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A FIRST APPROACH FOR AN EXERGY ANALYSIS OF THE HUMAN HEART WITH A PATHOLOGY

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Abstract. *One can notice the versatility of the Second Law of Thermodynamics by the constant outbreak of its principles' new applications. Among them, one of the most inquisitive is its use on living organism analysis, in all levels of organization. One of the highlights in the field is the application of exergy analysis to human organs, given that pathologies can greatly modify the destroyed exergy of the heart, for instance. The aim of these type of analyses is to help medical diagnosis. Bearing this in mind, the present work develops a thermodynamic model of the heart, considering the phenomenological behaviour of the circulatory system. Therefore, pressure volume curves of the left and right ventricles are predicted, being the input of the analysis. Moreover, data for healthy and hypertensive subjects, found on medical literature were used as a validation of the model. From this point it was possible to apply the First Law of Thermodynamics and exergy analysis for both conditions: healthy and unhealthy. Results may indicate a higher destroyed exergy when the subject is not healthy, even with no significant variations in exergy efficiency.*

Keywords: *Biothermodynamics, Heart, Second Law of Thermodynamics.*

1. INTRODUCTION

Any energy conversion process can be evaluated through the applications of the First and Second Law of Thermodynamics. While the first one states the conservation of energy in every conversion process, the Second Law quantifies the amount of irreversibility within any process. One approach to the Second Law of Thermodynamics is the use of exergy. Szargut (2005) defines exergy as a physical quantity capable of characterizing the quality of any kind of energy under consideration. In that way, exergy analysis presents itself as an useful tool for determining the losses and opportunities of improvement in a system or process using the same basis, the capacity of any system to perform work.

One of the most curious applications for the Second Law of Thermodynamics is the understanding of biological systems in all levels of organization, from cells to living organisms (de Oliveira Junior, 2013). Exergy analysis of the human body and its organs can deepen the understanding of energetic processes unclear to the medical area, enhancing the knowledge on how ageing and pathologies may affect the behaviour of the body.

Several works applied exergy analysis to biological systems. Batato *et al.* (1965) were the first to propose and perform an exergy analysis on the human body. Luo (2009) studied the entropy generated by cancerous cells, concluding that the entropy generation of those cells is always greater than that of healthy cells. Balmer (1982), Aoki (1991) and others analysed entropy generation of the human body and other living beings, noticing the tendency of organisms to an state of minimum entropy.

Henriques *et al.* (2016a) developed a model of the human heart for exergy analysis, applying it for normotensive and hypertensive subjects in different exercise conditions. Results showed a higher exergy destruction for hypertensive subjects, but no significant variation in cardiac efficiency. Henriques *et al.* (2016b) and Roll *et al.* (2017) further improved

the previous model and applied it to other pathologies (aortic stenosis and aortic valve stenosis), obtaining higher exergy destruction rates for unhealthy conditions.

In this context, this article develops a new approach for exergy analysis of the human heart, using different tools to evaluate the energy provided by the heart metabolism. The exergy analysis is than applied to healthy and hypertensive subjects, using predicted pressure-volume loops to evaluate the destroyed exergy and exergy efficiency of the heart.

2. METHODS

The human heart is composed of two pumps: the left heart, main pump of the cardiovascular system, responsible for the systemic circulation; and the right heart, an impulse pump, bounded to the pulmonary circulation. Each of these pumps is composed of one atrium and one ventricle. The atria are small chambers that work as passive reservoirs, while the ventricles are pressure generators, pumping blood through the circulatory system. Therefore, the left and right hearts can be compared to two pumps in series, demanding an adequate balance between their flow rates (Guyton and Hall, 2006; Boron and Boulpaep, 2012).

Therefore, the model used for thermodynamic analysis can be seen on Fig. 1. Since the atria are passive reservoirs, for the sake of simplicity, each side of the heart is composed of only one chamber. The model functions as a closed loop and steady state representation of the heart and cardiovascular system, and is based on previous work by Henriques *et al.* (2016a). In this figure, W represents the work done by the cardiac muscle; B_M is the exergy variation of metabolic reactions in the cardiac muscle; B_{QM} illustrates the exergy associated with heat transfer from the heart to the rest of the body; $B_{in,pul}$, $B_{out,aorta}$, $B_{in,cava}$ and $B_{out,pul}$ are the exergy flow rates carried in and out of the control volume by blood. The boundary of the control volume is placed on the outside of the cardiac muscle, confining work and metabolic exergy inside it.

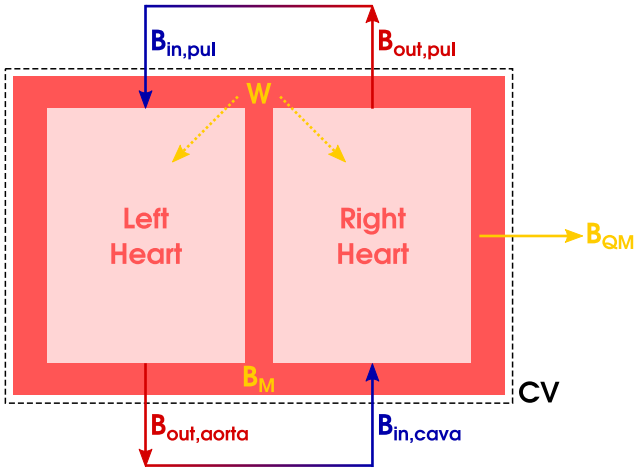

Figure 1. Control volume used for analysis.

For a first approach, the authors estimated pressure-volume loops for healthy right and left ventricles, based on data from Boron and Boulpaep (2012) and Redington *et al.* (1990). For the hypertensive case, the only adjusts made were changes in the pressures used for construction of the loops, considering data from Klabunde (2012) and Navadia (2014).

The exergy analysis for a generic control volume can be seen in Eq. 1, expressing the temporal variation of exergy as a function of heat exchange, power, exergy flow rates between inlet and outlet and destroyed exergy. For human organs, Keutenedjian Mady *et al.* (2012) introduces Eq. 2, which expresses the temporal variation of exergy as a combination of the metabolic exergy rate in the organ and the temporal variation of exergy due to transient environmental conditions.

$$\frac{dB}{dt} = \sum_j \left(1 - \frac{T_0}{T_j}\right) Q_j - W + \sum_{in} \dot{m}_{in} b_{in} - \sum_{out} \dot{m}_{out} b_{out} - B_d \tag{1}$$

$$\frac{dB}{dt} = B_{prod} - B_{reag} + \left.\frac{dB}{dt}\right|_{\Delta T} = -B_M + \left.\frac{dB}{dt}\right|_{\Delta T} \tag{2}$$

Assuming there are no variations of the environmental conditions , Eqs. 1 and 2 can be combined. The result is Eq. 3, that allows to estimate the destroyed exergy of an organ.

$$B_d = B_M + \sum_j \left(1 - \frac{T_0}{T_j}\right) Q_j - W + \sum_{in} \dot{m}_{in} b_{in} - \sum_{out} \dot{m}_{out} b_{out} \tag{3}$$

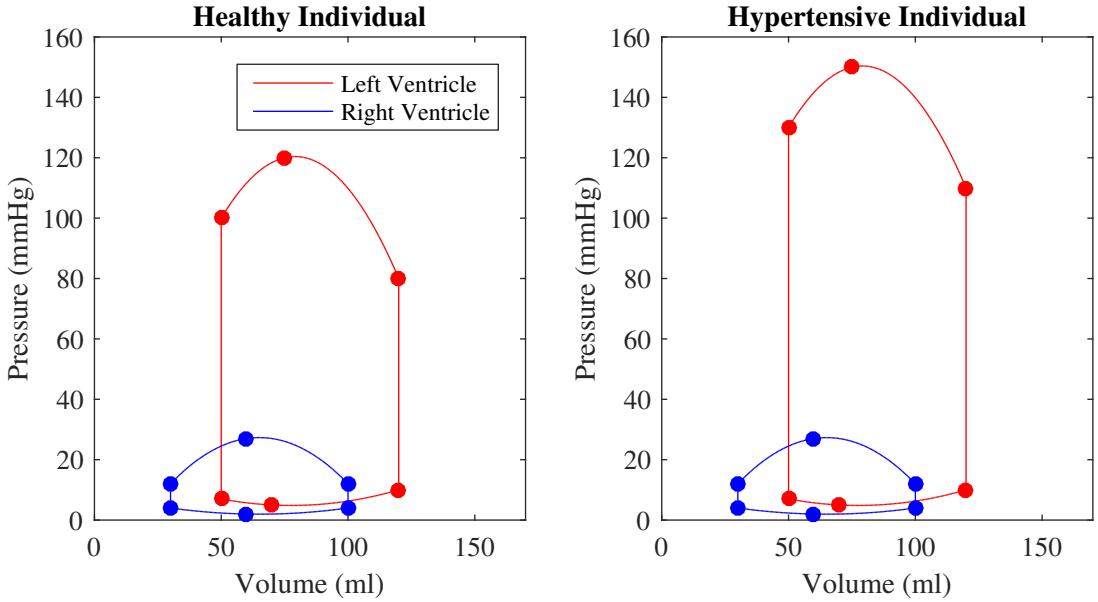

Figure 2. Estimated pressure-volume loops for healthy and hypertensive individuals, in medical units.

The power generated by the heart can be calculated as shown in Eq. 4, considering V_{min} and V_{max} the minimum and maximum volumes appearing on a cardiac cycle, for both left and right ventricles.

$$W = \int_{V_{min}}^{V_{max}} p(V)dV \tag{4}$$

Mady (2013) suggests the use of Eqs. 5 and 6 for estimating metabolic energy and exergy, respectively, in the human heart. However, those equations are functions of oxygen consumed and carbon dioxide produced by the heart, requiring the determination of those quantities.

$$M = 11371 m_{O_2} + 2366 m_{CO_2} \tag{5}$$

$$B_M = 9501 m_{O_2} + 3963 m_{CO_2} \tag{6}$$

The oxygen consumption of the myocardium, or $\dot{V}_{O_2}$ can be assessed through invasive methods or correlations that relate the consumption rate to the pressure-volume loops of the left ventricle (Suga, 2003; Steendijk and Brinke, 2008). The pressure-volume area (or PVA) is defined as the sum between the area defined by the pressure-volume variations of the cardiac chamber (EW, or cardiac work) and the triangular area between the end-diastolic pressure-volume relationship (EDPVR) and the end-systolic pressure-volume relationship (ESPVR) and limited laterally by the isovolumetric relaxation of the cardiac chamber (Fig. 3). It is important to define that the EDPVR and ESPVR are physical quantities, measured in the heart by filling until its maximum capacity.

Several authors have reported a linear relationship between $\dot{V}_{O_2}$ and the PVA (Izzi *et al.*, 1991; Rolett *et al.*, 1965; Takaoka *et al.*, 1993). Equation 7 was obtained by Takaoka *et al.* (1992), and correlates the oxygen consumption to the pressure volume area with a correlation coefficient of 0.911.

$$\dot{V}_{O_2} = (1.82 \cdot 10^{-5}) PVA + 0.0284 \tag{7}$$

Considering an energy balance applied to the control volume, it is possible to can obtain the heat transfer from the heart with Eq. 8, estimating the exergy of the heat exchange with Eq. 9, where T_0 represents the reference temperature (298 K) and T_{int} represents the average temperature of the human body (310 K).

$$Q_M = M - W \tag{8}$$

$$B_{Q_M} = Q_M \left(1 - \frac{T_0}{T_{int}}\right) \tag{9}$$

The exergy flow rates between inlet and outlet are divided in two parcels: a physical one and a kinetic one (Eq. 10). The physical component (Eq. 11) is obtained from the exergy of a stream equation for an incompressible substance, assuming no significant temperature variations between inlet and outlet, while the kinetic component (Eq. 12) accounts

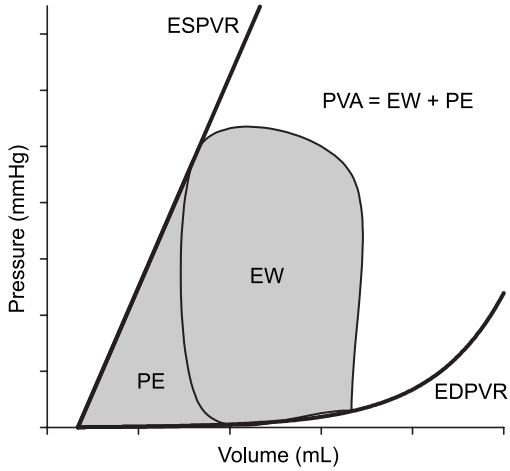

Figure 3. Pressure-volume loops for the left ventricle. The darker lines are the end-systolic and end-diastolic pressure-volume relationships, while the shadowed area represents the PVA (pressure-volume area). Adapted from Steendijk and Brinke (2008).

for the velocity difference between inlet and outlet. The flow rate $\dot{Q}$ is determined using the difference between V_{max} and V_{min}, considering the period of a heart cycle as the reciprocal of heart rate.

$$\Delta B_{fluxes} = \Delta B_{phys} + \Delta B_{kin} \tag{10}$$

$$\Delta B_{phys} = \dot{Q}\left(\frac{T_0}{T_{int}}\right)(P_{in} - P_{out}) \tag{11}$$

$$\Delta B_{kin} = \left(\frac{\dot{Q}^3}{2v_{blood}}\right)\left(\frac{1}{A_{in}^2} - \frac{1}{A_{out}^2}\right) \tag{12}$$

For an exergy efficiency analysis, Henriques *et al.* (2016a) suggests the use of Eq. 13, considering work as the desired effect and the metabolism as the source of exergy. However, another way to define the exergy efficiency of the heart is proposed in Eq. 14, considering as desired effect the exergy acquired by blood while passing through the heart, or ΔB_{fluxes}.

$$\eta_1 = \frac{W}{B_M} \tag{13}$$

$$\eta_2 = \frac{|\Delta B_{fluxes}|}{B_M} \tag{14}$$

3. RESULTS

Considering the method for application of exergy analysis to the human heart described in the previous section and the pressure-volume cycles proposed, the destroyed exergy rate and exergy efficiencies were evaluated for healthy and hypertensive individuals with a heart rate of 76 BPM. Results can be seen on Tab. 1.

Table 1. Results obtained for the defined exergy efficiencies, destroyed exergy and its components for both healthy and unhealthy subjects.

Balance [W]	**Healthy**	**Hypertensive**
W	1.15	1.43
B_M	3.90	4.68
B_{QM}	0.12	0.14
ΔB_{fluxes}	-1.20	-1.55
B_d	1.66	1.85
η_1	29.48%	30.52%
η_2	30.97%	33.00%

It can be seen that a hypertensive heart have a exergy destruction rate 0.19 W higher than a healthy one. All components of the destroyed exergy rate are higher for the hypertensive heart, once it has a bigger pressure-volume area in relation to

healthy individuals. Considering an individual who develops hypertension by the age of 30 and lives up to 80 years old, by integration of the destroyed exergy rate, one can conclude that an hypertensive individuals destroys around 300 MJ more than healthy ones.

Both exergy efficiencies defined resulted in values of about 30%. The efficiency based on the variation of exergy flow rates gave results 1-2% higher than the one based on cardiac power. For both definitions, there was no significant variation between healthy and hypertensive individuals, agreeing with results found by Henriques *et al.* (2016a). However, hypertensive hearts presented a slightly higher efficiency, for both cases.

The similarity found on exergy efficiencies can be explained by the fact that, in hypertension, higher pressures are achieved, increasing the power output of the heart (and the exergy flow rate) but also the pressure-volume area, and, therefore, the metabolic exergy provided to the cardiac muscle. Nevertheless, it seems that the increase in the metabolic activity is not as high as the effect of hypertension in other physical quantities.

4. CONCLUSIONS

A methodology for exergy analysis of the heart using the pressure-volume area as a prediction of cardiac metabolism was proposed and applied to healthy and hypertensive hearts. Hypertensive hearts presented a higher exergy destruction rate and similar efficiencies in relation to healthy hearts.

There is still room for improvement in the modelling of the unhealthy heart, once little medical information could be found about the complete effects of systemic hypertension in the human heart.

5. ACKNOWLEDGEMENTS

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