Atrial Fibrillation in the Athlete

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Abstract
Atrial fibrillation (AF) is the most common arrhythmia in athletes. Evidence supports that it occurs more frequently in endurance athletes than in nonathletes and that it can result in decreased performance or even ineligibility for athletes. Although there is no clear etiology of why the increase in athletes exists, three supported mechanisms include morphologic adaptation, autonomic alteration, and chronic systemic inflammation. Although treatment in athletes can be challenging, type 1C antiarrhythmics are accepted generally as a first-line therapy in addition to risk factor-based anticoagulation. Radiofrequency catheter ablation also has become a recommended treatment for symptomatic paroxysmal AF that is refractory to at least one class 1 or 3 antiarrhythmic medication and a reasonable treatment in symptomatic paroxysmal AF prior to initiation of antiarrhythmic therapy.

Introduction
Atrial fibrillation (AF) is the most common arrhythmia in athletes. Studies have shown there is a higher incidence and prevalence of AF among athletes when compared with the general population. This is more notable in middle-aged athletes than young athletes and more so in endurance athletes or athletes with intense short-term training and long-term sports participation. AF in athletes is found generally to be lone AF that is paroxysmal in nature. Although AF in this population is benign generally, it may result in decreased performance or ineligibility for the athlete, which has been shown to have severe emotional, mental, and physical effects. There is no clear evidence of the causal mechanism of AF in athletes, but possibilities include morphologic adaptation, autonomic alteration, and chronic systemic inflammation. Treatment guidelines suggest treating AF in athletes similar to that in the general population; however, long-term adverse effect profiles, toxicity, impairment of performance, and even sport-specific bans on treatment medications make treatment in athletes more challenging. Radiofrequency catheter ablation (RFCA) in drug-resistant lone AF has been shown to be as effective in athletes as in the general population and has emerged as a possibility to consider in treatment regiments.

Discussion
Definitions
It is important to understand the terms used to describe AF since study results and treatment recommendations are related often to a specific type of AF. The American College of Cardiology (ACC), American Heart Association (AHA), and the European Society of Cardiology (ESC) recommend in their guidelines the following classification system: first diagnosed AF, paroxysmal AF, persistent AF, long-standing persistent AF, and permanent AF. Every patient who presents with AF for the first time is considered a patient with first diagnosed AF. Paroxysmal AF is self-terminating, usually within 48 h, but up to 7 d. Persistent AF is when the episode lasts longer than 7 d or requires termination by cardioversion with drugs or direct current. When AF has lasted for more than 1 year, it is considered long-standing persistent AF. Permanent AF is said to exist when the patient and physician accept the presence of the arrhythmia and rhythm control interventions are no longer pursued. Additional terms that are often seen are lone-AF, nonvalvular AF, and secondary AF. When there is an absence of clinical or echocardiographic findings of other cardiovascular disease (including hypertension), related pulmonary disease, or cardiac abnormalities and age is less than 60 years, it is considered lone AF. Absence of rheumatic mitral valve disease, a prosthetic valve, or mitral valve repair is considered nonvalvular AF. Secondary AF occurs in the setting of a primary condition that may be the cause of the AF such as an acute myocardial infarction or hyperthyroidism.

Epidemiology
Several studies have reported a significant increase in incidence, prevalence, and relative risk of AF in athletes versus the general population. This increase was less in earlier studies that included younger athletes with relatively fewer years of training. For example, studies by Furlanello et al. (9) had athletes with mean ages of 21 and 24 ± 6 years and AF prevalence of 0.2% and 0.3%, respectively (18,21). However, the prevalence in the general...
population is <0.5% at 40 to 50 years and 5% to 15% at 80 years (5). Later studies (Table 1) with mean ages of 40 to 66 years reported prevalence rates of 5% to 63% in athletes (1,14,19,21). A large 12-year prospective cohort study of 16,921 patients by Aizer et al. (2) found that middle aged subjects <50 years old who performed moderate exercise 5 to 7 times per week had a relative risk of 1.74 (95% confidence interval (CI), 1.23 to 2.47, P < 0.01) after controlling for all potential confounding variables and medical conditions associated with AF (21). In that same study, men that jogged ≥5 d wk⁻¹ had a 53% increase risk for developing AF compared with those that did not exercise. This is consistent with other studies that show the increased risk in endurance type athletes. Interestingly, the Aizer study did not show a significant increased risk if exercise was 3 to 4 d wk⁻¹ or less or if exercise was 5 to 7 d wk⁻¹ but the patient age was >50 years old. Mozaffarian et al. (17) also showed in a prospective cohort study of 5,446 patients who were all older than 65 years that light to moderate physical activities, particularly leisure-time activity and walking (600 kcal·wk⁻¹), are associated with significantly lower AF incidence. Results from the Grup Integrat de Recerca en Fibril·lacio Auricular (GIRAfA) study showed that intense physical activity of >546 h was associated with an odds ratio (OR) of developing AF of 7.31 (95% CI, 2.33 to 22.96, P < 0.01), but 1 to 563 h of activity had an OR of only 1.7 (95% CI, 0.22 to 14.26, P = 0.5) (21). These results show there is an increased OR of AF related to exercise, but that its relationship to the intensity of exercise and the beneficial effects of exercise on other comorbidities that are risk factors for AF, especially in patients >65 years old, requires further prospective controlled studies.

Postulated mechanisms

The mechanism of AF in athletes is unclear and likely multifactorial. The three suspected etiologies include anatomic changes, alterations in the autonomic nervous system, and a sustained inflammatory response predisposing athletes to AF (14). Endurance training is associated with increased left and right atrial size; however, the clinical and arrhythmic consequences of this enlargement have been debated widely (19). A study by Pelliccia et al. (18) of 1,777 highly trained athletes examined at the National Institute of Sports Medicine from 1992 to 1995 with an average age of 24 ± 6 years was assessed for the prevalence, clinical significance, and arrhythmic consequences of left atrial enlargement. They found 347 athletes (20%) to have left atrial enlargement with transverse dimensions ≥40 mm. However, AF occurred equally in athletes that had atrial enlargement (0.28%) and in athletes that did not have atrial enlargement (0.27%) (19). While this study showed a significant number of athletes having atrial enlargement (20%), its ability to detect a relationship with AF may have been related to the low prevalence of AF (0.3%). This low prevalence was related likely to the relatively young age of the study population. A small case-control study of 45 athletes showed biochemical evidence of distribution of the collagen equilibrium that would favor fibrosis compared with sedentary controls (15b,21). This concept of fibrosis was examined also in a study performed on male Wistar rats that were conditioned to run vigorously for 4, 8, and 16 wk and compared with time-matched sedentary rats. It was found that at 16 wk, the exercise rats developed eccentric hypertrophy and diastolic function together with atrial dilatation with collagen deposition in the right ventricle and that there were significantly greater protein expressions of fibrosis markers in both atria and the right ventricle than in the sedentary rats (21). These fibrotic changes were reversed after an 8-wk exercise cessation period. These findings are yet to be confirmed in human studies.

Swanson (20) has made the hypothesis that older athletes with AF, but otherwise healthy, who have engaged in rigorous aerobic endurance exercise for more than a decade will have C-reactive protein (CRP) levels that are higher than those athletes without AF. If this hypothesis was validated, one could then consider a prospective clinical trial of anti-inflammatory therapy in athletes. While there are studies on the individual pieces of this hypothesis, there is no study that addresses all three components: inflammatory markers, AF, and athletes. There have been studies that have shown that prolonged, intense endurance exercise acutely increases serum concentrations of the inflammatory markers: interleukin-6 and CRP (19,20). While regular moderate endurance exercise training generally reduces inflammatory markers, high-intensity exercise such as a marathon race may transiently produce a sustained systemic inflammatory response and elevated CRP levels (19,20). This systemic inflammatory response is postulated by some to be a central part of “overtraining syndrome” or excessive training that results in deterioration of performance (20). This would coincide also with the ESC statement that in population-based studies, the intensity of physical activity showed a U-shaped relationship with the incidence of AF, which may indicate that the positive antiarrhythmic effects of physical activity are negated partially when exercise is too strenuous (5). Separate studies have noted an association between elevated CRP levels and both the presence of AF and the risk of developing AF (19,20). Lastly, separate studies report that medications with anti-inflammatory properties, such as glucocorticoids, statins, angiotensin-converting enzyme inhibitor, and angiotensin II receptor blockers (ARBs), seem to reduce recurrence of AF (20).

Physiologic adaptations associated with intense exercise are known as “athletes heart” and generally serve to meet the increased oxygen demand of skeletal muscle during rigorous activity (22). Increased parasympathetic tone and reduced sympathetic tone at rest are characteristics of aerobically trained athletes and result in resting bradycardia (19). The GIRAfA study demonstrated that 70% of patients with lone AF (the predominant type of AF in athletes) had vagal AF (21). Augmented vagal tone shortens the atrial refractory period and facilitates reentry and the appearance of AF (19,21).

Why It’s a problem

Why is there significant concern of this increased prevalence of AF in athletes?

In the general population, death rates are doubled by AF independent of other known predictors of mortality (5). Approximately, every fifth stroke is due to AF, and stroke in AF is often severe and results in long-term disability or
<table>
<thead>
<tr>
<th>Author</th>
<th>Type of Study</th>
<th>No. of Athletes</th>
<th>No. of Controls</th>
<th>Variable of Interest</th>
<th>Age</th>
<th>Result in Athletes</th>
<th>Result in Controls</th>
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<tbody>
<tr>
<td>Mont et al. (15b,19)</td>
<td>Subgroup analysis of lone AF population</td>
<td>120 mixed sports</td>
<td>96</td>
<td>Percentage of sportsmen vs nonsportsmen with AF</td>
<td>48 ± 10</td>
<td>63%</td>
<td>15%</td>
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<tr>
<td>Elosua et al. (8a,19)</td>
<td>Retrospective case control</td>
<td>58 endurance athletes</td>
<td>51</td>
<td>Incidence of AF</td>
<td>Odds ratio, 2.87; CI, 1.39 to 7.05</td>
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<tr>
<td>Molina et al. (15a,19)</td>
<td>Retrospective cohort study</td>
<td>252 marathon runners</td>
<td>305</td>
<td>Incidence of AF</td>
<td>39 ± 9 athletes, 50 ± 13 controls</td>
<td>5%</td>
<td>0.7%</td>
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<tr>
<td>Karjalainen et al. (12a,19)</td>
<td>Nested case control</td>
<td>300 orienteers</td>
<td>495</td>
<td>Relative risk and incidence</td>
<td>48 ± 6</td>
<td>RR, 5.5</td>
<td>Incidence 0.9%</td>
</tr>
<tr>
<td>Baldersberger and Attenhofer (2a,19)</td>
<td>Longitudinal case control</td>
<td>134 cyclist</td>
<td>62</td>
<td>Incidence</td>
<td>67 ± 7</td>
<td>10%</td>
<td>0%</td>
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<tr>
<td>GIRAFA Study (15b,19)</td>
<td>Prospective case control</td>
<td>107 physically active adults</td>
<td>107</td>
<td>Incidence</td>
<td>Odds ratio, 7.31; CI, 2.33 to 22.9</td>
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<tr>
<td>Heidbuchel et al. (11a)</td>
<td>Cohort of patients with ablation of atrial flutter</td>
<td>31 mixed sports</td>
<td>129</td>
<td>Incidence</td>
<td>55 ± 10</td>
<td>81%</td>
<td>48%</td>
</tr>
<tr>
<td>Aiezer et al. (2)</td>
<td>Retrospective cohort study</td>
<td>2,127 athletes who are 5 to 7 d wk⁻¹ exercisers</td>
<td>6,321 nonexercisers</td>
<td>Relative risk at 3 yr</td>
<td>52.1 vs 52.6 controls</td>
<td>RR, 1.74; CI, 1.23 to 2.47</td>
<td></td>
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<tr>
<td>Mozaffarian et al. (17)</td>
<td>Prospective case control</td>
<td>1,877 adults ≥65 yr with moderate exercise</td>
<td>477</td>
<td>Hazard ratio</td>
<td>72 ± 5</td>
<td>HR, 0.72; CI, 0.58 to 0.89</td>
<td></td>
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<tr>
<td>Pelliccia et al. (18)</td>
<td>Prospective cohort study</td>
<td>1,777 mixed sports</td>
<td>Prevalence of AF</td>
<td>24 ± 6</td>
<td>0.3%</td>
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HR, hazard ratio; RR, relative risk.
death. One third of all admissions for cardiac arrhythmias are due to AF. Cognitive dysfunction may be related to AF (5). Studies report that the most typical type of AF in athletes is lone AF. Lone AF is thought generally of as benign even though results reporting mortality data and stroke data in athletes with AF is scarce and inconsistent (21). This could be mainly due to the reduced stroke risk in lone AF which by definition does not have structural heart disease, congestive heart failure, hypertension, diabetes, or prior stroke and in which the age is >75 years. Unfortunately, the quality of life and exercise capacity are impaired in patients with AF (3). In a study by Hoogsteen et al. (12), 60% of the athletes had moderate or severe symptoms from AF and required termination of all sporting activities. A study by McAllister et al. (16) showed that elite collegiate athletes that were unable to participate due to injury could experience severe emotional, mental, and physical effects on quality of life (21). In addition, the ESC acknowledges that treating athletes who have AF may be more challenging than treating the general population (5).

Treatmet guidelines

The two sources most often used for guidance regarding treatment of AF in the athlete are the 36th Bethesda Conference Eligibility Recommendations for Competitive Athletes with Cardiovascular Abnormalities and The ESC’s Guidelines for the management of AF. The four guidelines from the 36th Bethesda Conference for AF (in the absence of Wolff-Parkinson-White syndrome) and the five recommendations made by the ESC specifically for AF in athletes are listed in Table 2. Challenges achieving these guidelines for the athlete and their physician often arise from controlling their rate with activity, the presence of symptoms of hemodynamic impairment with activity, and disqualification from contact activities due to anticoagulation. It is noteworthy that the current Bethesda Guidelines do not make any participation guidelines based on antiarrhythmic treatment but purely focus on maintaining an appropriate ventricular rate with their chosen treatment. β-blockers are not tolerated well (or even prohibited), and digoxin or non-dihydropyridine calcium antagonists will not be potent enough to slow heart rate in exertional AF (5). If the patients are cleared to participate, they may be impaired significantly in their ability to compete (14). Furlanello et al. (10) documented a significantly reduced maximal effort capacity in athletes with AF of 176 ± 21 W compared with 207 ± 43 W in athletes without AF.

Initial evaluation for AF should start with a thorough history documenting details defining the type of AF and associated symptoms and also try to discover predispositions or triggers for AF such as hyperthyroidism, ischemic heart disease, alcohol use, sleep deprivation, emotional stress, and use of stimulants like caffeine or cocaine (11,19,21). This should include a family history since reports of genetic transmission of AF have been noted (11). All patients with AF should receive an electrocardiogram (ECG), transthoracic echocardiogram, and blood tests for thyroid, renal, and hepatic function (5,11,19,21). Additional testing with stress testing would be recommended if ischemia were a potential etiology (11). Coumel (8) stated that vagal AF and adrenergic paroxysmal AF are rather easy to identify by the clinical history and the heart rate changes prior to the arrhythmia

Table 2.

<table>
<thead>
<tr>
<th>Treatment guidelines for AF in athletes (23).</th>
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<tr>
<td><strong>36th Bethesda Conference Eligibility Recommendations</strong></td>
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<tr>
<td>• 1. Athletes with asymptomatic AF in the absence of structural heart disease who maintain a ventricular rate that increases and slows appropriately and is comparable with that of a normal sinus response in relation to the level of activity, while receiving no therapy or therapy with AV nodal-blocking drugs, can participate in all competitive sports. Note that the use of β-blockers is prohibited in some competitive sports (namely, rifle).</td>
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<tr>
<td>• 2. Athletes who have AF in the presence of structural heart disease who maintain a ventricular rate comparable with that of an appropriate sinus tachycardia during physical activity while receiving no therapy or therapy with AV nodal-blocking drugs can participate in sports consistent with the limitations of the structural heart disease.</td>
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<tr>
<td>• 3. Athletes who require anticoagulation should not participate in sports with danger of bodily collision.</td>
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<tr>
<td>• 4. Athletes without structural heart disease who have elimination of AF by an ablation technique, including surgery, may participate in all competitive sports after 4 to 6 wk without a recurrence or after an electrophysiologic study has confirmed noninducibility (20).</td>
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**ESC Recommendations**

• 1. When a “pill-in-the-pocket” approach with sodium channel blockers is used, sport cessation should be considered for as long as the arrhythmia persists and until 1 to 2 half-lives of the antiarrhythmic drug used have elapsed. Class IIa level C evidence. |
• 2. Isthmus ablation should be considered in competitive or leisure-time athletes with documented atrial flutter, especially when therapy with flecainide or propafenone is intended. Class IIa level C evidence. |
• 3. Where appropriate, AF ablation should be considered to prevent recurrent AF in athletes. Class IIa level C evidence. |
• 4. When a specific cause for AF is identified in an athlete (such as hyperthyroidism), it is not recommended to continue participation in competitive or leisure-time sports until correction of the cause. Class III level C evidence. |
• 5. It is not recommended to allow physical sports activity when symptoms due to hemodynamic impairment (such as dizziness) are present. Class III level C evidence (5).

AV, atrioventricular.
onset. He goes further to state that in his experience, the clinical pattern of vagally mediated paroxysmal AF never coincides with the existence of any structural heart disease and thus would be the expected pattern in the typical lone AF of the young athlete. Vagally mediated AF is more frequent in men than women; its age of first symptoms appears between ages 30 and 50 years and has a lack of tendency toward permanent AF (8). The usual history for vagally mediated AF is that of weekly episodes, lasting a few hours and occurring predominantly at night with the morning being the most common time for sinus conversion. Close attention would need to be paid to notice that neither physical nor emotional exertion trigger the arrhythmia but that the relaxed period that follows effort or emotional stress frequently coincides with the onset of vagally induced AF (8). Other precipitating factors are postprandial state, rest, and alcohol (8,11,14). Objectively, vagally mediated AF on ECG shows progressive heart rate slowing over a few hours or even a few beats (8). This makes a Holter monitor a useful test in assisting in establishing vagal versus adrenergic AF. Coulmel indicates that adrenergic AF predominates in paroxysmal AF for patients that have an identified heart disease or in disease states such as hyperthyroidism or pheochromocytoma. These attacks occur during daytime, stress, or with exercise and have a diurnal pattern (8,11,14). Identifying vagal versus adrenergic pattern AF has therapeutic implications, in that treatment options such as β-blockers and digoxin are not only not useful in vagally mediated AF but could be considered contraindicated as they may precipitate them and prevent the traditional antiarrhythmic treatment from being effective, whereas flecainide is noted to be effective (8). The 36th Bethesda Guidelines emphasizes that athletes must be able to maintain an appropriate ventricular rate during activity. Because of this requirement, the next imperative study for athletes with AF who desire to compete is exercise testing. Traditional exercise testing protocols are not designed for this purpose in the well-trained athlete, and protocols would have to be tailored individually to achieve the level of stress during anticipated competition.

Treatment will consist of either rate or rhythm control in addition to thromboembolic prevention. Rate control in athletes may prove challenging due to decreased medication efficacy and poorly tolerated adverse effects. For individuals that have no or minimal heart disease, type 1C antiarrhythmic agents such as flecainide or propafenone are recommended as initial antiarrhythmic therapy because these drugs generally are tolerated well and carry relatively little risk of toxicity (11,14,15). For patients with symptomatic recurrent AF that occurs monthly to yearly, an as-needed or “pill-in-the-pocket” approach can be used with these type 1C antiarrhythmics, with one study showing atrial flutter with rapid conduction in only 1/569 episodes and 94% efficacy (5,14). Because of the risk of induction of rapid atrial flutter by flecainide, concomitant use of a β-blocker or calcium channel blocker is recommended generally (15). The use of amiodarone is not advised because of the young age of the athletes and the long-term toxicity of amiodarone (15).

Anticoagulation recommendations for AF in the athlete are the same as in the nonathlete and are based on the risk of thromboembolism. However, given the preponderance of lone AF and lack of thrombotic risk factors, the indication for anticoagulation in the athlete with AF tends to be lower than the general population. An initial screen of risk factors can be done using the CHADS2 score. The patient would receive one point each for cardiac failure, hypertension, age ≥75 years, or diabetes and two points for a prior stroke or transient ischemic attack (TIA). Oral anticoagulation (OAC) therapy with a vitamin K antagonist is recommended for a score ≥2 (5,11). If the CHADS2 score is 0 to 1, it is recommended to use a more comprehensive approach such as the CHA2DS2-VASc score. In this system, the patient receives 2 points for “major” risk factors of previous stroke, TIA, or thromboembolism and for age ≥75 years but receives 1 point for clinically relevant nonmajor risk factors such as heart failure, hypertension, diabetes, vascular disease (prior myocardial infarction, peripheral artery disease, or aortic plaque), age 65 to 74 years, and female sex. In this system, it is recommended that a score of ≥2 be treated with OAC, a score of 1 be treated preferably with OAC over aspirin 75 to 325 mg, and a score of 0 be treated preferably with no antithrombotic therapy over aspirin 75 to 325 mg (5). Guidelines regarding anticoagulation and cardioversion in the athlete remain the same as in the nonathlete. For patients with AF of unknown duration or >48 h, OAC therapy is recommended for at least 3 wk prior to and for 4 wk after chemical or electrical cardioversion (5). However, there may be a high risk of recurrence of AF after cardioversion in the athlete (21).

The value of RFCA treatment in lone AF is emerging, and there are some data proving its efficacy in athletes (14,21). A study by Furlanello et al. (10) treated 20 athletes that had disabling palpitations from idiopathic drug-refractory AF with two pulmonary vein isolation (PVI) RFCA sessions. At 3 months, Furlanello found that 18/20 (90%) of the athletes were free of AF and 2/20 (10%) reported episodes of only minutes. Postablation maximal exercise capacity significantly improved by 40 W. In this study, all 20 patients had improved quality of life parameters, and all athletes obtained reeligibility after RFCA. Another study by Koopman et al. (13) compared 94 athletes with AF (excluding long-standing persistent and permanent AF or left atrial diameter ≥55 mm) with 41 nonathletes that underwent PVI RFCA and found that at 3 years, after a similar average of procedures (1.2 ± 0.4), 84.5% of athletes compared with 87.3% of nonathletes were free from AF. Calvo et al. (4) performed circumferential pulmonary vein ablation (CPVA) on 182 patients (42 of which were endurance athletes) that were referred for symptomatic drug-refractory AF. At 1 year after a single CPVA, 59% of lone AF athletes were free of AF compared with 48% of controls, and after repeat ablation, those free from AF increased to 81% and 63%, respectively. This study also identified left atrial size and long-standing AF as the only independent predictors for recurrence after ablation (4). A report of the Heart Rhythm Society Task Force on Catheter and Surgical Ablation of Atrial Fibrillation, developed in partnership with the European Heart Rhythm Association, a registered branch of the ESC and the European Cardiac Arrhythmia Society, and in collaboration with the ACC, AHA, the Asia Pacific Heart Rhythm Society, and the Society of Thoracic Surgeons
devised the following Consensus indications for catheter ablation of AF (3):

- Symptomatic AF refractory or intolerant to at least one class 1 or 3 antiarrhythmic medication

Paroxysmal AF: catheter ablation is recommended (class I level A evidence)

Persistent AF: catheter ablation is reasonable (class IIa level B evidence)

Longstanding persistent AF: catheter ablation may be considered (class IIb level B evidence)

- Symptomatic AF prior to initiation of antiarrhythmic drug therapy with a class 1 or 3 antiarrhythmic agent

Paroxysmal AF: catheter ablation is reasonable (class IIa level B evidence)

Persistent AF: catheter ablation may be considered (class IIb level C evidence)

Longstanding persistent AF: catheter ablation may be considered (class IIb level C evidence)

However, catheter ablation of AF is one of the most complex interventional electrophysiologic procedures, and it is therefore to be expected that the risk associated with AF ablation is higher than for ablation of most other cardiac arrhythmias (3). The first worldwide survey of AF ablation reported that at least 1 major complication was seen in 6% of patients but with only 4 early deaths recorded in 8,745 patients (6). In a retrospective case series by Cappato, 32 deaths (1 per 1,000 patients) were reported during 45,115 procedures in 32,569 patients. Causes of deaths included tamponade in 8 patients (1 later than 30 d), 2 in 5 patients (2 later than 30 d), atrioesophageal fistula in 5 patients, and massive pneumonia in 2 patients. Myocardial infarction, intractable tachycardia, septicemia, sudden respiratory arrest, extracardiac pulmonary vein (PV) perforation, occlusion of both lateral PVs, hemotherax, and anaphylaxis were reported to be responsible for one death each, while asphyxia from tracheal compression secondary to subclavian hematoma, intracranial bleeding, acute respiratory distress syndrome, and esophageal perforation from an intraoperative transesophageal echocardiographic probe were causes of one late death each (6). Cappato also sent a questionnaire to 521 centers from 24 countries in 4 continents gathering data on the safety of catheter ablation of AF for 16,309 patients.

The results showed cardiac tamponade to be the most common complication with a rate of 1.31%, followed by total femoral pseudoaneurysm at 0.93%, transient ischemic attack at 0.71%, total ater-venous fistula at 0.54%, PV stenoses requiring intervention at 0.29%, stroke at 0.23%, and permanent diaphragmatic paralysis at 0.17%. All other complications occurred <0.1% of the time (7).

**Conclusion**

AF continues to have a significant impact on athletes, in particular, endurance athletes. Its etiology is still unclear, and treatment can be challenging. While the mainstays of treatment are type 1C antiarrhythmic medications and risk factor-based anticoagulation, RFCA has emerged as an evidence-based recommended treatment for symptomatic paroxysmal AF refractory to antiarrhythmic medication and even as a reasonable treatment in symptomatic paroxysmal AF prior to initiation of antiarrhythmic therapy.

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**References**


