food industry needs to be regulated to produce healthy products otherwise the industry may one day have to pay damages to consumers who have been made fatally ill by eating their products. **Editor's Note:** Avoiding saturated fats and sugars, especially fructose and supplementing with fish oil would seem to be the simplest way of preventing syndrome X.

*Vines, Gail. Sweet but deadly. **New Scientist**, September 1, 2001, pp. 26-30*

Alternative medicine in ear, nose and throat disorders

HANOVER, NEW HAMPSHIRE. The American Academy of Otolaryngology – Head and Neck Surgery has established a Committee on Alternative Medicine. This move was partly prompted by the fact that Americans now spend over 23 billion dollars every year on alternative and complementary therapies. The Committee has just published a very interesting review of alternative/complementary therapies used for ear, nose and throat complaints. Among their conclusions:

- Homeopathic remedies can be effective in reducing hay fever symptoms.
- The homeopathic remedy Oscillococcinum probably reduces the duration of influenza.
- *Echinacea (purpurea and pallida, but not angustifolia)* is effective in the prevention and treatment of upper respiratory infections.
- Bromelain is effective in the treatment of sinusitis and has significant anti-inflammatory properties.

- High doses (1 to 2 grams/day) of vitamin C reduce the duration of a cold.
- The homeopathic remedy Vertigoheel is effective in the treatment of vertigo as is Ginkgo biloba (120-240 mg twice daily).
- Ginger (300-1000 mg 2 to 3 times/day) is effective in the treatment of nausea, indigestion and vomiting.
- Hawthorn berry has been shown to be beneficial for atherosclerosis, arrhythmia, and hypertension (160-900 mg extract 2 to 3 times/day).

The Committee report concludes that physicians need to educate themselves about alternative and complementary therapies so that they can responsibly advise their patients “without shutting the door on their interest”.

Asher, Benjamin F., et al. Complementary and alternative medicine in otolaryngology. Laryngoscope, Vol. 111, August 2001, pp. 1383-89 [69 references]

Angioplasty and inflammation

CLEVELAND, OHIO. Angioplasty (percutaneous coronary intervention) is a surgical technique used to free up blocked arteries. It is associated with a fairly significant mortality rate depending on the complexity of the blockage, the presence of unstable angina, and the use of stenting. Researchers at the Cleveland Clinic Foundation now report that the extent of active inflammation in the arteries at the time of surgery is an extremely important additional risk factor for death or heart attack within 30 days after angioplasty.

Their study involved 727 patients who underwent angioplasty between January and July 2000. The patients all had their level of C-reactive protein (CRP) measured prior to the procedure. CRP is a measure of systemic inflammation with higher levels indicating a more severe inflammation. The researchers found that patients with a CRP level greater than 1.01 mg/dL had 3.6 times greater risk of dying or having a heart attack within 30 days than did patients with a CRP level of less than 0.16 mg/dL. The actual rate of death or heart attack among patients with the lowest CRP level was

3.9 per cent as compared to 14.2 per cent for those with the highest level.

The adverse effect of active inflammation was even more pronounced in people with diabetes. Here those with the highest CRP had a 20.3 per cent risk of dying or having a heart attack within 30 days of undergoing angioplasty. The researchers have combined all the known risk factors associated with angioplasty (including CRP levels) into a mathematical model which is quite accurate in predicting the risk of dying or having a heart attack within 30 days of the procedure. The model predicts a death/heart attack rate of anywhere between 1 and 22 per cent depending on the risk factors.

Researchers at the University of Texas include their comments about the findings and suggest it may be desirable to postpone angioplasty in patients with high CRP levels until the underlying inflammation is under control.

Chew, Derek P., et al. Incremental prognostic value of elevated baseline C-reactive protein among established markers of risk in percutaneous coronary intervention. Circulation, Vol. 104, August 28, 2001, pp. 992-97

Yeh, Edward, T.H., et al. C-reactive protein: linking inflammation to cardiovascular complications.

Circulation, Vol. 104, August 28, 2001, pp. 974-75 (editorial)

NEWSBRIEFS

Study launched on “economy class syndrome”. The World Health Organization has received approval to launch a study to determine why the incidence of deep vein thrombosis (DVT) has increased so dramatically among airplane passengers. The study will cost about 12 million dollars and take 2.5 years to complete. **Editor’s Note:** Nobody had ever heard of DVT in airplane passengers until the airlines began cramming them in like sardines in economy class. The solution would seem fairly obvious – give the passengers more room!!
Reuters Health News, August 17, 2001

Hold the incense. Taiwanese researchers warn that burning incense releases smoke laden with cancer-causing chemicals. The researchers found that the level of highly carcinogenic polycyclic aromatic hydrocarbons inside a Taiwanese temple increased to 19 times the level found outside when incense was burned. Incense burning also releases large quantities of other pollutants especially suspended particles. The levels of these were 3 times higher inside the temple than at a busy traffic intersection and 11 times higher than in ambient air.
New Scientist, August 4, 2001, p. 5

Heart attacks kill fitness buffs. Ever since 16 Swedish elite orienteers at the peak of fitness died of heart attacks researchers have been trying to find out why. They now believe they know. Dr. Lars Wesslen of Uppsala University found two strains of the *Bartonella* bacterium in the hearts of four of the five victims he autopsied. *Bartonella* was found in the lungs of the fifth. Overall he found antibodies to *Bartonella* in 31 per cent of orienteers, but in just 7 per cent of ordinary blood donors. Dr. Wesslen speculates that the constant, vigorous exercise that orienteers experience somehow makes them more vulnerable to attacks by *Bartonella*. Dr. Didier Raoult of the University of the Mediterranean in Marseilles says that a growing number of diseases are now being blamed on *Bartonella*.
New Scientist, July 21, 2001, p. 20

Abraham Lincoln poisoned by mercury. Norbert Hirschhorn, a medical historian from New York, believes that Abraham Lincoln suffered from mercury poisoning. This would explain his mood swings and frequent bouts of rage. Apparently Lincoln was prescribed an ‘antidepressant’ called “blue mass”. This medication contained large amounts of mercury – so much so that taking just two tablets a day would have exposed the President to 9000 times the exposure limit set by the US Environmental Protection Agency. Says Hirschhorn, “The blue mass pill caused the very symptoms it was supposed to treat.”
New Scientist, July 21, 2001, p. 21

Obscene drug prices kill children every day. The cost of patented pharmaceutical drugs has now, in many cases, reached levels that put them out of reach of most people in developing countries. One pill for treating malaria (malarone) costs \$4.92 US. The disease kills over one million people every year. One pill for treating HIV-AIDS (flucanazole) costs \$7.66. AIDS kills three million people every year. Some countries like Uganda have chosen to ignore the patents and try to save lives by importing cheaper generic drugs from India. The major pharmaceutical companies are opposed to this practice and are calling for stronger enforcement of patent rights. The issue is due for discussion in global trade negotiations later this year. Meanwhile, another thousand children die every day of dysentery, a disease that is entirely curable with pills costing \$3.89 each (ciprofloxacin).
New Scientist, July 21, 2001, pp. 18-19

Helicobacter pylori – bad guy or good guy? There is ample evidence that the *Helicobacter pylori* bacterium is responsible for most stomach ulcers and that stomach ulcers can be permanently cured by eradicating *H. pylori* with antibiotics. Recent research has concluded that an *H. pylori* infection may also be involved in stomach cancer and cancer of the pancreas. Nevertheless, some researchers are now asking if it is always beneficial to eradicate *H. pylori*. They point out that people often develop GERD

(gastroesophageal reflux disease) after *H. pylori* eradication and also refer to a recent finding that associated *H. pylori* eradication with an increase in esophageal cancer. The general consensus is still that *H. pylori* is a bad actor. Says Dr.

Barry Marshall, the discoverer of the *H. pylori*/ulcer connection, "The risk from *H. pylori* is still four times greater than the risk from eradicating it."
New Scientist, August 11, 2001, pp. 31-33

THE AFIB REPORT

In this issue we conclude the evaluation of the LAF survey results. It has taken a lot longer to interpret the results than I originally estimated, but I believe the effort has been very worthwhile. Thanks again for your input and support! We also take a closer look at the question of stroke prevention for lone afibbers. Should you take aspirin or warfarin? Our findings may surprise you!

SURVEY RESULTS – PART VII

Since publishing part V of the survey results we have received an additional 24 completed questionnaires giving a total sample size of 99. Twenty respondents have chronic LAF, 35 have the vagal variety, 24 the adrenergic variety, and the remaining 20 a mixture of vagal and adrenergic. The additional 24 sets of data means that we should be able to draw more valid conclusions than with just the original 75 sets. So I decided to re-evaluate all variables using all available data.

Severity of Episodes

The average for all respondents with paroxysmal (intermittent) LAF is 127 hours spent in fibrillation over the six-month survey period. The minimum was 0 hours and the maximum 900 hours. In comparison, an afibber with chronic LAF would have spent 4320 hours in fibrillation over a six-month period. Vagal afibbers had the easiest time with an average of 83 hours spent in afib (range: 0-576 hrs). Mixed afibbers were next with an average of 142 hours (range: 0-750 hrs) followed by the adrenergic group at 151 hours (range: 0-900 hrs).

There is a strong, statistically significant correlation between time spent in afib and the number of episodes experienced over the six-month survey period ($r=0.4567$ $p=0.0001$). Adrenergic afibbers had an average of 11 episodes in six months (range: 0-90), vagal 16 episodes (range: 0-150), and those with the mixed variety 30 episodes (range: 0-180).

There is also a statistically significant correlation between total time spent in afib and the average duration of individual episodes ($r=0.2547$ $p=0.02$). The average episode lasted 10 hours for the mixed group (range: 0.1-48 hrs), 13 hours for the vagal group (range: 0.1-168 hrs), and 17 hours for the adrenergic group (range: 0.1-72 hrs).

Effect of Age

There is a statistically significant trend for the time spent in fibrillation to increase with age ($r=0.2632$ $p=0.02$). According to the trend line, the average time spent in fibrillation over the six-month survey period was about 30 hours at age 30 years, 75 hours at age 40, 110 hours at age 50, and 155 hours at age 60.

The average age of vagal afibbers was 49 years, adrenergic 53 years, mixed 54 years, and chronic 59 years. The age difference between vagal and chronic afibbers was statistically significant ($p=0.003$). The age difference between vagal and adrenergic afibbers was not statistically significant nor was the difference between vagal and mixed afibbers. Thus it would appear that the vagal variety is associated with a younger age while the chronic variety is associated with an older age.

Effect of Gender

There were 19 women in our sample. Six (32%) had chronic LAF, 6 (32%) the mixed variety, 5 (26%) adrenergic, and 2 (10%) vagal. This distribution is distinctly different from that of men (18% chronic, 18% mixed, 24% adrenergic, and 40% vagal).

Women with LAF (at least those that responded to the survey) were older than men with LAF. The average age for women was 61 years while that of men was 51 years. This difference was statistically significant ($p=0.001$).

It is tempting to speculate that the same mechanisms (estrogen?) that protects women against heart attacks for an extra 10 years (compared to men) is at work here, but I have no evidence of such a connection. The explanation could simply be that older women seek medical information on the Internet more often than do younger women.

Women with paroxysmal LAF spent less time in fibrillation (43 hours) than men (133 hours) over the six-month survey period. There was no significant difference in the number of episodes, but episodes among women tended to be shorter in duration (4.5 hours versus 15 hours for men). There was no significant difference in the percentage of women and men who were taking drugs to prevent future episodes (69% versus 62%).

Effect of Years of LAF

There was a slight trend for the time spent in fibrillation (over six months) to increase with the number of years since diagnosis of LAF. This trend was not statistically significant ($p=0.1357$). There was also a slight increase in the number of episodes with increasing years of AF, but again not of statistical significance ($p=0.1097$). There was no correlation between the duration of episodes and years of LAF nor was there a statistically significant association between age and years of LAF ($p=0.3774$). The average number of years of LAF was 6 years for vagal, 5 years for adrenergic, 7 years for mixed, and 8 years for chronic. The figure for chronic afibbers may be a bit misleading though in that many have had the condition (without symptoms) for several years prior to being diagnosed through a routine electrocardiogram.

Effect of Aspirin

About half (47%) of all respondents with paroxysmal LAF took aspirin on a daily basis (22% among chronic afibbers). There was no significant difference in episode severity between users and non-users of aspirin. Aspirin users were older than non-users (54 versus 49 years average age) and more likely to be women (22% versus 12%). Unfortunately, the limited data did not allow consideration of these potential biases. Since one would have a positive effect and the other a negative effect it is probably fair to say that aspirin usage has little or no effect on LAF episode severity.

Effect of Digoxin

Only 16% of paroxysmal afibbers had ever been on digoxin (Lanoxin, digitalis) and there was no significant difference in episode severity between those who had been or were on digoxin and those who were not. However, there was a clear difference in digoxin use between intermittent (paroxysmal) and chronic afibbers. Fifty per cent of chronic afibbers had used digoxin as compared to only 16% of intermittent afibbers. This finding lends further support to the contention that digoxin can convert LAF to the chronic form.

There was also a statistically significant difference ($p=0.04$) between the proportion of women (50%) and men (18%) who had been prescribed digoxin. This could, at least partially, explain why more women than men fell into the chronic LAF category (32% versus 18%).

Effect of Amalgam Fillings

Most (76%) paroxysmal afibbers had amalgam (silver) dental fillings as did most chronic afibbers (75%). There was a clear correlation between the time spent in fibrillation and the presence of amalgam fillings. Paroxysmal afibbers without amalgam fillings spent an average of 35 hours in fibrillation over the six-

month survey period while those with amalgam fillings spent 143 hours in afib. This difference was statistically significant ($p=0.04$).

There was also a clear linear relationship between time spent in fibrillation and the number of fillings ($r=0.4173$ $p=0.002$). An afibber with 0 fillings could expect to spend 35 hours in afib while someone with 8 fillings could expect 140 hours and someone with 20 fillings could expect an average 300 hours in afib over a six-month period. Afibbers with amalgam fillings also tended to have more episodes (20) and of longer duration (15 hours) than afibbers without (13 episodes of average duration of 9 hours). These differences were, however, not statistically significant.

The findings that afibbers with amalgams have more severe episodes than those without support the contention that at least part of the inflammatory response underlying LAF is caused by oxidative stress or electrical instability generated by the presence of mercury in the heart tissue.

Effect of Dissimilar Metals in the Mouth

Seventy-six per cent of chronic afibbers (100% of female chronic afibbers) had dissimilar metals (amalgam fillings, gold crowns, bridges) in their mouth as compared to only 44% of paroxysmal afibbers (54% of female paroxysmal afibbers). There was no overall significant difference in episode severity between paroxysmal afibbers with dissimilar metals and those without. It would thus appear that dissimilar metals are primarily a problem with chronic afibbers. However, this conclusion is somewhat confounded by the fact that amalgam fillings and dissimilar metals often go hand in hand.

It would be advisable for afibbers with dissimilar metals to have a measurement made of the galvanic currents in their mouth. This can be done by a holistic dentist who can also advise on remedial action.

Effect of Fish Oil Supplementation

Forty-two per cent of all paroxysmal afibbers supplemented with fish oils (44% among chronic afibbers). Somewhat surprisingly afibbers on fish oil spent more time in fibrillation than did those not taking fish oil (149 hours versus 97 hours over the six-month period). Fish oil users also had more episodes than non-fish oil users (25 versus 13). A closer look at the overall picture reveals that the fish oil group had quite a few "disadvantages" when compared to non-fish oil group. Afibbers who supplemented with fish oil were older (55 years versus 49), less likely to be women (12% versus 20%), and more likely to have amalgam fillings (82% versus 70%). All these factors would be expected to significantly increase the time spent in fibrillation.

An attempt to at least partially account for this bias was made by just considering the 18 respondents who did not have any amalgam fillings. In this case, fish oil supplementation appeared to be beneficial. Fish oil users had only 8 episodes and spent only 24 hours in fibrillation while non-users had 16 episodes and spent 40 hours in fibrillation. Adjusting for age and gender should further improve the picture.

Fish oil users were also significantly more likely to be supplementing with magnesium (76% as compared to only 33% among non-users). The effect of this confounding was eliminated by just considering non-magnesium users. In this group 21% took fish oil while 79% did not. The users of fish oil (average age of 54 years) had an average of 8 episodes and spent 89 hours in fibrillation. The non-users (average age of 45 years) had an average of 16 episodes and spent 106 hours in fibrillation.

Due to the significant confounding of the data it is difficult to draw a firm conclusion as to whether or not fish oils affect the severity of LAF. Nevertheless, considering the excellent stroke protection afforded by fish oils it is probably fair to say that they are overall beneficial to lone afibbers.

Effect of Magnesium Supplementation

Fifty-one per cent of paroxysmal afibbers supplemented with magnesium (50% among chronic afibbers). There was no significant overall difference in episode severity between those who supplemented and those who did not. Magnesium users were quite a bit older (55 years versus 48 years for non-users) and more likely to be women (20% versus 13%). They were also much more likely to be supplementing with fish oil; 63% of magnesium users also used fish oil as compared to only 21% among non-magnesium

users. The effect of this confounding was eliminated by just considering non-fish oil users. In this group 33% took magnesium while 67% did not. The users of magnesium (average age of 55 years) had an average of 6 episodes and spent 65 hours in fibrillation. The non-users (average age of 46 years) had an average of 16 episodes and spent 112 hours in fibrillation. Based on this data it is probably fair to conclude, especially in view of the considerable age difference between the groups, that magnesium is indeed beneficial for afibbers.

Effect of Digestive Problems

Fifty-one per cent of all paroxysmal afibbers had digestive problems (bloating, flatulence, belching) compared to only 28% of chronic afibbers. There was a slight, statistically non-significant trend for afibbers with digestive problems to spend less time in fibrillation than those without digestive problems. Why afibbers with digestive problems should have less severe LAF than those without digestive problems is certainly a mystery and one I have no explanation for at this time.

Effect of Physical Fitness

There was no correlation between episode severity and the level of physical fitness.

Comparison of the Best and the Worst

A rather intriguing way of looking at the survey data is to compare those afibbers (7) who had no LAF episodes over the six-month survey period with those (9) who spent 450 hours or more in afib during the same period. The "best" (zero hours) afib group was younger than the "worst" group (average age of 53 years versus 60 years); they had also had LAF for a shorter period (7 years versus 11 years). The best group was more likely to have the vagal variety of LAF (57% versus 33%). The best group was more likely to take aspirin (43% versus 22%). The worst group was more likely to take fish oil (67% versus 29%) and magnesium (51% versus 43%). Whether this is because the worst group is clearly "sicker" and therefore trying everything or whether it is because fish oil and magnesium have a detrimental effect on LAF severity is not clear, but it is likely to be the former.

All members (100%) of the worst group had amalgam fillings (an average of 15 fillings each) while only 43% of the best group did (an average of 2 fillings each). This again points to the crucial role of amalgam fillings as a major cause of LAF.

Perhaps the most intriguing finding is that the members of the best group were almost twice as likely to have digestive problems than were the members of the worst group (79% versus 50%). I have no explanation for this, but it is consistent with the findings of the entire survey.

This concludes our evaluation of the LAF survey data. I thank you for your input and hope the results have been beneficial to you and have broadened your understanding of LAF. It certainly has been of great benefit to me. There are still a few puzzles I would like to clear up, but I am afraid I do not have the time or the resources to do so at this time.

STROKE PREVENTION IN LAF

Stroke (cerebral infarction, cerebrovascular event) is the third leading cause of death in the United States. It strikes about half a million Americans and kills upwards of 150,000 every year. A stroke involves a sudden interruption of blood flow to the brain. This interruption can be caused by a blood clot (thrombus) that lodges in a small artery in the brain (ischemic stroke) or by the rupture of an artery wall (hemorrhagic stroke). An ischemic stroke is sometimes referred to as a "heart attack of the brain". The interrupted blood flow results in brain cells being starved of oxygen; if the interruption last more than 4 or 5 minutes the cells will die and irreversible damage will occur. If the cells that die are the ones that control your speech or your left arm then these functions will become impaired. If enough cells die (massive stroke) then so will you.

The risk of a stroke increases with age; it is estimated that 5% of the population over 65 years of age will suffer a stroke. A prior stroke, heart disease, diabetes, hypertension, atrial fibrillation, high homocysteine levels, and a bacterial infection of the lining of the heart cavity (endocarditis) are significant risk factors. Major surgery accounts for a large number of ischemic strokes. It is estimated that as many as 25,000 people suffer a stroke every year as a sequel to coronary bypass surgery[1-5].

Stroke and Atrial Fibrillation

Atrial fibrillation is a risk factor for ischemic stroke because of the inefficient pumping action of the atria during fibrillation. The fibrillating atrium basically sits and quivers like a bowl of jelly. This can cause blood to stagnate and if the fibrillation goes on long enough to coagulate and form blood clots (thrombi). If one of these blood clots finds its way to a small artery in the brain a stroke may result. The danger of this happening is actually highest when the fibrillation ceases. The increased pumping action, once the atria gets back to normal, flushes out the heart chamber and with it any newly formed blood clots. This is why anticoagulation with warfarin (Coumadin) and/or heparin is essential prior to cardioversion and for about 3 weeks after.

The stroke risk in patients with non-rheumatic atrial fibrillation has been evaluated in at least 5 major randomized clinical trials designed to evaluate the effects of aspirin and warfarin in stroke prevention. Without treatment the annual stroke incidence in patients under 65 and no risk factors is 1%. This is equivalent to the annual incidence in the general population. In other words, afibbers under 65 years of age with no other risk factors do not have an increased risk of stroke. The incidence of stroke, even with no other risk factors, does however increase with age; it is estimated at 4.3% between the ages of 65 and 75 years and 3.5% above age 75. Having one or more risk factors (hypertension, diabetes, prior stroke or heart attack, angina or congestive heart failure) materially increases the risk to 4.9% under age 65, 5.7% between the ages of 65 and 75 years, and 8.1% above age 75[6,7].

Investigators at the National Registry of Atrial Fibrillation have recently devised a new scheme for predicting stroke risk. This system, CHADS2, assigns a score of 0 to atrial fibrillation patients with no additional risk factors. One point is added for the presence of congestive heart failure, hypertension, age 75 years or older, and diabetes (1 point for each) and 2 points for a history of stroke or TIA (transient ischemic attack). Thus an 80-year-old patient (+1) with hypertension (+1) and a prior stroke (+2) would have a CHADS2 score of 4. The investigators have correlated the CHADS2 score with the actual annual incidence of stroke observed in a large study involving 1733 atrial fibrillation patients. Patients with a score of 0 had a stroke rate of 1.2%, which is equivalent to that found in the general population of the same age. Patients with a score of 1 had a rate of 2.8%, those with a score of 2 a rate of 3.6%, and those with a score of 3 a rate of 6.4%.

So what does with mean? Of most importance to lone afibbers is the conclusion that afibbers under 75 years of age with no additional risk factors have an incidence of stroke equal to that of the general population[8].

Stroke and Lone Atrial Fibrillation (LAF)

Lone atrial fibrillation, by definition, means that there are no underlying heart problems present. So unless you have hypertension, diabetes, are over 75 years of age or have suffered a previous stroke or TIA you are at no greater risk for stroke than the general population[8]. Medical experts are pretty unanimous on this point. Dr. Rodney Falk, MD of Boston University, a world-renowned expert on atrial fibrillation, says that the stroke risk in patients with lone atrial fibrillation is minimal[5]. Professor Michael D. Ezekowitz, MD of the Veterans Administration says, "patients with lone atrial fibrillation are not at higher risk for thromboembolism than the general population and can be managed without anticoagulation or anti-platelet therapy"[9]. Dr. Stephen L. Kopecky of the Mayo Clinic did the first study regarding stroke risk in patients with lone atrial fibrillation. He found that lone afibbers under the age of 60 years had an exceptionally low stroke risk (0.55%) and that this risk varied little whether the fibrillation was paroxysmal or chronic[10].

So why should lone afibbers under 75 years of age with no additional risk factors worry unduly about an increased stroke risk? They probably should not, but many cardiologists and physicians obviously do.

LAF and Anticoagulation

Current official guidelines for stroke prevention state that patients with lone atrial fibrillation and no additional risk factors under the age of 65 years do not need anticoagulation (warfarin) or antiplatelet (aspirin) therapy and there is no evidence that they are beneficial[5]. Those between 65 and 75 years of age are advised to take a daily aspirin and only those over 75 years should be considered for warfarin therapy bearing in mind the increased bleeding risk in older people[5]. In our initial survey of 53 afibbers 5 were prescribed warfarin even though they were under 75 years and had no risk factors. An additional 5 respondents with hypertension were also on warfarin. Fifty per cent of the larger sample of 79 paroxysmal afibbers were taking aspirin. It would seem that the prescribers of anticoagulation and antiplatelet therapy are erring on the side of caution. Surely this is a good thing? Not necessarily, both warfarin and aspirin have very serious side effects that may, in many cases, outweigh their benefits.

Platelet Therapy (Aspirin)

Aspirin inhibits blood clotting by preventing blood platelets from sticking together (aggregation). It is estimated that over 50 million Americans now take a daily aspirin to ward off a stroke or heart attack. There is evidence that aspirin is effective in preventing a second stroke, but much less so in preventing a first stroke[11]. Three trials have evaluated the effect of aspirin therapy on stroke incidence in atrial fibrillation patients. The conclusion is that aspirin reduced the risk of stroke by about 21%. This means that if a patient has a stroke risk of 2.8% (the risk for a 70-year-old afibber with hypertension) without aspirin it would drop to 2.2% with aspirin – significant, but not really that impressive. The participants in the aspirin evaluation trial were generally older, had underlying heart disease and one or more other risk factors so the results may not be applicable to lone atrial fibrillation. So while the benefits of taking a daily aspirin are somewhat dubious, at least for lone afibbers, the risks are anything but dubious.

Aspirin is not innocuous. It can cause serious bleeding in the gastrointestinal tract and can aggravate existing ulcers. The estimated death rate from gastrointestinal (GI) bleeding is 12%. Researchers at Oxford University have released the results of a very large study aimed at establishing the magnitude of aspirin-related bleeding incidents. They carefully studied the results of 24 major randomized clinical trials involving almost 66,000 participants. They conclude that when treated for a year 2.47% of aspirin users develop GI bleeding as compared to 1.42% among placebo users. Put in terms of the 50 million American now taking aspirin this means that the excess incidence of GI bleeding attributable to aspirin would be 525,000 and the excess mortality would be 63,000 every year. The researchers also investigated whether lower dosages of aspirin would be safer. They found that they were not. The incidence of GI bleeding among low-dose aspirin users was 2.30% compared with 1.45% for placebo users. Somewhat surprisingly, the study also found that enterically-coated or otherwise modified formulations were no safer than standard aspirin. The increase in GI bleeding among users of modified formulations was 93% as compared to 68% for all aspirin users and 59% for low-dose users. The researchers conclude that patients and their physicians need to consider the trade-off between the benefits and harms of long-term aspirin use. Dr. Martin Tramer of the Geneva University Hospitals in Switzerland wholeheartedly agrees with this conclusion and adds, "It may be more appropriate for some people to eat an apple rather than an aspirin a day." [11,12]

So while taking a daily aspirin may help protect against heart attacks and strokes (particularly the second incident) its use is definitely not without risk. An alternative approach, which you may wish to discuss with your physician, is to take an aspirin at the start and end of an afib episode and for a week or so after if the episode is a long one (greater than 24 hours). The protective effect of aspirin lasts for about a week. This suggested approach assumes, of course, that you can actually feel when you have an episode.

Anticoagulation with Warfarin (Coumadin)

Most of the proteins involved in blood clotting rely on vitamin K for their synthesis. Warfarin (Coumadin) destroys vitamin K and basically turns users into pseudo-hemophiliacs.

At least 5 major trials have investigated the effectiveness of warfarin in prevention of stroke in patients with atrial fibrillation. The majority of participants in all of these trials had underlying heart disease and some had suffered strokes or heart attacks prior to the trials. Pooling all of the results shows that anticoagulation with warfarin to an INR (International Normalized Ratio) of between 2.0 and 4.0 resulted

in a reduction in stroke risk of 64%[5]. This means that if one's stroke risk without warfarin was say 2.8% (the risk for a 70-year-old afibber with hypertension) then one could reduce this to about 1.0% (actual trial results showed a decrease to 1.7% only so the 64% may be overstated in some cases).

Because warfarin thins the blood to the point where it is difficult for the body to stop even a very minor internal bleeding incident major bleeding and hemorrhagic stroke are very real risks of warfarin therapy especially among older people. The second Stroke Prevention in Atrial Fibrillation (SPAF-II) study found that while warfarin therapy lowered ischemic stroke risk by 3.6% it also increased the risk of major bleeding by 4.2% in patients over 75 years of age[13].

Another very important consideration when evaluating the results of clinical trials of warfarin is the fact that INR control in daily practice is rarely as good as that obtained in a strictly controlled clinical trial. A study of 2376 patients receiving warfarin reported an incidence of life-threatening or fatal hemorrhage (bleeding) of 3.38% per year in those over 80 years. This excess risk of treatment exceeded the reduction in the rate of major disabling or fatal ischemic strokes resulting from the warfarin therapy[14].

Some researchers feel that the bleeding problem and the deaths resulting from it makes the benefit/risk ratio for warfarin therapy somewhat dubious. A review by the prestigious Cochrane Institute concluded, "the margin between benefit and harm for warfarin prophylaxis in patients with chronic non-valvular atrial fibrillation is uncomfortably thin. The low absolute risk reductions observed in trials would likely be overwhelmed in less controlled settings by problems associated with the use of warfarin."[15]

Just recently British researchers concluded that although anticoagulation with warfarin is more effective than antiplatelet therapy with aspirin, "major bleeding was more common in patients receiving anticoagulation, and the evidence to support long term anticoagulation is weak."[16]

Another problem with warfarin is that it can interact with many common medications, herbs and even foods. This can lead to higher or lower INR values with a concomitant increased bleeding risk or decreased stroke protection[17,18]. The interaction with Tylenol (acetaminophen, Paracetamol) is particularly serious and can raise the INR to 6.0 or higher, a serious bleeding risk[19,20]. To add insult to injury, warfarin also increases the risk of osteoporosis and fractures of the spine and ribs. A recent study at the Mayo Clinic found that women who had been taking warfarin for a year or more had a 5.5 times greater risk of having a spinal fracture and a 3.4 times greater risk of a rib fracture[21].

So all in all, aspirin and especially warfarin are not really that great a choice for stroke protection in lone afibbers – particularly as most of us don't even need additional stroke protection. So how can we protect ourselves? Fortunately, there are many highly effective alternative methods for minimizing your risk of stroke whether you are an afibber or not.

ALTERNATIVE METHODS OF STROKE PROTECTION

There are numerous alternative ways of obtaining stroke protection equal to or better than that afforded by aspirin and warfarin – and without the side effects. These methods have not been evaluated specifically in atrial fibrillation patients, but since lone afibbers with no additional risk factors have no greater stroke risk than the general population it would seem reasonable that the benefits would be the same.

Antioxidants

There is ample evidence that an adequate intake of dietary antioxidants is crucial in stroke prevention:

- Finnish researchers have observed that a low blood level of lycopene increases stroke risk by a factor of three[22].
- British researchers have found that people with extremely low levels of vitamin C have twice the risk of dying from a stroke than do those with normal levels[23,24].

- A recent study carried out by the US Centers for Disease Control and Prevention involving over 1 million adult Americans concluded that people who take multivitamins and antioxidants (vitamins A, C or E) have a 15% lower risk of dying from heart disease or stroke[25].
- Finnish researchers have found that a high intake of beta-carotene significantly reduces the risk of a stroke among male smokers[26].
- Vitamin E has been found to inhibit platelet aggregation and adhesion – important in preventing stroke (aspirin has a similar effect)[27].
- Two large studies carried out at Harvard Medical School concluded that people who had taken 100 IU of vitamin E for 2 years or more had a 30% lower incidence of ischemic stroke – this is better than the protection afforded by aspirin[28].

Fish Oil

A 1995 study concluded that men who ate fish 5 or more times per week had a 40% lower risk of having a stroke than did men who ate fish less than once a week. Researchers at the Harvard Medical School and the Brigham and Women's Hospital now report that the benefits of fish consumption are even more spectacular for women. Their just completed study involved 79,839 female nurses who were between the ages of 34 and 59 years at the start of the study in 1980. After 14 years of follow-up a total of 574 strokes had occurred in the group. Most of the strokes (303) were ischemic (caused by a blood clot). There were also 181 hemorrhagic strokes (caused by a ruptured artery) and 90 strokes of undetermined origin.

After adjusting for age, smoking and other cardiovascular risk factors the researchers concluded that women who ate fish once a week lowered their risk of having a stroke of any kind by 22% and those who consumed fish 5 or more times per week reduced their risk by 52%. They ascribe the protective effect of fish consumption to the commensurate intake of fish oils (omega-3 fatty acids). They estimate that women whose intake of fish oils is 0.5 gram/day or more have a 30% lower risk of suffering a stroke than do women whose intake is below 0.1 gram/day. There was no evidence that women with a high fish or fish oil consumption have an increased risk of hemorrhagic stroke. The researchers believe that the protective effects of fish oils are due to their ability to inhibit platelet aggregation, lower blood viscosity, suppress the formation of leukotrienes, reduce fibrinogen levels, and reduce blood pressure levels and insulin resistance. They also note that the beneficial effects of fish consumption were substantially more pronounced among women who did not take aspirin on a regular basis[29].

Fish oil can be a double-edged sword though. Some fish like swordfish, tuna, shark, king mackerel, and red snapper can have mercury levels exceeding the current US standard of 1.0 ppm. Many more species exceed the New Zealand limit of 0.5 ppm. Salmon usually has very low levels of mercury. If you plan on supplementing with fish oil it is a good idea to ask the manufacturer to certify the maximum level of mercury found in their product and obtain a statement that they use molecular distillation to remove impurities from their product. I asked Pronova, a major Norwegian producer of fish oils, for certification. They stated that their fish oils are molecular distilled and are certified to contain less than 0.1 ppm of mercury (10 times lower than the allowable limit). The actual mercury content of their products is even lower at 0.01 ppm or less. Pronova oils are used in the manufacture of such brands as Coromega, Omacor and Pikasol.

Ginkgo Biloba

Ginkgo biloba increases blood flow to the brain and inhibits platelet activation and adhesion. Animal experiments have shown that it materially reduces the extent of damage if an ischemic stroke does happen[30,31]. Ginkgo biloba may interact with aspirin and warfarin to increase bleeding tendency so should not be taken together with these medications[32].

Folic Acid

A high blood level of homocysteine doubles the risk of suffering an ischemic stroke. Homocysteine levels can be safely and effectively lowered by supplementing with folic acid and vitamins B6 and B12[2,3].

Lifestyle and Diet

Smokers have more than twice the risk of having a stroke than do non-smokers so if you are a smoker you can reduce your stroke risk by 50% by quitting[33]. Doctors at a Birmingham hospital have

concluded that lifelong exercise provides a very significant protection against stroke. People who had been involved in vigorous exercise (running, swimming, cycling, playing tennis or squash) between the ages of 15 and 40 were found to have a 5 times lower risk of suffering a stroke than had people who had never done any vigorous exercise[34]. Medical doctors at the Boston University School of Medicine have found that older men who maintain a medium level of physical activity reduce their risk of having a stroke by more than 50%[35].

Habitual tea drinking provides strong protection against suffering a stroke. This is the major finding of a study published by the Dutch National Institute of Public Health and Environmental Protection. An analysis of dietary data showed that men who consumed more than 4.7 cups of tea per day had a 69% lower risk of having a stroke than did men who drank 2.6 cups per day or less. The Dutch researchers believe that the protective effect of black tea is due to its high content of flavonoids (mainly quercetin). They calculate that men with a daily flavonoids intake of 28.6 mg or more have a 73% lower risk of suffering a stroke than do men with a lower intake (less than 18.3 mg/day). The researchers have previously reported that a high intake of flavonoids also protects elderly men against coronary heart disease. A high intake of beta-carotene from vegetables and the consumption of solid fruits (e.g. apples) also showed some association with a lower stroke risk, but not enough to be statistically significant[36].

Should you use alternative methods rather than conventional ones (aspirin and warfarin) for stroke protection? The choice is entirely yours. At least you now know that you do have options.

This is it for this edition of The AFIB Report. In the next issue we will carry on with our review of supplements of particular benefit for lone afibbers.

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