THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

NUMBER 125 **APRIL/MAY 2013** **13th YEAR**

This issue features a report by fellow afibber Michael W. who nearly died from spontaneous, internal bleeding caused by Pradax (Pradaxa, dabigatran). It is now abundantly clear that the new anticoagulants – Pradaxa (dabigatran), Xarelto (rivaroxaban), and Eliquis (apixaban) should never have been approved for marketing before an effective antidote to stop bleeding was readily available. This is just another example of the seeming inability of the FDA and Health Canada to protect the public against dangerous drugs.

I just came across a full-page ad for Celebrex (celecoxib). Celebrex is mainly used for pain control in rheumatoid arthritis. In the summer of 2001 (12 years ago) researchers at the Cleveland Clinic warned that Celebrex increases the relative risk of experiencing a heart attack by 54%. Since then, conclusive evidence has emerged proving that Pfizer, the manufacturer of Celebrex, suppressed clinical research that clearly showed that the drug greatly increases the risk of suffering a heart attack. In 2005 the FDA asked Pfizer to suspend television advertising for Celebrex for 2 years – as if this would make the drug less dangerous!! Now apparently, it is OK to advertise this dangerous drug again.

Also in this issue we report that yoga may be helpful for adrenergic afibbers, the risk of an AF-related stroke peaks during the winter, and that using a smartphone app can help detect AF episodes. A new protocol for preventing adrenergically-mediated AF episodes is reviewed.

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

LETTERS TO THE EDITOR

I am a 68 year old man and suffer from LAF. It mainly occurs during competitive running events (orienteering), which I have been doing for many years. I have to stop running and walk back. About 1 in 6 of the serious competitors in my age band (65-70 years) has this problem.

Recently, my AF has taken a bad turn, in that the irregular beats occasionally include a gap of 3 to 5 seconds without a beat. This is accompanied by a feeling of a big surge (perhaps adrenalin) making me feel woozy for a few seconds.

Have you heard of this? I feel that I may be at particular risk and may need to stop this sport, which is the passion of my life.

I do not take any drugs at all. I read with interest your references to nutritional deficiencies and will look into going onto courses of supplements.

IP

I am sorry to hear about your increasing problems with afib episodes during orienteering runs. It sounds like you may now have developed sick sinus syndrome which, if it continues, may require implantation of a pacemaker. I would highly recommend that you stop participating in competitive running events and limit your exercise to walking - your body is trying to tell you something! I know, from personal experience, that it can be very difficult to give up cherished activities and experiences, but LAF is an unforgiving master.

I would suggest you read my research report on endurance exercise.
http://afibbers.org/resources/endurancesports.pdf
You may also be interested in reading what is archived in my afib database on exercise and AF.
http://www.afibbers.com/atrial_fibrillation/exercise/index.htm

If you wish to explore the supplementation approach to LAF management, I would suggest you begin with magnesium and then add potassium after a couple of weeks if your serum potassium level is below 4.5 mEq/L. For more on this see
www.afibbers.org/resources/12stepplan.pdf
www.afibbers.org/resources/strategy.pdf

Hans

Hi Hans -- just like to say that ginger is wonderful! I can't remember how you said it worked, but let me tell you what it does for me. As I told you before, the supplementation program plus great care about what I eat at night has meant I have been afib free for a couple of years now except for one brief episode during a stomach flu attack which stopped after my magnesium/potassium drink stayed down!

But I regularly was waking up with a rapid heartbeat (my usual pulse is 60 -- and this would be 80/90) around 3-4 a.m. It took a while to come back down and probably longer because I was anxious it would turn into afib. Ever since I started drinking one very strong cup of ginger tea around midnight, that has not happened. I still wake up again around 4 but my pulse is perfectly normal and I feel calm. So thanks, once again, for your wonderful work!

KF

I have had afib attacks for several years now, and had been searching the Internet to find a way to stop an active episode, and I came across one of your articles. I, like the man in the article, have tried different drugs through cardiologists, with minimal success.

After my first afib attack in September 2009, I was diagnosed with lobular masses behind my breast bone, indicating probably a metastasized breast cancer. I began researching natural cancer cures and happened upon Dr. Joanna Budwig's cottage cheese and flaxseed oil protocol for cancer. I tried it along with many vitamins. By the time all further tests were completed, no cancer was found anywhere. I also did not have any further afib attacks but made no correlation at the time.

Approximately two years later I had stopped taking the flaxseed oil, and a funny thing happened. I had another afib attack, then another the next week, then two the following week. Each attack lasted about 12 hours. These attacks became increasingly frequent and I began searching for a solution.

Sensing a possible connection between afib and flaxseed, I did some more research and found that flaxseed helps maintain normal heart rhythm. So I went back on the flaxseed oil and added Shaklee's Omega Guard, which has fish oil, EPA, DHA and ALA. I also learned that dehydration was a trigger, so I stayed hydrated. I went nine months without an attack, until the day my grandson was born (July 23) and I was in the hospital all day with my daughter, and had failed to take flaxseed or Omega Guard, and did not get enough water either. I went back on the protocol and no attacks until two days ago when I caught a stomach bug. Threw up for 12 hours and, needless to say, was dehydrated and could not keep vitamins down.

Have I cured my afib? – No, of course not. But going 9 months or so between attacks is wonderful.

MF

Yoga may help adrenergic afibbers

KANSAS CITY, KANSAS. Yoga is a combination of physical exercises, breathing exercises and meditation that has proven benefits in improving flexibility, breathing and posture, and also in relieving stress and improving cardiovascular health. Now a group of researchers at the University of Kansas reports that yoga may also be beneficial in reducing the frequency of atrial fibrillation (AF) episodes and in improving the quality of life (QoL) in AF patients.

Their study involved 52 patients with paroxysmal AF. The average (mean) age of the study participants was 61 years (range of 50 to 72 years), 53% were female and the mean duration of AF since diagnosis was about 5 years. The average left atrial diameter was 40 mm (4.0 cm) and average left ventricular ejection fraction was 59%. Thirty-nine percent of patients had hypertension and 18% had coronary artery disease. The majority (78%) were taking antiarrhythmic medications and 63% were on beta-blockers.

The study involved two phases – a 3-month control phase followed by a 3-month yoga intervention phase. The frequency of AF episodes were compared between the two phases and clinical characteristics and QoL scores, anxiety and depression scores were determined at start of the control phase, end of control phase (beginning of yoga phase), and end of yoga phase. AF episodes were designated as **symptomatic** if the patient felt them and the event monitor recorded them, as **symptomatic non-AF** episodes if the patient felt them but the monitor did not show them, and as **asymptomatic** if the monitor recorded them but the patient did not feel them. The yoga intervention was based on the Iyengar protocol and consisted of 10 minutes of *pranayama* (breathing exercises), 10 minutes of warm-up exercises, 30 minutes of *asanas* (various yoga positions), and 10 minutes of relaxation exercises.

The average number of symptomatic AF episodes declined from 3.8 in the 3-month control phase to 2.1 during the yoga phase – a relative reduction of 45%. Similar reductions in AF frequency were observed for symptomatic non-AF episodes (down from 2.9 to 1.4 – a 52% relative reduction), and asymptomatic episodes (down from 0.12 to 0.04 or a relative reduction of 67%). Scores for depression and anxiety decreased significantly after the yoga phase with relative decreases of 7% and 24% respectively. Scores for various QoL domains improved significantly for physical functioning, general health, vitality, social functioning, and mental health. The yoga intervention also resulted in improved cardiovascular health as evidenced by lower blood pressure and heart rate.

The Kansas researchers conclude that yoga lessens arrhythmia burden, relieves anxiety and depression and, in general, improves quality of life in patients with paroxysmal AF. They do point out that their study was not designed to determine any possible difference in the benefits of yoga between adrenergic and vagal afibbers.

Lakkireddy, D, et al. Effect of yoga on arrhythmia burden, anxiety, depression, and quality of life in paroxysmal atrial fibrillation. **Journal of the American College of Cardiology**, *January 25, 2013 [Epub ahead of print]*

Editor's comment: Regular yoga practice decreases heart rate and increases parasympathetic (vagal) dominance of the autonomic nervous system. This would be highly beneficial to adrenergic and perhaps some mixed afibbers, but is likely to be detrimental to vagal afibbers.

Thyroid disorders and atrial fibrillation

COPENHAGEN, DENMARK. The relationship between hyperthyroidism (overactive thyroid gland) and atrial fibrillation (AF) is well established. There is also evidence that subclinical hyperthyroidism is associated with AF. However, when it comes to a possible association between an underactive thyroid gland (hypothyroidism) and AF the picture is less clear. An early LAF Survey found that 10 out of 22 respondents had low morning basal temperatures (an indication of low thyroid hormone output) and of the 10, four had actually been diagnosed with hypothyroidism.

A very large Danish study now sheds further light on the connection between thyroid dysfunction and AF. The study involved 586,000 adults (mean age of 50 years with 39% being male) who had their thyroid function evaluated for the first time during the period 2000 to 2010. None of the participants had a recorded history of thyroid disorder or AF at

their baseline evaluation and, in general, were quite healthy with less than 0.7% having heart disease. Most (96%) were euthyroid (normal thyroid function), but 0.3% had overt hypothyroidism, 2.0% had subclinical hypothyroidism, and 0.7% had overt hyperthyroidism with the remaining 1% having subclinical hyperthyroidism. The following definitions were used:

	TSH, mIU/L	Free thyroxine (T4), pmol/L	Total thyroxine (T4), mmol/L
Euthyroid	0.2 - 5.0	9 – 22	60 – 140
Overt hypothyroidism	> 5.0	< 9	< 60
Subclinical hypothyroidism	> 5.0	9 – 22	60 – 140
Overt hyperthyroidism	< 0.2	> 22	> 140
Subclinical hyperthyroidism	< 0.2	9 – 22	60 – 140

During a follow-up of 5.5 years (3,215,807 person-years), a total of 17,154 study participants (2.9%) were admitted to hospital with a first episode of AF. Amongst the euthyroid subjects, 2.9% developed AF. Corresponding numbers for hyperthyroidism and hypothyroidism subjects were 4.6% and 2.5%.

Overall, an almost linear relation was seen between thyroid function and the risk (incidence rate ratio) of developing AF. After adjusting for gender, age, comorbidity and socioeconomic status, the incidence rate ratio for the various types of thyroid dysfunction were as follows:

	Incidence Rate Ratio (IRR)
Euthyroid	1.0*
Overt hypothyroidism	0.67
Subclinical hypothyroidism	0.88
Subclinical hyperthyroidism	1.30
Overt hyperthyroidism	1.41

* Reference

The risk (IRR) of AF associated with overt hypothyroidism was particularly low (IRR = 0.51) amongst women below the age of 65 years, whereas for men the risk (IRR = 0.95) was not significantly different from the risk observed for euthyroid subjects.

At first glance, it is surprising that the presence of hypothyroidism would be associated with a lower risk of developing AF. However, it needs to be kept in mind that all participants diagnosed with hypothyroidism began immediate treatment with levothyroxine and at 12 months after the beginning of treatment had an IRR of 1.03; in other words, a risk of developing AF similar to that of euthyroid participants.

The authors of the study conclude that hyperthyroidism increases the risk of developing AF, whilst both subclinical and overt hypothyroidism is associated with a lower risk of AF. NOTE: It would have been more appropriate if the authors had stated that <u>treated</u> hypothyroidism is associated with a lower risk of AF. The authors offer no convincing explanation for the observed risk decrease except to suggest that the lower resting heart rate associated with hypothyroidism may protect against AF.

A recent report from Greece describes two cases of AF which were clearly related to subclinical hypothyroidism (elevated TSH levels) and which resolved with levothyroxine therapy.[1]
Selmer, C, et al. The spectrum of thyroid disease and risk of new onset atrial fibrillation. **British Medical Journal**, *November 27, 2012 [Epub ahead of print]*

Editor's comment: The finding that hyperthyroidism is associated with an increased risk of developing AF is not surprising and confirms earlier studies. However, the finding that treated overt hypothyroidism and even subclinical hypothyroidism is associated with a decreased risk of AF, at least in the early treatment stage, is puzzling. One possible explanation might be that hypothyroidism is associated with vagal (parasympathetic) dominance and that levothyroxine activates the sympathetic nervous system thus, presumably, resulting in a balanced autonomic nervous system.
[1] Kolettis, TM and Tsatsoulis, A. Subclinical hypothyroidism: An overlooked cause of atrial fibrillation? Journal of Atrial Fibrillation, Vol. 5, No. 4, December 2012-January 2013, pp. 6-8

Seasonal variation in AF-related stroke

AALBORG, DENMARK. There is evidence that hospital admissions for certain cardiovascular diseases such as heart attack, sudden death, and heart failure are substantially higher during the winter than during the rest of the year. A similar trend has been reported for hospital admissions related to atrial fibrillation (AF), which seem to be inversely associated with outdoor temperatures. Now a team of Danish and New Zealand researchers reports a significant seasonal variation in hospitalizations for AF-related stroke.

Their study involved 243,000 Danish men and women (48% female) diagnosed with AF during the period 1980 to 2008, and 51,500 New Zealand men and women (48% female) diagnosed with AF during the period 1991 to 2008. The median age at diagnosis was 75 years for men and 78 years for women in the Danish cohort, and 76 years for men and 78 years for women in the NZ cohort. About 33% of Danish study participants and 53% of NZ participants had one or more comorbid conditions with congestive heart failure at 28% being the most common amongst New Zealanders and hypertension at 11.5% being most common amongst Danes.

Hospitalization rate for AF was significantly higher amongst study participants above 65 years of age than for younger ones. It is also clear that the incidence (new cases on an annual basis) of AF-related hospital admissions increased significantly during the study period – about 5% annually in Denmark and 2.6% in NZ for patients older than 65 years. Corresponding numbers for younger participants were 5.4% in Denmark and 0.2% in NZ.

During follow-up, 36,000 Danish study participants and 7518 NZ participants (54.6% females) were hospitalized with stroke. The risk of stroke was found to be 22% higher in winter than in summer in Denmark, and 27% higher in NZ. The risk of dying within 30 days of suffering a stroke was about 20% in both countries as measured during the period 2000 to 2008.

The researchers conclude that the incidence of AF is increasing significantly and that the incidence of AF-related stroke peaks during the winter season in both Denmark and New Zealand.
Christensen, AL, et al. Seasonality, incidence and prognosis in atrial fibrillation and stroke in Denmark and New Zealand. **BMJ Open**, *Vol. 2, No. 4, August 24, 2012*

Editor's comment: The finding that the incidence of AF-related stroke peaks in winter in intriguing. The authors suggest that climate factors such as ambient temperature, humidity, and hours of sunshine may play a role. There is evidence that hypertension is more pronounced during winter and that the degree of blood pressure elevation is directly related to blood viscosity and fibrinogen level. Thus it would seem plausible that the observed increase in stroke rate during winter time is related to increased blood viscosity.

New protocol for arrhythmia management

PORTLAND, OREGON. Beta-blockers are often used as first-line treatment for arrhythmias related to an overactive sympathetic nervous system response such as for palpitations (PACs and PVCs) and adrenergically-mediated atrial fibrillation (AF). Unfortunately, there is no such thing as a natural beta-blocker. However, now Drs. Milner and Mikolai of the Center for Natural Medicine in Portland, Oregon suggest that rather than trying to correct an overly sympathetic (adrenergic) autonomic nervous system (ANS) by blocking adrenergic receptors, it may be possible to achieve a balanced ANS by increasing the activity of the parasympathetic branch of the ANS. The parasympathetic arm of the ANS is activated by the neurotransmitter acetylcholine, which is synthesized by the body from choline and vitamin B5 (pantothenic acid).

Drs. Milner and Mikolai now report good results in reducing palpitations (ectopy) and other supraventricular arrhythmias through the use of the Milner Acetylcholine Protocol (MAP). This protocol involves ingesting a water-based mixture of pantethine and choline bitartrate throughout the day. The drink is produced by mixing 500 mg of pantethine and 2000 mg of choline bitartrate in 32 fl.oz (1 liter) of pure drinking water. It is consumed during waking hours with 4 fl.oz (125 mL) being consumed continuously during the first 4 hours after arising, 8 fl.oz during the next 4 hours, 8 fl.oz during

the next 4 hours, and finally, 12 fl.oz during the final 4 hours of the 16-hour "awake cycle".

If results are not satisfactory, the concentration of pantethine can be increased to 1000 mg or 1500 mg with a commensurate increase in choline bitartrate to 4000 mg or 6000 mg. In order to save cost, pantethine can also be replaced by pantothenic acid (twice the amount of pantethine) and choline bitartrate can be replaced by lecithin (2 tablespoons of lecithin per 2000 mg of choline bitartrate).

The authors provide three case studies demonstrating the effectiveness of the protocol in eliminating palpitations. They point out that the MAP is contraindicated in the case of bradycardia and AV heart blocks, and also caution against the use of pantethine and pantothenic acid by patients who are taking antiplatelet agents or anticoagulants for stroke prevention.

Mikolai, J and Milner, M. The Milner Acetylcholine Protocol (MAP) for management of cardiac dysrhythmias. **Townsend Letter**, *February/March 2013, pp. 73-79*

Editor's comment: The MAP may be useful in preventing daytime ectopics and adrenergically-mediated atrial fibrillation, but would be contraindicated for vagal AF.

Aldosterone blockage in atrial fibrillation

WARSAW, POLAND. In my March 2003 research report "*Aldosterone: Villain of the Peace?"* (www.afibbers.org/resources/aldosterone.pdf), I speculated that excess aldosterone or cortisol was implicated in the initiation of paroxysmal atrial fibrillation (AF) episodes and also was responsible for fibrosis of the heart tissue eventually leading to persistent or permanent AF. I also suggested that blocking mineralocorticoid (MC) receptors with aldosterone antagonists (spironolactone or eplerenone) may be effective in preventing AF episodes.

Now Polish cardiologists suggest that blocking excess aldosterone with spironolactone or eplerenone may be effective in preventing paroxysmal and persistent AF and in inhibiting the formation of fibrosis in the heart muscle. They point out that aldosterone promotes inflammation, oxidative stress, dysfunction of the autonomic nervous system, attenuation of baroreceptor activity, fibrosis and necrosis (cell death) of cardiomyocytes (heart muscle cells). They also point out that patients with AF have more MC receptors in the atria than do subjects without AF, and that patients with primary aldosteronism (excessive blood levels of aldosterone) have a 12-fold higher risk of AF. They suggest that therapy with spironolactone or eplerenone may reduce the deleterious effects of aldosterone.

A recent trial (EMPHASIS) involving over 2700 patients with mild heart failure found that therapy with 25 – 50 mg/day of eplerenone reduced the incidence of new-onset AF by almost 50%. Another trial (SPIR-AF) found that a combination of spironolactone and the beta-blocker atenolol significantly reduced the incidence of paroxysmal AF episodes over a 12-month period. Spironolactone on its own has also been found to reduce left atrial dimension and extent of fibrosis when taken on a long-term basis (25 mg/day). The researchers conclude that aldosterone antagonist therapy may be a simple and valuable option to the treatment of paroxysmal and persistent AF.

Dabrowski, R and Szwed, H. Antiarrhythmic potential of aldosterone antagonists in atrial fibrillation. **Cardiology Journal**, *Vol. 19, No. 3, 2012, pp. 223-29*

Editor's comment: It is encouraging to see the idea of aldosterone blockage as a means of preventing atrial fibrillation taken up by other researchers. It would be even more encouraging to see large-scale clinical trials evaluating the merits of aldosterone blockage in the prevention of AF.

Running marathons – The bad, and the ugly!

KANSAS CITY, MISSOURI. Several studies have concluded that moderately vigorous exercise is healthy, whilst prolonged, strenuous exercise is not. A study involving 100 German marathon runners over the age of 50 years found that coronary calcification was far more pronounced in the runners than in healthy controls not engaged in strenuous, sustained exercise. A Danish study recently concluded that athletes who regularly engaged in intense physical endurance sports had a 5 times greater risk of developing atrial fibrillation (AF) than did less vigorously exercising or

sedentary controls. A study involving 17,000 male physicians in the USA concluded that regular jogging was associated with a 53% increased risk of developing AF in all age groups, with those jogging in excess for 4 miles a day having the greater risk.

Now a group of American medical doctors interested in sports medicine provide a prescription for just the right amount of exercise. They emphasize that too little exercise is still the major public health problem, but caution that too much exercise can have serious adverse consequences. There is evidence that higher "dosages" of running are not necessarily healthier. Maximal benefit is achieved from moderate-to-vigorous exercise (fast walking or slow jogging) for approximately 40 minutes. On the other hand, running marathons or even half-marathons is likely to do serious damage to the heart, a condition the authors name "cardiac overuse injury". Cardiac overuse injury may involve premature aging of the heart, arrhythmia, accelerated coronary atherosclerosis, and even sudden cardiac death.

The authors conclude that "more" is probably not "better" when it comes to exercise, and participants should not harbour the illusion that if one hour of vigorous activity is good for health, then doing 4 hours of strenuous physical activity will multiply the health benefits. Accumulating data suggests that it does just the opposite – destroys the protective health benefits of exercise. They suggest the following "prescription" for exercise:

- Run 2 or 3 miles at a comfortable pace 2 to 4 times a week.
- Make swimming a regular part of activity. Lift weights or do other strength training 2 or 3 times weekly.
- Do yoga or some other stretching exercises regularly.
- Walk or garden as much as your heart desires.

Bhatti, SK, Hagan, JC, et al. The lady doth protest too much, methinks. Missouri Medicine, Vol. 110, January/February 2013, pp. 17-20

Editor's comment: For more on the benefits/dangers of endurance exercise, see my research report *Endurance Exercise – Is it Worth it*? www.afibbers.org/resources/endurancesports.pdf

Episode frequency and ablation outcome

YOKOSUKA, JAPAN. Paroxysmal (intermittent) atrial fibrillation (self-terminating episodes lasting less than 7 days) has a tendency to progress to persistent and long-standing persistent (permanent) atrial fibrillation (AF). Dutch researchers have reported that the one-year average rate of progression is 15%, although only 7% for lone AF. It is well-established that it is significantly more difficult to achieve success in ablating persistent and permanent AF than in ablating paroxysmal AF (PAF). Thus it is pertinent to ask the question, "Are more frequent paroxysmal episodes associated with poorer ablation outcome?" A group of Japanese researchers from the Yokosuka Kyosai Hospital now provides the answer to this question.

Their study included 362 consecutive patients (average age of 61 years, 76% male) who had suffered from highly symptomatic, drug-refractory PAF for about 5 years. The majority (84%) had lone AF, that is, AF without underlying structural heart disease (cardiomyopathy, coronary artery disease, and valvular heart disease). Average left atrium diameter was 38 mm (3.8 cm) and mean left ventricular ejection fraction was 66%. The frequency of PAF episodes was daily in 40% of patients, weekly in 31%, monthly in 25% and yearly in 4%.

All patients underwent a pulmonary vein antrum isolation (PVAI) procedure using an 8 mm, non-irrigated ablation catheter under electrophysiologic guidance. The left atrial wall was also ablated and a cavotricuspid (right atrial flutter) ablation performed at the end of the procedure. If frequent atrial premature ectopics (PACS) were present, focal ablation was performed. Acute success was 100% and complication rate was low at 2.5% with cardiac tamponade at 1.4% and phrenic nerve injury at 0.6%. All complications were treated successfully or resolved on their own within a week.

A year from the initial procedure, 63.5% of all patients were free of AF recurrence without the use of antiarrhythmic drugs. At 2 years, the complete success rate was 61.8%. A Cox regression analysis of variables likely to affect ablation outcome including AF frequency, presence of

hypertension or structural heart disease, left atrium size, left ventricular ejection fraction, and body mass index (BMI) demonstrated that left atrium size was the only independent predictor of AF recurrence after a single procedure. The frequency of AF episodes prior to the PVAI procedure did not affect ablation outcome either as a single variable or after correction for possible confounders.

The Japanese researchers suggest that the trigger and substrate for PAF is likely still to be found within the isolated pulmonary veins and antrum irrespective of episode duration and therefore one would not expect a difference in outcome depending on episode frequency.

Miyazaki, S, et al. Impact of the preprocedural frequency of paroxysmal atrial fibrillation on the clinical outcome after catheter ablation. **PACE**, *Vol. 35, October 2012, pp. 1236-41*

Editor's comment: Electrophysiologists at Maastricht University have developed a scoring system (HATCH) for estimating the likelihood of progression of PAF to persistent or permanent. The formula for the HATCH score is similar to that of the $CHADS_2$ score and allocates points as follows:

- Hypertension – 1 point
- Age > 75 years – 1 point
- Chronic obstructive pulmonary disease (COPD) – 1 point
- Heart failure – 2 points
- Stroke or transient ischemic attack (TIA) – 2 points

They observed that only 6% of patients with a HATCH score of 0 progressed to persistent AF, whilst among those with a score above 5, almost 50% progressed to persistent.

A 2005 Canadian study concluded that 25% of patients originally diagnosed with paroxysmal afib progress to permanent within 5 years of initial diagnosis. Major risk factors for progression were aortic stenosis, an enlarged left atrium, moderate to severe mitral regurgitation, and cardiomyopathy. A 2005 afibbers.org survey of 188 lone afibbers concluded that the risk of progression from paroxysmal to permanent was associated with a family history of AF, having undergone one or more cardioversions, having developed hypertension after diagnosis, and having an enlarged left atrium.

Smartphone app for atrial fibrillation

WORCESTER, MASSACHUSETTS. The gold standard for detecting atrial fibrillation (AF) is the 12-lead electrocardiogram (ECG). Whilst this is an excellent choice for AF diagnosis in a hospital or clinical setting, it is not practical for afibbers who wish to determine whether or not they are actually experiencing an episode at a particular point in time.

A team of electrophysiologists, engineers, and mathematicians associated with the University of Massachusetts and Worcester Polytechnic Institute now propose that a smartphone can be used to quickly and accurately determine whether an afibber is in AF or in normal sinus rhythm (NSR). The user places their right index or second finger on the smartphone camera for 2 minutes. Applications software built into the phone uses some fairly complicated algorithms (root mean square of successive RR differences and Shannon entropy) to analyse the pulse waves picked up by the camera and then displays pulse rate and indicates whether one is in AF or NSR.

The team tested their idea on 76 persistent afibbers scheduled for cardioversion using an iPhone 4S. The average age of the study participants was 65 years, 59% were male, and 40% had either coronary artery disease or heart failure. Clinical characteristics and algorithm means before and after cardioversion were as follows:

	AF	**NSR**
Systolic BP (mm Hg)	131	112
Diastolic BP (mm Hg)	81	68
Heart rate (beats/min)	91	70
Respiration rate (breaths/min)	19	16
RMSSD/mean*	0.29	0.08
Shannon entropy	0.80	0.45

* RMSSD/mean = root mean square of successive RR difference

The algorithm combining RMSSD/mean and Shannon entropy was 100% accurate in diagnosing irregular pulse and 96% accurate in diagnosing NSR. The researchers conclude that the iPhone 4S equipped with the new app reliably distinguishes an irregular pulse (AF) from pulse wave forms obtained during NSR.

McManus, DD, et al. A novel application for the detection of irregular pulse using an iPhone 4S in patients with atrial fibrillation. **Heart Rhythm**, *Vol. 10, March 2013, pp. 315-19*

Editor's comment: Although most afibbers are well aware whether they are in AF or not, the iPhone app would clearly be useful in occasionally checking for asymptomatic episodes, or to resolve doubts about whether AF is present or not.

The Danger of Pradax – A Personal Story

by Michael W.

When Pradax first became available in October 2010 I was about to go on warfarin since my AF had become very frequent. When I read the story in the *Vancouver Sun* about dabigatran being released, I thought "Hey, that's for me. No need to get regular blood tests for INR that are needed with the rat poison". So I asked my cardiologist for a prescription. He asked me to spell dabigatran for him (that should have been a warning).

I took dabigatran for two years without any apparent ill-effect. During that period I had 3 pulmonary vein ablations (PVAs) – each one improving my AF to where I can hopefully now say it is fixed. I had my last PVA on 12 September 2012. In early November 2012 I was alone at our cabin on the east side of Manning Park. I went to bed on the night of 2 November feeling just fine. On the morning of 3 November I woke up barely able to get out of bed. Although I had a satellite phone with me it was downstairs and needed to be taken outside the cabin to make calls. There was no way that I could accomplish either of those steps. Luckily, one of our neighbours was coming round that morning to help me with some work on the cabin. He used the satellite phone to call the ambulance service but help was awhile in coming since my neighbour had to go out to the highway to meet the paramedics and then ferry them and their equipment through the river in his truck. Needless to say, ambulances are not designed to drive through rivers.

To cut a long story short, I was suffering from pericardial bleeding and my heart and other vital organs were shutting down. I was evacuated from the cabin by helicopter to Kelowna hospital and then by jet air ambulance to Vancouver General Hospital. It was touch and go but a combination of dialysis and draining my pericardium saved my life. I was very lucky. I subsequently had a conversation with a Lions Gate Hospital specialist who told me that they had recently had three similar cases – two of the patients had died.

The problems with Pradax are: (1) that there is no way of measuring its level in the body, (2) there is no antidote for it (unlike vitamin K for warfarin) (3) bleeding can happen very quickly and without warning (as it did with me). Also, it would appear that Amiodarone exacerbates the effect of Pradax (while also impeding kidney & liver function that gets Pradax out of the system). I happened to be taking Amiodarone at the time (although I have been off it since the incident).

Frankly, Pradax should be taken off the market until an antidote is found for it. So far it has killed over 500 people in the USA and the lawyers are having a field day with lawsuits against the drug manufacturer. The FDA and the Canadian equivalent are bumbling around as usual – undoubtedly succumbing to pressure from Big Pharma. It would not surprise me if the HMOs in the US are supportive of Pradax (since it avoids them paying the cost of monitoring INR levels).

As you can see, I have a pretty dim view of pharmaceutical companies. My experience with the array of antiarrhythmic drugs thrown at me before I had the PVAs confirms that the medical profession has a rather

cavalier attitude towards drug side-effects – “Hey, we sure fixed that arrhythmia! Pity the patient died from the side-effects of the drug…..”

I have to close by saying that the BC Health & Emergency Services and hospitals were magnificent. I owe my life to them.

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