THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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Editorial

*Welcome to the second issue of **The Afib Report**. Our aim is to keep you abreast of new discoveries concerning atrial fibrillation but, even more important, to ultimately help find a solution to the problem. In order to maximize your benefits from **The Afib Report** I would highly recommend that you read my earlier report entitled "Lone Atrial Fibrillation: Causes and Management" (www.yourhealthbase.com/atrial_fibrillation.html). Although this report covers many of the basics of LAF some elaboration is needed in order to gain a fuller understanding of the problem and be able to interpret the significance of new findings. So the next few issues will be a combination of basic background information and the latest news. If you are already conversant with cardiology and electrophysiology you can skip these sections.*

Yours in health,
Hans Larsen

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Cardiology 101

The heart, apart from what other mythical and emotional characteristics we may ascribe to it, is basically a living pump. It is one of the hardest working organs in the body; it contracts and expands about 100,000 times every day. It supplies a blood vessel network 96,000 kilometers long and pumps in excess of 10,000 liters of blood around the body every single day. The heart has four chambers, the right *atrium*, the left atrium, and the right and left *ventricles*. The atria are situated above the ventricles with the right atrium being connected to the right ventricle through the *tricuspid* valve and the left atrium being connected to the left ventricle through the *mitral valve*.

Returning blood enters the right atrium from the superior and inferior *vena cava*. It is propelled onward by contraction of the muscular tissue of the atrium and then enters the right ventricle which pumps it through the lungs and back to the left atrium through the *pulmonary veins*. The passage through the lung capillaries eliminates carbon dioxide and other waste products and re-oxygenates the blood. From the left atrium the "rejuvenated" blood flows through the mitral valve into the left ventricle which contracts with enough force to pump the blood through the *aorta* into the smaller arteries and capillaries, where the actual nutrient and oxygen exchange with individual body cells takes place, and then back to the heart through the veins. Immediately after exiting from the heart the aorta branches off into the right and left *coronary arteries* which supply the heart itself with fresh blood and the nutrients it requires.

Electrophysiology 101

The fibers and individual cells of the heart muscle are unique in that they are able to contract and relax spontaneously. Thus the heart will beat, albeit at a very slow rate, even if it receives no external stimuli. Normally though, the operation of the heart is under the control of the autonomic nervous system.

The autonomic nervous system (ANS) controls the body's internal organs including the heart and digestive system and is responsible for regulating blood pressure. It has its origin in the hypothalamus region of the brain from where it divides into two branches – the sympathetic (*adrenergic*) branch and the parasympathetic (*vagal*) branch. The neurotransmitter used in the adrenergic branch is *norepinephrine* (noradrenaline); the parasympathetic system uses *acetylcholine* to transmit its messages.

The adrenal gland is an outgrowth on the adrenergic branch and its medulla (the inner part of the gland) produces two neurotransmitters, norepinephrine and *epinephrine* (adrenaline) collectively known as catecholamines. Norepinephrine is normally synthesized (from the amino acid tyrosine) right at the nerve endings as needed, but when the body is under excessive stress the adrenal medulla kicks in and produces large amounts of both epinephrine and norepinephrine as part of the "fight or flight" reaction and this, as we shall see later, can spell big trouble for afibbers.

The autonomic nervous system is responsible for maintaining the body's inner balance (homeostasis). It does this by continuously adjusting the secretion of the two neurotransmitters, norepinephrine (from the sympathetic nerve endings) and acetylcholine (from the parasympathetic nerve endings). Norepinephrine speeds up muscle contractions and heart rate while acetylcholine slows them down. Constant maintenance of a finely tuned balance is necessary to keep the body functioning at its optimum.

Maintaining a blood pressure sufficient to ensure an adequate blood supply throughout the body but low enough to avoid bursting small capillaries in the brain is perhaps one of the most important tasks of the autonomic nervous system. The cardiac control center of the ANS constantly receives input from *baroreceptors*. These specialized muscle fibers are located in the walls of the heart and the major arteries and they "measure" the blood pressure by stretching and relaxing as the blood flows past them. A lower than desired pressure will cause the ANS to activate the sympathetic nervous system and thus make the heart beat faster while too high a pressure will activate the parasympathetic system.

The atria are suffused with nerve endings from the sympathetic system which also has a direct connection to the *sinoatrial* (or sinus) *node* located at the junction of the superior vena cava and the right atrium. Although nerve endings from the parasympathetic system can also be found throughout the tissue most of the parasympathetic activation takes place at the sinoatrial (SA) node.

Impulses from the SA node spread across the atrium and cause it to contract and relax at a rate of about 70-75 contractions (beats) per minute. When the impulses reach the *atrioventricular* (AV) *node* located near the tricuspid valve the cells of the *bundle of His* are activated. This is followed by activation of the *Purkinje fibers* resulting in contraction of the ventricles.

Sounds complicated? It is, but unfortunately it is absolutely essential to have a clear understanding of the interaction between the heart and the autonomic nervous system if we are to comprehend and eventually vanquish arrhythmias. To sum up, the heart's operation is controlled by the "cardiac control center" of the autonomic nervous system. This center receives input from baroreceptors regarding blood pressure and then activates either the sympathetic or the parasympathetic branch in order to bring the blood pressure into the desired range. An activation of the sympathetic system will speed up the heart and increase blood pressure while activating the parasympathetic system (increasing vagal tone) acts as a brake by slowing the heart and decreasing blood pressure.

The autonomic nervous system, of course, controls several other involuntary body functions and responds to many other stimuli than just blood pressure. However, in so far as arrhythmias and atrial fibrillation are concerned, the heart rate connection is clearly the most important.

The ANS is only capable of maintaining homeostasis within certain limits. Exposure to stressful stimuli such as low blood sugar, extreme temperatures or a visit from the tax inspector can throw it off balance and as a result impair the smooth functioning of the internal organs including the heart. If the heart tissue and SA node are sensitive to autonomic nervous system disturbances it is quite possible that an atrial fibrillation attack or other arrhythmia will result.

Atrial Fibrillation 101

Atrial fibrillation is caused by a dysfunction of the heart tissue or nodes, by a dysfunction of the autonomic nervous system or by a combination of both. As we saw earlier, individual heart cells are capable of "beating" on their own outside the control of the autonomic system. Sometimes agglomerations of very active cells form and create a focus for so called *ectopic beats* (beats originating outside the SA node). The junction between the left atrium and the pulmonary vein is a particularly popular spot for these "rogue" cell agglomerations and some arrhythmias can be successfully treated by removing them with radio frequency ablation. If the ectopic beats become very frequent they may run together and create atrial fibrillation.

Atrial fibrillation basically involves a chaotic movement of electrical impulses across the atria and leads to a loss of synchrony between the atria and the ventricles. Once an attack has begun the atria may quiver or fibrillate at a rate as high as 300 to 600 times per minute. This causes a very inefficient filling and emptying of the atria; the chaos is transferred to the ventricles causing them to lose their regular rhythm and begin to contract fast and in a totally irregular manner. This is what gives rise to the fast and irregular pulse rate felt during an AF attack (90-160 beats/minute).

Atrial fibrillation in itself is not a disease, but rather a symptom of some other disorder of the body. Atherosclerosis, angina, valvular (rheumatic) heart disease, hypoglycemia, hyperthyroidism, anemia, pheochromocytoma, strenuous exercise, binge drinking, consumption of tyramine-containing foods, and exposure to mental or physical stress can all trigger atrial fibrillation. All these conditions have one thing in common – when active they are associated with an excessive release of norepinephrine and, in some cases, epinephrine as well.

Lone (primary) atrial fibrillation (LAF), by definition, is atrial fibrillation without underlying heart disease. So it stands to reason that this arrhythmia is primarily related to a dysfunction of the autonomic nervous system. The dysfunction can be an overactive sympathetic system or an underactive parasympathetic system or perhaps an overactive parasympathetic system followed by a too vigorous correction by the sympathetic system. These are finer points that may be covered in a future issue of The Afib Report. For now suffice it to say that atrial fibrillation ultimately involves an excessive release of norepinephrine from the autonomic nervous system.

Control of Norepinephrine

So what does this mean in terms of preventing lone atrial fibrillation? Clearly the key is to control or inhibit an excessive norepinephrine release. The simplest way to do this is to assist the autonomic nervous system to stay in balance by avoiding trigger factors. Staying away from alcohol, caffeine and tyramine-containing foods, and avoiding excessive physical and emotional stress are a good start. Another very important preventive measure is to avoid large dips in blood sugar levels. There are at least two documented cases of atrial fibrillation associated with *hypoglycemia* (low blood sugar) and probably many more unrecorded ones(1,2).

Hypoglycemia 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 autonomic nervous system; 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 will more than likely result in an AF attack. Hypoglycemic episodes can be avoided by eating small meals throughout the day (including before bedtime) and eliminating sugar and sugar-containing products as well as white flour-based products from the diet. It is also important to base the diet on low glycemic index foods.

Norepinephrine Inhibition and Paroxetine

The idea of inhibiting norepinephrine secretion by pharmaceutical drugs is an intriguing one. Recent work done by Dr. Jack Gorman, MD at Columbia University concludes that the antidepressant paroxetine (Paxil) may normalize heart rate variability and, in turn, help prevent panic attacks(3). Panic attacks, in many respects, are similar to LAF attacks. I actually tried paroxetine a couple of years ago and found that 20 mg/day did indeed significantly reduce the frequency of my attacks.

Prior to starting on paroxetine I experienced a LAF attack every 7 to 14 days and each one lasted between 12 and 17 hours. I had an attack 10 days after starting the paroxetine, but then went 55 days without one. The interval before the next one was 37 days, but this attack lasted 20 hours. Then it was 76 days without an attack, but when it occurred it lasted 108 hours. The next one came 40 days later and lasted 58 hours. However, it took the form of severe *bradycardia* with heart rates as low as 39 beats/minute. I later came across an article by Erfurth et al [ECG changes after paroxetine: 3 case reports. Nervenarzt 1998 Jul; 69(7):629-31] that reported 2 cases of severe bradycardia in connection with paroxetine treatment. So to make a long story short, I found paroxetine very helpful in the beginning, but had to discontinue it after the bradycardia episode which I found very scary. It may be that paroxetine in smaller doses (5-10 mg/day) may be helpful and have fewer side effects. If anybody tries it please let me know how it works out.

News

Researchers at the Mayo Clinic report that sildenafil (Viagra) can increase sympathetic nerve activity by as much as 141% and norepinephrine release by more than 30%. Definitely not a good choice for afibbers(4).

Medical doctors at the St. James University Hospital in the UK have found that drinking large quantities (500 ml) of water significantly increases sympathetic activity. Athletes may want to consider this when they rehydrate after exercise(5).

That's all for now. I hope you found this issue of The Afib Report useful. In future issues we plan on covering the benefits of supplementation as well as the role of a magnesium deficiency and amalgam dental fillings in the promotion of lone atrial fibrillation. We will also take a detailed look at antiarrhythmic drugs, RF ablation surgery, and the maze operation. Please let me have your feedback.

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