

Evaluation of Blood Pressure Pulse Wave Velocity and Arterial Relaxation Constant

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Abstract

The paper discussed the relationship between blood pressure pulse wave velocity and arterial relaxation constant at brachial artery. The Windkessels model of brachial artery was assumed to simulate blood pressure decay waveform. The pulse wave velocity and noninvasive blood pressure wave form were acquired along with invasive aortic pressure. The result showed that the relaxation constant of aortic pressure waveform and brachial blood pressure waveform were -0.5 ± 0.067 and -0.549 ± 0.067 respectively. The relationship of higher pulse wave velocity and relaxation constant was 0.8.

1. Introduction

Non-invasive blood pressure pulse wave velocity has been developed and validated for accessing oscillatory systemic arterial compliance and stiffness for sometimes [1-2]. The pulse waveform analysis using Windkessel's model has discussed extensively to model the blood pressure waveform at locations of various arterial tree. The derivation of model that was proposed using periodic function to describe pulsatile hemodynamics in artery was commonly accepted [3-5]. Based on circuit theorem, arterial pressure wave function could be described as external source to the arterial tree. And, the output could be quantified using Windkessel's model to estimate the systemic arterial function. The decay waveform in Windkessel's model was the characteristic arterial impedance of the model. The decay function of pressure wave was modeled as aortic valve closed. It is similar to the impedance function of capacitor discharge in the Windkessels model. And, the time constant of the decay waveform is the feature of arterial system by the definition of Windkessels model.

On the other hand, there was a transmission line theory that assumed the finite pulse wave velocity in the artery. The spatial and temporal pressure gradients in arterial

system were recognized as the feature of compliance in the artery. That was utilizing the propagation models of delay and wave reflection in blood pressure that derived the pulse wave velocity (PWV) into indexing the pathological and physiological condition of arterial system. The higher the value of PWV is indicating the severer of the arteriosclerosis. The method has been used in screening early evidence of vascular disease and in monitoring the response to the therapy [5-10].

However, using pulse wave velocity to screening the status of subjects was difficult and without physical basis. There was a need to simplify the accessing method using only one pressure cuff. And, to derive an index which has physical meaning to the systemic artery tree. However, the relationship of the relaxation time constant of Windkessel's model and PWV was not described. This study was to correlate the number of PWV and the relaxation constant calculated from the windkessel's model.

2. Methods

The blood pressure waveform from 24 patients using oscillometric method (Colin VP1000) and invasive pressure waveform at aortic root were acquired (mean age 63.2 ± 12.7 years). Their pulse wave velocities were acquired at same the sitting (Colin VP1000). Invasive high-fidelity right brachial and central aortic pressure waveforms were obtained during cardiac catheterization. Individual and central aortic pressure waveforms and the brachial Pressure Volume Relation waveform (PVR) were obtained and recorded simultaneously. The brachial PVR was acquired using fix cuff pressure at 50 mmHg while recording PWV pressure waveform from brachial and femoral artery.

For calculation of PWV, the ECG signal was used as time marker for each heart beat. The timing marker for the pressure waveform was marked at the maximum of

pressure up slope. The PWV was calculated by the time difference between time marker of the brachial arterial pressure waveform and femoral pressure waveform.

To calculate the relaxation constant of the Windkessel's model, the lowest pressure point was marked before the R-peak of ECG signal as the diastolic pressure (DBP) point. The segment of data analyzed was starting from the minimum of down slope of pressure waveform to the DBP point, as shown in Figure 1, the black line.

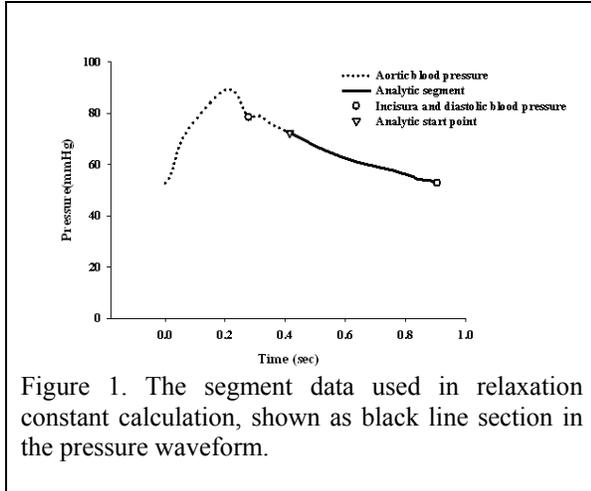


Figure 1. The segment data used in relaxation constant calculation, shown as black line section in the pressure waveform.

The invasive aortic data was used as the reference in this study. The relaxation constant of the aortic pressure waveform was calculated the same as brachial relaxation constant.

The formulas for calculating the the relaxation constant of the Windkessel's model is listed below. The C is the compliance and the R is the resistance. The α is the relaxation constant.

$$P(t) = P_{start} \times e^{\left(\frac{t-t_{start}}{RC}\right)} \quad (1)$$

or

$$P(t) = P_{start} \times e^{(-\alpha t)} \quad (2)$$

$$\frac{P(t)}{P_{start}} = e^{(-\alpha t)} \quad (3)$$

$$\ln\left(\frac{P(t)}{P_{start}}\right) = -\alpha t \quad (4)$$

When calculating the decay function of pressure waveform, one only looks for the dominate term of the model. Therefore, the $-RC$ term, $-\alpha$, in above formulas will be the relaxation constant. Therefore, the segment of analyzed data could be calculated using regression method to find the relaxation constant. The compliance, C, is, then, $1/R\alpha$. The derivation for the resistance is $R = MAP/CO$. The MAP is mean arterial pressure. The Co is

the cardio output. It was assuming a constant stroke volume and the extracted heart rate for the cardio output.

The arterial stiffness index or compliance index using area method assessed from aortic pressure waveform was also calculate. This is the common practice for assessing the compliance of the aorta or artery. It calculates the area ratio cover by systolic waveform and diastolic pressure waveform, as shown in Figure 2.

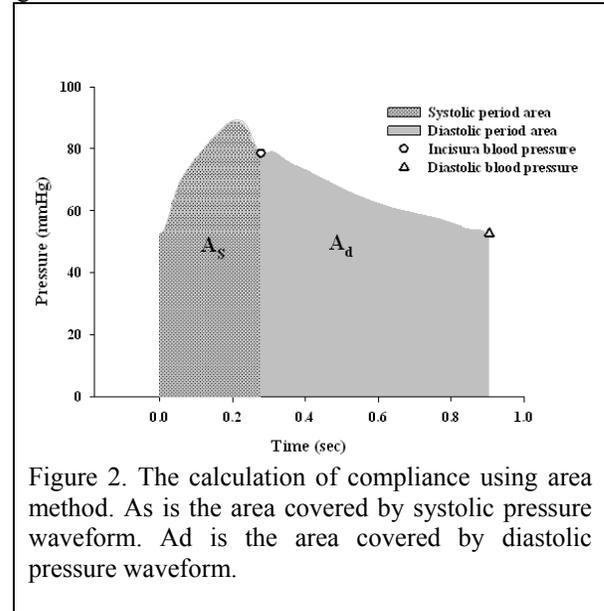


Figure 2. The calculation of compliance using area method. As is the area covered by systolic pressure waveform. Ad is the area covered by diastolic pressure waveform.

$$C = \frac{SV}{K(P_{incisura} - DBP)} \quad (5)$$

$$K = \frac{A_s + A_d}{A_d} \quad (6)$$

The SV is the estimated stroke volume. The DBP and $P_{incisura}$ were extracted from pressure waveform. K is the reciprocal ratio of diastolic area and total area.

3. Results

The mean and standard deviation of the pulse wave velocity was 1595.63 ± 301.56 cm/sec. The mean and standard deviation of the relaxation constant of the brachial arterial BP was -0.5 ± 0.067 and at aortic root was -0.549 ± 0.067 . The correlations of compliance using the relaxation constants method and area method at aortic root was 0.95, for aortic pressure waveform, as shown in Figure3. The R^2 is 0.83. The correlations of compliance using the relaxation constants method and area method at brachial artery was 0.909, for aortic pressure waveform as shown in Figure4. The R^2 is 0.795. The correlation of compliance between aortic and brachial pressure waveform is 0.967, and, the R^2 is 0.97, as shown in

Figure5. These data showed that the arterial compliance could be calculated from relaxation constant as well as using commonly accepted area method.

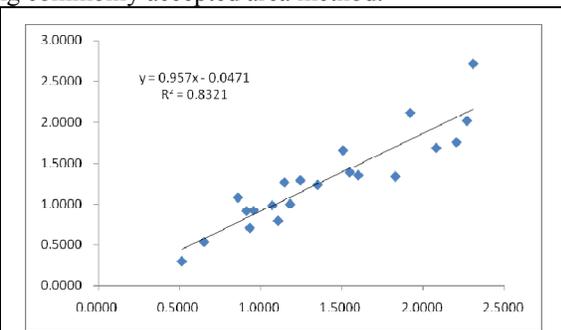


Figure 3. The correlation of compliance between area method and relaxation constant for aortic pressure waveform.

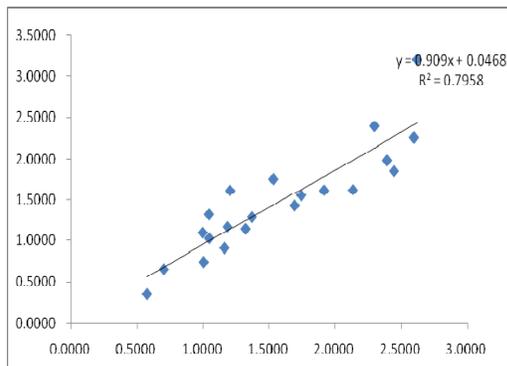


Figure 4. The correlation of compliance between area method and relaxation constant for brachial pressure waveform.

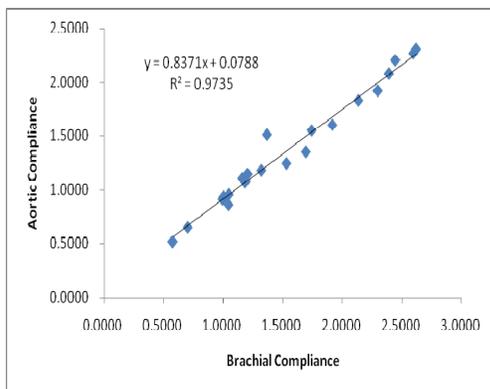


Figure 5. The correlation of compliance between aortic and brachial pressure waveform.

The correlation of the compliance between the PWV and brachial relaxation constant was 0.45. The correlation of compliance between the PWV and brachial using area method is 0.54. And, the correlation is much worst

between relaxation constant and PWV. These results showed that there were no correlation of PWV and the compliance. This result cannot be accepted because it did not reflect the common sense that the stiff vascular has higher PWV.

For a heart rate of 80, the normal relaxation time from the incisura point to the DBP point is less than 0.3 second. If one would have a DBP around 80 mmHg, the relaxation should no larger than -0.6. Therefore, if the one tested the data using the relaxation small than -0.6, the correlation of relaxation constant to the PWV is 0.87, as shown in Figure 6. For the group that the relaxation constant was larger than -0.6, the data showed that the correlation is 0.76, as shown in Figure 7.

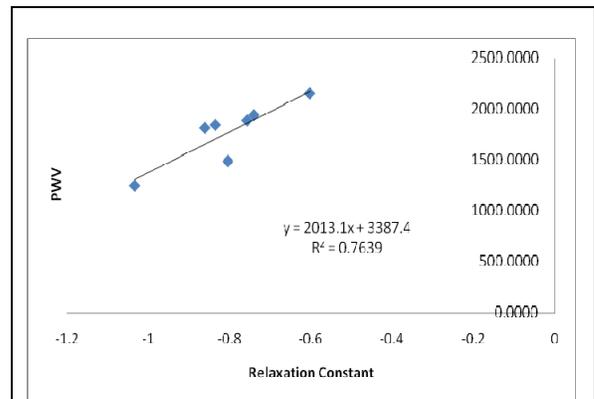


Figure 6. The correlation of relaxation constant and PWV for the group having smaller relaxation constant ($\alpha < -0.6$).

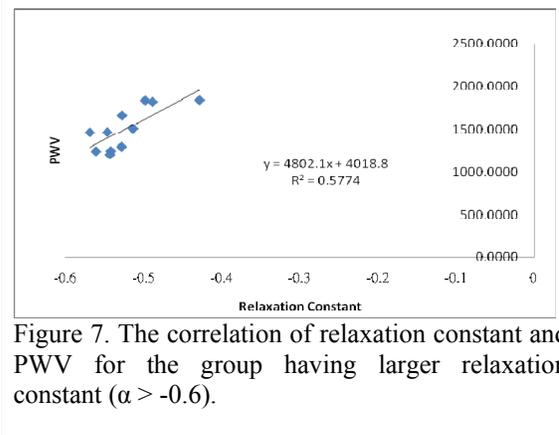


Figure 7. The correlation of relaxation constant and PWV for the group having larger relaxation constant ($\alpha > -0.6$).

These results has illustrated that the relaxation constant could be correlated to the pressure waveform velocity. The healthy and normal artery should have a smaller ($\alpha < -0.6$) relaxation constant.

4. Discussion and conclusions

The result showed that the higher the pulse wave

velocity the smaller the relaxation constant (more negative value). The faster the pulse wave velocity is associated with longer decay time.

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References

- [1] Cohn JN, Finkelstein S, McVeigh G, Morgan D, LeMay L, Robinson J, Mock J. Noninvasive Pulse Wave Analysis for the Early Detection of Vascular Disease Hypertension 1995;26:503-508.
- [2] Asmar R, Benetos A, Topouchian J, Laurent P, Pannier B, Brisac AM, Target R, Levy BI. Assessment of Arterial Distensibility by Automatic Pulse Wave Velocity Measurement. Hypertension 1995;26:485-490.
- [3] Peterson LH, Jensen RE, Parnell J. Mechanical Properties of Arteries in Vivo. Circulation Research 1960;13:622-639.
- [4] Goldwyn RM, WATT TB Jr. Arterial Pressure Pulse Contour Analysis via a Mathematical Model for the Clinical Quantification of Human Vascular Properties. IEEE Transactions on Bio-Medical Engineering 1967;14:1: 11-17.
- [5] McDonald DA. Regional pulse-wave velocity in the arterial tree. J of Applied Physiology 1968; 24:1:73-78.
- [6] Ziemann SJ, Melenovsky V, Kass DA. Mechanisms, Pathophysiology, and Therapy of Arterial Stiffness Arterioscler. Thromb Vasc Biol 2005;25:932-943.
- [7] Pannier B, Guérin AP, Marchais SJ, Safar ME, London GM. Stiffness of Capacitive and Conduit Arteries: Prognostic Significance for End-Stage Renal Disease Patients. Hypertension 2005;45:592-596.
- [8] Weber T, Auer J, O'Rourke MF, Kvas E, Lassnig E, Berent R and Eber B. Arterial Stiffness, Wave Reflections, and the Risk of Coronary Artery Disease. Circulation 2004;109:184-189;
- [9] Avolio AP, Deng FQ, Li WQ, Luo YF, Huang ZD, Xing LF, O'Rourke MF. Effects of aging on arterial distensibility in populations with high and low prevalence of hypertension: comparison between urban and rural communities in China. Circulation 1985;71:202-210
- [10] Qasem A and Avolio A. Determination of Aortic Pulse Wave Velocity From Waveform Decomposition of the Central Aortic Pressure Pulse. Hypertension 2008;51:188-195.

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