

Editorial

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## Dichotomy Argument, is Atrial Fibrillation Really a Physiological Protective Mechanism in Heart Failure?

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## Editorial

The paper, atrial fibrillation and left ventricle dysfunction relationship (new concept) proposed by Tilman [1], seems to be a very interesting paper showing a new aspect of atrial fibrillation. Their hypothesis suggested that atrial fibrillation is a physiological protective mechanism activated in the conditions of LV dysfunction. The hypothesis results from the comparison of the hydrodynamics of the sinus rhythm and atrial fibrillation. When the pressure in the left atrium (LA)-pulmonary vein (PV)-alveolar capillaries (AC) system increases to a certain critical value in the conditions of LV dysfunction or heart failure, the atrial fibrillation mechanism is activated. Atrial fibrillation terminates mechanical systole of the LA excluding the component of systolic left atrial pressure from the total pressure in the LA-PV-AC system, and of the RA with effect of reducing preload, and thus decrease the threat of the development of pulmonary congestion or edema [1]. Certainly, atrial fibrillation may prevent pulmonary congestion or edema due to a relative decrease of pulmonary arterial blood volume in patients with the decreased left ventricle systolic and/or diastolic function. Further, we had experienced some patients who had acute congestive heart failure which occurred immediately after the successful cardioversion of atrial fibrillation in patients with mitral stenosis many years ago. However, once this physiological protective mechanism has broken and pulmonary congestion has progressed, the loss of the atrial function associated with atrial fibrillation exacerbates heart failure in patients with left ventricular dysfunction as proved by many evidences regardless of the acute or chronic stage [2-4].

It is well known that the prevalence of atrial fibrillation increases with the severity of heart failure based on observations in major heart failure trials [2]. A recent Meta-analysis [3] of 104 eligible cohort studies involving 9,686,513 participants (587,867 with atrial fibrillation) showed that atrial fibrillation is associated with an increased risk of all-cause mortality (relative risk (RR) 1.46, 95% confidence interval (CI) 1.39 -1.54) and an increased risk of cardiovascular and renal disease, especially in heart failure (RR 4.99, 95%CI 3.04-0.22) [3]. In a cohort study [4] using a prospective registry of patients in 47 countries, 15,400 individuals were enrolled to determine the occurrence of death and strokes in this cohort over eight geographical regions 1 year after attending the emergency department, heart failure was the most common cause of death (30%) and stroke caused deaths (8%). Recently, it was proposed that development of new-onset atrial fibrillation identifies hypertensive [5] or atherosclerosis [6] patients increased risk at 2-3 folds of sudden cardiac death. Further, atrial fibrillation increased the mortality risk (Hazzard ratio (HR) 3.24, 95%CI 1.63-6.43) even if patients had implantable cardioverter-defibrillator generators replacement [7].

In comparison of rate control versus atrial fibrillation catheter ablation strategies in patients with atrial fibrillation and heart failure, atrial fibrillation catheter ablation is superior to rate control in improving LV ejection fraction in a meta-analysis of randomized, control trials [8]. They concluded that consideration should be given to atrial fibrillation ablation in patients with heart failure and atrial fibrillation before accepting a rate-control strategy in heart failure patients with persistent or drug-refractory atrial fibrillation. Moreover, AATAC multicenter randomized study [9] showed that catheter ablation (70%, 95%CI 60%-78%) of atrial fibrillation (an average of 1.4 ± 0.6 procedures) is superior to amiodarone (34%, 95%CI 25%-44%) in achieving freedom from atrial fibrillation at long-term follow-up and reducing unplanned hospitalization and mortality in patients with heart failure and persistent AF. Finally, the recently published CASTLE-AF [10] studied patients with symptomatic paroxysmal or persistent atrial fibrillation randomly assigned to undergo either catheter ablation (179 patients) or medical therapy (rate or rhythm control) (184 patients) for atrial fibrillation in addition to adequate guidelines-based therapy for heart failure [10]. All the patients had New York Heart Association class II, III, or IV heart failure, a left ventricular ejection fraction of 35% or less, and an implanted cardioverter-defibrillator. After a median follow-up of 37.8 months, catheter ablation for atrial fibrillation in patients with heart failure was associated with a significantly lower rate of death from any cause (13.4% vs. 25.0%; HR 0.53, 95% CI 0.32-0.86) or hospitalization for worsening heart failure (20.7% vs. 35.9%; HR 0.56,95% CI 0.37 -0.83) than was medical therapy [10]. The effects of atrial fibrillation catheter ablation were beneficial even in old patients, more than 75 years old [11].

These above-mentioned evidences have proved no doubt that atrial fibrillation is a harmful condition but not a physiological protective mechanism activated in the conditions of LV dysfunction. Although the hypothesis seems to be correct under certain conditions, it filled with contradictions when considering the whole picture of heart failure in patients with left ventricular dysfunction and atrial fibrillation. This may be like a dichotomy paradox of "Aristotle and the theory of tortoise", the quickest runner can never overtake the slowest, since the pursuer must first reach the point where the pursued started, so the slower must always hold a lead in a race [12].

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