THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

NUMBER 63 **OCTOBER 2006** **6th YEAR**

Welcome to our early October issue. Judi and I are taking a vacation, so thought we would send the issue out sooner rather than later. The centerpiece of this issue is, without a doubt, David Booth's poignant and thought-provoking description of his afib journey and the lessons it taught him. This deserves at least a two-time read! Thank you David for sharing.

Also in this issue we review the new 2006 guidelines for the management of atrial fibrillation. They are not materially different from the earlier guidelines but do allow a greater flexibility in choosing between aspirin and warfarin therapy in the case of moderate stroke risk (age over 75 years, hypertension, diabetes, or recent onset heart failure). As well, we report on the progress in finding a replacement for amiodarone, that magnesium improves bone strength, and robotic (magnetically-guided) radiofrequency ablation makes its debut.

Last, but not least, if you need to restock your supplements, please remember that by ordering through my on-line vitamin store you will be helping to defray the cost of maintaining the web site and bulletin board. You can find the store at http://www.afibbers.org/vitamins.htm - your continuing support is very much appreciated.

Wishing you good health and lots of NSR,

Hans

Highlights

Promising trials of dronedarone

FRANKFURT, GERMANY. Amiodarone (Cordarone) is probably the most effective antiarrhythmic drug on the market today and is widely used in the management of atrial fibrillation, especially in Europe. Unfortunately, the drug has many serious adverse effects and its long-term use can lead to pulmonary congestion, liver toxicity, severe thyroid problems, ventricular tachycardia, dermatitis, and visual disturbances. About 75% of patients taking amiodarone experience one or more adverse effects. Amiodarone also has a very long half-life (2-3 months) which means that any adverse effects can linger for a long time. Nevertheless, amiodarone is very effective in preventing the recurrence of atrial fibrillation, so a great deal of research has been directed toward finding a substitute which would maintain the benefits of amiodarone but avoid the adverse effects.

Early research pointed to the iodine part of the amiodarone molecule as being the culprit in most of its adverse reactions. This led to the development of dronedarone – a molecule similar in structure to amiodarone, but without the iodine moiety. Dronedarone also contains a methane sulfonyl group, which shortens the drug's half-life and decreases tissue accumulation. All in all, it is clear that dronedarone is a much safer drug than amiodarone, but is it as effective?

Preliminary studies showed that dronedarone is as effective as amiodarone in blocking Na^+, K^+, Ca^{2+} and slow L-type calcium channels and, as a result, is effective in prolonging action potential duration. Dronedarone also exhibits beta-blocking activities similar to those of amiodarone, but has no

significant effect on plasma levels of thyroid hormones (T3, T4 and reverse T3).

The DAFNE (Dronedarone Atrial Fibrillation Study After Electrical Cardioversion) clinical trial evaluated the effectiveness of dronedarone (400 mg twice a day) in maintaining sinus rhythm in 200 persistent afibbers who had undergone electrical cardioversion. The median time to recurrence of AF was 5 days in the placebo group and 60 days in the dronedarone group. There was no evidence of thyroid, ocular or pulmonary toxicity over the 6-month trial period, but about 3.9% of study participants did experience diarrhea, nausea or vomiting during the trial.

Two other clinical trials of dronedarone are now nearing completion. EURIDIS (European Trial in Atrial Fibrillation or Flutter Patients Receiving Dronedarone for the Maintenance of Sinus Rhythm) involves 612 patients at 65 centers in 12 European countries. ADONIS (American-Australian-African Trial with Dronedarone in Atrial Fibrillation/Flutter Patients for the Maintenance of Sinus Rhythm) involves 625 patients recruited from 101 centers in the USA, Canada, Australia, South Africa and Argentina. Both trials are placebo-controlled, multicenter, multinational, double-blind, parallel-group trials using 400 mg of dronedarone twice daily or a placebo to prevent recurrence of AF. Publication of final results is eagerly awaited.

Wegener, FT, et al. Dronedarone: an emerging agent with rhythm- and rate-controlling effects. **Journal of Cardiovascular Electrophysiology**, *Vol. 17, Suppl. 2, September 2006, pp. S17-S20*

Magnesium improves bone strength

MEMPHIS, TENNESSEE. Many afibbers have found magnesium supplementation highly beneficial in preventing ectopic beats (PACs and PVCs) and even afib episodes. Now there is evidence that an adequate daily magnesium intake also materially improves the density of skeletal bone and helps prevent osteoporosis and hip fractures.

Researchers at the University of Tennessee measured bone mineral density (BMD) in a group of older men and women (black and white between the ages of 70-79 years). The 2038 participants were enrolled in the Health, Aging and Body Composition Study initiated in 1997. The researchers also determined the participants' daily intake of magnesium, calcium, potassium, vitamin D, and vitamin C. Less than 26% of the study group met the Recommended Daily Allowance (RDA) for magnesium (320 mg/day for women and 420 mg/day for men over the age of 70 years). Twenty-five per cent took a magnesium supplement providing an average of 83 mg/day of elemental magnesium. Black men and women had a significantly higher BMD than did white persons and did not benefit from higher magnesium intake.

White women with the highest magnesium intake had a significantly higher BMD than women with lower intakes with an increase in daily intake from 220 mg/day to 320 mg/day corresponding to an increase of 0.020 g/cm^2 in whole body BMD (after adjusting for other relevant variables). For white men, an increase from 320 mg/day to 420 mg/day corresponded to an increase of 0.010 g/cm^2 in whole body BMD. These increases are roughly equivalent to those that would result from increasing daily calcium intake by about 400 mg. The researchers speculate that the beneficial effects of an increased magnesium intake on bone density may be due to one or more of the following factors:

- Improved synthesis of vitamin D with subsequent suppression of parathyroid hormone function.
- Increased alkalinity of a diet high in magnesium and lower net acid production.
- Substitution of calcium with magnesium in the formulation of bone hydroxyapatite, resulting in greater structural strength. NOTE: Strontium may have a similar effect.

The researchers conclude that a higher magnesium intake through dietary change or supplementation may provide an additional strategy for preventing osteoporosis.

Ryder, KM, et al. Magnesium intake from food and supplements is associated with bone mineral density in healthy older white subjects. **Journal of the American Geriatrics Society**, *Vol. 53, November 2005, pp. 1875-80*

Editor's comment: The finding that 100 mg/day of elemental magnesium is as beneficial in regard to bone strength as is 400 mg/day of calcium is welcome news to those afibbers, notably vagal, who

have found that calcium supplementation tends to increase the frequency of their episodes. I am not aware of any research on just how far one can go in replacing calcium with magnesium in the hydroxyapatite bone structure. However, it is quite possible that supplementing with 400-600 mg/day of highly absorbable magnesium (glycinate or citrate) may eliminate or vastly reduce the need for calcium supplementation in a normal diet.

Earlier cardioversion possible

CLEVELAND, OHIO. It is generally accepted practice that electrical cardioversion must be performed either within the first 48 hours after the onset of an AF episode, or after 3 weeks of anticoagulation with warfarin (Coumadin). Electrical cardioversion is usually followed with a 4-week course of anticoagulation to further reduce the risk of a stroke caused by blood clots (thrombi) released from the left atrium (particularly the left atrial appendage) after the return to regular sinus rhythm. A team of American, Australian and German researchers now report that electrical cardioversion can be performed safely without the 3-week pretreatment with warfarin if a transesophageal echocardiogram (TEE) taken immediately prior to cardioversion shows no signs of thrombi in the left atrium.

The clinical trial involved 525 patients assigned to TEE prior to cardioversion and 509 patients assigned to the conventional 3-week course of warfarin. The average age of the patients was 65 years and most of them had one or more comorbid conditions such as hypertension, or congestive heart failure. All patients had been in AF for at least 48 hours prior to enrolment and 82% were taking one or more antiarrhythmic drugs. The patients in the TEE group underwent TEE, anticoagulation with unfractionated heparin, and cardioversion within 3 days of enrolment, while patients in the conventional group underwent electrical cardioversion between 20 and 40 days after enrolment.

The immediate conversion rate (to normal sinus rhythm) was 82% in the TEE group and 78.4% in the conventional group. The TEE indicated the presence of thrombi in 62 patients and cardioversion was postponed for this group. After 6 months 62.5% of patients in the TEE group who had undergone cardioversion were still in sinus rhythm as compared to 53.9% in the conventional group. The incidence of ischemic (embolic) stroke and TIA (transient ischemic attack) was 1.9% in the TEE-guided group and 0.8% in the conventional group; however, this difference was not statistically significant. The rate of serious bleeding events was significantly higher in the conventional group (7.5%) than in the TEE-guided group (4.4%). Death from cardiovascular causes over the 6-month follow-up period was similar in the two groups at 2% and most were classified as sudden cardiac death not involving stroke or bleeding.

The researchers conclude that TEE-guided electrical cardioversion is a clinically effective alternative to the conventional anticoagulation strategy followed by cardioversion. They point out that the TEE-guided approach may be particularly useful in highly symptomatic, new onset AF and for patients at high risk for bleeding and stroke.

Klein, AL, et al. Efficacy of transesophageal echocardiography-guided cardioversion of patients with atrial fibrillation at 6 months: a randomized controlled trial. **American Heart Journal**, *Vol. 151, February 2006, pp. 380-89*

Robotic ablation makes its debut

MILAN, ITALY. Dr. Carlo Pappone and his team at the San Raffaele Scientific Institute has performed the first robot-assisted radiofrequency ablations in a group of 40 patients with paroxysmal (62.5%) or permanent (37.5%) AF. The circumferential pulmonary vein ablations (Pappone method) were carried out using a combination of the CARTO mapping system and a 4 mm *NaviStar* flexible, magnetically-guided catheter. The catheter contains a small permanent magnet in its tip and is guided by remote control through computerized input to two focused-field permanent magnets located on each side of the patient's body. The operator (electrophysiologist) sits in front of a computer screen in a separate room and performs the ablation from there, thus avoiding radiation exposure from fluoroscopy.

The remote-controlled ablation was successful in 38 of 40 patients with success being defined as voltage abatement greater than 90% of bipolar electrogram amplitude at the completion of the procedure. The median procedure time for the first 12 patients was 3 hours and 12 minutes, but this was reduced to 2 hours and 28 minutes for the following 28 patients indicating a rather short learning curve. The target temperature during the procedure was 65^{o} C with a power limit of 50 W. The mean overall procedure time for the robot-assisted procedure was significantly longer than normally experienced with manual ablation (mean procedure time of 1 hour and 50 minutes), but the average ablation time for the last 28 patients was somewhat shorter than for the manual procedure (49 minutes vs 58 minutes). No adverse effects occurred during the procedures.

The researchers conclude that magnetically-guided circumferential pulmonary vein ablation is safe and feasible with a short learning curve. Although all procedures during this first trial were performed by a highly experienced operator, the team predicts that it will eventually be possible for less experienced operators to perform the procedure successfully.

Pappone, C, et al. Robotic magnetic navigation for atrial fibrillation ablation. **Journal of the American College of Cardiology**, *Vol. 47, April 4, 2006, 1390-400*

Editor's comment: The advent of robot-assisted ablation is indeed an exciting event and its wider acceptance will hopefully improve the success rates of less skilled EPs. It is, however, unfortunate that the Pappone report made no mention of how many of the 40 patients were actually afib-free after the procedure and how many remained so after 6 months.

Azimilide not effective for AF prevention

VANCOUVER, CANADA. A group of researchers at the University of British Columbia (St. Paul's Hospital) has evaluated the efficacy of the experimental antiarrhythmic azimilide in maintaining sinus rhythm in a group of patients with paroxysmal AF and heart disease (congestive heart failure, coronary artery disease, or structural heart disease). The patients were randomized to receive a placebo (215 patients) or 125 mg of azimilide (216 patients) twice a day for 26 weeks after a recorded afib episode. The median time to afib recurrence was 10 days in patients with congestive heart failure or coronary heart disease irrespective of whether they were taking azimilide or the placebo. Overall, the median time to afib recurrence was 9 days in the azimilide group and 8 days in the placebo group – not a significant difference. The researchers conclude that azimilide does not have a significant benefit, compared with placebo, in delaying afib recurrence in patients who were in sinus rhythm when starting the drug.

Kerr, CR, et al. Efficacy of azimilide for the maintenance of sinus rhythm in patients with paroxysmal atrial fibrillation in the presence and absence of structural heart disease. **American Journal of Cardiology**, *Vol. 98, July 15, 2006, pp. 215-18*

SPECIAL REPORT

Review of 2006 Guidelines for Management of Patients with Atrial Fibrillation

A group of experts from the American College of Cardiology, American Heart Association, and European Society of Cardiology has published a new set of guidelines for the management of patients with atrial fibrillation. These guidelines supersede those issued in 2001.

Atrial fibrillation (AF) is the most common arrhythmia encountered in clinical practice. It is estimated that about 2.3 million people in North America and about 4.5 million in the European Union have AF. Most of these have associated structural heart disease, but 10-30% have what is known as "lone" AF. Lone AF was originally defined as being simply AF with no underlying structural heart disease; however, the 2006 guidelines narrow this definition. The term "lone" AF now applies to individuals younger than 60 years of age without clinical or echocardiographic evidence of cardiopulmonary disease, including hypertension. The term "nonvalvular" AF refers to cases without mitral valve disease, prosthetic heart valve, or valve repair. The terms paroxysmal

(terminating spontaneously within 7 days), persistent (episodes lasting longer than 7 days, but amenable to cardioversion), and permanent (lasting longer than one year and either not amenable to cardioversion or cardioversion foregone) have not been materially changed.

The guidelines cover the acute treatment of AF, rhythm and rate control, prevention of recurrence, and stroke prevention (antithrombotic therapy). There is also brief mention of maze surgery and catheter ablation, which are considered primarily as approaches to be tried if drug therapy fails.

Clinicians (cardiologists and electrophysiologists) should distinguish clearly between a first-detected episode, which generally requires no treatment and recurring episodes which may. Among the highlights of the guidelines are:

Acute treatment of an AF episode
Most episodes of AF terminate spontaneously, but if they do not attempts at electrical or pharmacological (drug-based) cardioversion should be made in a hospital setting. Pharmacological cardioversion is more effective the quicker it is initiated and loses its effectiveness if more than 7 days have elapsed since the onset of the episode. Flecainide (Tambocor) and propafenone (Rythmol) are most often used for pharmaceutical conversion, although there may be cases in which dofetilide (Tikosyn), ibutilide (Corvert), or amiodarone (Cordarone) are deemed desirable. The drugs can be administered intravenously or orally. Digoxin and sotalol may be harmful when used for pharmacological cardioversion of AF and are not recommended. It is now also accepted practice for lone afibbers to use the "pill-in-the-pocket" or "on-demand" approach to terminate paroxysmal episodes in a home setting. It is recommended that a beta-blocker or a calcium channel blocker (verapamil, diltiazem) be taken prior to taking the Class IC antiarrhythmic (flecainide or propafenone) in order to prevent rapid AV conduction in case atrial flutter is also present.

Electrical cardioversion involves the delivery of an electrical shock(s) synchronized with the intrinsic activity of the heart. It is performed in a sedated and, preferably, fasting state. Electrical cardioversion is usually performed within 48 hours of the onset of an episode, or after 3 weeks of anticoagulation with warfarin. A further 4 weeks of anticoagulation post procedure is necessary in order to prevent an embolic stroke precipitated by the heart's return to normal sinus rhythm. It is also acceptable to perform electrical cardioversion after ensuring, via transesophageal echocardiography (TEE), that there are no clots or clot precursors (SEC) in the left atrium prior to the procedure. It is very important to ensure that serum potassium level is within the normal range before attempting cardioversion. Low potassium levels are associated with a significantly poorer success rate for the cardioversion.

Maintenance of sinus rhythm
If episodes are infrequent and well-tolerated drug therapy is usually not indicated. However, if episodes are more frequent and highly symptomatic then antiarrhythmic therapy should be considered. The first choice drugs for vagal afibbers are flecainide and disopyramide (Norpace, Rythmodan). Propafenone is not recommend because its (weak) intrinsic beta-blocking activity may aggravate vagally-mediated paroxysmal AF. ***Editor's comment***: Some vagal afibbers have reported good results with propafenone. Whether or not it is effective or detrimental may depend on the speed with which it is metabolized in each particular case.

The first line treatment for adrenergic afibbers is usually a beta-blocker, but flecainide, propafenone or sotalol may be tried if the beta-blocker does not prove up to the task. Combinations of Class IC antiarrhythmics and beta-blockers or calcium channel blockers may also be efficacious in individual.

Rate rather than rhythm control may be a viable alternative for older patients in permanent and fairly asymptomatic AF. Rate control can be achieved with beta-blockers or calcium channel blockers (verapamil, diltiazem). It is desirable to achieve ventricular (pulse) rates of between 60 and 80 beats per minute at rest and between 90 and 115 beats per minute during moderate exercise. There is not evidence that afibbers assigned to rate control rather then rhythm control have a greater incidence of ischemic stroke.

Stroke prevention
The guidelines for antithrombotic therapy (stroke prevention) have changed considerably since the issuance of the 2001 guidelines. While the 2001 guidelines allowed for lone afibbers with no risk factors for stroke to forego

any stroke prophylaxis or take a daily aspirin, the 2006 guidelines require that all patients diagnosed with AF take either aspirin or warfarin even if they have no risk factors. Actually, the guidelines are not clear on this point. Another section (page 705) states, "*Antithrombotic therapy to prevent thromboembolism is recommended for all patients with AF, except those with lone AF or contraindications*." And on page 706 the following two statements appear:

- "*In patients with AF younger than 60 y without heart disease or risk factors for thromboembolism (lone AF), the risk of thromboembolism is low without treatment and the effectiveness of aspirin for primary prevention of stroke relative to the risk of bleeding has not been established*."

- "*Long-term anticoagulation with a vitamin K antagonist is not recommended for primary prevention of stroke in patients below the age of 60 y without heart disease (lone AF) or any risk factors for thromboembolism*."

So, it would seem that the option of no antithrombotic therapy is still open for lone afibbers.

Patients with only one moderate-risk factor (age over 75 years, hypertension, diabetes, or recent onset heart failure) may take aspirin or warfarin (target INR 2.5), while patients with two or more moderate-risk factors, or one high-risk factor (previous stroke, TIA or embolism, mitral stenosis, prosthetic heart valve) are advised to take warfarin (target INR 2.5). The same guidelines apply to patients with atrial flutter. The selection of antithrombotic therapy should not be influenced by whether the patient has paroxysmal, persistent, or permanent AF, but should be based strictly on the absence or presence of stroke risk factors.
ACC/AHA/ESC 2006 Guidelines for the Management of Patients with Atrial Fibrillation – Executive Summary. **Circulation**, *Vol. 114, August 15, 2006, pp. 700-52*
http://circ.ahajournals.org/cgi/reprint/CIRCULATIONAHA.106.177031v1

Editor's comment: One of the more controversial changes in the 2006 guidelines is the increased emphasis on the use of aspirin for stroke prevention. There is actually no evidence that aspirin is effective for the prevention of a first stroke (primary prevention) and the FDA has repeatedly refused to label the drug for this application. Aspirin is by no means innocuous and causes thousands of hemorrhagic strokes and serious internal bleeds every year. Thus, the "daily aspirin" ritual should not be embarked upon lightly, but only after careful study of the pros and cons in each particular case. An excellent start for this is the report "*Aspirin for the Primary Prevention of Cardiovascular Events: A Summary of the Evidence for the U.S. Preventive Services Task Force*" by Michael Hayden, MD et al. published in the **Annals of Internal Medicine**, Vol. 136, No. 2, January 15, 2002, pp. 161-72.
http://www.annals.org/cgi/reprint/136/2/161.pdf

Living with Vagal Lone AF

by David Booth

History

My first episode of AF occurred in the middle of winter. I was then 50, active, in good health and working under some stress. After 3 more episodes and a visit to emergency, I became a patient of the heart clinic. Tests revealed no obvious cause and the cardiologist prescribed Sotalol. It was considered to be a prescription for life, a prospect that grated against values that dated back to childhood when my parents mistrusted use of any unnecessary medicines. Within a couple of months, I had abandoned the medication in the hope that the whole experience was an isolated one.

Athletic sports were a major part of my life as teenager. Since then, non-competitive physical activity has remained imbedded in daily life. The body worked and performed well and I believed that I was looking after its well-being. There was, however, a tendency to push myself in order to achieve various goals, both at work and of a personal nature. Many of these had as an underlying aim to prove my own existence.

Four years after the first episode, again at a time of stress in the middle of winter, four more episodes hit me. With the heart thumping wildly, I got to emergency where another cardiologist gave me the same prescription as before. I left with the impression that indeed the problem was serious and followed instructions by taking the medication. Spring did not bring me the usual revival of energy and enthusiasm. My family doctor was concerned about high blood pressure. In June, more episodes of AF occurred with two more visits to emergency. The dose of Sotalol was increased. I spent the summer devoid of energy. The cardiologist explained that according to her experience AF was usually just the tip of the iceberg and that the symptoms increased with age. It was at that moment that the need to understand became urgent. Soon afterwards, I found the www.afibbers.org site, which gave me the tools to take responsibility for my own condition.

During the autumn, several more episodes occurred and the level of anxiety increased to a level that made work as a university teacher virtually impossible. My family doctor took me off work but learning that the insurance did not recognise AF as a valid reason for sick leave only added to the anxiety. The episodes became more frequent and I came to believe that this half existence was to be my lot for the rest of my life. Medication was again increased and the feeling of vulnerability became ever more present until I started to question the medication itself. In an attempt to find some solution, I stopped the medication. After a couple of weeks, some normality returned, and I started to regain a little energy and plan a return to work. The cardiologist accepted that the medication might be unsuitable and suggested a milder Beta-blocker. She also brought up the possibility of ablation but I was far from ready to accept such an intervention. I did try the new medication for a short while but regarded it with suspicion as the feeling of vulnerability returned.

The next series of episodes happened again in the middle of winter. By now, I was accustomed to the experience and the intensity of AF was much reduced. A further series occurred later in the spring, once again during a period of stress.

During this past year and a half, I have become more familiar with my own body. Some observations seem particularly important and it is these that I wish to share. Many of these observations are in agreement with those found in Hans Larsen's books.

Use of a journal

Although it may seem obsessive to the medical profession, writing a journal to keep track of episodes of AF has been beneficial. Not only does it help in identifying patterns but it also allows one to stand back from the experience.

Classification of AF

Initial episodes of fibrillation were generally intense and lasted several hours. Later, the intensity decreased and the duration became more variable, ranging from a few minutes to a whole day. Virtually all of my episodes of AF began just after a meal, getting into bed, while in bed or on getting up. The very occasional episode that did not fall into this pattern started soon after a séance of meditation. Although the classification of vagal AF is not generally recognised, the pattern in my case is very clear and undoubtedly important when deciding how to deal with the problem.

Symptoms

Apart from the discomfort of a crazy heart, other symptoms include a pressure and irritation in stomach, considerable belching, exhaustion, a salty taste in the mouth, and a need to urinate frequently. The most consistent symptom, however, is a tension in the region of the diaphragm, just under the solar plexus.

Triggers

Episodes of AF always occurred during periods of stress, often at work. After an initial episode, there were almost always a few more during the following days. An initial event made me more vulnerable, but the background of stress always seemed to be present during these periods. Foods that irritated the gut or the stomach, spicy or salty dishes for example, appeared to initiate episodes. Watching the television news just

before bed also increased my vulnerability, as did the obligation to be in a noisy or crowded place. In bed, I was more likely to have an episode while lying on my side, particularly the left side. When AF started in the middle of night, I had usually just woken up on my side with the impression that I had stopped breathing.

Strategies used to end an episode

Visits to emergency were, at first, the only way I knew of dealing with an prolonged episode, and indeed the official medical advise was to do just that. On the fourth visit, however, a doctor questioned the nurse as to why I had come in with just fibrillation. It was then that I realised that, despite the gloomy predictions of the cardiologist, this was something that could be dealt with. I have now come to use several ways of attempting to end an episode. The first "technique" is to stand with knees slightly bent and to shake the hands vigorously, if necessary for some minutes. This has worked for me many times. If not, I try relaxing in a warm bath: AF often calms and stops by itself. While in bed, the intensity of AF seems to be reduced by sitting up. If already in AF, I try sleeping in a reclining long chair rather than in bed. Free breathing, however, appears to be an important aspect of any attempt to stop AF. By free breathing I mean natural deep unforced breathing and it is particularly helpful to turn one's attention to a part of the body other than the abdomen area, the nose for example.

Strategies used to reduce risk

Probably the most important is an attempt to reduce stress, not by forcing anything, but by facing and dealing with it as soon as it arises. This, of course, is easier said than achieved, and sometimes requires considerable attention and honesty with oneself. For me, however, dealing with sources of stress has made me question many aspects of life, including my professional priorities and objectives. In general, however, it means being attentive to one's state of mind and responding to its needs. In my case, fighting against emotions may be one major source of stress. It may help to give oneself space to acknowledge them and allow them to be.

The second change I made to my daily life was to give myself whenever possible ample time to digest after meals. I now try to sit and read simply for relaxation after eating. This, apart from avoiding irritating foods and eating slowly, seems to be the most important way of avoiding episodes after meals.

My wife urges me to rediscover the pleasures of being. This may be rich advice indeed, for I wonder now if AF may be in some way a manifestation of a stressed-out body. I do notice that, in spite of fatigue, my body seems more at peace after an episode of AF than before. Maybe the heart needs to dissipate pent up tension and chaotic oscillation is its only way. If so, the pleasures of being may help us find a more balanced frame of mind.

Other strategies include avoiding prolonged work at the computer, a reduction in daily work objectives, an acceptance of need to relax at frequent intervals and a recognition that the body is no longer as young and able as it was.

Diet

My diet has become simpler. Although vegetarian, apart from an occasional meal of seafood, for the past two decades, I feel the increasing need for the most basic of foods prepared in simple ways. Added salt and all processed additives have been eliminated even to the extent that the food may appear fad to another, but this is what my body seems to want. Refined sugar too has been eliminated, and just this one change seems to have decreased considerably my everyday blood pressure. Fresh fruit now tastes sweet while a sugared muffin, for example, is now almost inedible. I limit my intake of bread, which seems to irritate the stomach. The breakfast that seems to suit me best is one based on cooked quinoa with some fruit, a soy based yogurt and flax oil. Lunch is simple, often a thick soup with some source of protein. Supper is still the main meal, but more modest and less rushed. I drink little but water and soymilk, which helps calm the stomach. A very occasional beer is sometimes welcome, but even this can sometimes instil a sense of vulnerability. I now distrust restaurant meals, particularly those with sauces. Although I prefer to eat at home, when eating out I choose the simpler dishes.

I take no supplements other than a little fish oil. For many months during the winter, however, I had the almost insatiable urge to eat cooked dates. Whether or not this has anything to do with a bodily need, I do not know, but the urge diminished as spring arrived.

Sleep

I accept now that the body needs seven or eight hours of sleep each day. I no longer force the body to get up in an attempt to get more out of the day. It may be simply an acceptance of corporal limits. The fact that vulnerability to AF increases when the body is fatigued is reason enough to give oneself ample sleep. I suspect that many of my episodes occurred during periods of sleep deficiency.

T'ai Chi

This slow precise form of exercise is of great benefit; at least it seems so in my case. Not only have I recorded a drop of 12 points in systolic blood pressure during a single half-hour of T'ai Chi, but I have also been able to terminate episodes of AF while exercising. In general, T'ai Chi does help to keep the body supple and relaxed, a condition that can only help minimise AF. It seems particularly important to keep the torso relaxed.

Meditation

Meditation has been an important part of me life for many years. Although simple meditation can be done for relaxation, on deepening the practice, one enters into the conflicting tensions of life. This can bring to the surface and thus amplify confusion, dread, depression and anxiety, all of which need to be faced for what they are. This level of meditation should be undertaken under the guidance of a competent master. Otherwise, it is very easy to slip into a practice of endless rumination or of a dreary stupor. What is of interest, however, is that meditation seems to change the functioning of the nervous system. In my case, episodes of AF often occur around periods of intense meditation, and in particular when the body tightens after a deep letting-go. Meditation can thus help us learn how we function, but working with this kind of practice is difficult and guidance is essential. Despite periods of difficulty, I am convinced that such practice can be deeply beneficial, although in the end meditation is of no use if undertaken with a particular objective in mind; doing so immediately puts a stick between the spokes.

Osteopathy

Treatment from a competent and sensitive osteopath has been of the utmost help. By feeling tension in the body and working with his hands, the osteopath is able to influence the nervous systems. On one occasion, I arrived for treatment with the heart already in AF. He was able to relieve the tension and stop the fibrillation. I now go for a monthly treatment in an attempt to limit the accumulation of tension. In my case, the sympathetic nervous system is generally overactive. Gradually, the upper body seems to be learning how to relax. Of interest however is the feeling after a treatment. For a couple of days, there is a general feeling of well-being and a desire for action. At the same time, the body feels unusually vulnerable to AF, especially during the first night. It thus appears important to stay particularly attentive and avoid excessive activity while the body readjusts itself. In this respect, I suspect that osteopathy and meditation can affect the body in similar ways.

Emotions

I have come to recognise the emotions that prevail during periods of frequent episodes. For me these are in the main a simmering anger or frustration with the "system" in which I work, often with a feeling of being up against a brick wall. I realise now that my values are not those of the institution, and this constantly puts me in a situation of tension. Within myself, the tension manifests as a tug of war between what I believe is essential and what I believe is expected of me. Coming to terms with this tension is, I suspect, an important step. To be able to step back and see this dynamic as a construction rather than a reality does take the sting out of the tension. Gradually it may be possible to enter into the simmering emotion and allow it dissipate before any accumulation of tension. Some might see this as an attempt to give more space to the heart. It is of interest that on the two occasions that I broke down into tears through despair during an episode of intense AF, the heart regained its normal rhythm within minutes.

Tension in the abdomen

Tension just below the solar plexus in the region of the diaphragm is the most persistent and frequent symptom during periods of AF. Whenever the heart is in fibrillation, the diaphragm muscles feel tight and taut. Breathing lacks its usual fluidity. Well before the onset of an episode, one of the very early signs is a conscious feeling of the pulse just below the solar plexus. This is an indication of the need to stop and relax and increasingly it is possible to avoid an episode before any clear symptoms in the heart rhythm. I now suspect that muscle system of the diaphragm may play an important part in the process.

Conclusion

My understanding of what happens is based on a mechanical viewpoint. This may be naive and simplistic but the model gives us a means to link some of the observations thus far. The heart is, in mechanical terms, a complicated forced oscillator, sitting just above the diaphragm and held in place by elastic suspenders. Some mechanical oscillators can go into chaotic movement as the elasticity is changed. The fact that AF symptoms change with orientation of the body does suggest that gravity affects the dynamic. I wonder therefore if tension in the muscular system simply limits the supporting elasticity and constricts the usual heart movement thus driving it into fibrillation. This does not reject the role of the autonomic nervous systems and indeed the suggestion that variations in the balance between the sympathetic and parasympathetic branches play a role in the onset of AF may be supported by what I experience after an osteopathy treatment or a period of deep meditation. Nevertheless, whether the process is a mechanical interaction or an adjustment in the nervous system, being mindful of tension in the upper abdomen and diaphragm region does appear to minimise and weaken episodes of AF. With this limited understanding of the workings of my own body, I try therefore in whatever way possible to allow the upper body to relax. All the strategies that seem to work for me, whether to reduce the risk of an episode or to weaken and stop the fibrillation, have this effect. I strongly suspect that when the diaphragm is relaxed and supple, risk of AF is considerably reduced. Even if a relaxed diaphragm is but a sign of something underneath such as balanced nervous system, tension in the diaphragm remains a clear warning indicator.

It would be tempting to claim that nothing is wrong with the heart and that it is all in the mind. There is obviously a weakness and fibrillation is its manifestation. But perhaps, AF in some cases may be a symptom of something much more general and should therefore be welcomed as an invitation to learn. Perhaps the cardiologist was right in saying that it is just the tip of the iceberg. This iceberg, I now suspect however, is not just physiological but encompasses the whole workings of the human being. It is my hope that by taking a holistic viewpoint, I may be able to learn and eventually find a way of allowing the body and all its being function in relative harmony, even in times of difficulty. I tend to take things to heart but do not allow the heart to express itself. Maybe my fibrillation is a call from the heart. Maybe it is no wonder that the heart is considered the centre of emotion.

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