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Editorial
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The Deleterious Effect of Atrial Fibrillation and Its Association with Mortality in Non-Responders to Cardiac Resynchronization Therapy

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Editorial

The well known complexity of the mechanism for developing atrial fibrillation (AF) accounts for the failure and relative success of the different therapeutic maneuvers in the management of AF. Despite medical improvements made in recent years, patients with heart failure (HF) are at increased risk of developing AF [1-3]. AF is known to worsen the clinical course of HF through multiple mechanisms including rapid ventricular response, irregularity of ventricular rhythm, loss of organized atrial contribution to cardiac output, and in some cases, tachycardia-induced cardiomyopathy. The prevalence of AF, in patients with HF, increases with the severity of the disease reaching up to 50% in advanced cases. In these patients with HF, AF is an independent predictor of morbidity and mortality increasing the risk of death and hospitalization in 76% [4-7].

As a result of more successful recognition and treatment of cardiovascular risk factors and diseases, mortality continues to decrease favoring an increase in the proportion of elderly population. It has been observed that normal histological changes in the atrial muscle occur with advancing age. These changes include a reduction in the number of myocardial cells within the sinus node, a generalized loss of atrial myocardial fibers in the nearness of the internodal tracts, as well as an increase in the quantity of connective tissue which leads to an apparent loss of myocardial fiber continuity, atrial electrophysiological changes, and increased incidence of AF [8-14]. Despite the excellent results obtained with some of the pharmacological agents in the treatment of HF and AF, the optimal medical treatment failed in the intention to improve symptoms and quality of life in certain patients with severe HF. Thus, the necessity to utilize cardiac devices emerges facing the failure of optimal medical treatment in order to achieve hemodynamic improvement and correction of the physio pathological alterations. In these patients, cardiac resynchronization therapy (CRT) can reduce the interventricular and intraventricular mechanical dissynchrony produced by left bundle branch block. Indeed, the simultaneous electric stimulation of both ventricles with CRT results in a significant hemodynamic improvement restoring a more homogeneous contraction pattern. It has been shown that CRT increases the left ventricular filling time, decreases septal disquinesia and mitral regurgitation, allowing a hemodynamic improvement [15-17]. These beneficial effects are, apparently dependent on continuous bi-ventricular stimulation since interruption of electric stimulation produce a progressive but not immediate loss of effect. Therefore, CRT reverts the ventricular reverse remodeling produced by chronic heart failure, and it is suggested that improvement in mechanical synchrony is the predominant mechanism. However, not all patients respond well to CRT. There are 30 to 40% of non-responders after successful implantation of a CRT device, and the most common reasons for interruption of CRT are the development of AF (18%) and loss of left ventricular capture (10%). Nevertheless, CRT can be re-instituted in a high proportion of patients so that only 5% of patients who successfully undergo implantation of a CRT device permanently lose CRT. Almost one fifth of patients who undergo successful implantation of a defibrillator capable of delivering CRT experience an AF with a rapid ventricular response, which at least temporarily results in the inability to deliver CRT.

Predictors of interruption of CRT as the result of the development of AF in the HF population include a previous history of AF, a relatively slow resting heart rate, and the absence of therapy with both beta-blockers and angiotensin converting enzyme (ACE) inhibitors [17]. These findings are consistent with the analysis of the SOLVD study which found that treatment with enalapril markedly reduces the risk of development of AF in patients with left ventricular dysfunction [18]. Therefore, although it is not clear whether the use of both beta-blockers

and ACE inhibitors directly influence the effectiveness of CRT, their use appears to improve the ability to deliver CRT. Similar finding were reported with angiotensin receptor antagonists. In the LIFE study, it was demonstrated that losartan reduced the incidence of new onset AF in 33% compared to atenolol despite a similar blood pressure control in both treated groups [19].

The goal should be to eliminate AF since it will improve the ability to deliver CRT. In this regard, it is very useful the atrial fibrillation suppression algorithm in dual-chamber permanent pacemakers. It promotes an atrial stimulation with adequate rates for the patient. It dynamically adjusts the stimulation rates in manner that stimulates the heart slightly over the intrinsic atrial rate regardless of the activity status [20-24]. Because patients with slower heart rates are more likely to develop AF, a dual-chamber rate-modulated pacing mode may reduce interruptions of CRT. The search for better pharmacological maneuvers should continue to provide the help needed to cardiac devices. The incorporation of the AF suppression algorithm to CRT devices may be very useful in eliminating AF, allowing a better performance of the CRT device without interruption. It has been clearly demonstrated that sinus rhythm is associated with a long-term improvement in left ventricular systolic function after AF ablation [25]. Therefore, AF catheter ablation may have a primary role as a rhythm control strategy in the definite treatment of AF in patients with HF [25-27]. There should always be a strong effort to convert and maintain to sinus rhythm by all means, after all, sinus rhythm is a God given rhythm.

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