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Modelling and Simulation in Optimization of High-frequency Oscillatory Ventilation

Modelování a simulace při optimalizaci vysokofrekvenční oscilační ventilace

Summary

High-frequency oscillatory ventilation is a non-conventional ventilatory regimen using small tidal volumes and high ventilatory frequencies. Animal experiment was conducted where lung lavage was used simulating acute respiratory distress syndrome. Proximal pressure and flow were recorded and analysed for studying the tidal volume delivery and alveolar pressure in experimental phases before and after lung lavage. It was shown that tidal volume even increases after lung lavage and alveolar pressure amplitude also increases with reduction of lung compliance after lung lavage.

Souhrn

Vysokofrekvenční oscilační ventilace je nekonvenční ventilační režim, který využívá malé dechové objemy a vysoké ventilační frekvence. Byl realizován animální experiment, při kterém byl plicní laváží simulován akutní syndrom dechové tísně. V průběhu experimentu byl nahráván proximální tlak a průtok, ze kterých byl analyzován dechový objem a alveolární tlak pro experimentální fázi před a po laváži. Z analýzy je zřejmé, že dechový objem se zvýšil po laváži plic a amplituda alveolárního tlaku se rovněž zvýšila při snížené poddajnosti plic po laváži.

Key Words

high-frequency oscillatory ventilation, tidal volume, alveolar pressure, protective ventilation, ARDS

Klíčová slova

vysokofrekvenční oscilační ventilace, dechový objem, alveolární tlak, protektivní ventilace, ARDS

Content

List of abbreviations

ARDS	Acute respiratory distress syndrome
CDP	Continuous distending pressure
COPD	Chronic obstructive pulmonary disease
HFV	High-frequency ventilation
HFOV	High-frequency oscillatory ventilation
MV	Mechanical ventilation
VILI	Ventilator induced lung injury

List of symbols

C_L	Lung compliance	L/cmH_2O
ΔP	Pressure oscillations	cmH_2O
f	Ventilatory frequency	Hz
L_{aw}	Airway inertance	$cmH_2O/L/s^2$
p_aO_2	Partial pressure of oxygen in arterial blood	mmHg
p_aCO_2	Partial pressure of carbon dioxide in arterial blood	mmHg
pH	pH of blood	-
R_{aw}	Airway resistance	$cmH_2O/L/s$
V_t	Tidal volume	mL

1. Introduction

Spontaneous breathing ensures oxygen delivery for a body and removal of carbon dioxide out of the body. When one breathes spontaneously, there is a negative pressure in the lungs during the inspiration supporting a circulation of blood by lung capillaries. Expiration is a passive part of breathing and a pulmonary mechanics determines character of flow.

Mechanical ventilation (MV) has to be used in patients with inadequate spontaneous breathing as a result of a lung disease or intentional inhibition. Conventional regimens of MV can be characterized by tidal volume and breathing frequency that are similar to spontaneous breathing. We can distinguish MV with positive and negative pressure during inspiration but most of the regimens use positive pressure during inspiration conversely to spontaneous breathing. Positive ventilatory pressure may adversely affect the blood circulation in the lungs.

Tidal volume depends on the ventilatory pressure and on the pulmonary mechanics; mainly airflow resistance and compliance of the respiratory system. It was shown in some clinical studies that mechanical properties of the respiratory system significantly affect ventilatory parameters and therefore they affect the efficiency of MV [1, 2, 3]. Airflow resistance and lung or chest wall compliance are changing during the pulmonary diseases such as acute respiratory distress syndrome (ARDS), chronic obstructive pulmonary disease (COPD), asthma etc. These diseases directly impact the efficiency of MV.

There are still strong adverse effects upon the patient's respiratory system during MV even though MV is investigated properly for a decades. The mortality of artificially ventilated patients remains still very high [4]. Adverse effects are generally described as ventilator induced lung injury (VILI). Barotrauma or volutrauma are main causes of VILI. Protective ventilation was introduced in order to minimize a lung injury during MV [5]. These regimens are characterized mainly by decreased tidal volume that should be in a range 6–8 mL/kg of body weight contrary to the previously used 8–10 mL/kg. Lower tidal volumes allow use of smaller ventilatory

pressures thus minimizing the volutrauma or barotrauma incidence. It may result in lower level of oxygen in arterial blood and higher level of carbon dioxide however the values of blood gases outside the recommended values are tolerated.

Some alternative ventilatory regimens were introduced to improve the patient's benefits from MV usage. These regimens are generally called nonconventional ventilatory modes. High-frequency ventilation (HFV) belongs to the nonconventional ventilatory regimens. Small tidal volumes compared with MV are used in HFV together with higher ventilatory frequencies. Tidal volume is similar to the anatomical dead space of the lungs and corresponds to a range 1–3 mL/kg and ventilatory frequency is from a range 3–15 Hz in high-frequency oscillatory ventilation (HFOV).

Continuous distending pressure (CDP) inflates the lung in HFOV and pressure oscillations ΔP are superimposed to CDP. The oscillations deliver the tidal volume into a patient. It is generally accepted that amplitude of the oscillations is attenuated in the lungs [6, 7]. Usage of the small pressure amplitudes in the lungs and breathing with the very low tidal volumes fulfils the main criteria of protective ventilator regimen. HFOV belongs to pressure controlled ventilatory regimens. Generally, CDP affects arterial oxygen tension PaO_2, whereas pressure oscillations ΔP and ventilatory frequency f affect arterial tension of carbon dioxide $PaCO_2$.

The monitoring of the ventilatory parameters is very limited in HFOV. Most common high-frequency oscillatory ventilator is 3100B (Carefusion, USA). In 3100B ventilator, CDP, pressure oscillations ΔP, time of inspiration and ventilatory frequency are set at the ventilator. No other parameters are commonly set or monitored during HFOV. Ventilator does not provide any information about the tidal volume V_t. Respiratory monitor Florian (Acutronic Medical, Switzerland) allows measuring of tidal volume in HFOV however the monitor is not approved for usage in USA. Furthermore, the production of the monitor was stopped in the past and there is no commercially available method for measuring of tidal volume in HFOV.

There is a group of patients that benefits from HFOV usage, mainly the patients with severe ARDS [3]. However the exact

description of target group of patients that should benefit from HFOV usage is not still exactly specified and there is a few studies describing successful use of HFOV in COPD and asthma patients [8, 9] despite a fact that increased airway resistance is generally considered as a contraindication to HFOV usage [10]. Lack of knowledge about the intrapulmonary parameters in HFOV is one of the reasons of this inconsistency.

Use of the models of the respiratory system is only the possibility how to gain a new knowledge in patient ventilated by HFOV. A design of the novel model of the respiratory system has to correspond with the future model usage. Ventilatory parameters in HFOV as oscillatory pressure amplitude and ventilatory frequency significantly differ from conventional MV and the models designed for MV have limited usage for studying HFOV. There is a higher velocity of the gas resulting in different character of the flow and different principle of gas mixing in the lungs. Considering these factors, unique models has to be implemented that can be used for simulation of HFOV.

2. Modelling and simulation in the respiratory care

MV is a method often used in the clinical practice. In cases where MV fails HFOV may be used as a rescue technique. There is a lot of effects that are observed in the clinical practice in mechanically ventilated patients and many of these effects are not still satisfactory explained. Use of the models of the respiratory system can contribute to better under-standing about these effects.

The real human lung structure is very complex to be modelled physically or described mathematically, therefore, some simplifications should be applied. The reasonable balance between the complexity of the model and its accuracy has to be established. Generally, mathematical, laboratory and experimental models based on living organisms can be applied to study MV.

2.1 Experimental Model of Acute Respiratory Distress Syndrome

An animal experiment was realized to develop and study an experimental model of ARDS. HFOV was used to ventilate the animal before and after ARDS induction to confirm some effects observed in the clinical practice and some results received from mathematical simulations.

The study was approved by the Institutional Animal Care and Use Committee of the First Faculty of Medicine, Charles University in Prague, on March 27, 2013. The study was performed in an accredited animal laboratory in accordance with Act No. 246/1992 Coll., on the protection of animals against cruelty.

Crossbred Landrace female pigs (Suss crofa domestica) with an average body weight of 48 kg were used in this study. Premedication of the animals was done by azaperone (2 mg/kg IM). The pigs were anaesthetized with atropine sulphate (0.02 mg/kg IM) and ketamine hydrochloride (20 mg/kg IM) and by initial boluses of morphine (0.1 mg/kg IV) and propofol (2 mg/kg IV). Animals were placed in supine position on a heated pad; body temperature was kept in the normal range (38–39 °C). Intubation of the animals was realized with a cuffed endotracheal tube (I.D. 7.5 mm) and Hamilton G5 ventilator (Hamilton Medical, Bonaduz, Switzerland) was used for MV. The animals received continuous infusion of propofol (8 to 10

mg/kg/h IV) combined with morphine (0.1 mg/kg/h IV) and heparin (40 U/kg/h IV) to maintain the anaesthesia. Boluses of 4 mg of myorelaxant pipecuronium bromide were administered every 45 min to supress spontaneous breathing during MV. Initial rapid infusion of 1 000 mL of normal saline was given intravenously, followed by a continuous IV drip of 250 mL/h to reach and maintain central venous pressure of 6 to 7 mmHg.

A cannulation of vein and arteria was performed to continuously measure central venous pressure and arterial blood pressure. Vigilance monitor was used for continuous measurement of cardiac output, mixed venous blood oxygen saturation and pulmonary artery pressure. Partial tensions of arterial blood gases were continuously monitored and recorded. Concretely, partial pressure of oxygen (PaO_2), carbon dioxide ($PaCO_2$) and *pH* were monitored and recorded by CDI 500 (Terumo, Tokyo, Japan). A catheter for continuous measuring of pressure and volume (Transonic, USA) was placed into right ventricle. All signals were recorded synchronously using a LabChart system (ADInstruments, Oxford, UK).

Animals were switched to HFOV with SensorMedics 3100B ventilator. A special sensor for measuring of pressure and flow was placed between the patient circuit of the ventilator and an endotracheal tube. Proximal pressure and flow were continuously recorded by data acquisition card (National Instruments, Austin, USA) and laptop. Settings of the experiment performed in Charles University in Prague (Czech Republic) is depicted in Fig. 1.

Model of ARDS was realized by double or triple lung lavage with 30–40 mL/kg 37 °C normal saline containing nonionic surfactant Triton X-100 (0.05%) causing surfactant deficiency. Stabilization period of 1 h was done before the start of the experimental measurement.

Normocapnia was titrated by changing of amplitude of pressure oscillations ΔP at the start of the experiment. CDP was changed during the experiment in two phases; before and after lung lavage. Initial value of CDP 8 cmH_2O was used and it was stepwise increased every 10 minutes about 2 cmH_2O. When low values of CDP were not tolerated in ARDS pigs, CDP was rapidly increased to prevent further deterioration in severe hypoxia. After reaching

maximum value CDP was stepwise decreased by 2 cmH_2O to its initial value. The representative recorded proximal pressure is depicted in Fig. 2 for whole experiment.

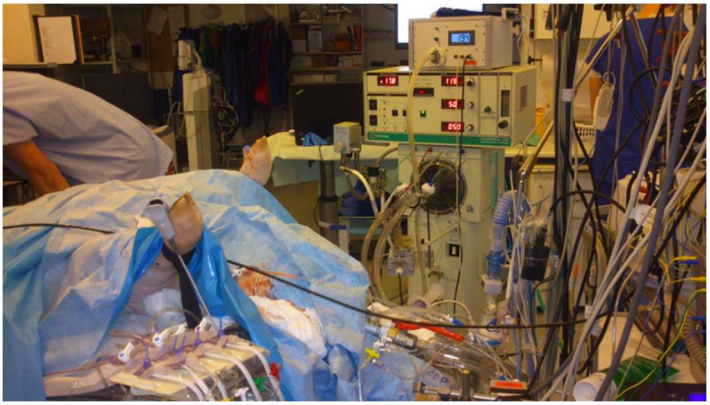

Fig. 1: Experimental settings in animal experiments.

Recorded signals of proximal pressure and proximal flow were used to identify a model of the respiratory system of the laboratory animal. The identification was realized for signals from experimental phases before and after lung lavage. Impedance of the model was computed for all frequencies contained in the measured signals. Modelled proximal flow was computed from measured proximal pressure and the impedance of the model in the Fourier domain. The optimization was done by minimizing the error function that was based on the sum of the absolute differences between the measured and modelled proximal flow.

Simple RLC model of the respiratory system was identified according to measured ventilatory signals. The model consists of resistance of the airways R_{aw}, inertance of the airways L_{aw} and lung compliance C_L.

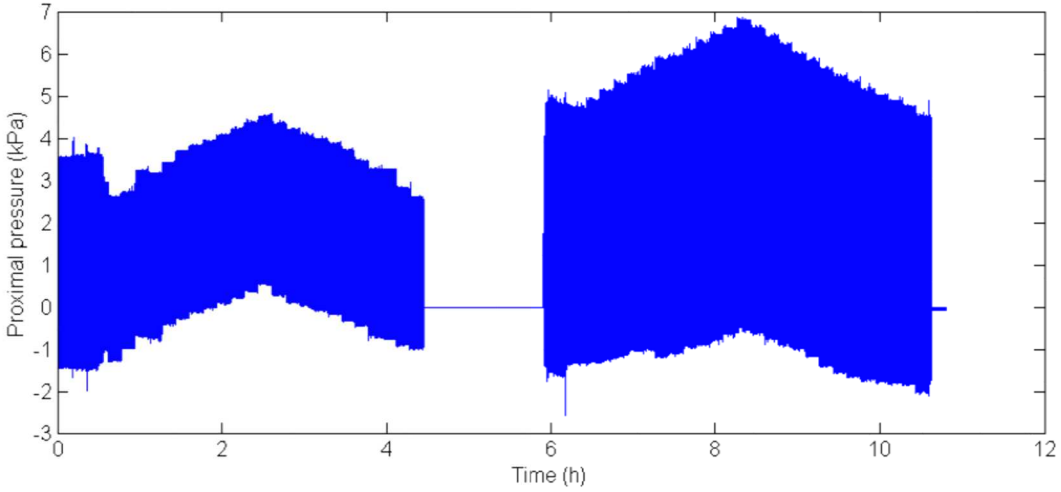

Fig. 2: Record of the proximal pressure in animal experiment before and after lung lavage of the laboratory animal.

3. Results of animal experiments

Time courses of proximal pressure and proximal flow were measured in laboratory pigs ventilated by HFOV and recorded during whole experiment.

Tidal volume was computed from measured proximal flow in two parts of the experiment; before and after lung lavage for different levels of CDP. Tidal volume was computed for the same levels of CDP for
9 series of measurement from 11 realized animal experiments. Two series of measured signals were excluded from processing because of significant artifact. Tidal volumes computed from the experiment are summarized in Tab. 1 for both parts of the experiment.

Tab. 1. Tidal volumes computed for laboratory animals in HFOV; before and after lung lavage simulating ARDS.

Series	Tidal volume V_t (mL)					
	before lung lavage			after lung lavage (ARDS)		
	CDP 12	CDP 18	CDP 24	CDP 18	CDP 24	CDP 30
1	145.1	152.9	149.7	176.9	173.0	172.4
2	147.5	155.2	150.9	178.4	182.4	180.3
3	172.2	180.9	176.5	190.5	196.8	202.0
4	149.1	162.4	163.9	156.0	168.7	175.0
5	165.3	175.0	175.9	164.8	175.0	177.7
6	132.4	141.1	141.4	176.8	174.6	172.4
7	116.2	130.8	137.6	183.3	187.8	195.4
8	157.7	158.6	156.7	165.9	173.8	177.2
9	130.8	135.8	140.8	196.8	199.6	201.2

Kolmogorov-Smirnov test was used and hypothesis about normal distribution of data was rejected. After that, Wilcoxon paired test was used to compare tidal volume among the groups with different level of CDP.

3.1 Effect of CDP upon tidal volume

Levels 12, 18 and 24 cmH_2O were chosen to study the effect of CDP upon the tidal volume in HFOV before lung lavage. The boxplot of data is depicted in Fig. 3.

The tidal volume statistically differs between the groups with CDP 12 and 18 cmH_2O ($p < 0.05$) and between groups with CDP 12 and 24 cmH_2O ($p < 0.05$). Statistical difference of tidal volumes was rejected in comparison of groups with CDP 18 and 24 cmH_2O.

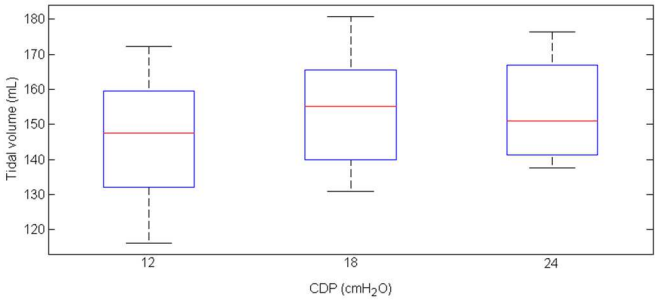

Fig. 3: Tidal volumes in HFOV before lung lavage for CDP levels 12, 18 and 24 cmH_2O.

Generally, higher CDP is used in patients with ARDS. Therefore, effect of CDP levels 18, 24 and 30 cmH_2O upon tidal volume was studied in laboratory animals after lung lavage modelling ARDS. The boxplot of data is depicted in Fig. 4.

The tidal volume differs between the groups with CDP 18 and 24 cmH_2O, between groups with CDP 18 and 30 cmH_2O and between groups with CDP 24 and 30 cmH_2O ($p < 0.05$) in HFOV after lung lavage.

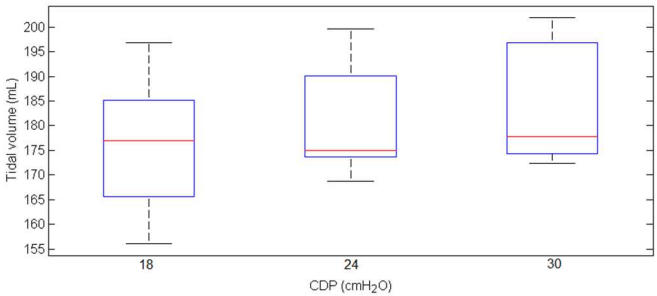

Fig. 4: Tidal volumes in HFOV after lung lavage for CDP levels 18, 24 and 30 cmH_2O.

3.2 Effect of ARDS upon tidal volume

Statistical difference ($p < 0.05$) was also confirmed when comparing the tidal volumes between the groups before and after lung lavage in level of CDP 18 and 24 cmH_2O. The boxplots of data for CDP 18 cmH_2O is depicted in Fig. 5 and for 24 cmH_2O in Fig. 6.

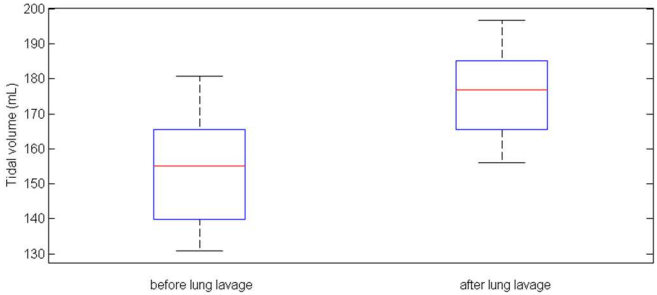

Fig. 5: Tidal volumes in laboratory animals before and after lung lavage for CDP 18 cmH_2O.

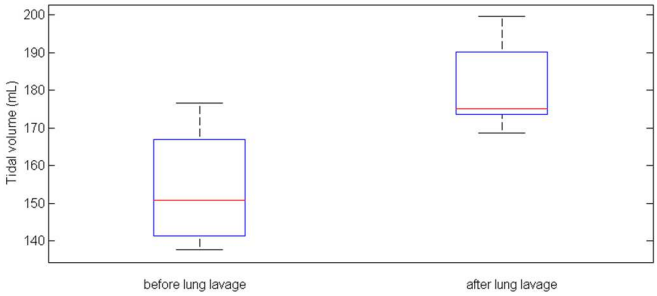

Fig. 6: Tidal volumes in laboratory animals before and after lung lavage for CDP 24 cmH_2O.

3.3 Identified model of the respiratory system

Proximal flow was computed from the measured proximal pressure and the impedance of the identified model. Basic RLC model was identified with signals from HFOV. The identification was processed for signals from one experimental measurement.

Following values of element were identified for HFOV before lung lavage:

$$R_{aw} = 10.5\ cmH_2O/L/s,$$
$$L_{aw} = 0.08\ cmH_2O/L/s^2,$$
$$C_L = 0.045\ L/cmH_2O.$$

The same model was identified with signals received in experiment part after lung lavage with following results:

$$R_{aw} = 12.8\ cmH_2O/L/s,$$
$$L_{aw} = 0.05\ cmH_2O/L/s^2,$$
$$C_L = 0.012\ L/cmH_2O.$$

4. Discussion

Tidal volumes in HFOV were studied in animal experiments in phases before and after lung lavage that was used for modelling of ARDS. The experiment was realized for different levels of CDP and results show that tidal volume statistically changes with CDP. This fact was observed in both groups; before and after lung lavage. We assume that lung compliance is changed with increasing CDP however it was earlier shown that tidal volume in HFOV does not change with a change of lung compliance. One can assume that higher CDP decreases airway resistance of smaller airways not containing cartilage rings in the structure and thus causing an increase of tidal volume.

Tidal volume was also significantly higher when comparing HFOV ventilation of the animals before and after lung lavage with the same level of CDP. Statistical difference was shown for CDP 18 cmH_2O and 24 cmH_2O. The increase of the tidal volume is caused probably by increased oscillations of alveolar pressure with reduced lung compliance as was showed in simulations with mathematical models based on the lung morphometry [11].

Identification of the model was realized according to proximal pressure and flow measured during the experiment. The identification was made with a few error functions and with a few weighting coefficients and exponents. The methods of least square error, minimizing maximum difference between measured and simulated value, with or without regarding the sign of difference were examined. Change of the functions, exponents and weights in error function had minimal effect on the results of identification. The airway resistance has substantial effect on the error function while effects of other parameters of the model were much lower. Another problem of identification may be caused by Fourier spectrum of the measured ventilatory signals that is very similar to the harmonic signal and it provides limited information for the process of identification.

It is probably a reason that model based on the lung morphology [12] that suppose harmonic signals of the ventilatory pressure and flow provides results that have been confirmed by laboratory measurement and also by animal experiment. It also suggests that main frequency of the ventilation in HFOV contains

most of the signal energy and results of conducted simulations sufficiently correspond to the reality.

The character of the flow is studied in [13, 14]. It was shown that the flow is laminar in normal condition. Turbulences may occur during obstruction of the central airways. If considering use of different gas mixture, i.e. heliox, it is necessary to consider different physical constants of the gas.

When comparing all results from measuring, modelling and simulation it is possible to say that even very simplified models can provide useful and reliable results. Another study should be made for explaining some effects in detail; for example the attenuation of the pressure oscillations in the alveolar space and its dependency on the pulmonary mechanics as observed in the simulations. The attenuation of the pressure oscillations is dependent on the lung compliance contrary to the tidal volume and the hypothesis is that it can be used for determining of CDP. The determining of optimal CDP is a challenge that may limit current use of HFOV.

It would be also useful to include endotracheal tube into the model when working with real measured signals from the animal experiments.

The identification of the models should be made with a few levels of CDP to study its effect upon the pulmonary mechanics.

5. Conclusion

The models provide useful results if they are designed properly. Even simple models can for example compute alveolar pressure in normal or diseased lung with changed pulmonary mechanics. It was shown that results from conducted simulations correspond with reality.

We have observed that tidal volume increases with CDP. Tidal volume was also higher comparing data from animal experiment before and after lung lavage that was used for modelling ARDS. The results of the model identification show that substantial decrease of lung compliance has no effect upon the tidal volume delivery.

The results of previously conducted simulations with mathematical models confirm that efficiency of HFOV is dependent on the pulmonary mechanics [11]. It was shown that tidal volume depends strongly on the airway resistance whereas reduction of lung compliance has almost no effect. It is in concordance with a fact that HFOV works close to the resonant frequency of the respiratory system.

Amplitude of the pressure oscillation is dependent on the lung compliance and it may be possible use ΔP for optimal choice of CDP.

It seems that HFOV is suitable for severe ARDS, whereas lung affected by obstructive disease is difficult to ventilate.

Literature

[1] Pelosi P, D'Onofrio D, Chiumello D, et al. *Pulmonary and extrapulmonary acute respiratory distress syndrome are different*. Eur Respir J 2003, 22:48s–56s.

[2] Gattinoni L, Pelosi P, Suter PM, et al. *Acute respiratory distress syndrome caused by pulmonary and extrapulmonary disease. Different syndromes?*. Am J Respir Crit Care Med 1998, 158:-11.

[3] Pachl J, Roubík K, Waldauf P, Fric M, Zábrodský V. Normocapnic *High-frequency oscillatory ventilation affects differently extrapulmonary and pulmonary forms of acute respiratory distress syndrome in adults.* Phys Res 2006, 55:15-24.

[4] Phua G J, Badia J R, Adhikari NK, et al. *Has mortality from acute respiratory distress syndrome decreased over time? A systematic review*. Am J Resp Crit Care Med 2009, 179:220-227.

[5] Ferguson ND, Frutos-Vivar F, Esteban A, et al. *Airway pressures, tidal volumes, and mortality in patients with acute respiratory distress syndrome*. Crit Care Med 2005, 33:21-30.

[6] Gerstmann DR, Fouke JM, Winter DC, et al. *Proximal, tracheal, and alveolar pressures during high-frequency oscillatory ventilation in a normal rabbit model*. Pediatr Res 1990, 28:367-73.

[7] Frantz ID 3rd, Close RH. *Alveolar pressure swings during high frequency ventilation in rabbits*. Pediatr Res 1985, 19:162-6.

[8] Frerichs I, Achtzehn U, Pechmann A, et al. *High-frequency oscillatory ventilation in patients with acute exacerbation of chronic obstructive pulmonary disease*. Journal of Critical Care 2012, 27:172-181.

[9] Duval EL, vanVught AJ. *Status asthmaticus treated by high-frequency oscillatory ventilation*. Pediatric Pulmonology 2000, 30:350-353.

[10] Fessler HE, Derdak S, Ferguson ND, et al. *A protocol for high-frequency oscillatory ventilation in adults: Results from a roundtable discussion*. Crit Care Med 2007, 35:1649-1654.

[11] Rožánek M, Roubík K. *Design of the Mathematical Model of the Respiratory System Using Electro-Acoustic Analogy*. International Journal of Biological and Life Sciences. 2009, vol. 4, no. 4, p. 232-235. ISSN 1307-7457.

[12] Horsfield K, Cumming G. *Morphology of the bronchial tree in man*. J. Appl. Physiol, 1968. 24: 373–383.

[13] Rožánek M, Roubík K. *Modelling of the Effect of the Gas Properties upon the efficiency of the Artificial Lung Ventilation*. In: Technical Computing Prague 2007, ISBN 978-80-7080-658-6.

[14] Rožánek M, Roubík K. *Use of Heliox during Spontaneous Ventilation: Model Study*. In: Proceedings of World Academy of Science, Engineering and Technology, 2008, pp. 460-462. ISSN 2070-3740.

Curriculum Vitae
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Degree M.Eng. (Ing.); Institution: Czech technical university in Prague, Faculty of Electrical Engineering, thesis: “Modelling of the effect of mechanical Ventilation upon the Pulmonary Perfusion”; 2002

Short-time course:

Vetram – Practical course of ventilatory physiology on an ARDS biomodel and its management, 2012.

Appointments:

Date:	2004 – present
Organization:	Czech Technical University in Prague, Faculty of biomedical engineering
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Position:	Lecturer

Date:	2006 – 2008
Organization:	Institute for Postgraduate Medical Education in Prague (IPVZ)
Position:	External lecturer

Research co-operation:

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Awards:

First price for the best poster presentation, conference Advanced engineering design 2004, Glasgow, Scotland.

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Professional Membership:

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Teaching Experience:

Czech Technical University in Prague, Faculty of biomedical engineering, since 2004 to present. Bachelor and Master subjects: Equipment for anaesthesiology and resuscitation, Respiratory therapy 1–2, Technique in Prehospital and Hospital Emergency Care, Physical Chemistry, Imaging Systems, Medical devices & equipment, Magnetic Resonance Imaging and Impedance Tomography, Devices in radiology, Ultrasound and Doppler Imaging Systems, Electronic Elements and Sensors in Medicine, Patient and Device Testers and Simulators, Intensive Care units and Mobile Care Units.

Charles University in Prague, First Faculty of Medicine, since 2004 to 2011. Subjects: Technical Aspects of Medical Devices 1–2.

Czech Technical University in Prague, Faculty of electrical Engineering, since 2002 to 2005. Bachelor and Maters subjects: Television technique, Biological signals.

Institute for Postgraduate Medical Education in Prague (IPVZ), 2006-2008. Education of subject in postgraduate programs: Biomedical engineer, Biomedical technician.

Invited Lectures:

Rožánek, M. Development of connection between the METI Simulator ECS and lung simulator ASL 5000. [Unpublished Lecture]. University of Medicine and Pharmacy "Grigore T. Popa" Iasi, Assoc.Prof. Radu Ciorap. June 02, 2012.

Rozanek M.: Electro-Acoustic Analogy and the Model of the Respiratory System. University Hospital, RWTH Aachen University, Pauwelsstraße 30, 52074 Aachen, Germany, November 19, 2010.

Rozanek M.: Electro-Acoustic Analogy and Its Use for Modelling of Biological Systems. Faculty of Electrical Engineering, "Gheorghe Asachi" Technical University of Iasi, Bd. Dimitrie Mangeron,67, 700050, Iasi, Romania, October 30, 2010.

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Deputy Editor-in-chief, The Clinician and technology Journal (in Czech: Lékař a technika). The official journal of Czech Society for Biomedical Engineering and Medical Informatics (2006-2008).

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Member, academic senate FBME, CTU in Prague (2004 – 2011).

Selected Publications:

Rožánek, M. – Hajný, O. – Čech, M. *Distribution of the ventilatory pressure in high-frequency oscillatory ventilation in the anatomically based model of the respiratory system*. Biomedizinische Technik / Biomedical Engineering. 2014, vol. 59, no. Suppl. 1, pp. S998-S1000. ISSN 0013-5585.

Kudrna, P. – Rožánek, M. – Hřibalová, B. *Flow measuring during neonatal high frequency jet ventilation using orifice plate*. Biomedizinische Technik / Biomedical Engineering. 2014, vol. 59, no. Suppl. 1, pp. S252-S254. ISSN 0013-5585.

Rožánek, M. - Horáková, Z. - Čadek, O. - Kučera, M. - Roubík, K. *Damping of the dynamic pressure amplitude in the ventilatory circuit during high-frequency oscillatory ventilation.* Biomedizinische Technik / Biomedical Engineering. 2012, vol. 57, no. Suppl. 1, p. 53-56. ISSN 0013-5585.

Rožánek, M. - Roubík, K. *Design of the Mathematical Model of the Respiratory System Using Electro-Acoustic Analogy.* International Journal of Mathematical, Computational, Physical, Electrical and Computer Engineering [online]. 2008, vol. 2, no. 11, p. 783-786.

Rožánek, M. - Roubík, K. *Mathematical Model of the Respiratory System - Comparison of the Total Lung Impedance in the Adult and Neonatal Lung.* International Journal of Medical, Health, Biomedical, Bioengineering and Pharmaceutical Engineering [online]. 2007, vol. 1, No. 6, p. 382-386.