Solutions to the Van der Pol Equation: a Model of Aortic Blood Flow

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Abstract—**Quantitative modeling, of large arteries, plays an important role in predicting and describing functional hemodynamic components. Here we present a descending thoracic aortic model based upon the nonlinear Van der Pol equation. The model is created by modification of the solution to this second order differential equation. The model displays a stroke volume of 97.82 ml and an average velocity of 22 cm/s for a heart rate of 70 bpm. An aortic radius of 1.16 cm is assumed.**

I. Introduction

Methods to model the cardiovascular system have taken an electrical form, namely the use of capacitance, resistance and inductance (inertance) to describe the arterial properties. The Windkessel model has been a widely accepted tool for analysis of the relationship between the components of an electrical circuit [4]. Generally attributed to Frank (1899), the Windkessel model can be described as an RC circuit [4]:

$$1/RC = - dP/PdV * dV/dt \qquad (1)$$

Modifications to this basic electrical model include nonlinear descriptions of compliance (8).

In 1926, the Dutch physicist Balthazar Van der Pol described a negative resistance oscillator represented by:

$$v'' - \alpha (1 - v^2)v' + \omega^2 v = 0 \qquad (2)$$

Van der Pol essentially described a tunnel diode or vacuum tube (3) in which resistance is a nonlinear function of current. Two years later, Van der Pol further applied this negative resistance oscillator to model the heartbeat as a function of circuits describing the sinus node, auricles (atria) and ventricles. The result of his model was an electrocardiograph that included both P waves and QRS complexes despite lacking the T waves of repolarization.

It is interesting to note that this electrical model shows a refractory period in which the condenser (capacitor) is not yet charged and thus will not excite the oscillator when an impulse is added. This mimicks the "all or nothing" response of the ventricles [2].

In 1969, William Conrad investigated pressure-flow relationships in collapsible tubes. His experiments showed that during steady flow, partially collapsed tubes exhibit negative resistance in pressure-volume plots, and noted a conversion of dc power (steady) to ac power (oscillatory). In his experiments, the pressure drop, for a given volumetric flow rate, exhibited a nonlinear flow during partial collapse.

Conrad established a link between a partially collapsed tube and Van der Pol oscillators succinctly, in which *v* of equation (4) describes dimensionless flow during a period of dimensionless time. Conrad suggested that this phenomenon can be applied to veins, hepatic venules and coronary arteries due to the periodic changes in transmural pressures in those areas. Moreover, the oscillation frequency depends on compliance and fluid inertance.

While Conrad's work describes steady flow, he asserted that nonsteady flows can approximated by a family of nonlinear resistances. Arteries also exhibit nonlinear stress-strain characteristics, mostly dependant upon the constitution of the vascular wall, that indeed vary in the circulatory system [4].

It then follows that a model of flow in the cardiovascular system may be described as a nonlinear resistance oscillator, namely a second order differential equation with dependence on inductance, capacitance and resistance of an analog circuit. This model would be an analogous to Windkessel, defining a nonlinear resistance, rather than compliance to approximate flow. In the current method, blood flow velocity waveforms are generated from the solution set of the Van der Pol equation (2) under certain restrictions and manipulations.

II. Methods

The Van der Pol equation may be solved computationally using a variety of software methods. Here Matlab version 6.5 release 13.01 (Mathworks™) provided the appropriate tools to solve the equation using the ODE45 routine (non-stiff, medium order method) for initial conditions [1 -3] over a span of [0 20]. Parameterizing the coefficients of equation (2) will be necessary to reproduce a graph of aortic blood flow velocity. Equation (2) contains coefficients α and ω^2 in which:

$$\alpha = R/L \qquad (3)$$

$$\omega^2 = 1/LC \qquad (4)$$

$$T_{rel} = 1.61RC \qquad (5)$$

Manipulation of these variables will recreate the characteristics of the waveform. Given the most basic form of equation (2), increasing the ratio, of resistance to inductance, produces changes that increase the period and magnitude of the waveform. Furthermore, increasing ω leads to an increase in the magnitude and a decrease in period.

Following initial parameterization, steps are needed in order to make the waveform physiologically significant. A Matlab M-file served to process the solutions of equation (2)

and produce a more meaningful graph. These steps included numerical addition, to scale the oscillations around a baseline, as well modulation of the oscillations. Modulation was needed to pass every other oscillation, creating the baseline flow and thus a diastolic period. Multiplication of the waveform was done in order to scale the velocities to physiological conditions. Time is then scaled by setting the distribution of points for a given time period. The result is scaled view of the solutions to the Van der Pol equation in which every other oscillation is passed centered about a new baseline. The parameterized equation to govern the model is then

$$v'' - (1-v^2)v' + 5.6v = 0 \qquad (6)$$

The above method is only one of many possible solutions to the second order differential equation. In this investigation, a model of the distal thoracic aorta is used to aid in direction.

III. RESULTS

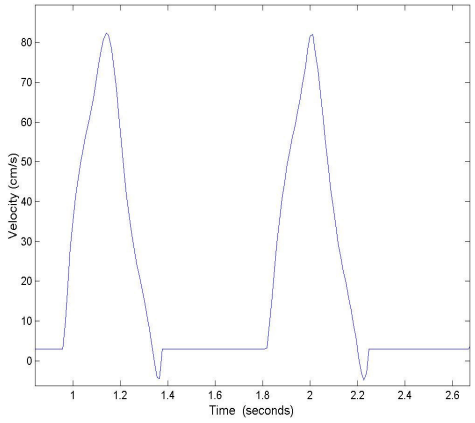

Figure 1. Van der Pol Model of Aortic Blood Flow

Quantity	Value
Heart Rate	69.75 bpm
Period	0.86 sec
Flow Time Flow Time Corrected	0.3871 sec 0.4174 sec
Systolic %	45%
Peak Velocity	82.34 cm/s
Avg. Velocity	21.69 cm/s
Stroke Distance	16.19 cm
Flow Volume	68.47 ml
Stroke Volume	97.82 ml
Cardiac Output	6.8 L/m

Table 1. Van der Pol Model Characteristics (Assuming a radius of 1.16 cm)

IV. EVALUATION

For the model to truly be physiologically accurate, it must contain significant information about the nature of flow. By treating the model as an aortic signal generated noninvasively from a transesophageal Doppler, cardiovascular parameters can be extrapolated. First, the heart rate is determined to be approximately 70 bpm, with a flow time (synonymous with left ventricular ejection time) of .3871 seconds. Alternatively, this corresponds to a systolic time of 45% of the cardiac cycle. This is close to the human cardiac cycle in which the majority of the cardiac period in diastole. Average velocity is calculated to be 22 cm/s, which is slightly higher than averages of 16-18 cm/s in the thoracic aorta [7]. Peak velocity is seen to be 82.34 cm/s, which is certainly reasonable in a vessel where the peak velocity has been noted to be as high as 1 m/s [6]. By applying Doppler analysis techniques, stroke distance is calculated to be 16.19 cm [6]. This also is useful in quantifying left ventricular function. Assuming an aortic radius of 1.16 cm from the thoracic aorta [5] and that 30% of blood flows to the head and upper extremities [7], stroke volume is determined to be 97.82 ml producing a cardiac output of 6.8 L/min. These values correlate well with physiological conditions of 53-133 ml stroke volume and 3.6-9.5 L/m of cardiac output in man [4].

V. DISCUSSION

The comparable nonlinear features, of both the cardiovascular system and the Van der Pol equation, make the current model applicable to physiological characteristics. Manipulation of the core equation can yield waveforms which closely match aortic blood flow velocity. This scaling is not solely contrived mathematics since the Van der Pol equation describes dimensionless time and dimensionless flow. The result of the Maltab solution, after manipulation, is a waveform of particularly interesting qualities if we assume a certain vessel radius. From a mathematical waveform analysis, it is evident that the model is a reasonable replication of thoracic aortic blood flow velocity. The model may be adapted to different rates and peak velocities. However, these changes will affect volume flow, as well as peak and mean velocities. Therefore, the physiological significance of these adaptations may limit the application of the model to other vessels in the periphery.

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