Heart and lung support interaction — modeling and simulation

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Received 9 October 1998; accepted 30 March 1999

Abstract—Mechanical support of the lungs used to preserve life or during any kind of surgery may have an adverse effect on the cardiovascular system. Usually, positive pressure in alveoli diminishes lung perfusion, venous return and cardiac output. Positive pressure during the respiratory cycle is transferred into the thoracic space. The aim of this study was to assess how synchronization of the respirator with spontaneous breathing influences the distribution of pressure and ventilation in non-homogeneous lungs and how it should influence hemodynamics. For this purpose a multicompart-mental model of respiratory system mechanics was used in the electrical analog of a respirator–lung circuit, which enabled us to simultaneously simulate ventilatory support and spontaneous breathing. Mechanical properties of the respiratory system were modeled by lumped parameters: resistances and capacitances of constant values, independent of lung volume or inspiratory flow changes. A multi-compartmental model of the respiratory system enabled us to simulate lung pathology characterized by non-homogeneity of the mechanical properties of the different parts of the lungs. The results of simulations presented in the paper enable us to conclude that lung volume increase, independent of the respirator–patient breathing synchronization, may be modeled as the increase in pulmonary vascular resistance and alveolar pressure increase, dependent on respirator–patient breathing synchronization, may be averaged by esophageous balloon measurements which show intrathoracic pressure changes.

Key words: Ventilatory support; pressure distribution; lung volume; hemodynamics.

1. INTRODUCTION

As we accept the obvious thing that the lungs, right and left ventricles as well as great vessels are inside the thorax, we have also to agree that both respiratory and cardiovascular systems (except systemic circulation) are influenced by cyclic pressure variations that are connected with different breathing patterns. During spontaneous breathing the mean intrathoracic pressure is negative, which creates good, physiological conditions for the blood to travel through pulmonary vessels, and venous return of the blood is also enhanced.
On the contrary, during mechanical ventilation of the lungs, which is used as a necessary means to support inefficient respiratory system, new physiological conditions for gas exchange are created. Positive airway pressure generated by a respirator is transferred into the thoracic cage and the mean intrathoracic pressure is always positive. This change in transpulmonary pressure gradient and increased lung volume during inspiration causes several harmful side effects: withdrawal of blood from the thorax into systemic circulation, increase of pulmonary vascular resistance (as a result of small intra-alveolar vessels compression), decrease of venous return and, finally, decrease of cardiac output.

As the pulmonary vessels are distensible, the fall in cardiac output and pulmonary arterial pressure decreases the pulmonary arterial resistance. This phenomenon is known as active vasoconstriction.

The hemodynamic consequences of mechanical ventilation have been considered as a complex problem by physiologists and are still the subject of various recent papers [1–4]. There are also some limitations in our understanding of the whole mechanism involved in the interaction between the respiratory and cardiovascular systems, especially in pathological cases. At beginning of providing mechanical lung ventilation by automatic pumps known as respirators (in 1952) the main mode of ventilatory support was so-called controlled ventilation, with the patient’s breathing excluded and the whole ventilation provided by a respirator.

This method enabled anesthesiologists to easily control minute ventilation of the lungs and adjust it to the patient’s metabolism. However, with the technological development of respirators during last decade, a better understanding of various physiological problems of ventilatory support simultaneously took place. This has resulted in today’s trend to keep patient’s breathing as long as possible during mechanical support by the respirator. It has a simple explanation in the fact that spontaneous breathing always gives negative pressure in the thorax, diminishing the mean positive intrathoracic pressure caused by mechanical ventilation. It means that, by preserving the patient’s breathing during ventilatory assistance, we can expect less harmful side-effects of positive pressure ventilation on hemodynamics. These effects should also depend on the actual conditions during ventilatory support, e.g. the synchronization of a respirator with spontaneous breathing and the state of the lungs (physiological or pathological).

The aim of this study was to access, by modeling and simulation, the influence of interaction between the lungs and a respirator on hemodynamics. In other words, to answer the question: how does synchronization of a respirator with spontaneous breathing influence the distribution of pressure and ventilation in (assumed) non-homogeneous lungs and how should it influence hemodynamics? The alveolar pressure distribution and ventilation distribution (as lung volume changes) are those indices which finally decide on changes of intrathoracic pressure and pulmonary vascular resistance, respectively.