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CHARACTERIZING THE CARDIOVASCULAR FUNCTIONS DURING ATRIAL FIBRILLATION THROUGH LUMPED-PARAMETER MODELING

STEFANIA SCARSOGLIO
Department of Mechanical and Aerospace Engineering, Politecnico di Torino
Corso Duca degli Abruzzi 24 Torino, 10129, Italy
stefania.scarsoglio@polito.it

ANDREA GUALA, CARLO CAMPOREALE, LUCA RIDOLFI
Department of Environment, Land and Infrastructure Engineering, Politecnico di Torino
Corso Duca degli Abruzzi 24, Torino, 10129, Italy
andrea.guala@polito.it, carlo.camporeale@polito.it, luca.ridolfi@polito.it

Atrial fibrillation (AF), causing irregular and rapid heartbeats, is the most common arrhythmia. Due to the widespread impact on the population and the disabling symptoms related to rapid heart rate, AF is a subject of growing interest under several aspects: statistical analyses on the heartbeat distributions, risk factors, impact on quality of life, correlation with other cardiac pathologies. However, several key points on the consequences induced by AF on the cardiovascular system are still not completely understood. The proposed work aims at quantifying the impact of AF on the most relevant cardiovascular parameters by means of a lumped-parameter modeling, paying particular attention to the stochastic nature of the irregular heartbeats and the reduced contractility of the heart. The global response leads to a rather impressive overall agreement with the clinical state-of-the-art measures regarding AF: reduced cardiac output with correlated arterial hypotension, as well as higher left atrial volume and pressure values are some of the most representative outcomes emerging during AF. Moreover, new insights on hemodynamic parameters such as cardiac flow rates, which are difficult to measure and almost never offered in literature, are here provided.

Keywords: Atrial fibrillation; lumped-parameter modeling; cardiovascular dynamics.

1. Modeling Description

In the lumped parameterization, the anatomical details are neglected and the cardiovascular system is described through a Windkessel model by a set of electrical components[1]. The lumped model here adopted extends the Windkessel approach to the whole (pulmonary and systemic) circulation system, and is combined to an active atrial and ventricular representation, through four time-varying elastance functions, for the left and right heart chambers. The present model consists of a network of compliances, resistances and inductances describing the pumping heart coupled to the systemic and pulmonary systems, along with an accurate description of the valve motion mechanisms[2]. The viscous effects are taken into account by the resistances, R, the inertial terms are considered by the inductances, L, while the elastic vessel properties are described by the compliances, . Three

cardiovascular variables are involved at each section: the blood flow rate, Q, the volume, V, and the pressure, P.

The main aim of the present work is to characterize the cardiovascular functions during AF with respect to the normal sinus rhythm (NSR). The fibrillated beating is simulated paying particular attention to the stochastic modeling of the irregular heartbeats and the reduced contractility function of the heart. The stochastic modeling of the heartbeat allows to have long fibrillated time series (e.g. 5000 cardiac cycles), thereby yielding statistically significant results.

The proposed approach has a double advantage. First, AF conditions can be analyzed avoiding the presence of other side pathologies, which usually accompany AF. Therefore, the outcomes should be read as purely consequent of a fibrillated cardiac status in a healthy young adult. Second, the main cardiac variables and hemodynamic parameters can all be obtained at the same time, while clinical studies usually focus only on a few of them at a time.

2. Results

In Fig. 1 we first report, through time series, some cardiovascular outcomes showing a striking agreement[3] with the state-of-the-art in vivo data regarding AF. During AF, there is a consistent reduction of both cardiac output (-9%) and stroke volume (-26%), with a consequent moderate arterial hypotension (diastolic: -7%, systolic: -11%), while the mean left atrial pressure increases by 13%.

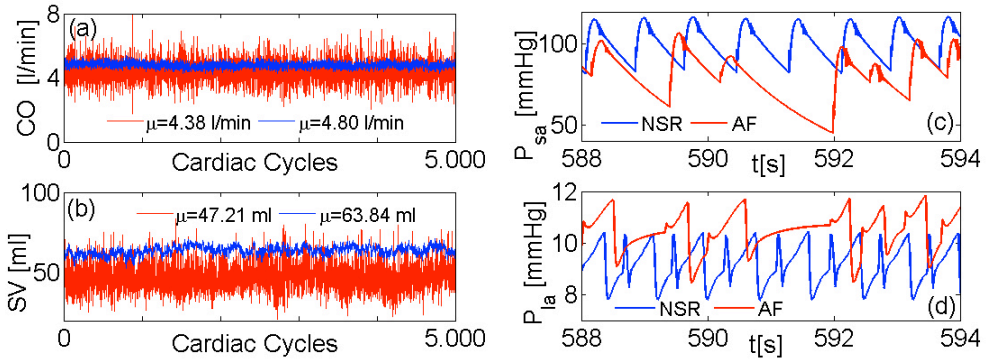


Fig. 1: (a)-(b): Cardiac output (CO) and stroke volume (SV) over 5000 cardiac cycles (average values are indicated with μ). (c)-(d) Systemic arterial (P_{sa}) and left atrial (P_{la}) pressure series. Blue: NSR, red: AF.

Among all the cardiovascular variables obtained through the present model, we here focus on hemodynamic parameters, such as cardiac flow rates, which are not easily measured during AF. In fact, to the best of our knowledge, there are no available trends or data of cardiac flow rates during AF in literature. However, the four valve flow rates can provide precious information to understand the effects of fibrillation events.

The time series of both mitral and tricuspid flows evidence the absence, in the fibrillated configuration, of the peaks due to atrial contraction in late diastole (see Fig. 2, panels a and d). A deeper investigation (see Fig. 2, panels b and e) highlights that short heartbeats lead to higher peaks of regurgitant mitral and tricuspid flows, while longer beats cause a decrease of the reflux (we recall that RR is the temporal range between two consecutive heart beats). For the direct flow, the contrary holds for the tricuspid flow: there is an inverse proportionality between the beat length and the value of direct flow peak (Fig. 2f). For the mitral flow, instead, the sparsity of values impedes to identify a clear trend between RR and the maxima of Q_{mi} (Fig. 2c). Therefore, the greater amount of regurgitant flow due to a rapid beat is in large part compensated by a greater amount of direct flow for the tricuspid valve. The situation is different for the mitral valve, where the increased portion of regurgitant flow during short beats is not systematically accompanied by a higher contribute of direct flow. Although the mean flow rates of mitral and tricuspid flows experience a similar decrease during AF (mitral flow: NSR μ=79.81, AF μ =71.10; tricuspid flow: NSR μ =79.80, AF μ =71.09), the net mitral flow

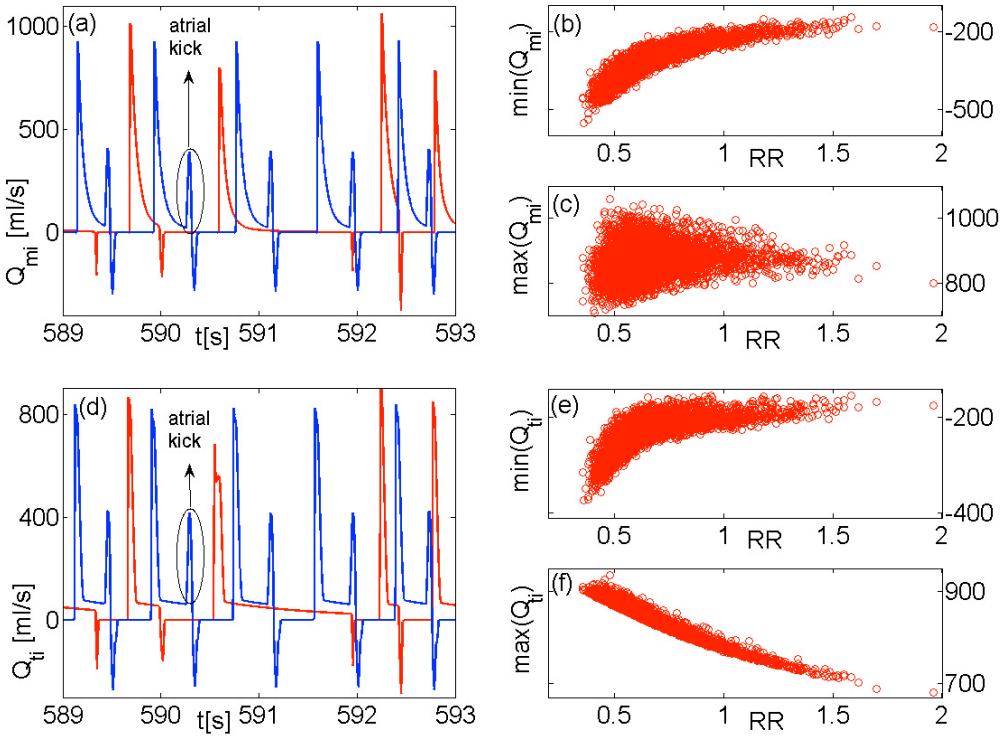
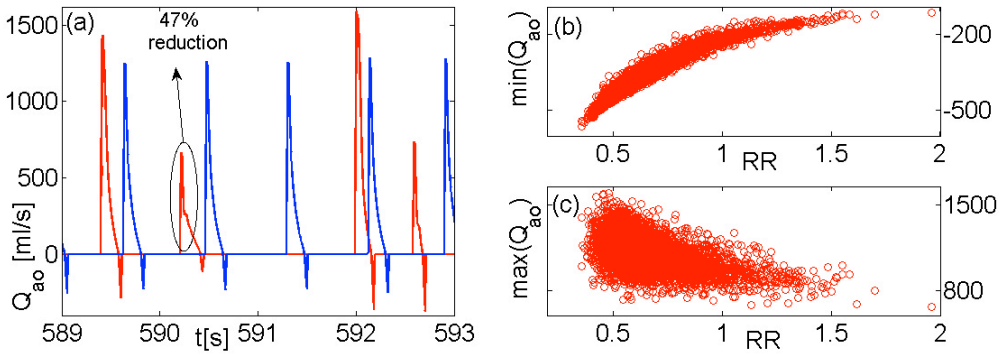


Fig. 2: Mitral (Q_{mi}) and tricuspid (Q_{ti}) flow rates. (a) Q_{mi} series. (b) AF: mitral flow regurgitant peak values as function of the heartbeat, min(Q_{mi})(RR). (c) AF: mitral flow direct peak values as function of the heartbeat, max(Q_{mi})(RR). (d) Q_{ti} series. (e) AF: tricuspid flow regurgitant peak values as function of the heartbeat, min(Q_{ti})(RR). (f) AF: tricuspid flow direct peak values as function of the heartbeat, max(Q_{ti})(RR). Blue: NSR, red: AF.

can be insufficient during rapid heartbeats, leading thereby to significant functional mitral regurgitation, as recently observed[4,5].
For the flows out of the left and right ventricles (aortic and pulmonary valve flows, respectively), the scenario partially reflects what happens in the corresponding atria. For both flows there is a marked positive correlation between the heartbeat length and the regurgitant peak (see Fig. 3, panels b and e). For the direct maximum peaks, the aortic flow rate presents an inverse but more sparse relation between RR and the maxima of Q_{ao} (Fig. 3c), while for the pulmonary flow dispersion of data is lower and the decreasing trend is better highlighted (Fig. 3f). These results may suggest that, due to AF, aortic valve insufficiency is more likely to occur than pulmonary valve insufficiency.

3. Conclusions

The main goal of characterizing the global response of the cardiovascular system during AF has been achieved by means of a lumped-parameter approach. Although some fine details as well as the spatial cardiovascular description are here missing, the present stochastic modeling turns out to be a synthetic and powerful tool for a deeper comprehension of the arrhythmia impact on the whole cardiovascular system. Furthermore, the current modeling can provide new comprehension on hemodynamic parameters (such as, for example, cardiac flow rates), which are difficult to measure and almost never treated in literature. The proposed approach can be exploited to predict the response to AF with the combined presence of altered cardiac conditions (e.g., left atrial appendage clamping), therefore recovering a clinical framework which often occurs in medicine.



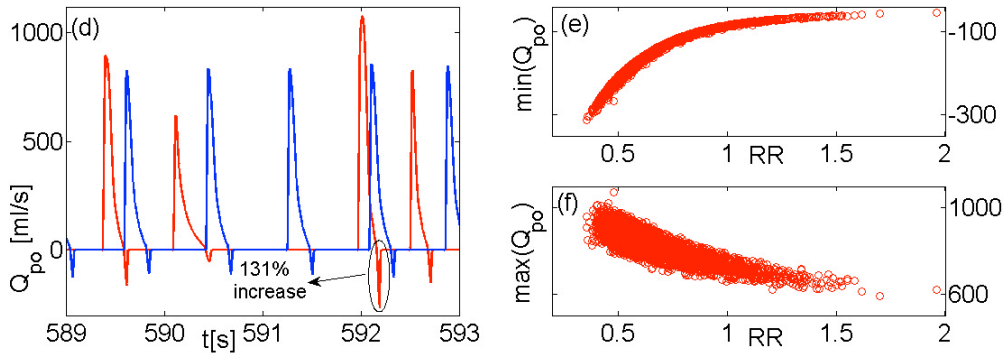


Fig. 3: Aortic (Q_{ao}) and pulmonary (Q_{po}) flow rates. (a) Q_{ao} series. (b) AF: aortic flow regurgitant peak values as function of the heartbeat, min(Q_{ao})(RR). (c) AF: aortic flow direct peak values as function of the heartbeat, max(Q_{ao})(RR). (d) Q_{po} series. (e) AF: pulmonary flow regurgitant peak values as function of the heartbeat, min(Q_{po})(RR). (f) AF: pulmonary flow direct peak values as function of the heartbeat, max(Q_{po})(RR). Blue: NSR, red: AF.

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