

The effect of *active* and *passive* dysfunction on right ventricle performance

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ABSTRACT

The study of the role of the right ventricle (*RV*) has lagged behind that of the left ventricle (*LV*). In this work, lumped parameter models are used to highlight the effect of *RV* on caval veins pressure and cardiac output.

1 INTRODUCTION

In the past, the importance of the right ventricle (*RV*) has been underestimated and lagged behind the one of the left ventricle (*LV*) [1, 2]. Specifically, it was thought that *RV* does not affect the cardiac output (*CO*) or the systemic venous pressure. However, in the last years, several studies demonstrated the role of the *RV* in cardiovascular diseases such as pulmonary hypertension [1]. The purpose of the present study is to evaluate the impact of *RV* dysfunction in terms of mean caval veins pressure (*pCVe*) and *CO* by assessing several degrees of *RV* impairment exploiting the lumped parameter model methodology. Starting from the definition of *RV* failure given by Haddad et al. in [1], in our model we implement the impaired ability of the *RV* to fill or to eject blood.

2 METHODS

The blood circulation has been replicated by means of a 0D model (see Figure 1). Two different geometrical configurations have been analyzed: a “normal” circulation (Figure 1a) and a “no*RV*” circulation in which the *RV* is missing and the venae cave (*CVe*) are directly connected to the pulmonary arteries (*PuA*) by the total cavo-pulmonary connection (*TCPC*) (Figure 1b), as in the so called Fontan circulation. For each circulation, only the main functional elements are reproduced considering compliance and resistance effects attributed to large vessels and microvasculature, respectively. Heart chambers are modelled as both passive elements, i.e., pressure

changes according to volume variations within the chamber, and active chambers, i.e., myofibers activation is replicated. In particular, ventricles are modelled according to Eq. (1)

$$P(t) = \varphi(t)E_{max}(V(t) - V_u) + (1 - \varphi(t))P_0(e^{K_E V(t)} - 1) \quad (1)$$

where $P(t)$ is the pressure within the chamber, $\varphi(t)$ is the activation function, E_{max} the ventricle elastance, $V(t)$ the volume within the chamber, V_u the unstressed volume, P_0 a parameter that characterizes the diastole and K_E the ventricle distensibility [3]. Finally, a resistance effect is applied to heart valves when open while the *TCPC* is simulated by the non-linear pressure flow relationship:

$$\Delta P(t) = K_{TCPC} Q^2(t) \quad (2)$$

where K_{TCPC} is the equivalent “resistance” and Q is the flow rate.

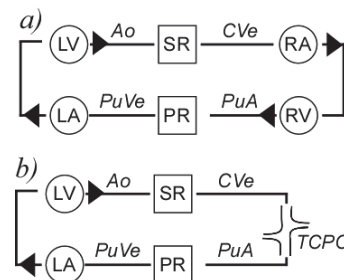


Figure 1. Geometrical configurations analyzed: a) *normal* circulation and b) *noRV* circulation. *LV*, left ventricle, *Ao*, aorta, *SR*, systemic resistance, *CVe*, caval veins, *RA*, right atrium, *RV*, right ventricle, *PuA*, pulmonary arteries, *PR*, pulmonary resistance, *PuVe*, pulmonary veins, *LA*, left atrium and *TCPC*, total cavo-pulmonary connection.

In order to analyze several degrees of *RV* dysfunction, different configurations have been reproduced. The passive and active functions were impaired separately: *i*) decreasing the ability of *RV* to fill by increasing K_E , i.e., making the ventricle stiffer, and *ii*) decreasing the ability to eject blood by reducing E_{max} , i.e., reducing the contractile force of the ventricle. In particular, starting from *normal* values, K_E

and E_{max} have been increased/decreased in the range of 10%-50%.

Calibration and validation of the model have been achieved according to literature data.

3 RESULTS AND DISCUSSION

Results have been analyzed considering $pCVe$ and CO . Here we refer to ad when examining the impairment of the ejection activity, i.e., *depressed active* function, and to pd when considering a stiffer ventricle, i.e., *depressed passive* function.

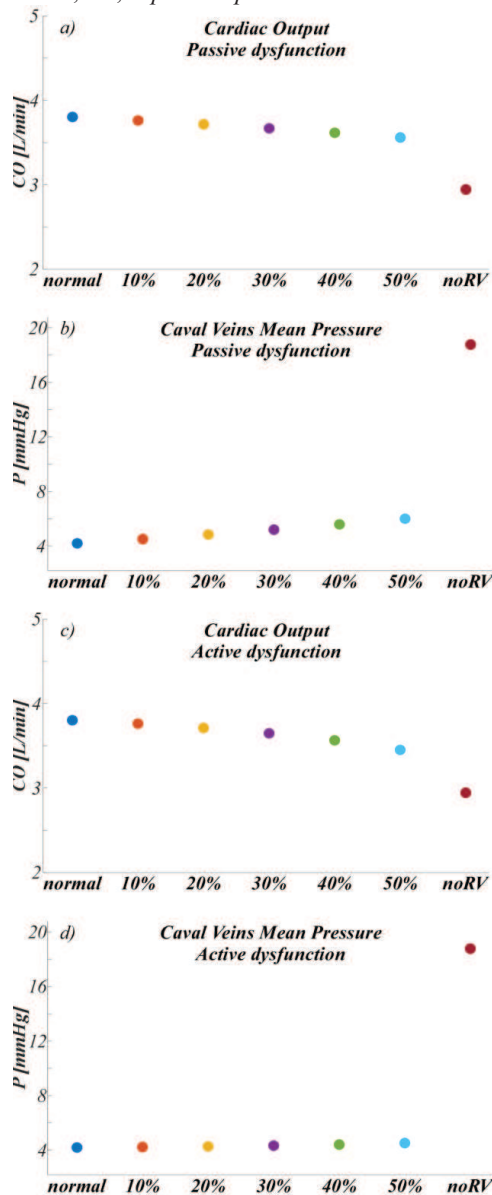


Figure 2. Results of the simulations. pd simulation results: a) CO and b) $pCVe$ and ad simulation results: c) CO and d) $pCVe$.

In Figure 2 the model outputs are reported. In all the configurations there is an almost continuous trend from *normal* to *noRV* that highlights the role of the right ventricle. An impaired RV can decrease the CO and increase the pressure on the caval veins. Moreover, comparing the pd configuration (Figure 2a and b) with the ad one (Figure 2c and d), it seems from the results that the RV ability to fill affects the performance of the system more than contractility does. Indeed, being equal the percentage of impairment, $pCVe$ (Figure 2b and d) is higher in pd than in ad , i.e., a stiffer RV creates higher pressure in the caval veins than a decreased contractile ability, and the same happens for CO (Figure 2a and c) that is lower in the pd configuration than in the ad one.

These preliminary results confirm the recent way of thinking that gives importance also to the right ventricle for the overall wellness of the body. The model, indeed, is able to show the effects of an impaired RV on $pCVe$ and CO .

Future work will be done in order to simulate a more realistic condition by adding important clinical concurrences. It is known from clinical practice that often RV impairment is related to tricuspid valve insufficiency when there is a diastolic dysfunction, and to changing in cardiovascular parameters, e.g. percentage of systole, when there is a decreased contractile activity.

REFERENCES

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