Proceedings of the 28th IEEE
EMBS Annual International Conference
New York City, USA, Aug 30-Sept 3, 2006

Model-based Analysis of Effects of Systolic Blood Pressure on Frequency Characteristics of the Second Heart Sound

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Abstract—Previous studies have shown that it is possible to estimate pulmonary and systemic blood pressure using the spectral information of the second heart sound. A mathematical model for the vibration of the closed aortic valve is proposed in the paper with the introduction of the nonlinear elasticity of the aortic wall tissue. Based on the parametric analysis, the effect of the aortic blood pressure at the moment of valve closure on vibration of aortic valve and produced sound is examined. The simulation results show that the increasing aortic pressure results in an increase in frequency and amplitude of produced sound. The results of this study also suggest that it is the increasing resonant frequency of the blood column induced by elevated distending pressure that plays significant role in the process. The application of the modeling study could be extended to interpret the relations of the second heart sound to pulmonary blood pressure.

I. INTRODUCTION

Frequency parameters of heart sounds have been gaining recognition as possible indicators of several heart and valve diseases. During past three decades bio-acoustic research has shown promising results in the evaluation of the severity of aortic stenosis, the identification of native and prosthetic heart valve degeneration and dysfunction, and the detection of coronary artery disease. More recently, several new areas of research in spectral phonocardiography have emerged with potential clinical applications. One of most attracting areas is the noninvasive estimation of the pulmonary and systemic arterial pressures using the spectral analysis of the second heart sound (S2).

The second heart sound (S2) marks the end of ventricular systole. It has two high-frequency components, A2 and P2, which relate to aortic and pulmonic valve closure, respectively. It has been known in medical diagnosis that the second heart sound of a hypertensive patient shows a characteristic "accentuation" [1, 2]. The utilization of spectrogram of S2 to estimate the systolic blood pressure (SBP) was investigated by Bartels and Harder using pattern recognition [1]. The technique requires to be calibrated to each individual by simultaneous recording of the PCG and intravascular pressure measurement of the brachial artery. In a recent study, the second heart sound signal was modeled using exponentially damping sinusoids and forward linear predication [3]. Damping and amplitude parameters of the model were shown to have high (statistically significant) correlation to the SBP. These studies indicated that it is possible to develop an alternative approach to measure SBP by the utilization of the frequency characteristics of second heart sound.

In light of the potential importance of these observations, quantitative modeling is an important step to help evaluate the effect of systemic SBP on the production of the second heart sound. In the present study a mathematical model of aortic valve vibration is developed based on the work preformed by Sikarskie et al [4] to evaluate the effects of SBP. It is followed by introducing the nonlinear relationship between the distending pressure and the aortic diameter according to a published experimental stress-strain curve of the aortic wall tissue. Finally, the computational simulation will provide the time course of the aortic pressure produced by vibration of aortic valve under the different SBP.

II. MATERIALS AND METHODS

A. A Mathematical Model of Aortic Valve Vibration

There is a considerable amount of evidence that clearly shows that valvular vibrations are a sufficient cause for the second heart sound [5, 6]. A mathematical model of aortic valve vibration has been developed from first principles by Sikarskie et al [4]. As shown in Fig.1, The model is a one-dimensional idealization (planar velocity profile) which assumes elastic, tapered, circular aorta with an elastic valve at one end (proximal boundary condition) and an assumed pressure at the other end (distal boundary condition). The

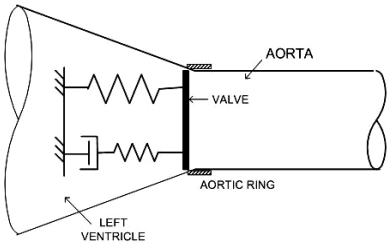

Fig. 1. Idealized one dimensional model of aortic valve vibration [4].

This work is supported by the Hong Kong Innovation and Technology Fund. The authors would like to acknowledge Jetfly Technology Ltd., Golden Meditech Company Ltd., Bird International Ltd. and Bright Steps Corporation for their support to the study.

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driving force on the valve is the pressure difference across the valve, i.e. the difference between the aortic pressure and the ventricular pressure.

The blood flow analysis follows the one-dimensional model of Streeter et al. [7] and Schaaf [8]. For an incompressible fluid (blood), and a flexible tube (aorta), the continuity equations and linear momentum are

$$\frac{\partial A}{\partial t}+\frac{\partial (AV)}{\partial z}+q(z,t)=0 \tag{1}$$

and

$$\frac{\partial V}{\partial t}+(1-\beta)\frac{V}{A}\frac{\partial A}{\partial t}+\beta V\frac{\partial V}{\partial z}+S_g g\frac{\partial P}{\partial z}+\frac{1}{\rho A}\tau_0 \pi D=0 \tag{2}$$

where t = time (s); z = distance along aorta from valve (cm); A = A(z, t) = aortic area (cm^2); average velocity $V=V(z,t)=1/A\int V_z dA$ (cm/s); P = P(z, t) = pressure head in mmHg; q = q(z, t) = flux of fluid through aortic wall (cm^2/s); β = momentum correction = $1/A\int (V_z/V)^2 dA$; D = D(z,t) = aortic diameter (cm); ρ = mass density of blood (g/cm^3); ρ_m = mass density of mercury (g/cm^3); Sg = ρ_m/ρ; g = acceleration of gravity (cm/s^2); μ = viscosity (poise) (1 poise = 1 dyn·s/cm^2); and $\tau_0=\tau_0(z,t)$ = wall shear stress (dyn/cm^2). For convenience, β is assumed to be 1.0 in Eq. 2.

An approximated expression for the wall shear stress is adopted from [8], i.e.

$$\tau_0=\frac{8\mu V}{D}+\frac{\rho D}{12}\frac{\partial V}{\partial t} \tag{3}$$

where D = aortic diameter under transmural pressure P. Assuming the aortic tissue is incompressible, the aortic area and pressure relationship is [7]

$$\frac{A}{A_0}=\frac{1}{1-\frac{D_0\rho_m g P}{Eh_0}} \tag{4}$$

where $A_0 = A_0(z)$ = aortic area under zero pressure (cm^2); $D_0 = D_0(z)$ = aortic diameter under zero pressure (cm); h_0, h = aortic wall thickness under zero pressure and pressure P respectively (cm) and E = aortic Young's modulus ($dynes/cm^2$). Setting β = 1, q = 0, and substituting Eqs.3 and 4 into Eqs. 1 and 2, there gives two non-linear coupled equations in V and P as

$$\begin{aligned}&\frac{\partial V}{\partial z}+\frac{S_g g}{a^2}V\frac{\partial P}{\partial z}+\frac{S_g g}{a^2}\frac{\partial P}{\partial t}=0\\ &\frac{4}{3}\frac{\partial V}{\partial t}+V\frac{\partial V}{\partial z}+S_g g\frac{\partial P}{\partial z}+\frac{32\mu}{\rho D^2}V=0\end{aligned} \tag{5}$$

where $a^2=Eh/\rho D$ (a is the speed of a pressure pulse in the artery).

The proximal boundary condition (at the valve) represents the principal point of departure from previous blood flow calculations. The equation of motion of the valve at the beginning of the diastole is [4]

$$M\frac{\partial V}{\partial t}+f(U)=A_v\rho_m g(P-Pv);t>0,z=0 \tag{6}$$

where M = valve mass (g); Av = valve area (cm^2); Pv = Pv(t) = ventricular pressure (mmHg), known U = U(t) = valve displacement = $\int_0^t V(0,t)dt$ and $f(U)$ = valve restoring force (dyn). The nonlinear elastic restoring force in the valve, $f(U)$ was modeled with a parabola based on the experimental pressure versus displacement of the closed valve cusp [4], i.e.

$$f(U)=\begin{cases}Q_0U+Q_2U^2 & U>0\\ Q_0U-Q_2U^2 & U<0\end{cases} \tag{7}$$

The ventricular pressure P_v is assumed to be a function of time as

$$P_v=-\Delta P_v-\frac{dP_v}{dt}t \tag{8}$$

where ΔP_v and dP_v/dt are initial pressure drop and the slope of the linear pressure decrease respectively [9].

B. A Nonlinear Elasticity Relationship

The elastic modulus of aortic wall is considered to be constant in the study of Sikarshie *et al* [4]. The assumption is reasonable only when no considerable variations of SBP are involved. However, biological soft tissue such as the aortic wall is known to be highly nonlinear in nature. When transmural blood pressure increase the aortic elasticity increases as a result of alterations in vessel wall properties. Since a wide range of blood pressure change will be investigated in the present study, the introduction of the nonlinear elasticity of aortic wall is necessary to obtain a full description of aortic hemodynamics.

The experimental stress-strain results of aortic wall tissue have been published in literature [10], as shown in Fig. 2. The challenge has been to incorporate them into computational models.

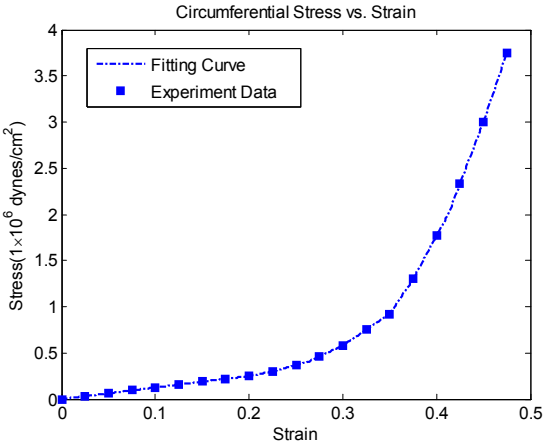

Fig. 2. An experimental stress-strain curve for normal aortic wall tissue described in the literature [10].

For the material having nonlinear elasticity, an incremental elastic modulus at a given point is calculated as the slope of the curve at that point in the stress-strain curve. Furthermore, the relations of transmural pressure to the elastic modulus and aortic radius can be derived.

For a very thin-walled cylindrical shell of radius R, the tension T under the distending pressure P is given by the relation commonly referred to as Laplace's law, i.e.

$$T=PR \tag{7}$$

A small increase in P or δP, will cause an increase in tension, δT, so that

$$(T+\delta T)=(P+\delta P)(R+\delta R)=PR+R\delta P+P\delta R+\delta P\delta R \qquad (8)$$

since δP and δR are small, and the radius change, on a percentage basis, is much less than the pressure change, the term δPδR and PδR is neglected, then

$$\delta T = R\delta P \qquad (9)$$

By dividing through by the wall thickness the change in tensile stress δτ (force per unit area) is obtained

$$\delta\tau = \frac{\delta T}{h} = \frac{R\delta P}{h} \qquad (10)$$

As a consequence of the constant volume of elastic wall material, there is $Rh = R_0 h_0$ under the assumption of Poisson's ratio of 0.50. Thus, we obtain

$$\delta P = E h_0 R_0 \frac{\delta R}{R^2}. \qquad (11)$$

After integrating, the distending pressure, P, is expressed as

$$P = \frac{E h_0 R_0}{2}\left(\frac{1}{R_0^2}-\frac{1}{R^2}\right) \qquad (12)$$

in which the condition has been used that R = R_0 when P = 0. By introducing the strain ε, P is expressed in terms of strain, i.e.

$$P = \frac{E h_0}{2R_0}\left(1-\frac{1}{(1+\varepsilon)^2}\right). \qquad (13)$$

Based on above derivation, the relations of the transmural pressure to the elastic modulus and aortic radius are presented in Fig. 3, providing that the inside radius R under zero distending pressure is 1.33 cm. The results are introduced into Eq.5 and 6 to characterize the nonlinear elasticity of aortic wall tissue.

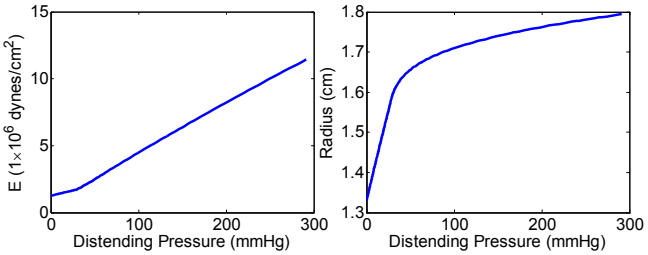

Fig. 3. (a) The relationship between the transmural pressure and the elastic modulus of the aortic wall; and (b) the relationship between the transmural pressure and aortic radius.

C. Sound Produced by Valve Vibration

The next step is to relate the vibration of the aortic valve to the production of the aortic component in the second heart sound. A previous study suggested that it was reasonable to assume that the closed semilunar valve, as it vibrated, acts analogously to a piston [10] [9]. Therefore, the amplitude of sound pressure generated by the aortic valve can be expressed as [11]

$$P_{amp} = \frac{G\pi\omega\rho\dot{x}_0 r}{D} \qquad (14)$$

where P_{amp} = sound-pressure amplitude; D = distance from the valve to the site of measurement of sound pressure; G = proportionality constant; $\dot{x}_0$ = velocity of centerline deflection; r = valve radius; ω = frequency of vibration; and ρ = density of blood (~ 1.05 g/cm^3); (1.1)

III. Results

An initial velocity and pressure distribution are specified as

$$V(z,0)=0 \qquad (15)$$
$$P(z,0)=P_0$$

The initial pressure refers to the aortic pressure at the moment of valve closure. A numerical solution is obtained using the method of characteristics developed in finite difference form as described in [4]. The normal parameters and other conditions are listed as following: ΔP_v = 0.25 cmHg; dP_v/dt = 2000mmHg/s; Δt = 0.0002 s; Δz = 5 cm; R0 = 1.33 cm; $2Q_0$ = 0.547 ×10^6 dyn/cm^2; $2Q_2$ = 45.4 ×10^6 dyn/cm^2; ρ = 1 g/cm3; ρ_m = 13.6 g/cm^3; S_g = 13.6; M = 5 g; Av = 4.91 cm^{-2}; g = 980 cm/s^2, μ = 0.04 Poise and G/D = 1/20.

Fig. 4 presents the simulated acoustic pressure generated by the valve vibration under different initial aortic pressures which varies from 80 to 140 mmHg in step of 20 mmHg. It is clearly shown that both the amplitude and frequency of the sound increase as the initial aortic pressure increases. The maximum amplitude, AMP, and the period of the sound, T, are defined as illustrated in Fig. 4. The frequency of the sound is characterized by 1/T. Fig.5. shows the trend that the frequency and amplitude of the sound changes with initial

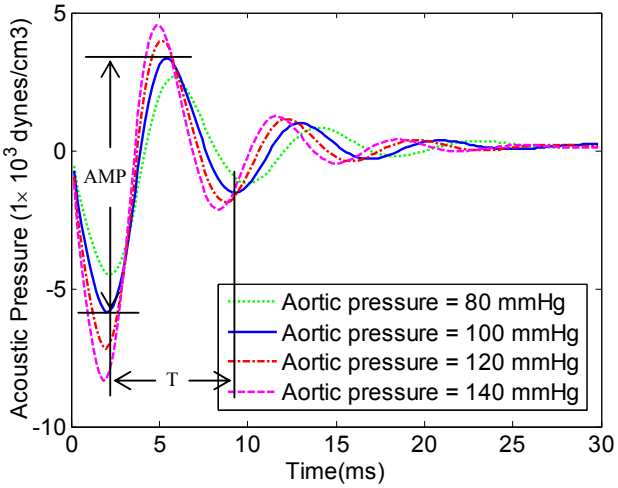

Fig. 4. The simulated acoustic pressure generated by the valve vibration under different initial aortic pressure.

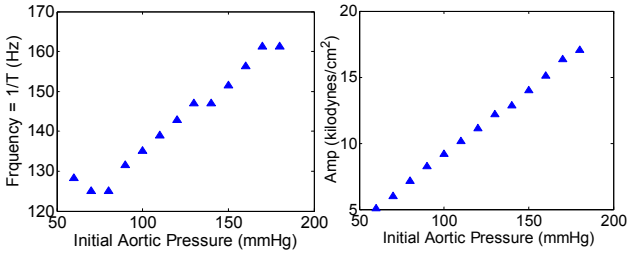

Fig. 5. (a) Effect of initial aortic pressure on the frequency of the second heart sound; and (b) Effect of initial aortic pressure on the amplitude of the second heart sound obtained from simulation.

aortic pressure. It is in agreement with the clinical finding that the second heart sound of a hypertensive patient shows a characteristic 'accentuation'. However, the theoretical frequencies derived from the model are somewhat higher than shown in the analysis of the data acquired from patients [12].

IV. Discussion

A mathematical model of aortic valve vibration has been developed based on the work carried out by Sikaskie and his colleagues [4]. The major improvement of the model in the present study is the introduction of the nonlinear elasticity of the aortic wall tissue. It is of great significant to obtain reliable theoretical results from the simulation in that the aortic wall tissue in nature has a nonlinear elasticity which will be exhibited clearly during large variation of distending pressure.

The contribution of the present study is that it is the first time to investigate the effect of aortic pressure on the second heart sound production from theoretical point of view, which is helpful in interpreting the clinical findings. The generation of the pulmonary component in the second heart sound has a similar mechanism with the generation of aortic component so that this study could extend to interpret the relations of the frequency characteristic of the pulmonary component to pulmonary blood pressure.

The previous study preformed by Sikarskie et al. has identified that the stiffness of the valve, the effective mass of the valve, and rate of development of a pressure drop across the valve after closure could affect the frequency of the vibration [4]. All of these studies have been preformed on the basis of the same initial aortic pressure at 100 mmHg and the assumption that the aorta is linearly elastic. In order to study the effect of aortic pressure variation, we have introduced the nonlinear elasticity based on the experimental stress-strain curve of the normal aortic wall tissue. With the experimental curve, the relations of aortic pressure to the elastic modulus and aortic radius have been derived. The results of the simulation indicate that the oscillation of blood column plays the significant roles in affecting the frequency of vibration, while the effect of valvular stiffening caused by increased tension is neglectable. It is also shown that in addition to the material and functional factors identified in previous studies the aortic pressure is also an important factor that significantly affect the frequency and amplitude of the sound produced by the vibrations of the aortic valve.

The theoretical frequencies derived from the model are somewhat higher than those obtained from patient's data. It is partly because the current model is one-dimensional that gives a maximum simplification of the valve and blood interaction in the aorta. It is also possible that the high frequency components of the sound were attenuated during transmission. The measurement of blood pressure using spectral information of external recorded heart sounds still needs a careful examination because the heart-thorax acoustic system plays significant roles for the transmission of the heart sounds.

In conclusion, a mathematical model for the vibration of the closed aortic valve has been developed in this study. The nonlinear elasticity of the aortic wall tissue has been introduced into the model. Based on the parametric analysis, the effects of the aortic blood pressure at the moment of valve closure on vibration of aortic valve and produced sound have been examined. It is demonstrated from the theoretical point of view that the increasing aortic pressure results in an increase in frequency of vibration and amplitude of produced sound. The results of analysis also suggest that it is the increasing resonant frequency of the blood column induced by elevated distending pressure that plays significant role in the process. However, the feasibility and simplification of the techniques for estimating of blood pressure based on frequency characteristic of second heart sound with simple calibration procedure still require further investigation.

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