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A numerical model to study the coupling between lung biomechanics and physiology

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1. Introduction

The Acute Respiratory Distress Syndrome (ARDS) is a critical condition. For patients suffering from this pathology, mechanical ventilation (MV) is needed to ensure their sufficient ventilation and oxygenation. Intensivists have several therapeutic tools at their disposal to design a correct MV. The most diffused are recruitment maneuvers with Positive End Expiratory Pressure (PEEP) and prone positioning (Meyer et al. 2021). However, practitioners face several issues when confronted to ARDS and using these two techniques:

- High variability between patients
- Limits in the understanding of the underlying mechanisms

This leads to a mostly experienced-based decision-making for physicians. Considering both the downside effects and the resources required to implement the recruitment maneuvers and prone positioning, being able to evaluate the probability to respond to different therapeutic approaches would be of high clinical significance. Moreover, better understanding the biomechanical and physiological phenomena underlying the patient response could inform new MV strategies to deal with ARDS. In order to tackle these issues, a coupled physio-mechanical computational framework was conceived and implemented.

2. Methods

2.1 Global framework

The global framework that was used to implement the physio-mechanical model is illustrated in Figure 1. Part of this framework, corresponding to the mechanical model, was presented in a previous publication (Bruna-Rosso and Boussen 2022). The model is a so-called multi-compartment model, which represents the lung as several “balloons” able to inflate and deflate according to a non-linear compliance. These compartments also have the ability to exchange gases with external capillaries. This later phenomenon, which extends the previously published model, is further detailed in the next subsection.

2.2 Physiological model

The physiological model aims at simulating the gas exchanges between the alveoli and the blood consecutive to an inspiration that is modelled within the mechanical part of the computational framework.

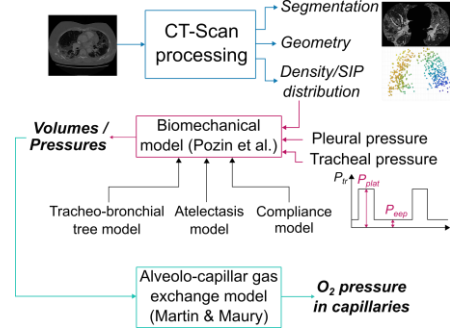


Figure 1: Physio-mechanical model workflow

The physiological model computes two main variables, namely the dioxygen fraction within the alveoli c_A and the partial pressure of O_2 P_c within the capillaries.

The equations that were used are based on the model from Martin & Maury (Martin and Maury 2013) and are the following:

- Variation of O_2 volume inside a compartment

$$\frac{d}{dt}(c_A V) = c_A^0 \dot{V} H(\dot{V}) + c_A \dot{V} (1 - H(\dot{V})) - Q$$

Where V is the total compartment volume (computed by the mechanical model) [L], $c_{O_2}^0$ is the fraction of dioxygen in the inhaled air (0.2), H is the Heaviside function and Q is the flow of oxygen through the alveolar-capillary membrane [$L \cdot s^{-1}$]. The first term of the right-hand side represents the inspiration, the second the expiration and the last one the oxygen that diffuses to the blood through the alveolar-capillary membrane.

- Variation of O_2 quantity in the capillary blood

$$\frac{d}{dt}(V_c (\sigma P_c + 4Cf(P_c))) = Q = D_m (P_A - P_c)$$

Where V_c is the volume of capillary blood for one compartment [L], σ is the oxygen solubility in plasma [$mol^{-1}L^{-1}mmHg^{-1}$], C is the concentration of hemoglobin [$mol \cdot L^{-1}$], P_A is the partial pressure of oxygen within the alveoli [cmH_2O] and D_m is the membrane diffusing capacity [$mol \cdot s^{-1} \cdot mmHg^{-1}$]. f represents the Hill's curve given by the following relationship: $f(P) = \frac{P^n}{\tilde{P}^n + P^n}$ where $n=2.5$ and $\tilde{P} \approx 26mmHg$. D_m was weighted with a coefficient z ($z = 1$: healthy tissue, $z = 0$: non aerated tissue) according to the corresponding voxel HU in the patient CT-scan to simulate a lower diffusion in pathological tissue. To close this system of equation, we need a relationship between P_A and c_A . According to (Jbaily et al. 2020), we have:

$$P_A = c_A \times \frac{M_G}{M_O} (P_A - P_{vap} + P_{atm})$$

Where M_G and M_O are respectively the gas mixture and oxygen molar masses [$\text{g}\cdot\text{mol}^{-1}$], P_A , P_{atm} and P_{vap} are respectively the alveolar (retrieved from the mechanical model), atmospheric and vapor pressures [cmH_2O]. M_G was deemed constant throughout the respiratory cycle. To simulate the heart beats that have a frequency that differs from the respiration one, the variable P_c was set to 40mmHg (venous blood O_2 partial pressure) at every τ_b , the heart period.

2.3 Implementation and simulation

Following the mechanical model framework, the physiological model was implemented using the Julia language. A simulation of an ARDS patient MV throughout 10 respiratory cycles was performed on $N=512$ compartments. The main parameters of this simulation are regrouped in Table 1.

Table 1: Simulation parameters

Peep	P _{plat}	τ_b	Respiratory rate	D _m
16 cmH ₂ O	26 cmH ₂ O	0.45s	20 min ⁻¹	3.5e ⁻⁶

3. Results and discussion

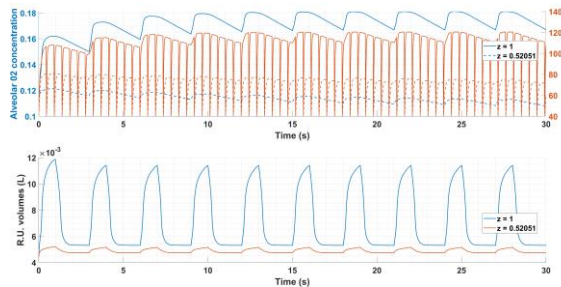


Figure 2 : Respiratory unit volume (bottom), O_2 concentration and capillary partial pressure (top) for 10 respiratory cycles.

The volume, alveolar O_2 fraction and capillary oxygen partial pressure time evolutions on 10 respiratory cycles are given in Figure 2. These curves clearly show the influence of the weighting parameter z , and thus the pathology, on both the ability of the lung to expand and diffuse gases through the alveolar-capillary membrane. We can see that the blood oxygenation stabilizes at a much lower value (76 mmHg against 120 mmHg for maximal P_c value on a respiratory cycle).

This figure also illustrates the interplay between lung biomechanics and physiology, since it shows how impaired inflation of a respiratory unit decreases the quantity of oxygen it diffuses. It reveals the potential of the model to investigate either the effect of MV parameters, such as Peep or prone positioning, on the patient oxygenation, and thus the potential of such a model to assist the clinicians in their decision-making process. This is further supported by the low computational cost of a simulation (around 10 minutes

for the simulation presented here). However, the diffusion model that is currently implemented is very simplistic. Discrepancies in the blood flow between respiratory units is not represented while it is known to be inhomogeneous throughout the lung, especially in case of ARDS (Cressoni et al. 2014). Future works should consider a spatial distribution for V_c , the capillary blood volume. Moreover, the way in which the pathology is modeled, i.e., through the weighting coefficient of both the compartments' compliance and diffusivity coefficient should be improved. Indeed, while it reproduces tendencies clinically observed, it is too simplistic to inform clinical decisions.

4. Conclusions

A homegrown computational framework designed to simulate pathological lung biomechanics was extended with a gas exchange model to describe the physio-mechanical coupling inside the respiratory system. Simulation on an ARDS patient was performed. Results were in accordance with tendencies observed in a clinical context, however the simplistic nature of the model implemented does not allow to make quantitative predictions. Future works will aim at making the diffusion model more sophisticated.

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