BHS Cardiology
CARDIAC ELECTROPHYSIOLOGY

Guide to Atrial Fibrillation

A. How does the normal heart work?

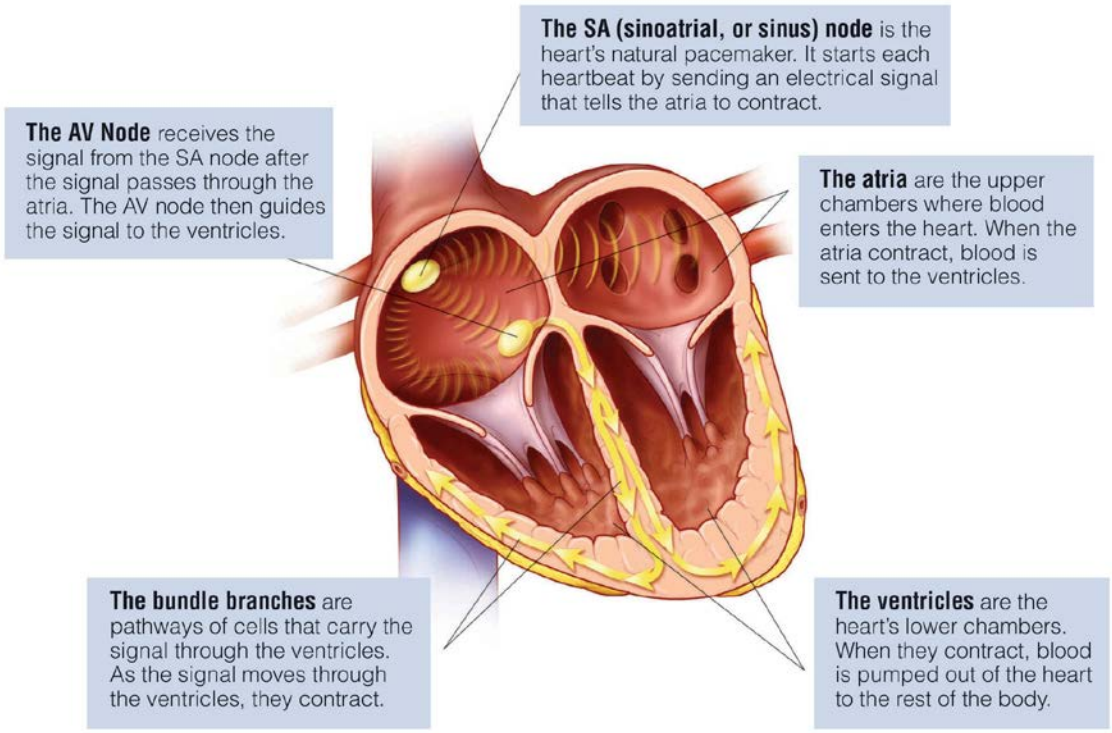
The heart is a pump, and is part of a system which circulates blood around the body to supply oxygen and nutrients to the body tissues. It has 4 key components:

1. A pumping mechanism, which in the case of the heart is a muscle which contracts around a cavity into which blood flows. When the muscle contracts, the cavity is obliterated and the blood is sent elsewhere.

2. valves which are embedded in the muscle and channel the blood efficiently.

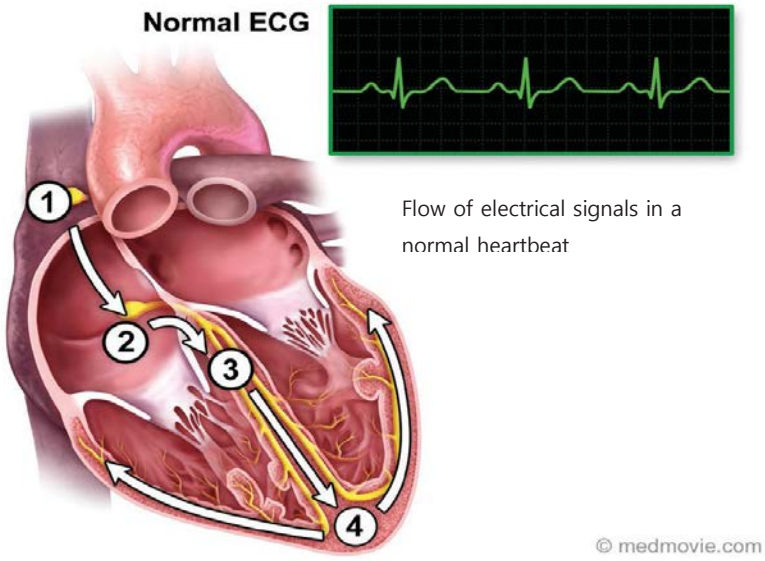
3. arteries, which supply the muscle with oxygen and nutrients.

4. an electrical system. Like most pumps, electricity makes the heart go. The brain determines what the body needs, and sends signals to the heart's electrical system via nerves, and the electrical system carries out the task of dictating the pumping rate. The figure below shows the setup of the heart's electrical system.



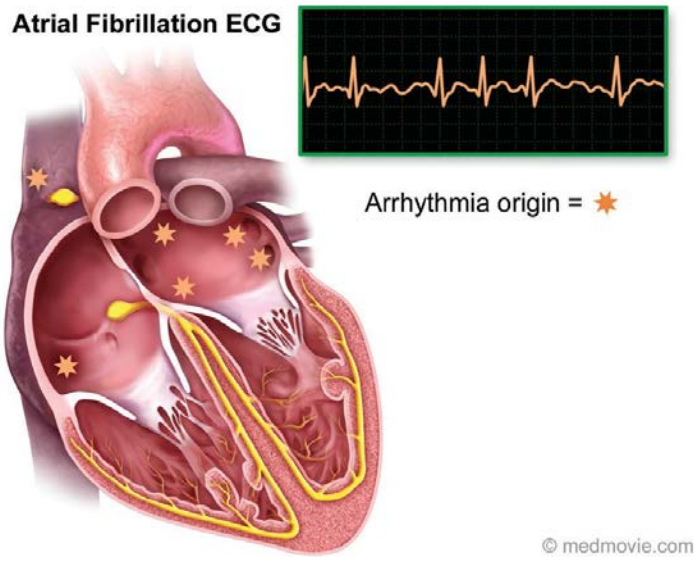
A normal heartbeat begins with an electrical impulse in the sinoatrial (SA) node, a small bundle of tissue located in the right atrium. The impulse sends out an electrical pulse that causes both atria to contract (squeeze) and move blood into the ventricles. The electrical current then passes through a small bundle of tissue called the atrioventricular (AV) node (the electrical bridge between the upper and lower chambers of the heart), which makes the ventricles squeeze (contract) and release in a steady rhythm. As the chambers relax and contract, they draw blood into the heart and push it back out to the rest of the body. This is what causes the pulse we feel on our wrist or neck. The figure below shows the normal

electrical function of the heart. An electrocardiogram, or ECG, is a tool we use to understand what the electrical system is doing at any given time by recording it from electrodes placed on the skin.



B. What is Atrial Fibrillation?

Atrial fibrillation is a disease of the electrical system of the heart. Abnormal heart timing (rhythm), or arrhythmia, is when the heart doesn't beat in a steady or regular pattern. Atrial fibrillation (AF) is one type of arrhythmia. AF occurs when the upper chambers of the heart are driven very rapidly ("fibrillate") by one or more (usually more) "renegade" timers which have awoken. These timers are much faster than the SA node timer, and they send impulses through the AV node to the ventricles, resulting in rapid pumping, which is inefficient and may result in symptoms. The figure below shows this.



C. Types of AF

AF occurs in one of two patterns:

i. Paroxysmal: refers to AF that comes and goes on its own. The AF may last for seconds, minutes, hours, or even several days before the heart returns to its normal rhythm.

ii. Persistent: refers to AF which does not stop by itself.

Although AF may be of either pattern when it is first detected, the classic sequence (if left untreated) is paroxysmal first (initially brief episodes which are few and far between, with gradual march over time - which may be years), eventually progressing to persistent. We do not know why this progression occurs, but it is likely explained in part by the factors which caused the AF to occur in the first place (see section F below).

D. Symptoms of AF

The symptoms caused by AF are varied between people, and may quality and/or severity over time for a given person. Some people with AF can tell immediately, whereas others are less aware. Some people have no symptoms. In such patients, AF is often first discovered accidentally, for example during a routine physical, preoperative testing for a non-heart related procedure, or when they present with a complication related to AF such as stroke (see below). This is why detection of AF is so important. Reasons for the absence of symptoms vary, but it is reasonable to think of the heart as part of a circulation system. Many patients are gifted with “slack” in this system, which can cover for inefficiency in one of its components. Symptoms caused by AF may include:

- Feeling tired or lacking energy.
- Shortness of breath, usually when exerting.
- Experiencing racing, pounding, or fluttering in the chest.
- Difficulty in performing usual activities.
- Experiencing pain, pressure, tightness, or discomfort in the chest.
- Dizziness, lightheadedness, or fainting
- Increased frequency of urination.

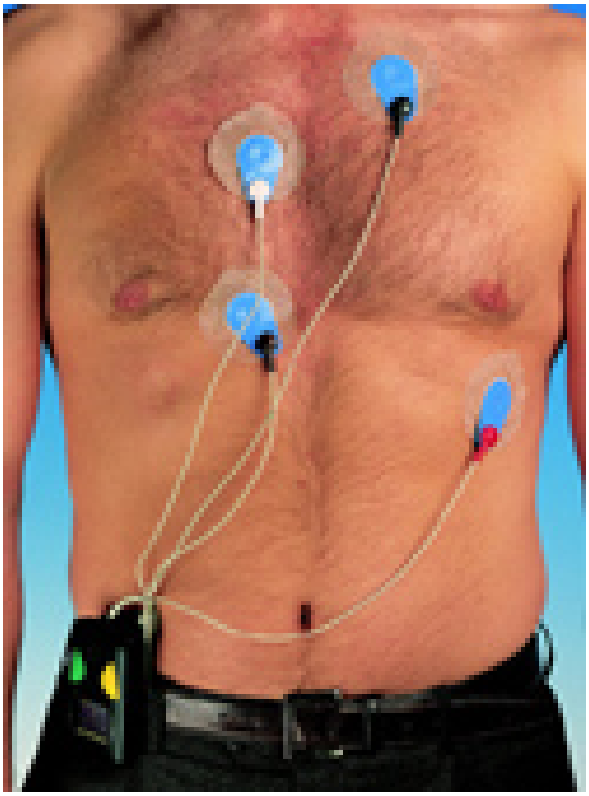
E. How is AF diagnosed?

There are several tests that may be done when someone may have an arrhythmia. Your doctor may order some or all of these tests, depending on the situation:

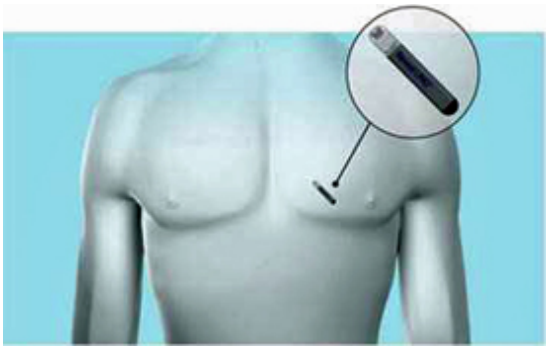
- Electrocardiogram (ECG): An ECG is a snapshot of your heart’s electrical activity. It is often performed in a doctor’s office. Stickers (electrodes) are attached to your chest, arms, and legs. These electrodes measure the rate and rhythm of your heart. An ECG is commonly used to diagnose AF (see figures above).

- Ambulatory electrocardiographic monitor: this can be thought of as a portable ECG machine which you wear for an extended period, typically 1-30 days,

during which your heart rhythm is monitored continuously. Stickers (electrodes) are placed on your chest and are then connected to a small recording machine that is usually worn around the waist (see figure below). This technology may make sense for patients whose AF is intermittent, does not cause symptoms, or in whom the relationship between symptoms and rhythm unclear.



- Implantable electrocardiographic monitoring device (IEMD): on occasion, much longer-term electrocardiographic monitoring may be necessary, for which the ambulatory electrocardiographic monitoring device is not tolerable. IEMDs are small devices (size of a matchstick) that continuously record the heart's rhythm, and are implanted under the skin in the chest area (see figure below). The device transmits your heart rhythm wirelessly to a small tabletop receiving unit in your home. Data are transmitted to a monitoring center that shares the information with your doctor. The battery in the IEMD typically lasts three or more years.



BHS Cardiology
CARDIAC ELECTROPHYSIOLOGY

F. Why do I have AF?

AF is the most common type of arrhythmia. There are approximately 2.3 million people in the United States who have AF, with 160,000 new cases diagnosed every year. As for many diseases, age is a key risk factor of AF, and it is rare before age 40. Genes do play a role, and some patients with AF are aware of parents or siblings with the disease. However, BY FAR THE LARGEST role is played by personal choices which, when taken together, we call "lifestyle." At any age, the following lifestyle elements will make AF more likely to occur (and progress):

1. Eating foods other than whole fruits, whole vegetables, whole grains, nuts, and seeds.
2. Not being fit (not exercising).
3. Inadequate or poor quality sleep.
4. Poor peace of mind.
5. Using tobacco or nicotine products.
6. Drinking alcohol to excess (more than 2 drinks per day)

We will return to lifestyle again below, because it is absolutely crucial that you understand its importance, regardless of which treatments you choose to deal with your AF.

G. What does AF bring to the table?

This is a two-part story, and BOTH are equally important:

PART 1: Implications of the diagnosis. Like many other "chronic" diseases such as high blood pressure, diabetes, sleep apnea, arthritis, erectile dysfunction, and depression, AF is a warning that you have taken a shortcut from the aging path we all walk, which eventually results in frailty and, hopefully soon afterwards, death. The shortcut will have you arrive at frailty at a younger age, and on the way place you at risk for medical catastrophe (eg. heart attack, cancer, dementia). Such catastrophes may be lethal, but are typically not, and when combined with frailty make many affected persons wish for death. The ONLY way to get off this road is to optimize personal choices (see F above). Treatment of AF will NOT get you off this road.

PART 2: The issues surrounding treatment. There are several issues which must be understood:

1. Although studies of patients with AF consistently show that people die sooner than if they did not have AF, we do not believe that it is the AF that kills. Rather, as noted above, we believe that it is the medical catastrophes which AF predicts which do the dirty work. Thus, AF is itself not life threatening.

2. Although AF may occasionally weaken the ventricles, placing one t risk for a problem which doctors unfortunately call “heart failure,” this is not typical.

3. Stroke: it is clear that people with AF have a higher rate of stroke than people without AF. The term “stroke” means damage to the brain caused by a problem with its blood supply, including blocked or burst blood vessels. In association with AF, the problem is blood vessel blockage, often caused by blood clots which are prone to form in the fibrillating atria and then travel from there via the ventricles and to the brain. Strokes caused by AF are typically devastating – either lethal or leaving the victim with a life-changing (often ruining) disability.

4. Diminished life quality: although symptoms caused by AF are the prime determinants of the degree to which it diminishes your quality of life, other factors also apply:

a. Impact of drugs used to treat AF, including risks, side-effects, costs, and inconveniences.

b. Other, more personal, factors which you feel are relevant.

H. What are the goals of treatment of AF?

1. Teaching you that AF is often the “canary in the coal mine,” or a warning of bad things in the years ahead. The ONLY way to decrease the likelihood of such things is by optimizing your personal choices (see section F above). Treating AF will NOT decrease the likelihood of bad things.

2. Minimizing the likelihood of AF causing or contributing to heart failure: as noted above, AF does not typically cause heart failure. When it does, it is because AF is driving the ventricles too quickly and for prolonged periods of time. As will be discussed below, ways to prevent this include slowing the ability of the AV node to conduct the renegade impulses or restoring normal rhythm.

3. Minimizing your risk of stroke: it turns out that several age, gender and medical history items, when taken together, give us an idea of stroke risk associated with AF in patients like you. It is important for you to understand that this measurement does not give us personalized information, just information on average risk in people like you. We utilize a scoring system called “CHADS-VASC,” in which points are assigned for a number of items:

CHA_2DS_2-VASc Risk Criteria	Points
Congestive Heart Failure	1
Hypertension (high blood pressure)	1
Age > 75 Years	2
Diabetes Mellitus	1
Prior stroke or mini stroke ("transient ischemic attack")	2
Peripheral Vascular Disease or Coronary Artery Disease	1
Age 65-74 Years	1
Sex Category (i.e., Female Sex)	1

The higher your point total, the higher your risk of stroke in the setting of AF. Based on this system, your doctor may recommend a "blood thinning" medication, also called an "anticoagulant" medication. These medications are make it harder for your blood to clot, and work to prevent clot formation in the fibrillating atrium (above). There are several different such medicines, including warfarin (Coumadin®), dabigatran (Pradaxa®), apixaban (Eliquis®), and rivaroxaban (Xarelto®). Although also technically blood thinners, aspirin and clopidogrel (Plavix®) are typically inadequate to prevent stroke due to atrial fibrillation.

Although anticoagulants are good at preventing stroke, they increase the risk of excess bleeding, which may be dangerous and, on occasion, life-threatening. Before starting on these drugs you, with information and advice from your doctor(s), must carefully weigh whether the potential benefits of anticoagulants outweigh their potential risks.

You may wonder whether restoring and maintaining normal rhythm will also prevent stroke associated with AF, and the answer is we don't know. Although the decision to take anticoagulants will always be yours alone, for patients at significant risk for stroke we typically advise continuation of anticoagulant drugs even if we think we have been successful in restoring normal rhythm.

For some patients, anticoagulant drugs cannot or should not be used. These are patients who have or are at risk to bleed already. Examples include patients who have experienced significant gastrointestinal bleeding in which the cause cannot be determined or eliminated, patients with prior brain bleeding, or patients who are unsteady on their feet and experience falls. For such patients, another option would be a strategy which specifically targets the region of your heart, called the "left atrial appendage (LAA)." Recall that during AF, blood clots are prone to form in the fibrillating atria. The region where clots typically form is the LAA, a structure which is not needed for normal heart function and we have a number of minimally invasive technologies which can be used to eliminate this structure.

While we are on the topic of stroke, here are the signs or symptoms to be aware of:

- Sudden numbness or weakness of face, arm or leg, especially on one side of the body.
- Sudden confusion, trouble speaking, or understanding.
- Sudden trouble seeing in one or both eyes.
- Sudden trouble walking, dizziness, loss of balance, or coordination.
- Sudden severe headache with no known cause.

If you or a loved one experiences any of these, it is crucial to understand that "time is brain," meaning that the faster that person gets to a hospital, the more likely it is for them to have a good outcome. Typically, this means calling 911.

4. Eliminating or minimizing the negative impact of AF on life quality: this becomes the second key issue (the first being stroke prevention) which should drive your decision making regarding how you wish to have your AF managed. As noted above, we do not have adequate information at this time to be sure that, even if we were to cure your AF, that it would be safe to stop your anticoagulant drug if you are taking one. Thus, in many ways stroke prevention and life quality improvement are separate issues.

Eliminating or minimizing the negative impact of AF on life quality begins with a decision on your part, with input from your doctor(s) who are informed by tests which s/he may perform, as to what aspect(s) of AF cause negative impact. The reason we mention this is that symptoms which may be caused by AF, such as fatigue to shortness of breath, may also be caused by many other things, such that treating the AF will not be of value to them. Conversely, in patients who cannot detect a significant negative impact caused by AF, no treatment may be needed.

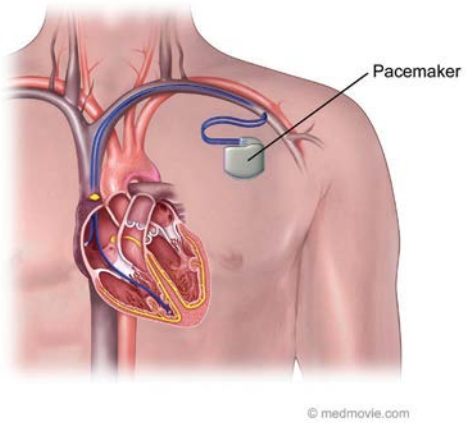
There are two general strategies for eliminating or minimizing the negative impact of AF on life quality:

1. Control the rate at which the heart pump is driven by the renegade timers. This "rate control" strategy attempts to slow or eliminate conduction of renegade timing impluses to the ventricle through the AV node (see figure above). There are 2 ways to do this:

A. Slow AV nodal conduction: in this approach, drugs are used which slow conduction of impulses through the AV node. There are a number of drugs which may achieve this, including beta blockers (example: Toprol) and calcium channel blockers (example: Cardizem). This approach is often limited by tolerability, as these drugs often cause side effects such as fatigue, sluggishness, shortness of breath, and depression.

B. Destroy AV nodal conduction: In this approach, the AV node is destroyed by a procedure called ablation, in which a thin wire is temporarily inserted into the heart via a vein in the groin which is accessed with a needle, placed against the AV node, and heated as to destroy the node. This is a painless, very low risk, and rapid procedure. It leaves the ventricles without any electrical input (good or bad), and so in tandem with this approach an artificial timer, called a pacemaker, is inserted (see figure below). The pacemaker provides slow and

regular beating of your ventricle, akin to your normal timer. It will adjust rate to your level of activity, also akin to your normal timer. Thus, even though the renegade timers may be active in your atrium, you will have no knowledge of it. This approach has the benefit of eliminating drugs used to treat AF. This approach is limited by the fact that you will be rendered deprendent on a pacemaker, which is highly reliable but which in theory may malfunction. If this were to occur the situation could be life-threatening.



2. Restore and maintain normal rhythm: this “rhythm control” strategy aims to suppress or destroy the renegade timers and allow your normal timer to resume take back control. There are several ways to do this:

A. Cardioversion: in this treatment, the shock was delivered to the heart, generally via pads which are placed on the front and back of the chest. You are put to sleep briefly, and a shock was delivered between these pads. The shock puts the renegade timers to sleep. It is worth noting that this procedure is often coupled with another procedure called a transesophageal echocardiogram (TEE). In this procedure, a sound wave recording device is passed into the food pipe connecting mouth and stomach (esophagus). The esophagus passes very close to the heart, and allows much clearer pictures than can be taken from the body surface. Certain features of the heart shown on TEE would predict complications from cardioversion, and thus would cause us to delay or cancel the procedure.

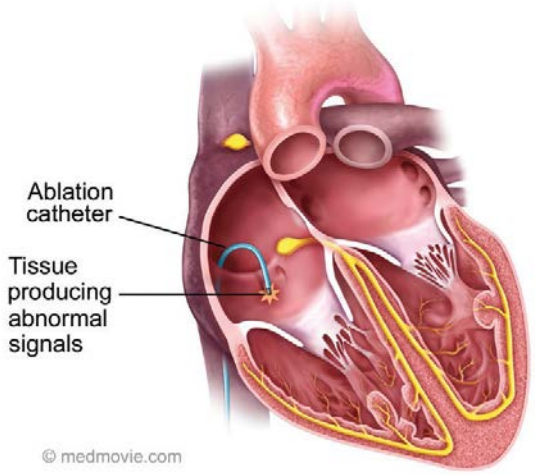
Although cardioversion is almost always successful in restoring normal rhythm, it does nothing to keep the renegade timers asleep, and typically they will wake up again at a later time, which may be anywhere from minutes to many months. Thus, as a generality cardioversion is not used as a stand-alone treatment, rather in association with another treatment such as drug or ablation (below).

B. Drugs: there are a handful of drugs, which we call “antiarrhythmics,” which may suppress renegade timers without disturbing your normal timer. Such drugs may be prescribed on a continuous or as-needed basis. Discovering whether a drug approach will work for you, and if so which drug, is a trial and error process which will often be initiated in

the hospital. We do this because, on occasion, drugs can cause adverse rhythm reactions which can be a threat, and which we will need to be aware of immediately. These reactions are almost always apparent within the first few doses of the drug, and in the hospital your heart rhythm is monitored continuously. This approach is limited by drug inefficacy (does not suppress renegade timers), intolerance (side effects), and/or toxicity (the drug does damage to an organ, which may or may not cause symptoms).

C. Ablation: the term “ablation” means destruction. You have seen the term already, applied to destruction of the AV node as one rate control strategy above. The use of this technique in rhythm control is far more complex, because as opposed to targeting a single discrete region which is always in the same place, ablation with the goal of AF cure must target all renegade timers, of which there may be many and can vary in locations from person to person. There are 2 ways to perform “curative-intent” ablation:

i. Catheter: in this procedure, you are laid on a table and put completely to sleep. Several wires are threaded into your heart via veins, which are accessed in your groin via needles. These wires are touched against the inner walls of your atrium, and extreme heat or cold is passed through them (see figure below). Extreme temperatures destroy the atrial tissue in the region of touch. These regions heal as scar tissue, which has no electrical activity. With luck, the regions which are destroyed are the ones which harbored the renegade timers. This approach is limited by procedural risk (for most people to whom it is offered, approximately 1%) of a serious complication (which may be life-threatening or altering) as well as failure (eg. does not cure you). Recovery time is brief – generally an overnight stay with return to normal function quickly thereafter. In some patients, it may make sense to do the procedure more than once.



ii. Surgical: as you might guess, the more atrial tissue which is ablated, the more likely it is that all renegade timers are killed off. Given their small size, catheters do not have the ability to kill off large amounts of tissue. In the surgical ablation procedure, the chest is opened and the heart stopped temporarily. Given direct access to atrial tissue, the surgeon can kill off large volumes. This correlates with a higher rate of cure than catheter ablation. In experienced hands, surgical ablation, which is commonly called the “maze procedure,” is more successful than catheter ablation. However, it is limited by a greater procedural risk, as well as a longer time to full recovery. For these reasons, the procedure is generally performed in patients who need heart surgery for other reasons or in those who have failed the catheter ablation approach and still seek normal rhythm.