CARDIOVASCULAR SIMULATOR

CONTENTS

1. Introduction

Purpose

The Cardiovascular Simulator is a dynamic computer simulation of human cardiovascular hemodynamics, primarily intended for students of physiology and medicine. It is implemented on engineering workstations running the X Window System, allowing the student to perform a variety of measurements in a real-time simulated cardiovascular system, not all of which would be possible in an animal laboratory. The simulation is based on the lumped-parameter mathematical model taught in Quantitative Physiology and Cardiovascular Pathophysiology courses in the Harvard-M.I.T. Division of Health Sciences and Technology. The computer simulation is implemented on the M.I.T. Project Athena student computing network.

2. The Model

The Basic System

The model which forms the basis of the simulator, based on an analog computer implementation by Defares [1], is shown in Figure 1. The cardiovascular system is divided into four major sections: the left heart, the systemic (peripheral) circulation, the right heart, and the pulmonary circulation. Each side of the heart is modelled by a variable capacitor, representing the pumping action of atrium and ventricle as a unit; two diodes, representing the atrioventricular and arterial valves; and inflow and outflow resistances. In total, the blood storage capacity of all vessels are lumped into six capacitances (C_l, C_a, C_v, C_r, C_{pa}, C_{pv}), connected by six resistances (R_{li}, R_{lo}, R_a, R_v, R_{ro}, R_{pa}), and four ideal flow valves (D_{li}, D_{lo}, D_{ri}, D_{ro}).

The program calculates experimental data based on constituent relations for each element and the continuity of flow through the system, analogous to Kirchoff's Current Law for electrical circuits. For the resistive pathways (resistors), flow is the pressure drop across the pathway divided by the resistance of the pathway, equivalent to Ohm's Law for linear resistors.

$$Q_r = P_r/R \qquad \text{Electrical Analog: } i = v/r \qquad (1)$$

For the capacitative compartments (capacitors), flow is equal to the time rate of change of volume in the compartment.

$$Q_c = dV_c/dt \qquad i = dq/dt \qquad (2)$$

The volume in the compartment can be expressed as the pressure times the compliance, plus the zero-pressure (atmospheric) filling volume.

$$V_c = P_c C + V_0 \qquad q = vc + q_0 \qquad (3)$$

Therefore, the change in volume is

$$dV_c/dt = d(P_cC)/dt \qquad\qquad dq/dt = d(vc)/dt \qquad\qquad (4)$$

This gives a complete equation for the continuity of flow:

$$Q_c = C\ dP_c/dt + P_c\ dC/dt \qquad\qquad i = c\ dv/dt + v\ dc/dt \qquad\qquad (5)$$

The lumped-parameter model is described fully by Eq. (5) applied to each of six circuit nodes, with initial conditions. These equations are solved numerically, giving pressure, volume, and flow through each compartment of the system. lnitial parameters are chosen which closely match experimental data from the literature. The variable capacitances in the heart are driven by a pre-computed time function, also derived from experimental data. The user of the simulator may modify all local and system parameters at any time; the instantaneous response to such perturbations can then be visualized for any pressure, volume, or flow in the system.
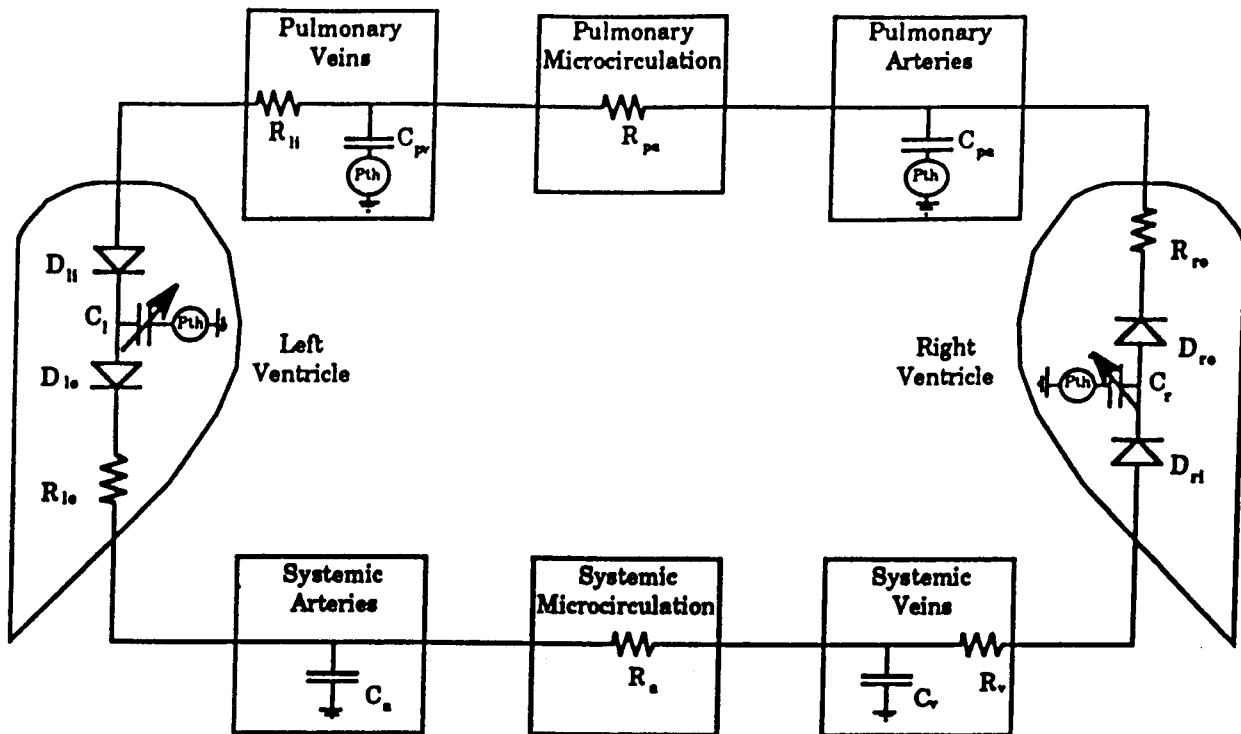

Figure 1. Circuit Diagram Equivalent of Lumped Parameter Model

The Baroreceptor Control System

The simulator incorporates a baroreceptor feedback control model in addition to the hemodynamics described above. The baroreceptor senses arterial pressure, P_a, producing an afferent signal, S, proportional to the deviation of P_a from the current setpoint pressure, P_{sp}. The physiological control pathways are described in Figure 2.

The control signal, S, is used to modulate autonomic activity to control heart rate and contractility, peripheral resistance, and venous tone. We have chosen a proportional control law which updates HR, $C_{l\,sys}$, $C_{r\,sys}$, R_a, and V_{v0} by adding to each of these parameters a deviation consisting of a moving average of the control signal multiplied by a gain factor. The model is based on the work of DeBoer [2]. Each limb of the response has a specific delay due primarily to the

response times of different autonomic nerve types and end-organs. Afferent nerve and central nervous system delays also contribute to delay in the control system (Table 1).

NERVE TYPE	LATENCY	MAX RESPONSE	LENGTH OF RESPONSE
Alpha-sympathetic	2.0 sec	5.0 sec	30.0 sec
Beta-sympathetic	2.0 sec	5.0 sec	30.0 sec
Parasympathetic	0.0 sec	0.5 sec	1.0 sec

Table 1. Delay values for reflex responses

Eq. (6) is the control equation for peripheral resistance. It incorporates a resistance setpoint R_0, an alpha sympathetic tone constant τ (usually zero), a gain factor γ, and a moving average of the control signal, S[n].
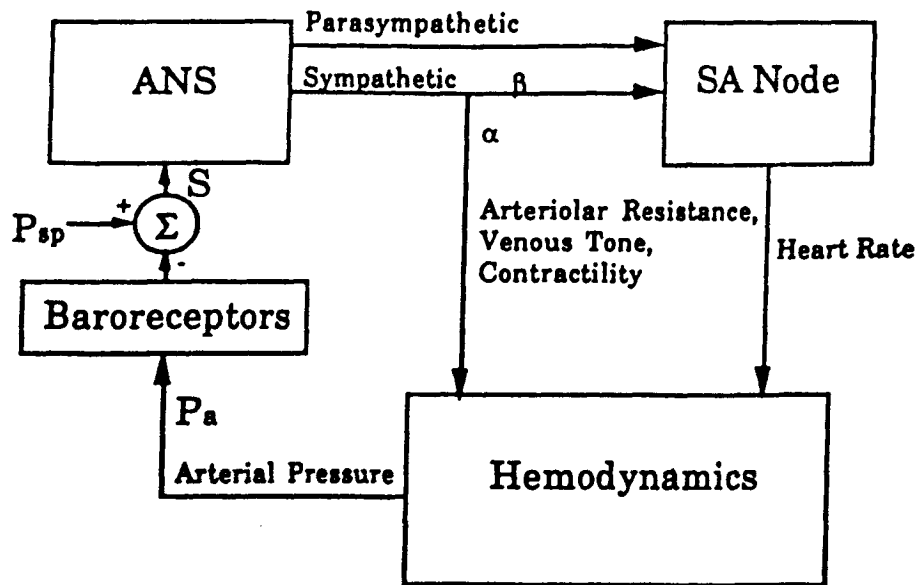

Figure 2. Baroreceptor Reflex Loop

$$R_a[n] = R_0 + \tau + \gamma \sum_{k=0}^{L} h[k]S[n-k]$$

(6)

The other controlled parameters are set similarly, but with different values for τ, γ, the weighting function h[k], and the nominal parameter value setpoint.

The gain values for heart rate and peripheral resistance were obtained from DeBoer [2], whereas contractility and venous tone gains were calculated directly from known near-maximal physiological responses in order to produce reasonable activity. This gross approximation may not hold for such a linearized operating-point control model; therefore caution should be used when making physiological inferences based on the control system model. For that matter, it should always be remembered that the cardiovascular simulator is not by any means a complete model of

hemodynamics—it merely serves as a linear simplification to facilitate understanding the principles of the real, nonlinear system.

REFLEX LIMB	GAIN	RESPONSE TIMING
heart rate	18 ms/mmHg	beta-sympathetic & parasympathetic
contractility	0.007 ml/mmHg/mmHg	beta-sympathetic
peripheral resistance	0.011 PRU/mmHg	alpha-sympathetic
venous zero-pressure volume	26.5 ml/mmHg	alpha-sympathetic

Table 2. Gain and timing for reflex responses

References

1.   Defares, J.G., J.J. Osborne, and H.H. Hara. Theoretical synthesis of the cardiovascular system. Study I: The controlled system. *Acta Physiol. Pharm. Neerl. 12* (1963), 189-265.

2   DeBoer, R.W., J.M. Karemaka, and J. Strackee. Hemodynamic fluctuations and baroreflex sensitivity in humans: a beat-to-beat model. *Am. J. Physiol. 253*, Heart Circ. Physiol. 22 (1987), H680-H689.