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Transition from sinus rhythm to atrial fibrillation – A mechanism inducing or delaying pulmonary congestion and edema

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ABSTRACT

Cardiogenic pulmonary edema (PEd) is a life-threatening condition where fluid accumulates in the lungs due to increasing hydrostatic pressure building up in the pulmonary vasculature (PV): veins, venules and capillaries. Atrial fibrillation (AF) is accepted as an arrhythmia which triggers and promotes the pathophysiological processes leading to pulmonary congestion and its final expression: PEd. We propose a different view, where AF is actually a physiological solution temporarily protecting from PEd. We hypothesize that the compliance of the left atrium (LA) increases with the onset of AF. Thus, it is possible that even if the volume of blood within the LA increases due to loss of atrial contraction, the pressure within the LA would still be lower than that prior to AF (because of the increased LA compliance during AF). Decreased LA pressure allows more blood to flow from the PV to the LA, abating the hydrostatic pressure buildup in the PV compartment. The ratio, *R*, between the LA volume gained from the transition to AF provided by the greater LA compliance, and the volume of blood retained in the LA due to loss of atrial contraction, determines the instant pressure in the LA, as AF begins. If *R* is >1, then the LA pressure will instantly decrease with the transition to AF and this may be beneficial in delaying PEd.

Introduction

This medical hypothesis focuses on the role of the left atrium (LA) when atrial fibrillation (AF) commences. Specifically, it is suggested that the transition from normal sinus rhythm (NSR) to AF may be beneficial in delaying the development of pulmonary congestion and edema. The general idea has recently been elegantly presented in the *journal* [1] however the present hypothesis differs and complements the idea. Whereas Tilman et al. base their hypothesis on "considering the heart as a hydrodynamic system" [1], we believe that hydrodynamics reflect the physical part of the events. It is the physiology of the system with the ability to change compliance of vascular compartments during AF which account for the suggested hypothesis. The conditions when this hypothesis holds and when it fails and the significance of heart rate are portrayed.

Background

Cardiac compartments

The left side of the heart is composed of compartments connected in series. Starting at the systemic circulation (and going against the direction of blood flow), the aorta is connected to the left ventricle (LV) via the aortic valve. The LV is connected to the left atrium (LA) via the mitral valve. (Valves throughout this hypothesis are assumed to be competent and unidirectional.) The LA and the pulmonary vasculature (PV, i.e. pulmonary veins, venules, and capillaries) are distinct compartments connected in series without valves separating them. Consequently, change of pressure in either compartment affects the pressure in the other.

Left atrium

Since the LA plays a major role in this hypothesis, several fundamental points are emphasized. Evaluating LA area using echocardiography reveals three main phases during the cardiac cycle [2]. (1) LA filling phase, where the LA serves as a reservoir, starts when

Abbreviations: AF, atrial fibrillation; CMR, cardiac magnetic resonance; CO, cardiac output; HF, heart failure; HFPEF, heart failure with preserved ejection fraction; HFREF, heart failure with reduced ejection fraction; HR, heart rate; LA, left atrium; LA-*C*, left atrial compliance; LA-*P*, left atrial pressure; LA-*V*, left atrial volume; LA-V_{max}, maximum LA-*V*; LA-V_{min}, minimum LA-*V*; LV, left ventricle; LVOT, left ventricular outflow tract; MDCT, multi-detector computed tomography; NSR, normal sinus rhythm; PEd, pulmonary edema; PV, pulmonary vasculature; SV, stroke volume; TTE, trans thoracic echocardiography.

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mitral valve closes and terminates when it opens. This period of time is equal to the duration of LV contraction (systole). The volume of the LA (LA-*V*) is lowest at the time mitral valve closes (LA-V_{min}), whereas it is greatest just before mitral valve opens again (LA-V_{max}). (2) LA emptying phase, where the LA serves as a conduit chamber, starts when mitral valve opens and terminates at a distinct LA-*V* before the onset of LA contraction. This LA-*V* is labeled here: LA-$V_{75\%}$, because approximately 75% of the LA stroke volume (SV, the volume transferred from the LA to the LV per beat) is transferred from the LA to the LV during this phase. (3) LA booster pump phase, where the LA muscle contracts and propels blood into the LV. At the end of this phase LA-*V* equals LA-V_{min}. The duration of phases 2 and 3 is equal to the duration of the LV diastole (approximately 2/3 of the duration of the cardiac cycle at normal HR). It is noted that during phases 1 and 2 LA wall tension is determined by the volume of blood distending the LA. During phase 3 LA wall tension is assumed to be greater than it is during the former phases (1 and 2), due to LA muscle contraction. The latter adds to the wall tension already produced by distention of blood in phase 2. We did not find literature supporting this assumption; however, two points logically favor this assumption. Phase 3 always follows phase 2 in time (during NSR), and wall tension at the end of phase 2 serves as the starting point for further development of wall tension due to LA contraction in phase 3. (For further reading see ref. [3] an elegant investigation of the LA-*V* using cardiac magnetic resonance imaging in healthy young and elderly subjects. Figs. 3 and 4 show LA-*V* as a function of time).

During atrial fibrillation (AF) booster pump phase (phase 3 above) is lost. Consequently, less blood is transferred from the LA to the LV per cardiac cycle, thus more blood is retained in the LA. LA-*V* varies during AF however the values of LA-V_{min} and LA-V_{max} are different from those obtained during NSR (see section evidence related to LA-*V* below).

Left atrial compliance

Compliance of an elastic component is defined by the change in pressure (ΔP) required to cause a change in volume (ΔV), $C = \Delta V/\Delta P$. When a large change in pressure is required to induce a small change in volume the compliance is low, and vice versa. LA compliance, LA-*C*, at a certain volume can be appreciated from the local derivative on the LA-*V* versus LA pressure (LA-*P*) plot (see Fig. 2 in Ref. [4]). Specifically, LA-*C* is greatest at low LA-*V*, and it decreases with the distention of LA walls. We claim, although it is not proven, that the LA-*C* during phase 3 is lower than that during phase 2.

Pathophysiology of pulmonary congestion and edema

In pulmonary congestion and edema fluid shifts from the PV (mainly pulmonary capillaries) into the surrounding supporting tissues (interstitium) and pulmonary alveoli, respectively. Two pathological forces account for the fluid shift: (a) increased hydrostatic pressure within the PV or (b) increased permeability of the pulmonary capillaries. This hypothesis relates to the former force only. Increased hydrostatic pressure within the PV is the result of various scenarios overloading the PV compartment with blood. For example, a sudden rise in the systemic blood pressure increases the resistance against which the LV must contract. This imposes a decrease in LV SV and therefore, more blood is retained in the LV at the end of contraction before the next filling. The excess of blood retained increases the LV pressure according to its compliance. Consequently, the LA-*P* required now for filling the LV must be greater than that required before systemic blood pressure had increased. The increased LA-*P* reflects backwards to the PV increasing the hydrostatic pressure [5].

Atrial fibrillation (AF) has 3 forms: paroxysmal (appears acutely and reverts to NSR spontaneously), persistent (appears acutely and does not revert to NSR spontaneously, but may be converted to NSR by medications or electric shock), and permanent (AF which cannot be converted to NSR) [6]. In this work we refer to the reversible forms only: paroxysmal and persistent.

AF is another cause for pressure buildup in the PV. Briefly, as AF initiates atrial contraction is instantly lost, diminishing the LA SV by as much as 20% [7–8]. Consequently, blood accumulates in the LA, increasing LA pressure, and in turn also increases the pressure within the PV. This process promotes fluid shift from the intravascular space to the interstitium first and then to the alveoli, culminating in pulmonary congestion and edema, respectively [9].

Case vintage

A 70 years old woman is admitted to the emergency room with progressive dyspnea which developed over the last few days, accompanied by palpitations. She also complains of leg edema and waking up at night with difficulty breathing over the last week. Medical history reveals 25 years for hypertension, 10 years of type II diabetes mellitus, and obesity. Her physical examination revealed: normal vital signs except for blood pressure 170/75, end-inspiratory crackles over lung bases, and bilateral leg edema. Blood tests were normal, including troponin level. EKG demonstrated rapid AF, 130 beats per minute, and signs of LV hypertrophy. Her chest X-ray displayed signs of PEd. Patient was treated immediately with oxygen, morphine, and diuretics. After initial improvement heart rate (HR) lowering drugs (rate control), and anti-coagulation therapy was added. Standard trans-thoracic echocardiography (performed 3 days after patient improved and converted spontaneously to NSR) demonstrated: LA enlargement, thickened left ventricular walls with a small LV cavity (concentric hypertrophy), normal LV systolic function (ejection fraction 60%), transmitral flow velocity measurements demonstrated findings consistent with a "pseudonormalization" pattern (diastolic dysfunction grade II) [10]. This clinical case is commonly encountered in internal medicine departments.

Hypothesis

Before presenting the hypothesis several assumptions are made: (1) LA and PV are compliant compartments containing blood, connected in series with no valves separating them apart. (2) Blood is a non-compressible fluid. (3) As a result of (1) and (2), a change in pressure in the LA compartment will instantly affect the pressure in the PV compartment. (4) Mitral valve remains competent during the transition from NSR to AF.

The following scenario may also explain the clinical case. Systemic blood pressure rises (for various common causes such as fluid, salt or medication indiscretion) forcing the LV to contract against a greater resistance at its outflow tract (LVOT). As the resistance increases less blood can be propelled from the LV to the circulation. (Consider the extreme case, where the LVOT is severely compromised, as in severe aortic stenosis. Then, the SV decreases significantly, clinically expressed by a weak palpated pulse, and events of fainting [11]). As SV diminishes, more blood per cardiac cycle accumulates in the compartments upstream to the LVOT that is in the LV, LA, and PV. As blood accumulates, it exerts pressure on the walls of the latter compartments, distending them. The walls of the LA are assumed here to be the "weakest part in the chain" because they are the thinnest. Data show that the risk for AF increases with LA-*V* [12]. Thus, at a certain point of LA distention AF initiates. In addition, the pressures within the LA, and PV compartments rise. This increase in hydrostatic pressure within the PV