Effects of Atrial Fibrillation on the Coronary Flow at Different Heart Rates: A Computational Approach

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Introduction

- Atrial fibrillation (AF) is the most common form of arrhythmia, with an estimated number of 14-17 million patients in 2030 in Europe only [1].
- The AF-induced alterations can promote extra cardiovascular disorders, apart from potential concomitant pathologies which usually arise with AF (e.g. hypertension, valvular disorders, etc…).
- The abnormalities of myocardial perfusion are possible in AF patients, even in absence of coronary artery disease [2].
- The relation between AF and coronary flow alterations is here investigated, since a clear understanding on this topic is still lacking.

Methods

Simulating AF

at 5 different mean heart rates (HR): 50, 70, 90, 110, 130 bpm

Each RR sequence forces the model.

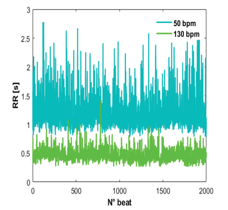


Fig.3: 2000 fibrillated heartbeat periods (RR) at 50 and 130 bpm [6].

Tab 1: (Columns II and III) Mean values ± standard deviations and coefficients of variation (in brackets) of RR_{sys} and RR_{dia} for each simulated frequency (50, 70, 90, 110, 130 bpm). (Column IV) Ratio between mean values of RR_{dia} and RR_{sys}.

HR [bpm]	RR_{sys} [s]	RR_{dia} [s]	RR_{dia}/RR_{sys}
50	0.41±0.049 (0.12)	0.79±0.26 (0.33)	1.93
70	0.35±0.034 (0.097)	0.50±0.17 (0.34)	1.43
90	0.32±0.026 (0.081)	0.35±0.14 (0.35)	1.09
110	0.31±0.019 (0.061)	0.24±0.11 (0.32)	0.77
130	0.30±0.019 (0.063)	0.17±0.096 (0.56)	0.57

Multiscale mathematical models

ARTERIAL NETWORK

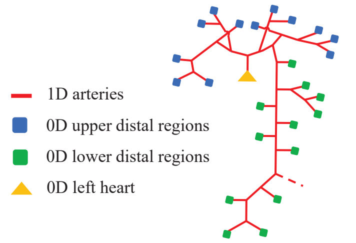


Fig.1: Scheme of the arterial network. It is closed by the left heart and the upper/lower body capillary units [3,4].

LEFT CORONARY CIRCULATION

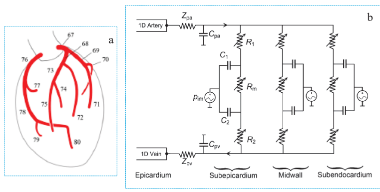


Fig.2: (a) 1D Left-coronary arteries included in the model. (b) Representation of the 0D model closing each coronary artery. Both figures are extracted from [5].

Hemodynamic parameters

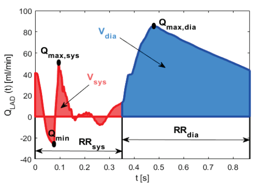


Fig.4: Example of fibrillated left anterior descending (LAD) flow rate waveform. $Q_{max,sys}$ and $Q_{max,dia}$ are the maximal flow rates during the systolic RR_{sys} and diastolic RR_{dia} periods, Q_{min} is the minimum flow rate, V_{sys} (red) and V_{dia} (blue) are the blood volumes flowing through the chosen LAD section within RR_{sys} and RR_{dia}.

$$V_{sys} = \int_{RR_{sys}} Q_{LAD}(t)dt$$

$$V_{dia} = \int_{RR_{dia}} Q_{LAD}(t)dt$$

$$SV = V_{sys} + V_{dia}$$

$$CBF = SV \ X \ HR$$

$$CPP = PA_{dia}^{*} - EDLVP^{**}$$

* Aortic diastolic pressure
** End-diastolic left-ventricular pressure

Results

Tab 2: Mean values ± standard deviations and coefficients of variation (in brackets) of $Q_{max,dia}$ and Q_{min} for each simulated cardiac frequency (50, 70, 90, 110, 130 bpm) in AF.

HR [bpm]	$Q_{max,sys}$ [ml/min]	$Q_{max,dia}$ [ml/min]	Q_{min} [ml/min]
50	48.97±5.36 (0.11)	73.92±7.07 (0.096)	-27.65±3.54 (-0.13)
70	56.36±10.65 (0.19)	86.75±8.60 (0.099)	-28.06±3.03 (-0.11)
90	71.11±15.07 (0.21)	94.30±9.70 (0.10)	-27.86±3.92 (-0.14)
110	84.68±15.08 (0.18)	96.61±11.15 (0.12)	-27.49±3.81 (-0.14)
130	87.41±14.85 (0.17)	89.52±17.24 (0.19)	-25.93±5.38 (-0.21)

Mean values, standard deviations and coefficients of variation of $Q_{max,sys}$ and $Q_{max,dia}$ increase moving towards the high frequency range. Q_{min} remains almost constant with HR.

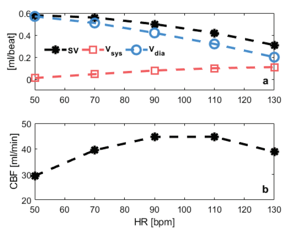
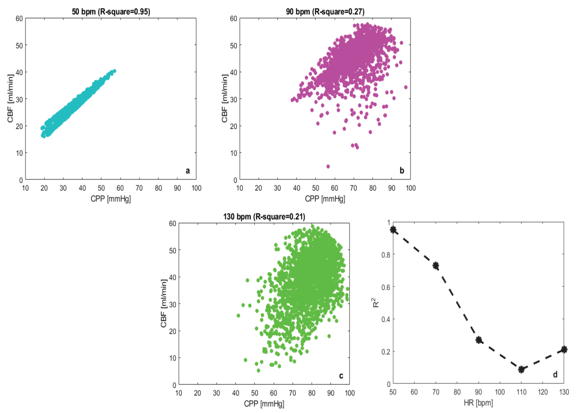


Fig. 5: (a) Mean behaviours of SV (LAD stroke volume), V_{sys} (LAD blood volume during RR_{sys}), V_{dia} (LAD blood volume during RR_{dia}). (b) Dependence of CBF (coronary blood flow) on HR.

In the range 50-130 bpm (Fig. 5a), SV decades with HR, V_{sys} increases from 0.01 to 0.11 ml/beat, and V_{dia} decreases from 0.57 to 0.20 ml/beat. CBF (Fig. 5b) exhibits a non-monotonic behaviour: it grows of the 34% in the range 50-90 bpm, remains almost constant between 90 and 110 bpm, and reaches the maximum value (44.8 ml/min) at 110 bpm. For HRs higher than 110 bpm, CBF decreases up to 38.8 ml/min.

CPP is often used in literature as a surrogate measure for myocardial perfusion [7]. Thus, we are interested in verifying if it is possible to consider CPP as a good estimate of CBF, even in AF.



Tab 6: (a-c) Scatter plots between CPP and CBF at a LAD section for different HRs, with the corresponding R-square values. (d) R-square behaviour of the scatter plots between CBF and CPP with HR.

CBF positively correlates with CPP up to 70 bpm, but data become sparse and very low correlation is detectable for higher HRs (Fig. 6a-c). In fact, there is a dramatic drop of the R-square (R^2) values with HR (Fig. 6d).

Conclusions

1) On average, an increment of the mean cardiac frequency during AF leads to higher maximal flow rates during the systolic and diastolic periods but it has a negligible effect on minimal flow rates, which remain almost unaffected. Moreover, fluctuations around mean values of maximal flow rates become more marked moving towards the high frequency range, with predominant systolic fluctuations up to 110 bpm and a progressive amplification in the diastolic oscillations from 50 to 130 bpm.
2) Coronary perfusion begins to be impaired exceeding 90-110 bpm.
3) CBF positively correlates with CPP up to 70 bpm, but this correlation is lost at higher frequencies, when data become sparse. Moreover, when HR is small enough (50/70 bpm) linear responses are possible even in case of AF; however, at high frequencies (above 70 bpm) any form of linearity disappears. Therefore, our results show that CPP cannot be considered a good estimate of CBF for high HRs in AF.

References

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