Modeling the Cardiovascular System to simulate the limitations of predicting fluid responsiveness by dynamic indices

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Introduction

Fluid management is one of the more challenging tasks in the peri-operative care, because the medical team cannot directly asses the volume status of a patient. Currently, it has been shown that fluid responsiveness of a patient can be predicted with help of dynamic indices like Pulse Pressure Variation and Stroke Volume Variation. However, their usage is limited[1]. In this study, a mathematical model of the cardiovascular system (CVS-Model) and its short-term control systems is developed that is used to investigate these limitations.

Methods

An earlier six compartmental model of the circulation with baroreflex as short-term control mechanism [2],[3] was extended to investigate the reliability of dynamic indices of fluid responsiveness. The systemic circulation was extended as suggested by Heldt et al., to include different intra- and extra-thoracic compartments[4]. The baroreflex control of systemic venous unstressed volume and peripheral resistance was modified accordingly. Furthermore, stress relaxation in the systemic veins and in the systemic arteries was implemented. A cardio-pulmonary interaction model was developed to simulate the respiratory modulation of the circulatory model.

The model was validated by simulating its steady state performances and its responses to a blood loss experiment. The limitations of the dynamic indices were investigated by simulation of baroreflex inhibition (to simulate general anesthesia) during the blood loss experiment and step-wise increases of the tidal volume and respiratory frequency under the assumption of mechanical ventilation.

Results

Simulation of the blood loss experiment showed comparable hemodynamic changes as found in the experimental results[5]. The mean arterial pressure and the dynamic fluid responsiveness indices in healthy subject were hardly affected by a hemorrhage of 1L. General anesthesia (simulated by a reduced baroreflex gain) caused an increase of the dynamic indices. Also, the dynamic indices increased as a result of an increase of tidal volume or a decrease of the respiratory frequency.

Conclusion

The main objectives of this investigation were to simulate the fluid responsiveness of a patient and the limitations of the dynamic indices of fluid responsiveness with a validated model. The developed mathematical model provided realistic simulation data of a patient in steady state and showed promising results of hemodynamic response to a blood loss experiment. Also, this investigation effectively showed that the dynamic indices are dependent upon the settings of the mechanical ventilator and amount of anesthesia a patient receives.

References

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