

MATHEMATICAL SIMULATION OF THE CARDIOVASCULAR RESPONSES TO LOWER BODY NEGATIVE PRESSURE UP TO -40 mmHg

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Abstract. This paper presents a mathematical model to simulate the human cardiovascular responses to lower body negative pressure (LBNP) up to -40 mmHg. The basal (initial) blood volume status, which includes the total blood volume and the initial blood volume distribution is the central determinant of the cardiovascular responses to LBNP and has not been included in previous models. Thus, the cardiovascular response to LBNP was regarded as an initial value problem in which the blood volume status and physiological parameters were regarded as initial conditions. When LBNP is applied to the model, the model tracks the blood volume status and physiological parameters from the initial steady state to the final steady state. Results from the model compared favorably with data from the LBNP literature.

Keywords. Blood pressure, cardiovascular modeling, lower body negative pressure.

Introduction. One of the objectives of this study was to develop a mathematical model in which the initial blood volume status was taken into account to simulate the physiology responses to LBNP stress. The other objective was to apply principles of fluid mechanics to explain the physiological phenomenon, instead of simply curve fitting equations to force the output of the model to mimic the experimental data.

Methods. The behavior of ventricle filling and contraction was simulated with a time-varying elastance model. The three-element Windkessel model was employed to model the arterial flow. A nonlinear spring and a frictionless motion piston were used to simulate the behavior of the venous system[1]. The heart rate, peripheral resistance and baroreflex were simulated by equations from Melchior[2]. The result of the mathematical model is an unsteady closed cardiovascular system simulation that includes the ventricle contraction model, the arterial system model, the venous system model, the ventricular filling model and the baroreflex control model. Available information such as the dynamics of ventricular filling and the mechanics of collapsed vessel walls is applied to obtain unknown parameters by utilizing engineering principles. Thus, all the parameters of the model, even those that usually are difficult to measure, are well defined by calculations. The result of this model is that complex physiological phenomena are simulated by a set of simple mathematical equations.

Results. Figure 1 shows the simulation of the aortic pressure under LBNP = -40mmHg. The systolic arterial pressure was reduced from 124 mmHg to 115 mmHg about 25 second after LBNP was applied.

The results of the model agreed very well with experimental data obtained from previous LBNP studies. Figure 2 shows the comparison between the model output and experimental data for systemic arterial blood pressure (SAP). Comparisons for heart rate show similar agreement.

Conclusions. A novel mathematical model of the cardiovascular responses to LBNP was developed. The model differs from previous models in that the initial blood volume and blood volume distribution is included, and the equations were developed independently from experimental data. That is, the model was considered an initial value problem and principles of fluid and solid mechanics were used to derive the equations to model the various components of the cardiovascular system. The model successfully reproduced existing experimental data.

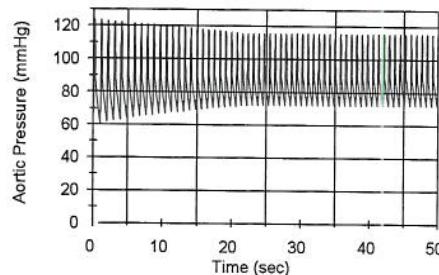


Figure 1 The aortic pressure variation under LBNP = -40 mmHg

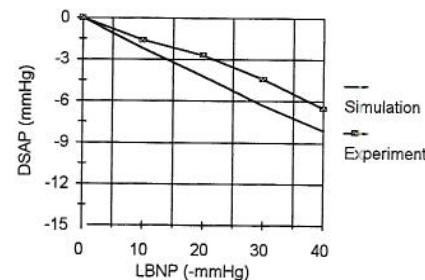


Figure 2 The simulated and experimental change of SAP under different LBNP

References.

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