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The Virtual Cardio-respiratory System: a Sub-model of Gas Exchange and Transfer

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A virtual cardio-respiratory system (CRS) is proposed for testing ventilatory support and scientific hypothesis. It may appear more convenient than experiments on animals or limited investigations on patients. In particular, there are no limitations for manipulation of virtual CRS parameters while such manipulation is difficult or impossible in the case of real CRS. The virtual CRS architecture: The proposed virtual CRS consists of: (a) the sub-model of respiratory system mechanics (RSM) previously used as the stand-alone virtual respiratory system, (b) a sub-model of gas exchange and transfer in the respiratory and circulatory systems (GET), which is constituted with three modules: gas transfer in respiratory system, gas exchange in lungs, and gas transfer in circulation. The GET utilizes airflows and pressures supplied by the RSM whereas the RSM utilizes volumes of gases supplied by the GET. Results: the CRS gave proper results for both respiration and respiratory arrest. In particular, if the CRS 'respired' with pure oxygen then arterial blood saturation with oxygen remained high for tens of minutes after respiratory halt when airways were open; otherwise atelectasis developed during 8–10 minutes. Like for real patients, carbon dioxide tension in blood decreased quickly when ventilation increased and it increased slowly when the ventilation fell.

K e y w o r d s: oxygen, carbon dioxide, respiratory arrest, respiratory system, saturation, virtual organs

Main abbreviations:

AGT — airway gas transfer module
CGT — circulatory gas transfer module
F_IG_j — inspiratory concentration of *j*-gas [%]
GE — gas exchange module
$i = 1..5$ — number of a lobe
$j = 0..4$ — number of a gas G (0-saturated H_2O vapor, 1-O_2, 2-CO_2, 3-N_2, 4-anasthetic)
P_AG_j — partial pressure of *j*-gas in lungs [kPa]
P_{atm} — the atmospheric pressure [kPa]
P_{H_2O} — saturated H_2O vapor pressure [kPa]

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P_{a_i} — pressure in *i*-lobe (with Patm as the origin) [kPa]
SaG_j — *j*-gas arterial saturation, e.g. SaO_2, $SaCO_2$ [mmol/l]
SvG_j — *j*-gas venous saturation [mmol/l]
V_{Gji} — amount of *j*-gas in *i*-lobe expressed with the volume that the gas would occupy at *Patm* and body temperature [liter]
V_{a_i} — the volume of *i*-lobe [liter]
V_{c_i} — the difference between $\sum_j V_{Gji}$ and V_{a_i} being the effect of compression, i.e. V_{c_i} is the gas volume that has to be added to Vai to increase the pressure from *P*atm to P_{a_i} [liter]

1. Introduction

Despite that proper mechanical behavior of the respiratory system is a very important factor for human health, proper gases gas exchange in lungs and gases transfer both in respiratory and cardio-vascular systems are those factors which have real meaning. Indeed, delivery of oxygen to tissues and carbon dioxide removal is the final task of the respiratory system. Therefore, although the mechanical activity may be one of the necessary conditions, it is not any sufficient condition of oxygen delivery. As it will be shown below (3.2. Respiratory Arrest), the mechanical activity does not need to be even a necessary condition of proper blood oxygenation.

Previously, a model of the respiratory system mechanics was developed in the Institute of Biocybernetics and Biomedical Engineering in Warsaw [1], originally for respirators and ventilatory support testing [2–3] as well as research [4] and education [5]. For the reason presented above, it had to be supplemented with a model of gas exchange and transfer to allow treating it as a true virtual organ.

As it is illustrated in Fig. 1, no simple modification of a model of the respiratory system mechanics can introduce gas transfer because of fundamental differences in phenomena connected with the mechanics and the transfer. Therefore, a separate model of gas exchange and transfer has been proposed. Then, like other authors did (e.g. [6]), such a model has been connected with the previously built model of the respiratory system mechanics to create a virtual cardio-respiratory system.

2. Virtual Cardio-respiratory System

The virtual cardio-respiratory system that is presented in the paper consists of:

- The sub-model of respiratory system mechanics (Fig. 2), previously developed by the authors as the stand-alone virtual respiratory system presented in several articles [1–5].
- A sub-model of gas exchange and transfer in the respiratory and cardio-vascular systems (Fig. 3), which is the subject of this paper. Three following modules constitute this sub-model: airway gas transfer (AGT), gas exchange (GE), and circulatory gas transfer (CGT).

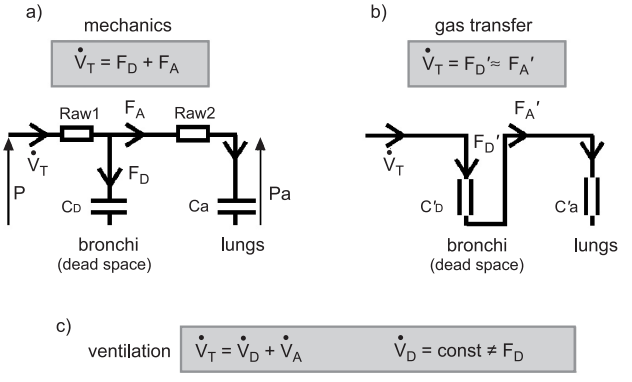

Fig. 1. An example illustrating difference between mechanics and transfer modeling; a) a simple model of lungs mechanics consists of compliant dead space (C_D) and lungs (C_a) connected with resistive bronchi (R_{aw1}, R_{aw2}). From the mechanical point of view, the dead space and the lungs are connected parallelly, and thus the total airflow being respiratory system ventilation is the sum of airflows F_D and F_A supplying C_D and C_a. Pressures and resistances determine these airflows; b) the gas transfer model corresponding to the above model of mechanics. Here, the dead space (C_D') and the lungs (C_D') are connected in series because the air volume that flows into the lungs (F_A') is approximately equal to the air volume that flows through the bronchi (F_D'), however, although the volumes are equal, the air is not the same. Pressures are not present in this model, airflows are determined by the respiratory system mechanics; c) typical, physiological approach to the ventilation different from the two above

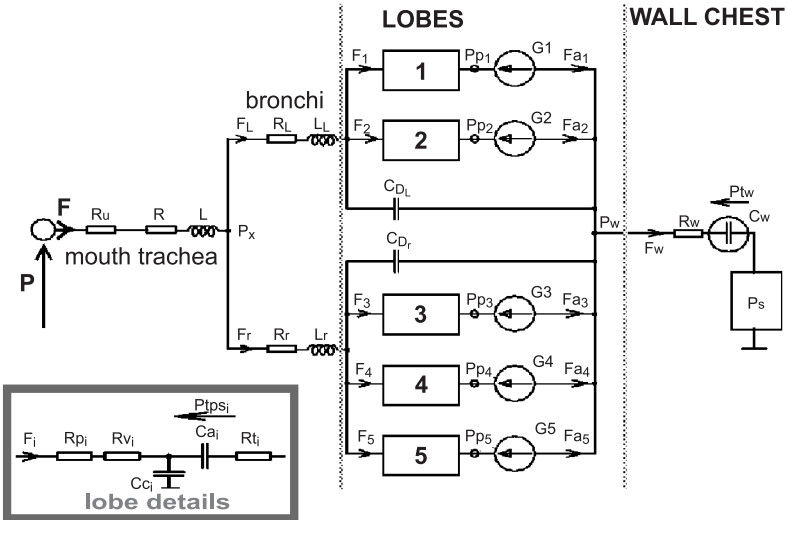

Fig. 2. Simplified scheme of the respiratory mechanics sub-model. *Ru*, *R*, R_L, R_r — upper airway resistances, *L*, L_L, L_r — inertances, C_{DL}, C_{Dr} — the parts of the dead space different from the trachea and the main bronchi (indices $_L$ and $_r$ concern the left and the right lung, respectively), *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 the smallest bronchi, Cc_i — air compressibility, Ca_i — the lobe compliance, Rt_i — lobe tissue viscosity

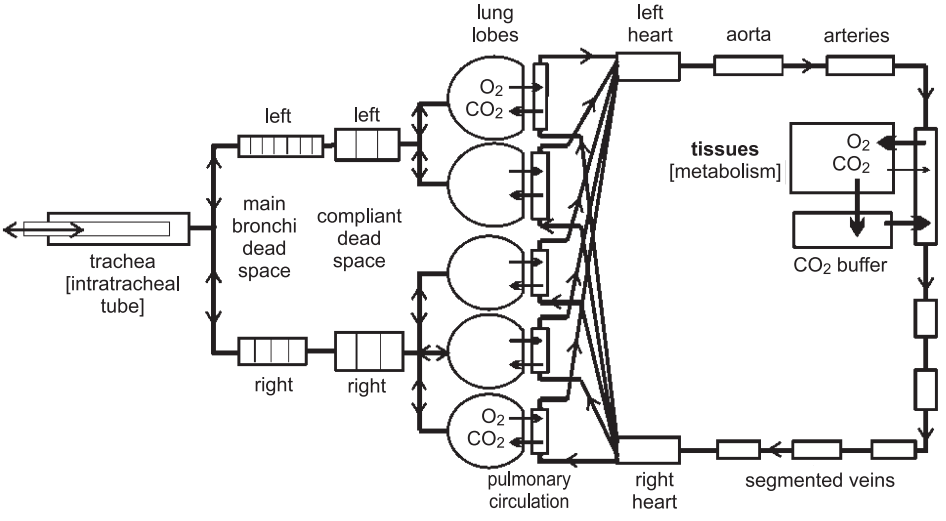

Fig. 3. An illustration of the gas transfer and exchange sub-model. The trachea (and an intratracheal tube, if used), the main bronchi, and the rest of the dead space are divided into segments. The gases move into/from lobes in accordance with their concentration in the segments and the airflows supplied by the mechanics sub-model. Blood, oxygenated and decarbonated in the 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. Carbonated and deoxygenated blood flows through veins to the right heart and the pulmonary circulation

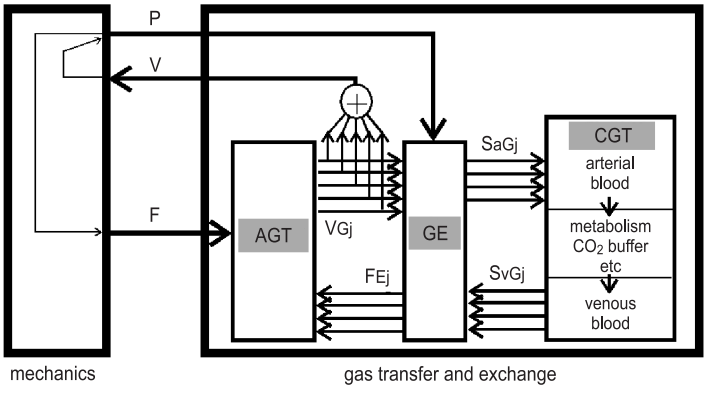

Fig. 4. Cooperation between the mechanics sub-model and modules of the gas transfer and exchange sub-model. The airway gas transfer module (AGT) calculates the volumes (V) of respiratory system elements utilizing convective gas movement (i.e. airflows F supplied by the mechanics sub-model) and gas inflow/outflow F_{Ej} being the result of gas exchange in the lungs. The mechanics sub-model utilizes V to calculate pressures P (using formulas describing compliances) and airflows F (using resistances and the pressures). The gas exchange module (GE) determines: (i) alveolar partial pressures and thus arterial blood saturations SaG_j utilizing P and volumes of the particular gases in the lungs V_{Gj}, (ii) gas exchange, i.e. F_{Ej}, utilizing SaG_j and SvG_j, where SvG_j are venous blood saturations calculated by the circulation transfer module (CGT)

It has been assumed that there are five gases in the respiratory system: saturated water vapor (SWV), oxygen (O_2), carbon dioxide (CO_2), nitrogen (N_2), and some anesthetic. All these gases, except SWV, may be compressed and can be transferred to/from blood. As SWV is the saturated vapor, its partial pressure is constant. Therefore, quantum of SWV in a lobe depends on the lobe volume only (i.e. it does not depend on the pressure in the lobe and thus SWV is not 'stored' in Cc symbolizing compressibility — see "Mechanics sub-model" below).

Modules and mechanics sub-model cooperation (Fig. 4): the AGT module utilizes airflows supplied by the mechanics sub-model whereas the mechanics sub-model utilizes volumes calculated by the AGT module. The GE module utilizes gases saturations in venous blood supplied by the CGT module and partial pressures determined with data supplied by the AGT module and the mechanics sub-model. Details are presented below.

2.1. The Sub-model of the Respiratory System Mechanics

Figure 2 presents the general structure of the computer model that is utilized as the mechanics sub-model of the virtual cardio-respiratory system. The main features of the sub-model are:

1) separation of the lungs and chest;

2) division of the lungs into lobes (i = 1..5 identifies upper left lobe, ….bottom right lobe, respectively);

3) division of the airway resistance into:

(a) the resistances that depend on the lung volume (Rv_i in Fig. 2) — resistances of the smallest bronchi that are a component of the lung tissue;

(b) the resistances that depend on the transmural pressure (Rp_i in Fig. 2) — 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 sub-model elements in Fig. 2 represent parameters, which are described by single numbers. Such parameters are: Ru, R, RL, Rr, Rti, Rw and L, LL, Lr — (resistances of mouth, trachea, left and right main bronchi, lung tissue, chest tissue, and inertance of trachea, left and right main bronchi, respectively). R, RL, Rr, L, LL, Lr were calculated from dimensions of trachea or main bronchi, while Rt_i, Rw were estimated on the basis of data from [7].

The most important non-linear elements of the sub-model are calculated with the following formulas:

1. The air compressibility 'compliance' symbolized by Cc_i in Fig. 2, i.e. the dependence between the alveolar pressure (Patm + Pa_i), the lobe volume Va_i, and the volume (Va_i + Vc_i) that the air in the lobe would occupy at Patm

$$Pa_i = P\text{atm} \cdot Vc_i/Va_i \tag{1}$$

2. The chest wall compliance symbolized by *Cw* in Fig. 2, i.e. the dependence of the trans-wall pressure *Ptw* on the chest volume V_w (*C*1, *C*2, and *C*3 depend on the patient state and properties)

$$p_{tw} = C1 \cdot V_w - \frac{C2}{\sqrt{V_w - C3}} \tag{2}$$

3. The lobe compliance symbolized by Ca_i in Fig. 2, i.e. the dependence of the trans-pulmonary pressure $Ptps_i$ on the lobe volume Va_i (*C*4 and *C*5) depend on the simulated patient, u_i — coefficient determining what part of the whole lungs is this particular lobe (u = [0.2, 0.25, 0.15, 0.2, 0.2])

$$Ptps_i = 1[kPa] \cdot \exp\left(C4 + Va_i \cdot \frac{C5}{u_i} \right) \tag{3}$$

4. The resistance Rv_i that depends on the lobe volume Va_i (a — a proportionality coefficient, $ccVa_i$ — critical closing volume)

$$Rv_i = a/(Va_i - ccVa_i) \tag{4}$$

5. The resistance symbolized by *Rp* in Fig. 2, i.e. the dependence of the airflow f on: the pleural pressure *Pp*, the pressure in the main bronchi *Pb*, and the pressure in the lungs *Pa* (b and k — coefficients dependent on the simulated patient)

$$f = \frac{Pb - Pa - b \cdot arctg\left(b \cdot \dfrac{Pb - Pa}{b^2 + (Pb - Pp) \cdot (Pa - Pp)} \right)}{k} \tag{5}$$

Data for the "standard" (healthy) human being have been collected on the basis of accessible literature. Details, esp. the derivation of the formula (5) and explanations of other formulas, as well as model verification have been presented precisely in [5].

When the mechanics sub-model 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 AGT module of the gas transfer and exchange sub-model (V in Fig. 4). 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. 2). Pressures and airflows calculated by the mechanics sub-model are the input of the gas transfer and exchange sub-model (P and F in Fig. 4).

2.2. The AGT Module — Gas Transfer in the Respiratory System

It has been assumed — in the gas exchange and transfer sub-model — that the respiratory system consists of (Fig. 3): 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 sub-model), and the lobes.

To allow simulating of different concentration of gases in various points of airways, each mentioned above element of the airways is divided into segments. The trachea and the intratracheal tube are divided into 2 cm long segments (assumed to be 22 mm and 18 mm in diameter, respectively). The main bronchi are not equal, therefore, the left main bronchus is divided into seven segments (11 mm in diameter) whereas the right one — into four segments (13 mm in diameter). Both the lengths and diameters (and thus the volumes) of the trachea, the tube, and the main bronchi are assumed to be unchangeable during respiration, i.e. they are not model variables (certainly, they can be changed as model parameters to simulate a patient different from the standard one). The rests of the dead spaces are divided into three segments of the volumes that change during respiration (the dead space of compliant airways).

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 its volume. Therefore, a lobe is treated as one segment despite that its volume is greater than the whole dead space.

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 sub-model. In the case of the lobes, the airflows are supplemented with particular gases exchange supplied by the *GE* module (F_{Ej} in Fig. 4). Although gas transfer with diffusion is not simulated directly, it is present, in fact, because of finite volumes of segments (the gas portion that flows into a segment 'diffuses' into the whole segment volume).

The volume of a gas in a segment at (n)-moment is equal to the volume at (n-1)-moment increased by the gas inflow and decreased by the gas outflow. The gas concentration in a segment is equal to the gas volume divided by the segment volume. The gas concentration in the inspired air is a parameter of the model. Since artificial ventilation or ventilatory support is mainly considered, it is assumed that the inspired air is saturated with H_2O by a respirator.

Sum of the volumes of all gases in elements of changeable volume gives the volumes that are utilized by the mechanics sub-model in formulas determining pressures (V in Fig. 4). For example, such sum gives Va_i and Vc_i used by the mechanics sub-model in the formula (3) for trans-pulmonary pressure calculation and in the formula (1) for alveolar pressures determination. Note: *SWV* is not a "component" of Vc_i, which has been explained above.

2.3. The GE Module — Gas Exchange

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 the all four gases. The assumption usually holds good except such cases as extreme exercises (blood flows very quick) or a diffusion disturbance (gas exchange needs more time).

The following formula determines the partial pressures (P_{Gji}) using the alveolar pressure (Pa_i) supplied by the mechanics sub-model and the amount of the gases (V_{Gji}) in a lobe calculated by the AGT module:

$$P_{Gji} = \left(Pa_i + Patm - P_{H_2O}\right) \cdot \frac{V_{Gji}}{Va_i \cdot \left(1 - P_{H_2O} / Patm\right) + Vc_i} \tag{6}$$

($Patm$, P_{H_2O}, Va_i, Vc_i, j — see Main abbreviations).

Saturations of O_2 and CO_2 are calculated with the following formulas:

$$\mathrm{SaO_2} = \sin\left(\frac{0.5 \cdot \pi \cdot P_{O_2}}{2 + P_{O_2}}\right) \cdot \left(\frac{9 \cdot \arctan\left(6.21 \cdot \left(P_{O_2} - 4\right)\right)}{\pi} + 5\right) \tag{7}$$

$$\mathrm{SaCO_2} = 9.5 \cdot P_{CO_2}{}^{0.45} \tag{8}$$

where: SaO_2 and $SaCO_2$ — arterial saturations (in mmol/l) of O_2 and CO_2, respectively, P_{O_2} and P_{CO_2} — the partial pressures (in kPa) calculated with the formula (6). The above formulas fit the dependences between the pressures and saturations very good**.**

It has been assumed that saturation of N_2 and an anesthetic is proportional to their partial pressures because they do not create chemical compounds with blood components.

The saturations calculated as above are the input of the CGT module (SaG_j in Fig. 4).

The quantum of a gas that flows from a lobe into the blood (F_{Gj} in Fig. 4) is proportional to the blood flow rate and the difference between arterial and venous saturations of this gas (SaG_j–SvG_j). $SaGj < SvG_j$ means inverse direction of F_{Gj} (e.g. CO_2 usually flows from the blood into the lungs because of $F_{CO_2} < 0$ unless the inspiratory concentration of CO_2, i.e. F_ICO_2, is significantly increased to avoid hypocapnia).

2.4. The CGT Module — Gas Transfer in the Cardio-vascular System

It is assumed that in the future, the CGT module will cooperate with a model of the cardiovascular system mechanics, as the AGT module cooperates with the lungs mechanics sub-model. In particular, the CGT module will utilize pulsatile blood flow supplied by such a sub-model, like the AGT module utilizes airflows supplied by the lungs mechanics sub-model. For now, however, it is assumed that the blood flow is constant with the cardiac output equal to 5 l/min for the standard patient.

Distribution of the blood flow between the lobes (i.e. perfusion of the lobes) depends on patient position. Since there is not any circulation mechanics model, differences in perfusion are treated as model parameters (ventilation of lobes also depends on the position, however, differences in ventilation of particular lobes are the result of mechanics sub-model simulation).

Blood from the lobes, saturated with the four gases (the saturations are supplied by GE module — SaG_j in Fig. 4), is mixed in the left heart, where the final arterial blood saturations are determined. Such homogenously saturated blood flows through aorta and arteries to capillaries in tissues, where O_2 is consumed with the constant rate equal to 0.25 l/min for the standard patient (metabolism in Fig. 4).

CO_2 production depends on respiratory quotient ($RQ = CO_{2\,produced}/O_{2\,consumed}$). RQ depends on fuel source that is metabolized, e.g. RQ=1 for glucose while for a saturated fat $RQ = 0.667$. Only small part of produced CO_2 (about 10% [8]) is transferred directly to the blood. Although the rest of CO_2 is stored in the various CO_2 buffers of different properties, it is assumed a single resultant buffer in the CGT module (Fig. 3) because of problems with accurate data collection. The rate of CO_2 transfer from the buffer to the blood is proportional to the difference between CO_2 tensions in the buffer and the blood. Under the steady-state conditions, the CO_2 amount that is transferred to buffers is equal to the CO_2 amount that is transferred from the buffers to the blood.

The blood with decreased O_2 saturation and increased CO_2 saturation, i.e. the venous blood, flows through several vein segments to the right heart, then to the lobes. The saturations of the venous blood that flows through the lobes are the input of the GE module (SvG_j in Fig. 4).

3. Results

A model of an organ may be treated as a virtual organ if its behavior is proper in different situations, even unusual ones. For example, the mechanics sub-model could be treated as a stand-alone virtual respiratory system because it was verified successfully [5] with spirometry dependent on the most sophisticated properties of the respiratory system. Accuracy of the gas transfer and exchange sub-model has been examined in a similar way, i.e. results of simulation of interesting situations were compared with data known from physiology.

3.1. Partial Pressures

Figure 5 presents exemplary courses of partial pressures of O_2 and CO_2 in the lungs (P_AO_2, P_ACO_2) and in the end of an intratracheal tube during artificial volume controlled ventilation with minute ventilation equal to 10 l/min of saturated air with F_IO_2=19.8%. As it could be expected, P_AO_2 was greater and P_ACO_2 was smaller than

the norm (should be: $P_AO_2 \approx 13.3$ kPa, $P_ACO_2 \approx 5.4$ kPa) because such ventilation (10 l/min, $F_IO_2 = 19.8\%$) is too intensive for the standard human being at rest.

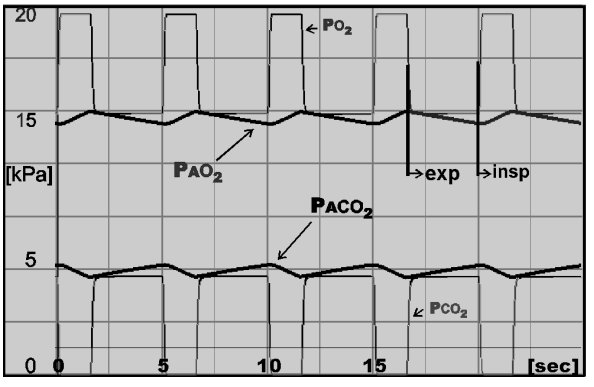

Fig. 5. Courses of O_2 and CO_2 partial pressures: in the lungs (P_AO_2, P_ACO_2) and in the intratracheal tube end (PO_2, PCO_2) during volume controlled ventilation with saturated atmospheric air (minute ventilation equal to 10 l/min)

3.2. Respiratory Arrest

Changes of venous CO_2 tension (PCO_2) and arterial oxygen saturation expressed as percentage of oxygenated hemoglobin (HbO_2%) after respiratory arrest were analyzed for the four following cases:

I. Airways closure caused respiratory arrest and F_IO_2, i.e. O_2 concentration in the inspired air (during respiration) and in the ambient air (after respiratory arrest), was equal to 20% as it is in the normal saturated atmospheric air;

II. Airways remained open after respiratory arrest and F_IO_2= 20%;

III. Airways closure caused respiratory arrest and F_IO_2 was equal to 100% (respiration with pure oxygen);

IV. Airways remained open after respiratory arrest and F_IO_2=100%.

The case I corresponds to the respiratory arrest during everyday breathing that is caused by some obturation or is intentional, e.g. during diving. As it occurs usually in the real world, arterial blood oxygenation fell below the safety level ($HbO_2\% \approx 70\%$ [8]) after about 1.5 minute. When airways remained open (the case II) and the fresh air might contact with the lungs, there was only about 15 sec more. For both cases, PCO_2 did not increase so quickly as HbO_2 fell because of the CO_2 buffer (the greater PCO_2, the smaller the rate of CO_2 transfer from the buffer to the blood).

The respiratory arrest, as in the case III, may occur when obturation appears during ventilation of a seriously ill patient with the pure oxygen. As in the case of real patients [9], HbO_2% did not fall significantly during 10 minutes, however, the lungs volume fell, which led to atelectasis (Fig. 6). It was because of staying of P_AO_2 at almost constant level and the blood could be oxygenated all the time. Since

oxygen passed from the lungs to the blood all the time and the volumes of the gases in the lungs could be supplemented: (a) neither with ambient oxygen because of airways obturation, (b) nor with significant amount of CO_2 from blood because of the CO_2 buffer, the volume of the lungs had to decrease. However, if the airways were open, as in the case IV, the vacuum that was caused by oxygen passage to the blood induced O_2 flow through the airways. In the consequence (Fig. 6): (a) the lungs volume remained constant, (b) HbO_2% also remained at almost constant level for hours [8]. Such a patient would be alive without respiration for a long time as long as CO_2 might be removed from the blood. Mottaghy et al. [10] proposed such ventilatory support with extracorporeal CO2-removal. It would be especially useful in the case of patients with hurt chest or lungs when movement of the chest during conventional respiration is not possible or undesirable [11].

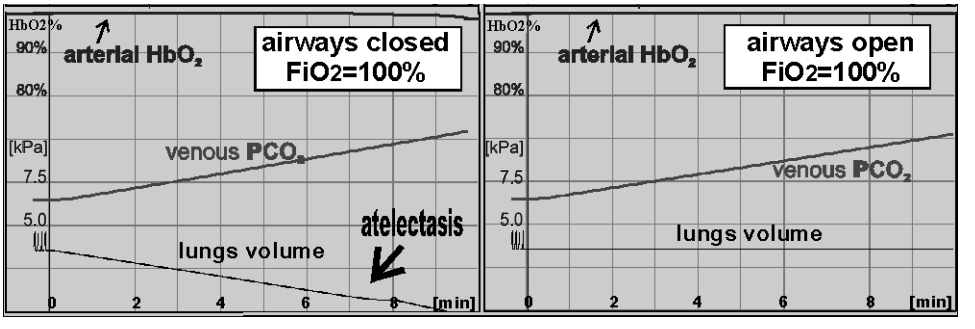

Fig. 6. Respiratory arrest. Changes of volume of lungs, arterial oxygen saturation expressed with HbO_2%, and carbon dioxide tension in venous blood (P_{CO_2}) after respiratory arrest when the airways are open/closed and oxygen concentration in the inspired air (FiO_2) is equal to 100% (pure oxygen). Note: the lungs collapse (atelectasis) when the airways are closed

3.3. Changes of Ventilation Intensity

It is well known that if ventilation increases then arterial PCO_2 decreases. It is comprehensible since the greater ventilation means the better CO_2 removal from lungs causing a decrease of P_ACO_2 (arterial PCO_2 is usually equal to P_ACO_2 — the assumption of GE module). Venous PCO_2 falls slower because CO_2 in venous blood comes

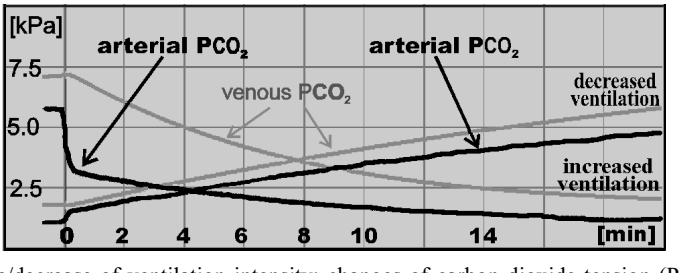

Fig. 7. Increase/decrease of ventilation intensity: changes of carbon dioxide tension (P_{CO_2}) in arterial and venous blood after increase of low ventilation and decrease of intensive one

mainly from the CO_2 buffer, which keeps higher level of CO_2 tension for some time (it empties slowly because of big capacity). It is less comprehensible but known [8] that if ventilation decreases then both arterial and venous PCO_2 increases very slowly. The same result was obtained for the model (Fig. 7).

4. Conclusions

The respiratory system mechanics sub-model was verified previously [5]. Since such phenomena as atelectasis or long-term high blood oxygenation during respiratory arrest have appeared, the gas exchange and transfer sub-model that is the subject of this paper seems to be accurate, too. Therefore, the whole virtual cardio-respiratory system seems to be accurate. It could be useful in analysis of such problems as, for example, whether an increase of ventilation or oxygen concentration in inspired air is more effective for blood oxygenation and decarbonation in a particular case of an artificially ventilated patient.

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