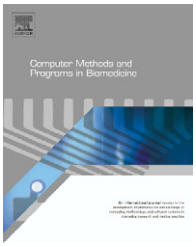
COMPUTER METHODS AND PROGRAMS IN BIOMEDICINE 109 (2013) 190–196

journal homepage: www.intl.elsevierhealth.com/journals/cmpb



A simplified model for mitral valve dynamics

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ARTICLE INFO

Article history:
Received 3 May 2011
Received in revised form
27 October 2011
Accepted 27 October 2011

Keywords:
Cardiac cycle
Mitral valve
Non-linear rotational spring
Damping
Hysteresis

ABSTRACT

Located between the left atrium and the left ventricle, the mitral valve controls flow between these two cardiac chambers. Mitral valve dysfunction is a major cause of cardiac dysfunction and its dynamics are little known.

A simple non-linear rotational spring model is developed and implemented to capture the dynamics of the mitral valve. A measured pressure difference curve was used as the input into the model, which represents an applied torque to the anatomical valve chords. A range of mechanical model hysteresis states were investigated to find a model that best matches reported animal data of chord movement during a heartbeat. The study is limited by the use of one dataset found in the literature due to the highly invasive nature of getting this data. However, results clearly highlight fundamental physiological issues, such as the damping and chord stiffness changing within one cardiac cycle, that would be directly represented in any mitral valve model and affect behaviour in dysfunction. Very good correlation was achieved between modeled and experimental valve angle with 1–10% absolute error in the best case, indicating good promise for future simulation of cardiac valvular dysfunction, such as mitral regurgitation or stenosis. In particular, the model provides a pathway to capturing these dysfunctions in terms of modeled stiffness or elastance that can be directly related to anatomical, structural defects and dysfunction.

1. Introduction

Valvular dysfunction is a relatively common and costly heart disease, typically requiring mechanical valve replacement. It has two primary forms, stenosis and regurgitation. Mitral stenosis is the abnormal narrowing of the mitral valve, which slows blood flow and is the only heart disease that is caused predominately by rheumatic fever. Mitral stenosis accounts for 10% of single native valve diseases [1]. Mitral regurgitation is more common in the elderly and is the leaking of blood through the mitral valve from the left ventricle into the left atrium. Regurgitation occurs due to a dysfunction of the valve leaflets, papillary muscles or the chordae tendineae, and has an occurrence of 2% [2].

The mitral valve separates the left atrium and ventricle. When functioning correctly, it allows blood to flow from the atrium to the ventricle during diastole, and prevents it flowing back during systole, thus maximizing flow out of the left ventricle into the systemic circulation. For a normal mitral valve, about 70–80% of the blood flow occurs during the early filling phase of the left ventricle. After this phase, the left atrial contraction contributes approximately 20% more to the volume in the left ventricle prior to mitral valve closure and ventricular

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doi:10.1016/j.cmpb.2011.10.012

systole. Similar behaviour is seen in the right ventricle with the tricuspid valve.

Abnormal dynamics in this filling phase in either ventricle may suggest valve dysfunction, but could also suggest a wider problem with the circulation, for example pulmonary embolism [3]. Pulmonary embolism is often characterised by tricuspid regurgitation and causes the secondary effect of increased volume in the atrium and/or ventricle [3]. Thus, significant research concentrates on the understanding of detailed flow dynamics and pressure around the valves and atria.

Methods for measuring flow include Doppler echocardiography [4–6], cineangiography, and cinefluoroscopy with radiopaque marker implantation [7]. All of these methods are either invasive or complex and time-consuming. Hence, none lend themselves to regular use for ongoing patient monitoring in regular clinical care.

Another approach for characterising valvular dysfunction is through physical models [8,9], or, more commonly, fluid flow modeling around the valves. Flow around the valves can be turbulent and represent a highly non-linear system that is challenging to model. Current models of this type are usually based on Navier–Stokes [10], or more simplified models of Bernoulli flow [11,12]. However, all of them require difficult to obtain, detailed geometric information to construct, and are very difficult to validate. Thus, they too are not useful for patient-specific regular clinical monitoring or diagnosis.

This research uses simple dynamic models of the stiffness of the valve leaflets to characterise the fundamental effects on flow and pressure, rather than concentrating on highly detailed fluid flow models. The valve is treated as a non-linear rotational spring or a 'hinge' with the change in angle under pressure driven flow being related to the stiffness and damping of the valve. Thus, the essential flow and pressure dynamics can potentially be used to back calculate the strength of the different chord structures in the valve, giving a physiological measure of valve disease with respect to the overall non-linear stiffness defined. Perhaps more importantly, this approach has the potential to directly relate valvular dysfunction to the anatomical, structural defects that cause it, via such simplified models. This model and the methods developed are tested and compared with clinical data from literature for initial proof-of-concept validation.

2. Methodology

2.1. *Generalised model*

The model treats the valve's fundamental response to pressure as a non-linear rotational spring or a hinge with a stiffness force dependent on the angle. Fig. 1 shows this concept schematically as a piecewise linear function, where the K_3 stiffness acts to limit valve opening at maximum angle.

In the first section, $0<\theta<\theta_1$, the force changes rapidly with a slope $K_1 = F_K(\theta_1)/\theta_1$, which corresponds to the stiffness of the valve during this period. In the middle section, $\theta_1<\theta<\theta_2$, the stiffness is much lower, and in the final section, $\theta_2<\theta<\pi/2$, the stiffness is high again. The $F_K(\theta)$ curve in Fig. 1 could also be represented by many more piecewise linear sections, which, in the limit, would define a continuous curve, with the stiffness as a continuous function of θ defined:

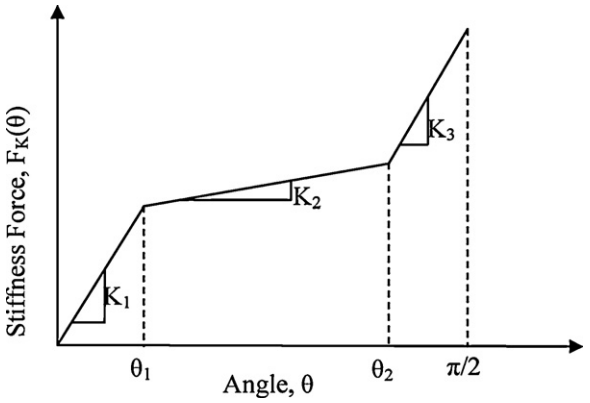

Fig. 1 – Force as a function of angle, representing three different stiffness profiles.

$$K(\theta) = \frac{dF_K(\theta)}{d\theta} \tag{1}$$

The following continuous description of $F_K(\theta)$ is used, as it can capture the fundamental dynamics illustrated in Fig. 1.

$$F_K(\theta) = a_1 + a_2\theta + a_3\ e^{-b_1\theta} + a_4\, e^{-b_2\theta} \tag{2}$$

yielding $K(\theta)$:

$$K(\theta) = \frac{dF_K(\theta)}{d\theta} = a_2 - b_1 a_3\ e^{-b_1\theta} - b_2 a_4\, e^{-b_2\theta} \tag{3}$$

For example, substituting $a_1 = -a_3 - a_4$, $a_2 = 0.04$, $a_3 = 0.002$, $a_4 = -0.35$, $b_1 = -3.7$, $b_2 = 11$ into (2), yields the force versus θ curve in Fig. 2. Note that in this case, the force dramatically increases for $0<\theta<0.2$, has a small slope for $0.2<\theta<1$, and increases more rapidly again for $1<\theta<\pi/2$. It thus captures the fundamental behaviour of Fig. 1. The corresponding stiffness, $K(\theta)$ from (3) is also shown in Fig. 2. Note that in Figs. 1 and 2, the stiffness in the first and last sections is assumed higher

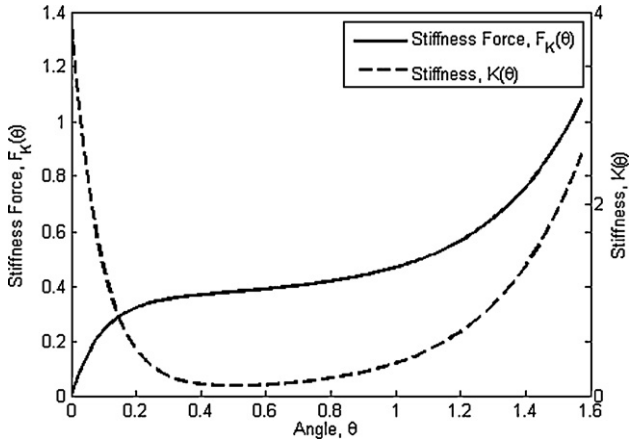

Fig. 2 – Force as a function of angle (continuous curve) for $a_1 = -a_3 - a_4$, $a_2 = 0.04$, $a_3 = 0.002$, $a_4 = -0.35$, $b_1 = -3.7$, $b_2 = 11$, and corresponding stiffness profile.