

A NUMERICAL MODEL OF LEFT VENTRICLE AND AORTIC VALVE FUNCTION OF ITS AFTERLOAD (I)

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(Received Dec. 18, 1987; Communicated by Wu Wang-yi)

Abstract

Due to the study of the function of heart and aortic valve, we set up a physical model of left ventricle, aortic valve and afterload and derive theoretical equation of each part from the model. Then we calculate the basic equations within physiology and impair parameters. Based on this, we will discuss fully in the next paper the effect of left ventricular afterload on valve opening, ejection and valve function, etc.

I. Introduction

Human heart is composed of four elastic chambers(left and right atria and left and right ventricles) and four valves (mitral valve, tricuspid valve, aortic valve and pulmonic valve). Aortic valve plays one-way switch role in blood flow. The four-chambered mammalian heart is actually two pumps lying side by side but hydraulically connected in series along a circular pathway so that the output of each pump eventually becomes the input of the other. Each pump ejects the same volume of blood per minute, or cardiac output, although there are small beat-to-beat variations. The heart's role as a pump propels blood to circulate for regularly systolic and diastolic motion and aortic valve's one-way flow. Generally speaking, we can get to know normal aortic valve's function and foresee its impair with hemodynamic parameters (pressure, flow, power, the area at the valve orifice and the cusp tip, etc.), which can provide guidance for our designing artificial heart and valve. It can be foreseen that left ventricle and its afterload play an important role on hemodynamic parameters apart from the valve characteristic itself. Afterload is bound to affect valve characteristics, especially valve function. It is dispensable that we study the effects of valve function on afterload for our coming to know that fluid dynamic changes result from the impair function valve and for designing and applying artificial valve. Because we mainly discuss the effects of aortic valve function on afterload, we study the whole effect of afterload instead of some parts of hemodynamics. People often adopt elastic chamber, which can represent all the characteristics of arterial tree. Lewis K. Waldman^[1] once used four-element model (see figure 2), but there is a 1% error in its input impedance compared with actuality. For instance, impedance modulus rises with frequency rising in higher frequency, the time of its minimim valve doesn't conform to reality and its phase doesn't rise with frequency rising and so on. To compensate for the shortcoming, this paper

adopts Noordergraaf's model (1978) (see figure 4), the input impedance of which more tallies with reality. Besides this, we use the modified model which Y.C. Fung^[2] set up in 1970 in the part of left ventricle and use one-dimensional flow model which Lee and Talbot^[3] set up in 1979 in the part of aortic valve. Following that, we set up physical model of left aortic valve and afterload and derive basic equation of each part from the model, then we calculate the basic equations within physiology and impair parameters. Based on this, it will be discussed fully in the next paper that left ventricular afterload influences valve opening, ejection and valve function, etc. The conclusions represent that every parameter of left ventricular afterload has influence on left ventricle, it results in cardiac output and external cardiac power reducing but ventricle and afterload average pressures rise with total peripheral resistance R or aortic resistance Z_c rising or with aortic and big arterial compliance C_1 or small arterial compliance C_2 or aortic inductance I_a reducing. Blood energy in valve rises with every parameter above rising.

II. Ventricular Afterload

It is known to us all that heart blood ejection of discontinuity changes into continuous flow as artery is elastic though heart ejects blood periodically. Generally speaking, we have Windkessel model^[4] modify arterial system for which, despite its simplicity and attendant limitations, continues to be extremely useful both for understanding vascular physiology and for investigating ventricular/vascular coupling, so the model is current since the nineteenth century.

To raise precision, many Windkessel models have been set up, for instance, classical Frank's model (two-element model). Westerhof's model (three-element model), Goldwyn's and Watt's (1967)^[5] model (four-element model). We use these models' input impedance to judge which most conforms to reality. For Goldwyn and Watt's model-four-element model (see figure 2) and Noordergraaf's model-five-element model (see figure 4), their impedance modulus and phases are shown in figure 6 and figure 5^{[6],[7]}.

A series of mammalian ascending aortic input impedance is shown in Fig. 7^[7], which represents that the input impedance modulus of human being or animal starts from maximum in zero frequency to reduce rapidly and then to swing up and down with frequency rising and finally to tend to a steady value, which is called characteristic impedance Z_c . Its phase starts fast from zero in zero frequency to lower and to cross zero point when the impedance gets the first minimum and then to tend to zero with frequency rising.

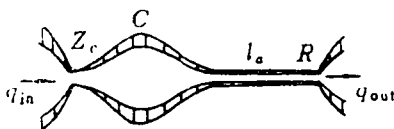


Fig. 1

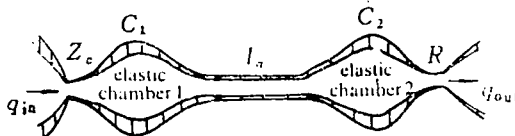


Fig. 3

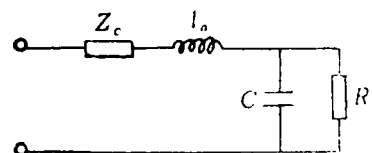


Fig. 2 Goldwyn and Watt's model

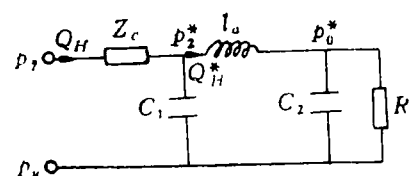


Fig. 4 Noordergraaf's model

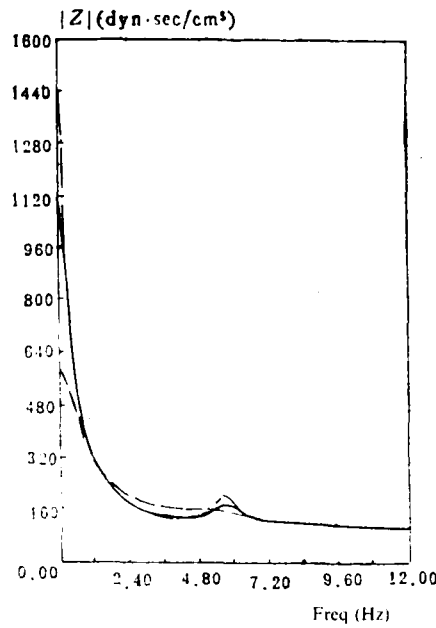


Fig. 5

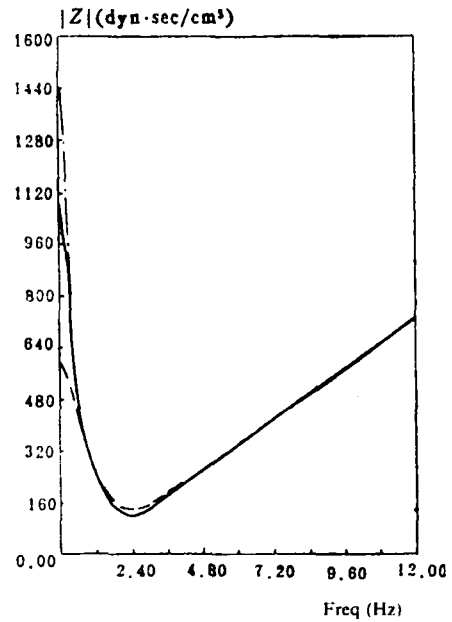
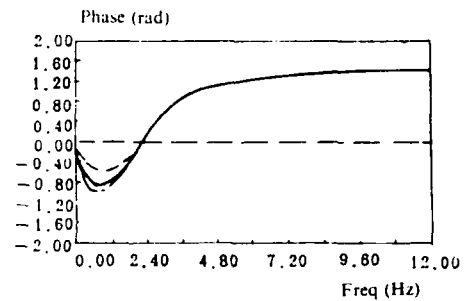
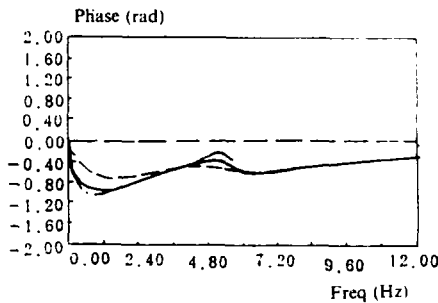


Fig. 6



We find it doesn't accord with reality at best after the figure of Goldwyn and Watt's model is compared with that of reality, so it will make a greater error when the model is used. It is interesting that Noordergraaf's model is a great improvement on the four-element model. Its input impedance more tallies with reality. Below we make use of the model as ventricular afterload. Equivalent circuit and pressure and flow in every point are shown in Fig. 4. We find

$$Z_c = \frac{p_z - p_z^*}{Q_H} \quad (2.1)$$

$$q_{in} = q_{out} + \frac{dV_1}{dt} \quad (2.2)$$

where q_{in} and q_{out} denote the rate of flow into the system from ventricle and from aorta and big arterial system into small arterial system, respectively. V_1 denotes the volume of the arterial system. The total arterial compliance is defined as

$$C = \frac{dV}{dp} \quad (2.3)$$

$$\frac{dV_t}{dt} = C_1 \frac{dp_i^*}{dt} \quad (2.4)$$

According to eqs. (2.2) and (2.4)

$$Q_H = Q_H^* + C_1 \frac{dp_i^*}{dt} \quad (2.5)$$

Eq (2.1) changes into

$$p_i^* = p_2 - Q_H \cdot Z_c \quad (2.6)$$

Substituting (2.6) into (2.5)

$$Q_H^* = Q_H - C_1 \frac{dp_i^*}{dt} = Q_H - C_1 \frac{d}{dt} (p_2 - Q_H \cdot Z_c)$$

we have

$$Q_H^* = Q_H - C_1 \left(\frac{dp_2}{dt} - \frac{dQ_H}{dt} Z_c \right) \quad (2.7)$$

the p_0^* point between C_2 elastic chamber and peripheral resistance R uses the continuity equation

$$q_{in} = q_{out} + \frac{dV_2}{dt} \quad (2.8)$$

and total peripheral resistance R

$$R = \frac{p_i^* - p_v}{q_{out}} \quad (2.9)$$

Therefore

$$Q_H^* = \frac{p_i^* - p_v}{R} + C_2 \frac{dp_i^*}{dt} \quad (2.10)$$

where p_v denotes the effective downstream pressure. Usually this is assumed to be mean venous pressure and is ignored now.

Inductance is defined as

$$p_i^* - p_v^* = l_a \frac{dQ_H^*}{dt} \quad (2.11)$$

Eq. (2.10) is changed into

$$Q_H^* = \frac{p_i^* - l_a \frac{dQ_H^*}{dt}}{R} + C_2 \frac{d}{dt} \left(p_i^* - l_a \frac{dQ_H^*}{dt} \right)$$

$$\frac{dp_i^*}{dt} = l_a \frac{d^2 Q_H^*}{dt^2} + l_a \frac{dQ_H^*}{dt} \frac{1}{RC_2} + \frac{Q_H^*}{C_2} - \frac{p_i^*}{RC_2} \quad (2.12)$$

When eqs. (2.6) and (2.12) are combined, we obtain a differential equation with respect to pressure

p_2

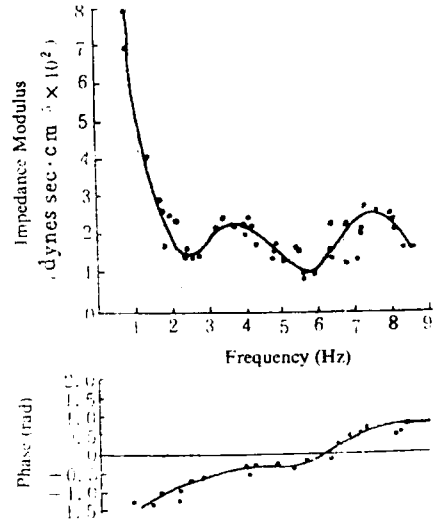


Fig. 7 Aortic input impedance of dog

$$\begin{aligned}
 \frac{d^3 p_2}{dt^3} = & \left[\left(-I_a \frac{C_1}{RC_2} \right) \frac{d^2 p_2}{dt^2} + \left(-1 - \frac{C_1}{C_2} \right) \frac{dp_2}{dt} + \left(-\frac{1}{RC_2} \right) p_2 \right. \\
 & + (I_a C_1 Z_o) \frac{d^3 Q_H}{dt^3} + \left(I_a + I_c \frac{C_1 Z_o}{RC_2} \right) \frac{d^2 Q_H}{dt^2} \\
 & \left. + \left(\frac{I_a}{RC_2} + \frac{C_1 Z_o}{C_2} + Z_o \right) \frac{dQ_H}{dt} + \left(\frac{1}{C_2} + \frac{Z_o}{RC_2} \right) Q_H \right] \frac{1}{I_a C_1}
 \end{aligned} \quad (2.17)$$

III. Cardiac Mechanics

Below we analyze cardiac mechanics by muscle mechanics.

Hill's equation^[8] (due to Archibald Vivian Hill, 1938) is the most famous equation in muscle mechanics. This equation is

$$(a+T)(v+b)=b(T_0+a) \quad (3.1)$$

Hill's equation refers to the ability of a tetanized skeletal muscle to contract. It is an empirical equation based on experimental data on frog sartorius muscle and on the equation of balance of energy. Let us recapitulate. Hill's equation refers to a property of skeletal muscle in the tetanized condition. A muscle bundle is fixed at a certain length L_0 . It is then stimulated electrically at a frequency that is sufficiently high to tetanize the muscle to the maximum tension T_0 (henceforth we shall use T for tension in place of P). In this tetanized condition the muscle is released to a new length L , which is smaller than L_0 , at a new tension T which is smaller than T_0 . Immediately after release, the velocity of contraction $v = -dL/dt$ and the tension T are measured. Then an empirical relationship between T and v is Hill's equation (see eq. (3.1)). Especially, $v = v_0 = v_{\max}$, when $T = 0$. We can get the relationship

$$v_0 = \frac{bT_0}{a} \quad (3.2)$$

Assuming

$$C = \frac{T_0}{a}$$

So equation (3.1) can be rewritten as

$$\frac{v}{v_0} = \frac{T_0 - T}{T_0 + CT} \quad (3.3)$$

where v_0 is the maximum velocity when $T=0$.

Hill's equation reveals only one aspect of the muscle behavior. T_0 remedy the shortcomings a more comprehensive approach is needed. Several methods have been proposed. Of these the best known is the Hill's three-element model. Two of the elements are arranged in series: (a) a contractile element, which at rest is freely extendible (i.e. it has zero tension), but when activated is capable of shortening; and (b) an elastic element arranged in series with the contractile element. T_0 account for the elasticity of the muscle at rest, a "parallel elastic element" is added. This parallel element was ignored in Fig. 8 and included in Fig. 9. 1s, 1p and 1c represent the length of series element parallel element and contractile element.



Fig. 8

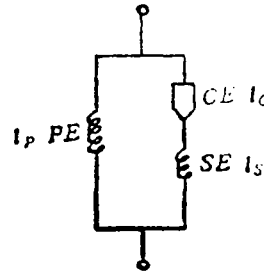


Fig. 9

Most of the authors would identify the contractile element with the sliding actin-myosin molecules, and the generation of active tension with the number of active cross-bridges between them. The series elasticity may be due to the intrinsic elasticity of the actin-myosin molecules and cross-bridges, and of the Z band connective tissues. The parallel elasticity may be due to connective tissues, cell membranes, mitochondria and collagenous sheaths. Assuming that the contractile element contributes no tension in a resting muscle, then the stress-strain-history relationship of a resting muscle determines the constitutive equation of the parallel element.

Y.C.Fung (1970) condensed Edman and Nilsson's experimental results by modifying Hill's equation, then obtained the form (Fung, 1970)^[9] that is called modified Hill's equation

$$v = -\frac{dl_c}{dt} = -\frac{b}{a} \frac{S}{S_0 + S} \left(\frac{f(t) - S_0}{S_0 + S} \right)^n \quad (3.4)$$

where $f(t)$ is a function of time after stimulation. We shall call $f(t)$ the characteristic time function. Y.C.Fung's results suggest that $f(t)$ can be represented by a half-sine wave

$$f(t) = \sin \frac{\pi}{2} \cdot \frac{t + t_0}{t_m} \bigg/ \sin \frac{\pi}{2} \cdot \frac{t_1 + t_0}{t_m} \quad (3.5)$$

In eqs. (3.4) and (3.5) the constants a and b are functions of muscle length L at the time of stimulation, n is a numerical parameter, S_0 is the peak tensile stress arrived at in an isometric contraction of length L , $S_0 = k_1 L + k_2$, k_1 and k_2 are "physiologic" constants, t is the time after stimulus, t_0 is a phase shift related to the initiation of the active state at stimulation, t_p and t_m correspond to be the time to reach the peak isometric tension and the peak tension after the instant of stimulation, and an exponent n is an additional physiologic constant.

From the three-element model, Sonnenblick^[10] connected with the experiment of quick release, isometric contraction and isotonic contraction, and then obtained

$$\frac{dS}{dt} = -\alpha(S + \beta) \quad (3.6)$$

where α and β are physiologic constants, S is series element tension as well as contractile element one.

On the other hand, we can obtain from the figure of three element model

$$l = l_s + l_c \quad (3.7)$$

On differentiating equation (3.7) with respect to time, we obtain the basic kinematic relation:

$$\frac{dl}{dt} = \frac{dl_s}{dt} + \frac{dl_c}{dt} \quad (3.8)$$

and from eq. (3.6) we have

$$\frac{dl_s}{dt} = \frac{dl_s}{dS} \cdot \frac{dS}{dt} = \frac{dS/dt}{\alpha(S + \beta)} \quad (3.9)$$

Substituting equations (3.9) and (3.4) into (3.8)

$$\frac{dl}{dt} = \frac{dS/dt}{\alpha(S + \beta)} - \frac{b[(k_1 L + k_2)f - S]}{\gamma(k_1 L + k_2) + S} \quad (3.10)$$

Usually $L = 1\text{cm}$.

We assume heart ventricle to be spherical. By Laplace relation we get

$$S = \frac{rp}{h} \quad (3.11)$$

Here r, p are the midsurface radius, the internal pressure. h is the thickness from endocardium to epicardium. Pay attention to

$$\frac{l}{L} = 1 + \frac{\Delta l}{L} = 1 + \epsilon_\theta \quad (3.12)$$

When $L = 1\text{cm}$, we have

$$dl/dt = d\epsilon_\theta/dt \quad (3.13)$$

$$\frac{dS}{dt} = \frac{r}{h} \frac{dp}{dt} \quad (3.14)$$

We use the small strain hypothesis above. After combining with eq. (3.10) – (3.14), we get

$$\frac{d\epsilon_\theta}{dt} = \frac{(r/h)(dp/dt)}{\alpha(pr/h + \beta)} - \frac{b[k_1(1 + \epsilon_\theta) + k_2]f - pr/h}{\gamma[k_1(1 + \epsilon_\theta) + k_2] + S} \quad (3.15)$$

As heart ventricle is assumed to be spherical, there is

$$\epsilon_\theta = \frac{w}{r} \quad (3.16)$$

where w and ϵ_θ are ventricular membranous radial direction and strain, respectively. Substituting eq. (3.16) into eq. (3.15)

$$\frac{dp_1}{dt} = \frac{K_1 dw/dt + wabfk_1(p_1 r/h + \beta) + r\alpha(br/h)[f(k_1 + k_2) - \beta - pr/h]p + \alpha\beta b(k_1 + k_2)r}{wr\gamma[k_1/h + r^2(\gamma(k_1 + k_2) + \beta)] + pr/h} \quad (3.17)$$

where

$$K_1 = \alpha \frac{r^2}{h^2} p^2 + \alpha \frac{r}{h} p [\gamma(k_1 + k_2) + \beta] + \alpha\beta\gamma(k_1 + k_2) \quad (3.18)$$

On the other hand,

$$\frac{V}{V_0} = \frac{(r_i + w)^3}{r_i^3} = \left(1 + \frac{w}{r_i}\right)^3 \quad (3.19)$$

where r_i and V_0 represent heart ventricular endocardial radius and volume at the beginning of ejection. V is ventricular volume, so mass conservation for the ventricle demands that

$$dV/dt = -A_i u \quad (3.20)$$

where A_i and u are the area and velocity at the valve orifice. Taking the derivative with respect to time of eq. (3.19), one obtains

$$dw/dt = -\frac{r_i A_i u}{3V_0} \cdot \frac{1}{(1 + w/r_i)^2} \quad (3.21)$$

Now, we obtain three basic first-order nonlinear differential equations (3.17), (3.20) and (3.21) which describe the heart ventricle properties. There are four unknown quantities, so these coupling equations are not closed. The model of the peripheral circulation here must be coupled with the valve model.

IV. Theory of Valve Opening

During its opening, the valve trembles. If we ignore the inertia and blood flow influence of valve, valve opening shows not only the change of area at the cusp but also the influence of valve boundary. And the valve is easy to crack for valve curves. To make the discussion simple, we select the simplest model below. We assume

- (1) Blood is incompressible fluid, and its flow is one-dimensional.
- (2) Velocity and Reynold number are very great during ejection (acceleration is up to $1000 - 5000 \text{ cm/s}^2$), so viscosity is ignored.
- (3) Aortic orifice is constant in area and shape.
- (4) Velocities are constant along the length of the valve. We justify this simplification on the basis that the fluid going through the valve during opening behaves like a jet.
- (5) Valve is made of two stiff thin pieces that have no weight. Valve geometry can be used during opening i.e. the planar straight valve illustrated in Fig. 10.

$2a$ and b_v are the width and breadth of the valve, respectively. At the cusp tips the variable width is $2r_v$, while the breadth remains constant. A_2 is the area at the cusp tip which is a dependent variable. The velocity u is the spatially uniform velocity of the jet. l is the length of the cusps. Here, we restate their continuity equation for opening and follow it with an unsteady Bernoulli equation in order to incorporate the pressure drop needed later for coupling. First, conservation of mass for valve configuration shown in Fig. 10 gives

$$2ab_v u_1 - 2r_v b_v u_2 = \frac{dV_v}{dt} \quad (4.1)$$

in which

$$V_v = (a + r_v) l b_v \quad (4.2)$$

We assume

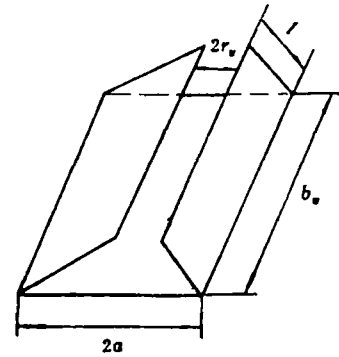
$$u_1 = u_2 = u \quad (4.3)$$

Combining equation (4.1)–(4.3) gives

$$(A_1 - A_2)u = \frac{dV_v}{dt} \quad (4.4)$$

or

$$dA_2/dt = 2(A_1 - A_2) \frac{u}{l} \quad (4.5)$$



u_1, P_1, A_1 , in valve orifice
 u_2, P_2, A_2 in valve exit

Fig. 10

The pressures p_1 and p_2 are uniform pressures at the orifice and cusp tip, respectively, while ρ is the density of blood. An unsteady Bernoulli equation along the central streamline is

$$\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} = - \frac{1}{\rho} \frac{\partial p}{\partial x} \quad (4.6)$$

for assumption

$$\frac{\partial u}{\partial x} = 0 \quad (4.7)$$

Eq. (4.6) changes into

$$\frac{\partial u}{\partial t} = - \frac{1}{\rho} \frac{\partial p}{\partial x} \quad (4.8)$$

$$\frac{du}{dt} = \frac{p_1 - p_2}{\rho l} \quad (4.9)$$

Eqs. (4.5) and (4.9) are basic equations that describe valve opening.

V. Basic Coupling Equations of Heart Ventricle Valve and Vascular System

Above we obtain basic equations of ventricular afterload ventricle and aortic valve. Now we combine with them to get five basic equations that are ordinary differential ones.

$$\frac{dA_2}{dt} = 2(A_1 - A_2) \frac{u}{l} \quad (5.1)$$

$$\frac{du}{dt} = \frac{p_1 - p_2}{\rho l} \quad (5.2)$$

$$\frac{dw}{dt} = - \frac{r_v A_1 u}{3V_0 (1 + w/r_v)^2} \quad (5.3)$$

$$\frac{dp_1}{dt} = \frac{K_1 dw/dt + wabfk_1(p_1 r/h + \beta) + rab(r/h)[f(k_1 + k_2) - \beta - r p/h]p + \alpha \beta f b(k_1 + k_2)r}{w r \gamma k_1/h + (r^2/h)[\gamma(k_1 + k_2) + p r \gamma h]} \quad (5.4)$$

$$\begin{aligned}
\frac{d^3 p_2}{dt^3} = & \left[-l_a \frac{C_1}{RC_2} \frac{d^2 p_2}{dt^2} - \left(1 + \frac{C_1}{C_2} \right) \frac{dp_2}{dt} - \frac{1}{RC_2} p_2 \right. \\
& + l_a C_1 Z_c \frac{d^3 Q_H}{dt^3} + l_a \left(1 + \frac{C_1 Z_c}{RC_2} \right) \frac{d^2 Q_H}{dt^2} \\
& \left. + \left(\frac{l_a}{RC_2} + \frac{C_1 Z_c}{C_2} + Z_c \right) \frac{dQ_H}{dt} + \left(\frac{1}{C_2} + \frac{Z_c}{RC_2} \right) Q_H \right] \frac{1}{l_a C_1}
\end{aligned} \quad (5.5)$$

where

$$K_1 = \alpha \frac{r^2}{h^2} p^2 + \alpha \frac{r}{h} p [\gamma(k_1 + k_2) + \beta] + \alpha \beta \gamma(k_1 + k), \quad Q_H = A_2 u$$

There are five unknown quantities in five coupling equations, so these equations are closed. We can change them into seven first order ordinary differential equations with respect to seven quantities A_2 , u , w , p_1 , p_2 , dp_2/dt , $d^2 p_2/dt^2$. Based on this, we shall calculate these equations numerically and it will be discussed fully in the next paper that left ventricular afterload reacts on valve opening, ejection and valve function, etc. and that the theoretical results are compared with experiments.

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