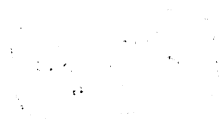


Interaction between Heart and Lung

Edited by Severin Daum

48 Figures, 36 Tables



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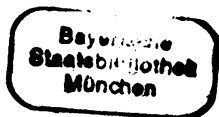
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Table of Contents

H. Denolin

Heart and Lung Interaction – Introduction	1
---	---

Mechanical Interaction between Heart and Lung

J. L. Robotham

Mechanical Heart-Lung Interaction	5
---	---

M. Mohr, H. Schulz, K.-D. Schuster, S. M. Lewis, M. Meyer, J. Piiper

Mechanisms of Cardiogenic Oscillations in Expired Gas	9
---	---

A. S. Menon, J. G. Abel, S. V. Lichtenstein, T. A. Salerno, A. S. Slutsky

Internal Cardiac Assist – A Novel Approaches	12
--	----

T. Podszus, T. Penzel, J. H. Peter, P. von Wichert

Blood Pressure Variation in the Pulmonary Circulation of Patients with Severe Obstructive Sleep Apnea	15
--	----

A. Rustici, R. Fogari, M. Grossoni, R. Tommasini, G. Ukmar, M. Morpurgo

Influence of Spontaneous Respiration on Right Ventricular Systolic Time Intervals and Pulmonary Arterial Pressures in the Dog	18
--	----

M. Eriksen

Stroke Volume Changes during the Respiratory Cycle Measured by Ultrasound Doppler Methods	25
--	----

J. Peters, M. K. Kindred, J. L. Robotham

Negative Intrathoracic Pressure during Diastole Distends the Aorta and Can Cause Retrograde Aortic Blood Flow	28
--	----

J. J. Koolen, A. C. A. Visser, E. Weber, N. G. Meyne, A. J. Dunming

Transesophageal Two Dimensional Echocardiographic Evolution of Biventricular Dynamics during Positive Endexpiratory Pressure Ventilation	30
---	----

H. Forst, B. Zwißler, J. Racenberg, M. Menger, K. Meßmer

“Paradoxic” Septal Motion during PEEP Ventilation	34
---	----

Humoral and Nervous Interaction between Heart and Lung*P. Harris*

Effects of Chronic Alveolar Hypoxia on the Myocardium 37

A. Hornykch

Humoral Interaction between Heart and Lung: Role of Eicosanoids 42

H. P. Koepchen

Nervous Interaction between Heart and Lung 46

*M. Kneussl, C. H. Chiang, P. Pappagianopoulos, B. Hoop, H. Kazemi*Central Regulation of Respiration, Cardiovascular Function and Metabolism:
The Role of Gamma-Aminobutyric Acid and Glutamic Acid 53*C. Miravalle, F. Galietti, G. E. Giorgis, G. M. Massaglia, S. Barberis, A. Ardizzi*Plasmatic Levels of TxB₂ and 6-Keto-PGF_{1α} in Thromboembolic Disease
of the Lung Treated with Fibrinolytic Drugs 57*H. Sinzinger*Prostaglandin (PG) E₁ in Heart and Lung 60*J. Herget, V. Hampl, F. Palecek*

Effect of Perinatal Hypoxia on Hypoxic Vascular Reactivity in Adult Rats 63

*S. V. Baudouin, N. T. Bateman*The Effect of Chronic Exposure to Hypoxia and Hypercapnia on the Contractile
Function of Rat Right Ventricular Papillary Muscle 67*V. Pelouch, B. Ošťádal, J. Procházka, D. Urbanová, J. Widimsky*Effect of High Altitude Hypoxia on the Protein Composition of the Right
Ventricular Myocardium in Young Rats 69*Ošťádal F. Kolár, V. Pelouch, J. Procházka, J. Widimsky*Spontaneous Reversibility and Pharmacological Reduction of Cardiopulmonary
Changes Induced by intermittent High Altitude Hypoxia in Young and
Adult Rats 72*J. Widimsky, B. Ošťádal, D. Urbanová, V. Pelouch, J. Procházka*Intermittent or Continuous Hyperoxia in the Treatment of Chronic Hypoxia –
Induced Cardiopulmonary Changes 75*C. A. Wagenvoort*

Interaction between Heart and Lung. Morphologic Aspects 77

The Heart in Lung Diseases

<i>A. Polonska, L. Polonski, J. Wodniecki, A. Krzywiecki, D. Zawila, A. Wycisk, M. Tendera</i> Cardiac Arrhythmias in Patients with Chronic Obstructive Pulmonary Disease	80
<i>I. Marozsán, L. Szatmáry, S. Kelemen</i> The Potential Damage of Ischaemic Heart Diseases in Asthma Bronchiale	82
<i>A. Bush, C. M. Busst, W. B. Knight, E. A. Shinebourne</i> Cardiopulmonary Interactions in Bronchopulmonary Dysplasia (BPD)	85
<i>R. J. Cusimano, K. A. Ashe, J. G. Abel, S. V. Lichtenstein, T. A. Salerno</i> The Cardiac Response to Pulmonary Hypertension	88
<i>J. E. Wolf, D. Fagret, B. Bertrand, J. Godart, M. Comet, J. Machecourt, B. Paramelle, B. Denis</i> Synchronous Measurements at Rest and during Exercise of the Right Ventricular Ejection Fraction (RVEF) with a 81m Krypton Perfusion, of the Pulmonary Pressure and Cardiac Output, in Chronic Obstructive Pulmonary Disease Patients (COPD)	90
<i>R. A. Incalzi, R. Pistelli, L. Fusco, C. L. Maini, M. G. Bonetti, R. Muzzolon, F. Gliozzi</i> Radionuclide Assessment of Left Ventricular Function in Stabilized Chronic Respiratory Failure	93
<i>B. Král, A. Hamet, D. Cernohorsky, J. Jandik, A. Štrasová, P. Tilšer, J. Eliáš, L. Vokrohlicky, V. Havel</i> Left Ventricular Function in Patients with Chronic Lung Disease – a Long Term Follow-up Study	96
<i>D. C. Flenley</i> Physiological Aspects of the Interactions between the Heart and Lungs	99

The Lung in Heart Disease

<i>M. Pistolesi, M. Miniati, M. R. Bonsignore, F. Andreotti, G. Di Ricco, C. Marini, M. Rindi, A. Biagini, E. N. C. Milne, C. Giuntini</i> Pulmonary Circulation in Patients with Chronic Left Heart Failure and Pulmonary Edema	102
<i>G. Rolla, C. Bucca, E. Caria, E. Scappaticci, S. Baldi</i> Bronchial Reactivity in Chronic Lung Congestion	105

<i>J. Kowalski, D. Brzostek, I. Hawrylkiewicz, W. Droszcz, J. Zielinski</i> Bronchial Hyperreactivity in Patients with Mitral Valve Disease	108
<i>M. Šamánek, A. Zapletal, J. Šulc</i> Disturbances of Lung Function in Transposition of the Great Arteries	111
<i>W. Petermann, J. Barth, P. Entzian</i> Influence of Severe Heart Failure on Lung Function	113
<i>F. Pelliccia, N. De Luca, F. del Monte, G. Spunticchia, M. Iamele, F. Dotta, A. Reale</i> Idiopathic Dilated Cardiomyopathy: Relationship between Heart and Lung Function	115
<i>N. De Luca, F. Pelliccia, G. Spunticchia, M. Iamele, M. Ciavolella, F. Dotta, A. Reale</i> Idiopathic Dilated Cardiomyopathy: Prognostic Relevance of Lung Function Test Data	118
<i>T. Allhoff, U. Sander, R. Müller, M. Meythaler, W. Rödl, J. Rein, B. Kunkel</i> Left Ventricular Function and Pulmonary Parameters in Various Forms of Heart Disease	120
<i>C. Tantucci, E. Boschetti, M. L. Dottorini, C. A. Sorbini, V. Grassi</i> Control of Breathing in Cardiac Patients: Pulmonary Function and Pattern of Breathing in Pulmonary Congestion and Edema before and after Recovery	123
<i>W. Schlick, M. Jung, A. Moritz, W. Klepetko, E. Wolner</i> Relation of Pulmonary Function, Hemodynamics and Clinical Symptoms in Patients with Valvular Heart Disease	127
<i>K. Andreopoulos, N. Margaris, G. Baltopoulos, P. Argirakopoulos, A. Papapaskali, E. Anagnostou, N. M. Siafakas</i> Serum Angiotensin Converting Enzyme (SACE) in Acute and Chronic Heart Failure	132
Heart and Lung in Special Situations (Surgery, Anaesthesiology, Extreme Situations)	
<i>K. Skarvan</i> Effects of Anesthesia on Heart and Lung	136
<i>K. W. Heinl, A. Hornyh, S. Daum, A. Steger</i> Effect of 100% Oxygen Breathing on Pulmonary Hemodynamic Parameters and Plasma Eicosanoids	140

<i>B. Šerf, M. Munclinger, J. Kofránek, M. Fusek, J. Kautzner, J. Hrudová, F. Boudik</i> Influence of Blood Pressure in Lesser Circulation on Ventilation-Perfusion Ratio in Supine Position	146
<i>I. Péntzes, T. Gondos, A. Bede</i> Changes of the Pulmonary Hemodynamic Parameters in ARDS	149
<i>P. Neidhart, P. M. Suter</i> How to Monitor Right Ventricular Function in Ventilated Patients?	152
<i>F. X. Schmid, S. Iversen, U. Hake, J. Thul, D. Duda, H. Oelert</i> Effect of Altered Ventilation Patterns during Cardiopulmonary Bypass on Hemodynamics, Pulmonary Gas Exchange and Extravascular Lung Water	155
<i>B. Reichart, H. Reichenspurner, D. K. C. Cooper, D. Novitzky, A. G. Rose, B. M. Kemkes, W. Klinner</i> Initial Experience in Heart-Lung Transplantation	160
<i>E. Fiehl, E. Hoefter, F. Krombach, H. Reichenspurner, G. König, B. Kemkes, G. Osterholzer, J. M. Gokel, C. Kugler, C. Hammer</i> Bronchoalveolar Lavage Following Combined Hetero-Orthotopic Heart-Lung Transplantation in the Dog	166
<i>J. M. Bishop</i> Outlook in the Evolution of Interaction between Heart and Lung	170

“Paradoxical” Septal Motion during PEEP Ventilation

H. Forst, B. Zwißler, J. Racenberg, M. Menger, K. Meßmer

Introduction

Mechanical ventilation with positive endexpiratory pressure (PEEP) often is associated with a fall in cardiac output and stroke volume. Not all the mechanisms leading to this serious side-effect are well understood. While the reduction in venous return resulting from the elevated intrathoracic pressure is widely accepted as the predominant causative factor, the role of a direct tamponade of the heart by the lungs (1), and of the effects of increased pulmonary vascular resistance compromising right ventricular systolic function (2) remain to be elucidated. Furthermore, the importance of direct mechanical interaction of both ventricles, mediated by the commonly shared interventricular septum and enhanced by the non-compliant pericardium, has not been clarified yet (3, 4).

For this reason a study was designed in dogs to answer the following questions:

1. Does PEEP ventilation influence the dynamic geometry of the left ventricle as a consequence of ventricular interference?
2. Does the expanding lung alter the diastolic configuration of the heart by direct mechanical interaction (cardiac “tamponade”)?

Material and Methods

The studies were performed in anesthetized dogs, using sonomicrometry and 2-dimensional transoesophageal echocardiography to assess the dynamic geometry of the heart. Pressures in both ventricles were measured by micromanometers, cardiac output was determined by the thermodilution technique. After a right-sided thoracotomy and after pericardiotomy a miniaturized piezoceramic transducer was inserted through the tract of a 16-gauge needle into the interventricular septum. Four corresponding transducers sutured to the epicardial surface of the right and left ventricular walls allowed continuous and simultaneous measurement of the septal-lateral (s.-l.) diameter of the right and left, and of the anterior-posterior (a.-p.) diameter of the left ventricle. Intrathoracic pressure was measured by a micromanometer fixed at the level of the right atrium on the pericardial surface. In all animals isovolemic hemodilution with dextran 60 was performed (mean het 29%) to obtain blood for volume expansion during PEEP. After closure of the pericardium and airtight closure of the chest the endexpiratory pressure was raised to 10, 15, and 20 cm H₂O and lowered to 10 and 0 H₂O respectively. PEEP levels 10, 20, and 0 were held for a 20 minutes period. To eliminate the well known effects of impaired venous return, the transmural right ventricular filling pressure (i.e. central venous pressure minus intrathoracic pressure) was kept constant by blood transfusion during the PEEP intervention. In a second series of experiments a short axis view of the heart was imaged by 2-D echocardiography prior and after induction of PEEP, with the probe inserted via the oesophagus and sutured

to the pericardium to avoid changes of the tomographic plane due to motions of the heart within the thorax.

Results

With stepwise increase of PEEP cardiac output (CO) and stroke volume (SV) decreased significantly (at PEEP 20: CO -21% and SV -31%) despite volume expansion (14 ml/kg body weight) and in presence of a constant transmural filling pressure. Thus, factors other than reduced preload have to be considered to explain the side effects of PEEP.

With increasing levels of PEEP right ventricular diameter increased (at PEEP 20: +6%), while left ventricular s.-l. diameter decreased concomitantly (-9%). The a.-p. diameter of the left ventricle remained unaffected. To demonstrate changes in ventricular dynamic geometry the ventricular pressure-diameter relationship was recorded at 2 ms intervals and pressure-diameter loops were constructed from single cardiac cycles. At control conditions the pressure-diameter loops of the s.-l. and the a.-p. axis of the left ventricle revealed a configuration, which is well known from pressure-volume loops (5).

During PEEP, however, the s.-l. loop was displaced to the left (indicating a decrease of the s.-l. axis) and revealed a marked deformation: the s.-l. diameter increased during early systole and decreased at the end of the isovolemic relaxation phase, thus suggesting "paradoxical" movement of the septum. The corresponding a.-p. loop did not show a comparable deformation: along this axis shortening during isovolemic contraction was even more pronounced with PEEP. From the pressure-diameter loops one can not differentiate conclusively, whether the rearrangement of dynamic geometry observed, was due to a paradoxical motion of the septum or of the left ventricular lateral wall. 2-D echocardiography verified diastolic flattening of the interventricular septum towards the left ventricle and abnormal systolic bulging of the septum, predominantly at the site of the right ventricular inflow tract when PEEP levels were greater than 10 cm H₂O.

Conclusion

These findings suggest that the shift of the interventricular septum to the left observed during diastole is caused by a dilatation of the right ventricle. There was no evidence for a tamponade of the left ventricle by the lung. Thus, PEEP induces a rearrangement of left ventricular dynamic geometry, characterized by a "paradoxical" motion of the septum, which could partly account for the hemodynamic side-effects of PEEP. Enthusiastic volume expansion during PEEP guided by pressure monitoring only, may aggravate these phenomena and result in a deterioration of cardiac performance.

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