THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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The importance of a thorough medical check-up before accepting the diagnosis of lone or, more accurately, idiopathic atrial fibrillation was brought home to me recently. A 6-year veteran of vagal afib reported that he had finally had extensive testing done on his thyroid gland and its function. A nodule was found and surgically removed last month. He has been in sinus rhythm ever since. Prior to the operation he had experienced 12- to 24-hour episodes every 12 to 14 days.

Thyroid disorders, hypoglycemia and electrolyte imbalances are known causes of LAF that should be ruled out before "joining the club" in search of the elusive "cure". In this issue we cover the most common known causes of LAF.

In addition we bring you the personal "journey" of Mark Murphy, MD. Mark underwent an ablation almost 3 years ago and has remained afib-free ever since. Many other afibbers have had "their life given back" as a result of ablation therapy and are understandably very enthusiastic about the procedure. However, according to our recent survey for every successful ablation there is an unsuccessful one. The overall success rate among 42 respondents to our survey was only 45%. An experienced surgeon like Dr. Andrea Natale at the Cleveland Clinic has an enviable 85% success rate, but there are a great number of inexperienced surgeons whose success rate is nowhere near this figure. One afibber reported that her surgeon only explored the right atrium because he did not feel comfortable going into the left. This does not make a lot of sense as at least 80% of the offending focal points are found in the left atrium. Needless to say, her ablation was not a success. We will discuss our survey results in greater detail in the next issue. In the meantime, unless you have a really experienced surgeon performing your ablation you may be better off waiting.

Yours in health and sinus rhythm,
Hans Larsen

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My Journey: A Happy Ending

by Mark Murphy, MD

My journey with LAF began with my first episode in September of 1997. I had been in excellent health up until this point. In addition, I routinely engaged in aggressive physical exercise. In any event, over time I began to develop recurrent episodes of PACs. These would occur after a heavy meal, or strenuous exercise. I did not give this much thought until these episodes degenerated into bursts of atrial fibrillation. I went to see a cardiologist who suggested that I take digoxin and not worry about it.

The episodes worsened and the discomfort, which included lightheadedness, dizziness, and extreme anxiety prevented me from working at my medical practice. My own research into this disease led me to begin regimens of various other medications. I first tried beta-blockers without success and the side effects, which included fatigue and decreased exercise tolerance precluded their use. Calcium channel blockers also failed. The more exotic antiarrhythmics including flecainide and propafenone helped only transiently and were associated with intolerable side effects.

I now realized I had to actively seek out some way of dealing with this disease process. I began to travel the country and interview electrophysiologists who were truly familiar with the pathophysiology of LAF. It seemed that a cure was a real possibility if the site of origin could be located and ablated with RF. I finally decided on an electrophysiologist in Provo, Utah. Although various centers around the country were performing the procedure and doing more research and development than this individual, I felt that his clinical skills offered me the best hope for a cure. In February of 2000, I underwent the procedure. It required one night's stay in the hospital and minimal discomfort. There was some associated chest soreness and groin pain from the catheter insertion sites. My postoperative course was complicated by one episode of AF, but after that time all ectopy gradually diminished and I was able to wean myself off all medications. I would say it took about three months to feel completely normal and without fear of going into AF.

I now feel as if I have my life back and could not be more grateful. I went from having episodes of AF every other day to now being in perfect normal sinus rhythm. My exercise routine is back to normal. I have regained fifteen pounds of lean weight that was lost secondary to side effects of medications. My only hope is that there would be a greater public and patient awareness of this nasty disease. I firmly believe a routine cure will be found in the near future, if the electrophysiologists who are actively ablating LAF are given credibility and support. As it is now, the experienced EPs are pushing a 90% success rate. This website provided me with tremendous hope throughout my journey with LAF, and Hans, I would like to thank you very much.

Causes of LAF

Lone atrial fibrillation is a bit like a fire. Just as it takes an ignition source (match) and a substrate (dry wood) to start and sustain a fire, so does it take an ignition source (trigger or precipitating cause) and a substrate (abnormally sensitive heart tissue) to begin and sustain a LAF episode. About 60% of the population experience PACs (premature atrial complexes) on a regular basis, but less than 1% has LAF, so just having PACs is clearly not sufficient to provoke and sustain an episode. Similarly, even though afib is common among patients with hyperthyroidism, not all such patients have LAF. Finally, afibbers who have their faulty heart tissue isolated via the maze procedure or ablation therapy are fully cured and no longer need to worry about exposure to triggers that used to set off an episode. Several studies have shown that the onset of an LAF episode is usually preceded by heightened sympathetic or parasympathetic activity in the autonomic nervous system. It is therefore likely that most, if not all, precipitating causes exert their effect on the heart through an autonomic nervous system that is dysfunctional, that is, too easily excitable. Thus it would seem that LAF only develops when three conditions are met:

- The autonomic nervous system is dysfunctional;
- The heart tissue is abnormally sensitive and capable of being triggered into and sustaining an afib episode;
- A trigger or precipitating cause capable of initiating an episode is present.

If indeed a sensitive heart tissue and a precipitating cause are both required to initiate an afib episode, then it follows that if the precipitating cause (underlying condition) is removed the episodes cease. I believe this is true up to a point. If, due to repeated episodes, the heart has been extensively re-modeled and made super sensitive to "ignition sources" then it is possible that another ignition source may take the place of the removed one and that episodes may continue. I am not aware of any medical research on this topic, so at this point, this is just speculation. Nevertheless, there are afibbers who have banished LAF by removing the underlying cause, so the possibility is certainly there.

Several conditions and diseases have been implicated in LAF. Alcohol intoxication, cocaine use, certain pharmaceutical drugs, viral and bacterial infections, pulmonary embolism (blood clot in the lungs), and tyramine-containing foods are well-recognized triggers. Specific conditions such as thyroid disorders, hypoglycemia, electrolyte imbalances, and pheochromocytoma may also be the underlying cause of LAF and chromosomal (gene) abnormalities may predispose some individuals to LAF[1-10]. The most likely trigger mechanism for lone atrial fibrillation is, however, an imbalance in the autonomic nervous system (ANS)[11].

A. Alcohol and Drugs

Alcohol intoxication (binge drinking) and withdrawal are both fairly common initiators of afib episodes[1-5,7-9]. It is likely that alcohol exerts its effects through changes in ANS activity particularly in the withdrawal (hangover) stage[7]. It has been estimated that as many as 40% of alcohol abusers suffer from periodic LAF episodes[5,9]. The effect of alcohol abuse may be reversible, that is, once alcohol abuse ceases the episodes may stop as well[7,12]. This assumes that the heart has not been remodeled by repeated afib episodes and has become super sensitive. So if you ever experience an afib episode after a night of heavy drinking, heed the warning and eliminate or at least seriously reduce your alcohol intake; the alternative may be life-long atrial fibrillation.

Cocaine and particularly cocaine in combination with alcohol markedly increases sympathetic (adrenergic) activity and can cause a variety of cardiac arrhythmias including fatal ventricular fibrillation[4].

Several pharmaceutical drugs including digoxin (Lanoxin, digitalis) and some drugs (theophyllines) used in the treatment of asthma, chronic bronchitis, and emphysema (chronic obstructive pulmonary disease) can trigger an afib episode. Most antiarrhythmic drugs can also act as initiators under certain conditions[3,5,6].

B. Tyramine-Containing Foods

Tyramine is a chemical (monoamine) that has been found to be a strong catalyst of norepinephrine release from cardiac nerve endings. It thus exerts its effect on the heart through the ANS[13]. Tyramine-containing foods have been associated with the initiation of both migraines and afib episodes[14,15]. Tyramine is found in many foods notably aged cheese, blue cheese, dark chocolate, overripe fruits (bananas and avocados), citrus fruits, and certain red wines. Tyramine related afib episodes are likely to cease if the consumption of the offending foods is stopped[14]. It is interesting that many of the triggers identified in our LAF surveys are also triggers for migraines. These include food additives, MSG, aspartame, caffeine, and alcohol. Migraines can also be triggered by hypoglycemia and, interestingly enough, by beer or whisky distilled in copper stills[15]. Tyramine activates the adrenergic branch of the ANS so it is likely that purely vagal afibbers would have a lot less problems with tyramine-containing foods than would adrenergic or mixed afibbers. Actually, a piece of aged cheese or dark chocolate after dinner may be just the ticket for vagal afibbers. See the following appendix for a listing of tyramine-containing foods.

C. Thyroid Disorders

An appropriate supply of thyroid hormones to the cells of the body is essential for normal functioning. The thyroid gland is situated at the base of the throat and produces two hormones, thyroxine (T4) and triiodothyronine (T3). Both of these hormones are iodinated amino acids and it is estimated that the thyroid gland requires an iodine supply of about 100-200 micrograms/day in order to produce them. The production of T3 and T4 is stimulated by the thyroid-stimulating hormone known as TSH or thyrotropin. TSH is produced in the pituitary gland and its secretion is regulated by the central nervous system via the thyrotropin-releasing hormone (TRH). Almost all the circulating thyroid hormones are bound to plasma proteins and only about 0.05% are actually found free in blood plasma. These free hormones can, however, have a highly significant effect and sensitive methods are available to measure them (FT4 and FT5)[16,17]. Some naturopaths and chiropractors use basal temperature (morning temperature taken under the armpit before arising) as a means of checking for

subclinical hypothyroidism and hyperthyroidism. A normal basal temperature is between 97.6 and 98.2 degrees F or 36.4 to 36.8 degrees C. A temperature above this range may indicate hyperthyroidism while a temperature below the normal range may indicate hypothyroidism.

Thyroid hormones may act directly on the heart tissue or can affect the functioning of the heart via the ANS[18]. Both an excess (hyperthyroidism) and a deficiency (hypothyroidism) of thyroid hormones have been linked to atrial fibrillation, but the evidence for a connection with hyperthyroidism (thyrotoxicosis) is much stronger than the evidence for a connection with hypothyroidism.

Hyperthyroidism (Thyrotoxicosis)

A toxic level of thyroid hormones in the blood (thyrotoxicosis) has been clearly linked to an increased risk of atrial fibrillation. However, the number of LAF patients who actually have hyperthyroidism is probably less than 3%[7]. Thyrotoxicosis is usually caused by an overactive thyroid gland (hyperthyroidism). Clinical (overt) hyperthyroidism is fairly simple to diagnose as both T3 and T4 levels are highly elevated and TSH levels are extremely low. Hyperthyroidism also has distinct clinical features such as weakness, fatigue, weight loss, heat intolerance, irritability, palpitations, and tremulousness. It is usually treated by inactivating part of the thyroid gland either by surgery or through injection of radioactive iodine. Unfortunately, it is easy to "overshoot" in this treatment with the result being hypothyroidism and a life-long dependency on synthetic thyroid hormones (levothyroxine).

There is increasing evidence that not just overt hyperthyroidism, but also subclinical hyperthyroidism can result in atrial fibrillation[19-21]. Subclinical hyperthyroidism is diagnosed when T3 and T4 levels are normal, but the TSH level is low and there are no overt symptoms of hyperthyroidism. Recent research has found that both clinical and subclinical hyperthyroidism have a profound effect on the ANS by increasing sympathetic (adrenergic) activity and decreasing parasympathetic activity[18,21]. Austrian researchers have found that people with undiagnosed (subclinical) hyperthyroidism are 5 times more likely to develop atrial fibrillation than are people with normal thyroid hormone levels. The researchers studied 23,638 people and found that those with low values of serum thyrotropin (TSH) (less than 0.4 mU/L) but normal values of free triiodothyronine (T3) and free thyroxine (T4) had an incidence of atrial fibrillation of 12.7%. This compared to an incidence of 2.3% among people with normal TSH levels and an incidence of 13.8% in those with diagnosed hyperthyroidism[19].

It is clear that both clinical and subclinical hyperthyroidism are important risk factors for atrial fibrillation and that all afibbers need to have their thyroid function tested in order to rule out these disorders as a precipitating cause. It is important to keep in mind that even normal levels of thyroid hormones (T3 and T4) may be toxic to some people so a very careful ("sensitive") analysis of TSH level is a must.

It is also a good idea to check the basal temperature. In our latest LAF survey 3 out of 22 respondents reported a high morning temperature (98.6 to 100 degrees F), which may indicate an overactive thyroid gland. The 3 high-temperature afibbers had considerably more and longer afib episodes than did afibbers with normal basal temperatures.

Hypothyroidism (Myxedema)

The first inkling that an underactive thyroid gland (hypothyroidism) could be associated with atrial fibrillation came in 1993[22]. In 1996 animal experiments confirmed the link[23]. More recent research has found a clear correlation between hypothyroidism and an increased sympathetic influence on the autonomic cardiovascular system[24]. Hypothyroidism is a fairly common disorder and is usually caused by an intrinsic defect in thyroid structure or biosynthetic mechanisms; insufficient secretion of TSH and TRH hormones are other possible causes. The disease tends to affect women more than men. Hair loss, fatigue, feeling cold, constipation, skin pallor, thick dry skin, and memory impairment are among its most common symptoms[16,17].

Hypothyroidism is diagnosed through the presence of low or normal levels of T3 and T4 hormones combined with an abnormally high level of TSH. It is treated with life-long medication with synthetic thyroid hormone (levothyroxine). As in the case of hyperthyroidism much attention has been focused lately on the prevalence of subclinical hypothyroidism. This condition is actually quite common with an estimated prevalence of 7-8% in women and about 3% in men. It is characterized by an elevated TSH level and normal T4 levels. Again it

should be kept in mind that the T4 level varies widely between individuals and a level that may allow one person to function quite well may be too low or too high for another person[25].

The reference range for TSH is 0.2 – 5.5 mU/L. This value was arrived at by measuring TSH levels in a large group of seemingly healthy people. A group of British researchers recently suggested that this range maybe too wide. They point out that when a group of individuals with no history of thyroid disease and no antibodies against thyroid peroxidase was tested their TSH values fell within a much narrower range of 0.48 – 3.60 mU/L. Other studies have shown that TSH levels above 1.9 mU/L are associated with thyroid peroxidase antibody positivity indicating abnormal pathology in the thyroid. There is also evidence that individuals with TSH values greater than 2.0 mU/L have an increased risk of developing clinical hypothyroidism over the next 20 years. The researchers believe that a TSH range of 0.5 – 3.5 mU/L rather than a range of 0.2 – 5.5 mU/L should be considered the norm, but point out that there is emerging epidemiological data suggesting that a TSH level above 2.0 mU/L may be associated with adverse effects[76].

In our recent LAF survey 22 respondents provided their basal temperature. Nine (41%) had a normal value while 10 (45%) had a low value. Of the 10 with low values 4 had actually been diagnosed with hypothyroidism; their basal temperatures were 96.5, 97.0, 97.1 and 97.3 degrees F respectively. The 6 undiagnosed respondents with low values had readings of 96.4, 96.8, 97.0, 97.1, 97.2 and 97.3. This may indicate that the 6 respondents with low values also have hypothyroidism. The finding that 45% of the, albeit, fairly small group of survey respondents had a low basal temperature lends support to the idea that hypothyroidism, whether diagnosed or subclinical, may be an important precipitating cause of LAF. It is also worth noting that afibbers with low basal temperatures tended to have more severe episodes than did afibbers with normal temperatures and that the two permanent afibbers who provided their temperatures both had low readings.

It is well established that both diagnosed and subclinical hyperthyroidism can cause atrial fibrillation and there is some evidence that both diagnosed and subclinical hypothyroidism can as well. Clearly, all afibbers should have a thorough evaluation of their thyroid function to rule out this possible cause of afib.

D. Hypoglycemia

Hypoglycemia (low blood sugar) can trigger an atrial fibrillation episode[3,26,27]. The disorder manifests itself as an excessive drop in blood sugar levels 3 to 6 hours after eating. A hypoglycemic episode is treated as a major emergency by the ANS; it proceeds to dump vast quantities of epinephrine into the blood stream in order to prompt the liver to release glucose for use by the starving brain. The chaos created by this sequence of events is what causes the afib episode.

Our first LAF survey showed that 25% of all respondents had hypoglycemia and another 24% had symptoms of hypoglycemia. Common among these symptoms are:

- craving for sweets;
- irritability or weakness if meal is missed;
- dizziness when standing up suddenly;
- heart palpitations;
- afternoon fatigue;
- tiredness an hour or so after eating;
- depression or mood swings.

The best way to diagnose hypoglycemia (idiopathic postprandial syndrome) is by measuring the blood level of glucose during an episode. A 2- or 5-hour postprandial glucose test may also reveal the presence of hypoglycemia if the glucose level is found to be below the morning fasting value. The actual blood glucose level that causes hypoglycemic reactions can vary considerably between individuals. A study carried out at the University of Montreal showed that a majority (83%) of patients with suspected postprandial hypoglycemia had average glucose levels of 4.3 mmol/L (76 mg/dL) at the time of their symptoms[28].

Hypoglycemia has been implicated in such diverse conditions as criminal behaviour, premenstrual syndrome, migraine headaches, atherosclerosis, and atrial fibrillation. It is best controlled by religiously avoiding foods with a high glycemic index (sugar, white and whole grain bread, bananas, raisins, potatoes, rice, and wheat cereal) and by eating frequent small meals throughout the day. Alcohol should also be avoided and the intake of dietary fiber increased. A daily multivitamin (and minerals) capsule is very important and a minimum intake of 200-400 micrograms/day of chromium may also be beneficial.

A hypoglycemic-induced LAF episode can sometimes be aborted by quickly consuming a "power bar" or a high glycemic index food like bananas or raisins. It is best to follow up with a snack of low glycemic index food (berries or nuts) in order to avoid a "yo-yo" effect. Hypoglycemia is relatively easy to keep in check and doing so may significantly reduce the number of LAF episodes.

It is also possible, but purely speculative on my part, that a blunted glucose response could be associated with LAF. Clinical trials carried out at the University of Illinois during the late 40s and early 50s established the existence of the so-called "flat" glucose tolerance curve in patients suffering from fatigue. These patients had slightly lower than normal fasting glucose levels, but the key difference was that their blood glucose level rose by an average of only 28% one-half hour after ingesting sugar. In contrast, the glucose level of the controls rose by about 73%. Also, after one hour the glucose levels of the controls were still 32% higher than fasting levels while the fatigue patients' levels were only 4% higher. The researchers concluded that flat curves are associated with excessive vagal (parasympathetic) stimulation[29]. Perhaps this excessive vagal stimulation could help explain the tendency among vagal afibbers to experience episodes after a meal. This connection is supported by the recent finding that the parasympathetic nervous system plays a major, if not dominant, role in postprandial (after a meal) insulin release. If the parasympathetic system is activated in order to cause insulin production to occur it is possible that this higher level of activation could be instrumental in initiating an episode. So restricting the intake of carbohydrates in the evening meal may be worth trying if episodes tend to occur after supper[30].

E. Electrolyte Imbalances

Heart cells (myocytes) are "powered" by the in- and out-flow of four ions (electrolytes) – sodium, calcium, potassium and magnesium. The cells contract when sodium and calcium flow in and potassium and magnesium flow out. They relax when sodium and calcium flow out and potassium and magnesium re-enter the cell. A proper balance between the four electrolytes is essential to ensure a steady heart beat and serious imbalances are recognized initiators of atrial fibrillation[2].

An excessive or inadequate amount of potassium in the intracellular fluids surrounding the myocytes (hyperkalemia or hypokalemia) can cause serious ventricular arrhythmias in patients with certain types of heart disease. A high level of calcium (hypercalcemia) has also been linked to arrhythmias, as has a low level of magnesium. There is also evidence that magnesium injections can stop certain arrhythmias[4]. Normal plasma levels of the electrolytes are[31,32]:

- Sodium 137-145 meq/L (137-145 mmol/L)
- Potassium 2.7-3.9 meq/L (2.7-3.9 mmol/L)
- Calcium 2.1-3.0 meq/L (1-1.5 mmol/L)
- Magnesium 2.0-2.5 meq/L (1-1.2 mmol/L)

Only 1% of the body's magnesium stores are found in the blood so plasma level is not a good indicator of the level in the cells of the heart.

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Appendix – Tyramine-Containing Foods

Aged cheese
Avocado
Baked goods [1]
Banana
Beer [2]
Blue cheese
Bologna
Bovril
Brewer's yeast
Broad beans
Caviar
Cheddar cheese
Chocolate
Cocoa
Cola drinks
Commercial gravy
Fermented soy products
Feta cheese
Flavoured gelatin
Ginseng
Granola and muesli [3]
Left over meat
Left over poultry or fish
Lentils
Lima beans
Lox
Marmite
Miso
Nutritional yeast
Over-ripe fruits
Over-ripe vegetables
Oxo
Oysters
Pecans
Pepperoni
Pickled herring
Pickled vegetables
Pickles and relishes
Plums
Potato
Prunes
Raisins
Raspberries
Rice and pasta [4]
Salad dressings [5]
Salami
Sauerkraut
Seasoning "flavour" packets
Sherry
Shrimp paste
Smoked and aged meats
Smoked or pickled fish
Smoked salmon
Soy sauce
Soybean paste
Soybeans
Spinach
Sweet potato
Teriyaki sauce
Tomato
Vegemite
Vermouth
Vinegar (all types)
Walnuts
Wine (especially red)
Yoghurt

[1] with excessive quantities of yeast
[2] including alcohol-free beer
[3] with raisins, banana or any over-ripe fruit and walnuts or pecans
[4] with "flavour" packets
[5] most commercial varieties

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