THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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Several afibbers have expressed the belief that there might be a cyclical nature to the timing of afib episodes and that this possible cyclical connection may be linked to hormonal fluctuations. I have now tabulated the results of a survey carried out in August with the aim of establishing whether a hormonal connection is likely.

Twenty-five female afibbers participated in the survey. This number of respondents, unfortunately, was not large enough to establish a connection (or lack of same) between hormonal fluctuations and afib episodes. My literature search did uncover a clear association between autonomic nervous system balance and fluctuations in estrogen and progesterone levels, but no reference to LAF and hormonal fluctuations. A much larger survey is clearly required to prove or disprove a hormonal connection.

Two or perhaps 3 afibbers have now managed to eliminate afib episodes through dietary changes. In this issue we bring you the story of Erling Waller "My Way Back to NSR". I am sure you will find it as inspiring and encouraging as I have.

Yours in health and sinus rhythm,
Hans Larsen

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The Hormone Connection

Several afibbers have expressed the belief that there might be a cyclical nature to the timing of afib episodes and that this possible cyclical connection may be linked to hormonal fluctuations. To the best of my knowledge, the only two hormones that fluctuate on a regular (monthly) basis are estrogen and progesterone. The levels of these two hormones are highest in menstruating women and, at least during the luteal phase, are considerably higher than the levels found in men[1].

There is no evidence that I know of indicating that estrogen or progesterone levels fluctuate in men. This may be because they don't or it may be because nobody has investigated whether they do. In any case, it seemed most appropriate to include just women in a survey aimed at uncovering a possible connection between LAF and hormonal fluctuations.

A. Survey Results

A total of 25 female afibbers (out of 62) answered the August 2002 questionnaire. Three had underlying heart disease, two had undergone a successful maze procedure, and two had undergone radiofrequency ablation therapy (one successful and one not).

The average age of the respondents was 56 years (range of 28 to 71 years) and the average age at diagnosis was 49 years (range of 14 to 68 years). The majority (50%) had the mixed variety of LAF followed by vagal at 40%. Only 10% (2 respondents) had the adrenergic form.

A little less than half (48%) of the respondents were taking drugs in order to prevent or reduce the severity of episodes. There was an almost statistically significant trend ($p=0.07$) for women on drugs to have longer lasting episodes than women who were not on drugs. The most popular drug was flecainide (4 respondents) followed by beta-blockers (3 respondents) and propafenone (2 respondents).

There were statistically significant trends for women taking pharmaceutical drugs to be childless, to be postmenopausal, to have undergone a hysterectomy, to have had bilateral oophorectomy, and to be on hormone replacement therapy. This could perhaps be taken as an indication that women who have had a greater involvement with the medical system are more likely to be taking drugs in an attempt to manage their LAF.

Seven respondents (28%) felt there was a definite connection between hormonal status and episodes while 14 (56%) felt that there was no association. The remaining 16% were not sure or thought that there might be a connection. Vagal afibbers were more likely to feel that there was a connection.

Eight respondents (32%) felt there was a definite association between their first LAF episode and major hormonal fluctuations (childbirth, onset of menopause, beginning of hormone replacement therapy).

Both commercially raised chicken and soy products may contain compounds that mimic the effects of estrogen. The 25 respondents consumed chicken an average of 5 times per month and soy products 12 times per month. There was no indication that consuming chicken or soy products had any effect on episode severity; however, there was a clear inverse association between chicken consumption and soy consumption with women consuming the most chicken consuming the least soy products.

Most (75%) of respondents had given birth to one or more children while the remaining 25% were childless. Most (64%) were postmenopausal with 20% being premenopausal and 16% being menopausal (perimenopausal). Postmenopausal women were significantly more likely to be on drugs to manage LAF. Four out of five premenopausal women experienced PMS.

None of the respondents used oral contraceptives. The average age at menopause was 49 years (range of 45 to 54 years). Younger women were more likely to have had menopause surgically induced (bilateral oophorectomy) and to be on hormone replacement therapy.

Five out of 24 respondents (21%) had undergone hysterectomy and 4 had undergone bilateral oophorectomy. 35% of respondents had had their estrogen level measured and found it within the normal range in 75% of the cases.

The 19 respondents who had true LAF (no heart disease and no maze or ablation procedure) experienced an average (mean) of 18 episodes (median 6 episodes) over the 6-month survey period with a range of 1 to 90 episodes. The mean duration of the episodes was 12 hours with a median of 5 hours and a range of 0.5 to 84 hours. Total average (mean) time spent in afib was 150 hours with a median of 48 hours and a range of 1 to 900 hours. There was a strong correlation between the frequency of episodes and the total time spent in fibrillation ($p=0.0002$).

B. Female Sex Hormones and LAF

The levels of female sex hormones (estrogen and progesterone) are highly dependent on menopausal stage. Thus it is necessary to investigate a possible connection between these hormones and LAF separately for each of the three stages – premenopausal, menopausal (perimenopausal), and postmenopausal.

Premenopausal stage
Sex hormone levels vary considerably between the luteal phase (early part of a new menstrual cycle) and the follicular phase (late part of the cycle). Researchers at the University of Ankara have found clear evidence of increased sympathetic (adrenergic) activity in the luteal phase[2]. Other researchers have found that norepinephrine levels increase markedly from the early to the late stage of the menstrual cycle. These same researchers also found that estrogen levels are highest between day 9-14 and days 21-25 of the cycle while progesterone levels rise continuously from the beginning until the end of the cycle[3]. Researchers at the University of The Andes in Venezuela have concluded that the autonomic nervous system balance is significantly changed during the luteal phase[4]. Researchers at the Mayo Clinic report that both estrogen and progesterone are elevated in the luteal phase and that the sympathetic baroreflex sensitivity (the ability to adjust heart rate in response to blood pressure changes) is greater as well. They speculate that estrogen may increase sympathetic activity while progesterone may decrease it[5].

What all this boils down to is that hormonal fluctuations markedly influence the autonomic nervous system balance and norepinephrine levels and that these effects are greater in the early part of the cycle than in the later parts. Could these changes be enough to initiate an episode?

Two out of five premenopausal women felt there could be an association between their menstrual status and LAF, two were not sure, and one did not think there was a connection. Two respondents thought their LAF episodes were more frequent at the beginning of the cycle while two felt they were more frequent at the middle or end. Clearly the survey data is far too incomplete to conclude whether or not there is a correlation between hormonal status and the timing of episodes. However, the evidence for a connection between autonomic nervous system balance and hormonal status is strong so it is certainly possible that there could be a connection, but obviously a much larger survey would be required to establish this.

None of the premenopausal women were using oral contraceptives so it is not possible to conclude anything about their possible effects on LAF. Theoretically an estrogen cream might help vagal afibbers while a progesterone cream might be beneficial for adrenergic ones (premenopausal). This, I would like to strongly emphasize, is pure speculation on my part and obviously requires a clinical trial to prove or disprove.

Menopausal (perimenopausal) stage
Four respondents were going through menopause (perimenopausal). Three of them felt there was no association between their hormonal status and the timing of their episodes. I have not come across any information that specifically addresses the question of changes in autonomic system balance during menopause so it is not possible to draw any conclusion for this group of respondents.

Postmenopausal stage
There is ample evidence that hormone replacement therapy can have a profound effect on the autonomic nervous system in postmenopausal women. Several independent research teams have concluded that replacement therapy with estrogen + progestin (HRT) increases vagal (parasympathetic) tone and decreases adrenergic (sympathetic) tone[6,7]. This effect is especially pronounced in women exposed to mental stress[8]. A German team found that heart rate increased during HRT despite the decrease in adrenergic activity[6].

Other researchers have found that transdermal, but not oral hormone replacement therapy with estrogen alone (ERT) reduces sympathetic (adrenergic) activity when awake, but not when in REM sleep[9-11].

It is also clear that surgically induced menopause (bilateral oophorectomy) induces adrenergic hyperactivity due to the abrupt loss of estrogen production from the ovaries[12]. This hyperactivity can be counteracted with ERT[12].

Our survey included 16 postmenopausal women. Five out of the 16 (31%) felt that there was a significant association between their first LAF episode and a major hormonal upset. Three cited the start of HRT with Premarin (unopposed estrogen) as the likely precipitating cause while one thought entering menopause was to blame. Two of the women who associated their first episode with the start of ERT had the vagal variety of LAF while one was mixed. It is thus possible that there could be a connection since ERT is know to tilt the autonomic system balance towards the vagal side. Four (25%) of the 16 postmenopausal respondents were taking

estrogen (either oral or transdermal), three (19%) were on HRT (estrogen + progestin) either oral or transdermal (cream) while one (6%) was using a progesterone cream. Eight (50%) of respondents did not use ERT or HRT.

Women using any kind of hormone replacement therapy had significantly longer lasting episodes (p=0.02) and tended to have more frequent episodes as well (not statistically significant). There was, unfortunately, not enough data to determine the individual effects of the different forms of hormone replacement therapy (ERT or HRT) in adrenergic, mixed and vagal afibbers.

It is clear that both ERT and HRT tend to shift the balance of the autonomic nervous system towards vagal predominance. This would clearly not be good news for vagal and perhaps even mixed afibbers. HRT does not reduce the risk of stroke and heart attack, doubles the risk of blood clots, and substantially increases the risk of gallstones, hip fractures, breast cancer and lung cancer[13-16]. In view of this, there would seem to be little reason for postmenopausal afibbers to begin or to continue hormone replacement therapy.

Conclusion

There is no question that the sex hormones estrogen and progesterone have a significant effect on autonomic nervous system balance both in the premenopausal and postmenopausal stages. Since autonomic balance is a key factor in lone atrial fibrillation it may well be that hormonal status could influence the frequency or duration of afib episodes. Unfortunately, the number of respondents to our survey was not sufficient to prove or disprove such an association. Nevertheless, there would seem to be nothing to gain and much to lose for postmenopausal women to begin or continue HRT in an attempt to reduce the severity of their afib episodes.

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My Way Back to NSR

by Erling Waller

I am a 74-year-old male enjoying excellent health, free of the afib that used to be my frequent and unwelcome companion. My afib career began about 10 years ago. I was doing some light carpentry at home mid-day, feeling fine, when all of a sudden my heart started racing. Not irregular at all, but a steady rhythm at 155 bpm. When my chest began to hurt I drove to my doctor's office and, in the ER, learned that I had something called atrial flutter. The episode lasted about 3 hours.

In hindsight I now realize that I had experienced PACs (premature atrial complexes) perhaps 3 or 4 years earlier and that starting in 1991 or 1992 I probably had experienced brief runs of atrial fibrillation now and then. The PACs had been dismissed as being of no concern by my cardiologist and I did not pay much attention to them until the autumn of 1995 when I had my first full-blown afib episode at the age of 67 years.

The second episode came about 3 months later, but from this time on they became more frequent, time between episodes varying from a month to a day, with more than 20 per year. The shortest ever was just a few minutes long (not including many very brief runs of a second or two), the longest about 44 hours. I always converted spontaneously. Episodes that would last more than a few minutes were almost always highly symptomatic, making me incapable of anything except lying down with feet up, or sometimes heading off to the ER for intravenous calcium channel blocker, ECG, and companionship. Once I went prepared with an article by an ER doctor who had used intravenous magnesium sulphate to successfully convert a patient. My ER doctor was interested and ordered it, but with no result. We agreed that it probably meant that I was not magnesium deficient to begin with. Later on I converted on my own, as always.

My episodes would almost always begin during the day, only occasionally at night while asleep. I did not discover any triggers as such although I suspect that at least some of my episodes involved a light reactive hypoglycemia following a meal or subsequent to enjoying a beer or two after work. Needless to say, I no longer drink beer. I believe it could precipitate a hypoglycemic reaction or that some substance in the "chemical brew" called beer might have been part of the complicated afib equation, but a direct cause-and-effect relationship was never clear to me.

I, of course, made the usual rounds of GPs and cardiologists and did learn that my heart was sound. At one time a "silent ischemia" was suspected because of a slight S-T segment depression during a stress test; however, this was disproved with a follow-up thallium stress test. Maximal heart rate exercise (treadmill testing) never provoked arrhythmia or angina. I tried the beta-blocker atenolol (Tenormin) for several weeks. It made me feel very tired and I suspect precipitated a very lengthy afib episode so I stopped using it. Eventually I was prescribed amiodarone (Cordarone), but decided against taking it.

At this point I finally realized that if I were to overcome my afib I would have to find the solution myself. So I began by asking 3 questions:

1. What changed within me to cause afib to arrive in my life at age 67?
2. What would change within me to initiate an episode?
3. What would change within me to terminate an episode?

Having learned that I had no underlying heart disease, I could assume that the chromosomal and mitrochondrial DNA codes for the substances and energies required for healing and maintenance of my cells were still intact. Obviously they used to be intact because I was healthy and without full-blown afib up until age 67. If the codes were indeed intact then the answers to my questions could be:

1. Chemical excesses or deficiencies, or both, had compromised the ability of the DNA codes to be actualized thereby making the cardiac tissues vulnerable to fibrillation.
2. Shifts in the amounts or types of internal chemicals would somehow initiate an afib episode.
3. Further chemical shifts would somehow terminate an episode.

After much study about cardiac cells, and the significance of cell membrane integrity and cellular energy in maintaining NSR, I finally focused in on the nutritional requirements of cells and the all important issues of omega-6 to omega-3 ratio, EPA and DHA fish oils, coenzyme Q10, l-carnitine, and magnesium.

Omega-3 (w3) and omega-6 (w6) are families of the essential polyunsaturated fats. They are essential in the diet because they are required and the body can't produce them. Probably everyone consumes too much w6 fats relative to the w3s since they are abundant in our food supply. The task for me was to know the sources and reduce their intake. The principal sources of w6s and w3s in our foods are the vegetable oils such as soybean, safflower, sunflower, canola, etc. If the food label lists polyunsaturated fats it's w6 and w3. The ratio of w6 to w3 in these food oils is too high to be conducive to health, and the methods used in extracting the oils make them unsuitable for consumption. "Virgin" applied to olive oil implies that gentle, low heat, non-destructive methods were used in extracting the oil, I've never seen that word used for other oils in our food. By reducing food oils and other common sources of polyunsaturates, and by adding supplemental w3s in the form of EPA/DHA fish oils I was able to improve my ratio. I have never aimed for a certain daily amount of w6, and would have a hard time doing so – I just watch my step. I figure that if I just stay low on most foods with oils I will still be getting plenty of w6, a required nutrient. But by doing so my intake of w3 is reduced. The most important w3s, EPA/DHA, are not in these oils anyway. They are either made in the body from other w3s in food (which for many is problematic), or they need to be supplemented. I usually take daily 4 capsules of fish oil providing 720 mg EPA and 500 mg DHA, but some days only 2 or 3 capsules. For a long time I was taking more than I am now. I absolutely stay away from hydrogenated oils which seem to be everywhere in processed foods. Hydrogenation produces "trans" fats with a molecular shape that screws up cell membranes. The book "Fats that Heal, Fats that Kill" by Udo Erasmus is powerful knowledge. Some days I only take 2 capsules, some days none, but I'm out of the woods now (in a maintenance mode) and am enjoying being less fussy about these things.

I also learned that I could likely improve my situation by ensuring that I had an adequate intake of two nutrients vital to proper cell functioning, l-carnitine and coenzyme Q10. For a long time I took about 2000 mg per day of l-carnitine, but now that I am just maintaining I'm down to about 1000. My doctor kindly wrote a prescription for Carnitor, the only prescription form in the US. Carnitine over-the-counter is a bit expensive, so with insurance I have only a moderate expense. Acetyl-l-carnitine is, by many accounts, superior for reasons having to do with entry into the cells, but the body converts l-carnitine to the acetyl form anyway. So I was never certain that the extra cost was justified. If I was starting over and knew what I know now I am sure I would go all out and buy the acetyl form.

I now take 100 mg per day of coenzyme Q10 in the form of an oil-based capsule, but for a long time it was 180 to 200 mg. There has been some discussion about taking CoQ10 while being on warfarin. For a period of time some years ago I was on warfarin and about 180 mg of CoQ10 daily, and there was never a problem with my INR nor was a question ever raised about the combination by my cardiologist or my other doctor. There is nothing in any of the voluminous scientific literature today that proscribes the combination. It's true that, because of its molecular similarity to vitamin K, CoQ10 does have a similar effect on clotting, but I was never told to limit my intake of spinach or other high vitamin K foods. A young person will perhaps not benefit from CoQ10 supplementation because the body normally produces sufficient quantities. Later in life (during the 30s) CoQ10 production falls off markedly. I was in my 60s when afib began and anyone with afib is probably not "normal" as regards to cellular energy and antioxidant protection, another important CoQ10 function.

I endeavoured to learn everything that I could about the minerals magnesium and potassium, because I learned that they are intimately linked to normal heart rhythm, and deficiencies can lead to arrhythmias. I read that magnesium is required in over 300 enzymatic reactions, including ones involved in the production of cellular energy. I also learned that magnesium is called "nature's physiologic calcium blocker". Since it is known that some 80% of people in our culture are magnesium deficient, I take 400 mg or so per day as a supplement. For some time I was using magnesium aspartate, but avoid it now because it seemed that it actually increased the frequency of a-fib events, probably due to the known "excitatory" effect of aspartic acid (aspartate). There are many other excellent supplement forms available. I don't supplement with potassium since there is a huge amount in common foods.

My other supplements include a mixed vitamin E supplement and about 1 to 2 grams per day of vitamin C. I also try to keep my calcium/magnesium ratio at about 2:1 versus the recommended 4:1. I believe this and eliminating most dairy products have also contributed to my healing.

Am I healed? I certainly believe so. I have been in normal sinus rhythm since January 2002, after experiencing a great reduction in frequency and intensity of afib events during the fall of 2001. I am experiencing the health and vitality of 30 years ago. I take no medications, my PACs have essentially ceased, and the fear of experiencing another afib episode has completely disappeared. Looking back, I now believe that the most important steps that I took to achieve this were to begin taking fish oils (EPA and DHA), reducing my intake of omega-6 fats, completely eliminating my intake of hydrogenated oils, and supplementing with CoQ10, l-carnitine, and magnesium. Another very important contribution was to stop putting into my body suspicious chemical ingredients and additives in processed foods. Besides avoiding like the plague MSG and aspartame in all of their guises, my general rule is, if I cannot pronounce it I probably should not eat it. I believe that dietary indiscretions resulting from an ignorance of sound nutritional principles had caused a gradual decline in my health over a period of many years, finally resulting in afib, a blessing in disguise. I deplore that having been given a good body, I let it deteriorate out of ignorance and blind obedience to false cultural dietary norms. I wish everyone with the nasty afib affliction could be as fortunate as I have been in finding a way out.

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