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 disynchrony 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.

References

1. Carson PE, Johnson GR, Dunkman WB, Fletcher RD, Farrell L, et al. (1993) The influence of atrial fibrillation on prognosis in mild to moderate heart failure: The V-HeFT studies. Circulation 87(6 Suppl): VI102- VI110.
2. Healey JS, Hohnloser SH, Exner DV, Birnie DH, Parkash R, et al. (2012) Cardiac resynchronization therapy in patients with permanent atrial fibrillation: Results from the Resynchronization for Ambulatory Heart Failure Trial (RAFT). Circ Heart Fail 5(5): 566-570.
3. Pozzoli M, Cioffi G, Traversi E, Pinna GD, Cobelli F, et al. (1998) Predictors of primary atrial fibrillation and concomitant clinical and hemodynamic changes in patients with chronic heart failure: A prospective study in 344 patients with baseline sinus rhythm. J Am Coll Cardiol 32(1): 197-204.
4. Dries DL, Exner DV, Gersh BJ, Domanski MJ, Waclawiw MA, et al. (1998) Atrial fibrillation is associated with an increased risk for mortality and heart failure progression in patients with asymptomatic and symptomatic left ventricular systolic dysfunction: A retrospective analysis of the SOLVD trials. J Am Coll Cardiol 32(3): 695-703.
5. Bourassa MG, Gurné O, Bangdiwala SI, Ghali JK, Young JB, et al. (1993) Natural history and patterns of current practice in heart failure. J Am Coll Cardiol 22(4 Suppl A): 14A-19A.
6. Mathew J, Hunsberger S, Fleg J, Mc Sherry F, Williford W, et al. (2000) Incidence, predictive factors, and prognostic significance of supraventricular tachyarrhythmias in congestive heart failure. CHEST 118(4): 914-922.
7. Kanek WB, Abbot RD, Savage DD, McNamara PM (1982) Epidemiologic features of chronic atrial fibrillation: The Framingham study. N Engl J Med 306(17): 1018-1022.
8. Davies MJ, Pomerance A (1972) Pathology of atrial fibrillation in man. Br Heart J 34(5): 520-525.
9. Lev M (1954) Aging changes in the human sinoatrial node. J Geront 9(1): 1-9.
10. Centurion OA, Fukatani M, Konoe A, Tanigawa M, Shimizu A, et al. (1991) Different distribution of abnormal endocardial electrograms within the right atrium in patients with sick sinus syndrome. Br Heart J 66(6): 596-600.
11. Centurion OA, Isomoto S, Shimizu A, Konoe A, Kaibara M, et al. (2003) The effects of aging on atrial endocardial electrograms in patients with paroxysmal atrial fibrillation. Clin Cardiol 26(9): 435-438.
12. Centurion OA, Shimizu A, Isomoto S, Konoe A, Kaibara M, et al. (2005) Influence of advancing age on fractionated right atrial endocardial electrograms. Am J Cardiol 96(2): 239-242.
13. Rensma PL, Allessie MA, Lammers WJ, Bonke FI, Schalij MJ (1988) Length of excitation wave and susceptibility to reentrant atrial arrhythmias in normal conscious dogs. Circ Res 62(2): 395-410.
14. Wiener N, Rosenbuehle A (1946) The mathematical formation of the problem of conduction of impulses in a network of connected excitable elements, specifically in cardiac muscle. Arch Inst Cardiol Mex 16(3): 205-265.
15. Moss AJ, Hall WJ, Cannom DS, Klein H, Brown MW, et al. (2009) Cardiac resynchronization therapy for the prevention of heart-failure events. N Engl J Med 361(14): 1329-1338.
16. Tang AS, Wells GA, Talajic M, Arnold MO, Sheldon R, et al. (2010) Resynchronization-Defibrillation for Ambulatory Heart Failure Trial Investigators. Cardiac resynchronization therapy for mild-to-moderate heart failure. N Engl J Med 363: 2385-2395.
17. Peterson PN, Greiner MA, Qualls LG, Al-Khatib SM, Curtis JP, et al. (2013) QRS duration, bundle-branch block morphology, and outcomes among older patients with heart failure receiving cardiac resynchronization therapy. JAMA 310(6): 617-626.
18. Vermes E, Tardif JC, Bourassa MG, Racine N, Levesque S, et al. (2003) Enalapril decreases the incidence of atrial fibrillation in patients with left ventricular dysfunction. Insight from the SOLVD study. Circulation 107: 2926-2931.
19. Wachtell K, Lehto M, Gerdtz E, Olsen MH, Hornestam B, et al. (2006) Different distribution of abnormal endocardial electrograms within the right atrium in patients with sick sinus syndrome. Br Heart J 66(6): 596-600.
20. Centurion OA, Shimizu A, Konoe A, Kaibara M, et al. (2003) The effects of aging on atrial endocardial electrograms in patients with paroxysmal atrial fibrillation. Clin Cardiol 26(9): 435-438.
23. Centurión OA (2009) Atrial fibrillation complicating congestive heart failure: Electrophysiological aspects and its deleterious effect on cardiac resynchronization therapy. J Atrial Fib 2(1): 37-49.

24. Centurión OA (2014) The Influence of Atrial Fibrillation on Cardiac Resynchronization Therapy. J Cardiol Curr Res 1(2): 00008.

25. Nedios S, Sommer P, Dagres N, Kosiuk J, Arya A, et al. (2014) Long-term follow-up after atrial fibrillation ablation in patients with impaired left ventricular systolic function: The importance of rhythm and rate control. Heart Rhythm 11(3): 344-351.

26. Heijman J, Voigt N, Abu-Taha IH, Dobrev D (2013) Rhythm control of atrial fibrillation in heart failure. Heart Fail Clin 9(4): 407-415.

27. Sairaku A, Nakano Y, Oda N, Uchimura Y, Tokuyama T, et al. (2014) Incomplete cure of tachycardia-induced cardiomyopathy secondary to rapid atrial fibrillation by heart rate control without sinus conversion. J Cardiovasc Electrophysiol 25(10): 1037-1043.