THE INFLUENCE OF OSTEOPATHIC TREATMENT ON THE LUNG FUNCTION OF COPD PATIENTS

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PREFACE

Einen herzlichen Dank möchte ich meinem Bruder, Dr. Michael Balleitner, aussprechen, der die spirometrischen Messungen durchführte.

Dank auch an Dr. Gebhard Woisetschläger, der mir mit Rat bei der Strukturierung der Arbeit zur Seite stand, sowie die statistische Auswertung und Übersetzung durchführte.

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ABSTRACT

BACKGROUND: COPD is projected to be the third leading cause of death worldwide by 2020. In Salzburg, Austria, overall prevalence of COPD at stage I or higher is 26.1%. Aim of this study is to evaluate possible effects of osteopathic treatment on tidal volume of COPD patients.

METHODS: Spirometry data of COPD-patients are measured before and one month after two osteopathic treatments.

RESULTS: No statistical significant improvement of Tiffeneau indices and FEV1 values could be achieved by osteopathic treatment. Improvements are only slight and within the methodical error of spirometry. Nevertheless, improvements are higher in the test group than in the control group.

CONCLUSIONS: Main limitation of this study might be the low number of treatments. Osteopathic long-term studies result in a higher reduction of severity of COPD.

KEY WORDS: COPD, osteopathic treatment, lung, spirometry, FEV1, Tiffeneau index

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1. Introduction and Purpose of the Study

Chronic obstructive pulmonary disease (COPD) is the fourth most frequent life-threatening disease after cardiovascular diseases, cancer and cerebrovascular diseases in western industrial countries and it is projected to be the third leading cause of death worldwide by 2020 (Schirnhofer et al., 2007). The current cost of treating COPD is already extremely high. In the USA 24 billion dollars are spent annually on the diagnosis and treatment of 16 million patients (Rufino and Lapa e Silva, 2006).

Characteristic for COPD is a progressive and almost irreversible obstruction of the air passages, leading to a chronic inhibition of the expiratory breath flow.
Most common reasons are the active as well as passive inhalation of tobacco smoke, air pollution, noxic substances in the working environment and frequent respiratory infections.

During my more than five year praxis in osteopathic treatment in the Upper Austrian capital, I noticed, that prevalence of respiratory diseases seems to be above-average in Linz and its catchment areas.

According to my opinion, predominant reasons are the following:

- Several thousand people are employed in iron and steel industries and engineering in this region.
- Among them are many blue collar-workers.
- A high percentage of the population smokes.
- Still, there are severe problems with air pollution, especially with particulate matter. In spite of high investments in pollution control during the last decades, population is still struggling with health consequences of economic growth in the nineteensixties and -seventies, when environmental and health impacts were not sufficiently considered.

I want to find out, if an improvement of the state of health of COPD patients can be achieved by osteopathic treatment, basing on the main problems of COPD.
I searched osteopathic literature for this topic already before I started the master study, but found only few studies and literature. With this thesis, I want to contribute to knowledge about COPD and with regard to osteopathy, to evaluate possible effects of

osteopathic treatment on tidal volume of COPD patients from an objective point of view.

In detail, I want to evaluate, whether objective parameters (expiratory capacity in the first second) on COPD patients are positively influenced straight after a first osteopathic treatment and/or after a second one (approximately four weeks after the initial treatment).

Additionally, osteopathic connections shall be worked out, with special focus on:

- thorax
- the upper cervical spine and the base of the scull (vagus nerve)
- Th2 – Th5 (symphaticus)
- fixation of the cervical pleura
- the diaphragm

The basic question of my study is, if there is a change of the tidal volume of COPD patients directly after or one month after osteopathic treatment.

My hypothesis is:

Selected osteopathic treatments result in a significant improvement of the tidal volume. Particularly, an enhancement of the forced expiratory volume in the first second is achieved.

2. Basicals

2.1. Definition of COPD and Related Diseases

In this chapter I will present only definitions and short summaries about the diseases. More detailed aspects will be described in chapter 2.3.

2.1.1. COPD

According to the World Health Organisation (WHO, 2007), COPD is
"*…a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases. A diagnosis of COPD should be considered in any patient who has symptoms of cough, sputum production, or dyspnoea (difficult or laboured breathing), and/or a history of exposure to risk factors for the disease. The diagnosis is confirmed by spirometry which measures lung function and capacity. Where spirometry is unavailable, the diagnosis of COPD should be made using all available tools. Clinical symptoms and signs, such as abnormal shortness of breath and increased forced expiratory time, can be used to help with the diagnosis. A low peak flow is consistent with COPD, but may not be specific to COPD because it can be caused by other lung diseases and by poor performance during testing. Chronic cough and sputum production often precede the development of airflow limitation by many years, although not all individuals with cough and sputum production go on to develop COPD. "*

According to the Global Initiative for Chronic Obstructive Lung Disease (GOLD, 2006), the chronic airflow limitation characteristic of COPD is caused by a mixture of small airway disease (obstructive bronchiolitis) and parenchymal destruction (emphysema). Most patients suffer from both, emphysema and chronic bronchitis and according to the predominant symptoms, it is distinguished between "blue bloaters" (bronchitis) and "pink puffers" (emphysema) (Richling, 2006: 11).
In NLM (2006/2) chronic bronchitis is additionally named as a main COPD disorder.
In contrary to asthma, COPD is not fully reversible with a ΔFEV1<15% and progredient with frequent exacerbations (Richling, 2006: 24).

2.1.2. Pulmonary Emphysema

Definition

According to the United States National Library of Medicine of the National Institutes for Health (NLM, 2006/1), pulmonary emphysema is a lung disease involving damage to the air sacs (alveoli). There is progressive destruction of alveoli and the surrounding tissue that supports the alveoli. With more advanced disease, large air cysts develop where normal lung tissue used to be. Air is trapped in the lungs due to lack of supportive tissue which decreases oxygenation. The air sacs are unable to completely deflate, and are therefore unable to fill with fresh air to ensure adequate oxygen supply to the body.

Symptoms

Symptoms are shortness of breath, chronic cough with or without sputum production, wheezing, a decreased ability to exercise. Additional symptoms that may be associated with this disease include anxiety, unintentional weight loss, ankle, feet, and leg swelling and fatigue.

A physical examination may show wheezing, decreased breath sounds, or prolonged exhalation (exhalation takes more than twice as long as inspiration). The chest may be barrel-shaped. There may be signs of chronically insufficient oxygen levels in the blood.

Complications accompanying emphysema are recurrent respiratory infections, pulmonary hypertension, cor pulmonale (an enlargement and strain on the right side of the heart), erythrocytosis (increased red blood cell count) and death.

Conservative treatment of emphysema

Smoking cessation is the most important and effective treatment.
Medications used to improve breathing include bronchodilators (hand-held inhaler or nebulizer), diuretics, and corticosteroids.
Antibiotics may be prescribed when respiratory infections occur. Influenza (flu) vaccines and Pneumovax (pneumonia vaccine) are recommended for people with emphysema.
Low-flow oxygen can be used during exertion, continuously, or at night.
Pulmonary rehabilitation can improve exercise tolerance and quality of life in the short-term.
Lung transplantation is an option for patients with severe disease.

Carefully selected patients may be eligible for lung reduction surgery. This procedure removes the damaged portions of the lung, which allows the normal portions of the lung to expand more fully and take advantage of increased aeration. When successful, those who undergo the surgery report improvement in walking distance and quality of life.

The outcome is better for patients with less damage to the lung. Prognosis is largely determined by initial shortness of breath, exercise tolerance, and results from lung function tests (spirometry). Death may occur from respiratory failure, pneumonia, or other complications. (NLM, 2006/1)

2.1.3. Chronic Bronchitis/Bronchiolitis

Definition (NLM, 2006/2)
Chronic bronchitis is an inflammation of the main airways in the lungs that continues for a long period or keeps coming back. To be diagnosed with chronic bronchitis, the cough and excessive mucus production must have occurred for three months or more in at least two consecutive years and not be due to any other disease or condition.
The same applies for Bronchiolitis, which in difference to bronchitis, takes place in the bronchioles.

Symptoms

Symptoms are cough that produces mucus (sputum), which may be blood streaked, shortness of breath aggravated by exertion or mild activity, frequent respiratory infections that worsen symptoms, wheezing, fatigue, ankle, foot, and leg swelling that affects both sides and headaches.

Complications, accompanying chronic bronchitis are acute bronchitis, pneumonia, cor pulmonale, emphysema, respiratory failure and cardiac arrhythmia.

Conservative treatment of chronic bronchitis

There is no cure for chronic bronchitis. The goal of treatment is to relieve symptoms and prevent complications. It is crucial to quit smoking to prevent chronic bronchitis from getting worse. Any other respiratory irritants should be avoided.
Inhaled medications that dilate (widen) the airways and decrease inflammation may help reduce symptoms such as wheezing.
Antibiotics may be prescribed for infections as needed.

Corticosteroids may occasionally be used during flare-ups of wheezing or in people with severe bronchitis that does not respond to other treatments.
Physical exercise programs, breathing exercises, and patient education programs are all part of the overall treatment plan.
Oxygen therapy may be needed in severe cases.
In very severe cases, a lung transplant may be recommended.

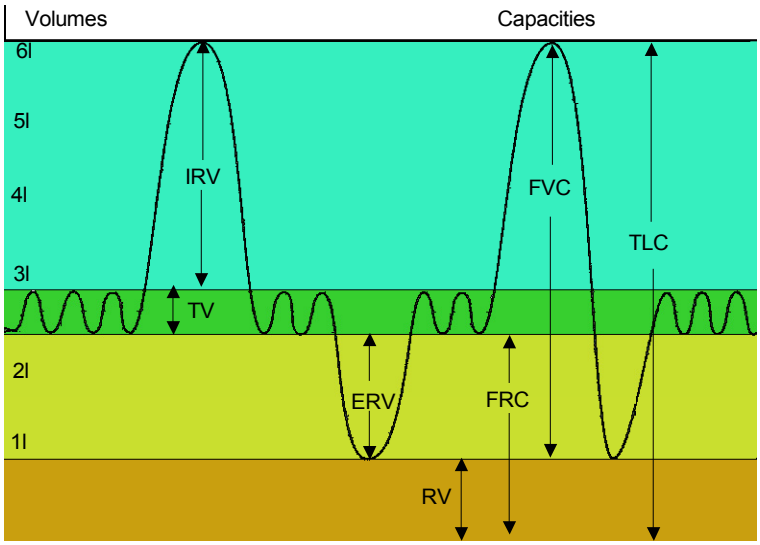
Mild or moderate cases of chronic bronchitis may often be controlled well with medicine and pulmonary rehabilitation. Advanced chronic bronchitis is more difficult to treat. Early diagnosis and treatment and stopping smoking significantly improve the odds of a good outcome (NLM, 2006/2).

2.2. Spirometry

Spirometry is the most common pulmonary function test (PFT) and is used for measuring the volume and flow of air that can be inhaled and exhaled.
Information gained by spirometry are:

- tidal volume (TV)
- inspiratory reserve volume (IRV)
- expiratory reserve volume (ERV)
- vital capacity (VC)
- forced expiratory volumes (FEVx)

cf. Ill. 1



Ill. 1: Lung volumina and capacities.

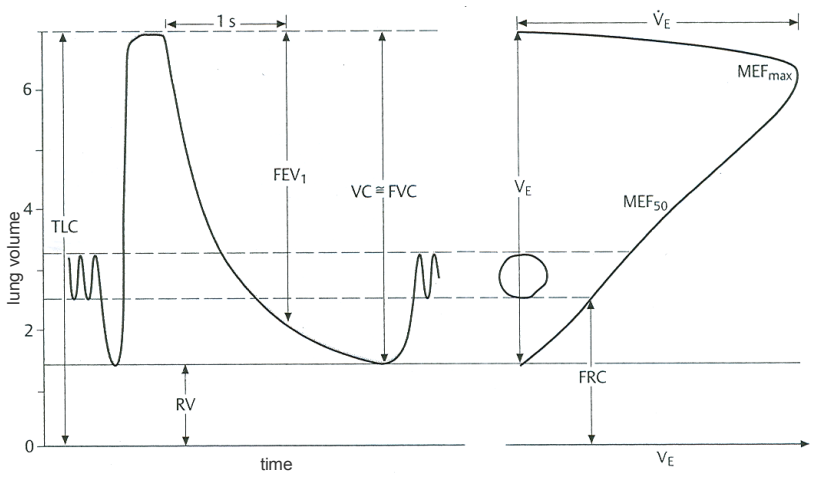
Vital capacity is the maximum amount of air moved in and out after of the lungs during a complete **forceful** inspiration. The tidal volume is the amount of air breathed in or out during **normal** respiration, Inspiratory reserve volume is the additional air that can be inhaled in addition to the tidal volume. The expiratory reserve volume is the maximal volume of air, that can be expelled from the lungs after normal expiration.
Forced expiratory volume is the volume of air that can be forced out in a distinct time after taking a deep breath. For example, FEV1 is the volume forced out in one second.
Spirometry is unsuitable for the measurement of total lung capacity (TLC) and residual volume (RV), and functional residual capacity of the lungs (FRC) can be only estimated. These air volumes (FRC, TLC and RV) can be measured via body plethysmography or gas (Helium) dilution technique.

Interpretation of spirograms

Dysfunctions in the respiratory system go ahead with typical changes in lung volumina and capacity. For this reason, spirometry data are interpreted in comparison with predicted values which are dependent on patient's height, age, sex, and sometimes race and weight.

Generally, it is recommended to use the Tiffeneau index (i.e. FEV1%/FVC) to decide whether there is or is not airway obstruction, and to quantify its severity on the basis of the FEV1 (American Thoracic Society (ATS, 1995), European Respiratory Society (Siafakas et al., 1995), British Thoracic Society (BTS, 1997)), but there is still discussion about the use of a fixed FEV1/FVC value (0.70) to define airway obstruction. According to Schermer and Quanjer (2007) evidence is emerging that this approach leads to an unacceptable percentage of false-positive diagnoses of mild and moderate COPD. Expressing FEV1 as a percent predicted value similarly introduces a bias: small people, elderly people, and especially small elderly people who are in good respiratory health will be incorrectly identified as having an abnormally low FEV1. Schermer and Quanjer propose the use of the lower limit of normal (LLN) for the FEV1/FVC ratio instead of a fixed ratio value of 0.70.

FEV1, the forced expiratory volume in one second (cf. Ill. 2) displays how many litres of air can be exhaled in one second. Obstructive dysfunctions (asthma, chronic bronchitis, emphysema) are characterised by an increase of static lung volumes. Narrow air passages result in a lower FEV1 value. Inspiration is not hindered to such an extent, therefore FEV1/VC relation is reduced (Gosselink, 2000: 53).



Ill. 2: Spirogram and flow-volume curve. MEF… maximum expiratory flow, MEF50… maximum expiratory flow at 50% of the vital capacity.

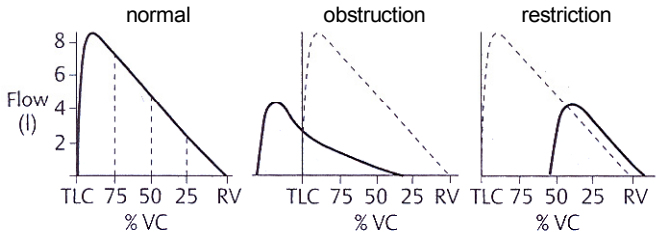
Classifications of the severity of COPD given by the Global Initiative for Chronic Obstructive Lung Disease (GOLD, 2006) are summarised in Table 1.

Stage I: Mild	FEV1 ≥ 80% predicted
Stage II: Moderate	50% ≤ FEV1 < 80% predicted
Stage III: Severe	30% ≤ FEV1 < 50% predicted
Stage IV: Very severe	FEV1 < 30% predicted plus chronic respiratory failure

Table 1: Spirometric classification of COPD. Severity based on post-BD FEV1.

Proportional changes of all lung volumes occur with **restrictive** pulmonary diseases. In these cases Tiffeneau indices are almost unchanged, too, because FEV1 and VC are reduced to a similar extent.

Typical deviations of the flow-volume curves of patients with obstructive and restrictive pulmonary diseases are shown in Ill. 3.



Ill. 3: Typical deviations of the flow-volume curve of patients with obstructive and restrictive pulmonary diseases.

For differential diagnosis of asthma versus COPD, the reversibility of a particular condition is tested by means of bronchiodilator tests ("Post-BD tests").
In this reversibility test a bronchodilator (BD) is administered before performing another test run for comparison. While asthma is a reversible airway obstruction and test values will normalise in the post-BD test, obstructions remain the same in COPD patients.

2.3. The Respiratory System

2.3.1. Embryology

A broad description of the embryological development of the lungs is given by Warburton and co-worker and will be quoted almost completely in the next two subchapters (Warburton et al., 2006: 668-672).

2.3.1.1 Embryological Development

The lung arises from the floor oft the primitive foregut as the laryngotracheal groove, at around 4- to 6-week gestation in humans. The proximal portion of this primitive structure gives rise to the larynx and trachea, which become separated from the esophagus, whereas progenitor cells located at the distal part of the primitive trachea give rise to the left and right main-stem bronchi.

Branching morphogenesis of the left and right bronchi to form specific lobar, segmental, and lobular branches extends through the canalicular stage of lung development up to about 20-wk gestation in humans. The first 16 of these 23 airway generations are stereospecific in humans, with the remainder being fractal in geometry, but with a distinct proximal-distal pattern of diameter and epithelial differentiation that are genetically hard-wired.

Alveolarization begins at about 20 wk in humans and continues at least up to 7 yr of age, giving rise to an alveolar gas diffusion surface 70 m^2 in area by 1 µm thick. This enormous surface is closely apposed to an alveolar capillary network capable of accommodating a blood flow between 5 l/min at rest and 25 l/min at maximal oxygen consumption. The entire developmental process of the lung is orchestrated by finely integrated and mutually regulated networks of transcriptional factors, growth factors, matrix components, and physical forces.

In utero, the lung is a hydraulic, fluid-filled system. Secretion of fluid into the airway lumen is osmotically driven by chloride channels. The larynx maintains an intraluminal hydraulic pressure of approximately 1.5 cm H_2O. Excess fluid drainage during fetal life results in hypoplasia of the lung. Conversely, obstruction of the trachea in embryonic lung in culture can result in a doubling of the rate of airway branching. Moreover, fluctuations in intraluminal pressure caused by coordinated peristaltic contractions of airway smooth muscle have recently been shown to play an important role in branching morphogenesis.

After cord clamping and a rush of catecholamines [adrenaline, dopamine, noradrenaline, …] *at birth, the lung lumen dries out and rapidly switches to air breathing. Clearance of lung intraluminal liquid is mediated by activation of sodium*

transport. Null mutation of sodium transporter channel genes (aEnaC) is neonatal lethal because it abrogates this fluid uptake. "Erection" of alveolar septa is relatively poorly understood. Nevertheless, correct organization of elastin matrix niche is important, as is remodelling of the alveolar capillary network. This suggests that vascular hydraulic perfusion pressure may play a key role in the emergence of septal structures into the alveolar space. This concept is further supported by a requirement for vascular endothelial growth factor secretion by the alveolar epithelium to maintain vascular integrity and hence correct epithelial branching as well as alveolar morphogenesis.

2.3.1.2 Adverse Impacts on the Developing Lung

Factors that adversely impact the developing lung, including human prematurity, oxygen exposure, corticosteroid exposure, incorrect amounts of growth factor (platelet-derived growth factor, fibroblast growth factor (FGF), vascular endothelial growth factor, transforming growth factor (TGF)-ß signalling, abnormal regulation, or injury of the pulmonary capillary vasculature, all result in hypoplasia of the alveolar epithelial surface, with a resulting deficiency in gas transport, particularly during exercise. For example, survivors of human prematurity with bronchopulmonary dysplasia will desaturate on maximal exercise during childhood, and some are now entering young adulthood with increasingly severe gas diffusion problems.

At the other end of the developmental spectrum, namely during aging, progressive involution of alveolar gas diffusion capacity occurs, especially over the last decades of life. This involution may be accelerated by exposure to adverse environmental factors, such as tobacco smoke, smog, industrial pollutants, toxic inhalants, infectious agents, and so forth. In industrialized societies where these risk factors are present, there is an epidemie of pulmonary failure due to chronic obstructive pulmonary disease (COPD), which is now the fourth leading cause of adult death in the United States.

COPD with emphysema has hitherto been considered to be an accelerated, involutional disease of aging smokers. However, because only a certain proportion (approx. 15%) of smokers develop COPD with emphysema, genetic susceptibility clearly must play a significant part. Recently, we have begun to wonder whether developmental issues may underlie at least some susceptibility to apparently adult-onset chronic lung disease. Certainly, the increasing numbers of survivors of human prematurity may be at risk. However, there may be other more subtle developmental genetic issues of concern.

In mice, interference with many key genes, either by null mutation, hypomorphism, or gain or loss of function, results in final common phenotypes comprising either neonatal lethal respiratory distress if the structural effect is severe, or reduced alveolarization and early-onset emphysema if the effect is milder. […] correct FGF signalling is absolutely required for lung morphogenesis distal to the carina, as well as for correct progress of all subsequent stages of lung development up to and including alveolarization.

In the lung, normal deposition and arrangement of elastin fibres is particularly important in the formation and maintenance of alveolar crests.
[…] failure to protect elastin from proteolytic degradation in acantitrypsin deficiency, or from excessive destruction of elastin, mediated by neutrophil elastase induced by chronic cigarette smoke exposure, results in the disease termed "emphysema," which is characterized by destruction of the alveolar walls. Elastic interdependence of the lung is an important concept in respiratory physiology, which accounts for orderly elastic recoil of the lungs during passive expiration. At the alveolar level, elastic interdependence is mediated by the correct expression, cross-linking, and orientation of elastin and collagen fibres. Thus, absence of correctly cross-linked and oriented elastin containing matrix predisposes to failure of correct establishment of elastic interdependence and alveolarization, whereas excessive degradation of elastin containing matrix underlies loss of elastic interdependence and alveolar degeneration. […] correct organization of the matrix during alveolarization may protect against subsequent proteolytic degradation and deterioration of the matrix, with subsequent loss of functional alveolar gas diffusion surface.

2.3.2. Basics of Anatomy and Physiology

Respiratory pump and lungs are a functional entity. The respiratory pump consists of the thoracic skeleton, respiratory muscles and the respiratory nervous system.
The respiratory pump of an adult ventilates approximately 10,000 litres of air via the bronchial system per day and keeps the large airways open by airway clearance.
Also for the arterialisation of oxygen in the lungs both, lungs and respiratory pump, are interdependent in their function. For example, during enhanced physical work, when the body needs more oxygen, tidal volume and/or breathing frequency is elevated by an increased activation of the respiratory pump.

Gas exchange is the most important process of breathing. Oxygen is released into the blood from tidal air and carbon dioxide from venous blood is delivered outside. This process is called "external respiration".
"Internal respiration" is the transfer of oxygen from arterial blood to the cell and the emission of carbon dioxide from cell to blood.
This chapter is dealing with external respiration under special consideration of the structures affected by COPD and mainly bases on Gosselink (2000). Structures, which are not directly affected by COPD are only touched (like the bronchi) or omitted (like the bony thorax). Alterations of anatomical structures and physiological processes will be summarised in the last subchapter (chapter 2.3.4). Specific osteopathic considerations will be discussed in chapter 3.1.

2.3.2.1 Respiratory Muscles

The respiratory system can be considered as a low-pressure air pump, reducing the oxygen partial pressure of about 160 mm Hg in the atmosphere, down to about 4-20 mm Hg in the mitochondrion (Anaesthetist, 2006) by ventilation (the transport of air into and out of the lungs and the transport to the alveoli via the airways), perfusion (the circulation of blood in the alveolar capilar membrane) and diffusion (the passage of oxygen from the air sacs of the lungs into the blood).
The lungs are connected with the thoracic cage by two pleura. The visceral pleura is covering the outside of the lung and the parietal pleura the inside of the thoracic cage. A slight vacuum between these two pleura (approx. 5 cm H_2O) is essential for the compensation of the opposed forces of the lung and the thoracic cage. The lung has an elastic inward tending force and the rib cage a force directed outwards (Gosselink, 2000).

The thoracic cage is expanded by the contraction of the respiratory musculature (muscles, their function and innervations are summarised in Table 2).
By this, lungs increase in volume and thus air pressure decreases inside and air flows in. During inspiration sagittal, longitudinal and transversal diameters of thorax and abdomen are increased simultaneously.

Muscle (group)	Innervation	Function
diaphragma	phrenic nerve (C3-5) intercostal nerves (Th6-12)	insp.
scalene muscles	cervical plexus (C2-8)	insp.
intercostal muscles	intercostals nerves (Th1-11)	parasternal: insp. interossal: stab.
levator costalis	intercostals nerves (Th1-11)	insp.
rectus abdominis	rami ventrales (Th 5-12)	exp./$insp_{acc}$
transversus abdominis	ilioinguinal nerve	exp./$insp_{acc}$
internal oblique muscle of abdomen	iliohypogastric nerve	
external oblique muscle of abdomen		
quadratus lumborum	lumbar plexus (Th12, L1-3)	exp_{acc}
erector spinae	rami dorsales (Th 1-12, L1-5)	$insp_{acc}$. + stab.
latissimus dorsi	thoracodorsal nerve (C7-8)	exp_{acc}
descending trapezius muscle	accessory nerve	$insp_{acc}$
serratus posterior muscle	rami ventrales (Th2-5)	superior: exp_{acc} inferior: $insp_{acc}$
serratus anterior muscle	long thoracic nerve (C5-7)	$insp_{acc}$
transversus thoracis muscle	intercostals nerves (Th4-7)	exp_{acc}
sternocleidomastoid muscle	accessory nerve	$insp_{acc}$
pectoralis muscles	pectoral nerves (C5, Th1)	exp_{acc}

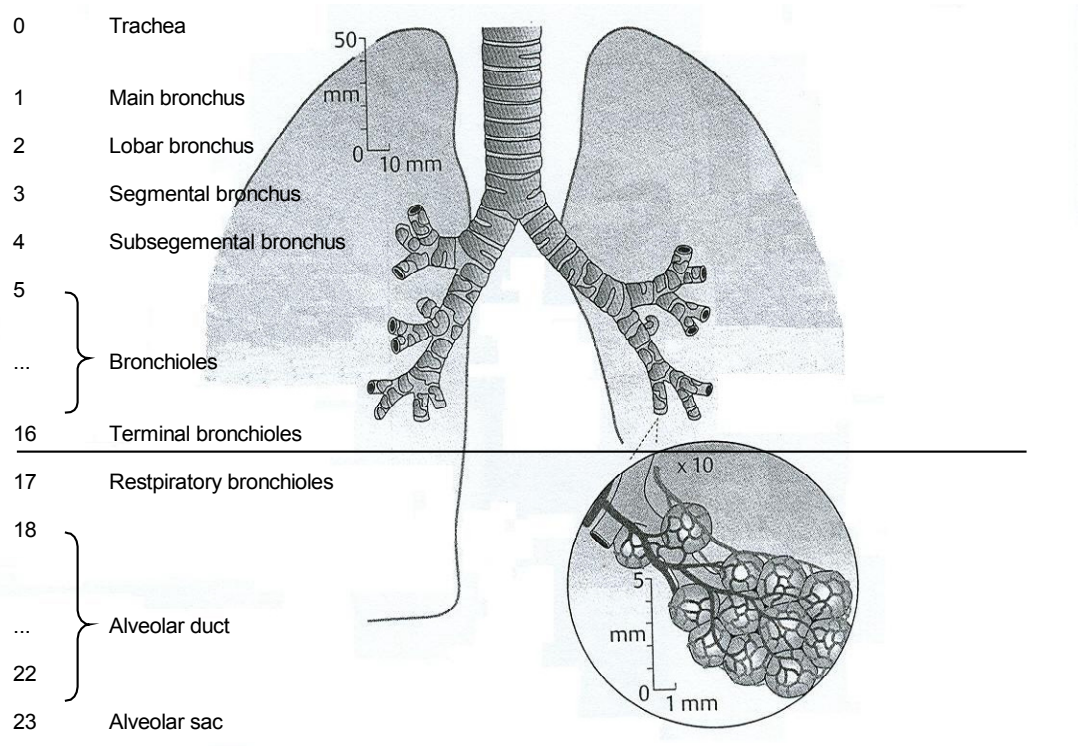
Table 2: Respiratory muscles, their innervation and function. Abbreviations: insp…. inspiratory, exsp… expiratory (Index: acc. … accessory respiratory muscle), stab… stabilising.

The potential energy stored in the lung during expansion (inspiration) is released as kinetic energy when the inspiratory muscles relax. Thus, after relaxation of the muscles, thorax is - almost passively - brought into pre-inspiration position again by the elastic recoil of the lungs. That means, that almost all of the respiratory work is performed during inspiration.

Function of respiratory musculature is dependent on the respiratory centre (neurons in the medulla oblongata), spinal nerves, peripheral nerves, neuromuscular components and the muscle fibres.
Additionally, respiratory movements are depending on the integrity of the trunk, body posture and the mechanic qualities (mobility, stability and flexibility) of the lungs (Gosselink, 2000).

2.3.2.2 The Airways

The respiratory tract may be seen as a branching tree-like structure, with about seventeen levels of branching between the trachea and the respiratory bronchioles (cf. Ill. 4). As on average the number of branches doubles at each level, we end up with about 130 000 (2^{17}) respiratory bronchioles.
The primary lobule is the zone supplied by one of these first order respiratory bronchioles. Each primary lobule contains about two thousand alveoli, and is about 3.5 mm in diameter. Branches within the primary lobule give rise to alveolar ducts, which in turn give off alveoli. All in all, there are about twenty-three intricate levels of branching within the respiratory tree (Anaesthetist, 2006).



Ill. 4: Branching levels of the airways.

The diameter of the central airways is approximately 1.8 cm and decreases to 0.05 cm in the peripheral airways (Gosselink, 2000).

2.3.2.3 Alveoli and Respiratory Bronchioles

While airways from the trachea to the terminal bronchioles have the main purpose to direct air (as well as to clean and to humidify it), gas exchange happens in the alveoli, which are supplied with air via the respiratory bronchioles. Off these open the respiratory exchange units, and not just at the end, but along the bronchiole. For efficiency, the branching, tubular architecture of air conductance overlaps slightly the honeycomb architecture of gas exchange (Beresford, 2007).

In the alveoli and alveolar sacs air passes over the capillary bed, where oxygen moves into the blood and CO_2 is removed from the blood. Air predominantly is distributed by molecular diffusion in this widespread area of approximately 50 - 100 m², since gas flow is very low. Gas concentrations only change little in the alveolar space and the gas mixture is homogenised by pulse (Gosselink, 2000).

2.3.2.4 Immune Reactions and Mucus Transport in the Lungs

Production and transport of entrapping mucus probably are the most important factors for the clearance of the airways. Additional to mucociliar clearance, some other mechanisms like alveolar clearance, immunologic and physiological reactions (coughing, sneezing, bronchiospasms) are of importance (Gosselink, 2000).

Mucus predominantly consists of water (95%), glycoproteins and other proteins (lactoferrine, immunoglobuline A and M), which are bactericidal and bacteriostatic. It is secreted by goblet cells and mixed glands, and is swept pharynx-wards by the ciliary beating action (Beresford, 2007)

For mucociliary transport, airways are covered with ciliated cells with approximately 200 cilia, which move coordinated three dimensional with a frequency of approx. 15 Hz. Fields of thin mucus on top of the cilia guard the epithelium cells against toxic gases and microorganisms. Additionally, they are responsible for humidity regulation of the airways and for the greasing of the epithelium (Gosselink, 2000).

If function of mucociliar transport is diminished or if mucus quantity is too high, another mechanism, two-phase gas-liquid flow (forced expiration and coughing) may be induced. If the expiratory airflow in the airways is high enough, it can move the film of mucus towards mouth. Airflow depends on the pressure loss in the airways (i.e. the difference of the pressure in the alveoli and the atmospheric pressure at the mouth). Alveolar pressure is the sum of the pleural pressure and the elastic recoil pressure of

the lungs and will be high at expiration and in large lungs. Additionally, diameters of the airways are influencing the velocity of the airflow.
In the alveoli a high pressure is built up at the beginning of expiration, air flows outside. Due to the higher diameters of the larger airways pressure there will decrease, but pleural pressure will remain the same.
If the difference of the bronchial pressure and pleural pressure exceeds the critical transmural pressure, pressure inside and outside the airways will be identical in one point ("equal pressure point" (EPP).
Downstream of this point a dynamic compression of the airway (flow limiting segment) will occur, where the velocity is increased and mucus can be moved. Localisation of the EPP is variable and is depending on the expiratory force, elastic retraction forces, and thus also lung elasticity and lung volume.
Due to the larger airway diameters and thus smaller pressure differences it can not be shifted further peripheral than the 4th generation of the airways.
In patients with emphysema, that means with instable airways, the EPP will be shifted to the smaller airways due to the loss of elasticity and a total closure decreases efficacy of coughing (Gosselink, 2000).

2.3.3. Histology

The histological structure of the airways is taken from Beresford (2007).
*The **trachea** is a flexible, extensible tube, with an always-patent lumen. Mucosa is mostly pseudostratified, columnar, ciliated epithelium with goblet cells, on a loose lamina propria rich in elastic fibres, mucous and mixed glands, leucocytes and sometimes lymphoid nodules.*
The cilia sweep towards the pharynx, but the elastic fibres run longitudinally as a layer between mucosa and submucosa.
Supporting C-shaped pieces of hyaline cartilage are incomplete on their esophageal side. The gap in the C is crossed by trachealis smooth muscle and connective tissue.
Outer adventitia is fibro-elastic connective tissue.

***Bronchi** resemble the trachea in structure, except that the cartilage pieces in the wall have very irregular shapes, and the smooth muscle forms a nearly complete layer - muscularis mucosae - between the cartilages and the lumen.*

Bronchioles *have no cartilages and their elastic fibres merge with those of the surrounding lung tissue.*
Their epithelium changes to simple, low ciliated columnar with a few goblet cells. In the lamina propria no mucous glands are present, where the smooth muscle is relatively substantial.
Sharing the connective tissue of the branching bronchi are blood vessels, nerves and lymphatic vessels, entering or leaving at the hilum or lung root.

Hilar structures *include arteries (bronchial and pulmonary), veins, lymphatics (from two systems), bronchi, lymph nodes, ganglia, nerves (to bronchial, bronchiolar, and vascular smooth muscles; and sensory), and adipose and other connective tissue. The carotid body-like glomus pulmonale in the pulmonary artery's adventitia is of uncertain function.*

Mucosa of the lower airway
Cell types in the epithelium:

- *ciliated columnar cells, with lysosomes and some microvilli*
- *mucus-secreting goblet cells*
- *basal 'undifferentiated' cells to replace the specialized kinds*
- *Clara's non-ciliated bronchiolar secretory cells with granules and granular endoplasmic reticulum (GER)*
- *neuroendocrine cells*
- *lymphocytes migrated from the lamina propria*

A sheet of sticky mucus is moved by ciliary action over the mucosa to catch and remove particles - the mucociliary escalator.
The basal lamina typically is thick.
Muco-serous mixed glands, where present in the lamina propria, are small, compound tubular, and respond under nervous control to irritant stimuli, e.g., smoke.

The Respiratory chambers
Respiratory bronchiole has simple, low columnar or cuboidal bronchiolar and ciliated cells; elastic fibres and smooth muscle support the epithelium's basal lamina. Opening out along the respiratory bronchiole are alveoli, whose openings are ringed by smooth muscle.

At the end of the respiratory bronchiole are one or more long alveolar ducts. Alveolar ducts can be viewed as being three to six atria, vestibules, leading to alveolar sacs, made up of varying numbers of alveoli.
One alveolus or cubicle shares an alveolar wall with the ones adjacent and backing on to it. The wall is thus interalveolar and carries the many capillaries, whose blood is to receive oxygen and give up carbon dioxide.

Interalveolar wall

- *Air side - continuous alveolar epithelium with:*
    - *type I pneumocytes/squamous cells and*
    - *pneumocytes type II/ septal or great alveolar cells, with prominent lipid cytosomes/ multilamellar bodies in their cytoplasm.*

Surfactant is a stabilizing fluid film of lipids (90%) and proteins (10%), covering the epithelium and lowering surface tension. The principal surface-active agent is the lipid, dipalmitoyl phosphatidylcholine (DPPC). The type II cells synthesize this film, but also are the stem cell to replace themselves and Type I cells. Cytosomes are stored surfactant. Surfactant comprises mainly lipids, with surfactant-associated glycoproteins SP-A, -B, -C, and -D, which variously cause the lamellar material to become a monolayer, enhance the lowering of surface tension, stabilise the lipids and counteract their oxidation, and modify host defences.

- *Alveolar macrophages/dust cells lie free in the alveoli.*
- *Alveolar epithelium lies on a basal lamina sometimes merging with, and sometimes separated from, the basal lamina of a blood capillary, on which lies an unfenestrated endothelium on the blood side. Where the two basal laminae are separated, the space - zona diffusa - is taken by elastic and reticular fibres, fibroblasts, macrophages and other connective tissue cells.*
  The pulmonary blood-air barrier can therefore be as thin as 300 nm, and has a very extensive area. Communication between adjacent alveolar sacs is through holes in the wall - alveolar pores.

Basal laminae, fibres, and surfactant maintain the shape and patency of alveoli during respiration.

- ***Pleurae*** *are fibro-elastic vascular membranes with mesothelial coverings. From the visceral pleura, connective tissue septa run in to subdivide the lung into lobules and carry lymphatic and venous vessels.*

Protective Mechanisms

Additionally to the mucociliary transport, other protective mechanisms of the respiratory system are solitary lymphoid nodules and tonsils, and their lymphocyte progeny, for immune defence, phagocytic alveolar macrophages/dust cells, reflex coughing, sneezing, and constriction of bronchioles, secretion of serous bacteriolytic materials, e.g., defensins and lysozyme and the recovery of water and heat, preventing too much loss in the expired air.

Some protection is hazardous in that enzymes from white blood cells can break down elastin; and activated lung macrophages stimulate fibroblasts to lay down movement-restricting collagen - an interstitial fibrosis (Beresford, 2007).

According to Aoshiba (2007), there is an increase in apoptotic alveolar epithelial and endothelial cells in the lungs of patients with emphysema, and since this is not counterbalanced by an increase in proliferation of alveolar wall cells as they become senescent, the net result is loss of alveolar unit leading to the development of emphysema. Furthermore, defective clearance of apoptotic cells in the lungs of patients with emphysema may promote inflammation.

2.3.4. Pathology, Prevalence, Pathogenesis and Therapy

2.3.4.1 Pathological Alterations

Symptoms caused by/accompanying COPD (ATS, 2007) are:

- Mucous hypersecretion and cilliary dysfunction
- Loss of lung elasticity and airflow limitation
- Gas exchange abnormalities
- Hyperinflation
- Pulmonary hypertension
- Other systemic effects

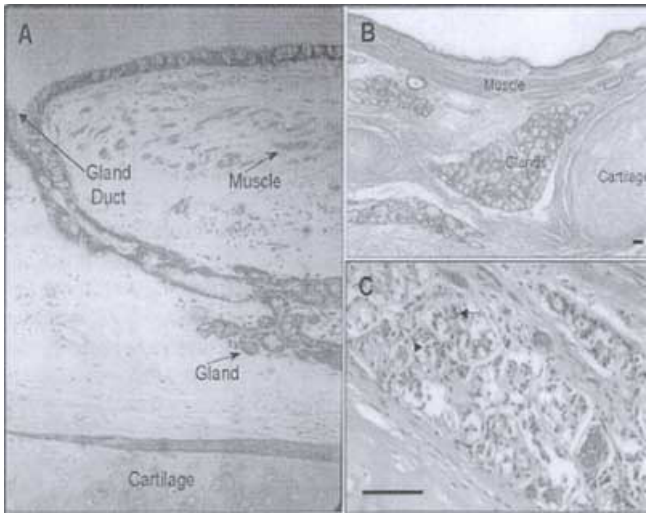
<u>Mucous hypersecretion and cilliary dysfunction</u>

Mucous hypersecretion is due to stimulated secretion from enlarged mucous glands. Cilliary dysfunction due to squamous metaplasia of epithelial cells (ATS, 2007).

In the central airways (cartilaginous airways >2mm of internal diameter bronchial glands hypertrophy and goblet cell metaplasia occurs. This results in **excessive mucous production** or chronic bronchitis. Cell infiltrates also occur in bronchial glands.

Airway wall changes include squamous metaplasia of the airway epithelium, loss of cilia and ciliary dysfunction, and increased smooth muscle and connective tissue.

Different inflammatory cells predominate in different compartments of the central airways. In the airways wall these are lymphocytes, predominantly of the CD8+ type, but as the disease progresses neutrophils also become prominent. In the airspaces, in addition to lymphocytes, neutrophils and macrophages can also be identified (ATS, 2007).

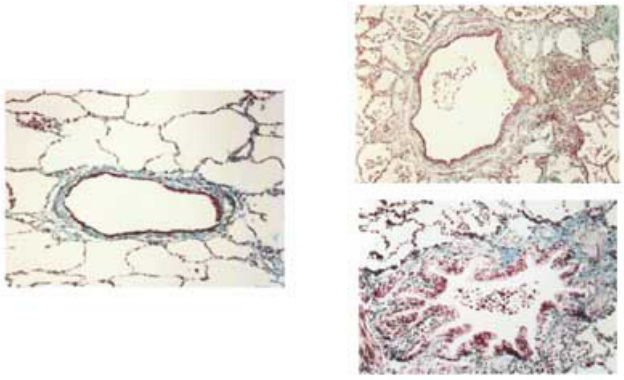


Ill. 5: a) A central bronchus from the lungs of a cigarette smoker with normal lung function; very small amounts of muscle are present; there are small epithelial glands. b) Bronchus from a patient with chronic bronchitis; shows a thick bundle of muscle and enlargement of glands. c) A higher magnification of the enlarged glands with evidence of chronic inflammation involving polymorphonuclear (arrow head) and mononuclear cells, including plasma cells (arrow).

Loss of lung elasticity and airflow limitation

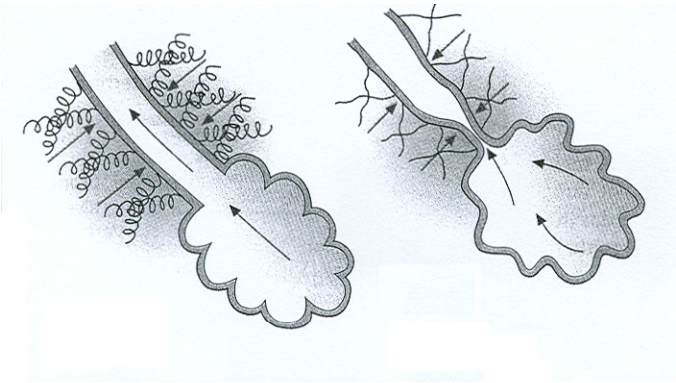
The major site of the airflow limitation is in the smaller conducting airways <2 mm in diameter and is mainly due to airway remodelling (fibrosis and narrowing). Other factors that also contribute include loss of elastic recoil (due to destruction of alveolar walls), destruction of alveolar support (alveolar attachments) [12], accumulation of inflammatory cells, mucous and plasma exudate in the bronchi, and smooth muscle contraction and dynamic hyperinflation during exercise. The latter is one of the major contributors to exercise limitation in these patients (ATS, 2007).

Bronchiolitis is present in the peripheral airways (noncartilaginous airways <2mm internal diameter) at an early stage of the disease. There is pathological extension of goblet cells and squamous metaplasia in the peripheral airways. The inflammatory cells in the airway wall and airspaces are similar to those in the larger airways.
As the disease progresses, there is fibrosis and increased deposition of collagen in the airway walls (ATS, 2007).



Ill. 6: Histological sections of peripheral airways from a cigarette smoker with a) a nearly normal airway showing some airway inflammation, b) the presence of an inflammatory exudate in the wall and lumen of the airway, and c) an airway with reduced lumen and structural reorganisation of the airway wall, increased smooth muscle and deposition of peribronchial connective tissue.

Stability of the peripheral airways is gained by the elastic recoil of the parynchematous tissue (a schematic view of the elastic forces of the parynchematous tissue is shown in Ill. 7). A lung with emphysema loses its elasticity, which is important to keep airways open, and thus air distal of the collapsed airways is trapped ("air trapping"). The patients experience great difficulty exhaling and respiratory dead space is higher due to the higher airway resistance (Gosselink, 2000).



Ill. 7: Elastic forces of the parenchymous tissue during expiration in a healthy lung (left) and in a lung with emphysema (right).

Changes by COPD in the Lung Parenchyma

Emphysema, defined as an abnormal enlargement of air spaces distal to the terminal bronchioles, occurs in the lung parenchyma in COPD.

As a result of emphysema there is a significant loss of alveolar attachments, which contributes to peripheral airway collapse.

There are two major types of emphysema, according to the distribution within the acinus: 1) centrolobular (which involves dilatation and destruction of the respiratory bronchioles); and 2) panlobular emphysema (which involves destruction of the whole of the acinus). The former is the most common type of emphysema in COPD and is more prominent in the upper zones, while the latter predominates in patients with α_1-antitrypsin deficiency and is more prominent in the lower zones.

In the early stages of the disease, these are microscopic lesions. During the course of the disease, they may progress to macroscopic lesions or bullae (defined as an emphysematous space >1 cm in diameter). Bullous disease can also occur in the absence of COPD.

The inflammatory cell profile in the alveolar walls and the airspaces is similar to that described in the airways and persists throughout the course of the disease (ATS, 2007).

Ill. 8: a) Common airway with alveolar attachments. b) Loss of alveolar walls, enlargement of alveolar spaces and decreased alveolar wall attachment in emphysema.

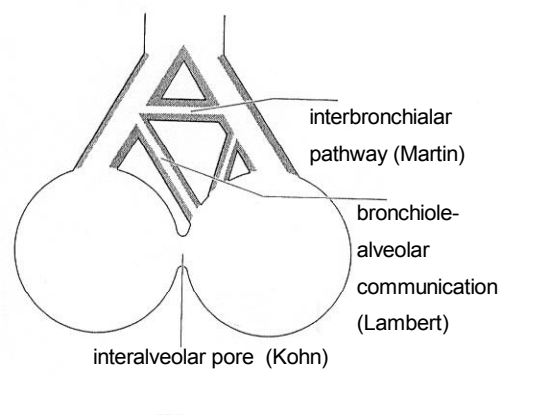
Hyperinflation

As a result of airway collapse and higher airway resistance, the respiratory pump is permanently doing additional work and muscles become fatigued without the necessary rest periods.

In patients with emphysema, diaphragm is contracted to a high extent and the accessory respiratory muscles fix the thorax almost in inspiration position, even

during resting ventilation. These restrictions in lung function are compensated by the respiratory pump as long as muscular force is still high enough.

As an additional response, in patients with obstructive pulmonary diseases additional airways can be observed. These "collateral airways" are classified in interbronchialar pathways, bronchiole-alveolar communications and interalveolar pores (cf. Ill. 9) and contribute to ventilation to a high extent. In healthy subjects collateral ventilation is unimportant. In collateral airways air resistance is essentially higher than in the normal peripheral airways, because passages are narrower and air needs more time to reach peripheral regions of the lung. Air resistance is dependent on lung volume: The higher lung volume, the lower is the resistance in the collateral airways. Normally, only intralobular collaterals can be observed, but interlobular links are possible, too (Gosselink, 2000).



Ill. 9: Collateral airways.

Mucus hypersecretion in combination with the loss of elasticity, moves the equal pressure point (EPP) to the smaller airways and a total closure **decreases efficacy of coughing** (Gosselink, 2000).

Gas exchange abnormalities

Gas exchange abnormalities occur in advanced disease and are characterised by arterial hypoxaemia with or without hypercapnia. An abnormal distribution of ventilation-perfusion ratios (due to the anatomical alterations described above) is the main mechanism of abnormal gas exchange in COPD.

An abnormal diffusing capacity of carbon monoxide per litre of alveolar volume correlates well with the severity of the emphysema (ATS, 2007).

Ideally, ventilation (V) and perfusion (Q) are in an equilibrium at the alveolar level. In reality, also in healthy subjects V/Q ratio is not perfect. There are regional differences in perfusion, depending on body posture and local oxygen partial pressures in blood. Since the latter has regulating influences on perfusion, lower ventilation in a lung region causes a local reflectory vasoconstriction and thus local perfusion is diminished. By this mechanism optimal V/Q ratio shall be re-established (Gosselink, 2000).

Pulmonary hypertension

Pulmonary Hypertension occurs late in the course of COPD, normally after the development of severe gas exchange abnormalities.

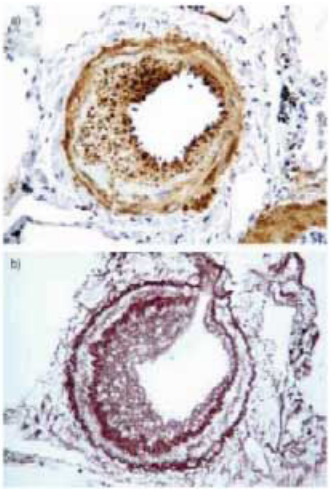
Factors contributing to pulmonary hypertension in COPD include vasoconstriction (mostly of hypoxic origin), endothelial dysfunction, remodelling of pulmonary arteries and destruction of the pulmonary capillary bed.

Pulmonary vascular changes begin early during the course of the disease.

Initially, these changes are characterised by thickening of the vessel wall and endothelial dysfunction.

These are followed by increased vascular smooth muscle and infiltration of the vessel wall by inflammatory cells, including macrophages and CD8+ T lymphocytes.

In advanced stages of the disease, there is collagen deposition and emphysematous destruction of the capillary bed. Eventually, these structural changes lead to pulmonary hypertension and right ventricular dysfunction (cor pulmonale) (ATS, 2007).



Ill. 10: Pulmonary muscular artery from a patient with chronic obstructive pulmonary disease, showing prominent intimal thickening and luminal narrowing. a) Immunostaining with monoclonal antibody against a-smooth muscle actin, showing abundant proliferation of smooth muscle cells in the intima. b) Orcein stain showing abundant deposition of elastic fibres in the intimallayer. Internal scale bar=42.2 mm.

In my opinion, this diminished blood circulation might also have negative effects on the immune response and thus be one reason for the inflammatory exacerbations in patients with COPD.

Other systemic effects

COPD is associated with extrapulmonary effects, including systemic inflammation and skeletal muscle wasting. These systemic effects contribute to limit the exercise capacity of these patients and to worsen prognosis, independent of their pulmonary function (ATS, 2007).

2.3.4.2 The Prevalence of COPD

To say in advance, there is no clear consensus as to what constitutes an obstructive ventilatory defect (OVD) and thus prevalence of obstructive ventilatory defect in a population depends on the definition chosen.
Prevalence is lowest with the criterion FEV1/**FVC** < lower limit of normal (LLN), with FEV1/FVC<0.70 slightly higher values can be found and with FEV1/**VC**<LLN prevalence is highest. The differences in calculations are approximately 10% absolute (Ben Saad et al., 2007).

Recently, data about the prevalence of COPD in Austria were published by Schirnhofer et al. in 2007, who surveyed a gender-stratified, population-based sample of 2.200 adults in Salzburg older than 40 years of age. The findings of prebronchodilator and postbronchodilator spirometry, as well as information on smoking and reported respiratory disease was recorded. Irreversible airflow obstruction was defined as a postbronchodilator FEV(1)/FVC ratio of < 0.70. For 1.258 participants with good-quality postbronchodilator spirometry findings, the overall prevalence of COPD at stage I or higher was 26.1%, and was equal in men and women. The prevalence of COPD stage II or higher (FEV(1)/FVC ratio, < 0.7; FEV(1), < 80% predicted) was 10.7%. A doctor diagnosis of COPD was reported by only 5.6% of participants. That means, that one quarter of residents of Salzburg County (Austria) who were ≥ 40 years of age had at least mild irreversible airflow obstruction, but only few of them had a diagnosis of COPD (Schirnhofer et al., 2007).

Some other prevalence data confirm the higher prevalence in industrialised nations and in men and give also insight into economic consequences of this disease:

In the United States prevalence of COPD in the workforce population is substantial with 46.5% of current employment among adults having the disease. In 2000, an estimated 10.5 million people had COPD, of which more than 7.2 million were from the under-age 65 employed population. COPD-related per patient total medical costs decreased from $1460 in 1999 to $1138 in 2003 largely because of a decrease in the cost of hospitalisations for COPD (Nurmagambetov et al., 2006).
According to Rufino and Lapa e Silva (2006) 24 billion dollars are spent annually on the diagnosis and treatment of 16 million patients in the USA.

In Brazil, COPD ranks as the sixth greatest cause of death, with a prevalence of 15.8% according to the fixed-ratio FEV1/FVC = 0.70 (Jardim and Nascimento, 2007).

The average prevalence of COPD (criterion post-BD FEV(1)/FVC) < 70%) in females in six Chinese rural areas was 5.4%. Statistical association of COPD was found with a family history of respiratory diseases (odds ratio (OR) no history vs. history = 2.46), frequent coughing during childhood (OR frequent coughing vs. never coughing = 3.93), lower body mass index (OR lower body mass index vs. normal body mass index = 2.20, age (OR 70 years or older vs. 40 - 49 years = 8.98, (OR smoking vs. no smoking = 1.68,), exposure to occupational dusts (OR exposure vs. no exposure = 1.45), worse ventilation in kitchen room (OR worse vs. good ventilation = 1.47) and lower educational level (OR low vs. high educational level = 2.19) (Ran et al., 2006).

2.3.4.3 Pathogenesis

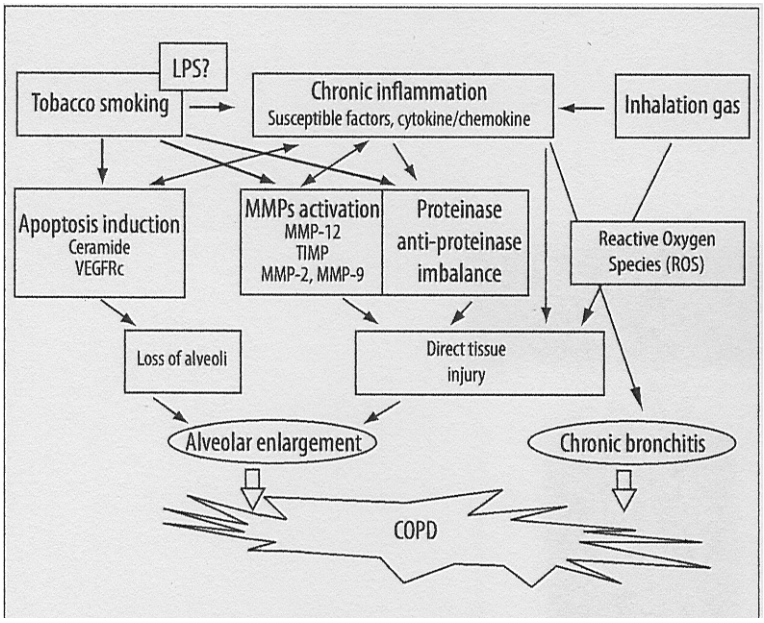
In this chapter, I will present a summary of the actual state of research, except the wide field of genetics which will not be touched due to my lack of knowledge.

According to Chan-Yeung et al. (2007) risk factors for COPD in Hong Kong-Chinese patients found were smoking (most important), being male, poor education and low body mass index. A dose-dependent relationship was found between the risk of COPD and pack-years smoked. Place of birth, exposure to environmental tobacco smoke and a history of asthma and tuberculosis were not associated with increased risk of COPD.
Since only 10-20% of smokers develop COPD, an additional influence of genetic susceptibility is considered. Genetic researches in this field mainly focused on variants of genes involved in protease anti-protease systems, defense against oxidative stress and inflammation (Keicho and Matsushita, 2007).

Inflammation, proteases-antiproteases imbalance, oxidative stress, tissue damage and tissue repair, apoptosis and several genes seem to be involved in the pathogenesis of the disease. The cellular and molecular events underlying COPD pathogenesis are driven by multifunctional molecules including enzymes, cytokines, chemokines, growth factors, lipid mediators and their respective receptors (Tzortzaki et al., 2007).

In a review by Fujiata and Nakanishi, actual theories about COPD-pathogenesis learned from in vivo animal models and discussed for humans are summarised (Fujiata and Nakanishi, 2007: RA22). These model is shown in Ill. 11.

The current paradigm for the pathogenesis of COPD is that a chronic airflow limitation results from an abnormal inflammatory response to inhaled particles and gases in the lung. Alveoli and bronchial inflammation are induced in susceptible smokers. In addition, a protease-antiprotease imbalance has been shown to be a major contributor to COPD. It has also been proposed that the development of emphysema may involve alveolar cell loss through apoptosis.



Ill. 11: Schematic drawing of chronic inflammation and COPD pathogenesis according to Fujiata and Nakanishi, 2007. LPS… Lipopolysaccharide, MMP… matrix metalloproteinase, VEGFRc… Vascular endothelial cell growth factor receptors.

Apoptosis induction

Apoptosis, the process of programmed cell death, plays a major role in cellular homeostasis, maintaining the delicate balance between cell proliferation and cell death. Various studies have demonstrated apoptosis in human emphysematous lungs, even without the accumulation of inflammatory cells. Vascular endothelial cell growth factor (VEGF) is a growth factor required for endothelial cell survival, and blocking VEGF leads to the apoptosis of endothelial cells.

Recently, **VEGF reduction** has even been considered to be an autoimmune disease, although others have ruled out the possibility of an acquired immune system in COPD. This contrasts with the traditional hypothesis of cigarette-smoke induced pulmonary inflammation leading to protease-mediated destruction of alveolar walls (Fujiata and Nakanishi, 2007).

Recently, **ceramide** has been reported to contribute to the development of COPD. Apoptosis blocked by VEGF receptor antibody induces inflammation by ceramide, a second messenger lipid. Ceramide is induced by endothelial apoptosis and the administration of ceramide induces MMP-12 expression. Ceramide is considered to be a link between excessive apoptosis and inflammation (Fujiata and Nakanishi, 2007).

MMP activation

Matrix metalloproteinase-12 (MMP-12) is nearly undetectable in healthy macrophages. In contrast, MMP-12 is expressed in the alveolar macrophages of human cigarette smokers. MMP-12 knock out mice did not develop emphysema in response to long-term cigarette smoke exposure. MMP-12 knock out mice also failed to recruit macrophages into their lungs in response to cigarette smoke. Neutrophil elastase deficient mice were significantly protected from the development of pulmonary emphysema after cigarette smoke exposure (Fujiata and Nakanishi, 2007).

Metalloproteinases (MMP) and elastases are enzymes, secreted by lyosomes, which themselves are released by macrophages. They are essential to the normal development of the pulmonary tissue, as well as to its remodeling and repair (Rufino et al., 2006).

Macrophages, one type of inflammatory cells, play an important role in the development of COPD and are found to be in increased numbers in the bronchial wall as well as in the lung parenchyma, especially in the alveolar spaces, in patients with COPD. Macrophages are cells derived from the bone marrow and the blood monocyte. They constitute the most common cell type among those that reside in the lung. They have various functions: they phagocyte particles or antigens; participate in the presentation of antigens to T lymphocytes; and can release various cytokines and active metabolites of arachidonic acid. In smokers, there is a greater release of lysosomes, up to five times more than in nonsmokers (Rufino et al., 2006).

Protease and anti-protease imbalance

In animal experiments considerable evidence describing a protease and anti-protease imbalance hypothesis has been published. The intratracheal instillation of elasatase results in a rapid and significant airspace enlargement, followed by acute neutrophil and subacute macrophage accumulation in the lung with hemorrhaging. In addition, other elastases including neutrophil elastase and proteinase-3, but not nonelastolytic enzymes such as bacterial collagenase causes COPD-like changes.

Protease is released by neutrophils, and is toxic to microorganisms but also injurious to the tissues.

Neutrophils are some of the central cells in the physiopathological mechanism of COPD, in which these cells are accumulated and activated. Neutrophils are abundant in blood. However, they are rarely found in the pulmonary tissue of healthy individuals. They have a short half-life, surviving for few hours (six hours on average) after being released by the bone marrow. In an inflammatory reaction, various factors

are produced that are chemotactic for neutrophils, which rapidly migrate to the site of inflammation, where they exert a phagocytic function against bacteria, fungi and viruses.

Protein substances, such as elastase, acid phosphatase, beta-glucuronidases, myeloperoxidase, metalloproteinases, lipocaine combined with gelatinase, proteinase 3 and cathepsin G, are released from the neutrophil granules. Such substances can participate, directly or indirectly, in the destruction of the lung parenchyma. Neutrophils also release other products that can promote chemotaxis and activation of other neutrophils, such as IL-8 and leukotriene B4. Therefore, they amplify and perpetuate the neutrophilic inflammatory process. These substances alter the balance between the production and degradation of proteins of the extracellular matrix, which results in the destruction of the alveolar wall (Rufino et al., 2006).

Reactive Oxygen Species

Cigarette smoke contains high concentrations of reactive oxygen species (ROS). This excess of ROS disturbs the balance between oxidants and antioxidants, resulting in oxidative stress. Gaseous irritants, like Sulfur dioxide (SO_2) and Nitrogen dioxide (NO_2) induce COPD-like lesions in animal models. SO_2 exposure leads to a more diffuse alveolar damage with more extensive damage with the destruction of lung tissue after a longer exposure. The administration of oxidants such as ozone (O_3) also causes significant lung injury with COPD-like lesions. Ultrafine particles such as silica and coal dust induce focal emphysema in animal experiments and exposure to diesel exhaust particles also lead to chronic airway inflammation in laboratory animals (Fujiata and Nakanishi, 2007).

Apart from macrophages and neutrophils, mentioned above, other inflammatory cells (e.g. lymphocytes) can be observed in COPD, too. Their influences on the disease are discussed in detail in Rufino et al. (2006).

2.3.4.4 Therapy

Since COPD is not fully reversible, treatment focuses largely on symptomatic relief and can be classified in pharmacological treatment, ventilation therapies, surgery and pulmonary rehabilitation.

2.3.4.4.1 Pharmacological Approaches

According to Kunisaki et al (2007) effective drug therapies for exacerbations of COPD include inhaled bronchodilators, systemic corticosteroids and antibacterials.

The two main classes of inhaled bronchodilators are beta-adrenoceptor agonists and anticholinergics. These drugs optimise lung function during exacerbations, with neither class demonstrating clear superiority over the other.
Systemic corticosteroids are effective when used either for inpatient or outpatient treatment of exacerbations. They hasten recovery from exacerbations and reduce relapse rates.
Antibacterials decrease morbidity from exacerbations and may decrease mortality in the more severe exacerbations.
Strategies to prevent COPD exacerbations include smoking cessation, long-acting inhaled beta-adrenoceptor agonists, inhaled long-acting anticholinergics, inhaled corticosteroids and vaccination. Mucolytic agents, pulmonary rehabilitation, and case management programmes may also reduce exacerbation risk, but the current evidence supporting these interventions is weaker.

Decramer and Ferguson, 2006 reviewed the safety of long-acting beta2-agonist (LABA) and inhaled corticosteroid (ICS) combination therapy which is recommended by international treatment guidelines for COPD.
Bronchodilators such as LABAs are key treatments for COPD due to their effects on bronchial smooth muscle and airflow limitation. LABAs are well-tolerated in patients with COPD, with a low incidence of reported adverse events. Most adverse events associated with LABA use are due to systemic exposure and include muscle tremor and cardiac effects. Placebo-controlled studies in patients with COPD demonstrate that there is no increase in risk of cardiac adverse events with LABA therapy.
ICS therapy targets airway inflammation in COPD, and is associated with a reduction in the frequency of COPD exacerbations, and improvements in symptoms, lung function and health status. Localized effects such as oropharyngeal irritation are common with ICS, but are not considered to be serious. Potential ocular effects with ICS therapy in patients with COPD have been identified and require further investigation. Rare, but more serious adverse events related to ICS use are the effects on bone and the suppression of endogenous cortisol production.
Clinical data indicate that LABA/ICS combination therapy is more effective in COPD than either agent used alone and is not associated with any additional adverse events.

Contrary, according to Ernst et al. (2007), there is an association of the use of inhaled corticosteroids with an excess risk of pneumonia hospitalization and of pneumonia hospitalization leading to death within 30 days, among elderly patients with COPD.

Recently, Calverley et al., 2007 studied the effect on survival of long-acting beta-agonists and inhaled corticosteroids. In spite of significant benefits in all other outcomes, the reduction in death did not reach the predetermined level of statistical significance compared to a placebo group: The mortality rate for salmeterol alone or fluticasone propionate alone did not differ significantly from that for placebo. As compared with placebo, the combination regimen reduced the annual rate of exacerbations from 1.13 to 0.85 and improved health status and spirometric values ($P<0.001$ for all comparisons with placebo). There was no difference in the incidence of ocular or bone side effects. The probability of having pneumonia reported as an adverse event was higher among patients receiving medications containing fluticasone propionate (19.6% in the combination-therapy group and 18.3% in the fluticasone group) than in the placebo group (12.3%, $P<0.001$ for comparisons between these treatments and placebo).

2.3.4.4.2 Non-Pharmacological Approaches

Ventilation therapies

According to Kunisaki et al. (2007), effective therapies for the treatment of acute exacerbations of COPD include oxygen and non-invasive ventilation. Oxygen can be safely administered in acute exacerbations associated with hypoxaemia, with titration of oxygen delivery to a goal oxygen saturation of 90%. Non-invasive ventilation reduces the morbidity and mortality associated with acute exacerbations complicated by hypercapnic respiratory failure.

Oxygen therapy is one of the principal non-pharmacologic treatments for severe chronic obstructive pulmonary disease (COPD) patients. Home oxygen therapy (HOT), or long-term oxygen therapy (LTOT) for 15 hours or more per day, can improve the survival rate of severe COPD patients with beneficial effects on hemodynamic state, hematological characteristic, exercise capacity, lung mechanics, and mental state. Oxygen therapy is indicated in cases of severe chronic respiratory failure with PaO2 of 55 Torr or less, or in cases with PaO2 of 60 Torr or less in whom there is remarkable hypoxia during sleep or during exercise. The induction of oxygen therapy needs evaluations of oxygen desaturation during exercise and sleep as well as hypoxia at rest (Nakamura and Ishizaka, 2007).

Noninvasive mechanical ventilation has been of use in the treatment of some forms of chronic and acute respiratory failure. According to Neme et al., a combination of continuous positive airway pressure (CPAP) and pressure support ventilation (PSV)

improves respiratory mechanics and alveolar ventilation, and reduces inspiratory muscle effort (Neme et al., 2007).

Melsom et al. tried to redistribute blood flow to well-ventilated lung regions in order to decrease V/Q mismatch. It has been suggested that inhaled nitric oxide (iNO) in physiologic concentrations (approximately 100 p.p.b.) could act as a local vasodilating agent in well-ventilated lung regions. Result of this study is, that neither low nor high concentrations of iNO improve oxygenation in patients with very severe COPD (Melsom et al., 2007).

Pulmonary Rehabilitation

Skumlien et al. investigated changes in health according to World Health Organization's International Classification of Functioning, Disability and Health (ICF) after four weeks of pulmonary rehabilitation (PR). Twenty-two men and 18 women with chronic obstructive pulmonary disease in Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage II-IV attended in-patient, multidisciplinary PR consisting of endurance training four to five times/week at 70% of peak work rate (WRpeak), resistance training three to four times/week at 72% of 15 repetitions maximum, educational sessions and individual counselling. The results were compared to those of 20 Chronic Obstructive Pulmonary Disease (COPD) patients included after the same criteria and investigated while waiting for admission to PR. In the rehabilitation group, significant improvements in health related quality of life (HRQoL) (-7 units, St. George's Respiratory Questionnaire), arm (6%) and leg (15%) maximal voluntary contraction, peak oxygen uptake (6%), WRpeak (60%) and treadmill endurance time (93%) were found. At iso-WR, ventilation and dyspnoea were significantly lower, but inspiratory capacity remained unchanged. Improvements in HRQoL correlated with increases in peak ventilation, but not in muscle strength or exercise capacity. Men improved their six-minute walking distance significantly in contrast to women. Clinically important improvements in HRQoL were found in two out of three of the men, and one out of three of the women. Four weeks of intensive PR generated significant health effects comparable to longer lasting programmes. Changes in exercise capacity and muscle strength were not related to improvements in HRQoL. (Skumlien et al, 2007).

Paz-Diaz et al. investigated the impact of an 8-week program of comprehensive pulmonary rehabilitation on depression, anxiety, dyspnea, and health-related quality of life in patients with chronic obstructive pulmonary disease (COPD) on 24 patients

with severe COPD randomized either to pulmonary rehabilitation (PR), (n = 10; FEV1 30 +/- 9%) or control (C; n = 14; FEV1 34 +/- 11%). The pulmonary rehabilitation program included disease education, energy conservation techniques, relaxation, and exercise including 20-min arm elevation with dumbbells and 20-min leg exercise sessions three times a week for eight weeks. At baseline and after completion of the program, all patients were evaluated using the Beck Depression Inventory, State Trait Anxiety Inventory (STAI), Modified Medical Research Council Scale (MRC), and St. George's Respiratory Questionnaire (SGRQ).

After PR, there was a significant improvement in the severity of depression ($P < 0.01$), a decrease in symptoms ($P < 0.05$), an increase in daily living activities ($P < 0.05$), and a decrease in the total score of the SGRO ($P < 0.01$). Dyspnea measured by the MRC scale was significantly better in the PR group ($P < 0.01$) (Paz-Diaz et al., 2007).

Surgical therapies

According to Vasko et al. three surgical options are currently employed: Bullectomy, lung volume surgery and lung transplantation. During volume reduction surgery the hyperinflated areas are resected, so the functional residual volume is getting reduced, the respiratory muscle function is increased (Vasko et al., 2006).

Bullectomy and lung volume reduction surgery may result in improved spirometry, lung volume, exercise capacity, dyspnoea, health-related quality of life and possibly survival in highly selected patients. Lung transplantation results in improved pulmonary function, exercise capacity, quality of life and possibly survival in highly selected patients. It has to be considered, that patients with a diagnosis of COPD have a 2.7-4.7-fold increased risk of postoperative pulmonary complications (ATS, 2007).

Alternative therapies

Wu et al. investigated the influence of acupressure on oxygen saturation in order to determine if acupressure would lessen dyspnea and reduce depression in patients with COPD.

In a randomized, double blinded experimental study 44 subjects were randomly assigned to either a true acupressure or a sham acupressure group. The true acupressure group received a program of acupressure using appropriate acupoints that promote relaxation and relieve dyspnea. The sham acupressure group received acupressure using sham acupoints different from the meridians and ganglionic sections of the true acupressure group. Both acupressure programs lasted four weeks, with five sessions per week that lasted 16 minutes per session. The Geriatric

Depression Scale (GDS) and Dyspnea Visual Analogue Scale (DVAS) were administered prior to the program as a baseline, and again following the completion of the four-week program. Oxygen saturation and other physiological indicators were measured before and after each session. The results of this study showed that the GDS scores, DVAS scores, oxygen saturation, and physiological indicators of the true acupressure group were significantly improved, compared to those of the sham acupressure group (Wu et al., 2007).

3. Osteopathic Aspects

In the beginning of this chapter, I will summarise the crucial points of pathogenesis and pathology for osteopathic considerations. Then, I will give an overview about the osteopathic scientific literature. Finally, I will describe my osteopathic approach to the patients, as a result of these considerations and literature data.

3.1. Osteopathic Principles and Pathology of COPD

3.1.1. Introduction

While traditional state of the art treatment of COPD largely focuses on symptomatic relief, osteopathic treatment aims at resolving or at least reducing causal pathological factors, in order to encourage the body to take over its **self healing mechanisms**.

But, what are the causal pathological factors for COPD?

Recent studies give hints to genetic predisposition and auto-immune reactions.
In these cases, osteopathic treatment predominantly has to aim at the strengthening of the resistance of the body.
Otherwise, having a genetic predisposition does not mean that disease will break out inevitably. Additionally, conditions have to be appropriate. Therefore, it is important to avoid or resolve additional risk factors:

Since **structure and function are interdependent and the body functions as an entity**, the therapist should support the patient to re-establish **free mobility** of the fasciae and joints and to harmonise body statics.

As an example, Beal and Morlock describe in a review of osteopathic literature on respiratory disease, that the majority of those with lung disease had changes in the

spinal area Th2-7. In their actual study, all 40 patients with lung disease had abnormalities of Th2-7 (Beal and Morlock, 1984).

The loss of mechanical flexibility and hyper-tension inside the tissue lead to a deterioration of the supply- and discharge condition (**law of the arteries)**. A proper circulation of all bodily fluids (blood, lymph and liquor) is a prerequisite for health. A disturbed removal of toxic substances accounts for the initial problems in COPD.
Thus, also visceral disturbances have to be resolved in order to support the auto-regulation processes of the immune system, the endocrine system, the autonomous nervous system and other regulative systems. This also because disturbed homeostasis is another reason for the formation of COPD.

Last but not least, patient's well-being should be the essential aim of osteopathic treatment. By resolving any dysfunctions which can be diagnosed, quality of life should be improvable.

3.1.2. Pathology of COPD from an Osteopathic Point of View

Respiratory effort is increased in COPD patients by chronic endo- and exobronchial obstructions of the airways. By obstructions air way resistance increases inversely proportional to the fourth power of the air way radius (e.g.: if the radius is halved, air way resistance is increased by the factor 2^4=16, Hagen-Poiseuille's law, Anaesthesist, 2006)
Thus, air way obstruction is a main factor in the increase of respiratory effort.

Another reason for the increased effort is the development of emphysema, with the proceeding of COPD. These are accompanied with a loss of elasticity of the lungs and a reduction of the diffusion area, resulting in a reduced gas exchange, an alteration of the respiratory mechanics (hyperinflation) and air trapping (cf. chapter 2.3.4.1).

The thoracic cage is expanded by the loss of the retraction forces of the lung and it draws the lung into inspiratory position. Concurrently, the diaphragm remains in caudal position. That means, that the inhaled air remains in the lungs.

By the contraction and shortening of the respiratory muscles, in many cases, the phrenic nerve is irritated and the intrathoracal fasciae are retracted.

Simultaneously to the reduced exhalation also inhalation is reduced and exercice capacity decreases.

Some of my patients had overburdened respiratory muscles even during rest.
Scalenic muscles and mm. sternocleidomastoidei, which draw the first rib to superior and dorsal, are hypertone and shortened, and thus a shortened distance between sternum and thyroid cartilage can be observed. Normally, this distance is three fingerbreadths, in COPD patients shorter.

As discussed in chapter 2.3.4.1 respiratory accessory muscles are used to compensate the lower gas exchange.
By the increased tonus of the tonic respiratory accessory muscles (m. pectoralis major and minor), they promote a cyphotic curvature of the thoracic spine, via an internal rotation of the glenohumeral joint and a protraction of the scapula, causing other severe dysfunctions of respiration and body static:

- Adductors and stabilisers of the scapula, the pars ascendens and pars transversus of the trapecius muscle and the extensors of the thoracic spine are weakened by a reciprocal restraint and continuing distension.

- The mobility of the spine in rotation becomes restricted.

- Derivation and attachment of the abdominal musculature converge and thus is actively insufficient with resulting dysbalances in the abdominal "muscle corset".
- The cyphotic curvature of the thoracic spine often is the reason for a cyphotic sitting posture (the lumbar spine is in flexion), which takes the pressure of the pelvic floor and thus attenuates (weakens) it, due to the lack of a permanent stimulus. Under stress (caughing and sneezing) ptosis may occur and the bladder declines to dorsal and its sphincter system may become insufficient. Involuntary loss of urine may be the consequence. Additionally, overweight may add to this process.

- Hyperextension of the cervical spine with an overload of passive joint structures and thus an affection of the vagus nerve.

3.2. Osteopathic Literature

Literature is divided into articles about the effect of osteopathic treatment on COPD patients in special and those about effects on lung function in general. Finally a chapter with miscellaneous articles of interest is added.

3.2.1. Effects of Osteopathic Treatment on COPD

Cosmai found an improvement of the small and medium airways due to a better functioning of the accessory and primary respiratory muscle fibres (improvement of FEV1) of patients suffering from chronic obstructive bronchopneumopathy after four weekly osteopathic treatments collaborating with medical therapy compared to a control group with medical therapy only (both groups n=15, Cosmai, 2003)

Allen et al. assessed the influence of osteopathic manipulative therapy in the management of patients with chronic obstructive lung disease. In a 9-month study on the effects of spinal manipulative therapy as a treatment for obstructive pulmonary disorders, there was a progressive decline in the severity of the condition. The average reduction in severity was approximately 10.7%. All of the patients were noted as having costotransverse dysfunction at the level of T3, as well as T2 being noted in patients with asthma. Joint motion between T3/T4 was restricted. Local tissue was edematous and tender to palpation (Allen et al., 1975).

Miller studied the influence of osteopathic manipulation on patients with chronic obstructive pulmonary disease. 92% of the patients stated they were able to walk greater distances, had fewer colds, experienced less coughing, and had less dyspnea than before treatment (Miller, 1975).

Gibb attempted to investigate the possibility of cervical HVT (C3-4 segment) to affect the phrenic nerve and thus the diaphragm as a way of treating chronic obstructive pulmonary disease (COPD) and asthma. Eleven normal male subjects performed spirometric maximal manoeuvres measuring lung function. They were given a placebo treatment and tested again. A week later the subjects were manipulated bilaterally at the C3-4 level and their lung function tested again resulting in **no significant difference** between the placebo and HVT treatment (Gibb, 2002).

Masarsky and Weber describe a case of a 53-year-old patient with a history of chronic obstructive pulmonary disease going back more than 20 years. He was treated with a

combination of chiropractic manipulation, nutritional advice, therapeutic exercises, and intersegmental traction. Improvements were noted in forced vital capacity, forced expiratory volume in one second, coughing, fatigue, and ease of breathing ($p < 0.005$). Improvement was also noted in laryngospasm (Masarsky and Weber, 1989).

3.2.2. Effects of Osteopathic Treatment on Lung Function

According to Gibellini F. (2002), both FEV and FVC improved significantly after four weekly specific osteopathic treatments of normal subjects compared to a placebo group (both groups n=10).

Rupp studied the influence of osteopathic treatment on lung function in Idiopathic Parkinson's Syndrome (IPS). The control group (13 subjects) received an up-to-date medical and physiotherapeutical treatment and the treatment group (13 subjects), additionally, received two individually adjusted osteopathic treatments within the intervention period. Lung function of Parkinson's patients in both groups was measured by means of spirometry at the beginning and the end of the intervention period.
In the treatment group all lung function parameters could be clearly improved. The changes in parameters FVC, FEV1, PEF, and MEF75 at $p<0.05$ were **significant**. Results of the control group did not show any significant differences (Rupp, 2007).

Wieser treated the mediastinum in order to gain a free mobility of the thorax. Since it is assumed that the mobility of the thorax is limited by the tension of fascia and ligaments, the relaxation of the pericardial ligaments might improve lung function. To investigate whether the treatment of the mediastinum can improve vital capacity of persons with palpable restrictions of fascia 60 adult persons were randomly divided into Test (n=30) and Control (n=30) groups. The osteopathic treatment consisted of a recoil technique to the sternopericardial ligaments and a stretch technique to the vertebropericardial ligaments. Lung function was assessed before and after the treatment using a forced expiration test. The treatment did **not result in a significantly increased vital capacity** (Wieser, 2007).

M'Lennan explored the effect of manual therapy on lung function. The purpose of her study was to investigate the effect of Muscle Energy Technique (MET) on lung function and chest expansion in a normal healthy non-smoking population. Ten male

and ten female subjects aged 20-35 years performed maximal expiratory manoeuvres before and after treatment. Chest expansion, forced vital capacity, forced expiratory volume in one second and peak expiratory flow were measured. Muscle energy technique increased chest expansion at all three levels measured ($p<0.05$) and forced vital capacity in both male and female subjects ($p<0.05$, M'Lennan, 2002).
Since no significant changes were found for FEV1, Tiffeneau indices of **healthy subjects** are not positively influenced by MET.

According to Schröder, lung function (VC and FEV1) can be improved by stretching thoracal fasciae. Intrathoracic fasciae of 28 healthy subjects were treated with stretching methods, in order to improve the ability of thorax and lung to expand. By this, VC was significantly improved (approx. 11%). FEV1 was improved, too, but to a smaller and not significant extent (approx. 3%) only.
Thus, no short- term improvements of the Tiffeneau indices are achieved by these techniques on **healthy subjects** (Schröder, 2003).

Fischer describes a significant increase (approx. 3%) of VC (n=30) after manipulation of the thoracic spine (Th1, 2, 3 and 5) in otherwise healthy subjects compared to a control group without spinal dysfunctions. No significance was found for Th4 (Fischer, 2003).

Effects of soft tissue technique and Chapman's neurolymphatic reflex stimulation on respiratory function were assessed by Lines and co-workers. Thirty asymptomatic subjects received care by means of these techniques. Measurements of forced vital capacity (FVC) were taken. A significant improvement in FVC was noted, suggesting that chiropractic may improve breathing capacity (Lines et al., 1990).

3.2.3. Other Articles of Interest

Since neutrophils and mononuclear cells are considered to be involved in pathogenesis of COPD, the following articles might be of interest, too.

Brennan et al. studied biological changes associated with the forces applied by spinal manipulation. They determined the priming of polymorphonuclear neutrophils for an enhanced [internal, Grabner] respiratory burst and its duration, the priming of mononuclear cells for enhanced endotoxin-stimulated tumor necrosis factor

production and plasma levels of substance P following a single thoracic spine manipulation (mean manipulation force: 878 +/- 99 N).
Both polymorphonuclear neutrophil (PMN) and monocyte concentrations from subjects who received spinal manipulation were significantly higher after than before treatment, and significantly higher than the response in sham or soft- tissue treated subjects. Measurement of the force applied by sham and spinal manipulation suggested a force threshold for the enhancement of the CL response.
There was a significant difference in the respiratory burst of polymorphonuclear neutrophils in response to a particulate challenge, depending on the time of blood sample collection. The response of polymorphonuclear neutrophils isolated from blood collected 15 min after manipulation was significantly higher than the response of cells isolated from blood collected 15 min before and 30 and 45 min after manipulation. Mononuclear cells were also primed for enhanced endotoxin-stimulated tumor necrosis factor production by spinal manipulation. Both of these priming effects were accompanied by a slight, but significant elevation in plasma substance P (Brennan et al., 1991, Brennan et al, 1992).

Since these substances are harmful to lung tissue, this could be a contraindication for spinal manipulation of COPD patients.
Therefore, it would be of interest if neutrophil and monocyte concentrations are enhanced due to additional release or to accelerated transport processes (e.g. from sites of inflammations).

3.3. Osteopathic Diagnosis and Treatment

3.3.1. Diagnosis

During diagnosis I attached importance to:

- Palpations for tensions and quality of the cervical fasciae and myofascial elements in the superior thoracic aperture and the suboccipital muscles.

- Assessment of the rib mobility - are there restrictions during inspiration of expiration (interapophysal spinal joints, costotransversal joints, costovertebral joints, chondrocostal joints, chondrosternal and sternal joints)

- Assessment of the mobility of the pulmonary lobes (lung expands and tilts three dimensionally during respiration)

- Test of the elasticity, symmetry and quality of movement of the diaphragm during respiration (a restriction of the superior diaphragm movement indicates prenoptosia)

- Assess of the quality of the lung tissues concerning circulation and drainage

- Local listening of the mediastinum

- Fascial tests of the mediastinum and thorax

- Palpation of the subclavial muscle (the space between the first rib and clavicle is compacted by a hypertension of this muscle and subjacent structures are compressed (trapezoid and conoid ligament of the clavicle, parietal pleura, phrenic nerve, subclavial vein)

- Palpation of the costopleural, transversopleural and vertebropleural ligaments. for assessing the attachment of the pleura.

- Ventral Chapman reflex points (2^{nd},3^{rd} and 4^{th} intercostals spaces near sternum)

3.3.2. Osteopathic Treatment

Taking into consideration the pathological aspects and osteopathic principles, apart from treatment of individual other dysfunctions, I mainly concentrated on the following aspects during therapy:

- Structural aspects of the spine, thorax and cranial base
- Fascial aspects of the thoracic cage and abdominal cage
- Aspects of the nerval supply
- Improvement of the supply- and discharge condition (blood and lymph flow)

3.3.2.1 Structural aspects of the spine, thorax and cranial base

- **Mobilisation of the first rib and relaxation of the subclavius.**

  by a technique for the suspensory ligaments and the cupula of pleura. This makes sense, because elasticity of the superior thoracic aperture

is improved and agglutinations are reduced. These techniques, primarily aim at the resolution of possible tensions.

3.3.2.2 Fascial aspects of the thoracic cage and abdominal cage

- **Treatment of fixations of the cupula of the pleura**:

  The suprapleural fascia is connected via the superior thoracic aperture with the deep cervical fascia (profunda, media and pharyngobasilar fascia) and the cranial base. Restricted mobility of the fasciae affects the outflow and the nerval supply by the phrenic and vagus nerve.

- **Treatment of the diaphragm**

  According to Paoletti (2001), the diaphragm is a kind of fascia, separating the thoracic cavity from the abdominal one. It connects the thoracal and abdominal fasciae. The superior part of the diaphragm is covered by the endothoracic fascia and superficially by the pleura. This fascia is continuing as the transverse fascia in the abdomen. The inferior side of the diaphragm is covered by the peritoneum, where the liver and stomach are attached and the renal fascia origins. Additionally, there is a connection with the fascia of the psoas muscle.

  In the superior part, the diaphragm is attached by the visceral layer, formed by the pericardium. This fascia is connected with the cranial base by the fasciea pharyngobasilaris, interpterygoidea and palatina. The visceral layer is stabilized by the vertebropericardiac and sternopericardiac ligaments in anterior-posterior direction.
  Thus, diaphragm acts as a connecting link of the fasciae between cranial base, neck, thorax and abdomen.

  There are two opposed tearing forces forces on the diaphragm:
  To superior peripheral and central thoracal fasciae and to caudal abdominal fasciae and the weight of the there attached organs.
  The hyperinflation of the thorax causes a shortening and flattening of the diaphragm and the auxiliary muscles (scalenic muscles) are needed more often even during resting ventilation (inspiration rather by breast breathing instead of diaphragmic respiration, resulting in the formation of a barrel chest).

That means, if diaphragm is restricted, all fascial connections have to be assessed (to mediastinum, to pleura, to pericardium, to peritoneum).

- **Treatment of the diaphragm at the transition of the thoracic and lumbar spine**

  There is a connection of the crus of diaphragm and the anterior longuitudinal ligament of the spine and further to the os sacrum. Lateral there are the lateral and medial acuate ligaments, which are connected with the greater psoas muscle and the quadratus lumborum.

3.3.2.3 Aspects of the nerval supply

- **Treatment of the innervation of the diaphragm**

  The diaphragm is innervated by a descending branch of the cervical plexus, the phrenical nerve (C3-C5), motoric as well as sensible. Besides others, the phrenical nerve is connected to the cervicothoracic ganglion and the vagus nerve and is split and supplies the diaphragmatic plexus. Here, there are connections with the pericardium, the neurovegetative fibers of the intercostal nerves, with the neurovegetative plexi of the lungs, the coeliac plexus, with the cervicothoracic ganglion, with the coeliac ganglia, the cardio-esophageal junction, the cardia and the vagus nerves, and others (Netter, 1987).

  Therefore, the lower cervical nerves and the phrenic nerve have to be treated, if necessary.

- **Vagus nerve:** Treatment of the **upper cervical spine** and the **cranial base** as well as of the lamina prevertebralis

  The cervical spine is brought into hyperextention and passive joint structures are overloaded by a cyphotic curvature of the thoracic spine. Additionally, the **parasympathetic nervous system**, which rises from the base of the fourth ventricle (dorsal nucleus of the vagus nerve) and passes the jugular foramen, may be irritated. If such irritations can be found, tensions of the occipitomastoid suture, the jugular foramen and C0-2 can be resolved by suboccipital soft tissue techniques and craniosacral techniques.

- **Phrenical nerve:** Treatment of the **cervical spine (C3-C5).**

  The phrenical nerve is a descending branch of the cervical plexus and is both, a sensible and motoric nerve. It is connected with the cervico-thoracic ganglion and the vagus nerve (and others). It is responsible for the innervation of the pleura and diaphragm (Barral, 1991). According to Barral, frequency and amplitude of the contractions of the diaphragm can be influenced, by treatment of the phrenical nerve running along the anterior scalene muscle.

  The phrenical nerve can be palpated at the 10th rib (Mussy's point), too. This point is painful on pressure in patients with pleuropulmonary disorders only, and can be relaxed by pressure in direction of the listening.

- **Treatment of the ribs, spine and sternum**

  Parietal restrictions of the thorax do not only affect mobility, but also influence the sympathetic share of the vegetative nervous system.
  By a harmonisation of the heads of the ribs at **Th1-Th6** and treatment of the dura mater spinalis (endothoracic fascia), via the the ganglionated cord (sympathetic chain - pulmonary plexus - peribronchial plexus - intramural plexus) sympathetic innervation can be influenced.

3.3.2.4 Improvement of the supply- and discharge condition (blood and lymph flow)

- **Relaxation of the diaphragm and expansion of the left and right pars costalis.**

  The treatment of the diaphragm is of particular importance, because it is *"the general compensation structure "*(F.Meert, 2007: 120, translated by Grabner, 2007). F. Meert designates the diaphragm as *"central motor or pump for the body fluids"* (F.Meert, 2007: 120, translated by Grabner, 2007), especially for the interstitial and lymphatic fluids. *"during exhalation, in a way, the diaphragm squeezes out the thoracic duct in cranial direction"* (F.Meert, 2007: 127, translated by Grabner, 2007).

- **Treatment of restrictions of the tissue**

  According to Carreiro (2004: 92), optimal function of the lymph vessels of the lungs is important for the humidity equilibrium in the airways. The ends of the vessels lay in the loose connective tissue and the peribronchial vascular area of the lung (O'Brodovich, 2000) and end in the bronchioles.

  The lymph of the left lung flows via the thoracic duct into the left venous angle, the lymph of the right lung moves via the lymphatic duct into the right venous angle.
  In order to avoid the collection of proximal lymph and an afflux, possible restrictions of the tissue in COPD patients have to be treated.

- **Lymphatic stimulation by the sternum pump**

  By the sternum pump a lymphatic stimulation in order to guarantee a free flow of the inflammatory exudations in the free lymph vessels and veins. By this method, predominantly vertical lymph vessels of the thorax and mediastinum (thoracic duct, parasternal trunk and bronchomediastinal trunk) are stimulated (F.Meert, 2007).

- **Treatment of the Mediastinum:** Mobilisation according to Barral

  Many structures, important for circulation, are located in the mediastinum (sympathetic chain, which is covered by the prevertebral lamina, vagus nerve, aorta, thoracic duct). Thus, mobilisation of the mediastinum should improve the supply conditions.

- **Visceral treatment of the subphrenic recess and of mediastinal tension**s

  Barral describes, that a restriction of the middle cervical fascia causes a narrowing of the veins running inside. If necessary it has to be treated (Barral, 1991).

- **Treatment of the first and 12th rib** in order to guarantee a sufficient lymphatic drainage via the cisterna chyli and the superior thoracic aperture.

  By this treatment a relaxation of the medial acuate ligament can be gained, resulting in an improvement of the arterial, venous and lymphatic flow. Additionally, the thoracic cage is enabled to expand further (Möckel and Mitha, 2006: 332).

Additionally, pressure is taken off the scalene and the costoclavicular hiati, and metabolic activity is stimulated by the treatment of the first rib.

- **Chapman reflex points**

  The Chapman reflex is described as a tension hindering the lymphatic drain and causing an inflammation in the myofascial tissue distal of the obstruction (Ward, 1997). For the lungs, these nodular points can be found dorsal near the periost of Th2-5 between the proc. spinosus and proc. transverses.

  Autoregulation of the neurovegetative and lymphatic circulation can be improved by treatment (vibratory treatment of approx. 10-15 seconds). Additionally, the internal organs are influenced via the vegetative nervous system.

4. Methodology

4.1. The Patients

Twenty-six patients with a doctor diagnosis of COPD by a pulmonologist in advance of this study were randomly assigned to a test- and to a control group by throwing a coin.

All spirometry measurements were performed by a medical doctor.

Patients of the test group had a first osteopathic treatment (cf. chapter 3.3.2) within the following five days and another after four weeks. A second spirometry measurement was done five weeks after the first one.

In my initial concept, I wanted the patients to have a spirometry directly after the first osteopathic treatment, too, but most of the patients did not show up at the doctor for spirometry. They came to spirometry only at the second date, when they needed another receipt for pharmacotherapy. Thus, these initially planned spirometry measurements were omitted.

Six of the patients (three of each group) did not show up for second spirometry and were excluded. The two groups were quite similar in age structure and body mass index, as can be seen in Table 3 and Table 4.

Var	Group	n	Mean	Std. Deviation
Age	Test group	10	50.9	8.20
	Control group	10	48.3	11.61

Table 3: Mean values and standard deviations of patients' age in the two groups.

Patients in the control group were slightly younger than the test persons in the study group.

Var	Group	n	Mean	Std. Deviation
BMI	Test group	10	29.5	6.94
	Control group	10	30.6	4.46

Table 4: Mean values and standard deviations of patients' body mass index (BMI) in the two groups.

Patients in the control group have slightly higher body-mass indices (BMI) than the test persons in the study group.

According to the medical doctor all patients had the same pharmacotherapy: bronchospasmolytics (beta2-agonists, systemic corticosteroids, combivent).

All of the patients were smokers or quit smoking within the last two years.

4.2. Anamnesis, Diagnosis and Treatment

For anamnesis I made use of two medical data sheets (my self administered and a standardized one for pulmonary diseases; cf. appendix 3).
Diagnosis was performed according to chapter 3.3.1, osteopathic treatment according to chapter 3.3.2.

4.3. Data Evaluation

Spiromety data before and after osteopathic treatment were collected in a datasheet (Microsoft® Excel 2000) and then were error checked. Software used for the evaluation was SPSS® 12.0.0.

It is the aim of this study to find out positive influences of osteopathic treatments on lung function (especially on vital capacity and the one second forced expiratory volume). Therefore, the null hypothesis "Osteopathic treatment has no influence on

vital capacity and one second forced expiratory volume" was tested by 1-tailed t-tests (level of significance α=0.05) for the patients before and after osteopathic treatments. Variables used are listed in Table 5.

Dependent variables		Independent variables
vital capacity	VC%pr	Spirometry: first measurement before treatment (1), second one after treatment (2).
forced expiratory capacity	FVC % pr	
forced expiratory volume in one second	FEV1 %pr	
forced expiratory capacity G in one second	FEV1 %Gpr	
forced expiratory capacity T in one second	FEV1 %Tpr	

Table 5: Variables used for the statistical evaluation.

Additional considerations

The spirometrical measurements were done on two summer days (16-06-06 and 28-07-06). Therefore, ozone concentrations may have a cross influence on COPD and thus spriometry data. Ozone predominantly is formed in rural areas and not in urban and industrial areas, where most of the precursors (NO_x), are emitted. Therefore, it is not possible to quantify the exposure of the individual patients to ozone exactly, because it is unknown where the patients have been before the measurements. Nevertheless, the level of ozone imissions on these two days is estimated by data of Austrian Federal Environment Agency (UMWELTBUNDESAMT, 2006):

Ozone concentrations		max. MV1 ([µg/m³])	max. MV8 ([µg/m³])	Changes (28-07 - 16-06)
Enzenkirchen	16-06-06	178	165	+2% / -1%
	28-07-06	182	163	
Zöbelboden	16-06-06	167	153	-8% / +7%
	28-07-06	153	163	

Table 6: Ozone concentrations in two Upper-Austrian environmental monitoring stations. Threshold values prescribed by Austrian ozone law for the maximum one hour mean value (max. MV1) are an information value of 180 µg/m³, and an alarm value of 240 µg/m³ (BGBl No. 210, 1992 in the actual version). A WHO guide line suggests a maximum eight hour mean value (max. MV8) of 120 µg/m³ for the protection of human beings (World Health Organization (WHO), 2006).

There are only little differences in ozone concentrations between the two days, the spirometry measurements were performed.

It is obvious, that most of the patients tend to overweight. Therefore body mass indices were calculated, too. Nevertheless, according to Amra et al., 2005, higher BMI

is not a risk factor for obstructive pattern in pulmonary function test and Ran et al. found a strong association of **low** BMI with COPD, possibly as a risk factor for COPD independent of smoking, and a potential predictor for COPD severity (Ran et al., 2006).

Since sample size would be too small, no additional statistical evaluations were done with the factor BMI.

5. Results

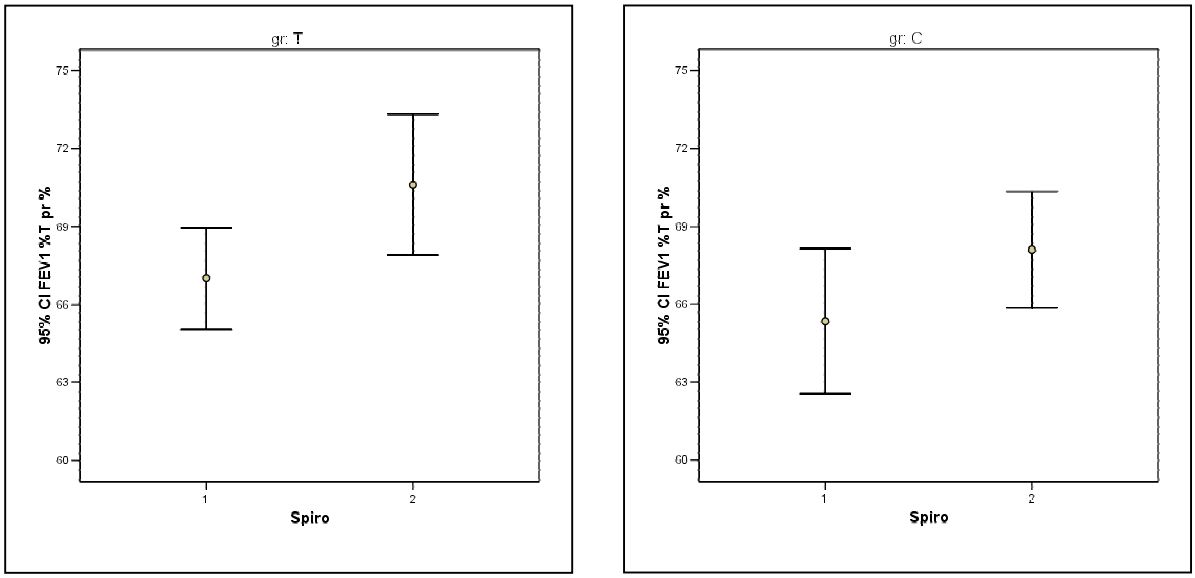
5.1. Tiffeneau Indices (FEV1/VC)

COPD has been diagnosed by a pulmonologist in advance of this study. One criterion for COPD diagnosis is a Tiffeneau index (FEV1/VC) lower than 0.7. The mean values in both groups (cf. Table 7) are lower than 0.7 and do not differ significantly.

1st spirometry	FEV1 %T pr % (Tiffeneau indices)			
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	67.01	65.34	1.105	0.143
Std. Deviation	2.72	3.91		
Min-Max.	62 - 70	59 - 70	no significant differences	
Median	67.8	66.0		

Table 7: Tiffeneau indices of both groups do not differ significantly in the first spirometry. In the control group slightly lower values can be observed.

In the second spirometry similar changes can be observed in both groups (mean values ± 95% confidence intervals cf. Ill. 12). Improvements are higher in the test group, but not statistically firm at a level of significance of 0.05.



Ill. 12: Higher Tiffeneau indices of the test persons can be observed in both groups in the second (2) spirometry compared to the first one (1). Left chart: Test group (T), right chart: Control group (C).

2nd spirometry	FEV1 %T pr % (Tiffeneau indices)			
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	70.62	68.10	1.614	0.06
Std. Deviation	3.80	3.14		
Min-Max.	65 - 76	63 - 73	no significant differences	
Median	71.7	69.1		

Table 8: In the second spirometry, one month after osteopathic treatment, Tiffeneau indices of both groups do not differ significantly. Again, in the control group slightly lower values can be observed.

No significant differences between the two groups can be observed in the second spirometry (descriptive data cf. Table 8). Again, in the control group slightly lower values can be observed.

Also the relative changes (in percent) of the Tiffeneau indices in the two groups (cf. Table 9) do not differ significantly. Nevertheless, slightly higher improvements in the test group can be observed.

Relative changes (2-1)/1	FEV1 %T pr % (Tiffeneau indices)			
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	5.62	4.69	0.230	0.41
Std. Deviation	8.19	9.78		
Min-Max.	-5.4 - 16.7	-6.4 - 24.5	no significant differences	
Median	7.3	3.8		

Table 9: Relative changes of the Tiffeneau indices are similar in both groups. No significant influence of the osteopathic treatments can be observed.

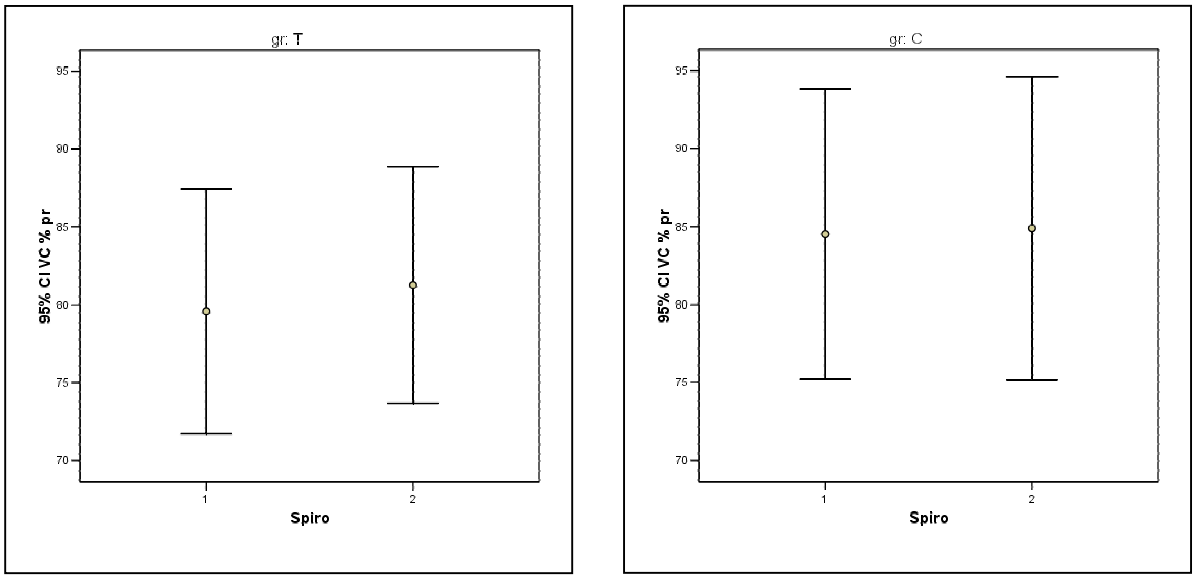
5.2. Vital Capacity (VC)

Vital capacity is another measure of spirometry and is not affected by COPD to a high extent. In order to evaluate possible cross influences on the Tiffeneau index, these data are evaluated, too.

1st spirometry	VC % pr			
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	79.57	84.51	0.917	0.19
Std. Deviation	10.96	13.02		
Min-Max.	62 - 98	59 - 100	no significant differences	
Median	80.9	87.5		

Table 10: Vital capacities in both groups do not differ significantly in the first spirometry. In the control group slightly higher values can be observed.

In the second spirometry similar changes in the two groups can be observed (mean values ± 95% confidence intervals cf. Ill. 12).



Ill. 13: Higher vital capacity of the test persons can be observed in both groups in the second (2) spirometry compared to the first one (1). Left chart: Test group (T), right chart: Control group (C).

As can be seen in this illustration, but also in Table 12, (the slight) improvements are higher in the test group, but not statistically firm at a level of significance of 0.05. Descriptive data of the second spirometry are summarised in Table 11.

2nd spirometry VC % pr				
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	81.27	84.90	0.665	0.26
Std. Deviation	10.60	13.63		
Min-Max.	63 - 98	59 - 101	no significant differences	
Median	80.4	88.3		

Table 11: In the second spirometry, one month after osteopathic treatment vital capacities of the members of both groups do not differ significantly. Again, in the control group slightly higher values can be observed.

Relative changes (2-1)/1 VC % pr				
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	2.26	0.40	-1.500	0.08
Std. Deviation	3.01	2.51		
Min-Max.	-2.8 - 8.7	-2.6 - 6.5	no significant differences	
Median	2.4	-0.2		

Table 12: In the second spirometry, one month after osteopathic treatment vital capacities of the members of both groups do not differ significantly. Slightly higher improvements can be observed in the test group.

Since spirometry is error-prone and highly dependent on the co-operation of the patients these changes are within the methodical error of spirometry.

5.3. Forced Vital Capacity (FVC)

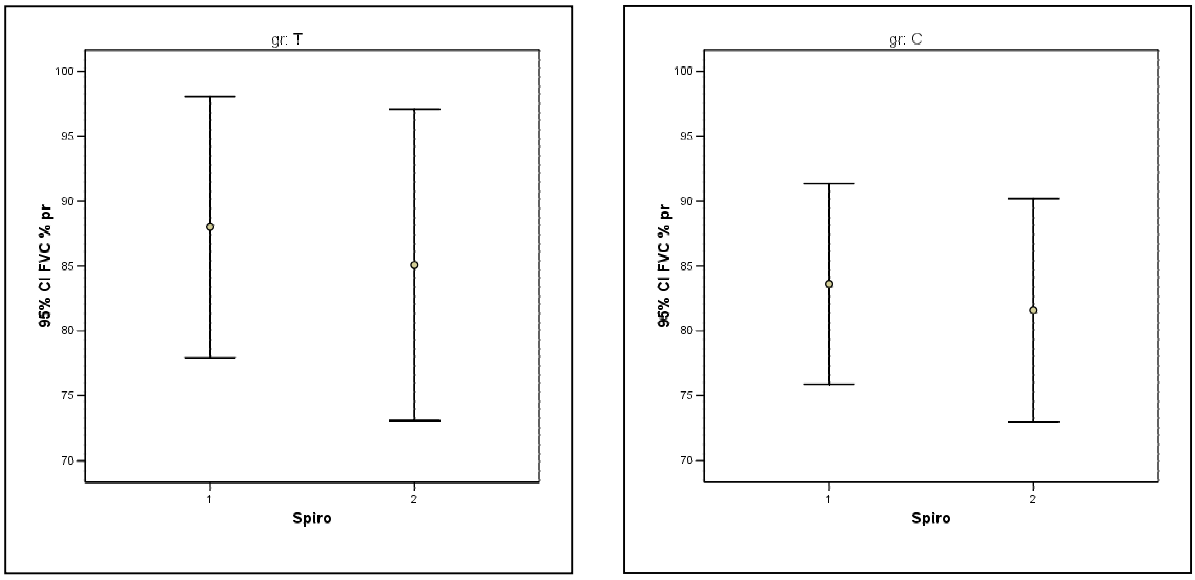
The same already said about vital capacity applies for the forced vital capacity. It is not affected by COPD to a high extent.

1st spirometry	FVC % pr			
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	88.01	83.62	0.785	0.22
Std. Deviation	14.04	10.80		
Min-Max.	70 - 106	69 - 97	no significant differences	
Median	89.6	87.3		

Table 13: Forced vital capacities in both groups do not differ significantly in the first spirometry. In the control group slightly lower values can be observed.

Statistically, there are only insignificant differences in the both groups. Before osteopathic treatment, forced vital capacity is slightly higher in the test group.

In the second spirometry similar changes in the two groups can be observed (mean values ± 95% confidence intervals cf. Ill. 12).



Ill. 14: A lower forced vital capacity of the test persons can be observed in both groups in the second (2) spirometry compared to the first one (1). Left chart: Test group (T), right chart: Control group (C).

As can be seen in Ill. 14, deterioration is slightly higher in the test group, but not statistically firm at a level of significance of 0.05 (cf. Table 15).

Thus, in the second spirometry (Table 14) no significant differences can be observed, too.

2^{nd} spirometry FVC % pr				
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	85.08	81.57	0.537	0.30
Std. Deviation	16.79	12.07		
Min-Max.	63 - 105	61 - 99	no significant differences	
Median	89.8	81.8		

Table 14: In the second spirometry, one month after osteopathic treatment forced vital capacities of the members of both groups do not differ significantly. Again, in the control group slightly lower values can be observed.

Relative changes (2-1)/1 FVC % pr				
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	-3.75	-2.38	0.422	0.34
Std. Deviation	6.55	7.98		
Min-Max.	-15.3 - 6.3	-19.9 - 5.2	no significant differences	
Median	-1.7	0.2		

Table 15: Relative chances in the forced vital capacity are higher in the test group compared to the control group, but differences are not statistically significant.

5.4. Forced Expiratory Volume in One Second (FEV1)

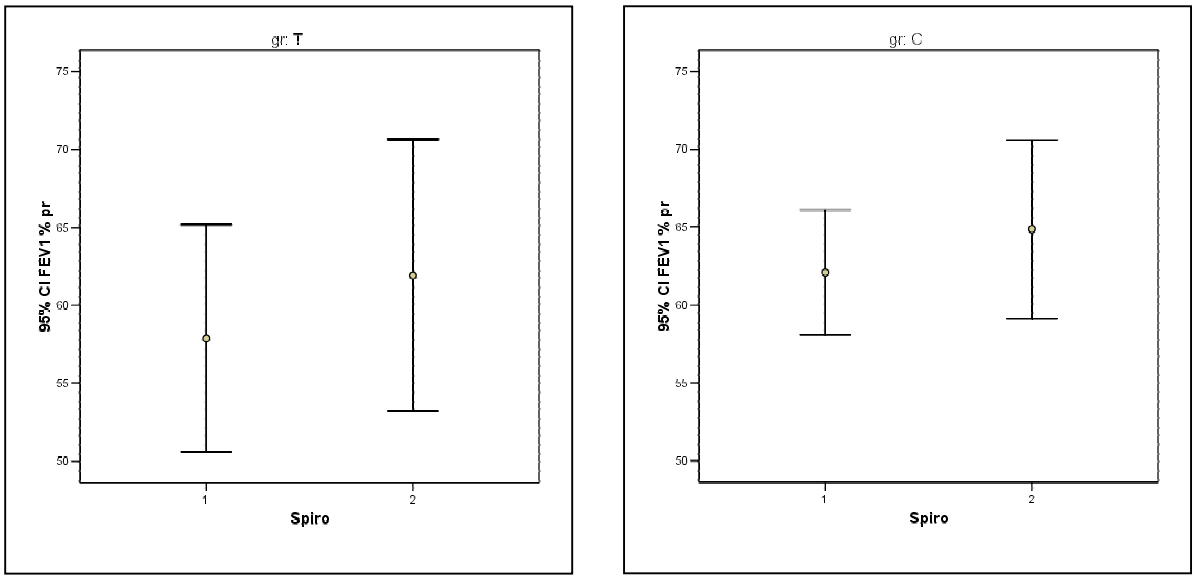
FEV1 is the parameter which is most sensitive towards COPD and a measure of the severity of this disease. Descriptive data of the first spirometry are summarized in Table 16.

1st spirometry	FEV1 % pr			
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	57.88	62.08	1.138	0.135
Std. Deviation	10.23	5.62		
Min-Max.	40 - 68	53 - 70	no significant differences	
Median	60.4	62.5		

Table 16: Forced expiratory volumes in one second of the members of the two groups do not differ significantly in the first spirometry. In the test group slightly lower values can be observed.

Forced expiratory volumes in one second of the members of the two groups do not differ significantly in the first spirometry. In the test group slightly lower values can be observed.

In the second spirometry similar changes in the two groups can be observed (mean values ± 95% confidence intervals cf. Ill. 12).



Ill. 15: Higher FEV1 values of the test persons can be observed in both groups in the second (2) spirometry compared to the first one (1). Left chart: Test group (T), right chart: Control group (C).

Improvements are higher in the test group, but not statistically firm at a level of significance of 0.05, as can be seen in Table 18. Data of the second spirometry, one month after osteopathic treatment, are listed in Table 17.

2^{nd} spirometry FEV1 % pr				
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	61.92	64.86	0.636	0.27
Std. Deviation	12.21	7.99		
Min-Max.	43 - 76	49 - 73	no significant differences	
Median	63.3	67.9		

Table 17: In the second spirometry, one month after osteopathic treatment, forced expiratory volumes in one second (FEV1) of both groups do not differ significantly. Again, in the test group slightly lower values can be observed.

Since improvements are only slightly higher in the test group, FEV1 values are still worse in this group, compared to the control group. Differences are not statistically firm.

Relative changes (2-1)/1 FEV1 % pr				
Group	Test group (T)	Control group (C)	1-tailed t-test (α=0.05)	
n	10	10	t	p
Mean	6.83	4.52	0.599	0.28
Std. Deviation	6.83	10.14		
Min-Max.	-3.3 - 16.7	-8.7 - 24.5	no significant differences	
Median	6.7	4.4		

Table 18: Relative changes of the FEV1 values are slightly higher in the test group, but again, differences are not statistically firm.

6. Discussion

6.1. Discussion of the Method

Changes of my concept

To begin with, my initial plan to gain spirometry data subsequent to the first osteopathic treatment could not be fulfilled due to lacking co-operation of the patients. Therefore, only data of one spirometry measurement after osteopathic treatment are available.

In this connection a study by Fischer et al. (2007) may show up reasons for the low cooperation, correlating the outcomes of pre-treatment interviews prior to pulmonary rehabilitation with the drop out rate. According to the authors, anticipated reasons for drop-out were identified: the intensity of the programme, barriers to attending (e.g. transportation problems, sudden illness and other duties/responsibilities), lack of improvement and social factors. Additionally, four different attitudes towards pulmonary rehabilitation could be distinguished: optimistic, 'wait and see', sceptic and pessimistic. Follow-up data revealed that whereas a pessimistic attitude (high disability, low self-confidence, many concerns) was related to decline, the 'sceptic' patients had dropped out during the course (Fischer et al, 2007).
Probably, expectations of the patients, who have had a long history of disease and treatment (with only little improvements) are low and thus cooperation, too.

Sample size

Sample size is very low (10 patients in each group) and thus may not represent the population. Generally, for statistical methods, higher sample sizes give more reliable results.

Number of treatments

The patients of the test group were treated only twice. COPD is a chronic disease and any conservative treatment has to be applied frequently. Probably, the same applies for osteopathic treatment. For example, Allen et al. (1975) describes an average reduction of severity of COPD of 10.7% by osteopathic treatment in a 9-month study.

Spirometry data

Generally, spirometry is error-prone and needs the co-operation of the patients.
As usual, data of the best of three trials were used.

Seasonal and environmental influences

Since oxidative stress is considered to have an impact on COPD, systematic studies should not be performed in summer, when environmental ozone concentrations are highest, as was done in this study. Probably, early spring, when concentrations of heating fumes are low and ozone concentrations not yet that high, would be the best season to minimise environmental influences.

Anamnesis

Additionally to my anamnesis sheet a specific list of risks for exposition to noxes would have been helpful. Nevertheless, none of the patients knew about any problems after birth (preterm delivery, immature respiratory tract, …)

Variables

The variables used, Tiffeneau index and FEV1, are the most objective measures to characterize the severity of COPD.

Since genetic predisposition seems to plays an important role in COPD, it is questionable if this disease can be cured. Other or additional variables, like a questionnaire dealing with patients' quality of life would complete the osteopathic approach and perhaps might show other influences of osteopathic treatment, like a reduction of the frequency of exacerbations.

Treatment

Osteopathic treatment was individually adjusted to the patients, according to their different dysfunctions that became obvious in anamnesis and diagnosis.

Limitations arising from the therapist

Last, but not least, osteopathic treatment was performed by only one single therapist and thus results cannot be considered as having a general application i.e. one applying to other therapists.

6.2. Discussion of the Results

No statistical significant improvement of Tiffeneau indices and FEV1 values could be achieved by osteopathic treatment compared to the control group. Improvements are only slight and within the methodical error of spirometry.

Nevertheless, results are slightly better in the test group compared to the control group.

7. Summary and Conclusions

Chronic obstructive pulmonary disease (COPD) is the fourth most frequent life-threatening disease after cardiovascular diseases, cancer and cerebrovascular diseases in western industrial countries and it is projected to be the third leading cause of death worldwide by 2020 (Schirnhofer et al., 2007). The current cost of treating COPD is already extremely high. In the USA 24 billion dollars are spent annually on the diagnosis and treatment of 16 million patients (Rufino and Lapa e Silva, 2006).

Characteristic for COPD is a progressive and almost irreversible obstruction of the air passages, leading to a chronic inhibition of the expiratory breath flow.
Most common reasons are the active as well as passive inhalation of tobacco smoke, air pollution, noxic substances in the working environment and frequent respiratory infections.

I wanted to find out, if an improvement of the state of health of COPD patients can be achieved by osteopathic treatment, basing on the main problems of COPD.
Since disturbed homeostasis seems to have a major contribution to COPD, osteopathic treatment might have positive influences on this disease.

Twenty-six patients with a doctor diagnosis of COPD by a pulmonologist in advance of this study were randomly assigned to a test- and to a control group.
Six of the patients (three of each group) did not show up for second spirometry and were excluded.
The ten test persons of the test group had spirometry in advance and a second one after two osteopathic treatments. The ten test persons of the control group had spirometry only at the same days.

During therapy, I mainly concentrated on the following aspects apart from treatment of individual other dysfunctions:

- Structural aspects of the spine, thorax and cranial base
- Fascial aspects of the thoracic cage and abdominal cage
- Aspects of the nerval supply
- Improvement of the supply- and discharge condition (blood and lymph flow)

No statistical significant improvement of Tiffeneau indices and FEV1 values could be achieved by osteopathic treatment compared to the control group. Improvements are only slight and within the methodical error of spirometry. Nevertheless, results are slightly better in the test group compared to the control group (5.6% vs. 4.7% relative to the initial Tiffeneau indices).

The main reason for this disappointing result might be the low number of treatments. The patients of the test group were treated only twice. COPD is a chronic disease and any conservative treatment has to be applied frequently. Probably, the same applies for osteopathic treatment. For example, Allen et al. (1975) describes a significant average reduction of severity of COPD of 10.7% by osteopathic spinal manipulative therapy in a 9-month study.

Thus, a long-term study might have resulted in better outcomes.

Finally, since genetic predisposition seems to plays an important role in COPD, it is questionable if this disease can be cured. Other or additional variables, like a questionnaire dealing with patients' quality of life would complete the osteopathic approach and perhaps might show other influences of osteopathic treatment, like a reduction of the frequency of exacerbations.

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9. Table of Illustrations

*Original source. Illustrations marked with an asterisk are edited by Woisetschläger G., 2007.

Ill. 8 from: http://www.thoracic.org/sections/copd/for-health-professionals/pathology-pathogenesis-and-pathophysiology/pathology/lung-parenchyma.html

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Appendix 1

Concept (German)

Konzept für Diplomarbeit im Rahmen des Universitätslehrganges Gruppe M

Andrea Grabner

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Studientyp	Kontrollierte Studie
Autor	PT Andrea Grabner
Titel	Auswirkungen der osteopathischen Behandlungen auf die Lungenfunktion bei COPD-Patienten
Stichworte	Osteopathische Behandlung; Toraxbeweglichkeit; bronchiale Reinigung; Diaphragma; Pleurabänder; Spirometrie
Einleitung	Ziel der Studie ist es herauszufinden, ob es bei COPD-Patienten unmittelbar nach der Durchführung einer ersten osteopathischen Behandlung und/oder nach einer zweiten osteopathischen Behandlung (ca. 4 Wochen nach der 1. Behandlung) zu einer Veränderung des objektiv messbaren Parameters (Ausatemkapazität innerhalb der ersten Sekunde) kommt. Dabei soll der ostheopathische Zusammenhang herausgearbeitet werden. Besonderes Augenmerk liegt dabei auf: - dem Brustkorb - der oberen HWS und Schädelbasis (N. vagus) - Th2 – Th5 (Symphatikus) - Pleurakuppelfixation - Diaphragma
Fragestellung der Untersuchung	Verändert sich unmittelbar oder im Abstand von einem Monat das Atemzugsvolumen bei COPD-Patienten nach osteopathischen Behandlungen?
Osteopathische Relevanz	Bei der Studie werden mögliche Auswirkungen der osteopathischen Behandlungen auf das Atemzugsvolumen bei COPD-Patienten objektiv erfasst.
Hypothese	Das Atemzugsvolumen wird verbessert.

Konzept für Diplomarbeit im Rahmen des Universitätslehrganges Gruppe M

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Methodologie

Geplanter Probenumfang	Es sollen 30 Patienten, welche die Einschlusskriterien erfüllen, in die Studie aufgenommen werden.
Einschlusskriterien	Leichte Pathologie dh. I: FEV1 zw. 70% und 80% der Vitalkapazität
Ausschlusskriterien	- Keine COPD-Patienten - sehr starke Pathologie dh. III. FEV1 < 70% der Vitalkapazität - Veränderung der Medikamentation während der Studie
Vorgangsplanung	- Anamnese - Osteopathische Untersuchung - Spirometrie: Nachweis der Obstruktion durch den Tiffenau-Test (1-Sekunden-Ausatem-Kapazität < 80% der Vitalkapazität) - 1. osteopathische Behandlung - Spirometrie - 2. osteopathische Behandlung (nach 4 Wochen) - Spirometrie

Variablen

Abhängige Variablen	Ausatemkapazität innerhalb der ersten Sekunde
Unabhängige Variablen	- Raucher/Nichtraucher - Allergiker - Stress / Kein Stress während der Behandlungen - Alter - Medikamentation
Validität und Verlässlichkeit der Variablen	Alle abhängigen Variablen werden mit Gold-Standards gemessen.

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Zusätzliche Informationen	Anamnese und Untersuchung der Patienten vor den Behandlungen
Geplante Dauer der Studie	- 1. Behandlung inkl. Anamnese und Befund ca. 60 Minuten - 2. Behandlung nach rd. 4 Wochen ca. 60 Minuten - insgesamt 6 Monate
Literatur	Barral J.-P., Lehrbuch der Viszeralen Osteopathie, Band 1, Urban & Fischer Verlag München – Jena 2002 Berg F., Angewandte Physiologie Drenckhahn D. / Zenker W. (Hrsg.), Benninghoff Anatomie, Band 1, 15. völlig neu bearbeitete Auflage, Urban & Schwarzenberg München-Wien-Baltimore 1994 Ewig S., Atemwegserkrankumngen bei Erwachsenen Gross R. / Schölmerich P. (Hrsg.), Lehrbuch der inneren Medizin, 6. völlig neu bearbeitete Auflage, F. K. Schattauer Verlag Stuttgart-New York 1982 Liem T., Leitfaden der Viszeralen Osteopathie Lorenz J., Pneumologie Netter F. H., Atlas der Anatomie des Menschen, 2. überarbeitete und erweiterte Auflage, Georg Thieme Verlag Stuttgart – New York 2000

Appendix 1

Concept (German)

Abbreviation		**Source**	**Formula**
Spirometry	Spirometry		
group	Group		
ID	Identification number		
sex	Sex		
age	Age		
h	Height	Measurement	
w	Weight	Measurement	
VC pr	Vital capacity (predicted)	Tabular	
FVC pr	Forced vital capacity (predicted)	Tabular	
FEV1 pr	Forced expiratory volume in one second (predicted)	Tabular	
VC act	Vital capacity (measured)	Measurement	
FVC act	Forced vital capacity (measured)	Measurement	
FEV1 act	Forced expiratory volume in one second (measured)	Measurement	
VC % pr		Calculated	VC act/VC pr
FVC % pr		Calculated	FVC act/FVC pr
FEV1 % pr		Calculated	FEV1 act/FEV1 pr
FEV1 %G pr		Calculated	FEV1 pr/FVC pr
FEV1 %G act		Calculated	FEV1 act/FVC act
FEV1 %G pr %		Calculated	FEV1 %G act/FEV1 %G pr
FEV1 %T pr		Calculated	FEV1 pr/FVC pr
FEV1 %T act		Calculated	FEV1 act/FV act
FEV1 %T pr %		Calculated	FEV1 %T act/FEV1 %T pr
BMI		Calculated	w/(h/100)^2

independent v.										dependent v.											dep. v.	
Spirometry	**group**	ID	sex	age	h	w	VC pr	FVC pr	FEV1 pr	**VC act**	**FVC act**	**FEV1 act**	VC % pr	FVC % pr	FEV1 % pr	FEV1 %G pr	FEV1 %G act	FEV1 %G pr %	FEV1 %T pr	FEV1 %T act	**FEV1 %T pr %**	BMI
1	C	1	m	43	179	104	5,22	4,7	4,4	4,7	4,11	2,7	90,00%	87,45%	62,00%	92,66%	65,69%	70,90%	92,66%	57,45%	62,00%	32,46
1	C	3	m	51	177	106	5,41	4,76	4	4,76	4,15	2,52	88,00%	87,18%	63,00%	84,03%	60,72%	72,26%	84,03%	52,94%	63,00%	33,83
1	C	4	m	30	172	76	4,99	3,64	2,7	3,64	3,23	1,82	73,00%	88,74%	67,00%	74,63%	56,35%	75,50%	74,63%	50,00%	67,00%	25,69
1	C	5	m	52	187	117	5,58	3,29	3,2	3,29	2,4	1,98	59,00%	72,95%	61,00%	98,66%	82,50%	83,62%	98,66%	60,18%	61,00%	33,46
1	C	6	f	42	167	60	3,82	3,63	2,5	3,63	2,5	1,62	95,00%	68,87%	65,00%	68,66%	64,80%	94,38%	68,66%	44,63%	65,00%	21,51
1	C	7	m	37	183	117	5,7	5,7	4,7	5,71	5,5	3,31	100,18%	96,49%	69,83%	83,16%	60,18%	72,37%	83,16%	57,97%	69,71%	34,94
1	C	8	m	67	181	99	4,49	4,49	3,6	3,39	3,39	1,89	75,50%	75,50%	52,65%	79,96%	55,75%	69,73%	79,96%	55,75%	69,73%	30,22
1	C	9	f	65	165	83	2,98	2,98	2,5	2,33	2,09	1,33	78,19%	70,13%	54,07%	82,55%	63,64%	77,09%	82,55%	57,08%	69,15%	30,49
1	C	9	f	52	159	70	3,2	2,78	2,7	2,78	2,54	1,59	87,00%	91,37%	59,00%	96,94%	62,60%	64,57%	96,94%	57,19%	59,00%	27,69
1	C	10	m	44	178	112	5,15	5,15	4,3	5,11	5,02	2,88	99,22%	97,48%	67,29%	83,11%	57,37%	69,03%	83,11%	56,36%	67,82%	35,35
1	**T**	**1**	f	56	163	81	3,25	3,25	2,7	2,02	2,29	1,07	62,15%	70,46%	39,63%	83,08%	46,72%	56,24%	83,08%	52,97%	63,76%	30,49
1	**T**	**2**	f	42	170	68	4,02	4,02	3,4	3,33	3,11	1,95	82,84%	77,36%	57,52%	84,33%	62,70%	74,35%	84,33%	58,56%	69,44%	23,53
1	**T**	**2**	f	56	161	74	3,14	2,48	2,6	2,48	2,24	1,62	79,00%	90,32%	61,61%	106,03%	72,32%	68,21%	106,03%	65,32%	61,61%	28,55
1	**T**	**3**	f	45	165	66	3,75	3,75	3,2	2,72	3,33	1,59	72,53%	88,80%	50,16%	84,53%	47,75%	56,48%	84,53%	58,46%	69,15%	24,24
1	**T**	**4**	m	41	178	143	5,25	5,25	4,4	3,38	3,68	1,9	64,38%	70,10%	43,48%	83,24%	51,63%	62,03%	83,24%	56,21%	67,53%	45,13
1	**T**	**5**	f	43	163	55	3,65	3,65	3,1	3,58	3,5	2,08	98,08%	95,89%	67,31%	84,66%	59,43%	70,20%	84,66%	58,10%	68,63%	20,7
1	**T**	**6**	m	64	170	85	3,95	3,95	3,2	3,34	2,95	1,91	84,56%	74,68%	59,13%	81,77%	64,75%	79,18%	81,77%	57,19%	69,93%	29,41
1	**T**	**7**	m	56	176	94	4,62	3,6	3,8	3,6	3,74	2,53	78,00%	103,89%	67,00%	104,89%	67,65%	64,49%	104,89%	70,28%	67,00%	30,35
1	**T**	**8**	m	47	169	77	4,44	3,82	3,8	3,82	4,03	2,55	86,00%	105,50%	68,00%	98,17%	63,28%	64,46%	98,17%	66,75%	68,00%	26,96
1	**T**	**10**	m	59	180	116	4,75	4,19	3,8	4,19	4,32	2,5	88,20%	103,10%	65,00%	91,79%	57,87%	63,04%	91,79%	59,67%	65,00%	35,8
2	C	1	m	43	179	104	5,22	4,68	4,4	4,68	4,11	2,91	89,62%	87,82%	66,82%	93,05%	70,80%	76,09%	93,05%	62,18%	66,82%	32,46
2	C	3	m	51	177	106	5,41	4,7	4	4,7	4,31	2,77	86,89%	91,70%	69,25%	85,11%	64,27%	75,52%	85,11%	58,94%	69,25%	33,83
2	C	4	m	30	172	76	4,99	3,59	2,7	3,59	3,07	1,75	72,00%	85,52%	64,42%	75,67%	57,00%	75,34%	75,67%	48,75%	64,42%	25,69
2	C	5	m	52	187	117	5,58	3,31	3,2	3,31	2,51	2,25	59,36%	75,83%	69,32%	98,06%	89,64%	91,41%	98,06%	67,98%	69,32%	33,46
2	C	6	f	42	167	60	3,82	3,7	2,5	3,7	2,48	1,72	96,83%	67,03%	69,01%	67,36%	69,35%	102,96%	67,36%	46,49%	69,01%	21,51
2	C	7	m	37	183	117	5,74	5,74	4,8	5,82	5,68	3,41	101,39%	98,95%	71,64%	82,93%	60,04%	72,40%	82,93%	58,59%	70,65%	34,94
2	C	8	m	67	181	99	4,49	4,49	3,6	3,39	3,39	1,89	75,50%	75,50%	52,65%	79,96%	55,75%	69,73%	79,96%	55,75%	69,73%	30,22
2	C	9	f	65	165	83	2,98	2,98	2,5	2,27	1,83	1,21	76,17%	61,41%	49,39%	82,21%	66,12%	80,42%	82,21%	53,30%	64,84%	30,49
2	C	9	f	52	159	70	3,2	2,96	2,7	2,96	2,78	1,98	92,63%	93,92%	73,47%	91,04%	71,22%	78,23%	91,04%	66,89%	73,47%	27,69
2	C	10	m	44	178	112	5,15	5,15	4,3	5,08	4,02	2,68	98,64%	78,06%	62,62%	83,11%	66,67%	80,22%	83,11%	52,76%	63,48%	35,35
2	**T**	**1**	f	56	163	82	3,36	3,36	2,8	2,1	2,29	1,25	62,50%	68,15%	44,64%	83,33%	54,59%	65,50%	83,33%	59,52%	71,43%	30,86
2	**T**	**2**	f	42	170	67	4,1	4,1	3,5	3,3	2,72	2,06	80,49%	66,34%	59,37%	84,63%	75,74%	89,49%	84,63%	62,42%	73,76%	23,18
2	**T**	**2**	f	56	161	74	3,14	2,51	2,6	2,51	2,41	1,89	79,96%	96,02%	71,87%	104,77%	78,42%	74,86%	104,77%	75,30%	71,87%	28,55
2	**T**	**3**	f	45	165	66	3,69	3,69	3,1	2,91	3,09	1,64	78,86%	83,74%	52,73%	84,28%	53,07%	62,97%	84,28%	56,36%	66,87%	24,24
2	**T**	**4**	m	41	178	143	5,31	5,31	4,4	3,56	3,68	1,92	67,04%	69,30%	43,44%	83,24%	52,17%	62,68%	83,24%	53,93%	64,79%	45,13
2	**T**	**5**	f	43	163	55	3,65	3,65	3,1	3,58	3,5	2,08	98,08%	95,89%	67,31%	84,66%	59,43%	70,20%	84,66%	58,10%	68,63%	20,7
2	**T**	**6**	m	64	170	85	3,84	3,84	3,1	3,32	2,43	1,79	86,46%	63,28%	57,19%	81,51%	73,66%	90,37%	81,51%	53,92%	66,15%	29,41
2	**T**	**7**	m	56	176	94	4,62	3,71	3,8	3,71	3,78	2,74	80,38%	101,89%	72,56%	101,78%	72,49%	71,22%	101,78%	73,85%	72,56%	30,35
2	**T**	**8**	m	47	169	77	4,44	3,92	3,8	3,92	4,1	2,86	88,25%	104,59%	76,27%	95,66%	69,76%	72,92%	95,66%	72,96%	76,27%	26,96
2	**T**	**10**	m	59	180	116	4,75	4,31	3,8	4,31	4,38	2,84	90,73%	101,62%	73,84%	89,24%	64,84%	72,66%	89,24%	65,89%	73,84%	35,8

Appendix 3
Medical History Sheets

Anamnese

Name: Geb.Dat.:
Adresse: Telefon:

Beruf: Familienstand:

Aktuelles Problem:

Lokalisation:
Seit wann?
Qualität:
Wodurch wird es besser?

Ursache: mechanisch /toxisch /entzündlich

Erblich bedingte Einflüsse:

Einflüsse während der Schwangerschaft:

Anzahl und Verlauf der vorherigen Schwangerschaften:

Geburtsvorgang:

Erscheinung und Verhalten des Neugeborenen:
(asym.Schädelform)

Funktionsstörungen:
- abnormes Schreien
- Unfähigkeit zu saugen
- Schluckstörungen
- Augenstörungen
- Opisthotonus
- Spastik oder Lähmung
- Konvulsionen

Entwicklung des Kindes:

Schwere Krankheiten in der Kindheit:

Vergangene med. Geschichte:

(Trauma ,OP
schwere und /oder chron.Erkrankungen
Zähne
Regulierung
Impfung)

Medikamente:

Ernährung:

Trinken: allgem.
Alkohol
Kaffee

Sportliche Aktivitäten:

Schlaf: (Müdigkeit Position):

Harndrang / Verdauung:

Zyklus: (Pille,Spirale)

Geburt:

Psyche:

Herz-Kreislauf System:

RR ,Schwindel, kalte Hände/Füße
Atemnot, Claudicatio, Schwellung

Atmungssystem:

Infektionen, Husten, Atemnot, Astma

Gastrointestinal-Trakt:

Magen (Atemnot ,Husten, Acidose, Aufstoßen, Gürtel auf)

Leber + Gallenblase
(diffuse Muskelprobleme, Krämpfe, Verstopfung, Blähung;
Müdigkeit, Depression, hormonelles Ungleichgewicht, Süß)

Duodenum + Pankreas, Empfindlichkeit Nabel
Gürtelschmerz,Beschw.3-4 h, nach dem Essen

Darm

Urogenitaltrakt:

Niere (RR, Sturz Coccygis,
Infektion; Ödeme)

Blase (Infekte; Schmerzen,
Senkung, Inkontinenz

Prostata

Uterus (Mens., Schmerzen vor/während)

Autointoxikation:

Migräne, schlechter Schlaf,
Müdigkeit, Konzentration,
Juckreiz, Schwindel,
Kopfschmerz, Photosens.,
Mundgeruch, Zunge belegt

Atembefund
Therapeut: .....
Datum: ....
Adressette oder Name: .....
Station/Zimmer:.....
Alter:.....
Diagnose:.....
Nebendiagnose:.....
allgemeine Belastbarkeit (nach ärztlicher Vorgabe):
 im Bett
 Gehen in der Ebene
 Sitz an der Bettkante
 Treppe
Stand
 Gehen im Zimmer
max. Blutdruck:.....
max. Puls: .....
 blutdruck-/pulssenkendes Medikament
 ß-Sympathomimetikum
 sonstige Medikamente .....

1. Activity of Daily Life (ADL)
Hilfsmittel:
 zusätzliche Gabe von Sauerstoff
 Handstock
 Unterarmgehstützen
 Rollator
 Rollstuhl
 sonstige Hilfsmittel: .....

	kann/darf nicht ausgeführt werden	1 (völlige Unselb-ständigkeit)	2	3	4	5	6	7 (völlige Selb-sändigkeit)
Transfer RUSL								
Transfer SL(RL)/Sitz								
Schuhe anziehen								
Transfer Sitz/Stand								
kleine Gegenstände aufheben								
Treppe steigen								

Timed up and 90 (aufstehen, 3 m gehen, drehen, zurückgehen und setzen): .....

2. Thoraxverhältnisse (in die Skizzen einzeichnen)

a Atembewegung ---
b Gewebetonus ///
c Schmerz *
d Besonderheiten

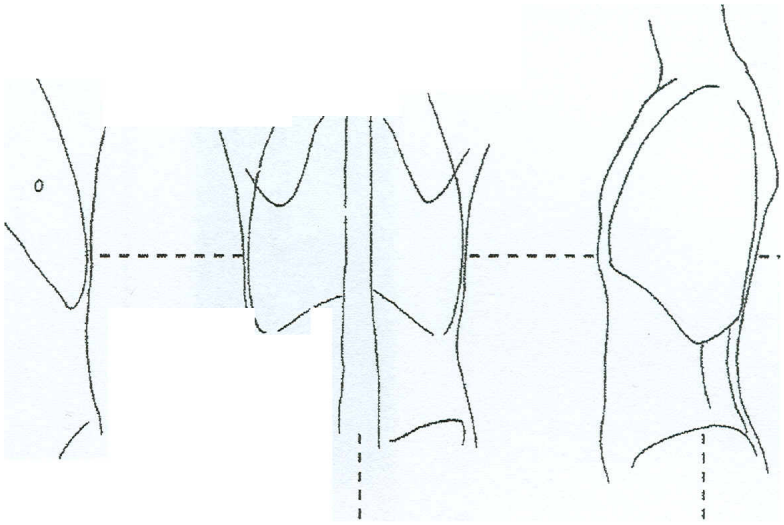
von vorne **von hinten** **von der Seite**

rechts lateral links lateral links latera1 rechts lateral ventral dorsal

costal

costo-

abdominal



	0 (kein Schmerz	1	2	3	4	5	6	7	8	9	10 (max. Schmerz)
subj. Schmerz-empfindung											

Schmerzqualität: .....

3. Atemform in Ruhe

Atemfrequenz: ..... /min

Atemweg: .....

Atemrhythmus:

□ Fehlen der Atempause

□ Länge EA = Länge AA

□ unregelmäßig

□ EA verlängert

□ häufiges Seufzen

□ AA verlängert

Atemnebengeräusche:

□ rau hörbar

□ Rasseln

□ Giemen

4. spontanes Husten

□ produktiv

□ unproduktiv

□ Räuspern

Sekret:

□ sehr zäh

□ zäh

□ lässt sich leicht abhusten

□ rot/braun

□ gelb/grün

□ klar/weiß

Muskelaktivität beim Husten:

□ zu schwach

□ angemessen

□ zu stark

sonstige Besonderheiten des Hustenverhaltens:.....

5. Einsatz der Atemhilfsmuskeln in Ruhe

	kein	mäßig	stark
Bauchmuskulatur			
Intercostalmuskulatur			
M. pectalis major			
M. sternocleidomastoideus			

6. Beweglichkeit
Einschränkung: ↓, ↓↓, ↓↓↓
Schmerz: *
Bewegungsausmaß ohne Befund: ˇ
kann/darf nicht ausgeführt werden

Beweglichkeit der Wirbelsäule

	FLEX	EXT	LAT FLEX		ROT	
			li konk	re konkav	pos.	neg.
HWS						
BWS						
LWS						

Beweglichkeit der Schulter

	rechts	links
hand behind neck		
hand behind back		

7. Muskeltonus

	hypoton		normoton		hyperton		schmerzhaft hyperton	
	re	li	re	li	re	li	re	li
M. trapezius	□	□	□	□	□	□	□	□
M. pectoralis	□	□	□	□	□	□	□	□
Bauchmuskeln	□	□	□	□	□	□	□	□
autochthone Rückenmuskeln	□	□	□	□	□	□	□	□

8. Belastbarkeit

6-min-Gehen in der Ebene .....m mit .....5 Pause

Puls in Ruhe: ..... Puls nach Belastung: .....

Blutdruck in Ruhe: ..... Blutdruck nach Belastung: .....

Atemfrequenz in Ruhe: ..... Blutdruck nach Belastung: .....