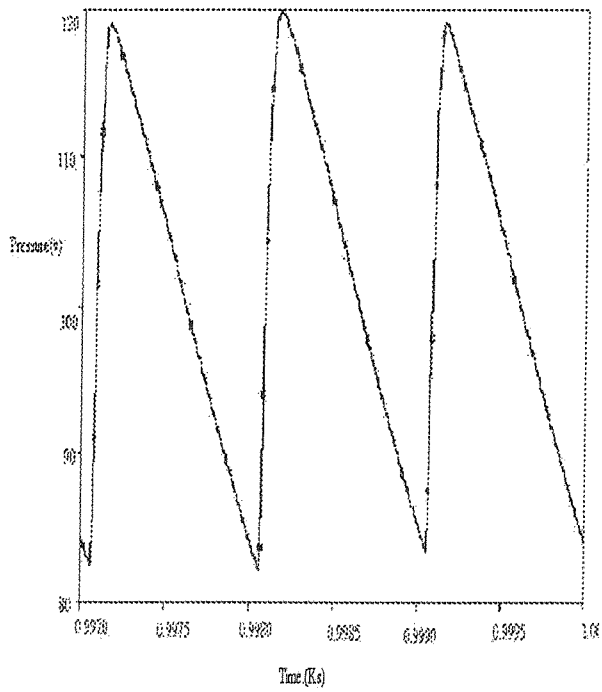

Figure 5: Pressure graph of aorta varying between 80-120 v (mmHg).

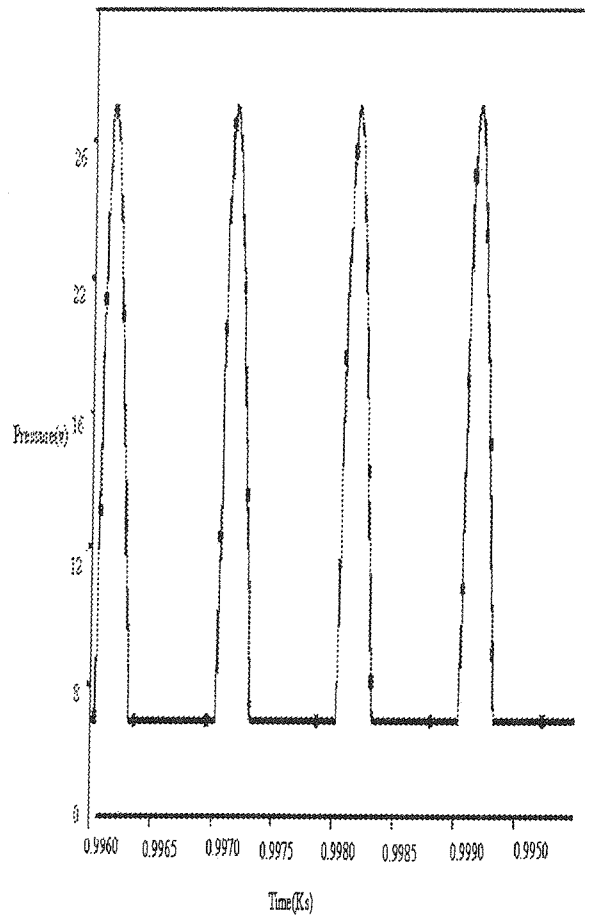

Figure 6: Pressure graph of right ventricle Varying between 6-25 v (mmHg).

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diastole. The experimental data of physiological reference [16] confirm it. In cardiovascular system, the volume graph of left ventricle varies between 60-120 ml but regarding the nature of the model, volume graph of Figure 4 varies between 0-60 ml. This can be interpreted by keeping the 60 ml difference. The effort was to keep the 60 ml difference of systole and diastole according to Figure 4. Figure 5 shows that aorta pressure varies between 80-120 mmHg (diastole-systole) and the results are in agreement with physiological article [16]. The physiological data show the variation of blood pressure in aorta to be between 78-115 mmHg. Figure 6 shows the pressure graph of right ventricle varying between 6-25 mmHg. The actual graph of right ventricle, which shows the variation of pressure between 6-27 mmHg, confirms that this simulation result is in accordance with physiological data [17]. Corresponding resistors should be decreased to keep the same time constant for contraction and relaxation of the heart, respectively. If contraction of atria is required, both atriums can be simulated as low-pressure pumps. Its simulation circuit should be the same as that for the ventricle however, because the four pumps are connected in series the circuit will be extremely complex. In this model, for didactic reasons, the right and left ventricles are not identical. The values of resistors and capacitances are not the same value but the resemblances to conditions *in vivo* are improved. All of valves have been simulated by diodes and a very small, so-called physiologic back flow of blood occurs. However, this back flow is negligible compared with cardiac output. Using the circuit, it is possible to simulate heart valves by switches driven by voltage and no back flow is observed but the circuit will be complex and likely unstable. The ultimate potential of the cardiovascular system modeling is to provide a clinician with an intelligent, sensitive tool for monitoring a patient's cardiovascular state over time so as to guide therapy. This model is very sensitive to the changes of its parameters because the elements are not passive. This sensitivity enables us to study the cardiovascular diseases changing the parameters values. The theoretical evaluation of our model, therefore provides confidence in the performance of the electronic system with respect to experimental data and demonstrates the power of the electronic systems for modeling of the cardiovascular system.

5. CONCLUSIONS

This study supports electronic system as a powerful approach for the intelligent patient monitoring of cardiovascular function. Moreover, the forward model, on which the theoretical validation is based, provides a convenient test bed of data. This may facilitate the development of new methods that could be incorporated with cardiovascular system identification method so as to provide a more detailed picture of cardiovascular state. In conclusion, based on the results of the modeling, we have found a very good agreement between the performance of this electronic system and the experimental data. Our understanding is that use of this equivalent electronic circuit of cardiovascular system is useful for studying of whole cardiovascular system and is a useful tool in teaching physiology and pathology for students.

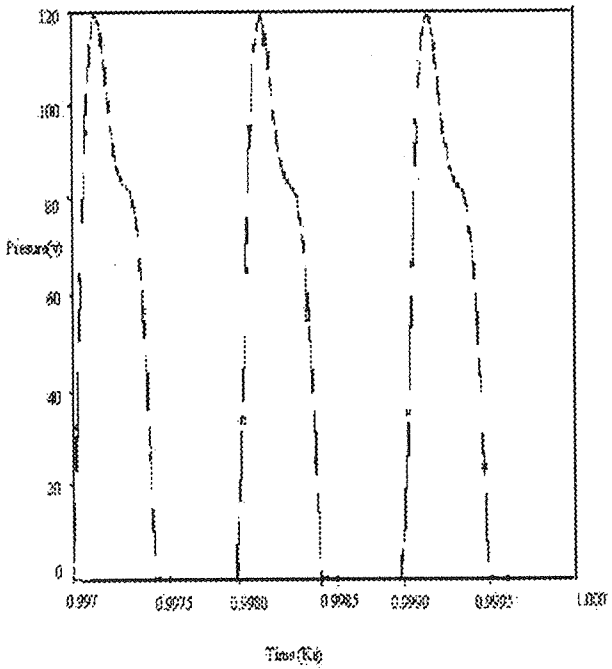

Figure 3: Pressure graph of left ventricle varying between 0-120 v (mmHg).

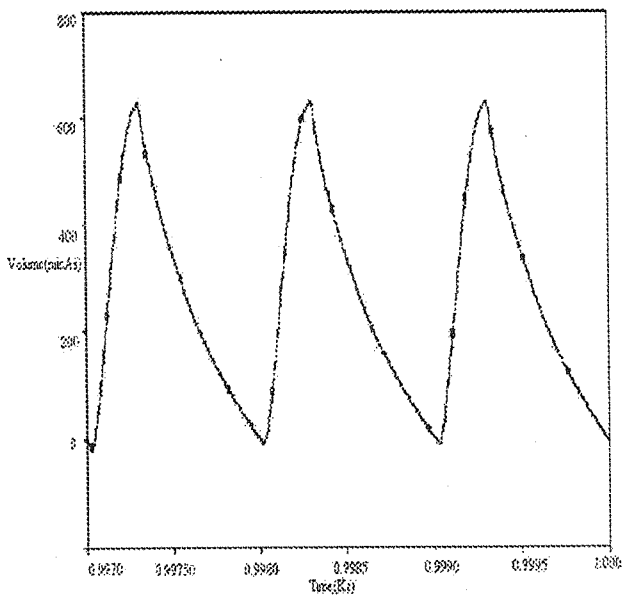

Figure 4: Volume graph of left ventricle and right ventricle varying between 0-60 μAs (ml).

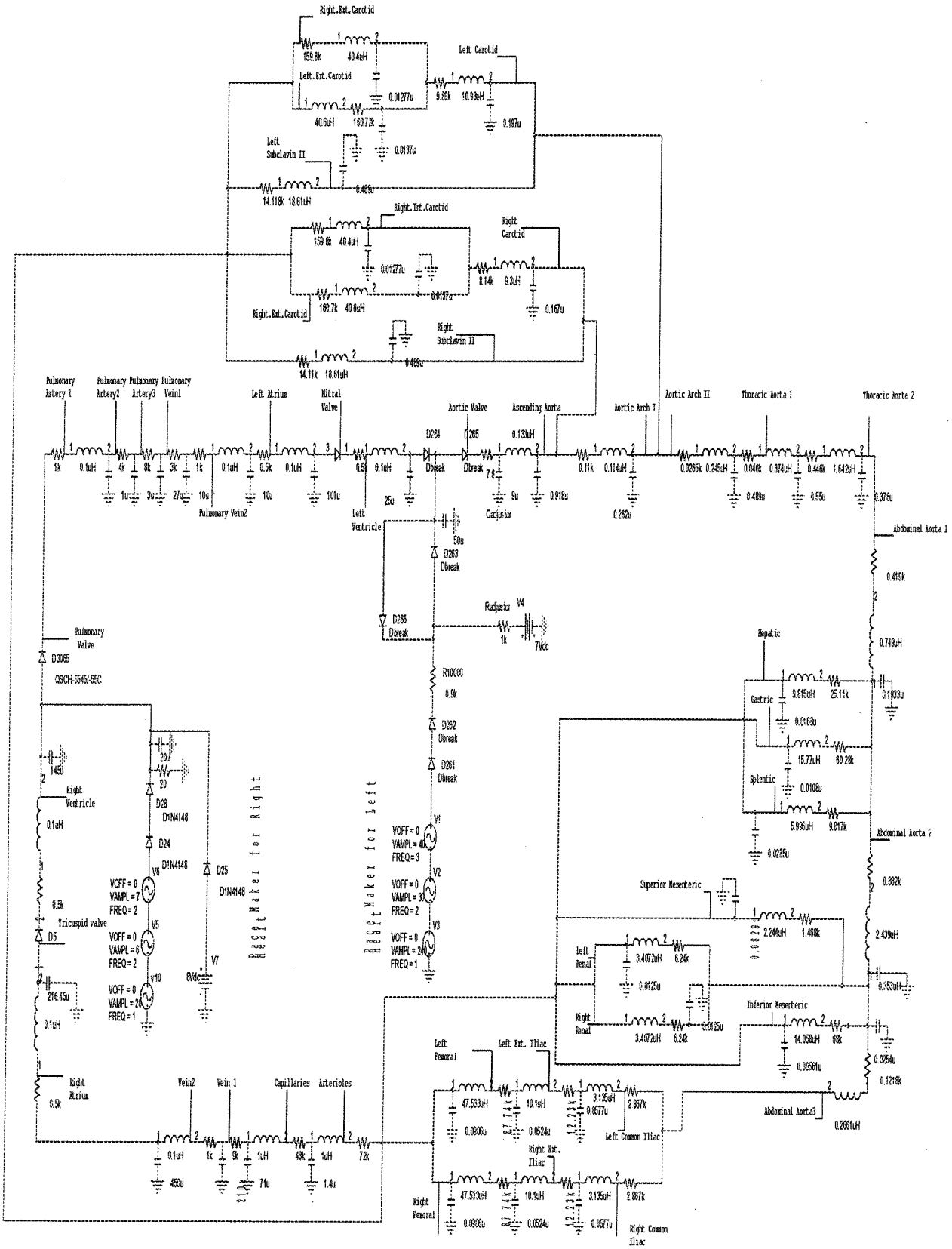

Figure 2: Electronic circuit of cardiovascular system.

5 mmHg and the peak is in 120 mmHg, therefore the result of simulation is in a very good agreement with real data. It shall be noted that each cycle is 0.8 s according to *in vivo* condition. Also, the volume graph of left and right ventricle which is shown in Figure 4, indicating the difference of 600 μAs (60ml) between systole and

contracting-relaxing action of the left and right ventricles are achieved by the pacemaker. The pacemaker consists of three AC power supply and two diodes (DIN4148). The pacemaker has the pulse generator of 1 Hz.

TABLE 1: THE SPECIFICATIONS OF THE MODEL SEGMENTS

N0.	DESCRIPTION	R(kΩ)	L(μH)	C (μF)
1	Left Atrium	0.5	0.1	101
2	Left Ventricle	0.5	0.1	25
3	Ascending Aorta	7.6	0.133	0.918
4	Aortic Arch1	0.011	0.144	0.262
5	Aortic Arch 2	0.0265	0.245	0.489
6	Right Subclavin II	14.118	18.61	0.489
7	Right Carotid	8.14	9.3	0.167
8	Right.int.Carotid	159.8	40.4	0.01277
9	Right.ext.Carotid	160.7	40.6	0.0137
10	Left Subclavin II	14.118	18.61	0.489
11	Left Carotid	9.89	10.93	0.197
A	Left.int.Carotid	159.8	40.4	0.01277
B	Left.ext.Carotid	160.72	40.6	0.0137
12	Thoracic Aorta 1	0.046	0.374	0.556
13	Thoracic Aorta 2	0.446	1.642	0.376
14	Abdominal Aorta 1	0.419	0.749	0.1933
15	Abdominal Aorta 2	0.882	2.439	0.353
16	Abdominal Aorta 3	0.1218	0.2661	0.0254
17	Left Common Iliac	2.867	3.135	0.0557
18	Left External Iliac	12.23	10.1	0.0524
19	Left Femoral	87.74	47.533	0.0906
20	Right Common Iliac	2.867	3.135	0.0577
21	Right External Iliac	12.23	10.1	0.0524
22	Right Femoral	87.74	47.533	0.0906
23	Hepatic	25.11	9.815	0.0168
24	Gastric	60.28	15.77	0.0108
25	Splenic	9.817	5.996	0.0285
26	Left Renal	6.24	3.4072	0.0125
27	Right Renal	6.24	3.4072	0.0125
28	Superior Mesenteric	1.468	2.244	0.0829
29	Inferior Mesenteric	68	14.058	0.00561
30	Arterioles	72	1	1.4
31	Capillaries	48	1	71
32	Vein 1	9	-	210
33	Vein 2	1	0.1	450
34	Right Atrium	0.5	0.1	216.45
35	Right Ventricle	0.5	0.1	150
C	Pulmonary Artery 1	1	0.1	1
D	Pulmonary Artery 2	4	-	1
E	Pulmonary Artery 3	8	-	3
F	Pulmonary Vein 1	3	-	27
G	Pulmonary Vein 2	1	0.1	10

For each of the ventricles, one pacemaker is used in order to drive the voltage into the circuit. As it can be seen in the Figure 2, an 8 DC voltage source is used for the right ventricle to enable the related pressure graph to vary between 8 to 25 volt (mmHg). The values of R.L and C were either directly extracted from references [8]-[9] or Were calculated using formulas of reference [10]. The circuit consists of the largest arteries and arterioles have been simulated as one separated compartment with resistance of 72 kΩ and capacitance of 1.4 μF. The capillaries and veins are also modeled by one and two segments. The upper body arteries such as carotid have been attached to arterioles by one connection. The generated current of suppliers distribute to left ventricle, aorta and upper body arteries. It continues its path toward the body arteries such as femoral, renal or gastric. The current passes the arterioles, capillaries and veins and enters the right atrium. Another amplifier, pacemaker, generates the required current for circulation in the pulmonary arteries and veins and eventually the final current enters the left atrium. One of the major problems of these types of complex circuits is stability. The stability of the circuit was checked by running it for long periods of time (1000s to 5000 s) and no sign of instability was observed. All of the current and voltage graphs have been checked for oscillating and there is no sign of oscillation in these graphs. The model is capable of showing pressure and volume signals for different arteries throughout carotid to femoral triggered by initial systolic contraction. The pressure (voltage) graphs can be obtained from the different points of the circuit easily but volume graphs can not be obtained directly meaning that integration of current–time graph shall be obtained as charge-time graph for each element. It shall be noted that there is no leakage of charge in the system and the output voltage is proportional to the input voltage. The proportionality factor reflects the contractility of the ventricles. To meet this end, both terminals of ventricles capacitors are connected to the pacemakers connections. Due to their high input impedance, there is no appreciable leakage of charge from the circuit. In this circuit, there is no point of constant pressure (voltage). Therefore, total capacitance of the system (905 μF) is one of the important determinants of its functions. The ratios of capacitances determine the distribution of total charge in the circuit. The ratio between total systemic arterial and systemic venous capacitance is about 0.01. The ratio between total systemic and pulmonary is 0.15. These ratios are similar to those reported for the human cardiovascular system [13]-[14].

4. RESULTS AND DISCUSSIONS

The results of the simulation performed for a heart rate of 60 beats per minute or 1 Hz are given in Figure 3 to Figure 6. The pressure-time graph of left ventricle is shown in Figure 3, where the waveform varies between 5-120 mmHg (volt).

From the experimental human data [15], we have found the normal blood pressure of left ventricle varies between 5-120 mmHg. The wave form in our model starts from

peripheral resistance [9]. A non-linear computer model for pressure and flow propagation in the human arterial system was developed [10]. The model with 55 arterial segments is based on one-dimensional flow equation and simulates different homodynamic effects on blood flow. The simulation of steady-state and transient phenomena using the electronic circuit has also been done but the arteries have not been included in their model widely [11]. A few forward models of the cardiovascular system have been previously developed for the purpose of using electronic system. One presented a beat-to-beat model, which incorporated *windkessel* and *starling* properties with arterial baroreflex control of blood flow and systemic arterial resistance [12]. Another model was developed to study the generation of waves [13]. This model included *windkessel* properties, constant stroke volume and arterial resistance. There are some other models which mainly incorporated *windkessel* properties, arterial resistance and assumed stroke volume but none of these simple models are nearly as comprehensive as our forward model in terms of accounting for pulsatility, the short term regulatory system, and resting physiological perturbations. The model which is presented here describes simulation of the cardiovascular system using a complex electronic circuit. In this study, we have taken a slightly different approach to the modeling of the system. We have tried to advance existing electrical models by increasing more segments and parameters. Compared to previous studies, the arterial system of this model is more detailed. Therefore, normal and abnormal performance of arterial system can be studied.

2. MODEL SIMULATION PRINCIPLES

The block diagram of cardiovascular system is shown in Figure 1. This diagram is an actual presentation of physiological cardiovascular and arterial system [14]. In our model, every blood vessel, atrium, ventricle and set of all capillaries and arterioles have been presented by a block consisting of a resistor, an inducer and a capacitor. Voltage, current, charge, resistance and capacitance in the electronic circuit are equivalent to blood pressure, flow, volume, resistance and compliance in the cardiovascular system. Ground potential (reference for voltage measurements) is equivalent to zero. The correlation between electrical characteristics of the system and their mechanical counterparts are as follow:

1mmHg = 1 volt (pressure ~ voltage)
1ml = 10 μAs (volume ~ charge)
0.01ml/Pa = 1 μF (compliance ~ capacitance)
1 Pa.s/ml = 1 kΩ (resistance)
1 Pa.s2/ml = 1 μH (inertia ~ inducer)
1s=1s (unit of time)

Blood vessels resistance, depending on blood viscosity and vessel diameter, are simulated by resistors. The vessel compliance is simulated by capacitors. The blood inertia is considered using inducers. Atriums are simulated as part of the venous system without any contraction. Atriums are modeled as a resistor-capacitor segment. Ventricles are simulated as a section of blood vessels in which its resting capacitance (diastole) can be decreased (systole) and then returned to the resting capacitance. Essentially, energy of systolic contraction of left and right ventricles are modeled by superposition of three AC power supplies and diodes. These voltage sources are amplifiers and the input of them are connected to the capacitors simulating atriums. Thus, during systole, the voltage from the ventricular capacitor is amplified and applied to the aortic capacitor. The specification of each block is explained in Table 1. As can be seen, each block consists of a resistor(R), an inducer (L) and a capacitor(C).

3. SYSTEM DESCRIPTION

The equivalent circuit of the cardiovascular system is shown in Figure 2. In this simulation, blood volume, cardiac output and aortic pressure should be 5000 ml, 83 ml/s and 80-120 mmHg. For better understanding of the circuit, Figure 1 and Table1 should be noted together. Similarly, as the preparation *in vivo*, also the equivalent circuit can be subdivided into two parts: heart (atrium and ventricle) and the arterial circulation. The left atrium and

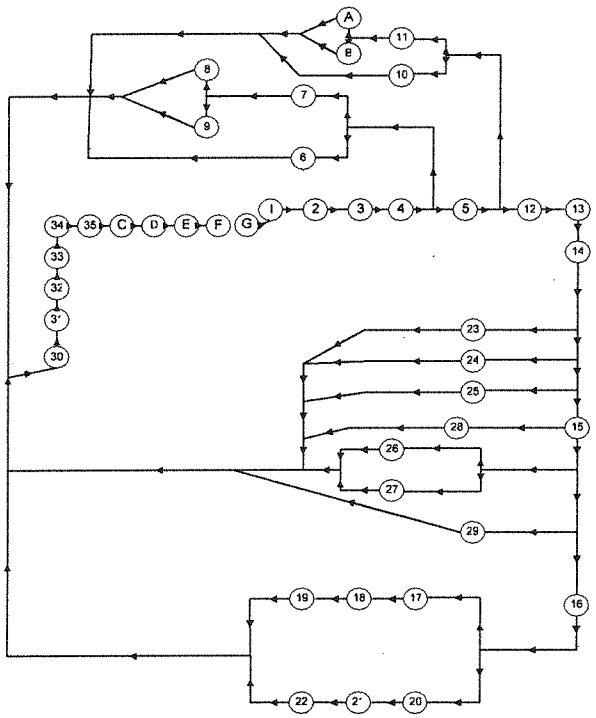

Figure 1: Block diagram of cardiovascular system.

ventricle are modeled by two capacitors 216.45 μF and 150 μF. Using of suitable diodes were very important matter in this circuit. The aortic, mitral, tricuspid and pulmonary valve are simulated with ideal diodes: SD41, SD41, 120NQ045 and QSCH-SS45/-55. Using these types of diodes have helped the circuit to show the operation of the cardiovascular system properly. The

Simulation of the Cardiovascular System Using Equivalent Electronic Circuit With Active Elements

K.Hassani [i]; M.Navidbakhsh [ii]; and M.Rostami [iii]

ABSTRACT

This paper describes simulation of the cardiovascular system using a complex electronic circuit. In this study, we have taken a slightly different approach to the modeling of the system and tried to advance existing electrical models by increasing more segments and parameters. The model consists of 42 segments representing the arterial system which are active. Anatomical and physiological data for circuit parameters have been extracted from medical articles and textbooks. The frequency of heart is 1 Hz and the system operates in steady state condition. Each artery is modeled by one capacitor, resistor and inductor. The left and right ventricles are modeled using AC power suppliers and diodes. The results of the simulation including pressure and volume graphs exhibit operation of the cardiovascular system under normal condition. The results of the simulation have been compared with the relevant experimental observation and are in good agreement with them.

KEYWORDS

Modeling, Simulation, Electronic Circuit, Cardiovascular System.

1. INTRODUCTION

The diseases of the human cardiovascular system are one of the main problems in contemporary health care in industrial countries. They cause the majority of deaths and also often afflict people in their most productive age. In this context, different approaches were used with the aim of providing better understanding and simulation of the blood flow in the human cardiovascular system. A three - parametric model of heart muscle mechanics was introduced [1] and a modification of this model was presented later [2]. It successfully predicted force development during both isometric and isotonic contractions, showing deactivation of the contractile element during isotonic shortening and the apparent dependence of series stiffness in time. The study of the series elasticity of cardiac muscle was presented [3]. Afterwards, the time-varying elastance model of the left ventricle was introduced and later the relationship between pressure-volume area and cardiac oxygen consumption was described [4]. Finite element methods are commonly used to simulate the left ventricular performance. The first computer models describing the arterial system constructed a multi-branched model of the systemic arterial tree in a form usable for digital computer which allowed simulation of different physiological and pathological conditions. This model was described in detail later [5]. Also blood flow through sites of particular interest of the arterial tree such as anastomoses, stenoses and bifurcations can be described with finite element method simulation. The mathematical description of the whole human cardiovascular system remains a complex task, and for that reason, the models are simplified with respect to particular parts of interest. A pulsatile-flow model of the left heart and two-segment aorta was constructed and the changes in flow work investigated. Time-tension index and stroke volume of this model are produced by an intra aortic balloon pump [5]. Later, an open-loop lumped-parameter model was developed and the sensitivity of an intra aortic balloon pump to timing, rate of inflation, balloon placement and stroke volume was determined [6]. The model later yielded good results for a short-term interaction between the assist device and the circulation [7]. The model of an assisted failing canine circulation was presented from which cardiac oxygen supply and consumption could be calculated [8]. The influence of drugs on the circulation was studied later in a 19-compartment model. Also, the use of computer-aided design of control systems was improved to produce a simple electrical analog of the human cardiovascular system and used to simulate steady-state circulatory conditions with transients introduced by varying the

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