# Characterizing the cardiovascular functions during atrial fibrillation through lumped-parameter modeling

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- In the USA and Europe 7 million people are currently affected by AF ⇒ incidence is expected to double within the next 40 years;
- AF is responsible for **substantial morbidity** and **mortality** in the general population;



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- In the USA and Europe 7 million people are currently affected by AF ⇒ incidence is expected to double within the next 40 years;
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- Broad interest: statistical analyses on the heartbeat distributions, risk factors, correlation with other cardiac pathologies.



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### Open key aspects

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- The anatomical and structural complexity of some regions (e.g., right ventricle) makes estimates not always feasible and accurate
  substantial absence of well-established information;
- Presence of other pathologies (hypertension, atrial dilatation, mitral stenosis, ...) ⇒ the specific role of AF is not easily detectable and distinguishable. Side pathology is cause or effect?



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### Motivation and Goal

 Understand and quantify, through a stochastic modeling approach, the impact of paroxysmal AF on the cardiovascular system of a healthy young adult (structural remodeling effects neglected);



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- The main cardiac parameters can all be obtained at the same time, while clinical studies usually focus only on a few of them at a time ⇒ overall good agreement with the clinical state-of-theart measures;



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- The main cardiac parameters can all be obtained at the same time, while clinical studies usually focus only on a few of them at a time ⇒ overall good agreement with the clinical state-of-theart measures;
- Accurate statistical analysis of the cardiovascular dynamics, which is not easily accomplished by in vivo measurements.



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Mathematical framework Cardiac cycle simulation

### Cardiovascular scheme



Mathematical framework Cardiac cycle simulation

# Physiologic and fibrillated beating

#### • Normal Sinus Rhythm (NSR)

- RR extracted from a correlated pink Gaussian distribution;
- Time varying (right and left) atrial elastance;
- Full left ventricular contractility;



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Hemodynamic parameters Systemic arterial pressure Left atrium Flow rates

## Left ventricle





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## Left ventricle



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### Arterial pressure: time series and statistics



P <sub>sas</sub> [mmHg]	Mean	Systolic	Diastolic	Pulsatile
NSR	99.52	116.22	83.24	32.99
AF	89.12	103.66	77.24	26.42

Scarsoglio et al., Med. & Biol. Eng. & Comput., 2014 (in press).



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### Pressure and volume behaviour



V <sub>la</sub> [ml]	Mean	End-Systolic	End-Diastolic
NSR	56.53	64.41	55.37
AF	65.95	71.41	68.84



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Hemodynamic parameters Systemic arterial pressure Left atrium Flow rates

### Left heart: mitral and aortic flows



*Q<sub>mi</sub>* and *Q<sub>ao</sub>*: the increased portion of regurgitant flow during short beats is not systematically accompanied by a higher contribute of direct flow ⇒ possible functional mitral regurgitation and aortic valve insufficiency;



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Hemodynamic parameters Systemic arterial pressure Left atrium Flow rates

## Right heart: tricuspid and pulmonary flows



*Q<sub>ti</sub>* and *Q<sub>po</sub>*: the greater amount of regurgitant flow due to a rapid beat is in large part compensated by a greater amount of direct flow ⇒ right valves insufficiency is less likely to occur.



Conclusions

### **Discussion and Conclusive Remarks**

 First attempt to quantify, through a stochastic modeling, the role of acute AF on the whole cardiovascular system;



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  - Anatomical remodeling due to long-term effects and short-term regulation effects of the baroreceptor mechanism are absent;
  - Reduced contractility of the right ventricle and the ventricular interaction should be properly accounted for;



Conclusions

- First attempt to quantify, through a stochastic modeling, the role of acute AF on the whole cardiovascular system;
  - Anatomical remodeling due to long-term effects and short-term regulation effects of the baroreceptor mechanism are absent;
  - Reduced contractility of the right ventricle and the ventricular interaction should be properly accounted for;
- Isolate single cause-effect relations, a thing which is not possible in real medical monitoring:
  - the drops of systemic arterial pressure and cardiac output are entirely induced by the reduced ventricular contractility during AF;
  - the decrease of the ejection fraction and the LA enlargement are primarily caused by the irregular heart rate;



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 Moderate systemic hypotension and left atrial enlargement should be interpreted as pure consequences of AF alone and not induced by other pathologies;



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- **New information** on hemodynamic parameters (e.g., flow rates), difficult to measure and almost never treated in literature;



Conclusions

- Moderate systemic hypotension and left atrial enlargement should be interpreted as pure consequences of AF alone and not induced by other pathologies;
- Accurate statistical description of the cardiovascular dynamics, a task which is rarely accomplished by in vivo measurements;
- **New information** on hemodynamic parameters (e.g., flow rates), difficult to measure and almost never treated in literature;
- Future work:
  - Response to AF with the combined presence of altered cardiac conditions (e.g., left atrial appendage clamping);
  - Modeling response to real beating series for both NSR and AF.

