Arrhythmia recurrence after ablation of atrial fibrillation: should we be concerned about sleep apnoea?

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This editorial refers to ‘Low efficacy of atrial fibrillation ablation in severe obstructive sleep apnoea patients’ by M. Matiello et al., on page 1084.

In recent years, cardiology research has learned to put its interest in some entities that have long been regarded more as annoyances than diseases that need aggressive treatment. Two examples are left bundle branch block in heart failure, which had been regarded merely as a bystander phenomenon indicating severe disease but now is regarded as an aggravating factor in the heart failure syndrome that should be corrected by resynchronization therapy. Another is atrial fibrillation (AF) which was once considered a benign arrhythmia but now is known to be associated with significant morbidity and mortality, and thus strong efforts are made to cure the arrhythmia by catheter ablation. The ‘new kid on the block’ seems to be sleep apnoea. Whereas ‘snoring’ has been regarded simply as something that sometimes frustrates spouses we now realize that both obstructive sleep apnoeas (OSA) and central sleep apnoea are associated with significant morbidity making them an important target for effective therapy.

Matiello et al.1 study the influence of the severity of OSA on success of catheter ablation for AF. The results of this study indicate that (i) 24% (42 of 174) of all patients undergoing AF ablation have some degree of obstructive sleep apnoea (OSA) and central sleep apnoea are associated with significant morbidity making them an important target for effective therapy.

The seemingly high incidence of sleep disorders in patients undergoing AF ablation is not surprising, given an estimated incidence of 25% of OSA in middle-aged men2 and the male preponderance in this study. However, as the authors correctly point out, the results on OSA as a negative predictor of ablation success have some degree of obstructive sleep apnoea and (ii) that the presence of severe OSA, together with an enlarged left atrial (LA) diameter, is an independent predictor of AF ablation failure.

For AF recurrence after ablation, there have been many baseline differences between the group with severe OSA and other groups that may explain why these patients had a higher recurrence rate after ablation: patients with severe OSA were older, had a higher incidence of persistent or long-standing persistent AF, a higher body mass index, and a higher incidence of hypertension and structural heart disease. Although some of these differences did not reach the level of statistical significance in this rather small group of patients, the impression remains that patients with severe OSA in this study were sicker than those with low risk of OSA and had a more advanced AF substrate. This is not surprising because OSA is associated with obesity, older age, and the presence of coronary artery disease. We do not know what type of structural heart disease was present in the different groups. This would be an important piece of information because, for example, heart failure as an underlying heart disease may be associated with more atrial fibrosis and thus a more difficult substrate for AF ablation.3 It is also important to note that it is difficult to separate OSA from hypertension as a predictor for AF recurrence after ablation because OSA has also been shown to be an independent risk factor for hypertension.4

The authors state that treatment of OSA with continuous positive airway pressure (CPAP) did not affect the results; they assume that once an atrial substrate for AF has developed, treatment of underlying disease does no longer influence AF burden and thus postulate that CPAP should be initiated early enough to prevent AF from occurring. This seems a likely scenario and would represent yet another piece of information showing the necessity for early intervention in AF and its underlying aetiologies or modulating factors before the vicious circle of atrial remodelling has led to irreversible structural changes of the atrial myocardium. However, CPAP is not the only treatment option for OSA. Weight reduction5 or cessation of alcohol consumption may influence the severity of OSA and thus can have an influence on the recurrence of AF episodes. In fact, alcohol per se is known to be...
a triggering factor in paroxysmal atrial fibrillation. The paper does not provide data on any of these variables over the course of the study. Moreover, antiarrhythmic drugs are frequently prescribed after AF ablation. Although the authors used strict criteria for the definition of ablation success, i.e. freedom of AF recurrence without antiarrhythmic drugs, they do not provide any data on how many patients nevertheless received antiarrhythmic agents throughout the trial and what drugs were administered.

The ablation approach in this study was not only confined to pulmonary venous isolation, but also included linear ablation at the LA roof and the posterior wall. Although this approach may offer some advantage with regard to ablation success in patients with persistent and long-standing, persistent AF, it bears the problem that linear lesions frequently have gaps which may be the substrate for LA macrore-entrant tachycardias. In particular, in patients with paroxysmal AF, this type of substrate modification may not be necessary and may even be pro-arrhythmic. The authors did not distinguish between LA flutter or a true AF recurrence in their analysis. It is unclear how a limitation of the ablation approach to pulmonary vein isolation without linear lesions or a more individualized approach according to the arrhythmia type would have influenced the results.

Despite these limitations, this study sheds some important light on the fact that underlying disease should not be overlooked in patients with AF presenting for ablation. There may be many triggering factors such as OSA that are generally being regarded as unimportant (just as AF itself has been regarded in the past), but may in fact significantly influence success of this invasive procedure. Other candidates include uncontrolled hypertension or heart failure, pulmonary disease, subclinical thyroid function abnormalities, or drugs. Despite the exciting progress, AF ablation has made in recent years, the still high recurrence rate should teach us that treatment should probably not be purely anatomical but should also take into account various intrinsic and extrinsic modulators of arrhythmogenesis.

One can only speculate about the reasons why the presence of severe OSA can be a negative predictor for AF ablation success. It has been shown that blood oxygen desaturation during apnoea episodes may lead to an adrenergic response that may, in turn, activate triggers of AF. Not only systemic but also pulmonary hypertension can result from OSA that may eventually lead to right atrial distension causing initiation of AF in the right atrium rather than the pulmonary veins. No data on the right-sided heart chambers are provided in this study, although the authors also speculate that other foci than pulmonary venous may be responsible for the low success rate in this particular group of patients.

In summary, this small but well-designed study underscores the importance of underlying factors that may promote AF recurrence after left atrial ablation procedures. Although these data need confirmation in larger trials, one should probably treat OSA as early and as aggressive as possible before AF develops and be prepared that these patients may pose a challenge to a procedure that has raised such great enthusiasm among electrophysiologists in recent years. It may well be that, in the future, we will need a more individualized approach to AF ablation which takes into account the underlying aetiology rather than performing the same ablation lesions in every patient no matter what the cause of AF is.

Conflict of interest: none declared.

References