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Atrial fibrillation: In the light of new hypothesis

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ABSTRACT

Background: Atrial fibrillation (AF) is the most common cardiac arrhythmia. Many studies have investigated the cause for the development of AF, however, the question remains unanswered.
Methods: A comparison of hydrodynamics between AF and sinus rhythm was performed.
Results: I proposed the hypothesis that atrial fibrillation is a protective physiological mechanism, based on the termination of atrial mechanical systole. This reduces the pressure in the system of the pulmonary veins and alveolar capillaries in pathological situations, and thus reduces the likelihood for development of pulmonary congestion and edema.
Conclusions: The hypothesis is well correlated with the known facts and phenomenons associated with AF, and explains the causes of the onset of AF in different conditions and diseases.

Atrial fibrillation (AF) is the most common arrhythmia in clinical practice. Disease prevalence is increasing at an alarming rate worldwide. AF is also associated with a large escalating cost [1]. Many studies regarding the causes and the mechanisms of the development of AF have been carried out with a large amount of information accumulated, however, there still is significant controversy.

I am proposing a hypothesis which may explain many of the known facts associated with AF that have led to conflicting interpretations, and help in understanding the results of various studies. This hypothesis explains the cause of AF and leads to a new approach to the role of AF in the pathophysiology of left ventricular (LV) dysfunction not only in heart failure (HF) [2], but also in other diseases and conditions leading to LV dysfunction, and may possibly lead to different perspectives on the issue of treatment.

(1) Comparison of AF and sinus rhythm

	AF	Sinus rhythm
Regularity of rhythm	Irregular	Regular
Form of QRS complex	Supraventricular	Supraventricular
Rate due to AVnode function	Normo, tachy, brady	Normo, tachy, brady
Atrial mechanical systole	No	Yes

It is obvious that there are two differences between AF and SINUS RHYTHM. First, AF is an irregular rhythm, which is usually asymptomatic or associated with the subjective discomfort. Second, the essential difference is the termination of atrial mechanical systole that has a significant impact on the hydrodynamics of the heart.

(2) Comparison of the hydrodynamics of AF and sinus rhythm

In a normal heart, a powerful active diastole begins at the termination of left ventricle (LV) systole. There is an increase in volume of the LV with a significant decline in the pressure causing a suction effect that opens the mitral valve (MV) with blood being directed into the cavity of the LV. The left atrial (LA) systole begins 0.12–0.2 s before the ending of the LV diastole, thus compressing the atrial volume and increasing its internal pressure. An algebraic addition therefore occurs by combining the "positive" pressure of LA systole (atrial kick) with the "negative" pressure of the LV diastole, resulting in the end-diastolic pressure (EDP). At the end of the LV diastole, while the MV is still open, the EDP is immediately transmitted and equalized through the communicating system of the LV, LA, pulmonary veins (PV), and alveolar capillaries (AC), as there are no obstacles to spread of the EDP wave. EDP is thus equal to pulmonary wedge pressure (PWP), with a normal range of ~8–12 mmHg.

AF stops mechanical systole of the LA excluding the component of systolic LA pressure from total pressure in the LA–PV–AC system, thus leading to reduced EDP and PWP. The right atrium (RA) is also involved in AF. As is in the case with the LA, there is a termination of the mechanical systole of the right atrium (RA), and a reduction in end-diastolic

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pressure in the right ventricle (RV). Thus at the end of diastole of the RV when the tricuspid valve is still open, pressure of systemic venous inflow to the heart is decreased, resulting in a reduction of the preload. It is known that reduction of the preload is one of the first urgent components in the treatment of pulmonary congestion and edema. Thus, AF leads to a synergy in the hydrodynamic effect, due to the termination of a mechanical systole in both the LA and RA.
It is known that the main manifestation of LV dysfunction (systolic and diastolic) is a significant increase of EDP which is transmitted through the entire intercommunicated system (LV–LA–PV–AC), causing increased PWP with increased likelihood for development of pulmonary congestion and edema. Pulmonary edema is usually present when PWP increases to 20 mmHg. It is logical to assume that the termination of both left and right side mechanical atrial systole, as a result of the development of the AF, creates more favorable, more optimal hydrodynamic conditions for the heart in a status of LV dysfunction, which is a manifestation of many cardiovascular diseases.

(3) I am proposing a HYPOTHESIS that AF acts as the physiological protective mechanism. The main purpose of AF is to eliminate the mechanical systole of the LA in order to reduce the intercommunicated pressure in the system (LA–PV–AC), and of the RA with the effect of reducing preload, and thus prevent pulmonary congestion and edema in the conditions and the diseases which cause to increase this pressure. By means of this hypothesis it is possible to explain a number of the known facts.

Direct correlation AF and heart failure

AF and HF have emerged as new cardiovascular epidemics over the last years. The prevalence of AF in patients with heart failure increases in parallel with the severity of the disease, ranging from 5% in patients with mild, from 10% to 26% among patients with moderate, and up to 50% in patients with severe heart failure [3,4]. It is known that all forms of LV dysfunction leading to HF are accompanied by an increase of EDP and PWP with a threat of developing pulmonary congestion and edema. The hypothesis clearly explains the cause of the development of AF in HF and their direct correlation [2]. Thus the AF can be a marker of advanced HF.

Direct correlation with the diseases

It is known that AF is associated with a number of diseases, most often hypertension (HTN), mitral stenosis (MS), and hyperthyroidism.

(a) Hypertension is one of the most important prevalent contributors to disease and death. AF and HTN often coexist. Because most people have high blood pressure, HTN is the primary risk factor for AF. Cohort studies suggested that HTN was present in 50–53% patients with AF [5]. The main manifestation of HTN is increased peripheral vascular resistance, that leads to hypertrophy of LV with gradual development of LV dysfunction. As it was already noted above LV dysfunction is associated with increased EDP that leads to the onset of AF, that was explained by the hypothesis.

(b) Mitral stenosis is characterized by obstruction to LV inflow at the level of the mitral valve due to structural abnormality of the mitral valve apparatus. As the valve progressively narrows, the resting diastolic mitral valve gradient, and hence LA pressure, increases. This leads to retrograde transmission of LA pressure to intercommunicated system (LA–PV–AC) with a threat to transudation of fluid into the lung interstitium with development of pulmonary congestion and edema. 40% of patients with MS have AF [6]. The hypothesis explains emergence of AF and its direct correlation with MS.

(c) Hyperthyroidism is a disorder that involves excess synthesis and secretion of thyroid hormones by the thyroid gland, which leads to the hypermetabolic condition of thyrotoxicosis. One important symptom in this condition is tachycardia [7]. It is known that prolonged tachycardia leads to tachycardia-mediated cardiomyopathy, that causes LV dysfunction with increased EDP and development of AF. 10–15% of patients with thyrotoxicosis have AF.

Direct correlation AF with age

It is known that age is a major risk factor for all cardiovascular diseases. The ATRIA study projected that the prevalence of AF will increase 2.5-fold by the year 2050, an estimate based on the growing proportion of elderly individuals [8]. Congestive heart failure (CHF) is the most common diagnosis in hospital patients age 65 years and older. As it was noted the above hypothesis completely explains the mechanism of developing AF in HF and other conditions. The prevalence of AF is related to age, so approximately 70% with AF are between 65 and 85 years of age.

(4) Many facts confirm that the termination of mechanical systole of both atriums by the development of AF creates optimal hydrodynamic conditions in the heart in situations when the pressure in the intercommunicated system (LV–LA–PV–AC) increases, and therefore, it appears that the heart "needs" AF:

(a) Several large randomized, controlled clinical trials have been performed in order to compare rate versus rhythm-control strategies in patients with AF (AFFIRM, RACE, STAF, AF-CHF etc.). These studies did not demonstrate any significant benefit of a rhythm-control strategy and actually showed a trend toward harm in the general population of patients compared with rate-control strategy. Some studies concluded that rate-control is more effective and less costly than rhythm-control and should be the initial treatment for AF among patients with coexisting HF [9–11].

(b) In many cases, after successful treatment of AF via rhythm-control (by medications or ablation), there is a spontaneous return of AF. Only 23–64% of patients assigned to rhythm-control treatment remained in sinus rhythm [12,13]. This phenomenon is consistent with the above proposed hypothesis that the heart in the condition of LV dysfunction "needs" AF, which gives him more optimal hydrodynamic benefits.

(c) In clinical practice, it is not uncommon to find the occurrence of pulmonary edema after successful return of the AF to sinus rhythm [14]. Proposed hypothesis explains this phenomenon by the fact that the hydrodynamics with AF is preferable for the heart in this condition.

(d) In the same way it is possible to explain the development of pulmonary edema in cases where a pacemaker was implanted for AF. This is one of the known complications of so-called pacemaker syndrome [15]. In these cases not only desynchronization of atrials and ventricles take place, but also increased pressure in the system LA–PV–AC occurs as a result of imposing atrial regular rhythm instead of the former AF.

(5) There is a lot of research and, accordingly, a lot of controversy regarding the source of AF, but localization of an anatomical or histological substrate as a source of AF has