Physiome of Coronary Circulation: Simulation of Autoregulated Coronary Flow

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Abstract

The purpose of this study was to explain intramyocardial coronary microvascular diameter changes during a cardiac cycle observed with our needle-probe CCD videomicroscope by a model simulation. An autoregulated coronary flow model was constructed by including a control element that regulates blood flow by tuning myocardial elastance to our previous model. The relationship between mean flow and purfusion pressure by model experiments coincided well with the published data. The relative arteriolar diameters maximal in a cardiac cycle were calculated based on simulation. The changes of diameters were small, coinciding with the experimental observation.

1. Introduction

Coronary blood flow is affected by physical factors, such as blood pressure and intramyocardial pressure and by physiological factors such as nervous and hormonal control. Hence, autoregulation is brought about. We observed intramyocardial coronary microvascular diameter changes during a cardiac cycle with our needle-probe CCD intra-vital videomicroscope[1] [2].

We analyzed the mechanism of the myocardial coronary vascular blood flow dynamics by introducing the myocardium pump model[4] that took account of both intramyocardial pressure and myocardial elastance(contractility)[3].

The purpose of this study is to explain further intaramyocardial coronary microvascular diameter changes during a cardiac cycle by a model simulation. The proposed model includes an autoregulating mechanism with the element that regulates blood flow by tuning myocardial elastance to the previous model.

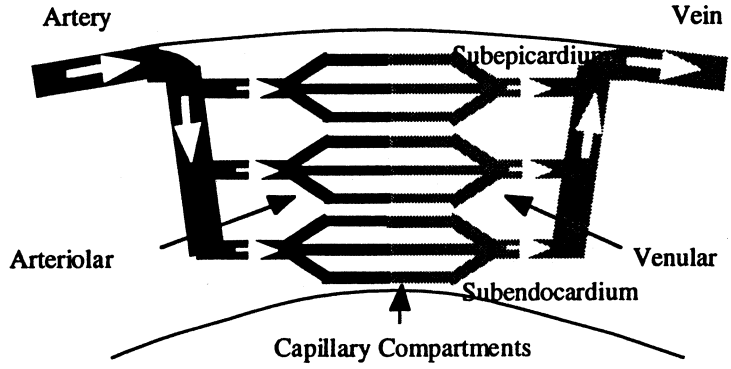

Figure 1. Three layered model of coronary circulation. Each layer consists of three compartmental models, i.e., arteriole, capillary and venule.

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2. Previous model without autoregulation

Myocardium is divided into three layers as shown in Fig.1. Each layer consists of three compartmental models, i.e., arteriole, capillary and venule[3]. Figure 2 shows a compartmental model of blood vessel.

Resistance is inversely proportional to sequence of the compartmental volume V,

$$R=k_r/V^2 \qquad (1)$$

where k_r is a constant. As Bruinsma indicated, V is related to the transmural pressure P_{tr}, intravascular pressure P_{in} and vascular elasticity[6],

$$P_{tr}=P_{in}-P_{im}=a\mathrm{Exp}(Ke(t)V) +b\mathrm{Log}(Ke(t)V)-P_0 \qquad (2)$$

where a, b and P_0 are constants, and Ke(t) is given as follows,

$$K_e(t)=K_0+\Delta K(1-\cos(\pi t/T_s)) +1-\cos(2\pi t/T_s))/2 \quad for\ systole\ t\le T_s \qquad (3a)$$

$$K_e(t)=K_0+\Delta K(1-\cos(\pi t/T_s))/2 \quad for\ early\ diastole\ t\le T_s \qquad (3b)$$

$$K_e(t)=K_0 \quad for\ late\ diastole\ t\le T \qquad (3c)$$

where K_0 is time-invariant component and ΔK is the maximum time-variant component. Ts and Td are the duration of systole and diastole, respectively, and t is the time.

3. Model with autoregulation

When oxygen demand is constant, the autoregulation tries to keep mean arteriolar blood flow constant at a reference volume, even if the mean purfusion pressure changes one value to another.

We modified our previous model, such that autoregulation mechanism can be simulated. In the new mechanism can be simulated. In the new model, K_0 is replaced by the following formula so that flow is controlled,

$$K_0=K_{0ref}\{1+\alpha(F-F_{ref})^n\} \quad for\ F\ge F_{ref} \qquad (4a)$$

$$K_0=K_{0ref}\{1-\alpha|F-F_{ref})|^n\} \quad for\ F<F_{ref} \qquad (4b)$$

where F is the mean flow, F_{ref} is the mean flow at the reference state, K_{0ref} is the value of K_0 when $F = F_{ref}$, and n and α are parameters.

The value of mean F is obtained by solving the dynamical equations (1) to (4) for given coronary artery pressure and left ventricular pressure. If simulated flow exceeds the value at maximal dilatation, F is replaced with the value.

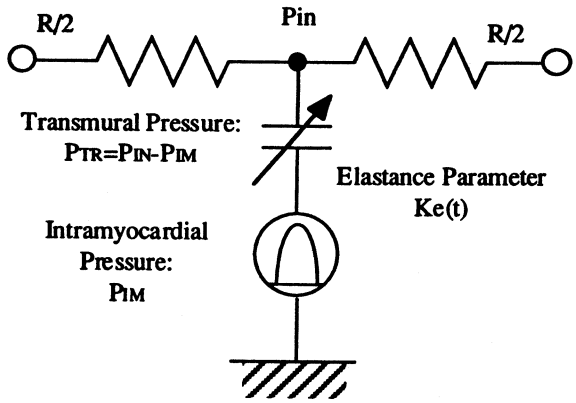

Figure 2. Compartmental model of blood vessel. R is vessel resistance.

4. Results

We input a typical pulsatile coronary arterial flow and left ventricular pressure, which were measured in our laboratory. The sum of three reference values becomes 1.7ml/s/100g. The values of constants in (2) were set based on published data[5]. K_{0ref} was determined so that F_{ref} becomes 1.7ml/s/100g.

Simulation were performed for n=1/10, 1/3, 1 and 3, and α=0.3 to 1. The results for the case that n=1/10 and α=0.5 are shown in Fig.3. Mean flow vs. pressure curve by our simulation agreed well with that of the published autoregulated coronary flow[6].

Figure 4 shows a phasic waveform of coronary arteriolar flow in three layers when purfusion pressures are 30, 50, and 100mmHg. Both systolic retrograde flow and diastolic forward flow in subendcardium were increased with the decrease of purfusion pressure. These results also showed good agreement with our previous study in which the degree of stenosis was increased stepwise[3].

The simulated relative diameter maximal in a cardiac cycle normalized with the value in the reference purfusion pressure P=100mmHg were 1.16,1.14, 1.10 and 0.92 in all layers for Pa=30, 50, 70 and 120mmHg, respectively. The changes of diameters were quite small, coinciding well with the experimental observation[7]

5. Concluding remarks

The present model was found to be useful to simulate autoregulated coronary flow. This model is promising to analyze the effect of autoregulation on the stenosed coronary flow and autoregulated flow in more microscopic level. In future we need to develop our model so that arteriolar compartments are subdivided into more segments.

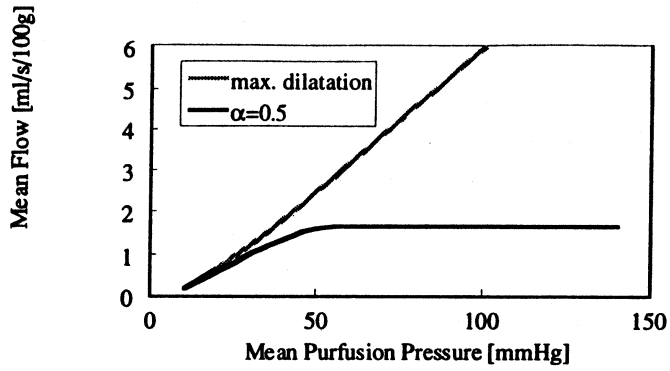

Figure 3. Relation between mean blood flow and purfusion pressure obtained by simulation. α is a parameter expressing the intensity of control element appeared in (4).

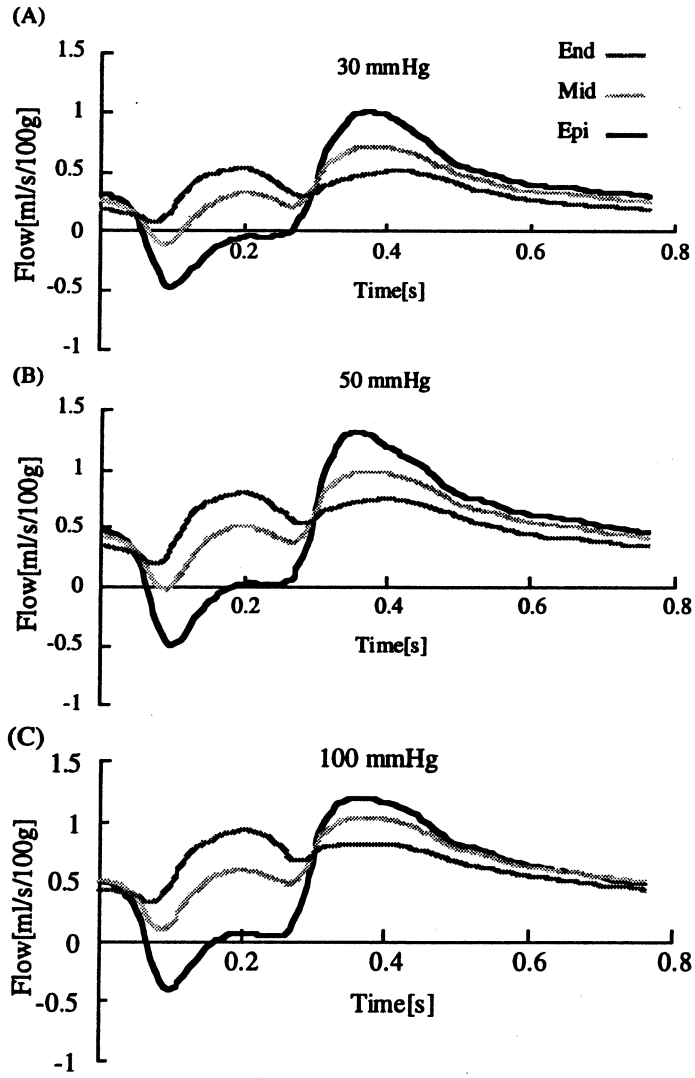

Figure 4. A phasic waveform of coronary arteriolar flow in three layers at purfusion pressures (a) 30mmhg, (b) 50mmHg and (c) 100mmHg. End, Mid and Epi means endocardial, midcardial and epicardial layer, respectively.

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