NASPE POSITION STATEMENT

# NASPE Policy Statement on Catheter Ablation: Personnel, Policy, Procedures, and Therapeutic Recommendations

MELVIN SCHEINMAN,* HUGH CALKINS,† PAUL GILLETTE,‡ RICHARD KLEIN,§ BRUCE B. LERMAN,‖ FRED MORADY,# SANJEEV SAKSENA,** and ALBERT WALDO††

From the *University of California-San Francisco, San Francisco, California, †Johns Hopkins Hospital, Baltimore, Maryland, ‡Cook Children's Medical Center, Fort Worth, Texas, University of Utah Medical Center, Salt Lake City, Utah, ‖The New York Hospital, Cornell Medical Center, New York, New York, #University of Michigan Hospital, Ann Arbor, Michigan, **Cardiac Medicine & EP, Warren, New Jersey, ††University Hospitals of Cleveland, Cleveland, Ohio

## Catheter Ablation Policy Statement

The initial NASPE policy statement on catheter ablation was published in May 1992 and was largely related to use of DC ablation. Radiofrequency catheter ablative procedures are currently accepted as primary therapy for most patients with supraventricular tachycardia and for several forms of ventricular tachycardia. The purpose of this report is to update the rationale and indications for catheter ablative procedures. We also provide recommendations regarding electrophysiological laboratory personnel and facilities. Emphasis is placed on procedures that have been extensively used throughout the world and for which there is adequate follow-up information. A detailed review of the mechanism of arrhythmia was felt to be outside the mandate of this committee. Where possible, data are culled from large, prospectively designed trials, where available, as well as evidence from numerous clinical trials.[1–6] The recommendations for use of catheter ablation are formatted in the style used in ACC/AHA documents.

## Definition of Terms and Procedures

Catheter ablation refers to the intentional destruction of arrhythmogenic myocardial tissue, atrioventricular connections or parts of the specialized conduction system in order to cure or control cardiac arrhythmias. These procedures currently almost exclusively use radiofrequency energy and the mode of tissue destruction involves membrane disruption by delivery of thermal energy. These procedures are performed in specialized laboratories, using fluoroscopy and equipment to record surface and intracardiac electrograms and pace the heart.

Address for reprints: Stephanie Mascetta, NASPE, 6 Strathmore Rd., Natick, MA 01760-2499. Fax: (508) 647-0124; e-mail: smascetta@naspe.org

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## Training and Roles of Nonphysician Personnel

Personnel in the procedure room should include a minimum of 2 support personnel (nurses, technicians, or engineers). One nurse, physician assistant, or anesthesiologist is required to administer sedation and carefully monitor vital signs throughout the procedure. The precise responsibilities of the staff who assist with these procedures may vary depending on the particular hospital situation. In most hospitals, the staff is responsible for preparing the patient for the procedure, operating the fluoroscopy system, the radiofrequency generator, and the computerized ECG electrogram recording and mapping (as required) equipment. Despite the absence of clinical studies in this area, it is our recommendation that the staff assisting with catheter ablation procedures have been involved with at least 30 catheter ablation procedures before working independently in the electrophysiology laboratory and that they continue to be involved with at least 30 catheter ablation procedures per year. It is optimal to have support personnel who are dedicated to electrophysiological studies. There is no consensus relative to the specifics of EP catheter nurse performance. Some centers have instituted annual tests to assess knowledge about arrhythmias and resuscitation together with testing of skills relating to procedures and equipment.

It is felt that all complications should be logged, and that physicians and lab personnel meet every 3–4 months to discuss complications and methods of improved practice as mandated by the Joint Commission on Hospital Accreditation Guidelines.

The required skills and training of personnel may vary depending on the number and experience of physicians/electrophysiologists in the procedure room and the complexity of the case.

## Guidelines for Use of Heparin

The overall risk of developing a thromboembolic complication after catheter ablation has recently been estimated to be 0.6%.[7] The risk of a thromboembolic complication is higher when ablation is performed in the left heart (1.8% to 2.0%) and/or for ventricular tachycardia (2.8%).[7] A recent survey that examined this question reported a lower incidence of thromboembolic complications with only 1 cerebrovascular accident among the 3,357 ablation procedures.[3] This lower incidence of thromboembolic complications may represent, at least in part, the greater recent use of closed loop temperature control that has been shown to decrease the incidence of coagulum formation, as well as greater attention to systemic anticoagulation during catheter ablation procedures that involve catheter manipulation in the left heart.

Despite the absence of data from clinical trials, systemic anticoagulation with heparin is suggested during ablation procedures that involve catheter manipulation in the left heart. Patients are typically treated with a loading dose of heparin (60 units/kg with a maximum of 6,000 units). Subsequent doses of heparin can be administered either as a continuous infusion (12 units/kg per hour) or as intermittent boluses of 500 to 2,000 units based on the activating clotting time (ACT). These doses may vary depending on individual patients or ablative procedures. The ACT should be checked 15 to 30 minutes after the initial loading dose and every 30 to 60 minutes thereafter.[8] The ACT should be maintained within a range of 250 to 300 seconds. Higher levels of anticoagulation (ACT > 300 sec) are often used during procedures that are associated with the highest risk of thromboembolic complications (i.e., use of a basket catheter, focal pulmonary vein isolation, or linear lesions in the left atrium for atrial fibrillation). Patients undergoing catheter ablation of atrial fibrillation or atrial flutter who are in these arrhythmias immediately prior to the catheter ablation procedure should be anticoagulated according to the guidelines that apply for external cardioversion of these arrhythmias.[10] While current guidelines suggest an INR of 2–3 (prior to cardioversion), it was felt that discontinuation of anticoagulants for 2 to 3 days prior to the procedure should suffice for most patients. For high-risk patients, consideration should be given to use of low molecular weight heparin injections or intravenous heparin as the anticoagulant effect of warfarin dissipates during the days prior to the procedure.

For procedures involving the right heart, anticoagulation should be strongly considered if an intracardiac defect that could allow paradoxical embolism is present, or when mapping or ablation devices that increase the risk of thrombus formation are used. Anticoagulation may also be considered on an individual basis, particularly for long procedures.

### Conscious Sedation

Conscious sedation is used widely to facilitate patient comfort during catheter ablation procedures.[10–12] Patients are typically sedated with midazolam alone or with a combination of midazolam and fentanyl throughout the procedure. The sedation can be administered either by a physician, nurse, an anesthesiologist, or nurse anesthetist. Propofol is another agent that is commonly used during cardioversion procedures. The specific agents, which can be used by various personnel, are often dictated by hospital policy.[10–12]

### Defibrillation

Physicians who perform catheter ablation procedures should be familiar with the indications, precautions, techniques, and complications associated with internal and external cardioversion/defibrillation procedures. These issues are discussed in detail in the recently published American College of Cardiology/American Heart Association Clinical Competence Statement.[13] All patients who undergo catheter ablation procedures should be attached to an external defibrillator with skin patch-electrodes. It is now clear that biphasic defibrillation waveforms are more efficacious than monophasic defibrillation waveforms.[14] Low energy internal cardioversion is currently being used by many centers during procedures such as catheter ablation of atrial fibrillation where multiple defibrillations are required.[15]

## Facilities and Equipment

Comprehensive catheter ablation programs require a fully equipped invasive electrophysiological laboratory and ready access to surgical support and facilities. It was felt that full cardiac surgical support was desirable but that at minimum, facilities performing ablation should have thoracic surgical backup. Imaging equipment with pulsed fluoroscopy and image storage capability is recommended. Image storage is particularly helpful during catheter ablation near vital structures. These include selective AV nodal pathway ablation in perinodal regions, septal accessory tracts, or ablation of aortic valve cusp tachycardia. Image storage is also necessary to obtain static frames of angiographic recordings of the coronary vasculature, including the coronary or pulmonary venous system, aortic root, and cardiac chambers prior to or during the mapping and ablation procedure. Biplane or C-arm x-ray equipment, which allows rapid acquisition of fluoroscopic images in different planes is desirable and recommended.[16]

Ideally, cine or digital imaging should be available for more complete recordings of angiographic images for left atrial, pulmonary vein, coronary venous system, left ventricular and right ventricular ablation procedures. During all of these procedures, hemodynamic monitoring should be performed for patient safety. Minimum standards include monitoring of systemic arterial blood pressure by automatic cuff or intra-arterial catheter, together with continuous monitoring of peripheral oxygen saturation. Other intracardiac or intravascular pressures may be recorded during specific procedures such as transseptal puncture or in patients with severely depressed ventricular function. Additional imaging equipment may include intracardiac ultrasound for catheter placement or transseptal puncture, monitoring lesion location and development, and atrial or ventricular function.[17,18] It may help reduce radiation exposure from fluoroscopic imaging.[19]

Mapping equipment, which creates three-dimensional anatomic reconstruction, helps to relate physiologic data to cardiac anatomy. These systems may further assist in the placement of multiple catheters, application of serial linear ablative lesions, and reduce radiation exposure by providing nonfluoroscopic guidance to electrode catheter locations. Systems that allow for single beat mapping may facilitate mapping and shorten the procedure time.[20,21] Such equipment is highly desirable when complex ablative procedures are planned in the atrium or the ventricle, when multiple tachycardias may be present, or brief evanescent arrhythmias are mapped.

Radiation exposure precautions, including shielding of personnel and patients, are necessary. Ceiling suspended and/or floor mounted radiation shielding is highly recommended, and is essential in laboratories performing prolonged complex imaging and ablative procedures. The laboratory should establish guidelines for total radiation exposure and should monitor patient exposure as outlined in the NASPE position paper on radiation exposure.[22] Upon completion of the procedure, radiation exposure and fluoroscopic time should be recorded.

Recording equipment should include a multichannel digital or analog recorder capable of monitoring a minimum of 3, preferably 12, surface electrocardiographic leads simultaneously with a minimum of 4, preferably 12 to 24, intracardiac recordings. Hard copy recordings should be available for immediate review at paper speeds of at least 100 mm/sec. Data storage preferably on optical disc is recommended for subsequent review and analysis. A digital or computerized programmed stimulator is required for arrhythmia induction, diagnostic testing, and tachycardia termination. Availability of at least one fully functional external DC defibrillator with noninvasive cardiac pacing capability is mandatory during these procedures. A biphasic waveform defibrillator is recommended. Facilities for temporary or permanent cardiac pacing should be available. Application of internal catheter DC shocks may be needed during procedures or resuscitation efforts. Catheter electrodes and defibrillation energy sources designed for such applications may be required in such clinical situations.

Ablation energy should include a radiofrequency current generator with a minimum power output of 50 watts with temperature and impedance monitoring. Other energy sources may be used in ablative procedures and all such equipment should be utilized in a manner conforming to hospital, local, and national regulations governing their application. In addition, the ablation catheter should be inspected periodically during the procedure if monitoring suggests the presence of coagulum. If reprocessed catheters are used for ablation, these should meet national and institutional standards for performance and sterility.

The laboratory and site should be equipped to care for patients with acute cardiac or extracardiac complications of ablative procedures such as coronary thrombosis, cardiac tamponade, vascular dissection, or pneumothorax. Oxygen, suction and airway maintenance instrumentation should be present. Availability of general anesthesia is recommended and is essential for pediatric patients. Ventilatory support, with intubation facilities and mechanical ventilators, should be available.

## Indications for Ablation Procedures

### Atrial Tachycardia

*Mechanisms*

Atrial tachycardia (AT) can be classified according to anatomic sites or mechanism. Most AT originate in the right atrium, are focal in origin, and have electropharmacological responses consistent with cAMP-mediated triggered activity.[23] Preferential sites of origin include the long axis of the crista terminalis and the circumference of the tricuspid annulus. Other focal sites that give rise to AT and that may also immediately precede atrial fibrillation include the pulmonary veins, ligament of Marshall, coronary sinus ostium, and superior vena cava. The mechanism of these focal tachycardias has not been definitely defined but are thought to be triggered. Finally, focal AT may be due to an automatic mechanism, examples of which include atrial tachycardia that clusters near the mouths of the atrial appendages (most often observed in children) and inappropriate sinus tachycardia.

Macroreentrant atrial tachycardia may involve complex reentrant circuits involving either the right or left atrium. (These mechanisms are discussed in the flutter section below.)

*Success/Complications*

Success rates for AT are highly dependent on whether the tachycardia is focal or related to atrial scarring (macroreentry). Relative to the other paroxysmal supraventricular arrhythmias, the number of patients in whom ablation has been performed is small. Because the complexity of cases considered appropriate for ablation of AT has dramatically changed over the last several years, we have compiled data from published reports since 1995.[24–29] Data from each AT subtype comprise less than 100 patients. Published success rates for incisional reentrant tachycardia are approximately 85%. Recurrence rates may be as high as 50% and these patients often have between 2 and 4 reentrant circuits, not all of which are targeted for ablation. Complications associated with this procedure are approximately 1%.

Success rates for focal AT are in general, higher than for surgical "scar" related AT. For example, the acute success rate in focal AT is 70–90% with better results for right atrial foci. Complications are uncommon. Although published reports of patients with inappropriate sinus tachycardia suggest that acute ablation success rates approach 75%–90%, enthusiasm for this procedure is tempered by high recurrence rates. Complications of the procedure include transient superior vena cava syndrome, right phrenic nerve injury with right diaphragmatic paralysis, and inadequate subsidiary atrial escape rates necessitating pacemaker implantation.[30]

Ablation of atrial tachycardia originating from the crista terminalis is successful in approximately 95% of patients. Complications include damage to the right phrenic nerve, and sinus node dysfunction in arrhythmias originating from the superior aspect of the crista terminalis. Finally, ablation of rare forms of AT originating from the anteroseptal annular region can result in complete heart block.

## Catheter Ablation for the AV Junction, AV Node Reentry, and Accessory Pathways

### Mechanisms or Rationale for Ablation

*AV Junctional Ablation*

AV junctional ablation is usually performed for patients with atrial fibrillation and rapid ventricular response whose rate cannot be controlled with drug therapy. Available data have documented improved quality of life, together with a sustained increase in ejection fraction for those with depressed systolic function.[31–40]

*AV Nodal Reentry*

These patients have dual AV nodal pathways and reentrant arrhythmias involving the AV node. The usual mechanism involves antegrade "slow" pathway and retrograde "fast" pathway conduction. Ablation is directed towards elimination of the slow pathway since the risk of AV block is clearly lower. Other forms of tachycardia manifestations including antegrade fast, retrograde slow conduction, or so-called slow-slow AV nodal reentry have been described but the slow pathway is preferentially targeted.[1–5]

*Accessory Pathway*

Tachycardias supported by extra-nodal accessory pathways are usually characterized by antegrade AV nodal conduction and retrograde accessory pathway conduction. In unusual circumstances conduction may proceed antegrade over the pathway and retrograde over the node. In either instance ablation of the accessory pathway is the desired approach.

Many prior reports have adequately documented the safety and efficacy of ablative procedures for control or care of supraventricular arrhythmias.[1,2] Three large prospective trials have focused on the safety and/or efficacy of catheter ablation of the AV junction and for patients with AV node reentry or accessory pathways.[3–5] The studies show remarkable concordance and the results of one study is detailed below.

The NASPE voluntary registry was a prospective study of 3,423 ablative procedures performed at 68 centers in the United States for the calendar year 1998. Both acute and, where possible, 6 month follow-up data were obtained by a trained nurse coordinator.

For the group of patients requiring AV junctional ablation, slow pathway ablation for AVNRT, or accessory pathway ablation, the acute success rate was found to be in excess of 90% (Tables I and II). Significant complications for those undergoing AVJ ablation included a death due to pacemaker malfunction. Others have reported a

**Table I.**

Percent of Successful Ablations and of Complications for Teaching Versus Nonteaching Hospitals

| Procedure | Teaching Hospital % Success | Nonteaching Hospital % Success |
|---|---|---|
| AVJ | 171/176, 97.2% | 458/470, 97.4% |
| AVNRT | 456/476, 95.8% | 705/732, 93.2% |
| AP (total) | 255/275, 92.7% | 372/399, 93.2% |
| Left free wall | 160/172, 93.6% | 232/247, 93.9% |
| Right free wall | 26/27, 96.3% | 54/56, 96.4% |
| Septal | 75/83, 90.4% | 90/103, 87.4% |

**Table II.**

Percent of Successful Ablations and Percent of Complications for the Medical Centers Which Did >100 Cases Versus <100 Cases in 1998

| Procedure | With >100 Cases % Success | With <100 Cases % Success |
|---|---|---|
| AVJ | 354/366, 96.7% | 275/280, 98.2% |
| AVNRT | 603/627, 96.2% | 558/581, 95.8% |
| AP (total) | 315/339, 92.9% | 312/335, 93.1% |
| Left free wall | 201/216, 93.1% | 191/202, 94.6% |
| Right free wall | 37/39, 94.9% | 43/44, 97.7% |
| Septal | 144/160, 90.0% | 85/98, 86.7% |

small incidence of sudden death due to polymorphous ventricular tachycardia (VT) early after ablation and recommend cardiac pacing at relatively rapid rates for several weeks after AVJ ablation.[6]

*Ablation*

Even after successful AV junctional ablation, the patient may still have symptoms related to pacemaker mode switch and/or pacemaker syndrome. In addition, the need for anticoagulant therapy for atrial fibrillation or flutter is not abrogated.

For patients undergoing slow pathway ablation for AVNRT, the incidence of second degree AV block was 0.16%, 0.74% developed complete AV block, and there were no other significant complications. For patients with accessory pathways, success rates varied depending on pathway location. Patients with septal pathways had a slightly lower incidence of successful ablation compared to those with free wall pathways. Complications included instances of cardiac tamponade, AV block, and rare instances of coronary occlusion and pulmonary embolism. The results for various ablative procedures are listed in Table I, which compares procedures performed at teaching hospitals versus community hospitals. No significant difference was found in incidence of successful ablation or complications between the two groups. In addition, comparison of data for large volume centers (i.e., those performing >100 cases per year) versus lower volume centers ($\leq$ 100 cases per year) (Table II) showed no significant difference between the groups.

We conclude from these data that ablation of the AV junction, slow AV nodal pathway for patients with AVNRT, and accessory pathway ablation, have matured to the point where these procedures can be performed effectively and safely in either academic centers or in experienced community hospitals.

## Atrial Flutter

The vast majority of atrial flutter cases, estimated at about 90%, involve reentry through an isthmus bounded on one side by the tricuspid valve annulus and on the other side by the inferior vena cava orifice, the Eustachian ridge, and the coronary sinus os. This isthmus is often referred to as the subeustachian isthmus. The tachycardia circuit may either show a pattern of counterclockwise (typical atrial flutter) or clockwise (reverse typical atrial flutter) progression around the tricuspid annulus as viewed fluoroscopically in the LAO position.[41]

Other less common forms of atrial flutter include: (1) lesion reentry atrial flutter in which a lesion, often from a surgical incision associated with repair of a congenital heart defect, serves as the central line of block around which the reentrant wavefront circulates - some forms of lesion reentry may have a very complex reentrant circuit and in others the reentrant circuit may involve the subeustacian isthmus; (2) left atrial flutter, in which the atrial flutter reentrant circuit is confined to the left atrium, and (3) atypical atrial flutter, which includes all other forms of reentrant atrial tachycardia in the rate range of atrial flutter that are not described by the above types.

*Indications for Atrial Flutter Ablation*

(i) Atrial flutter in which the reentrant circuit involves the subeustachian isthmus.

Subeustachian isthmus dependent atrial flutter may be cured by using radiofrequency energy delivered via a catheter electrode that achieves bidirectional block of impulses in this isthmus.[42–45] A variant of this is when atrial fibrillation, treated with an antiarrhythmic drug, recurs as subeustachian isthmus dependent flutter. In such instances, complete ablation of the atrial flutter isthmus, i.e., achieving bidirectional block in the subeustachian isthmus plus continuation of the antiarrhythmic drug will prevent recurrence of both the atrial flutter and the atrial fibrillation in 70%–80% of cases.[46,47]

Use of ablative therapy to achieve bidirectional subeustachian isthmus block is associated with a high rate of acute success. However, an atrial flutter recurrence rate of about 7% has been reported, invariably associated with recovery of conduction through the subeustachian isthmus.[29,32] It should be appreciated that in some cases of subeustachian isthmus dependent atrial flutter, anatomic variations make achieving bidirectional isthmus block quite difficult. Some uncommon types of atrial flutter have a reentrant circuit which utilizes the subeustachian isthmus, including lower loop reentry.[48] In these instances, ablation to create bidirectional block in the subeustachian isthmus provides effective treatment. Many cases of atrial flutter associated with surgical repair of congenital heart disease are due to lesion reentry or are subeustachian isthmus dependent.[49,50]

(ii) Lesion reentry atrial flutter

When a reentrant circuit due to lesion reentry can be demonstrated, atrial flutter can be cured with ablation by establishing a line of bidirectional block from the lesion to a nearby inexcitable atrial boundary.[49,50] The efficacy of applying ablative techniques to cure this type of atrial flutter depends on the ability to identify (map) the reentrant circuit, and find a vulnerable pathway (isthmus) to ablate. Nevertheless, atrial flutter associated with surgical lesions may be very complex and difficult to ablate. Should ablation therapy be contemplated, mapping of the reentrant circuit with identification of a vulnerable isthmus is critical to achieve success. Ablation should be considered in the absence of ability to control the ventricular rate or in the presence of ventricular dysfunction suspected to be related to the arrhythmia, otherwise it is desirable to demonstrate failure of suppressive antiarrhythmic drug therapy with at least one antiarrhythmic drug before proceeding to ablative therapy.

*Success and Complications*

The only large, prospective, multicenter report on the success of ablation of atrial flutter comes from the 1998 NASPE prospective catheter ablation registry (3). In this registry, a total of 477 patients underwent ablation of the atrial flutter isthmus for attempted cure of atrial flutter. Interestingly, 97 (20.4%) of the patients had failed a prior atrial flutter ablation. The data do not indicate whether there was any confirmation that the atrial flutter isthmus was a part of the atrial flutter reentrant circuit, nor whether bidirectional conduction block in the atrial flutter isthmus was achieved or even tested following the ablation. For the group as a whole, acute success was achieved in 85.8% of patients. Of particular note, the mean age was 61 + 14 years, and 134 patients (28.3%) were over age 70.

On follow-up, recurrent atrial flutter was documented in 64 (l4.7%) of patients, and 28 of these patients underwent a repeat ablation procedure. The recurrence rate reported may be related to criteria used to diagnose bidirectional isthmus block. Complications occurred in 1.2% of procedures and were largely those typical of right heart catheterization.[3] They included bleeding/hematoma in 3 patients, cardiac tamponade in one patient, deep vein thrombosis in one patient, and hemopneumothorax in one patient. Complications perhaps more in line with the risks peculiar to atrial flutter included 2 patients with inadvertent complete AV block, one patient with AV nodal Wenckebach conduction, and one patient with significant tricuspid regurgitation. Also, one patient had hypoxia, and another patient had hypotension. At follow up, 3 patients had died. One had congestive heart failure, and the cause of death was unknown in the other 2 patients.

## Atrial Fibrillation

Atrial fibrillation may occur as a result of functional abnormalities or structural changes involving the atria. Functional abnormalities include alternations in autonomic tone (either increased vagal or increased sympathetic may produce atrial fibrillation). In addition, the arrhythmia may appear in patients with hyperthyroidism. Patients with supraventricular tachycardia, of course, may develop atrial fibrillation in response to more rapid rates and/or premature beats.

For experimental models of atrial fibrillation, multiple circulating reentrant waves appear to be present. In some, atrial fibrillation may be due to a dominant rotor (mother wave) that serves to perpetuate the arrhythmia. Premature atrial depolarizations or bursts of atrial tachycardia may trigger and/or perpetuation of atrial fibrillation. In humans, these triggering foci often arise from the pulmonary veins, but also may occur in other areas, such as the superior vena cava or coronary sinus.

Because catheter ablation of atrial fibrillation is still a young discipline, and because the various ablation techniques used to cure atrial fibrillation are still evolving, conclusive data regarding the efficacy and safety of these techniques are not available.

In studies that ranged in size from 8–45 patients, linear ablation in the left and/or right atrium has been reported to eliminate atrial fibrillation in 0%–58% of patients.[51–55] Better results have been obtained in patients with paroxysmal atrial fibrillation, either by focal ablation (usually within the pulmonary veins) of the premature depolarizations that trigger atrial fibrillation,[56,57] segmental isolation of pulmonary veins guided by pulmonary vein potentials,[58] or complete electrical isolation of the pulmonary veins by circumferential ostial ablation. Circumferential ostial ablation has been accomplished with contiguous applications of radiofrequency energy. The acute success rates have ranged from 62%–90% at mean follow-up intervals of less than 1 year, with recurrence rates of 25%–40%. The best candidates for ablation procedures directed at the pulmonary veins appear to be patients with idiopathic paroxysmal atrial fibrillation.

The risk of major complications resulting from catheter ablation of atrial fibrillation has been higher than with catheter ablation of other supraventricular arrhythmias. Because only a small number of clinical studies have described the results of catheter ablation of atrial fibrillation, and because these studies have included small numbers of patients and have used different techniques, a reliable estimate of risk is not yet available. While complications of procedures in the left atrium have been reported as high as pericardial effusion/tamponade in up to 11% of patients, a cerebral ischemic event in up to 8% of patients, and 5%–20% of patients pulmonary vein stenosis; current estimates of serious complications from experienced centers are in the 2%–3% range. Ablation in the right atrium has been associated with sinus node dysfunction and pericardial effusion, each in up to 8% of patients. The techniques used to ablate atrial fibrillation are still in a development stage, and the risk:benefit ratio of these techniques remains to be determined. Because catheter ablation of atrial fibrillation is technically challenging and associated with a risk of serious complications, the procedure should be performed only in electrophysiology laboratories staffed by experienced and well-trained operators and support personnel.

## Catheter Ablation of Ventricular Tachycardia

### Mechanism of Ventricular Tachycardia

Ventricular tachycardia usually occurs in patients with structural cardiac disease. In patients with structural heart disease, particularly prior infarction, the most common mechanism for monomorphic ventricular tachycardia is related to reentry within and/or around scarred myocardium, although nonreentrant mechanisms also occur. Ventricular tachycardia may occur in patients with normal hearts and may be related to triggered or automatic rhythms (right ventricular outflow tract ventricular tachycardia) or reentry (i.e., left fascicular ventricular tachycardia).

In patients without structural heart disease idiopathic ventricular tachycardia most commonly arises either in the right ventricular outflow tract or involves the left posterior fascicle in the left ventricle. Radiofrequency catheter ablation of these types of ventricular tachycardia has had a long-term success rate ranging from 85%–100%,[59–62] and complications have been rare, with only one instance of myocardial perforation and death being reported. This must be considered a minimal figure. Less common sites include foci arising from the left ventricular outflow tract (LVOT) or from the left anterior fascicle. LVOT ablation carries the risk of damage to the coronary arteries.

In contrast, for patients with structural heart disease (i.e., ischemic heart disease, dilated cardiomyopathy, or RV dysplasia), radiofrequency catheter ablation is usually not curative. In patients with structural heart disease, multiple morphologies of ventricular tachycardia are often present, recurrences are potentially lethal, and unpredictable changes in the myocardial substrate can occur. Catheter ablation often is used as adjunctive therapy in association with an implantable cardioverter/defibrillator (ICD). Conventional mapping techniques require the ventricular tachycardia to be hemodynamically stable, and this traditionally has limited the feasibility of catheter ablation to a small proportion of patients with ventricular tachycardia in the setting of structural heart disease. In selected patients with coronary artery disease and hemodynamically stable ventricular tachycardia reported in single center studies, the success rate of conventional radiofrequency catheter ablation has ranged from 67%–96%, with a serious complication in <2% of patients.[63–78] In contrast, in patients with ventricular tachycardia due to bundle branch reentry, ablation of either right or left bundle branch is usually curative.[79–81]

In the only prospective, multicenter study of radiofrequency catheter ablation of ventricular tachycardia, 146 patients with structural heart disease underwent ablation with a saline-irrigated ablation catheter, which results in larger lesions than conventional radiofrequency ablation.[78] The acute success rate in eliminating all mappable ventricular tachycardias was 75%, but the 1-year recurrence rate was 56%. Lethal and nonlethal major complications occurred in 2.7% and 5.3% of patients, respectively.[78] Therefore, although radiofrequency ablation of ventricular tachycardia with a saline-irrigated catheter has a favorable acute success rate, the short-term benefit from the procedure must be carefully weighed against the risk of serious complications and a high recurrence rate of ventricular tachycardia during follow-up.

Newer mapping technologies available for clinical use include basket catheters, electroanatomic mapping, and noncontact mapping. Catheter ablation of hemodynamically unstable ventricular tachycardia by placing lines of radiofrequency lesions through areas of low voltage scar, appears feasible. However, further studies are needed to determine the long-term efficacy of catheter ablation of unstable ventricular tachycardia guided by these new approaches.

**Radiofrequency Catheter Ablation for the Pediatric Population**

The ablation of arrhythmias in children poses unique challenges compared with adults due to differences in body habitus, vascular and cardiac dimensions, the presence of congenital cardiovascular defects in 12%, and the psychological make up of the patients. The types of arrhythmias treated are the same but the prevalence is different. In pediatric patients automatic atrial tachycardias are seen more frequently than in adults and accessory pathway mediated tachycardias are also more frequent. The equipment used for pediatric ablation is virtually identical with greater use of smaller ablation catheters. The techniques are very similar except that general anesthesia is used more frequently in children.

The Pediatric Radiofrequency Ablation Registry of the Pediatric Electrophysiology Society is a voluntary data collection registry focusing on acute results from 49 centers.[82] Data comprised 6,504 patients who underwent 7,305 procedures. Their ages ranged from 0.1–20.9 years (mean 12.3 years) and their weight from 1.9–139 kg mean. Eighty-eight percent had anatomically normal hearts. Of the congenital defects, Ebstein's anomaly and congenitally corrected transposition were the most common.

Successful ablation was achieved in 91% of patients with accessory pathways, 97% for those with AV nodal reentry, 87% for those with focal atrial tachycardia, 75% for those with atrial flutter, and 48% of those with ventricular tachycardia. Factors associated with success included the presence of a left free wall accessory pathway and AV node reentry. Factors associated with failure included anterior septal pathways, ventricular tachycardia, and intra-atrial reentry. Complications occurred in 7.6% of cases and included 2.9% major complications. Complications were more frequent in patients with anteroseptal pathways. Twenty-seven percent of major complications were second or third degree AV block while 22% involved perforation and/or a pericardial effusion. Major complications increased significantly in patients <4 years of age. Death occurred acutely or during follow-up in 0.46% of patients but at least half were unrelated to the procedure. Factors favoring fewer complications were the presence of right free wall pathways and the experience of the operator (>20 previous ablations). Factors associated with increased likelihood of death were presence of congenital heart disease and left-sided foci or pathways.

Thus, radiofrequency catheter ablation has been carried out in children by experienced pediatric teams, with similar success rates and complication rates as in adults. The natural history of frequent spontaneous resolution of all mechanisms of tachycardia in the first year of life favors a cautious approach in this age group.

RECOMMENDATIONS: The recommendations for catheter ablation of specific tachycardias are categorized in the standard ACC/AHA format as follows:

Class I: Conditions for which there is general agreement that a given procedure or treatment is beneficial, useful, and effective.

Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

Class IIa: Weight of evidence/opinion is in favor of usefulness/efficacy.

Class IIb: Usefulness/efficacy is less well established by evidence/opinion.

Class III: Conditions for which there is evidence and/or general agreement that a procedure/treatment is not useful/effective and in some cases may be harmful.

In addition, the recommendations are evidence based where possible:

Level A: Data supporting recommendation is derived from multiple randomized clinical trials that included large numbers of patients.

Level B: Data supporting recommendation is derived from a limited number of randomized trials including small numbers of patients or from data analysis of nonrandomized studies or observational data registries.

Level C: Recommendation is based on consensus of expert opinion without evidence from clinical trials.

**Class I**

1. AV node reentry: slow pathway ablation: For those patients in whom treatment of AVNRT is deemed necessary ablation can be offered as an initial therapy option. Ablation should be recommended for those patients who have failed ≥1 antiarrhythmic drug or who have significant side effects to drug therapy. (Evidence level B.)

Fast pathway ablation: Because of the risk of complete heart block, ablation directed at the fast pathway should be reserved for those patients who have failed drug therapy as well as prior attempts at slow pathway ablation.

2. AV reentry: Recommendation for ablation therapy for patients with accessory pathway mediated SVT are the same as for AV node reentry

above, with the exception of those patients with atrial fibrillation and rapid ventricular response who should undergo ablation as initial therapy. Patients with anteroseptal pathways deserve special consideration because the increased risk of complete heart block from catheter ablation reduces the benefit/risk balance. (Evidence level B.)

3. AV junction: Ablation of the AV junction with subsequent complete heart block should be recommended for those patients with atrial tachycardias, particularly persistent or permanent atrial fibrillation in which the ventricular response rate cannot be adequately controlled with AV nodal blocking agents. (Evidence level A.)

4. Focal atrial tachycardia: In general patients with focal atrial tachycardias should receive at least one trial of antiarrhythmic drug therapy prior to catheter ablation. However depending on the experience of the electrophysiologist and resources such as specialized mapping systems, ablation could be offered as an initial therapeutic approach when therapy to suppress the arrhythmia is required. (Evidence level B.)

5. Isthmus dependent atrial flutter: Ablation can be considered initial therapy for patients with recurrent isthmus-dependent atrial flutter. (Evidence level A.)

### Class IIa

1. Nonisthmus-dependent macroreentrant atrial tachycardias: These atrial tachycardias include scar-related macroreentrant atrial tachycardia, left atrial macroreentrant tachycardia, and other atypical atrial flutters. Because of the potential complexity of these reentrant circuits, ablation should be recommended only after a trial of drug therapy. (Evidence level B, C.)

2. Idiopathic ventricular tachycardia: Because of the small numbers of patients reported and the limited follow-up, the accumulated evidence is insufficient to determine the complications of ablation and the long-term outcome in these patients. Ablation should be considered as therapy for these tachycardias after an appropriate trial of antiarrhythmic drug therapy. Ablation should be considered as therapy for these tachycardias after an appropriate trial of antiarrhythmic drug therapy or as initial therapy for patients unwilling or unable to take drugs.

RV outflow tract: In the typical individual with exercise-induced ventricular tachycardia ablation should be considered after a trial of $\beta$-blocker therapy. (Evidence level C.)

LV fascicular tachycardia: similarly for patients without structural cardiac disease and fascicular tachycardia, ablation should be considered after a failed trial of drug therapy. (Evidence level C.)

3. Ischemic ventricular tachycardia: Ablation should generally be considered as adjunctive and not curative therapy for ischemic VT and as such should only be recommended as treatment for drug-refractory recurrent VT. (Evidence level B, C.)

### Class IIb

1. Inappropriate sinus tachycardia: Because of a high recurrence rate and persistence of nonspecific symptomatology postablation, catheter ablation can be considered only after trials of drug therapy including $\beta$-blockers. (Evidence level C.)

2. Atrial fibrillation: Because of the small numbers of patients reported, the short duration of follow-up, and the developing approach to the specific technique of performing ablation, the accumulated evidence is insufficient to determine the complications and long-term outcome in these patients. Ablation could be considered as therapy for patients with paroxysmal (focal) atrial fibrillation after an appropriate trial of antiarrhythmic therapy. Patients with permanent atrial fibrillation who might be considered for catheter-based ablative therapy should be referred to centers with a specific interest and experience in performing more complex procedures required for these cases. (Evidence level B, C.)

3. Left ventricular outflow tract: Because of the possibility of the *potential for* significant complications with ablation at the LV outflow tract, ablation should be recommended only after a trial of drug therapy (Evidence level C.)

4. Arrhythmogenic RV dysplasia VT: Because of the high recurrence rate of VT associated with ARVD, ablation should only be considered adjunctive therapy and should be recommended only in cases of recurrences after adequate drug therapy. (Evidence level C.)

### Class III

1. Tachycardias with reversible causes: Any tachycardia or treatable trigger should be managed medically with treatment directed at the precipitating mechanism. Such tachycardias might include VT due to acute ischemia and drug-induced tachycardias.

2. Sinus node modification or ablation prior to trials of drug therapy.

3. Sinus node modification or ablation for patients with postural orthostatic hypotension.

4. Accessory pathway ablation for patients with pre-excitation, but who are asymptomatic (except possible extenuating circumstances relating to pediatric population or high-risk occupational situations).

## References


1. Jazayeri MR, Hempe SL, Sra JS, et al. Selective transcatheter ablation of the fast and slow pathways using radiofrequency energy in patients with atrioventricular nodal reentrant tachycardia. Circulation 1992; 85:1318–1328.
2. Jackman WM, Beckman KJ, McClelland JH, et al. Treatment of supraventricular tachycardia due to atrioventricular nodal reentry by radiofrequency catheter ablation of the slow-pathway. N Eng J Med 1992; 327:313–318.
3. Scheinman MM, Huang S. The 1998 NASPE prospective catheter ablation registry. PACE 2000; 23:1020–1028.
4. Calkins H, Yong P, Miller JM, et al. Catheter ablation of accessory pathways, atrioventricular nodal reentrant tachycardia, and the atrioventricular junction. Final results of a prospective, multicenter clinical trial. Circulation 1999; 99:262–270.
5. Hindricks G, on behalf of the Multicenter European Radiofrequency Survey (MERFS) Investigators of the working group on arrhythmias of the european society of cardiology. The Multicentre European Radiofrequency Survey (MERFS): Complications of radiofrequency catheter ablation of arrhythmias. Eur Soc Cardiol 1993; 14:1644–1653.
6. Calkins H, Epstein A, Packer D, et al. Catheter ablation of ventricular tachycardia in patients with structural heart disease using cooled radiofrequency energy: Results of a prospective multicenter study. Cooled RF Multi Center Investigators Group. J Am Coll Cardiol 2000; 35: 1905–1914.
7. Zhou L, Keane D, Reed G, et al. Thromboembolic complications of cardiac radiofrequency catheter ablation: A review of the reported incidence, pathogenesis and current research directions. J Cardiovasc Electrophysiol 1999; 10:611–620.
8. Albers GW, Dalen JE, Laupacis A, et al. Antithromobotic therapy in atrial fibrillation. Chest 2001; 119(1 suppl):194S–206S.
9. Hirsh J, Warkentin TE, Shaughnessy SC, et al. Heparin and low-molecular-weight heparin: Mechanism of action, pharmokinetics, dosing, monitoring, efficacy and safety. Chest 2001; 119(1 suppl):64S–94S.
10. Fuster V, Ryden LE, Asinger RW, et al. ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation-executive summary. J Am Coll Cardiol 2001; 38:1231–1266.
11. Bubien RS, Fisher JD, Gentzel JA, et al. NASPE expert consensus document: use of i.v. (conscious) sedation/analgesia by nonanesthesia personnel in patients undergoing arrhythmia specific diagnostic, therapeutic, and surgical procedures. PACE 1998; 21:375–385.
12. Bryson HM, Fulton BR, Faulds D. Propofol. An update of its use in anesthesia and conscious sedation. Drugs 1995; 50(3):513–559.
13. Tracy CM, Akstar M, DiMarco JP, et al. American College of Cardiology/American Heart Association clinical competence statement on invasive electrophysiology studies, catheter ablation, and cardioversion. Circulation 2000; 102:2309–2320.
14. Dell'Orfano JT, Naccarelli GV. Update on external cardioversion and defibrillation. Curr Opin Cardiol 2001; 16(1):54–57.
15. Boriani G, Biffi M, Camanini C, et al. Transvenous low energy internal cardioversion for atrial fibrillation: A review of clinical applications and future developments. PACE 2001; 24(1):99–107.
16. Wittkampf FH, Wever EF, Vos K, et al. Reduction in radiation exposure in the cardiac electrophysiology laboratory. PACE 2000: 23:1638–1644.
17. Marchlinski FE, Ren JF, Schwartzmann D, et al. Accuracy of fluroscopic localization of the crista terminalis documented by intracardiac echocardiography. J Intervent Card 2000: 4:415–421.
18. Dauod EG, Kalbfleisch SJ, Hummel JD. Intracardiac echocardiography to guide transseptal left heart catheterization for radiofrequency catheter ablation. J Cardiovasc Electrophysiol 1999:10:358–363.
19. Rosenthal LS, Mahesh M, Beck TJ, et al. Predictors of fluoroscopy time and estimated radiation exposure during radiofrequency catheter ablation procedures. Am J Cardiol 1998: 82:451–458.
20. Kottkampf H, Hugl B, Krauss B, et al. Electromagnetic versus fluoroscopic mapping of the inferior isthmus for ablation of typical atrial flutter: A prospective randomized study. Circulation 2000: 102: 2082–2086.
21. Saksena S. Benefits of new catheter mapping technology: Fact or fiction? J Intervent Card Electrophysiol 2000; 4(2):339–343.
22. Limacher MC, Douglas PS, Germano G, et al. ACC expert consensus document. Radiation safety in the practice of cardiology. J Am Coll Cardiol 1998; 31:892–913.
23. Markowitz SM, Stein KM, Mittal S, et al. Differential effects of adenosine on focal and macroreentrant atrial tachycardia. J Cardiovasc Electrophysiol 1999; 10:489–502.
24. Marchlinski F, Calland D, Gottlieb C, et al. Magnetic electroanatomical mapping for ablation of focal ATs. PACE 1998; 21:1621–1635.
25. Kottkamp H, Hindricks G, Breithardt G, et al. Three-dimensional electromagnetic catheter technology: Electroanatomical mapping of the right atrium and ablation of ectopic AT. J Cardiovasc Electrophysiol 1997; 8:1332–1337.
26. Schmitt H, Weber S, Schwab JO, et al. Diagnosis and ablation of focal right AT using a new high-resolution, non-contact mapping system. Am J Cardiol 2001; 87:1017–1021.
27. Hsieh MH, Chen SA. Catheter ablation of focal AT. In DP Zipes, M Haissaguerre, (eds.): Catheter Ablation of Arrhythmias, 2nd edition, Armonk NY, Futura Publishing Co. Inc., 2002.
28. Weiss C, Willem S, Cappato R, et al. High frequency current ablation of electropic AT: Different mapping strategies for localization of right and left-sided origin. Herz 1998; 23:269–279.
29. Anguera I, Brugada J, Roba M, et al. Outcomes after radiofrequency catheter ablation of AT. Am J Cardiol 2001; 87:886–890.
30. Man KC, Knight B, Tse HF, et al. Radiofrequency catheter ablation of inappropriate sinus tachycardia guided by activation mapping. J Am Coll Cardiol 2000; 35:451–457.
31. Kay GN, Ellenbogen KA, Giudici M, et al. The Ablate and Pace Trial: A prospective study of catheter ablation of the AV conduction system and permanent pacemaker implantation for treatment of atrial fibrillation. APT investigators. J Interv Card Electrophysiol 1998; 2:121–135.
32. Ozcan C, Jahangir A, Friedman PA, et al. Long-term survival after ablation of the atrioventricular node and implantation of a permanent pacemaker in patients with atrial fibrillation. New Engl J Med 2001; 344:1043–1051.
33. Marshall HJ, Harris ZI, Griffith MJ, et al. Prospective randomized study of ablation and pacing versus medical therapy for paroxysmal atrial fibrillation: Effects of pacing mode and mode-switch algorith. Circulation 1999; 99:1587–1592.
34. Wood MA, Brown-Mahoney C, Kay GN, et al. Clinical outcomes after ablation and pacing therapy for atrial fibrillation: A meta-analysis. Circulation 2000; 101:1138–1144.
34. 35.Levy T, Walker S, Mason M, et al. Importance of rate control or rate regulation for improving exercise capacity and quality of life in patients with permanent atrial fibrillation and normal left ventricular function: A randomised controlled study. Heart 2001; 85:171–178.
36. Ueng KC, Tsai TP, Tsai CF, et al. Acute and long-term effects of atrioventricular junction ablation and VVIR pacemaker in symptomatic patients with chronic lone atrial fibrillation and normal ventricular response. J Cardiovasc Electrophysiol 2001; 12:303–309.
37. Yeung-Lai-Wah JA, Qi A, Uzon O, et al. Long-term survival following radiofrequency ablation of atrioventricular junction for atrial fibrillation: Clinical and ablation determinants of mortality. J Interv Card Electrophysiol 2002; 6:17–23.
38. Brignole M, Gianfranchi L, Menozzi C, et al. Influence of atrioventricular junction radiofrequency ablation in patients with chronic atrial fibrillation and flutter on quality of life and cardiac performance. Am J Cardiol 1994; 74:242–246.
39. Lee SH, Chen SA, Tai CT, et al. Comparisons of quality of life and cardiac performance after complete atrioventricular junction ablation and atrioventricular junction modification in patients with medically refractory atrial fibrillation. J Am Coll Cardiol 1998; 31:637–644.
40. Fitzpatrick AP, Kourouyan HD, Siu A, et al. Quality of life and outcomes after radiofrequency His-bundle catheter ablation and permanent pacemaker implantation: Impact of treatment in paroxysmal and established atrial fibrillation. Am Heart J 1996; 131:499–507.
41. Saoudi N, Cosio F, Waldo A, et al. Classification of atrial flutter and regular atrial tachycardia according to electrophysiologic mechanism and anatomic bases: A statement of the joint expert group from the Working Group of Arrhythmias of the ESC and NASPE. J Cardiovasc Electrophysiol 2001; 12:852–866.
42. Poty H, Saoudi N, Abdel Aziz A, et al. Radiofrequency ablation of type 1 atrial flutter: Prediction of late success by electrophysiological criteria. Circulation 1995; 92:1389–1392.
43. Cauchemez B, Haissaguerre M, Fischer B, et al. Electrophysiological effects of catheter ablation of inferior vena cava bicuspid annulus isthmus in common atrial flutter. Circulation 1996; 93:284–294.

44. Poty H, Saoudi N, Nair M, et al. Radiofrequency ablation of atrial flutter. Further insights into the various types of isthmus block: Application to ablation during sinus rhythm. Circulation 1996; 94:3204–3213.
45. Huang DT, Monohan KM, Zimetbaum P, et al. Hybrid pharmacologic and ablative therapy: A novel and effective approach for the management of atrial fibrillation. J Cardiovasc Electrophysiol 1998; 9:462–467.
46. Nabar A, Rodriguez LM, Timmermans C, et al. Radiofrequency ablation of "Class IC atrial flutter" in patients with resistant atrial fibrillation. Am J Cardiol 1999; 83:785–787.
47. Tai C-T, Chen S-A, Chiang C-E, et al. Long-term outcome of radiofrequency catheter ablation for typical atrial flutter: Risk prediction of recurrent arrhythmias. J Cardiovasc Electrophysiol 1998; 9:115–121.
48. Cheng J, Cabeen WR Jr, Scheinman MM. Right atrial flutter due to lower loop reentry: Mechanism and anatomic substrates. Circulation 1999; 99:1700–1705.
49. Chan DP, Van Hare GF, Mackall JA, et al. Importance of the atrial flutter isthmus in post-operative intra-atrial reentrant tachycardia. Circulation 2000; 102:1283–1289.
50. Collins KK, Love BA, Walsh EP, et al. Location of acutely successful radiofrequency catheter ablation of intraatrial reentrant tachycardia in patients with congenital heart disease. Am J Cardiol 2000, 86:969–974.
51. Haissaguerre M, Jais P, Shah DC, et al. Right and left atrial radiofrequency catheter ablation therapy of paroxysmal atrial fibrillation. J Cardiovasc Electrophysiol 1996; 7:1132–1144.
52. Gaita F, Riccardi R, Calo L, et al. Atrial mapping and radiofrequency catheter ablation in patients with idiopathic atrial fibrillation. Circulation 1998; 97:2136–2145.
53. Pappone C, Oreto G, Lamberti F, et al. Catheter ablation of paroxysmal atrial fibrillation using a 3D mapping system. Circulation 1999; 100:1203–1208.
54. Garg A, Finneran W, Mollerus M, et al. Right atrial compartmentalization using radiofrequency catheter ablation for management of patients with refractory atrial fibrillation. J Cardiovasc Electrophysiol 1999; 10:763–771.
55. Ernst S, Schlutter M, Ouyang F, et al. Modification of the substrate for maintenance of idiopathic human atrial fibrillation. Efficacy of radiofrequency ablation using nonfluoroscopic catheter guidance. Circulation 1999; 100: 2085–2092.
56. Haissaguerre M, Jais P, Shah DC, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. N Engl J Med 1998; 339:659–666.
57. Chen SA, Hsieh MH, Tai CT, et al. Initiation of atrial fibrillation by ectopic beats originating from the pulmonary veins: Electrophysiological characteristics, pharmacological responses, and effects of radiofrequency ablation. Circulation 1999; 100: 1879–1886.
58. Haissaguerre M, Jais P, Shah DC, et al. Electrophysiological end point for catheter ablation of atrial fibrillation initiated from multiple pulmonary venous foci. Circulation 2000; 101:1409–1417.
59. Klein LS, Shih HT, Hackett FK, et al. Radiofrequency catheter ablation of ventricular tachycardia in patients without structural heart disease. Circulation 1992; 85:1666–1674.
60. Nakagawa H, Beckman KJ, McClelland JH, et al. Radiofrequency catheter ablation of idiopathic left ventricular tachycardia guided by a Purkinje potential. Circulation 1993; 88:2606–2617.
61. Wen MS, Yeh SJ, Wang CC, et al. Radiofrequency ablation therapy in idiopathic left ventricular tachycardia with no obvious structural heart disease. Circulation 1994; 89:1690–1696.
62. Coggins DL, Lee RJ, Sweeney J, et al. Radiofrequency catheter ablation as a cure for idiopathic tachycardia of both left and right ventricular origin. J Am Coll Cardiol 1994; 23:1333–1341.
63. Stevenson WG, Khan H, Sager P, et al. Identification of reentry circuit sites during catheter mapping and radiofrequency ablation of ventricular tachycardia late after myocardial infarction. Circulation 1993; 88:1647–1670.
64. Kim YH, Sosa-Suarez G, Trouton TG, et al. Treatment of ventricular tachycardia by transcatheter radiofrequency ablation in patients with ischemic heart disease. Circulation 1994; 89:1094–1102.
65. Gonska BD, Cao K, Schaumann A, et al. Catheter ablation of ventricular tachycardia in 136 patients with coronary artery disease: Results and long-term follow-up. J Am Coll Cardiol 1994; 24:1506–1514.
66. Bogun F, Bahu M, Knight BP, et al. Comparison of effective and ineffective target sites that demonstrate concealed entrainment in patients with coronary artery disease undergoing radiofrequency ablation of ventricular tachycardia. Circulation 1997; 95:183–190.
67. Strickberger SA, Man KC, Daoud EG, et al. A prospective evaluation of catheter ablation of ventricular tachycardia as adjuvant therapy in patients with coronary artery disease and an implantable cardioverter-defibrillator. Circulation 1997; 96:1525–1531.
68. Gonska BD, Cao K, Schaumann A, et al. Catheter ablation of ventricular tachycardia in 136 patients with coronary artery disease: Results and long term follow-up. J Am Coll Cardiol 1994; 24:1506–1514.
69. Rothman SA, Hsia HH, Cossu SF, et al. Radiofrequency catheter ablation of post infarction ventricular tachycardia: Long-term success and the significance of inducible non clinical arrhythmias. Circulation 1997; 96:3499–3508.
70. Stevenson WG, Friedman Pl, Kocovic D, et al. Radiofrequency catheter ablation of ventricular tachycardia after myocardial infarction. Circulation 1998; 98:308–314.
71. Soejima K, Suzuki M, Maisel WH, et al. Catheter ablation in patients with multiple and unstable ventricular tachycardias after myocardial infarction: Short ablation lines guided by reentry circuit isthmuses and sinus rhythm mapping. Circulation 2001; 104:664–669.
72. Wilber DJ, Kopp DE, Glascock DN, et al. Catheter ablation of the mitral isthmus for ventricular tachycardia associated with inferior infarction. Circulation 1995; 92:3481–3489.
73. Hadjis TA, Stevenson WG, Harada T, et al. Preferential locations for critical reentry circuit sites causing ventricular tachycardia after inferior wall myocardial infarction. J Cardiovasc Electrophysiol 1997; 8:363–370.
74. De Chillou C, Lacroix D, Klug D, et al. Isthmus characteristics of reentrant ventricular tachycardia after myocardial infarction. Circulation 2002; 105:726–731.
75. Marchlinski FE, Callans DJ, Gottlieb CD, et al. Linear ablation lesions for control of unmappable ventricular tachycardia in patients with ischemic and nonischemic cardiomyopathy. Circulation 2000; 101:1288–1296.
76. Soejima K, Delacretaz E, Suzuki M, et al. Saline-cooled versus standard radiofrequency catheter ablation for infarct-related ventricular tachycardias. Circulation 2001; 103:1858–1862.
77. Della Bella P, De Ponti R, Uriate JA, et al. Catheter ablation and antiarrhythmic drugs for hemodynamically tolerated post-infarction ventricular tachycardia; Long-term outcome in relation to acute electrophysiological findings. Eur Heart J 2002; 23:414–424.
78. Morady F, Harvey M, Kalbfleisch SJ, et al. Radiofrequency catheter ablation of ventricular tachycardia in patients with coronary artery disease. Circulation 1993; 87:363–372.
79. Blanck Z, Dhala A, Deshpande S, et al. Bundle branch reentrant ventricular tachycardia: Cumulative experience in 48 patients. J Cardiovasc Electrophysiol 1993; 4:253.
80. Touboul P, Kirkorian G, Atallah G, et al. Bundle branch reentrant tachycardia treated by electrical ablation of the right bundle branch. J Am Coll Cardiol 1986; 7:1404.
81. Tchou P, Jazayeri M, Denker S, et al. Transcatheter electrical ablation of the right bundle branch: A method of treating macroreentrant ventricular tachycardia due to bundle branch reentry. Circulation 1988; 78:246.
82. Friedman RA, Walsh EP, Silka MJ, et al. NASPE Expert Consensus Conference: Radiofrequency catheter ablation in children with and without congenital heart disease. Report of the writing committee. North American Society of Pacing and Electrophysiology. PACE 2002; 25:1000–1017.