REVIEW

An Overview of Cardiac Ablation

Soquel Rey, BS,[1] Shel Levine, MS, ACSM-CEP[1]

ABSTRACT

Although cardiac ablation is the preferred method of treatment for several supraventricular tachycardias, its efficacy has also been assessed for treatment of more complicated arrhythmias. In this article we review cardiac ablation and other treatment options for supraventricular tachycardia, atrial fibrillation, and ventricular tachycardia. *Journal of Clinical Exercise Physiology.* 2017;6(2):29–35.

Keywords: atrial fibrillation, arrhythmia, exercise

INTRODUCTION

During normal function the conduction system, cells of the cardiac conduction system, transmit action potentials from the sinoatrial node (SA node) throughout the cells of the right and left atria as well as directly to the atrioventricular node (AV node). The AV node delays the action potential to allow for atrial-ventricular synchronization while the atria contract and force the final 1/3 of filling of the ventricles. The AV node releases the action potential to the His-Purkinje conduction cells and spreads throughout the right and left ventricles via the bundle branch/Purkinje fiber system resulting in coordinated contraction of the ventricles (1). Normal sinus rhythm describes the rhythm of the heart in which the action potential is initiated by the SA node and follows the path described earlier resulting in a heart rate between 60–99 beats·min^{-1}. Any change or variation in the normal sequence/timing of the conduction system is described as an arrhythmia (1).

There are many different types of arrhythmias, or abnormal cardiac conduction, with varying levels of severity and persistence. Some can be short lived (i.e., isolated premature contraction) and have a very small effect on the heart's overall rhythm, whereas others may be long lasting/permanent and result in significant reductions in cardiac output (1). Some common types of arrhythmias include conduction disorders within the AV junction, isolated premature contractions, and atrial/ventricle dysrhythmias. The type of arrhythmia can often be diagnosed by analyzing the electrocardiogram (ECG) (1).

The type, duration, symptoms, and risk factors associated with the arrhythmia all contribute to its overall clinical significance, which influences the decision to implement treatment (2). Treatment methods for arrhythmias are also highly variable, ranging from drug therapy to invasive surgery, depending on the type and severity of the arrhythmia. A rapidly evolving form of treatment for some arrhythmias is cardiac ablation (2). The American Heart Association defines cardiac ablation as "a therapeutic method used to destroy a small section of heart tissue causing abnormal electrical activity or irregular heartbeat" (3). The ablation technique is a 2-step process. First, the site of abnormal activity must be identified using electrode catheters inserted through the femoral or brachial arteries and positioned inside the heart (i.e., cardiac catheterization). This step is referred to as cardiac mapping. Second, the abnormal tissue is destroyed via radiofrequency (RF) ablation, a form of heat energy, or cryoablation, extremely cold temperatures, delivered through a catheter to the site of abnormal tissue (4). This article will discuss cardiac ablation treatment techniques for various types of supraventricular tachycardia (SVT), atrial fibrillation (AFib), and ventricular tachycardia (VT) and compare its treatment efficacy to pharmaceutical options.

History of Cardiac Ablation

The first cardiac ablation was performed on humans in 1981 using a high-energy direct-current shock to destroy the abnormal tissue (5). The direct-current shock created a high-voltage form of energy that lacked both precision and control (4,6). Radiofrequency cardiac ablation was developed shortly thereafter. Radiofrequency cardiac ablation uses a

[1]Eastern Michigan University, Ypsilanti

Address for correspondence: Shel Levine, MS, ACSM-CEP, Eastern Michigan University, School of Health Promotion & Human Performance, Ypsilanti, MI 48197; (734) 487 2818; e-mail: slevine@emich.edu

Conflicts of interest: None

low-voltage form of energy, thus enhancing the safety and applicability of the cardiac ablation technique (4,6). Cryoablation, the most recently developed, utilizes a rapid-cooling catheter tip that destroys the abnormal tissue by delivering temperatures from –60ºC to –80ºC to the target area (7). Both RF cardiac ablation and cryoablation are commonly used today; however, other methods that utilize acoustic and laser technology to destroy the abnormal tissue are currently under investigation (4,6,7).

Cardiac Ablation Procedure

For the ablative therapy to be successful, the arrhythmia must be correctly diagnosed and precisely mapped. When possible, arrhythmias can be correctly diagnosed through 12-lead, event, and Holter ECG monitoring; however, ECG monitoring cannot always capture short-term, nonpersistent arrhythmias. In this case, diagnostic electrophysiological studies (EPSs) are necessary to ensure accurate diagnosis (4). During diagnostic EPSs, catheters (typically entering via the femoral vein) are positioned around the heart to electrically stimulate and map the arrhythmia so that it can be recorded and diagnosed. Furthermore, diagnostic EPSs can provide conduction information detailing SA nodal, AV nodal, and His-Purkinje functioning. Once diagnosed, the origin of the arrhythmia is mapped to ensure proper catheter positioning during the ablative procedure (4).

Mapping allows the location of origin of an arrhythmia to be identified by providing real-time 3-dimensional images of the cardiac anatomy in addition to catheter positioning and electrophysiological information about the tissue (4). In some cases, magnetic resonance imaging, computed tomography, and echocardiographic images are also required to assist with catheter placement and mapping. After the catheters have been properly placed, the cells causing the arrhythmia can be destroyed (4).

Cardiac Ablation: Risks and Recovery

According to Bashore et al. (8), cardiac ablation has proven both safe and effective with a rate of procedural complications ranging from 1% to 5% and a mortality rate of 0.6% (9). The most common risks associated with the procedure are related to the catheterization process leading to damage of the vascular system with occurrences at a rate of 2%. Although low risk, other possible complications include pericardial tamponade, cardiac perforation, AV nodal damage, heart valve damage, coronary artery damage, and systemic embolization (4,8).

After surgery, the ablative procedure results in varying lengths of recovery from days to months; however, in most cases, the patient can return to previous activity levels within 1–2 weeks (10). Several studies analyzed the long-term effects of the cardiac ablation and found that the ablative therapy yields a higher quality of life and exercise capacity compared with traditional drug therapy (11–14). Brignole et al. (11) performed a 12-month evaluation comparing symptoms, exercise tolerance, and activity level of ablation-treated patients and pharmacologically treated patients. They found that the patients who underwent ablative therapy reported significant reductions in several symptoms, including palpitations, dyspnea, exercise intolerance, fatigue, and chest discomfort. However, there was no significant difference in cardiac performance, evaluated by ejection fraction and exercise duration during an exercise echocardiogram, between the groups (11). Jaïs et al. (12) also found that ablative therapy yielded higher quality of life and exercise scores during stress testing by comparing maximum workload, peak metabolic equivalents of task, and maximum heart rate in ablation-treated patients compared with patients who were treated pharmacologically (12). Brignole et al, (14) analyzed cardiac performance by comparing the duration of a standardized stress test in 22 patients before and after AV junction RF ablative surgery. Results indicated increased stress testing duration and, thus, improved cardiac performance (14). Moreover, Furlanello et al. (15) compared maximal exercise capacity of 20 competitive athletes with disabling, symptomatic AFib before and after catheter ablation. After ablation all athletes successfully passed a preparticipation evaluation. including an exercise test, holter monitoring, and an echocardiogram. and were able to return to the same level of training and competition as before the occurrence of AFib episodes (15).

Cardiac ablation has become the primary form of treatment for several SVTs (Table 1), including AV nodal reentrant tachycardia (AVNRT), atrioventricular reentrant tachycardia, focal atria tachycardia, and atrial flutter. Ablative techniques are also being used and tested for their efficacy in treating more complicated arrhythmias, such as AFib and VT (8).

Overview of Supraventricular Tachycardia

In general, SVT describes several arrhythmias in which premature atrial depolarizations arise from an ectopic stimulus located in the atria or AV nodal tissue. Most forms of SVT are triggered by an electrophysiological mechanism known as reentry. In reentry, a propagating impulse continually restimulates the atria, creating a reentrant circuit. The exact reentrant path, severity, symptom manifestation, and treatment strategy are widely variable depending on the specific type of arrhythmia (16).

As stated earlier, SVTs are often caused by the maintenance of rapidly firing ectopic cells, which can result in single or multiple reentrant circuits, thereby inducing the arrhythmia. The normal electrical conduction system often has more than one pathway to and from the same location; this serves as a preventive measure by providing a means for conduction to proceed in the case of damage to one pathway (16). Although this duel pathway structure provides an important backup function for the conduction system, it can also serve as an outlet to create dangerous reentrant circuits under certain circumstances. Such a circumstance is depicted in Figure 1. This specific example depicts the dual pathway system located in the AV nodal tissue, resulting in a reentrant circuit leading to a specific form of SVT: AVNRT. As seen in AVNRT, a slow pathway and a fast pathway connect the atria

TABLE 1. Commonly ablated arrhythmias.

Arrhythmia	Triggers	Success Rate	Complication Risk
AV nodal reentrant tachycardia (4,16)	Premature atrial contraction triggers reentrant circuit within AV nodal tissue	>95%	1%–5%
Atrioventricular reentrant tachycardia (4,16)	Conduction through accessory pathway bypassing AV node triggers reentrant circuit	~95%	1%–2%
Focal atrial tachycardia (4,16)	Abnormal automaticity, externally triggered activity, or reentry within atrial musculature	>80%	1%–2%
Atrial flutter (4,16)	Premature atrial contraction triggers large reentrant circuit in right atrium	~95%	1%–5%

AV = atrioventricular.

to the ventricles through the AV node (16). The reentrant circuit is triggered by a premature atrial contraction. When the premature stimulus reaches the AV node (left image, Figure 1), the slow pathway may be ready to conduct but the fast pathway may still be in recovery period from the previous contraction and unable to conduct. If this occurs, the slow pathway will conduct the stimulus like normal in an antegrade fashion, during which time the fast pathway is still recovering (16). By the time the conduction makes it through the slow pathway to the ventricles, the fast pathway may be fully recovered and carry on the conduction in a retrograde fashion, from ventricles to atria (middle image, Figure 1). By the time the conduction makes it through the fast pathway to the atria, the slow pathway may be fully recovered and the conduction can reenter the slow pathway, creating the tachycardic reentrant circuit (right image, Figure 1) (16).

Treatment of SVTs requires interruption of the reentrant circuit. Current available treatments that aim to end the tachycardic episode include vagal maneuvers, drug therapy, and cardioversion. Because of their low risk and immediate availability, vagal maneuvers are usually the first treatment method attempted. The goal is to increase vagal tone, thereby ending the tachycardic episode. The Valsalva maneuver and carotid sinus massage are commonly used examples of vagal maneuvers (8).

If vagal maneuvers are unsuccessful, drug therapy is the next mode of treatment. The medication chosen (adenosine, a calcium channel blocker, or a beta-adrenergic receptor blocker) for treatment is highly dependent on the patient's medical history and symptomology. Although vagal maneuvers may be successful in ending the tachycardic episode, only drug therapy and ablation offer preventive or permanent measures for recurrent SVT (8).

Due to the side effects associated with long-term drug therapy, cardiac ablation has been established as the preferred method of treatment for many SVTs (4). Oftentimes,

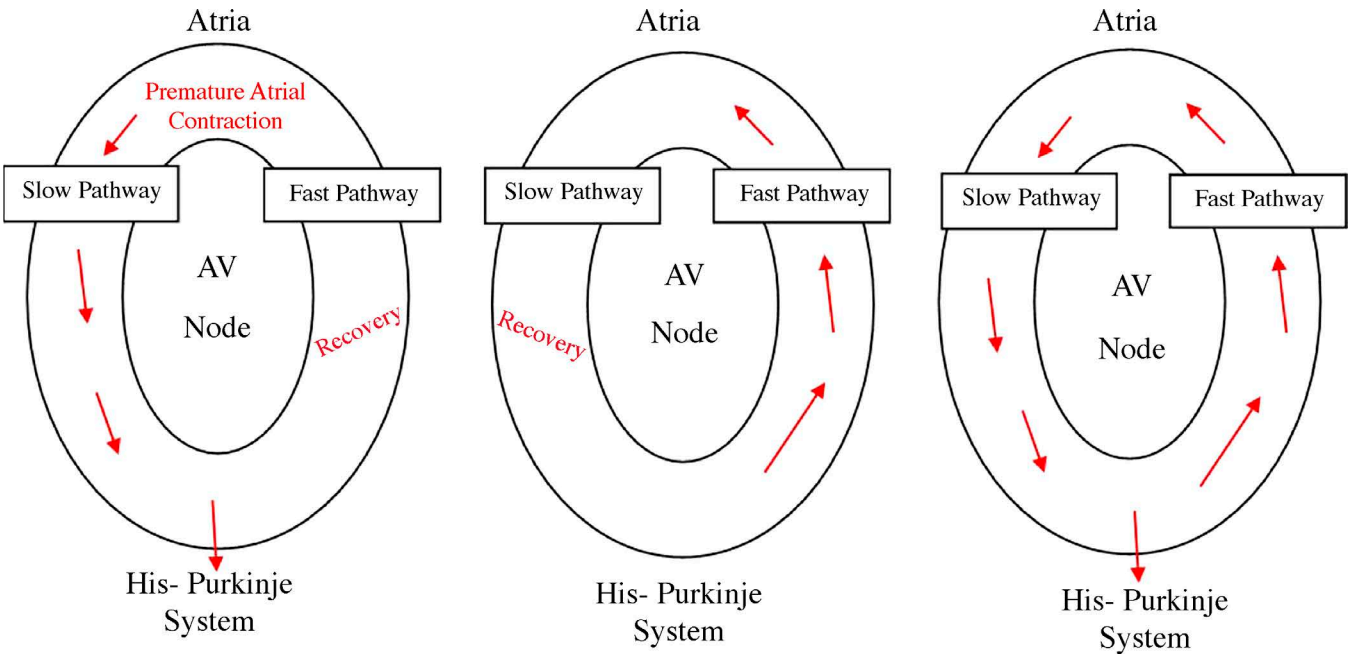

FIGURE 1. Illustration of AV nodal reentrant tachycardia. The image on the left represents the premature contractile trigger, with only the slow pathway ready to pass on the antegrade conduction. The middle image represents retrograde conduction through the fast pathway. The image on the right represents the full tachycardic reentrant circuit.

SVTs have a specific location of reentry. In describing AVNRT earlier (Figure 1), the slow conduction pathway is the location of reentry. During ablation, the catheter is positioned to ablate this location and avoid damaging uninvolved conduction tissue, thereby maintaining normal conduction through the AV node (4).

Overview of Atrial Fibrillation

The most common chronic arrhythmia, AFib, affects 2.2 million to 5 million Americans and has a prevalence of 0.4% in the general population and as high as 8.8% in the elderly (older than 80 years old) (17). Although AFib can develop in an otherwise healthy heart, "it is commonly associated with rheumatic heart disease, hypertension, ischemic heart disease, pericarditis, thyrotoxicosis, alcohol intoxication, disorders of the mitral valve, and digitalis toxicity" (16). Though AFib alone is rarely fatal, because the atrial hyperactivity results in an inability to effectively pump blood, patients with AFib are at a higher risk of embolism formation leading to a stroke (8,18). Other possible consequences of AFib include hypotension, increased dementia risk, decreased cardiac output, hemodynamic collapse, myocardial ischemia, and tachycardia-induced myocardial dysfunction (8,18).

Ablative therapy has become the preferred method of treatment for several arrhythmias, but its uses are still being evaluated for more complicated arrhythmias, such as AFib (17). In AFib, the atria are contracting at an irregular rate of 300 to 600 beats·min^{-1}. Thus, the baseline on the ECG appears erratic, there are no visible P-waves, and the R-to-R interval is irregular (17).

Despite some discrepancy, cases of AFib are usually classified into 1 of 3 broad categories: paroxysmal, persistent, or permanent (17). Paroxysmal AFib describes cases where AFib episodes abruptly start and stop and last for less than 7 days, though most cases end within 24 hours. Paroxysmal AFib is typically caused by rapidly firing ectopic cells or reentrant circuits located in the atrial tissue or the roots of the pulmonary veins. Risk factors associated with the spontaneous ectopy and reentrant circuits leading to AFib include hypertension, congestive heart failure, and coronary artery disease. High alcohol consumption or alcohol withdrawal, are additional triggers known to initiate paroxysmal AFib (17). Furthermore, several studies (19–21) have found a correlation between increased physical activity and risk of developing AFib possibly due to increased left atrial size and volume in active people.

In cases where AFib lasts for longer than 7 days, it is categorized as persistent and requires electrical or pharmacologic cardioversion (17). When AFib persists for more than a year despite treatment attempts, it is categorized as permanent AFib. The longevity of persistent and permanent AFib is most often due to single or multiple reentry circuits, as previously discussed, in addition to changes in the structure and anatomy of the atria to further support reentry and sporadic, random contraction. Loss of myofibrils, the contractile component of cardiac muscle, buildup of glycogen granules, and lack of cell-to-cell communication/regulation at gap junctions are all common atrial structural changes opposing return to sinus rhythm (17). The initial strategy for AFib treatment requires management of symptoms and lowering the risk of stroke or further complications associated with the arrhythmia. The next step involves choosing a permanent rate or rhythm control strategy. Most strategies that are intended to control cardiac rate or rhythm are also effective in terminating AFib. Anticoagulants are generally required for managing stroke risk. Despite the positive effects of anticoagulation therapy, long-term usage increases risk of hemorrhage and bleeding (17).

As stated earlier, a rate or rhythm control strategy is required for long-term AFib management. Several studies have shown no difference in success or mortality rates when comparing rate versus rhythm control strategies; therefore, the method of treatment is determined on a case-by-case basis (17). The goal of rate control is to decrease the ventricular rate to less than 100 beats·min^{-1}, thus increasing cardiac output and alleviating symptoms. Rate control strategies exist in 2 stages: acute and chronic. The acute stage involves pharmacologic therapy, whereas the chronic stage involves ablation of the AV node resulting in complete heart block followed by pacemaker implantation (17). Rhythm control strategies aim to maintain sinus rhythm. This can be achieved through pharmacologic cardioversion, electrical (via direct current) cardioversion, or cardiac ablation. Each method has its own risks and benefits (Table 2). The method of therapy is chosen based on symptoms, risks, patient profile, and needs of the individual (17). Typically, the method of therapy can be individualized to each patient by (1) history of dealing with AFib or VT, (2) past response to medications, (3) level of symptomology, (4) level of recurrence, and (5) presence of other heart complications/disease.

Ablative techniques aim to destroy the foci causing the rapid, erratic contractions; typical targets include both electrical triggers, such as pulmonary vein foci and atrial muscular foci, and atrial substrate triggers, such as fibrosis, hypertrophy, mutations of ion or gap junctions, and electromechanical remodeling (22). As stated earlier, the pulmonary veins have been identified as a common location for abnormal automaticity triggering AFib making them a primary target for ablative therapy. Furthermore, the junction of the pulmonary vein and left atrium has been identified as a common location of reentry providing a system to maintain the arrhythmia. The autonomic tone of the ganglionated plexi that innervate the pulmonary veins and left atrium is another source of AFib genesis (23). Not all cases of AFib require the same ablative technique; ablation producing pulmonary vein isolation may be a suitable form of treatment for paroxysmal AFib, whereas permanent AFib may require extensive ablation techniques (23).

Ablation has been most successful in treating paroxysmal AFib with success rates as high as 70% to 80% in patients without other structural heart complications (4). The most common ablative techniques for treating paroxysmal AFib include pulmonary vein isolation and box isolation

TABLE 2. Risks and benefits of atrial fibrillation (AFib) management.

	Risks	Benefits
Pharmacologic cardioversion (17)	• Drug toxicity (~20% long-term users) • May be unsuccessful in AFib termination	• May reduce frequency, duration, and symptoms • 30%–50% efficacy
Electrical cardioversion (17)	• May induce ventricular fibrillation • AFib recurrences	• Immediate termination of AFib episode
Cardiac ablation (17)	• 2%–5% complication rate ○ Pericardial tamponade ○ Phrenic nerve injury ○ Stroke ○ Pulmonary vein stenosis	• 50%–60% effective after 1 treatment • 70%–80% effective overall

(23). The effectiveness of ablation therapy was compared to drug therapy in treating paroxysmal AFib in a meta-analysis of 5 studies (24). Of the patients treated with catheter ablation, 77% did not experience any episodes of paroxysmal AFib during the first year following the procedure with 17% requiring multiple ablative procedures and 2.6% enduring major complications. In comparison, only 29% of the patients who received drug therapy did not experience any episodes of paroxysmal AFib after the 1-year period. Another meta-analysis found similar results (25). The ablative procedure was successful in 57% of patients who received 1 treatment, 71% of patients who received multiple treatments, and 77% of patients who received multiple treatments in combination with drug therapy. The success rate of drug therapy alone was lower than all the success rate for all 3 ablative therapy groups at 52%. Of the ablation patients, 5% had major complications compared with 30% of the drug therapy patients; however, the complications due to drug therapy were much less severe than the complications due to ablative therapy (25).

Cardiac ablation has been less successful in treating persistent and permanent cases of AFib, but new techniques and strategies are still being developed. To treat persistent AFib, more extensive ablation techniques are required. These techniques ablate specific areas of atrial tissue identified as a key location for vagal innervation, slow conduction, reentry, and AFib maintenance. One study demonstrated ablation to be successful in 93% of paroxysmal AFib cases, 87% for persistent AFib, and 78% for permanent AFib; however, a similar study had much less success: only 12% of patients receiving ablation returned to sinus rhythm. Due to the difficulty in identifying the mechanism of AFib, more study and testing needs to be done affirming the success of this ablative technique (23). Overall, many studies have demonstrated the superiority of catheter ablation over drug therapy for terminating AFib; however, data are still lacking on the overall mortality and morbidity rates comparing the 2 different forms of treatment.

Overview of Ventricular Tachycardia

Cardiac ablation is also used to treat VT, which is rapid arrhythmia defined as 3 or more premature, consecutive beats arising in the ventricles as manifested on the ECG with widened QRS complexes often lacking a preceding P-wave. As a result, the ventricular rate typically rises to 160 to 240 beats·min^{-1}. The typical mechanism for consecutive reoccurrence of premature ventricular complexes leading to VT is ventricular reentry and automaticity (26,27).

There are two common ways to classify VT. The first is based on duration; nonsustained VT describes a case in which the VT episode lasts less than 30 seconds. whereas sustained VT episodes last longer than 30 seconds (26). Sustained VT requires intervention for termination (26,27). The second way VT is classified is according to ECG morphology. In monomorphic VT, each QRS complex has the same appearance because each ventricular contraction arises from the same focus and thus is conducted through the same pathway. In contrast, in polymorphic VT (traditionally known as torsades de pointes) several ventricular foci initiate the conduction and is distinguished by beat-to-beat variation in the QRS morphology (26,27).

Furthermore, VT may present as asymptomatic or may result in several of the following symptoms: palpitations, exercise intolerance, dizziness, lightheadedness, syncope, or sudden death. In some cases, symptom evaluation can influence the treatment strategy by providing information about the severity of VT and risk of sudden death (26). Another important distinction for VT treatment is whether or not the arrhythmia is idiopathic or occurs in conjunction with structural heart disease. In 15% to 20% of cases, VT is associated with structural heart disease (30). The most common disease associated with VT is chronic ischemic heart disease; however, cardiomyopathy, previous myocardial infarctions, and congenital heart disease are associated with increased risk for VT as well (26,27).

The typical cause of VT in the presence of a structural heart disease is reentry through areas of myocardial scarring. Treatment strategies include pharmacologic therapy, implantable cardioverter defibrillator (ICD), and ablation. The mortality rate for VT with associated heart disease is much greater than that for idiopathic VT; as such, an ICD or ablative procedure is often necessary. For patients at increased risk for sustained VT in the presence of structural heart disease, ICD is the standard treatment method (4,30). Although an ICD can be successful at ending the VT episode, the defibrillator shocks have been associated with a higher risk of death and hospitalization. Ablative therapy can be used in conjunction with an ICD to decrease the

frequency of shocking. Ablative therapy for monomorphic VT with associated structural disease and scarring has a success rate of approximately 70% with a 3% mortality rate. Complications of the procedure include stroke, femoral hematomas, and heart block. Furthermore, VT recurrence following ablation occurs in approximately half of all patients, (4,30).

In addition, VT can occur in the absence of a structural heart disease (i.e., idiopathic VT). Idiopathic VT can present as monomorphic or polymorphic and is often caused by a discrete focal point or reentry circuits identifiable thorough EPS (4). Idiopathic VT can be terminated with carotid massage, antiarrhythmic drugs, or cardioversion. Treatment for idiopathic VT may utilize 1 of 3 strategies, depending on the severity, duration, and symptom manifestation of the arrhythmia: preventive strategies (i.e., antiarrhythmic drugs), acute treatment strategies (i.e., carotid massage, cardioversion), and long-term treatment strategies (i.e., ablation, ICD) (4). Idiopathic focal VT often originates near the right ventricle outflow tract; in contrast, idiopathic reentrant VT often originates from reentry in the fascicles of the left ventricle. In either case, ablation targets the area with the earliest ventricular activation. Ablative therapy for treatment of monomorphic, idiopathic VT has a success rate greater than 80% (4). However, ablation for polymorphic, idiopathic VT has been much less successful and in most cases, antiarrhythmic drugs are the preferred therapy (4).

Summary

Cardiac ablation is a rapidly developing form of therapy that has already become an effective treatment for many tachycardic arrhythmias. Ablative therapy has shown superiority over pharmaceutical therapy in many cases of SVT. Despite its proven success, its necessity is usually assessed on a case-by-case basis to ensure that every patient receives the safest and most effective treatment method.

Ablation has also become an option for AFib and VT. Ablation has shown promise and is quickly becoming a primary treatment of AFib, although data on long-term efficacy and effectiveness are lacking. Ablation has become a common long-term therapy for idiopathic monomorphic VT treatment. An ICD or pharmacologic therapy is currently more common for polymorphic VT and VT occurring in the presence of identifiable heart disease.

REFERENCES

1. American Heart Association website [Internet]. Dallas (TX). About Arrhythmia; [updated 2014 Oct 23; cited 2016 February]. Available from http://www.heart.org/HEARTORG/Conditions/Arrhythmia/AboutArrhythmia/About-Arrhythmia_UCM_002010_Article.jsp#.VrIFnvkrJn4
2. American Heart Association website [Internet]. Dallas (TX). Prevention and Treatment of Arrhythmia; [updated 2016 Oct 27; cited 2016 December]. Available from http://www.heart.org/HEARTORG/Conditions/Arrhythmia/PreventionTreatmentofArrhythmia/Prevention-Treatment-of-Arrhythmia_UCM_002026_Article.jsp#.WEMK-_krI2x
3. American Heart Association website [Internet]. Dallas (TX). Heart and Stroke Encyclopedia online; n.d. [cited 2016 February]. Available from http://www.heart.org/HEARTORG/Encyclopedia/Heart-Encyclopedia_UCM_445084_Encyclopedia.jsp?levelSelected=13&title=cardiac%20ablation
4. Tedrow UB, Asirvatham SJ, Stevenson WG. Electrophysiology and Catheter-Ablative Techniques. In: Fuster V, Walsh RA, Harrington RA. Hurst's The Heart, 13e [Internet]. 2011 [cited 2016 February]. Available from: http://accessmedicine.mhmedical.com/content.aspx?bookid=376&Sectionid=40279773.
5. Scheinman MM, Morady F, Hess DS, Gonzalez R. Catheter induced ablation of the atrioventricular function to control refractory supraventricular arrhythmia. In: JAMA [Internet]. 1982 [cited on 2017 February];248:851.
6. Hassett J, Swartz J, Bednarek M. Process and device for the treatment of atrial arrhythmia [Internet]. 2001 [cited 2016 February]. Available from https://www-google-com.ezproxy.emich.edu/patents/US6251109.
7. Andrade J, Khairy P, Dubuc M. Catheter cryoablation. In: Circulation: Arrhythmia and Electrophysiology [Internet]. 2013 [cited on 2017 February];6:218-7. doi: 10.1161/CIRCEP.112.973651.
8. Bashore TM, Granger CB, Jackson K, Patel MR. Heart Disease. In: Papadakis MA, McPhee SJ, Rabow MW. Current Medical Diagnosis & Treatment [Internet]. 2016 [cited 2016 February]. Available from: http://accessmedicine.mhmedical.com/content.aspx?bookid=1585&Sectionid=96304476.
9. Curley M, Berger M, Roth, J, et al. Predictors of mortality and major in-hospital adverse events associated with electrophysiology catheter ablation. In: JAMA Intern Med [Internet]. 2014 [cited on 2017 February]. doi:10.1001/jamainternmed.2014.415.
10. Cleveland Clinic. After Catheter Ablation. [Internet]. N.d. [cited 2016 July 13].Available from http://my.clevelandclinic.org/services/heart/home-recovery/arrhythmias-procedures-devices/after-catheter-ablation.
11. Brignole M, Menozzi C, Gianfranchi L, Musso G, Mureddu R, Bottoni N, Lolli G. Assessment of Atrioventricular Junction Ablation and VVIR Pacemaker Versus Pharmacological treatment in patients with heart failure and chronic atrial fibrillation. Circulation [Internet]. 1998 [cited 2016 July 10];98:953-60. Available from: 10.1161/01.CIR.98.10.953.
12. Jaïs P, Cauchemez B, Macle L, Daoud E, Khairy P, Subbiah R. Catheter ablation versus antiarrhythmic drugs for atrial fibrillation. Circulation [Internet]. 2008 [cited 2016 July 10];118:2498-2505. Available from: 10.1161/CIRCULATIONAHA. 108.772582.
13. Natale A, Zimerman L, Tomassoni G, Newby K, Leonelli F, Fanelli R, Beheiry S, Pisano E. AV Node Ablation and pacemaker implantation after withdrawal of effective rate-xontrol medications for chronic atrial fibrillation: effect on quality of life and exercise performance. pacing and clinical electrophysiology [Internet]. 1999 [cited 2016 July 10];22:1634-9.
14. Brignole M, Gianfranchi L, Menozzi C, Bottoni N, Bollini R, Lolli G, … Gaggioli G, Influence of atrioventricular junction radiofrequency ablation in patients with chronic atrial fibrillation and flutter on quality of life and cardiac performance. Am J Cardiol [Internet]. 1994 [cited 2016 July

10];74:242-6. Available from: http://dx.doi.org/10.1016/0002-9149(94)90364-6.
15. Furlanello F, Lupo P, Pittalis M, Foresti S, Vitali-Serdoz L, Francia P, De Ambroggi G, Ferrero P, Nardi S, Inama G, De Ambroggi L, Cappato R. Radiofrequency catheter ablation of atrial fibrillation in athletes referred for disabling symptoms preventing usual training schedule and sport competition. J Cardiovasc Electrophysiol [Internet]. 2008 [cited on 2017 February];19(5):457-62. doi: 10.1111/j.1540-8167.2007.01077.x.
16. Gugneja M, Kraft P. Paroxysmal supraventricular tachycardia. Medscape [Internet]. 2015 [cited 2016 February]. Available from http://emedicine.medscape.com/article/156670-overview#a4.
17. Prystowsky EN, Padanilam BJ, Waldo AL. Atrial fibrillation, atrial flutter, and atrial tachycardia. In: Fuster V, Walsh RA, Harrington RA. Hurst's The Heart, 13e [Internet]. 2011 [cited 2016 February]. Available from http://accessmedicine.mhmedical.com/content.aspx?bookid=376&Sectionid=40279769.
18. Michaud GF, Stevenson WG. Supraventricular tachyarrhythmias. In: Kasper D, Fauci A, Hauser S, Longo D, Jameson J, Loscalzo J. Harrison's Principles of Internal Medicine 19e [Internet]. 2015 [cited 2016 February]. Available from: http://accessmedicine.mhmedical.com/content.aspx?bookid=1130&Sectionid=79742251.
19. Molina L, Mont L, Marrugat J, Berruezo A, Brugada J, Bruguera J, Rebato C, Elosua R. Long-term endurance sport practice increases the incidence of lone atrial fibrillation in men: a follow-up study. Europace [Internet]. 2008 [cited 2016 December];10(5):618-23. Available from: http://dx.doi.org/10.1093/europace/eun071.
20. Andersen K, Farahmand B, Ahlbom A, Held C, Ljunghall S, Michaëlsson K, Sundström J. Risk of arrhythmias in 52755 long-distance cross-country skiers: a cohort study. Eur Heart J [Internet]. 2013 [cited 2016 December];34(47):3624-31. Available from: http://dx.doi.org/10.1093/eurheartj/eht188.
21. Myrstad M, Løchen ML, Graff-Iversen S, Gulsvik AK, Thelle DS, Stigum H, Ranhoff A. Increased risk of atrial fibrillation among elderly Norwegian men with a history of long-term endurance sport practice. Scand J Med Sci Sports [Internet]. 2014 [cited 2016 December];24(4):e238-44. Available from: 10.1111/sms.12150
22. Lubitz S, Fischer A, Fuster V. Catheter ablation for atrial fibrillation. Br Med J [Internet]. 2008 [cited 2016 February];336:819-26. Available from: http://dx.doi.org/10.1136/bmj.39513.555150.BE.
23. Kumagai K. Catheter ablation of atrial fibrillation. Circulation [Internet]. 2011 [cited 2016 February];*75*:2305-11. Available from:10.1253/circj.CJ-11-0658.
24. Piccini J, Lopes R, Kong M, Hasselblad V, Jackson K, Al-Khatib S. Pulmonary vein isolation for the maintenance of sinus rhythm in patients with atrial fibrillation. Circ Arrhythm Electrophysi [Internet]. 2009 [cited 2016 March];2:626-33. Available from: 10.1161/CIRCEP.109.856633.
25. Calkins H, Reynolds M, Spector P, Sondhi M, Xu Y, Martin A, Williams C, Sledge I. Treatment of atrial fibrillation with antiarrhythmic drugs or radiofrequency ablation. Circ Arrhythm Electrophysiol [Internet]. 2009 [cited 2016 March];2:349-61. doi: 10.1161/CIRCEP.108.824789.
26. John RM, Stevenson WG. Ventricular Arrhythmias. In: Kasper D, Fauci A, Hauser S, Longo D, Jameson J, Loscalzo J, Harrison's Principles of Internal Medicine 19e [Internet]. 2015 [cited on 2016 March]. Available from: http://accessmedicine.mhmedical.com/content.aspx?bookid=1130&Sectionid=79742328.
27. Badhwar N. Chapter 13. Ventricular Tachycardia. In: Crawford MH. Current Diagnosis & Treatment: Cardiology, 4e [Internet]. 2013 [cited on 2016 March]. Available from: http://accessmedicine.mhmedical.com/content.aspx?bookid=715&Sectionid=48214545.