ELECTRO-FLUID-DYNAMIC SIMULATOR OF THE CARDIOVASCULAR SYSTEM FOR THE RESEARCH IN ASSISTED CIRCULATION AREA

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Abstract. *Human cardiovascular system models have been used to study dynamics of heart and circulation. They help understanding elastic behavior of great vessels, the action of valves, the blood flow and pressure, in normal physiological and pathology conditions. Moreover, they are a useful tool for the development of Ventricular Assist Devices (VAD). Simulation systems reduce the number of experimental surgeries and permit analysis of some conditions that are of difficult to establish "in vivo" experiments. This paper describes an electro-fluid-dynamic simulator of the cardiovascular system to be applied in the development of a VAD. This simulator, under development at our laboratories, consists of three modules: 1) electrical circuit model of the cardiovascular system that operates in the PSPICE simulator environment: it represents the arterial trees in a reduced form – "windkessel" – and the heart as variable elastances; 2) electronic controller (under development): based on the LabView® acquisition and control tool, that will act on the physical simulator; 3) physical simulator (under development): a fluid-dynamic simulator composed by linear actuators, for simulation of the active heart chambers, and compliance tubes for simulation of great vessels. This module is proposed based on results obtained with the electric circuit model (module 1). Simulation studies, using the electrical circuit model, provided physiological parameters acceptable for normal human conditions.*

Keywords: *cardiovascular system model, ventricle assist device, electro-fluid-dynamic simulator.*

1. Introduction

The cardiovascular diseases are the first cause of human death all over the world (AHCP, 2005). Big portion of them is related to severe cardiac congestive disease and heart transplantation becomes the unique available procedure. However, either due to difficulties to obtain the organ or due to the patient's weakness, the surgery cannot be immediately performed. Therefore, 30% of patients die waiting for heart transplantation (Kaye, 1993). The Left Ventricle Assist Devices (LVAD) appears as an important option to support those patients, during the period they are waiting for the heart transplantation. Efficient and reliable LVAD, that is able to improve the patient's life quality, still is considered a challenge for several research groups.

LVAD prototype cannot be evaluated directly in patients due to the device risks of malfunction. To solve this problem, three LVAD evaluation modes use to be applied: 1) mathematic models provide a numerical evaluation of the problems; 2) mock loop test setups simulate the problems and physical experiments can be performed, the so called "in vitro" tests; 3) device implantations in experimental animals evaluate the device characteristics working in real physiological conditions, the so called "in vivo" experiment. One evaluation mode cannot exclude another. However, they complement each other.

In this work, an electro-fluid-dynamic simulator of the cardiovascular system for development of ventricular assistance devices is presented. This simulator is under development at São Judas Tadeu University (USJT) in conjunction with Institute Dante Pazzanese of Cardiology (IDPC) and has three modules: 1) cardiovascular system electric analog model is operating in PSPICE electric simulator environment: it represents the reduced form of arterial aortic tree (windkessel), and the left cardiac chamber through an elastance variable in time (E(t)), in fact, a compliance varying with time: $C(t)=1/E(t)$; 2) electronic controller: currently, it's been developed, based on the LabView® acquisition and control tool, to actuate over the physical simulator; 3) physical simulator: it consists of a fluid-dynamic equipment with linear actuators to simulate the heart chamber (left ventricle) and with compliance tubes to simulate the great vessels (pulmonary vein and aorta artery). The instantaneous parameters, such as hydraulic resistances and tube compliances, are fixed by the electronic controller (module 2), which is "fed" with parameters previously tested through simulation performed in module 1.

2. Model Characterization

Chilbert (1988) states that the most important step for physiological system modeling is the accurate determination of model application. A cardiovascular system model, used to simulate assisted circulation, at first, must respect the

cannulation technique. IDPC's research group has been studying and improving a LVAD in experimental implantation in animals with apical-aortic cannulation (Andrade, 1999) - Fig. 1. In this technique, the LVAD receives blood through a cannula introduced in the apex of the natural left ventricle and pumps the received blood volume to the aorta artery (in series assistance).

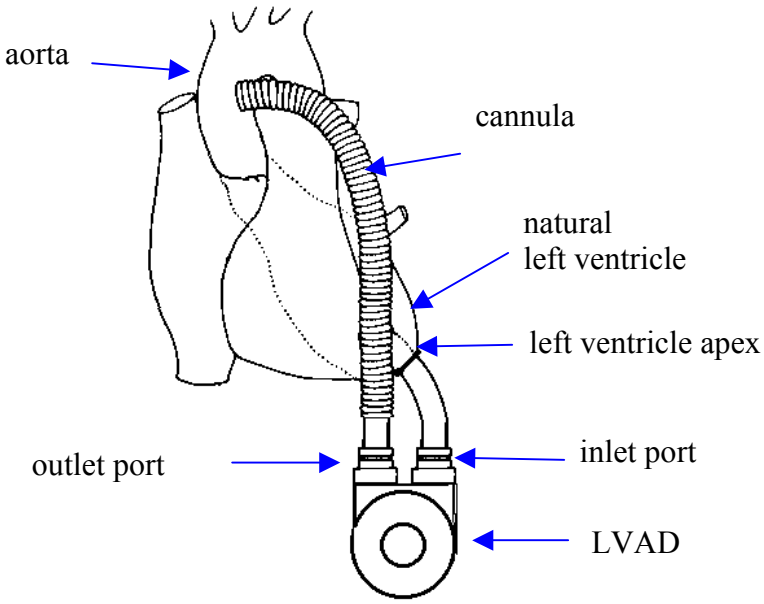


Figure 1. Schematic drawing showing LVAD's apical-aortic cannulation.

The physical simulator represents the human circulatory system with apical-aortic cannulation. The following items are represented: the left ventricle with an active cavity and the vascular system in a closed loop. Respecting Frank-Starling's law, the ventricular elastance activation function as well as the heart debit are pre-load dependent, in the mathematical model. Also respecting the same principle, in the electro-fluid-dynamic equipment, the pump will be servo-controlled (LabView®).

2.1. Left Ventricle Model

Ventricles are cavities with muscular walls contracting periodically. The effect of such contraction is ventricular pressure variation as function of volume and time (Borges, 1975). Ventricular elastance is defined as relation between ventricular pressure and ventricular volume. Generally, ventricle is modeled as time-variable elastance (Fu, 1998), (Kelley, 1995), (Mitsui *et al*., 1996), (Yu, 1998). The representation of time-variable elastances is made as Pressure x Volume (PxV) diagram through the heart cycle (Timmons, 1995), as shown in Fig. 2 (b) left portion. Figure 2 (b) right portion presents the ventricular pressure in a cardiac cycle (Tc).

Suga and Sagawa (1974), Suga, Sagawa and Demer (1980) and Sagawa (1978) identified several properties for time-variable elastance. In their experiments, pre and after loads suffered variation, as well as myocardium contractility and heart rate. It is possible to observe that: 1) the point at end of systolic phase (*Vsf, Pfs*) is kept over the static PxV curve for the activated myocardium; 2) the PxV curve, at the end of the systolic phase, as well as the time to reach the aortic valve closing are not sensitive to pre and after loads, but they are affected by the myocardium contractility and by the heart rate (*tc*); 3) finally, the ventricular filling phase follows the static curve for relaxed myocardium.

By approximating the PxV relationship, at the end of the systolic and diastolic phases, through straight line distances, Suga and Sagawa (1974) proposed a simplified relationship for the time-variable elastance: $E(t) = P(t)/(V(t)-Vo)$. $E(t)$ is the instantaneous ventricular elastance, $P(t)$ is the instantaneous ventricular pressure, $V(t)$ is the instantaneous ventricular volume and Vo is the point where the straight line E intercepts the axis of abscissas. Considering that the ventricular elastance is located between its "*Emax*" values, at the end of the systolic phase, and "*Emin*", at the end of the diastolic phase, and still defining the activation function $\alpha(t)$, it is possible to write the elastance time function expression as : $E(t) = \alpha(t)Emax+[1-\alpha(t)]Emin$.

In order to describe the elastance time behavior during the systolic phase, several researchers have been using activation functions $\alpha(t)$ of the following types: rectified sine, square sine, triangular wave, square wave, etc. (Timmons, 1995).

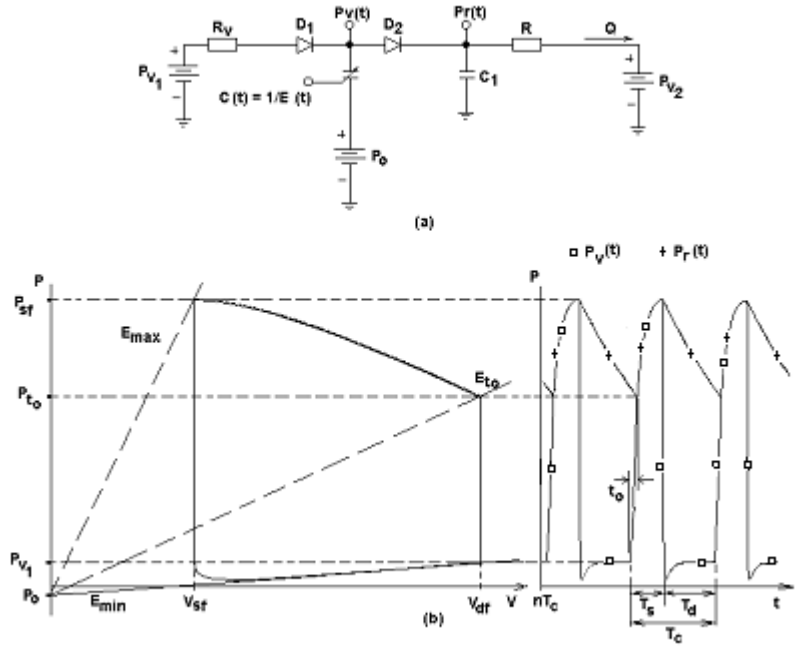
The *Vo* volume does not remain constant during all cardiac cycle, and presents a continuous decrease during the ejection phase (Timmons, 1995). Such size is still difficult to be measured in humans and, in general, it is obtained through the extrapolation of the curve generated by the end points of the systolic phase (Beringer and Kerkhof, 1998).

The adopted option in our ventricular model is the use of a negative and constant *Po* pressure (fixed and extrapolated point under the origin in ordinate axis) instead of *Vo*.

By using *Po*, the effect of increasing inclination of *E,* during the ejection phase (from *Emin to Emax*), is the same one as decreasing *Vo*, with the advantage that *Po* is a parameter easy implement in electric analog model (Lucchi, 1999).

In left ventricle electric analog model, (Fig. 2 a), $C(t) = 1/E(t)$ is a capacitance varying in time and *Po* is simply a constant voltage generator. The heart valves are modeled by ideal diodes (D_1 and D_2) and the figure presents the ventricle pumping between constant pressure sources (*Pv1 and Pv2*).

Pv_1 source represents average venous pressure, at natural ventricle entrance; Pv_2 is average pressure in the output of the "windkessel" model (used as ventricular preload). Also, the model has a hydraulic resistance *R* and a compliance C_1. The hydraulic resistance (*Rv*) limits the venous flow (*Q*) and establishes a limited time for the ventricle filling phase.



P = pressure, R = resistance, D = diode, C = compliance, E = elastance, Q = flow, V = volume, T and t = time

Figure 2. Chart showing the ventricle model pumping between two pressure sources (a) and PxV cycle and temporal behavior of the ventricular pressure $P_v(t)$ and arterial pressure $P_r(t)$ (b).

2.2. Vascular System Model

The vascular system model is divided in two groups: with distributed parameters (of transmission line type) and with concentrated parameters (of "windkessel" type). The first group considers details of the vascular geometry and bifurcations, which may produce reflections from pressure and flow waves. The vascular system has a big number of bifurcations. It is impossible to describe each one since arterial system has more than a billion of them (Palladino, Drzewiecki and Noodergraaf, 1995). Several simplified models with distributed parameters have been proposed to study pressure and flow wave reflection phenomena (Jager, Westerhof and Noodergraaf, 1965), (Pollack, Reddy and Noodergraaf, 1968), (Westerhof, 1968), (Schwarzaupt *et al*., 1997).

If reflection properties for distributed systems are not the most important part of the study (which is the case in this work), the arterial pressure "seen" by the heart, specifically by the left ventricle, may be obtained through a model of "windkessel" type (Palladino, Drzewiecki and Noodergraaf 1995).

Goldwyn and Watt (1967) proposed a physical model of vascular system formed by four components: two elastic chambers (represented by their compliances C_1 and C_2), connected by a circular section tube (the fluid inertial effect can be observed in a tube with length *L* and transversal section *A*), ending by a "Poiseuille hydraulic resistance" (*R*). The Goldwyn and Watt's model, schematically represented in Fig. 3 (a), is provided by enough details and can predict the pressure wave form and, at the same time, it is simple enough for clinical applications (the parameters can be estimated from experimental tests).

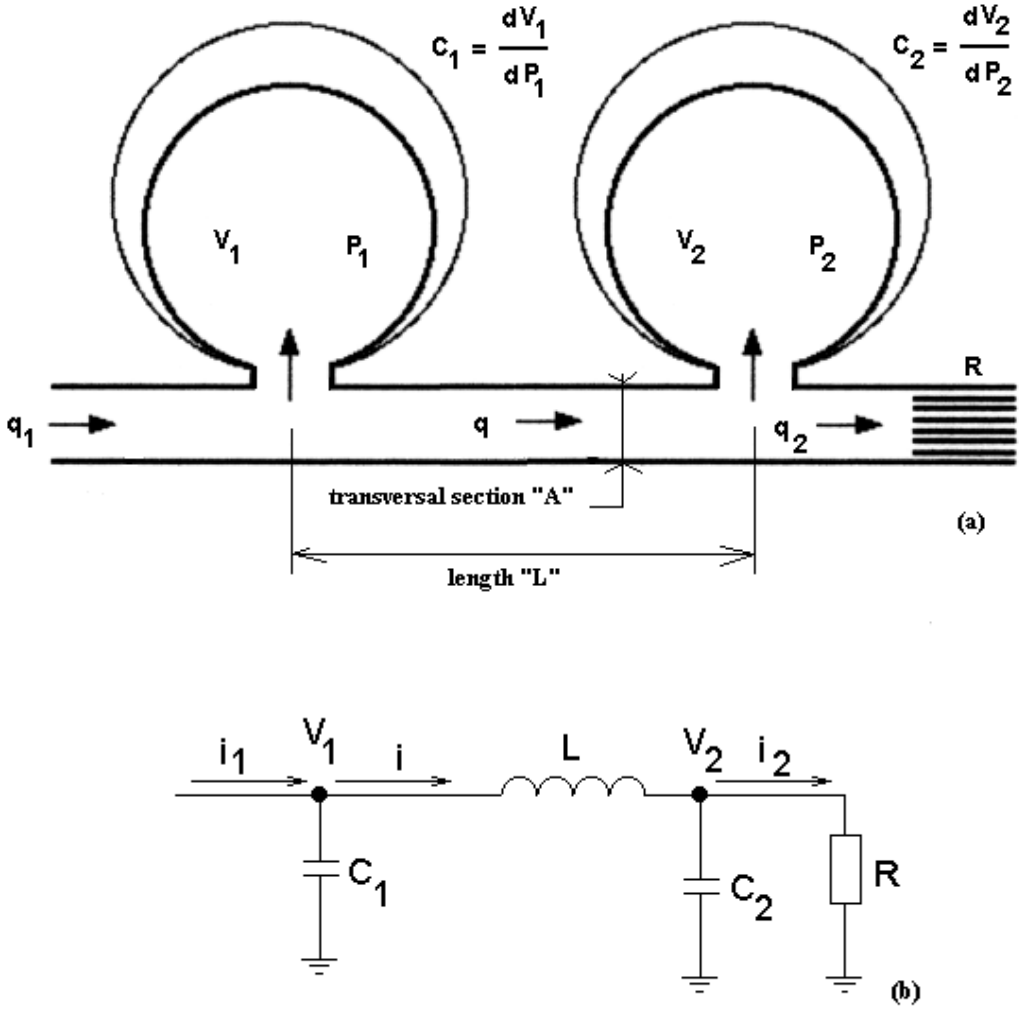


Figure 3. Vascular system model proposed by Goldwyn and Watt (1967) (a) and electric analog circuit (b).

In the model, the mass conservation equation for the first reservoir is: $q_1 - q = dV_1/dt$, and for the second one is: $q - q_2 = dV_2/dt$. Since constant compliances and reservoir external pressures are null (referential pressure), we can write: $q_1 - q = C_1.dP_1/dt$, e $q - q_2 = C_2.dP_2/dt$. Considering the blood flowing between the reservoirs in laminar regime through a rigid tube with transversal section A and length L, and ignoring the dissipative effects in this length, the mass conservation equation is: $d[\rho AL(q/A)]/dt = P_1A - P_2A$, where ρ is the density, ρAL is the mass, and q/A is the average velocity of flow.

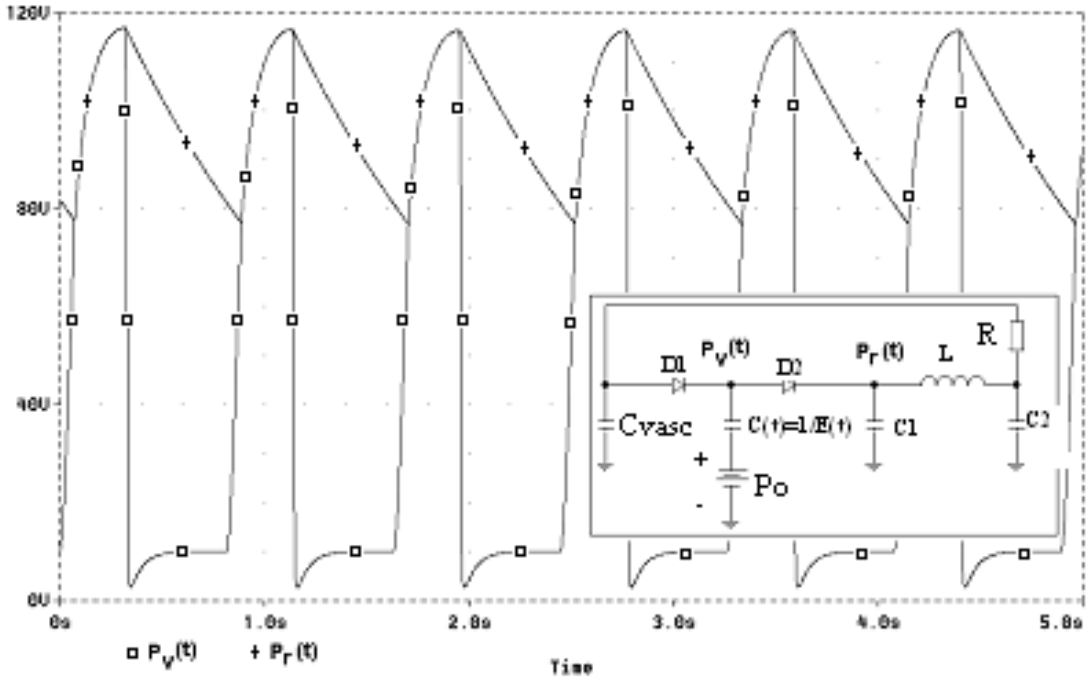
By defining the inertia as $M = \rho L/A$ and observing that output flow is $q_2 = P_2/R$ (Hagen-Poiseuille's equation), the system can be represented by the electric circuit showed in Fig. 3 (b), using the electric-hydraulic analogy presented in Tab. 1.

Table 1. Electro-Hydraulic Analogy.

HYDRAULIC MODEL	ELECTRIC MODEL
Flow (q_1, q_2, q)	Current (i_1, i_2, i)
Pressure (P_1, P_2)	Voltage (V_1, V_2)
Compliance (C_1, C_2)	Capacitance (C_1, C_2)
Inactivation (M)	Inductance (L)
Hydraulic Resistance (R)	Resistance (R)

2.3. Electric Analog Model of the Human Cardiovascular System

The electric analog model of the human cardiovascular system, operating in closed loop (module 1) and the simulation results for normal conditions are presented in Fig. 4. The activation function of ventricular elastance is controlled by the baroreceptor reflection and the voltage over capacitor C_1 (aortic pressure) affects *E(t)* (Lucchi, 1999). The ventricular and aortic pressures are presented as time function for 5 seconds of simulation. Each volt corresponds to 1 mmHg of pressure and the cardiac output was of approximately 5 L/min. Therefore, the model represents physiological parameters acceptable for normal human conditions. Alterations in the model parameters, such as maximum and minimum ventricular elastance, can simulate pathological conditions.



1V is equivalent to 1mmHg

Figure 4. Results obtained with Cardiovascular System Simulation and Electric Analog Model.

2.4. Electronic Controller and Physical Simulator

In the physical simulator (fluid-dynamic equipment under development) the ventricle must have an elastance varying by a linear actuator, and its instantaneous value will be controlled by LabView®. Two types of linear actuators have been developed for posterior comparison: diaphragm with pneumatic activation, similar to the proposal of Bustamante *et al*. (2004) and diaphragm with electric activation. The large vessels, aorta and pulmonary vein, are been constructed with compliance tubes. Their compliance will be controlled electrically using restrictive plates, Fig. 5(a). Fluid temperature will be maintained at 37°C (approximately) in a tank with thermostat, Fig. 5(b). Systemic hydraulic resistance will be controlled by electrically adjustable clamp, Fig. 5(c). The mathematical relationships used in the electric analog model are easily transported to the controller environment.

The system allows data acquisition. The pressure signals will not only be used to control the process in a closed loop, but also will be viewed in real time and stored for posterior analysis and report generation.

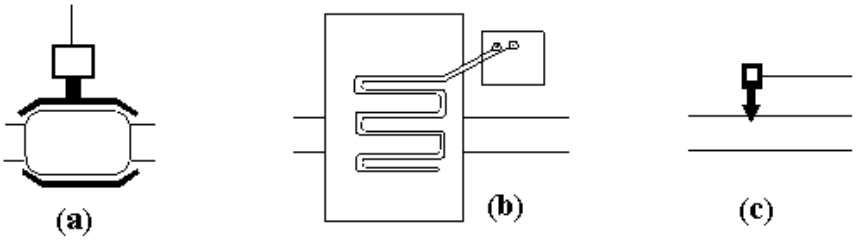


Figure 5. Parts of the physical system: (a) compliance tubes; (b) venous tank with temperature control; (c) controlled hydraulic resistance.

The schematic representation of the electro-fluid-dynamic simulator of the cardiovascular system (under development) to be applied in the development of LVAD is shown in Fig. 6.

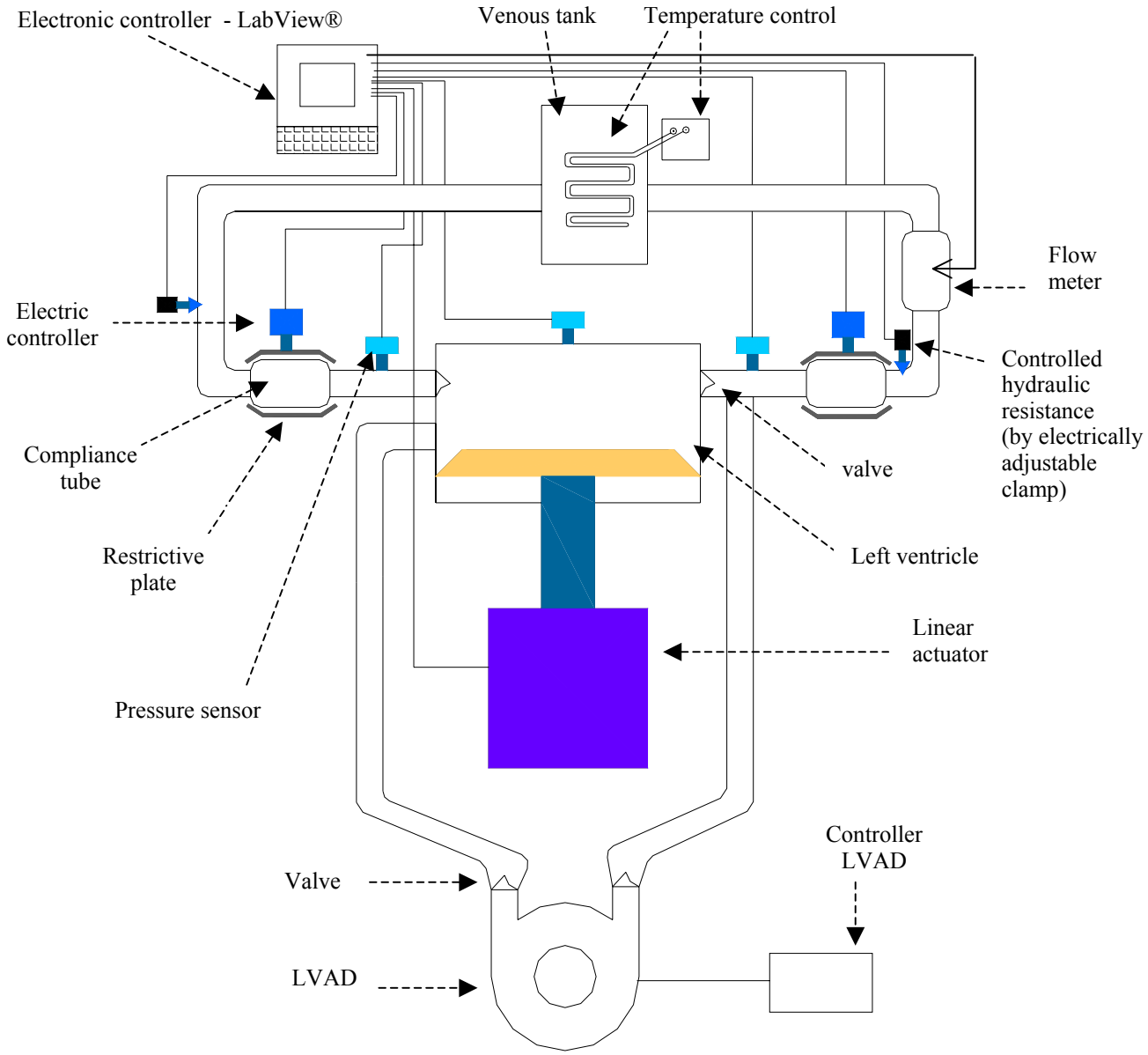


Figure 6. Schematic representation of the electro-fluid-dynamic simulator of the cardiovascular system.

3. Conclusions

The results obtained with the simulations using electric analog model (module 1) indicate physiological parameters acceptable for normal human conditions. This is considered to be very promising for this project, in its initial phase. Parameter alterations allow simulation of several pathological conditions. The simulation results makes up a group of information that will be essential for the development of a useful and versatile physical simulator.

4. Acknowledgements

The authors acknowledge the support from Research Center at São Judas Tadeu University and from Department of Bioengineering at Institute Dante Pazzanese of Cardiology.

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