# Simulation of Cardiovascular Diseases Using Electronic Circuits

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#### **Abstract**

We have developed an electronic circuit to simulate the behavior of the cardiovascular system under normal or pathological conditions. The current, voltage, charge, resistance and capacitance of the circuit correspond to flow, blood pressure, volume, resistance and capacitance of the simulated cardiovascular system, respectively. We have implemented a circuit with two ventricles separated from the systemic and the pulmonary circulation to simulate normal conditions. Vital parameters of the cardiovascular system such as aortic pressure, cardiac output, end-diastolic or systolic volume of right or left ventricle, left and right ventricular pressure, blood volume, pulmonary artery pressure and stroke volume can be obtained. Qualitative and quantitative agreement with the Starling preparation has been achieved for a 120 sec simulation time. We have also simulated pathological situations such as left ventricular failure, mitral stenosis, aortic valvular stenosis, hypertension, pulmonic valve stenosis adjusting the parameters of the circuit components.

# 1. Introduction

Mathematical models are powerful tools for investigating and understanding complex systems. Several models for the cardiovascular system have been proposed in the past which enhance knowledge about cardiovascular physiology [1,2]. These models provide understanding of the underlying mechanisms for normal conditions and diseases of the cardiovascular system such as arrhythmia [3], ischemia and hypertension [4] and permit exploitation of several therapy strategies [4]. Some of those models are based on electronic circuits [3,5].

Electronic circuits are used for two purposes: (a) to study wave propagation when an excitation is taking place in the heart [6], (b) to model the cardiovascular system under normal conditions [7-11]. Modeling of cardiovascular diseases has been achieved using complicated models which are based on the solution of

complex systems of equations [4,12] and not electronic circuits.

It is the aim of our work to address simulation of the cardiovascular system using an electronic circuit under normal and pathological conditions. The principles of equivalent quantities have been used to design the circuit. Adjusting the parameters of the circuit components we have simulated diseases of the cardiovascular system. More specifically, we present simulation of mitral valve stenosis, triscupid valve stenosis, pulmonic valve stenosis, aortic valve stenosis, hypertension and changes in heart rate.

#### 2. Materials and methods

The electronic circuit which is used for the simulation of the cardiovascular system is shown in Fig. 1. The design of the circuit is based on the principles of equivalent quantities as it is demonstrated in Table 1.

Table 1. Equivalent quantities.

Cardiovascular System	Electronic Circuit	Equivalence Relations
Voltage	Pressure	1V ↔ 10mmHg
Current	Blood Flow	$1\mu A \leftrightarrow 100 \text{ml/sec}$
Resistance	Resistance	$1M\Omega \leftrightarrow 1U$
Capacitance	Capacitance	1μF↔100ml/10mmg
Charge	Volume	$1\mu As \leftrightarrow 100ml$

In the design of the circuit we have taken into account the Starling law of the heart [1,2], which states that within physiological limits the heart pumps all the blood that returns to it without undue damming of blood in the veins. In addition, we assumed that the inertia of the blood is ignored and the left and right ventricular have the same capacitance. The circuit consists of three parts as the cardiovascular system: the heart (ventricle and atrium), the pulmonary circuit and the systemic circuit. The capacitance ratio between systemic and pulmonary circuit is approximately 1/7 and between systemic arterial and systemic venous about 1/40.

A pacemaker is connected in the circuit to drive the

two ventricles. The pacemaker is a square wave generator with adjustable frequency. The pacemaker operates in 1Hz frequency with duration of systole and diastole 0.25 and 0.75 sec, respectively. The A-V valves are simulated by a voltage-controlled switch and a 20 k $\Omega$  resistor. When the A-V valves are open, the switch is "on" and as the result the ventricles are "off" simulating diastole phase. The outflow tract valves (pulmonary, aortic) are simulated by diodes with adjustable internal parameters. The parameters of the cardiovascular system are measured in several points of the circuit. These points correspond to circles in Fig. 1.

The circuit must be modified to be used for the simulation of cardiovascular diseases. In our case, this can be achieved adjusting parameters of the circuit components.

To simulate mitral stenosis we use an adjustable resistor outside the mitral valve and two time-controlled switches. When the simulation starts, a 60V battery is used for 5 sec to simulate the venous reservoir. Steady state is established in 16 sec. After 60 sec a 1  $M\Omega$ resistor is connected to the circuit. Increasing the resistance we reduce the charge from the LA to the LV simulating the mechanism of the mitral stenosis [2]. Pulmonic valve stenosis and aortic valvular stenosis have been simulated using similar procedures. Hypertension [1,2] is simulated increasing the resistance in small arteries of the systemic from 7.5 M $\Omega$  to 12 M $\Omega$ . The 12 M $\Omega$  resistor replaces the 7.5 M $\Omega$  after 60 sec of steady state simulation. We have also simulated hear rate changes adjusting the value of the pacemaker frequency.

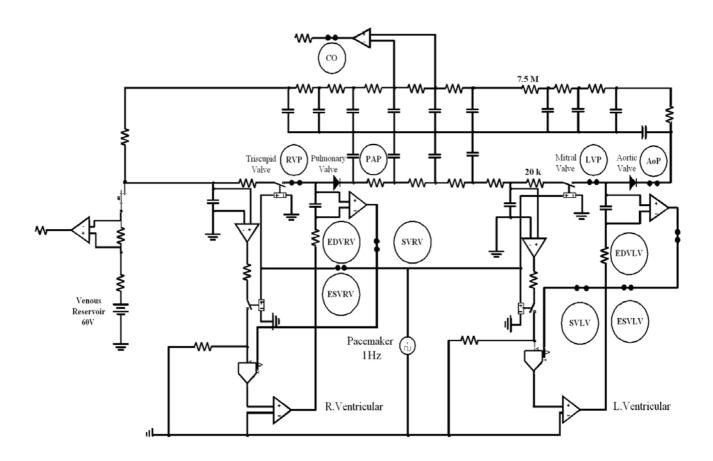


Figure 1. The proposed electronic circuit.

Table 2. Results for normal condition, mitral stenosis, hypertension and hear rate increase.

Measurements	Normal Conditions	Mitral Stenosis	Hypertension	Heart rate increase (2Hz)
RVP	29mmHg	31mmHg	28mmHg	27.4mmHg
LVP	136mmHg	137mmHg	156.2mmHg	130mmHg
PAP	23.3/14.3	26/19	22.3/14.3	21.9/17
LAtP	6.29mmHg	12mmHg	6.5mmHg	6.08mmHg
RatP	4.5mmHg	4.2mmHg	4.27mmHg	3.98mmHg
EDVLV	5.4mmHg	3.55mmHg	5.7mmHg	4.8mmHg
ESVLV	2.4mmHg	1.05mmHg	2.9mmHg	2.8mmHg
SVLV	3mmHg↔450ml/min	2.5mmHg↔375ml/min	2.8mmHg↔420ml/min	2mmHg↔300ml/min
EDVRV	3.80mmHg	3.5mmHg	3.6mmHg	2.9mmHg
ESVRV	0.80mmHg	1 mmHg	0.8mmHg	0.9mmHg
SVRV	3mmHg↔450ml/min	2.5mmHg↔375ml/min	2.8mmHg↔420ml/min	2mmHg↔300ml/min
CO	4650ml/min	3675ml/min	4050ml/min	5150ml/min
AoP	130/79mmHg	129/65mmHg	152.5/102.5mmHg	125/95mmHg

#### 3. Results

Results for the cardiovascular system under normal conditions, with mitral stenosis, with hypertension and with changes in heart rate are presented in Table 2. A list of all abbreviations used is provided in Appendix A. The results for normal conditions are similar to those reported in the literature for the cardiovascular system (Table 3). A remarkable increase of the AoP (15.25/10.25) and LVP (156 mmHg) is observed in hypertension when the resistance in capillaries increases by 50%. CO increases (10.75 %) when the HR increases from 1 to 2 Hz without changing the systole time of ventricles 0.25 sec. CO reaches the maximum value when HR = 2 Hz. A decrease in CO is observed for HR > 2 Hz, which agrees with the observation described in [1,2]. In the case of mitral stenosis we obtain: LatP = 12 mmHg, SVLV =2,5 mmHg (375 ml/min), CO = 24.5 mmHg (3675 ml/min) and AoP 12.9/6.5 which are close to those reported in literature [1,2].

### 4. Discussion

We have designed an analog circuit, which is able to simulate normal condition and diseases of the cardiovascular system. The measurements we obtain for mitral stenosis are close to what is reported in the literature: increase of the LatP (50%), significant decrease of the SVLV (16%) and reduced the CO (26 %) [2,13].

Our model is advantageous compared to other models since it requires only fundamental knowledge in the design of electronic circuits [14,15] and does not involve

complex equations. Compared to other electronic circuit models it is also advantageous since both normal conditions and diseases of the cardiovascular system can be simulated. The proposed model can be further upgraded to achieve more accurate simulations with fewer assumptions simply modifying the diastole time, systole time and the capacitance of LV and RV.

Table 3. Comparison of simulation results in normal condition vs physiological measurements of the cardiovascular system.

Quantity	Physiological Measurements	Simulation
RVP	25-30mmHg	29mmHg
LVP	120-130mmHg	136mmHg
PAP	21-25/7-13	23.3/14.3
LAtP	7-8mmHg	6.29mmHg
RatP	4-6mmHg	4.5mmHg
CO	5000ml/min	4650ml/min
AoP	120-130/80-90mmHg	130/79mmHg

## 5. Conclusions

We have developed electronic circuits to simulate the behavior of the cardiovascular system under normal and pathological conditions using the concept of the equivalent quantities. A circuit similar to human cardiovascular system with two ventricles separated from the systemic and the pulmonary circulation has been implemented. Assuming that the capacitance of the LV and RV is the same the simulation for 120 sec (total

charge is circulated once through the system within 60 sec) of the normal conditions and several diseases of the cardiovascular system provided with results which are very close to those reported in the literature. Further upgrade of the circuit is possible to explain more complicated mechanisms causing disorders of the cardiovascular system.

# Appendix A

Table 3. List of Abbreviations

Aortic pressure	AoP
Atrio-ventricular valve	A-V valve
Atrial pressure	AtP
Cardiac output	CO
Stroke volume	SV
Ventriculo-aortic valve	V-Ao valve
Blood volume	BV
End-diastolic volume of right ventricle	<b>EDVRV</b>
End-diastolic volume of left ventricle	EDVLV
Ejection fraction of the right ventricle	EFRV
Ejection fraction of the left ventricle	EFLV
End-systolic volume of left ventricle	ESVLV
End-systolic volume of right ventricle	ESVRV
Left ventricle	LV
Left ventricular pressure	LVP
Left atrial pressure	LatP
Pulmonary artery pressure	PAP
Right atrial pressure	RatP
Right ventricle	RV
Right ventricular pressure	RVP
Stroke volume of the right ventricle	SVRV
Stroke volume of the left ventricle	SVLV
Aortic pressure	AoP
Atrio-ventricular valve	A-V valve
Atrial pressure	AtP
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