SIMULATION OF THE CARDIOVASCULAR AND AUTONOMIC NERVOUS SYSTEM FOR OPTIMIZATION OF AURICULAR VAGUS NERVE STIMULATION

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INTRODUCTION

Percutaneous electrical stimulation of the auricular vagus nerve (pVNS) is a promising approach in management of postoperative and chronic low back pain. pVNS has shown beneficial effects on systemic physiological parameters, such as heart rate variability. Although there are some efforts on modelling pVNS and its effects on the cardiovascular system, in order to control and optimise pVNS, the contribution of the autonomic nervous system is not modelled sufficiently yet. In this study, we model jointly the cardiovascular and autonomic nervous system, to provide a basis for the development of a feedback-based control of pVNS stimulation parameters.

METHOD

A model composed out of 3 main blocks, cardiovascular system (CVS), a chronotropic autonomic model and pVNS. CVS was optimized to keep the nominal systolic and diastolic pressure in the range of 120/80 mmHg and receives heart period as an input from baroregulation and pVNS blocks. Baroregulation system consist of carotid baroreceptors and chronotropic effect of vagal and sympathetic and total effects on heart period are summed up with pVNS effect (Fig.1).

Cardiovascular system: A minimal cardiovascular system [1] which describes the hemodynamics of heart and blood circulation was established. This model is stable in long-term and does not depend on initial values. In addition, cardiac cycles were simulated by taking into account the inertia effect on cardiac valves. Moreover, the change of thoracic pressure and its effect on cardiac output was included. Systemic resistance, which plays a major role in blood pressure, was modelled as part of the closed loop model. The model works based on pressure volume relation of every component, heart period, and systemic resistance or respiration, as changing thoracic pressure influences the heart rate variability.

Autonomic nervous system: A pulsatile heart simulation provides input for the carotid baroregulation. We used a model of carotid baroregulation [2] to simulate the baroreflex effect based on arterial pressure changes in the cardiovascular system. In this work, we focused on chronotropic effects on RR-intervals through the sympathetic and vagal efferent branches. Therefore, the vagal and sympathetic contribution will be added to the intrinsic heart period as autonomic nervous system input. Since the main contribution of pVNS on the system is not clear yet, cervical vagus nerve stimulation effects (stimulation of the cardiac vagal branch) on RR- intervals was adopted from [3], to simulate the whole structure. This can be substituted in future by pVNS.

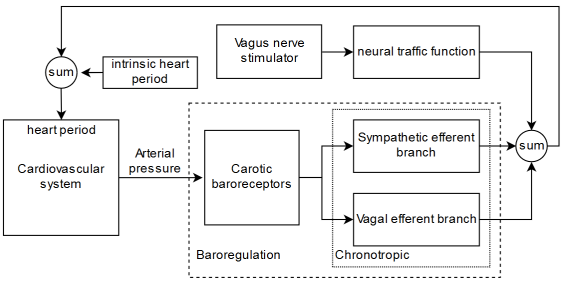

Figure 1: system block diagram.

RESULTS AND DISCUSSION

The cardiovascular system model was physiologically verified by altering the thoracic pressure, the elasticity of ventricle walls, and the systemic resistance. An increase in left ventricular stroke volume and pressure, due to doubled systemic resistance, and a decrease due to the half systemic resistance, are shown in Fig. 2. In order to investigate the respiration effect on stroke volume, the thoracic pressure was decreased from -4 mmHg to 0 mmHg. A decrease in left ventricular stroke volume and pressure was observed.

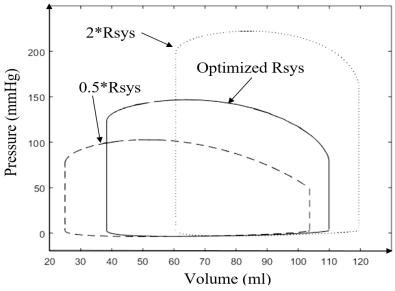

Figure 2: Systemic resistance effect.

Respiratory sinus arythmia was simulated by applying the RR- intervals from a real ECG signal to the cardiovascular system model. Fig. 3 shows aortic pressure for both conditions. A small change in the range of 5% in amplitude and different systolic and diastolic duration due to the varying RR-intervals is obvious.

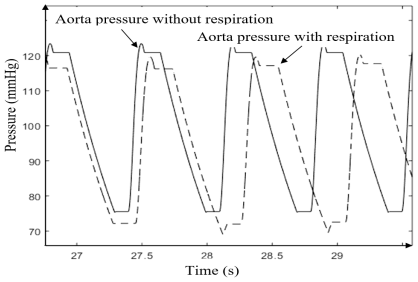

Figure 3: Respiratory effect on aortic pressure.

Autonomic nervous system simulation showed a fast response to the increase in mean aortic pressure via an increased RR-interval. A slow response was seen for a decrease in aortic pressure via a decrease in RR-intervals. Direct vagus nerve stimulation of the cardiac branch increased the RR-intervals.

CONCLUSION

In this study, we modelled a minimum dynamic cardiovascular system with autonomic nervous system and direct vagus nerve stimulation input. The model is stable and easy to solve. We verified the model by checking its response to physiologic changes. This model will allow us to optimize pVNS stimulation and build feedback based solutions. A feedback-controlled pVNS can avoid blind treatment or even help detecting non-responders by monitoring bio-signals. Simulations facilitate manipulation of physiological and stimulation parameters. In addition, simulations reduce the experimental costs and allow individualized models.

REFERENCES

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