

Mathematical Model of Venous Occlusion Plethysmography for Diagnosing Deep Vein Thrombosis

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Abstract-Although the results of VOP can determine the being of DVT, it has difficulty in analyzing quantitatively the effects of the degrees of thrombosis, blood pressure and cuff pressure on VOP. In this paper, by developing a combined model that is composed of pulsatile cardiovascular model and venous occlusion model, we showed the availability of more realistic simulation. Using the proposed combined model in simulation about the effects of different head pressure and degree of thrombosis on VOP, we can get the basic understanding what relationship is between the state of patient and measured VOP.

Keywords - Deep venous thrombosis, venous occlusion plethysmography, mathematical model

I. INTRODUCTION

Deep venous thrombosis(DVT) is the formation of blood clots in the deep venous system. It is a source of morbidity or mortality after surgery and a complication in many other branches of medicine. While invasive methods for DVT detection such as venography are most accurate, they could cause some risk to the patient. As a result there has been many studies on non-invasive techniques, including venous occlusion plethysmography(VOP), to screen patients for the presence of DVT[1-3].

Although the results of VOP are helpful to detect DVT, being an indirect measure, there are difficulties in quantitative comparison of results that come from different degrees of thrombosis. So, there has been a study which estimates the effects of thrombosis size on patient's venous outflow with a numerical model[4].

In this study, by developing a mathematical model which consists of cardiovascular system model and venous occlusion model, basic understanding of relationships between the hemodynamic changes caused by varying VOP related parameters and the indirect measurements such as impedance plethysmography (IPG) and strain gauge plethysmography (SPG), are offered.

II. METHODOLOGY

Total cardiovascular system is a loop that has many blood branches and at individual compartment of system, blood pressure, flow and blood volume provides meaningful clinical information. Each compartment has their own resistance, inertance and compliance and, from the value, cardiovascular system can be modelled[5]. Fig. 1 shows a simple cardiovascular model.

In Fig. 2, a venous occlusion model is shown. R_a is the arterial resistance, R_{cap} the capillary resistance, R_{calf} the total resistance of anterior tibial, posterior tibial and peroneal resistance, R_{fem} the femoral resistance, R_{saph} the saphenous resistance, C_v the compliance of calf vein.

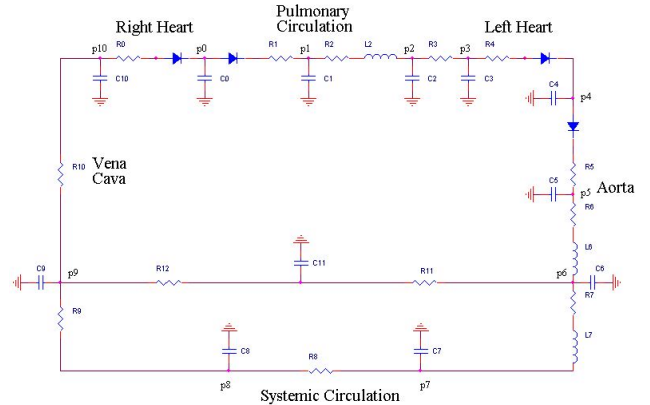


Fig. 1. A simple cardiovascular model

The resistance of each vein was calculated using Poiseuille's formula assuming laminar flow.

$$R = \frac{\Delta P}{Q} = \frac{128\mu L}{\pi D^4} \quad (1)$$

From the experimental results that, after cuff inflation, the venous pressure rises to approximately 90% of the cuff pressure[4], continuity of flow gives

$$\frac{P_a - P_h - 90\%P_{cuff}}{R_a + R_{cap}} = \frac{90\%P_{cuff} + P_h}{R_v + R_{cuff}} \quad (2)$$

where P_a is the arterial pressure, P_{cuff} is the cuff pressure, P_h is the head pressure due to leg raising, R_v is the venous resistance and R_a is the arterial resistance.

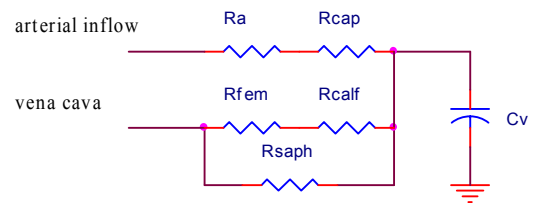


Fig. 2. A venous occlusion model

To combine two models, the mean arterial pressure of the cardiovascular model was used as the arterial inflow.

In this study, the effects of leg raise and the changes of vein resistance and compliance caused by DVT were simulated. Additionally, the waveform of IPG contaminated by the arterial pulses was generated. Numerical values of equation parameters correspond approximately to an adult male human weighing 70kg at rest. And the heart period is assumed to be 0.8 sec. The solution of model was found by the 2nd order Runge-Kutta method, implemented by a commercial mathematical solver, ACSL(Aegis Technologies Group, Inc.)

Report Documentation Page

Report Date 25 Oct 2001	Report Type N/A	Dates Covered (from... to) -
Title and Subtitle Mathematical Model of Venous Occlusion Plethysmography for Diagnosing Deep Vein Thrombosis	Contract Number	
	Grant Number	
	Program Element Number	
Author(s)	Project Number	
	Task Number	
	Work Unit Number	
Performing Organization Name(s) and Address(es) Department of Medical Engineering College of Health Science Yonsei University South Korea	Performing Organization Report Number	
Sponsoring/Monitoring Agency Name(s) and Address(es) US Army Research, Development & Standardization Group (UK) PSC 802 Box 15 FPO AE 09499-1500	Sponsor/Monitor's Acronym(s)	
	Sponsor/Monitor's Report Number(s)	
Distribution/Availability Statement Approved for public release, distribution unlimited		
Supplementary Notes Papers from 23rd Annual International Conference of the IEEE Engineering in Medicine and Biology Society, October 25-28, 2001, held in Istanbul, Turkey. See also ADM001351 for entire conference on cd-rom. , The original document contains color images.		
Abstract		
Subject Terms		
Report Classification unclassified	Classification of this page unclassified	
Classification of Abstract unclassified	Limitation of Abstract UU	
Number of Pages 2		

III. RESULTS AND DISCUSSION

The clinical use of the VOP implies measurement of the venous capacitance(V_c), the maximal fraction increase in calf volume during occlusion and the venous outflow(V_o), the rate of volume outflow between 0.5 and 2 sec after cuff deflation. First, by varying the P_h from 0 to 5 mmHg by 1 mmHg, the effects of leg raise were simulated. And, the changes of the venous capacitance and the venous outflow were observed. The results are presented in Fig. 3.

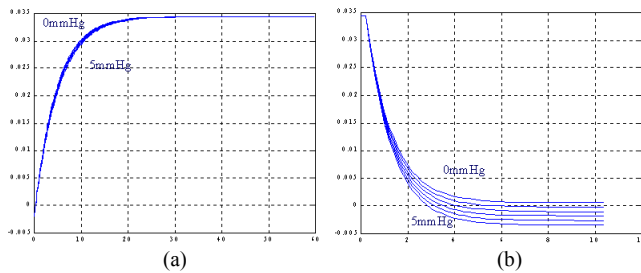


Fig. 3. The effects of leg raise (a) during cuff occlusion and (b) after deflation

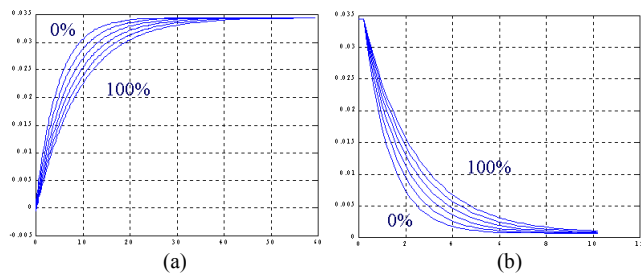


Fig. 4. The effects of venous resistance change (a) during cuff occlusion and (b) after deflation

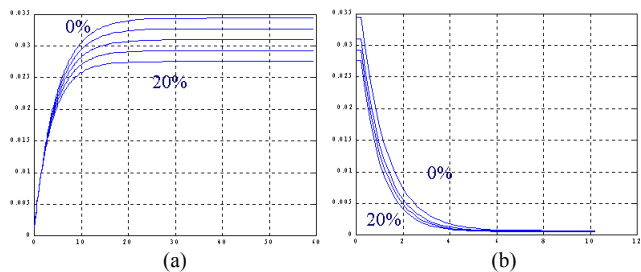


Fig. 5. The effects of venous compliance change (a) during cuff occlusion and (b) after deflation

Clots, in deep vein system, get various dimensions and are attached to some different veins so that vein's mechanical parameters would be changed and not be predicted easily. In general, they, by disturbing the blood flow, make the venous resistance increase and, by hardening the vein, the compliance decrease. By increasing the resistance in 10% steps from 0%(normal) to 100%, the effects of venous resistance changes on the calf volume change were simulated and the results are described in Fig. 4. As increasing the resistance, the venous capacitance is not changed but the venous outflows are decreased. And Fig. 5 shows the effects of the compliance changes when the venous compliance is decreased in 5% steps from 0%(normal) to 20%. The results above are well agreed with the fact that the venous capacitance of DVT patient is smaller than normal.

Additionally, the waveform of arterial pulse added into VOP that found IPG is generated by (3).

$$imp(VOP) = imp(Vc) + \alpha \times imp(AP) \quad (3)$$

where AP means the arterial pulse and α is the scaling factor which determines the magnitude of arterial pulse measured by the electrodes of IPG.

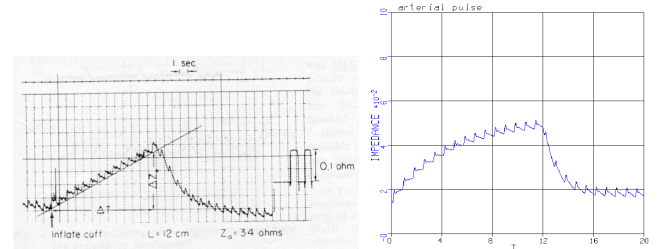


Fig. 6. A measured waveform include the arterial pulse (left, by D. W. Hill, H. J. Lowe, 1973) and a generated waveform(right)

Developing the mathematical model of VOP that consists of cardiovascular model and venous occlusion model, more realistic VOP curve, in which pulsatile shapes are included, can be generated. And, the effects of occlusion cuff pressure, arterial pressure on VOP and other factors, could be simulated as well as the effects of DVT.

IV. CONCLUSION

This model has demonstrated the availability of more realistic VOP curve. In the further study, for the other factor that affects VOP curve, simulations could be performed.

ACKNOWLEDGMENT

This work was supported by RRC program of MOST and KOSEF, South Korea.

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