Today the majority of tachyarrhythmias can be treated with percutaneous ablation.1 To summarize, the ablation procedure consists of transmitting energy (generally radiofrequency) to the distal electrode of a catheter introduced into the heart via a vein or artery. This electrode, once placed at the site chosen by means of electroanatomic references, selectively destroys the substrate of each tachycardia, whether they are abnormal electrical connections, ectopic foci, or isthmus of myocardial tissue. In this manner, percutaneous ablation has become the treatment of choice for curing the majority of patients with Wolf-Parkinson-White syndrome, re-entrant tachycardias via nodal re-entry, atrial tachycardias, atrial flutter, and some ventricular tachycardias.

Nevertheless, in 2 types of arrhythmia it seems to be difficult to establish ablation via catheter as a therapeutic option: these arrhythmias are ventricular fibrillation and atrial fibrillation.

With respect to ventricular fibrillation, this has very recently been treated with ablation in a reduced number of patients but, on a day-to-day basis, the most efficacious and safe treatment for the majority of individuals with the risk of suffering this arrhythmia continues to be the implantable defibrillator.2 With respect to atrial fibrillation, treatment has been and continues to be basically pharmacological, but ablation may be used in the near future, at least in certain patients. This is due to the fact that recent investigations have shown that atrial fibrillation may have a focal origin, and that if this focus remains active for a long enough period of time it can change the electrical properties of the atrial myocardium in such a way that the atrial fibrillation may continue to exist despite the disappearance of the focus.3-5 This phenomenon is called atrial remodeling and, in clinical practice, it is attributed to the transformation, with the passage of time, of an atrial fibrillation that was initially focal in nature into chronic atrial fibrillation in which, as has been observed previously, multiple activation sources coexist erratically in the atria, maintaining the arrhythmia.5-7

When atrial fibrillation is focal, the foci are located in small areas of the myocardial tissue that are generally found in the origin of the pulmonary veins, or, less frequently, in other atrial areas, in the vena cava, or in the coronary sinus. From these areas surge bursts of very rapid and irregular electrical activity that initiate and sometimes maintain atrial fibrillation. This has been proven by eliminating the electrical activity in these areas and verifying the subsequent disappearance of atrial fibrillation.3-4

The article by Silva et al published in this issue of the REVISTA ESPAÑOLA DE CARDIOLOGÍA is presumably the first study published in Spanish in which treatment via radiofrequency ablation is described in 33 patients who presented with frequent symptomatic episodes (daily episodes in 70% of the patients) of atrial fibrillation.8 The mean age of the patients was 51 years, the majority of patients did not have structural heart disease, and various anti-arrhythmia drugs had been ineffective for all of them. In this group of patients, the authors decided to directly isolate all the pulmonary veins with electrical activity (59 of the 115 pulmonary veins explored), regardless of whether there was evidence of abnormal electrical activity or not. In order to carry out the isolation of the vessels, selective radiofrequency was applied to the areas in which they presumably identified, via a circular catheter located near the ostium of each pulmonary vein, greater precocity of the potential of said vein. They performed, therefore, segmental ablations rather than completely circular ablations around the pulmonary veins. In this manner, the authors were able to isolate electrically 58 of the 59 veins they attempted to ablate, without the occurrence of any significant complications. Following a mean followup period of 6 months, only 8 patients had new episodes of atrial fibrillation and all but 2 patients, clinical situations clearly improved. It is interesting to note the observation made in 19 patients:
anti-arrhythmia drugs that had failed previously were efficacious following the procedure in controlling atrial fibrillation crises.

These results are similar to those recently published by other authors and highlight the fact that percutaneous ablation of atrial fibrillation is viable, efficacious, and safe, even though the methods used may be different.\(^3\)\(^4\)\(^9\) Although some authors defend the practice of indiscriminately isolating all the pulmonary veins, others recommend acting solely on the responsible pulmonary vein. In the same manner, agreement does not exist as to whether the energy should be applied to the atrium surrounding the focus, or directly to the focus in the interior of the pulmonary vein. What is certain is that with the difficulty in extracting conclusions from the preliminary series published, the success of the procedure appears to depend above all on the proper selection of candidates for the ablation procedure. The characteristic Holter of patients with focal atrial fibrillation reveals frequent atrial extrasystoles and series of rapid atrial tachycardia, some of which degenerate into atrial fibrillation that is self-limited after a variable period of time. In these cases, the result of ablation may be excellent, as was seen in the Hospital Clínico de Barcelona series presented in this issue of the REVISTA. In extreme contrast would be the patients with chronic atrial fibrillation, in whom with the underlying remodeled atrium, once the sinus rhythm has been re-established, a single extrasystole from any origin would be sufficient to trigger a new episode of persistent atrial fibrillation. In conclusion, at a time when some articles appear to demonstrate the lack of benefits of a strategy directed toward maintaining the sinus rhythm through repeated cardioversion and anti-arrhythmia drugs,\(^12\) we must remember that percutaneous ablation should be considered a therapeutic option of choice in certain patients with symptomatic and relapsing atrial fibrillation, despite medical treatment. And this is only the beginning, as, probably, in the near future our knowledge of the physiopathology of this arrhythmia will increase, ablation techniques will be optimized, and the indications for this procedure will be extended. Given the good prospects, we cannot deny that, in the not very distant future, as we can now cure arrhythmias, we will be able to cure atrial fibrillation.

REFERENCES


