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Influence of Ventilation Mode on Blood Oxygenation – Investigation with Polish Virtual Lungs and Italian Model of Circulation

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Positive alveolar (P_A) and thoracic (P_T) pressures during artificial ventilation disturb pulmonary circulation, and might influence arterial blood oxygenation (PaO_2). Initial analysis of such influence of different artificial ventilation modes is the goal of this paper. Previously elaborated virtual respiratory system (IBIB PAS, Warsaw, Poland) and cardiovascular system model (ICP CNR, Rome, Italy) were connected with two files-buffers to work as one virtual cardio-pulmonary system. Dependence of PaO_2 on two methods (continuous inspiratory airflow (VCV) or pressure (PCV)), two ventilatory frequencies (fV = 15 or 7.5/min), and two values of the minute ventilation (Vmin = 6 or 8L/min) was investigated. Perfusion dependence on gravity was neglected as the virtual patient was in the supine position. Simulations showed that when fV = 15/min, neither the used method nor Vmin influence pulmonary blood flow significantly, whereas they influence the flow during expiration when fV = 7.5 (blood flow falls more for PCV and Vmin = 8 L/min). Vmin more significantly influences alveolar partial pressure of oxygen (PO_2) when fV = 15/min. PO_2 was greater for PCV. As effects on the flow and PO_2 were contradictory, PaO_2 was almost independent of the used method and fV. It depended on Vmin more significantly if fV = 15/min.

K e y w o r d s: virtual physiological human, respiratory system, cardio-pulmonary interaction

1. Introduction

Although it is possible to ventilate lungs without their movement [1], conventional ventilation is connected with thoracic pressure (*Pt*) cyclic changes caused by

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a respirator. Cardiovascular system (CVS) work is influenced by *Pt* as the chest contains a significant part of the cardiovascular system: the whole pulmonary circulation, the left and right atriums and the ventricles as well as the large vessels. Hence it appears that there is a mechanical interaction between the respiratory system (RS) and CVS. Additionally, both systems realize the common task, i.e. they have to cooperate to perform the gas exchange and gases transfer from/to ambient air to/from cells. The existence of both interaction and cooperation suggests that CVS modeling cannot be really accurate without RS modeling unless a specific problem is analyzed and the influence of RS may be neglected. On the other hand, ventilation efficacy analysis with RS models should be connected with CVS modeling since blood oxygenation is the final task of ventilation.

Despite that respirators have been used for more than 50 years and many different modes of ventilation were tested during that time, some effects of positive *Pt* and airway pressure being dangerous to the patient could not be completely eliminated. Such unfavorable influence of artificial ventilation concerns lungs as such and their ability to perform gas exchange. The influence of *Pt* is favorable if breathing is spontaneous, because negative *Pt* makes pulmonary blood flow easier during inspiration, i.e. when the partial pressure of oxygen (P_AO_2) is higher, which makes gas exchange more effective. However, non-physiological positive pressure *Pt*, which appears in the lungs during artificial ventilation or ventilatory support, influences the hemodynamics unfavorably [2, 3]. In particular, positive *Pt* decreases pulmonary blood flow pressing pulmonary vessels, and thus increasing their resistance.

Generally, it is assumed that the mean *Pt* is the best measure of influence of artificial ventilation on hemodynamics [2, 3]: the greater the mean *Pt*, the more unfavorable the influence. Therefore, for example, keeping spontaneous breathing, even weak, is beneficial because it partially compensates positive pressure caused by respirator.

Inspiratory airflow pattern may influence gas exchange efficacy. The airflow that declines from the maximal value to zero is assumed to be optimal [4, 5]. However, such airflow causes the greatest mean Pt since almost the whole tidal volume is pressed into lungs at the inspiration beginning. As blood oxygenation being the final task of ventilation depends on both gas exchange and pulmonary blood flow, it seems to be necessary to analyze how different methods of ventilation influences the oxygenation, in particular, which mechanism dominates: whether influence on blood flow or on gas exchange. Initial analysis of the problem can be performed with modeling.

2. Methods

2.1. Models

A model of gas transfer and exchange is the heart of the computer system that has been used to model dependence of blood oxygenation on the cardio-pulmonary

interaction. As the gas transfer in airways depends on the airflow rate and the gas exchange depends on the alveolar pressure, the model of gas transfer and exchange has to cooperate with a model of respiratory system mechanics. On the other hand, as gas exchange depends on blood flow through pulmonary capillaries and gas transport with blood depends on blood flow through the other vessels, the model of gas transfer and exchange has to cooperate with a model of cardiovascular system mechanics. Therefore, the used system is an ensemble of three models. Each of them was previously used as a stand-alone model in different studies. In this paper, all the models were connected each other to simulate the cardio-pulmonary interaction.

2.1.1. Virtual Respiratory System – a Model of Respiratory System Mechanics

The virtual respiratory system that is used in the analysis presented here has been elaborated in the Institute of Biocybernetics and Biomedical Engineering, PAS (Warsaw, Poland). The system consists of:

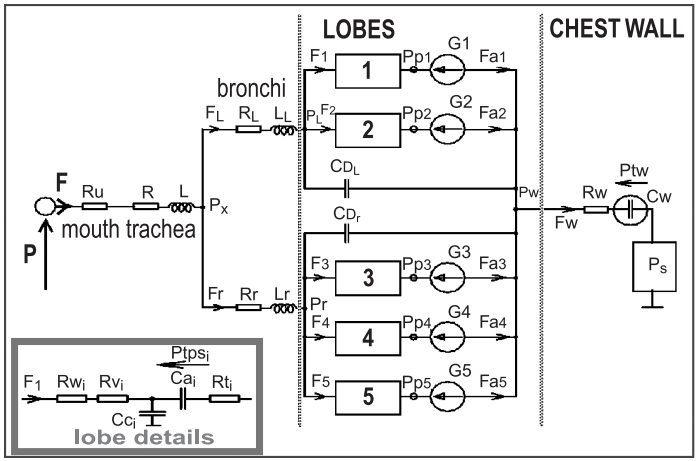
- the model of respiratory system mechanics, previously developed as a stand-alone virtual respiratory system presented in several articles (e.g. [6–8]),
- the model of gas transfer and exchange presented in other papers (e.g. [1]).

Figure 1a presents the general structure of the mechanics model. The main features of the model are:

1) separation of the lungs and chest;
2) division of the lungs into lobes (in Fig.1a, $i = 1...5$ identifies upper left lobe, bottom right lobe, respectively);
3) division of the airway resistance into:
   (a) resistances that depend on the lung volume (Rv_i in Fig. 1a) – resistances of the smallest bronchi that are a component of the lung tissue;
   (b) resistances that depend on the transmural pressure (Rp_i in Fig. 1a) – resistances of the bronchi that may collapse;
   (c) resistances of the large bronchi;
4) many others such as: nonlinearity of parameters, influence of gravity (G_i), air compressibility (Cc_i), etc.

Some of the model elements shown in Fig.1a represent parameters, which are described by single numbers. Such parameters are: Ru, R, R_L, R_r, Rt_i, Rw and L, L_L, L_r – (resistances of mouth, trachea, left and right main bronchi, lung tissue, chest tissue, and inertance of trachea, left and right main bronchi, respectively). Data for the model for the "standard" (healthy) human being have been collected on basis of the accessible literature. In particular, R, R_L, R_r, L, L_L, L_r were calculated from dimensions of trachea or main bronchi, while Rt_i, Rw were estimated on the basis of data from [9]. Equations describing non-linear elements as well as their derivations have been presented in detail in [7] (the model is also presented in free online available [8]).

a)



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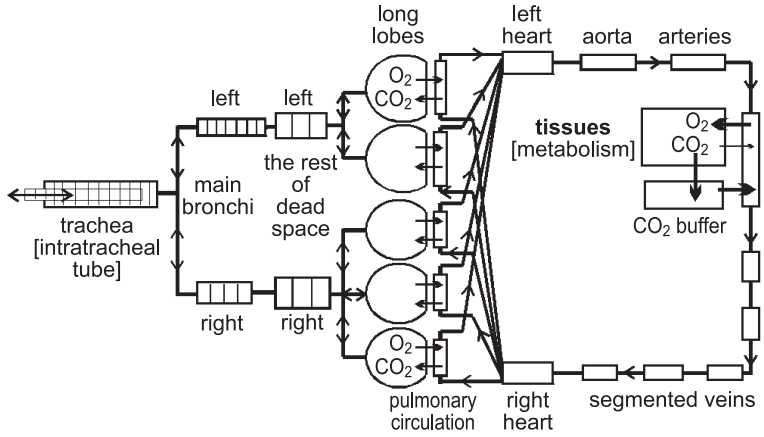

Fig. 1. Virtual respiratory system:

a) Simplified scheme of the mechanics model. *Ru*, *R*, R_L, R_r – upper and main airways resistances, *L*, L_L, L_r – inertances, C_{DL}, C_{Dr} – the parts of the dead space different from the trachea and main bronchi (indices $_L$ and $_r$ concern the left and right lung, respectively), *Cw*, *Rw* – chest wall compliance and viscosity, *Ps* – respiratory muscles, *Pw* – intrapleural pressure. Numbered boxes describe lobes (i = 1, 2 – left upper, lower lobe, 3, 4, 5 – right upper, middle, lower lobe): Rp_i – the "resistance" of the bronchi that may collapse, Rv_i – the resistance of smallest bronchi, Cc_i – air compressibility, Ca_i – the lobe compliance, Rt_i – lobe tissue viscosity b) An illustration of the gas transfer and exchange model. The trachea (and an intratracheal tube, if used), main bronchi, and the rest of the dead space are divided into segments. Gases move into/from lobes in accordance with gas exchange as well as their concentration in the segments and airflows supplied by the mechanics model

When the model of respiratory system mechanics was used as the stand-alone virtual respiratory system, it calculated the volumes of all respiratory system elements. Now, the volumes are supplied by the gas exchange and the transfer model. The above concerns the volumes of: lobes (Va_i), dead spaces (V_{DL} and V_{DR}), and compressed air (Vc_i), i.e. the 'charge' of Ca_i, C_{DL} and C_{DR}, Cc_i, respectively (Fig. 1a). Pressures and airflows calculated by the mechanics model are the input of the gas transfer and exchange model.

2.1.2. Virtual Respiratory System – a Model of Gas Exchange and Transfer

The model of gas transfer and exchange was presented in details in [1]. It consists of gas exchange module (GE), airway gas transfer module (AGT), and gas transport in CVS (CVGT).

AGT utilizes airflows supplied by the mechanics model whereas the mechanics model utilizes volumes calculated by AGT. GE utilizes gases saturations in venous blood supplied by CVGT and partial pressures determined with data supplied by the mechanics model and AGT.

AGT: It has been assumed that the respiratory system consists of (Fig. 1b): the trachea, an intratracheal tube (if it is used), the main bronchi (left and right), the rests of the left and right dead spaces (corresponding to C_{DL} and C_{DR} in the mechanics submodel), and the lobes. Alveoli are small, and thus it can be assumed that if a portion of a gas flows into an alveolus, it is present immediately in the whole volume of the alveolus. Therefore, a lobe is treated as one segment despite that its volume is greater than the whole dead space. The other parts of RS are divided into several segments. Transfer of each gas from a segment to neighbor one(s) is proportional to its concentration in this segment and to the airflows that have been supplied by the mechanics model. In the case of the lobes, the airflows are supplemented with particular gases exchange supplied by GE. The sum of the volumes of all gases in elements of changeable volume gives the volumes that are utilized by the mechanics model in formulas determining pressures.

GE: It has been assumed that blood flowing through pulmonary capillaries has enough time to be fully saturated. It means that end-capillary tensions are equal to the alveolar partial pressures for all gases in lungs. Partial pressure of a gas is determined using the alveolar pressure supplied by the mechanics model and the amount of the gas in a lobe calculated by AGT. Saturations of O_2 (SaO_2) and CO_2 ($SaCO_2$) are calculated from the partial pressures P_{O_2} and P_{CO_2} with formulas fitting the real dependences between the pressures and saturations very good**.** It has been assumed that saturations of the other gases are proportional to their partial pressures because they do not create chemical compounds with blood components (carbon monoxide is not considered). The saturations are the input of CVGT. Gas exchange, i.e. the quantum of a gas that flows from/into a lobe into/from blood, is proportional to the blood flow rate and the difference between the arterial and venous saturations of this gas.

CVGT: This module describes gases transport with blood and their metabolic changes. Blood, oxygenated and decarbonated in particular lobes, is mixed in the left heart and flows through arteries to the systemic microcirculation, where O_2 is consumed. About 10% of produced CO_2 passes directly to the blood, the rest moves to the buffer [1]. Carbonated and deoxygenated blood flows through veins to the right heart and the pulmonary circulation. Both the gas exchange and transport require blood flow rate determination.

2.1.3. Model of Cardiovascular System Mechanics

Like AGT cooperates with the model of RS mechanics, CVGT has to cooperate with a model of CVS mechanics. In particular, gas exchange depends on blood flow rate through the pulmonary capillaries whereas gas transport depends on the systemic circulation. On the other hand, pulmonary blood flow depends on the alveolar pressure (P_A) and *Pt*. Both the pressures have to be supplied by some model of RS mechanics.

The above means that it would be impossible to analyze influence of a ventilation method on blood oxygenation without the use of a model of CVS mechanics unless a specific problem is studies as in [1]. Therefore, the virtual RS was connected with Cardiosim™ – the CVS model (Fig. 2) elaborated in the Institute of Clinical Physiology, CNR (Rome, Italy). It was derived from a previous numerical model [10] and was the basis for other applications including surgical and intensive care unit data analysis [11] and hybrid modeling [12]. The model is composed of six modules: left and right heart, systemic and pulmonary arterial circulation, systemic and pulmonary venous circulation. Its modular structure permits the easy replacement of the models included in any of its modules.

The left and right heart modules comprise two variable elastance functions, relating ventricular pressure to ventricular volume through isovolumetric time varying elastance in order to represent right and left ventricular ejection [13]. The ejection function includes an internal ventricular resistance. The left and right ventricular fillings are represented as a sum of exponentials [14]. The connection of the ventricle to the circulatory network is achieved by valves, that are assumed to be ideal, i.e. when the valves are open the flow through is proportional to the pressure drop, and when these are closed there is no flow. Atria are passive and represented by a single compliance. Arterial trees are represented by modified windkessels.

Values of pulmonary and systemic arterial compliances were taken from literature or estimated, when necessary and possible, using a decay time method applied to experimental data. Impedance of the pulmonary microcirculation (Z_{ap} in Fig. 2) depends on the arterial pulmonary, the venous pulmonary, and the alveolar pressures. Pulmonary arterial resistance (R_{ap} in Fig. 2) depends on the difference between the arterial pulmonary pressure and *Pt*, whereas pulmonary venous resistance (R_{vp} in Fig. 2) depends on the difference between the venous pulmonary pressure and *Pt*. As those differences are transmural pressures, they depend on blood volumes contained in the vessels. Details of the model were presented in [10, 15].

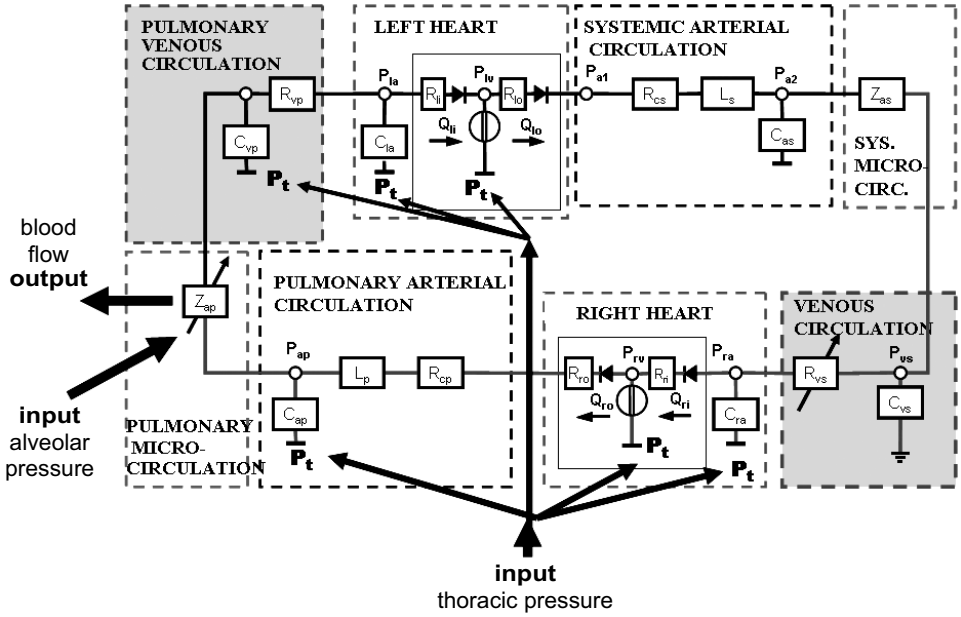

Fig. 2. A scheme of the cardiovascular system model (see [15] for details) that supplies blood flow through pulmonary capillaries (the model output) utilized by the virtual respiratory system to calculate gas exchange. Although blood flows through the other parts of the cardiovascular system are also model outputs (utilized by the virtual respiratory system to determine gas transport), they are not indicated to make the figure more clear. The thoracic and alveolar pressures being outputs of the virtual respiratory system are the model inputs

2.2. Models Connections

Since the virtual RS and CVS models are two different, stand-alone models written in different computer languages, their connection required special procedures. On the one hand, the procedures should transfer data between the models. On the other hand, the procedures have to synchronize work of both the models.

Taking the above into account, the models were connected with two files-buffers: resp_to_cardio and cardio_to_resp. The virtual RS wrote the values of alveolar and thoracic pressures to the resp_to_cardio buffer (after each 1 msec of simulated time). The CVS model used these values to calculate the pulmonary blood flow. The pulmonary and systemic flows were written by the CVS model to the cardio_to_resp buffer and used by the virtual RS to calculate gas exchange and transport. Figure 3 shows the flow-diagram of the cooperation with both buffers.

2.3. Simulation Procedures

The parameters of artificial ventilation of the “standard” virtual patient with the metabolic oxygen consumption equal to 0.3 L/min were as follows:

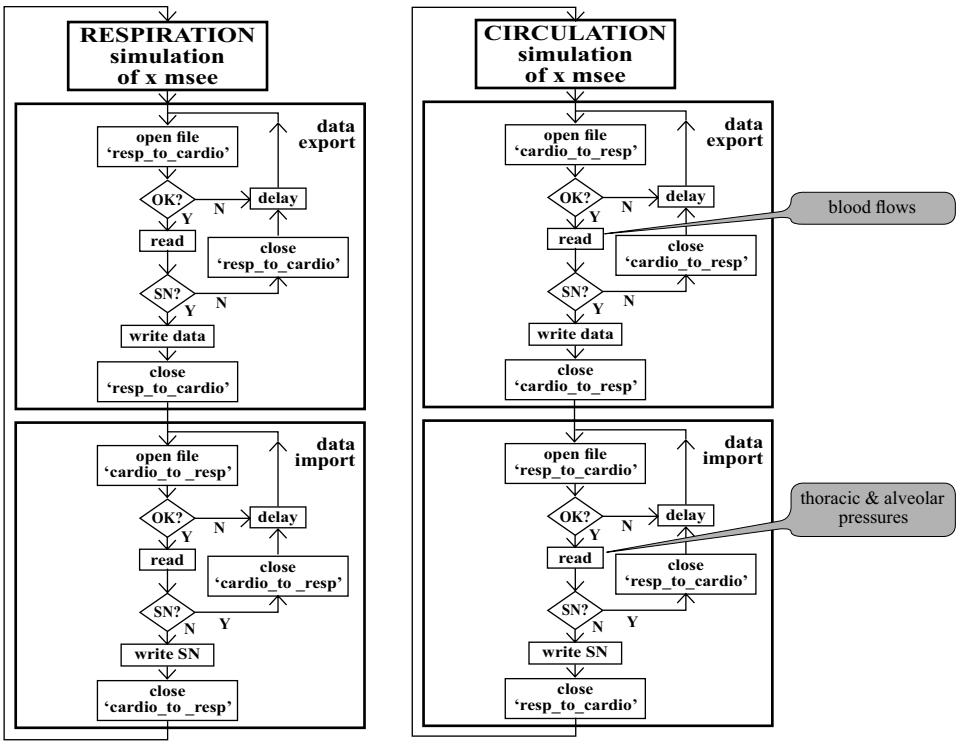

Fig. 3. The flow-diagram of the use of buffers resp_to_cardio and cardio_to_resp by the virtual respiratory system (on right) and the cardiovascular system model (on left). SN is a number that cannot be true data (e.g. 99999). SN in a buffer means that data has been already used by one model, and thus the second model can write own data for the next step of simulation

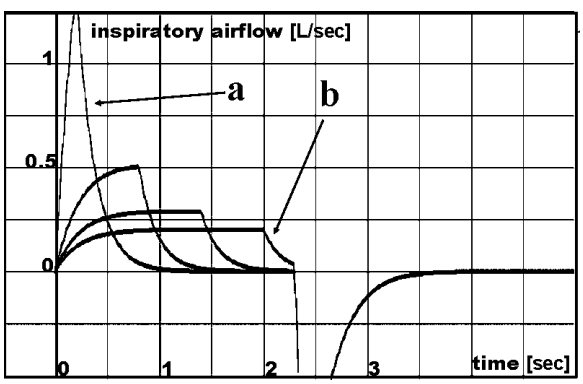

Fig. 4. The analyzed airflow patterns (the flow values for ventilation with frequency 12/min when the minute ventilation was equal to 6 L/min are shown, for different frequency or ventilation the values are adequately different). a – falling airflow as during the conventional pressure controlled ventilation (PCV) with continuous inspiratory pressure; b – continuous (except the inspiration beginning and end) airflow as during the conventional volume controlled ventilation (VCV)

– the ventilation frequency (f_V) equal to 15 or 7.5/min,
– the minute ventilation (Vmin) equal to 6 or 8 liters/min,
– the inspiratory airflow patterns as shown in Fig. 4.

For each combination of the values of the above parameters, blood flow through pulmonary capillaries, alveolar partial pressure of oxygen (P_AO_2), and arterial blood oxygenation expressed as the percentage of blood saturation (SaO_2) were measured (i.e. simulations were logged). As there is a slow buffer of carbon dioxide in CVGT (Fig. 1b) simulating real slow buffers [1], measurements were performed not before 6 minute of simulated time after a change of one of the above ventilation parameters to obtain the steady state of cyclic changes of partial pressures in lungs and gases saturations in blood.

3. Results

Neither the airflow pattern nor Vmin influenced pulmonary microcirculation when f_V = 15/min (Fig. 5a). If f_V = 7.5/min then both the pattern and Vmin influenced the blood flow through pulmonary capillaries, however, the changes were more significant during expiration than inspiration. The airflow pattern that corresponded to the conventional pressure controlled ventilation (PCV – see Fig. 4) decreased the flow more than the pattern that corresponded to the volume controlled ventilation (VCV – see Fig. 4).

As it could be expected, Vmin influenced P_AO_2, however, for the same Vmin, $P_AO_2$was greater for (Figs. 5b and 6b):

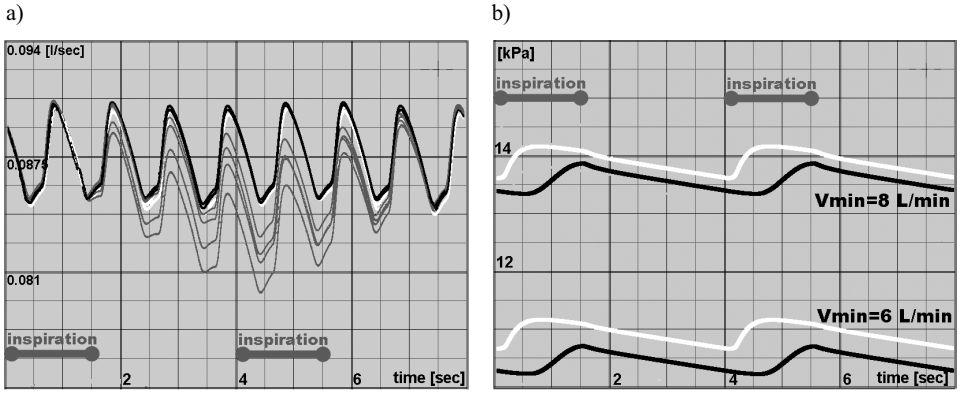

Fig. 5. Simulations for the pressure controlled (white) and volume controlled (black) ventilation with frequency equal to 15/min and minute ventilation Vmin = 6 and 8 L/min.
a) blood flow through pulmonary capillaries (gray curves – the flow for the frequency equal to 7.5/min); note that neither the ventilation mode nor minute ventilation influences the flow (hence white curves are almost invisible)
b) alveolar partial pressure of oxygen

– PCV,
– F_V = 7.5/min.

Table 1 presents values of SaO_2 for each combination of values of the ventilation parameters. SaO_2 for Vmin = 8 L/min is higher than for Vmin = 6 L/min, however, SaO_2 is more sensitive to Vmin for f_V = 15/min. SaO_2 is higher for f_V = 7.5/min than for f_V = 15/min, and higher for PCV than for VCV, however, influence of the airflow pattern and f_V on SaO_2 is weak.

4. Discussion

The paper seems to be the first report on the connection of two virtual organs, such as a model of the cardiovascular system mechanics and a model of the respiratory system simulating both its mechanics and gas transfer as well as gas exchange, into one ensemble. They were created by different authors, written in different computer languages. That is an interesting approach as the European Alliance for Medical and Biological Engineering and Science recommended to the attention of the European Commission to create virtual organs, which would be connected into one Virtual Physiological Human in the future [16].

Such an ensemble enables the authors to analyze interaction between the respiratory and cardiovascular systems from both mechanical and gas transfer/exchange points of view. There were published a few papers concerning the interaction. However, either mechanics of one system or gas transfer and exchange was not taken into account. For example, the mechanical interaction between both systems during breath-hold diving was simulated in detail by Fitz-Clarke [17], but neither gas transport nor exchange was analyzed. On the other hand, both respiratory system mechanics and airway gas transfer as well as gas exchange were taken into account in [18], however, the mechanical interaction between the systems was neglected. Moreover, in that model gases are transported by a liquid, whereas the gases 'transport themselves' during normal ventilation. Also in some other models (e.g. [19, 20]), the cardiovascular system mechanics is either simplified as in [1] or neglected at all.

Hence it appears from the above that the ensemble presented here covers the greatest range of phenomena. Certainly, some limitations concern also the models of the ensemble, and thus the simulations and analyses of their results that are presented in this paper have to be treated as an initial investigation only. The following limitations may have significance:

- the venous return simulation is not absolutely correct (e.g. collapse of the main veins is not simulated by the cardiovascular system model);
- the pulmonary circulation in the cardiovascular system model is not divided into parts, therefore it has been impossible to simulate differences between ventilation and perfusion, asymmetrical lungs pathology, etc.;

- additionally, since the lungs are divided into large parts (lobes), it is not possible to take into account such microscopic phenomena as diffusion screening (therefore, only 'macroscopic', averaged values of saturations, partial pressures, etc. have been considered);
- interaction between O_2 and CO_2 saturations are not taken into account in the model of gas exchange and transfer;
- influenced by chemoreceptors, autonomic nervous control of heart rate and other parameters of the cardiovascular system work was omitted (however, such a control would have meaning for conclusions only if different ventilation modes caused significantly different saturations of O_2 and CO_2; since the differences in saturation appeared small, the autonomic control would have no reason to change the cardiovascular system work).

Despite the above limitations, the qualitative results seem to be reliable although it may seemingly be a surprise that neither *V*min nor the pattern of the inspiratory airflow influences the blood flow through the pulmonary capillaries when f_V =15/min (Fig. 5a). The mean *Pt* during the PCV ventilation is higher than during the VCV ventilation. Both the mean *Pt* and its amplitude it higher for *V*min = 8 L/min than for *V*min = 6 L/min. The same concerns P_A. Therefore, it might be expected that pulmonary vessels are stressed, what should result in a resistance increase and blood flow fall. However, as the simulations showed (Fig. 6a), an increase in *Pt* or P_A, needs time to influence the flow. It could be explained as follows. Since the right and left heart separate the pulmonary circulation from the systemic one, changes in the pulmonary blood volume cannot be continuous: a volume increase may appear only during the right heart systole, whereas a decrease – during the left heart diastole. Moreover, those increase and decrease are limited by properties of both hearts. Therefore, the volume may change only

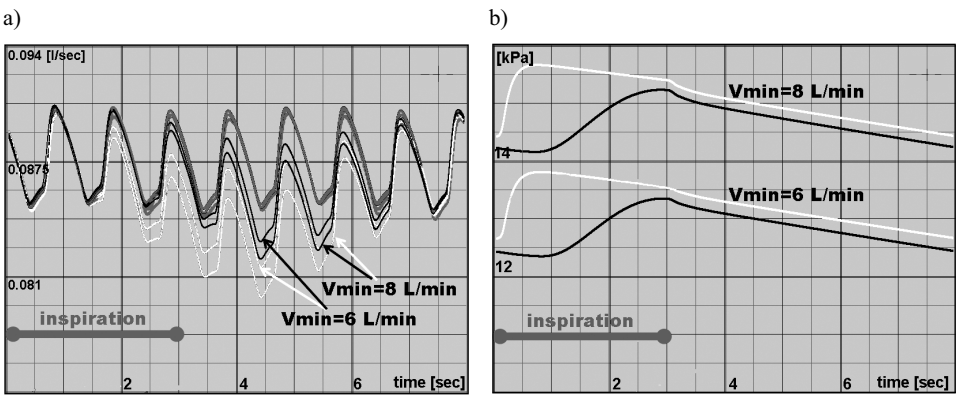

Fig. 6. Simulations for the pressure controlled (white) and volume controlled (black) ventilation with frequency equal to 7.5/min and minute ventilation *V*min = 6 and 8 L/min. a) blood flow through pulmonary capillaries (gray curves – the flow for the frequency equal to 15/min), b) alveolar partial pressure of oxygen

step by step according to the heart work, and if the number of the steps (i.e. the number of heart evolutions) is small, the volume cannot change significantly. As resistance of vessels depends on their cross-section being proportional to the volume of vessels, also the resistance as well as the mean blood flow may change only if there is a sufficient number of heart evolutions during the inspiration. If the number of the evolutions is sufficient to decrease the volume, some time is necessary to increase the volume during the expiration. Hence the mean flow is decreased during the expiration beginning, too (Fig. 6a), despite that *Pt* is already negative and P_A is equal to zero. As during PCV *Pt* and P_A attain high values earlier than in the case of VCV, more significant decrease of the mean flow is observed in PCV. Certainly, the periodic instantaneous blood flow depends on periodic pulmonary arterial-venous pressure gradient.

The fact that the mean P_AO_2 is greater for PCV than for VCV (Figs. 5b and 6b) is obvious: in the case of PCV the maximal value is attained earlier, near the inspiration beginning. It causes that both arterial (Table 1) and venous blood is a little better oxygenated. It was not analyzed in details why P_AO_2 is also greater during expiration (it is probably connected with blood as a O_2 container because P_AO_2 does not change rapidly but it alters step by step after ventilation change before achieving the steady state).

Table 1. Oxygen saturation in arterial blood (in %) for different minute ventilations (*V*min), ventilation frequencies (f_V), and patterns of inspiratory airflows (PCV – conventional pressure controlled ventilation, VCV – conventional volume controlled ventilation)

	f_V	15/min	7.5/min
*V*min	pattern	PCV ÷ VCV	PCV ÷ VCV
8 L/min		93.8 ÷ 93.6	94.5 ÷ 94.2
6 L/min		91.2 ÷ 90.6	93.4 ÷ 93.0

The fact that the mean P_AO_2 is greater for $f_V = 7.5$ than for $f_V = 15$ (Figs. 5b and 6b) is also understandable. Indeed, the alveolar ventilation is equal to *V*min less the dead space ventilation. As the dead space ventilation is equal to the dead space volume multipied by f_V (i.e. the smaller the f_V, the smaller the dead space ventilation), *V*min less the dead space ventilation is greater for the smaller f_V.

For the evident reason, P_AO_2 is greater for greater *V*min. The fact that P_AO_2 is more sensitive to *V*min change when $f_V = 15$/min is also connected with the dead space ventilation (relative increase of the alveolar ventilation with the same increase of *V*min is less significant when f_V is lower).

As positive P_A contradictorily influences P_AO_2 and the blood flow through pulmonary capillaries, effects of their changes seem to partly compensate one another. Hence the influence of the airflow pattern on blood oxygenation is so small (Table 1).

Influence of f_V is also small unless the alveolar ventilation is significantly smaller than Vmin because of the relatively great dead space ventilation, i.e. when Vmin is small and f_V is high (Table 1).

PCV with low f_V seems to be the most favorable from the point of view of blood oxygenation (Table 1), despite that such ventilation influences the blood flow in the greatest degree. However, to keep Vmin unchanged PCV requires higher mean P_A and low f_V requires both greater tidal volume and higher peak P_A. Both high mean and peak P_A are dangerous for lungs because of risk of barotrauma, whereas big tidal volume is dangerous because of risk of volutrauma. As the method of ventilation moderately influences blood oxygenation, the mechanical effect on lungs rather than the oxygenation should be taken into account during the ventilation parameters settings. For example, despite almost the same oxygenation (Table 1), PCV with f_V = 7.5/min and Vmin = 6 L/min is worse than VCV with f_V = 15/min and Vmin = 8 L/min because such PCV requires the tidal volume equal to 0.8 L and the peak pressure equal to 0.85 kPa, while such VCV needs 0.53 L and 0.52 kPa, respectively. Certainly, the given values of the pressure concern the virtual patient with standard (average for the human being) respiratory system compliance. In the case of a sick patient with a lower compliance, the difference between pressure values would be greater.

5. Conclusions

The ensemble of the virtual respiratory system and the cardiovascular system model seems to simulate quite reliably the cardio-pulmonary interaction since the obtained results can be explained qualitatively with the physiological knowledge. Quantitative results appeared a little surprising as the positive thoracic and alveolar pressures seem not to influence noticeably the pulmonary flow if a patient is ventilated with such frequency that the inspiration time is not significantly greater than the duration of two heart evolutions.

The positive alveolar pressure influences blood oxygenating in two contradictory ways: a) the greater the pressure, the smaller the blood flow through pulmonary capillaries may be, b) the greater the pressure, the greater the alveolar partial pressure of oxygen.

They partially compensate one another, and thus influence on blood oxygenation is weak. Hence it appears that risk of lungs injury rather than oxygenation should be taken into account when ventilation parameters are set.

Acknowledgments

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