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Effect of Upper Arm Cuff Pressure on Pulse Morphology using Photoplethysmography

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Abstract—**Study of Arterial pressure and flow variation when external pressure applied is critical in understanding clinical relevance of pulse. We propose a distributed model of entire human arterial tree to describe the hemodynamic changes due to pressure applied on the brachial artery. Input to this distributed model is from a four element Windkessel model. We measured the blood volume during brachial occlusion in six healthy subjects using finger photoplethysmography (PPG). We analyzed the morphological changes in both the model and the experiment. We found that both the model pressure pulse and the finger volume pulse morphology changing with the applied pressure. We calculated the normalized values of pulse height, maximum systolic slope, maximum diastolic slope and peak to peak interval (PPI) for each beat. Mean values of all the morphological parameters show an initial rise and in the model the maximum occurs at 60-80mmHg while in the experiments it occurs at 40mmHg.The predicted model parameters are positively correlated with measured parameters (for maximum systolic slope r = 0.54; for maximum diastolic slope r = 0.77; for pulse height r = 0.89 when *P* < 0.05). The PPI variation is different for each subject reflecting the reflex properties of the individual. Variable elastance and reflex system should be incorporated in the model to accurately predict the experimental results.**

I. Introduction

ARTERIAL pressure and flow pulse are important resources in the diagnosis of heart diseases. The variations of the pulse shape are strongly related not only to cardiac function but also to the peripheral circulation. Various models have been proposed to simulate human systemic circulation [1]-[4]. Windkessel models, one of the lumped models, are widely used for such simulation [4].Lumped models do not consider the effect of wave reflection and hence such models do not predict amplification, shape alteration in pressure and flow waveforms and variations in input impedance spectra of arterial tree [2],[10]. In order to overcome this limitation distributed parameter model based on transmission line theory can be used [1], [2].

Study of arterial pressure and flow variation when external pressure applied is critical in understanding clinical relevance of pulse. Vascular occlusion induces hemodynamic changes that allow for noninvasive, optical measurements of physiologically important parameters and also useful in designing non-invasive patient monitoring systems. In arterial occlusion applied external pressure should be more than systolic blood pressure, and in venous occlusion it should be above the central venous pressure [5]. Anesthesiologists should be aware of the changes in local vascular resistances and bypass flow due to cuff inflation in the left forearm for the evaluation of left internal mammary artery and left anterior descending artery anastomosis [6]. It has been experimentally observed that, large external pressure, like cuff pressure, has a significantly higher effect on the mean PTT of the upper arm than that of the lower arm [7]. Though such experiments have been reported well in the literature, no distributed model of whole arterial tree is found for such study.

In this work we have analyzed the response of the human arterial system with electrical transmission line model when the external pressure is applied on the upper arm brachial artery. In order to validate the model we used Photoplethysmography (PPG) which is a simple and low cost optical technique that can be used to detect blood volume changes in the micro vascular bed of tissue [8]. We have examined the beat to beat morphological changes such as pulse height, systolic slope, diastolic slope and peak to peak interval in the model as well as in the finger PPG.

II. Methodology

A. Aortic Pressure waveform duplication using a four element Windkessel model

Windkessel models describe the blood flow using closed electrical circuits. The 4-element model as shown in Fig. 1 includes inductance L represents inertia of blood flow which is neglected in the two element and three element Windkessel models [4]. We have used the 4-element model as it predicts better than the two and three element models. We modeled aortic flow as a current source to a Windkessel model. The aortic pressure is the voltage measured across the four elements. This pressure signal is used as input to the transmission line model.

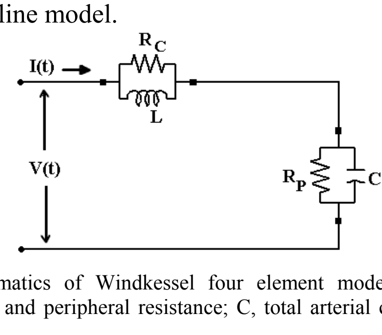

Fig. 1 Schematics of Windkessel four element model Rc and Rp characteristic and peripheral resistance; C, total arterial compliance; L, inertance.

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B. Electrical Transmission Line model

The similarity between the one dimensional *Navier-Stokes* equations of fluid dynamics and telegraph equations of the electromagnetic waves propagation has been long used in modeling arterial system [2].

Fluid dynamic system parameters such as fluid volume, pressure, flow, fluid inertia, hydraulic resistance, compliance and proportionality constant of radial (leakage) flow are analogous with electrical system parameters such as charge, potential, current, inductance, resistance, capacitance and conductance respectively.

The electromagnetic waves propagation is described by equations (1) and (2).

$$\frac{-\delta V}{\delta x} = IR + L\frac{\delta I}{\delta t} \tag{1}$$

$$\frac{-\delta I}{\delta x} = IR + L\frac{\delta V}{\delta t} \tag{2}$$

where V is the electrical voltage, I is the electrical current, R is the electrical resistance, L is the electrical inductance, G is the electrical conductance, x is the distance and t is the time. Using the above equation a hydraulic system can be modeled as an electrical circuit and compliant tubes can be modeled as small segments of transmission line [2].

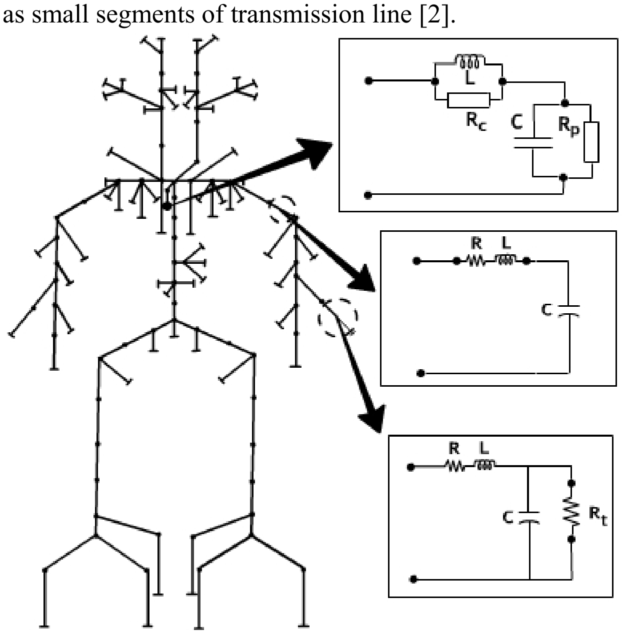

Fig. 2. Schematic model of human arterial tree with 128 electrical equivalent segments. In the inset Windkessel model by which pressure input to the arterial tree is given, brachial artery segment and radial artery segment in forearm are shown.

The *RLC* values can be calculated from the mechanical parameters of compliant tubes using the following relations. $R = \frac{8\mu}{\pi r^4}$, $L = \frac{\rho}{\pi r^2}$, $C = \frac{3\pi r^2}{2Eh}$ where R is the resistance per unit length, L is the inductance per unit length, C is the capacitance per unit length, μ is the blood viscosity, r is the radius of the tube (or artery), ρ is the blood density, E is the *Young's* modulus. We synthesized the model of whole human arterial tree combining 128 individual arterial segments which is shown in Fig. 2. Previously published data were used for finding the parameters R, L, C and R_t [1]. The conductance G which represents leakage is not considered in the model.

C. External cuff pressure as a voltage source

We modeled the external cuff pressure as voltage source in series with a diode on the brachial artery segment of the transmission line model as in [10] which is shown in Fig. 3. The external pressure reduces the radius of the arterial segment and hence the resistance calculated increases. The variable voltage source increased in steps to simulate the increments in cuff pressure.

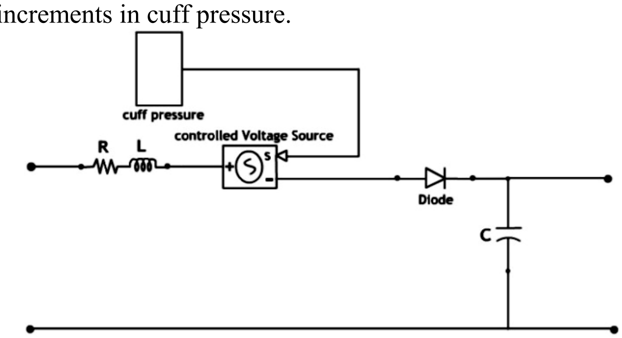

Fig. 3. Modification of brachial artery segment of the distributed model shown in Fig.2

We used SIMULINK to generate a realistic pressure waveform using the 4-element Windkessel model. The input aortic flow rate can be changed to control the duration of the output pressure waveform. Typical input flow and output pressure of the Windkessel model are shown in Fig. 4(a), 4(b). The radial pressure pulse and its first derivative when the external pressure applied are shown in Fig.5. Changes in the pulse morphology with different external pressure, as predicted from the model, are shown in Fig.6.

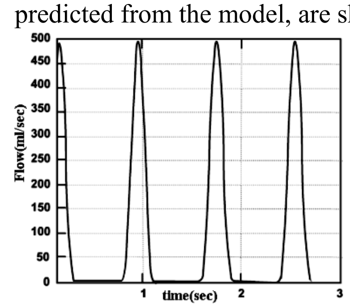

Fig. 4(a).Aortic Flow

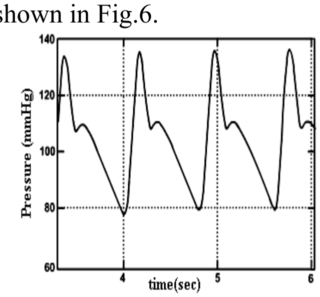

Fig. 4(b)Aortic Pressure

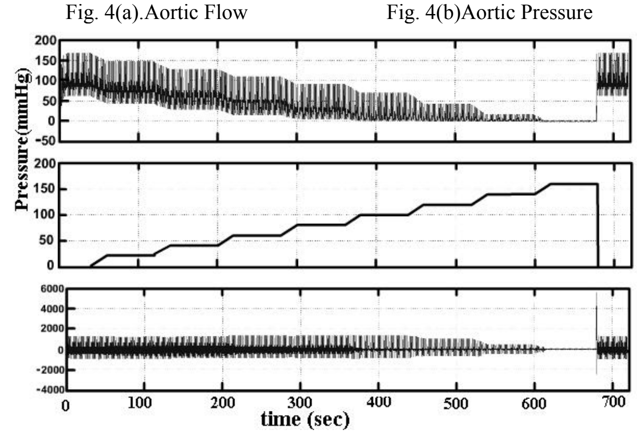

Fig. 5. Radial pressure when external pressure is applied (Top), its derivative (bottom) and the applied cuff pressure (middle).

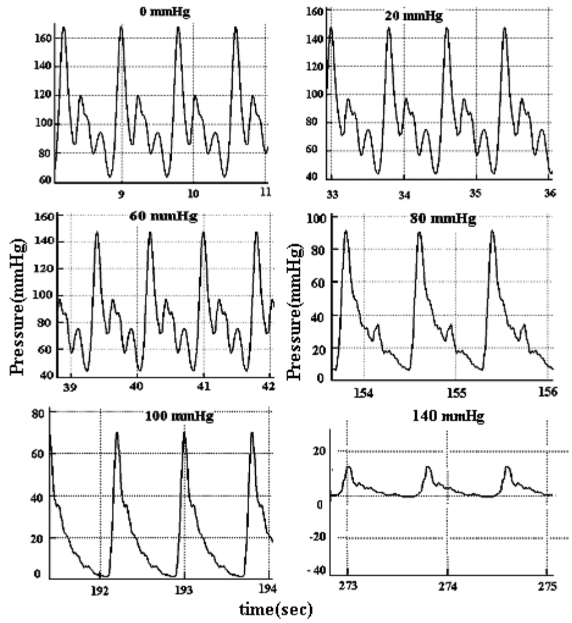

Fig. 6. Radial pressure waveform shape changes with different external cuff pressure.

D. Experimental Protocols

We measured volume pulse using PPG in six healthy volunteers with no history of cardiovascular disease. The age of the subjects were in range of 22 to 29 years and Body mass index of 24.31±2.73. All the measurements were undertaken in a temperature-controlled room (20±1°C). We measured the systolic, diastolic pressures and pulse rate using blood pressure monitoring device, not considered in this study. The cuff was wrapped around the upper arm of the left hand and the PPG was placed on the forefinger of same hand. The subject initially lied in supine position and relaxed for five minutes. During the whole measurement, the subject breathed normally. The hands were kept at heart level and the subject was also asked to remain still in order to reduce the motion artifacts. We raised the cuff pressure from 0mmHg to 140 mmHg in 20 mmHg steps. We acquired 30sec recording of the signal at 100Hz at each cuff pressure level. We increased the pressure levels in 10 sec. For each subject we performed three trials with 5 minutes gap between each trial.

E. Data Acquisition and Preprocessing

We used Biopac model MB35 system for acquiring PPG. We filtered the pulse signal below 0.3Hz and above 10Hz. We manually removed the prominent motion artifacts in the data.

F. Statistical Analysis

We performed paired student's t- test for calculating correlation coefficient between measured and model parameters. P<0.05 was considered to indicate statistically significance.

III. Results

We calculated the following parameters for each beat: maximum value, minimum value, maximum time, maximum of first derivative (maximum systolic slope) and minimum of first derivative (maximum diastolic slope). Calculating the difference between maxima and minima of amplitude, we found the pulse height and then normalized it with corresponding values at zero cuff pressure, similarly the peak to peak interval (PPI) from the time interval of successive peaks and normalized with zero pressure PPI. Mean values of normalized height, normalized PPI, and normalized slope of a single subject are shown in Fig.8, Fig.9 and Fig.10. Fig.11 shows the comparison of the normalized parameters of both the model and the experiment.

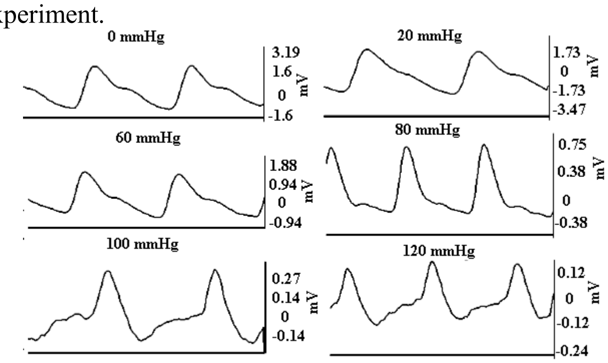

Fig. 7. PPG waveform shape changes with external cuff pressure.

The measured and model parameters are compared with paired student's t-test, indicating that all measured parameters except PPI are statistically significant and positively correlated with model parameters (mean normalized maximum systolic slope: r = 0.54, $P<0.05$, mean normalized maximum diastolic slope: r = 0.77, $P<0.05$, normalized mean pulse height: r = 0.89, $P<0.05$, mean normalized peak to peak interval: r = 0.22, $P<0.05$).

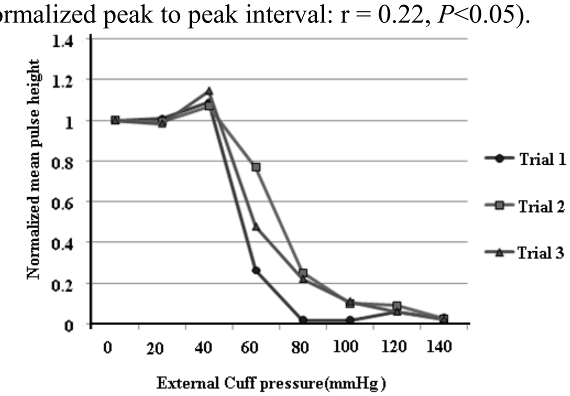

Fig. 8. Normalized mean pulse height of left hand PPG

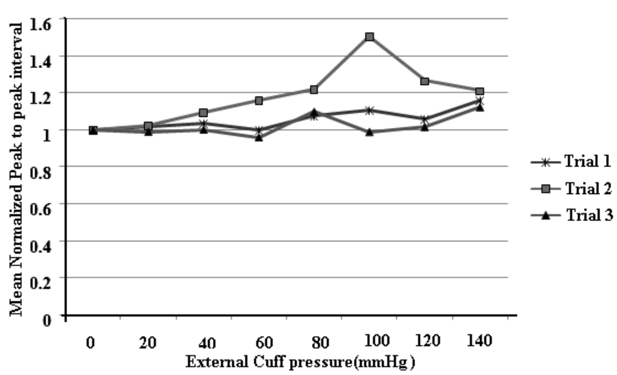

Fig. 9. Mean of normalized PPI of left hand PPG

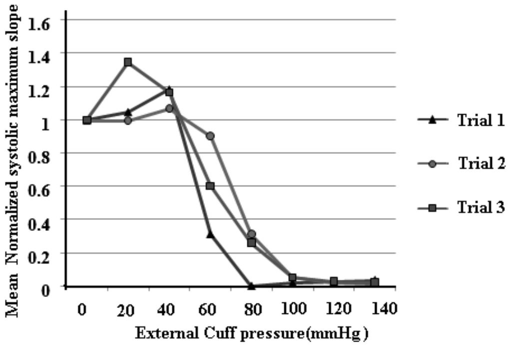

Fig. 10. Mean of normalized Maximum slope of Left hand PPG

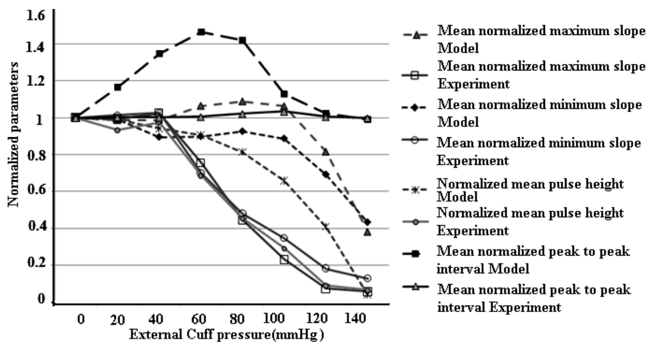

Fig.11. Comparison of the various normalized parameters in the transmission line model and experiment.

IV. Discussion

It is to be noted from Fig.6 that the diastolic phase of radial pressure signal is changing with applied external pressure, specifically the duration of the diastolic phase is increasing and the dicrotic notches are reducing. Also the pulse amplitude reduces with external applied pressure. The slope of the pressure pulse slightly rises until 80mmHg and then it reduces. The same trend can be observed in the experimental data as shown in Fig.7. The results from the model as shown in Fig.11 indicate that maximum slope, minimum slope and PPI, all normalized, slightly rises with external pressure, while the normalized pulse height does not show such a trend. These parameters reach the maximum at 60 to 80mmHg. In the experiment all the four parameters show the initial rise with the pressure and the maximum occurs at 40 mmHg. Above this external pressure, venous gets completely occluded, and the incoming pulsatile blood flow is added to the already pooled blood volume down the limb, results in initial rise in the maximum slope and pulse height and then a fall. The initial rise of the above parameters predicted in the model is much more than in the experiment. Specifically PPI variation predicted by the model is much more while the experiment shows very little variation (the model PPI is less correlated with measured PPI $r = 0.22$, $P<0.05$). This may be due to the lack of feedback mechanism incorporated in the model. In the intact human being baroreflex and cardio pulmonary reflex are activated in response to increase in pressure and reduction in volume.

In the model we evaluated only the radial artery pressure while in the experiment we observed digital volume pulse using PPG. The derivative of arterial pressure is directly proportional to that of the arterial volume with the elastance as the proportionality constant. While the model elastance does not change, in the external cuff experiment the elastance does not remain constant. This could be one of the reasons for the discrepancy between the model and experiment.

It is observed that volume pulse starts to disappear and becomes irregular for all the subjects above 100mmHg. The PPI variation graph is different for each subject reflecting the reflex properties of the individual. Also, it is interesting to note that all the normalized parameters from the experiment follow the same trend. This means that measuring the four parameters is redundant and any one parameter could predict the others.

V. Conclusion

We have used distributed model of entire human arterial tree instead of lumped model for simulating the externally applied cuff pressure. We analyzed the variation of pressure pulse height and slope using the transmission model and experiment. While the model could generally predict the changes in the above parameters, they do not exactly match with those in the experiment. We need to incorporate the reflex feedback in the model in order to correctly predict the experiment results. Instead of using Windkessel model, we can use lumped model of heart with baroreflex. While the elastance in the experiment varies with pressure, in the model it is constant and might be another reason for the mismatch. More number of subjects with different age group should be considered for the experiment to find the arterial elastance change with the external pressure. With the reflex and elastance changes incorporated in the model, its clinical relevance can be studied for different diseased conditions.

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