CARDIOVASCULAR SYSTEM MODELLING OF HEART-LUNG INTERACTION DURING MECHANICAL VENTILATION

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Abstract: Choosing suitable ventilation strategies for critically ill patients with lung disorders involves considering the difficult and poorly understood trade-off between achieving adequate ventilation, while maintaining suitable perfusion. This study presents a minimal cardiovascular system model that includes variations in pulmonary vascular resistance during mechanical ventilation. The model is shown to capture transient haemodynamics from experimentally measured data. It also enables investigation into the effects of time varying resistance on pulmonary haemodynamics. The study shows the potential usefulness of this model in a tool to assist clinical staff in optimising ventilation pressures while maintaining adequate pulmonary perfusion.

Keywords: Biomedical systems, Physiological models, Dynamic modelling, Dynamic models, Lumped constant models.

1 INTRODUCTION

Although the lungs and the circulation system are usually simulated independently, the interaction between them has significant consequences for both systems. One important example of this is the interaction between lung pressures and pulmonary haemodynamics in critically ill patients with lung disorders. For example, patients suffering from acute respiratory distress syndrome (ARDS), or post cardiac surgery patients, require ventilation at high pressures to increase lung ventilation and improve gas exchange (Claxton *et al.*, 2003; Dyhr *et al.* 2002). However, these high pressures can also cause a reduction in pulmonary perfusion, which can have a negative impact on gas exchange, as well as reducing cardiac output (Parrillo and Dellinger, 2002). The trade-off between achieving adequate ventilation, while maintaining adequate pulmonary perfusion, is an important consideration when treating intensive care patients.

High ventilator pressures affect circulation in a number of ways (West, 2005; Parrillo and Dellinger, 2002). Increased pressure acting on the outside of the heart limits ventricle filling. Increased pressure on the large pulmonary arteries and veins causes a reduction in the pulmonary circulation blood volume. Increased pleural pressure and lung volume impedes blood flow through the capillaries, increasing the pulmonary vascular resistance.

Pulmonary vascular resistance (R_{pul}) is generally defined as:

$$R_{pul} = \frac{P_{pa} - P_{pu}}{CO} \quad (1)$$

where the pressure drop across the pulmonary circulation is defined as the pulmonary artery pressure (P_{pa}) minus the pulmonary venous pressure (P_{pu}), and cardiac output (CO) represents the average blood flow through the pulmonary circulation. This resistance can vary during both the cardiac cycle and the respiratory cycle by two main mechanisms (West, 2005). Firstly, the cross-sectional area of a capillary, and thus the resistance, is dependent on the pressure drop across the capillary wall. A decreased blood pressure in the capillary, or a reduced pleural pressure acting on the outside of the capillary, will cause the capillary to expand and its resistance to be reduced. Secondly, due to hydrostatic effects, the blood pressure in the capillaries is lower at the highest point in the lungs than at the lowest point. A pressure increase in the pulmonary artery can cause capillaries to become recruited, thus reducing the total pulmonary resistance. Both of these effects are largely dependent on the pressure difference between the pulmonary artery and the pleural pressure.

There are many models of the circulation system and the respiratory system in the literature (Beyar *et al.* 1987; Ursino, 1999; Barbini *et al.*, 2003). However, there are very few models that simulate the impact of changes in lung pressure and volume on haemodynamics, especially relating to pulmonary vascular resistance. Probably the most comprehensive compartmental cardiopulmonary interaction models are described by Liu *et al.* (1998) and Lu *et al.* (2001). Both these models combine detailed circulation system and respiratory system models. Liu *et al.* (1998) divides the pulmonary vascular resistance into three different resistances that are dependent on pleural pressure, alveolar volume and capillary volumes. Lu *et al.* (2001) uses a much simpler definition where pulmonary resistance is dependent on lung volume. However, in both cases, these models are not verified against experimentally measured pulmonary artery pressure, which is an important factor in pulmonary perfusion.

Scharf *et al.* (1980) have carried out experimental studies where they measured pulmonary and systemic artery pressures and flow rates simultaneously with pleural pressure variations during mechanical ventilation. If the respiratory drive has been suppressed, it can be assumed that thoracic cavity compliance remains constant during mechanical ventilation. Under these conditions, according to West (2005), pleural pressure, lung volume and pulmonary vascular resistance should all vary proportionally to one another. So during the mechanical inspiration, pulmonary vascular resistance will increase as pleural pressure increases and visa versa.

This research presents a model based investigation into the affect of mechanical ventilation on pulmonary haemodynamics. All of the interaction mechanisms discussed above are considered, with specific focus on the effects of variations in pulmonary vascular resistance during respiration. A minimal cardiovascular system model, described by Smith *et al.* (2004a), is used to simulate experimentally measured haemodynamic changes during mechanical ventilation (Scharf *et al.* 1980). A simple proportional relationship is defined between the pleural pressure and the pulmonary vascular resistance. The investigation focuses on the importance of including a time varying pulmonary vascular resistance and the ability of this simple model to capture the measured haemodynamic trends in the experimental data.

2 METHOD

2.1 The cardiovascular system model

The minimal cardiovascular system model used in this study, shown schematically in Figure 1, is defined in detail in Smith et al (2004a). The systemic circulation is made up of two chambers representing the vena-cava (vc) and the aorta (ao), connected by a resistor to simulate the net systemic resistance (R_{sys}). Similarly, the pulmonary circulation is simulated as the pulmonary artery (pa) chamber connected to the pulmonary vein (pu) chamber by the pulmonary vascular resistance (R_{pul}).

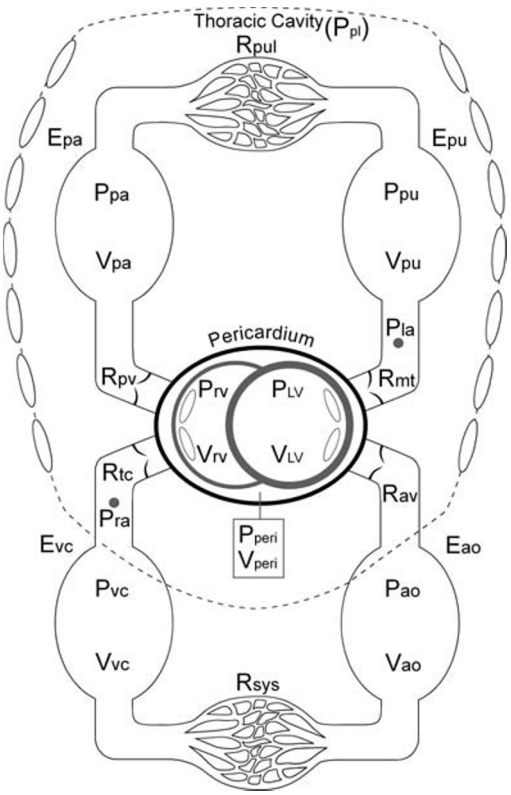

Fig. 1. Diagram of the cardiovascular system model used in this study.