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 its 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. Ra is the arterial resistance, Rcap the capillary resistance, Rcalf the total resistance of anterior tibial, posterior tibial and peroneal resistance, Rfem the femoral resistance, Rsaph the saphenous resistance, Cv the compliance of calf vein.

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

\[ R = \frac{\Delta P}{Q} = \frac{128\mu L}{\pi D^4} \tag{1} \]

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

\[ \frac{P_a - P_{h} - 90\%P_{\text{off}}}{R_v + R_{\text{cap}}} = \frac{90\%P_{\text{off}} + P_{h}}{R_v + R_{\text{off}}} \tag{2} \]

where \( P_a \) is the arterial pressure, \( P_{\text{off}} \) is the cuff pressure, \( P_{h} \) is the head pressure due to leg raising, \( R_v \) is the venous resistance and \( Ra \) is the arterial resistance.

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.)
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### Subject Terms

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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.

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

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

where $AP$ means the arterial pulse and $\alpha$ 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.

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REFERENCES