

## **Master's Thesis:**

Can intraocular pressure be influenced  
by osteopathic treatment?

Thesis to attain the Master of Science degree  
in osteopathy

at the **Donau University of Krems**

submitted  
at the **School for Osteopathy, Vienna**

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Davos, September 2009

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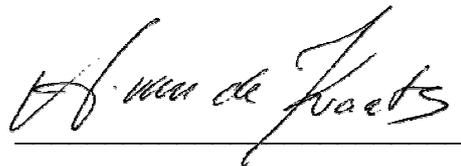
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A handwritten signature in black ink, appearing to read 'A. van de Ven', written over a horizontal line.

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## ABSTRACT

This single-blind, randomized, clinical study seeks to determine if intraocular pressure can be influenced with two osteopathic interventions. The goal is to decrease intraocular pressure. An experimental group (25 test subjects) and a control group (20 subjects) were selected according to inclusion/exclusion criteria. The experimental group was treated twice osteopathically according to the Lien Mecanique Osteopathique (LMO) concept developed by Paul Chauffour and Eric Pratt (1978). The control group was measured but not treated in the same timeframe. An average decrease in median intraocular pressure of 2.5% resulted within the experimental group over the total observation period. At the same time, the eyes were affected to a sharply varying degree: while a lesser decrease of intraocular pressure (0.5%) was recorded in the right eye, it decreased by 4.6% in the left eye. It is also noticeable that intraocular pressure decreased directly after treatments, while it had recurred before the second treatment. The average level of change in intraocular pressure in the control group registered a lower mean in both eyes than in the experimental group: a decrease in the mean intraocular pressure of 0.4%. The issue and the hypothesis have only been conditionally confirmed. Intraocular pressure is influenced by osteopathic intervention, even if not to a significant degree. Further studies in this area are recommended in which one clearly lengthens the duration of the study and increases the number of treatments.

Eight study subjects (32%) averaged a higher intraocular pressure after the **first treatment** than before it, five maintained the same intraocular pressure (20%), and 12 achieved a reduced intraocular pressure (48%).

After the **second therapy** unit's treatment, four test subjects (16%) indicated an average higher intraocular pressure than before treatment. The intraocular pressure of nine others remained the same (36%), while 12 subjects achieved a lower intraocular pressure (48%).

The issue and the hypothesis have only been confirmed conditionally. The intraocular pressure was influenced by osteopathic intervention even if not to a significant degree. Other studies on this topic are recommended, but one should lengthen the duration of the study and increase the number of treatments.

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## **1 PREFACE**

During autumn of 2008 I registered at the School of Osteopathy in Vienna for master-of-science training. A master's thesis must be written to attain this title. The topic and cornerstone of this work were set through referral of a patient with increased intraocular pressure by a local ophthalmologist. The ophthalmologist's question was: Can osteopathic treatment influence increased intraocular pressure? Consequently the idea of this topic took root, and the physician agreed to collaborate in the study.

## **2 INTRODUCTION**

There have been few osteopathic studies to date that assess the potential of influencing intraocular pressure. The issue concerns either case examples or clinical pilot studies without control groups (Esser, 2002) or tasks resulting from a sequence of osteopathic techniques (Bilgeri, 2006).

In recent years the funding authorities for in- and outpatient services in the healthcare sector have required increased evidence concerning the efficiency of applied treatment methods. Particularly treatments in complementary medicine sectors must fulfill such a task. Osteopathy is no exception to this rule. I myself have been confronted with these issues repeatedly in my everyday practice: How can the osteopathic method be described in standard terms? How can the effectiveness and reliability of results be measured?

## **3 GOAL**

The study's goal is to examine if an osteopathic treatment approach can lower intraocular pressure.

The patients were only treated with medicines previously. The goal here lies in establishing if it is possible to bring about a decrease in intraocular pressure with the osteopathic treatments described in further detail under 6.8. A further goal is to review and confirm what earlier osteopathic studies have measured and determined (Esser, 2002; Bigeri, 2006).

## 4 CURRENT LITERATURE ON INCREASED INTRAOCULAR PRESSURE

There are more than 66 million glaucoma patients worldwide, and blinding of both eyes occurs in at least 6.8 million people as a result of glaucoma. Thus this ailment has become the second most common cause for blindness in the developing world (Weinreb, 2004). Glaucomas are a group of eye diseases that, if not recognized and treated early, can lead to destruction of the optic nerve. Chronic glaucoma usually occurs without symptoms (Scheible, *et al.*, 2009). On the other hand, since glaucoma can be treated effectively but the illness causes irreversible blindness, early detection has gained crucial importance. However, studies of the general population have shown regularly that up to 50% of glaucoma cases remain undiagnosed.

The clear connection with age shows that the probability of glaucoma illness occurring doubles with each decade of life starting at age 60. The increase in frequency of glaucoma illness for 2030 is estimated at 34.1% (Michelson, *et al.*, 2008).

Though the intraocular pressure values of 20 - 60% of patients with glaucomatized ocular nerves and field-of-vision damage lie beneath 21 mm Hg (mercury), between 40-80% still remain for whom intraocular pressure is increased (Gerste, 2008). Intraocular pressure measurement is comparable to blood pressure measurement: increased values do not necessarily mean that an ailment is present. It only means that the intraocular pressure measurement should be carried out more often and regularly in order to detect and avoid the emergence of more severe illnesses (*e.g.*, glaucoma) early enough.

One research team from the USA and Switzerland pointed out in this context that a compartmentalizing of the subarachnoid space within the optic nerve as well as a disturbed and inflamed change in composition of the fluid can be responsible for altering the optic nerve pathologically (Gerste, 2008).

### 4.1 OSTEOPATHIC LITERATURE, CITATIONS

Short introduction into the world of osteopathic thought

*Osteopathy is a „symmetrical, harmonious, technical accompaniment of the total living mechanism in which all communications with the brain remain open and all hindrances in the circulation of blood and other fluids are overcome.“*

*„An osteopath must master the anatomy and physiology and possess a very good knowledge of chemistry.“*

*„The point which I want you to observe is that you must know the precise structure of the human body, the precise location of each bone, nerve, each fiber, each muscle and organ, their origin, the course and flow of all fluids in the body, their relationship to each other, and the function they play in maintaining life and health.“*

*(Still, A.T., 1886, 159)*

#### **4.1.1 A.T. Still**

It was important for A.T. Still to consider the upper region of the thoracic spine more precisely. He placed great value on examining all vertebral joints, the upper ribs, as well as the collarbone. A further concern for him was to optimize the blood and nerve supply, especially in the upper thorax area. From A. T. Still's point of view, the causes of eye diseases usually do not lie in the eye itself but result from an suboptimally functioning cervical vertebral column or disturbances in the upper dorsal spine area (Still, 1896, 477-507).

#### **4.1.2 Cipolla, 1975**

There were four studies between 1975-1984 conducted from an osteopathic viewpoint. The oldest study by Cipolla *et al.* took place in 1975. Cipolla made a preliminary study. A group of 20 people was involved in the study, 10 of whom were treated once with osteopathic, manipulative cervico, myofacial techniques. The measurements concerning intraocular pressure occurred before and directly after the treatment as well as four hours later. The experimental group showed a clearer reduction in intraocular pressure than the control group. Unfortunately no long-term measurements were carried out in this study. Hence the statement rested on a single treatment. According to current scientific standards, the study is unfortunately not convincing. At most it could be determined that osteopathic treatment has a short-term influence on intraocular pressure.

#### **4.1.3 Misischia, 1981**

In 1981 Misischia tried to see how osteopathic techniques influence intraocular pressure and to measure it. Misischia also took a measurement before the treatment, directly afterward, and a final measurement 60 minutes later. His treatment lasted 10

minutes and was limited to the cervical region. Immediately after the manipulation the intraocular pressure sank about 2-4 mm Hg. Then, 60 minutes after the treatment, its decrease amounts to 3-5 mm Hg. He assumed a connection between the reduction in pressure and influence on the autonomic nerve system.

#### **4.1.4 Feely, 1982**

Feely *et al* developed another interesting theory in 1982. They put together the hypothesis that one could influence intraocular pressure with treatment of the cervical vertebral column and the upper thorax region. To test this thesis they selected people with chronic open-angle glaucoma and tested them against people without this ailment, forming an experimental group as well as five control groups. The experimental group was treated with manipulative osteopathic techniques at the cervical vertebral column and thorax region. One control group was not treated with manipulation, while the second one was treated manipulatively in the lower thoracic spine and the passage to the lumbar vertebral column. The third control group, which had no glaucoma ailments, was treated the same as the experimental group. The fourth control group, also disease free, was not treated manipulatively. The fifth control group of non-diseased people was treated the same as the second control group affected. Measurements occurred before and after treatment at intervals of 5, 10, 20, 30, and 60 minutes. The measurements resulted in a decrease of 3-7 mm Hg – especially in the group affected with glaucoma. No long-term measurements were made in this study either. Unfortunately the treatments here as well were regionally limited and do not correspond entirely to the osteopathic concept. With too many control groups comprised of too few test subjects, the significance also decreases.

#### **4.1.5 Fowler, 1984**

Fowler's interest applied to the sympathetic nerve system. His motivation in this study was the effect of treating the lower cervical spine and the passage to the thoracic spine with osteopathic manipulations, thus influencing the sympathetic nerve system and hence intraocular pressure. He limited the study to test subjects who had a somatic dysfunction of C8-Th2. The experimental group was treated in the cervicothoracic passage region; the first control group was treated in the thoracolumbar passage region, and the second control group was not treated. This study too was measured immediately before and after intraocular pressure treatments at intervals of 5, 15, and 30 minutes. In the case of the group treated, measurements after 15

minutes indicated a significant increase of intraocular pressure. There was no clear difference between the groups that had only been treated at the cervical vertebral columns or their passages. Fowler's conclusion was that one should not limit treatment to single regions of the vertebral columns. He also suggested that one should not limit the regions to be treated. Fowler's second finding was that measurements after treatment should be carried out over a prolonged period of time.

#### **4.1.6 Esser, 2000**

His hypothesis asked: „Can a decrease in intraocular pressure be affected by an osteopathic technique in case of primary chronic open-angle glaucoma?“ Esser's study concerned a clinical pilot study that was carried out without control groups.

Some 25 patients were recruited. These patients had a verified primary open-angle glaucoma in which intraocular pressure did not exceed 30 mm Hg. Esser selected seven osteopathic techniques, which were applied with all patients in the same manner. The Verum group was treated three times a week, and its values were measured. The results found that intraocular pressure had decreased in the intermediate term. The P value ( $p < 0.001$ ) produced confirmed the significance in terms of an intraocular pressure decrease with osteopathic techniques.

#### **4.1.7 Bilgeri, 2006**

Bilgeri had a total of 20 patients available, who she distributed into two groups by means of a randomization list. It resulted in an experimental group and a control group. The experimental group was treated according to a holistic osteopathic method. All patients were treated according to the same criteria and given the same course of treatment. Bilgeri's study was able to show that an osteopathic treatment had a positive effect on intraocular pressure.

## **5 BASES OF THE THESIS**

The eye as an optical analysist absorbs more than 80% of all the human being's information. Human ability to imagine and recall as well as one's thought process and part of one's sense of fantasy touch ultimately on what one sees (Sachsenweger, M., 2003, 1-3). Possessing the ability to see means being self-sufficient in life. Earlier one's survival depended upon it. Today it's the basis for freedom of movement in the entire world and the related quality of life. Being blind is an especially severe form of

suffering for many people. Thus physicians and therapists should be constantly aware of how vital eyesight is for each one of us (Lang, 2004, II).

## 5.1 ANATOMY OF THE EYE

An eye measures about 24 mm in size and roughly weighs 7.5 g. The eyeball (*Bulbus oculi*) lies well imbedded in the boney eye socket (*orbita*). The eyeball has the form of a ball. At its front, like a watch glass, lies the translucent cornea. It has a greater curvature index than the rest of the eyeball. The axis of the eye, *Axis bulbi*, links the frontal and rear poles of the eye. Medially from the rear pole, the visual nerve (*Nervus Opticus*) leaves the *Bulbus*. Laterally from it lies the *Fovea retina*, the location of sharpest vision. The visual axis (*Axis opticus*) goes through the *Fovea centralis*, which binds together the means of curvature pointing to the boundary surfaces breaking media lying in the ray path (frontal and rear cornea and lens surface). The equator of the eyeballs (*Aequator bulbi*) refers to the largest diameter of the eyeball; it divides the *Bulbus* into front and rear hemispheres of nearly the same size (Ugli, 2004, 4-7).

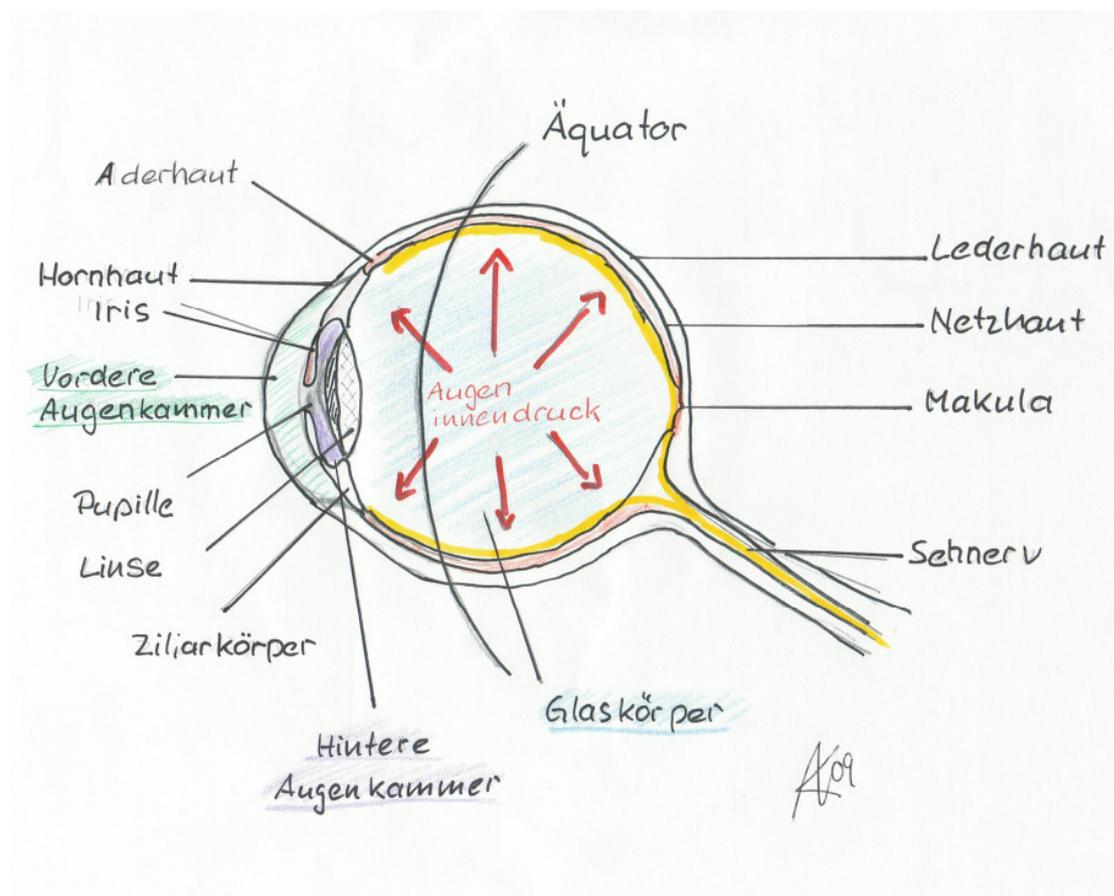


Figure 5.1: Structure of the eye

*Tunica fibrosa bulbi*, exterior membrane, with two sections

- a) *Sclera*, white, hard membrane [*protective covering*]
- b) *Cornea*

*Tunica vasculosa bulbi*, middle membrane uveal tract. This consists of the:

- a) Black layer circulation (*Coroidea*) [*blood supply of the retina*]
- b) Ciliary body (*Corpus ciliare*) [*accommodation, production of aqueous humor*]
- c) Iris [*regulation of light incidence*]
- d) Uvea

*Tunica interna bulbi*, inner eyeball membrane, retina. This consists of the:

- a) *Pars optica*
- b) *Pars ciliaris retinae*
- c) *Pars iridica retinae*
- d) *Pars ciliaris* and *Pars iridica* together comprise the *Pars caeac retinae*

The *Bulbus oculi* exhibit the following inner areas:

- a) Front chamber of the eye, *Camera bulbi anterior*, in front of the iris
- b) Rear chamber of the eye, *Camera bulbi posterior*, behind the iris
- c) Vitreous body area with the vitreous body, *Corpus vitreum*.

**The lens** hangs from zonula fibers in the rear eyeball membrane behind the iris.

**The cornea**, lens, vitreous body base, content of the eye chambers, and aqueous humor (*Humor aquosus*) comprise the eyes' optical media.

**The musculature** of the lens, ciliary body, fracture membrane, and iris enable short- and long-distance sight and form the accommodation apparatus (Schmidt, 1999).

The **optic nerve** (*Nervus opticus, Fasciculus opticus*) is an extracranial pathway emanating in the brain. The *Nervus opticus* contains about one million nerve fibers, as well as existing *septae* from connective tissue (Moore, 1996).

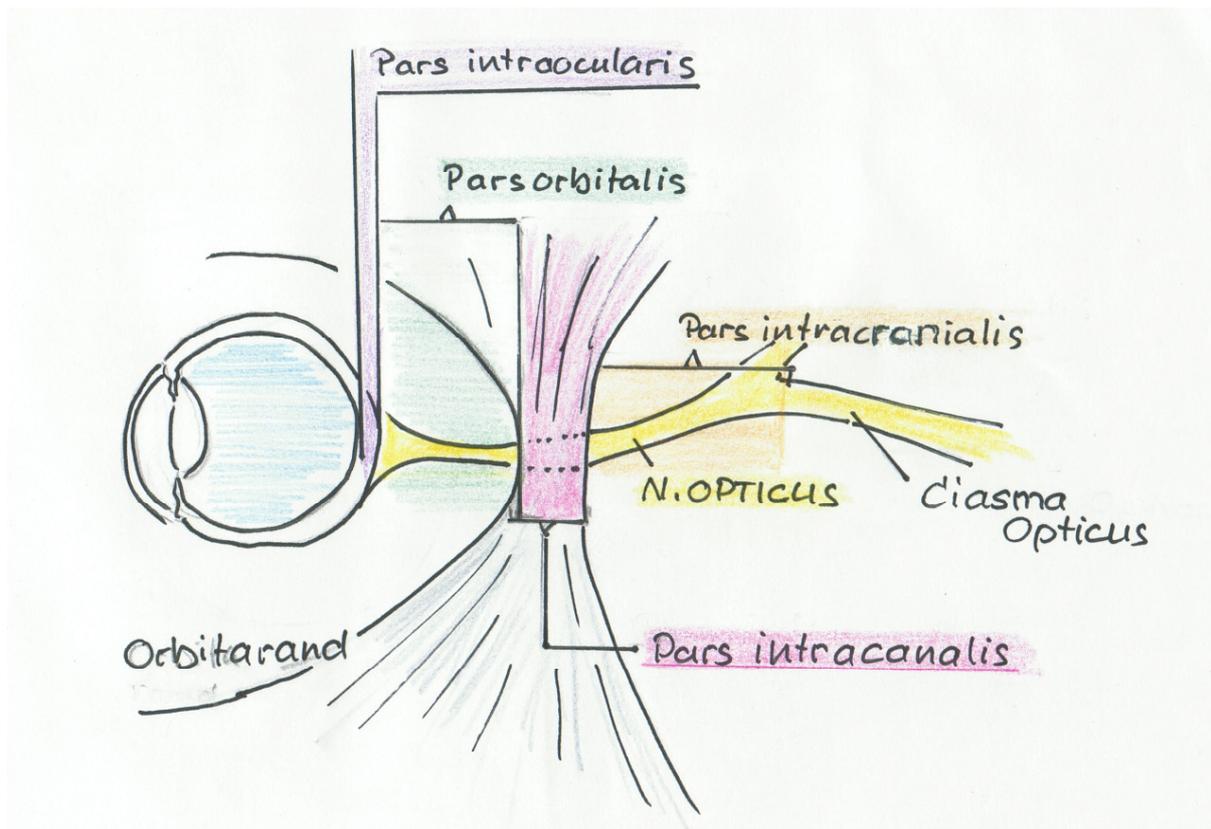
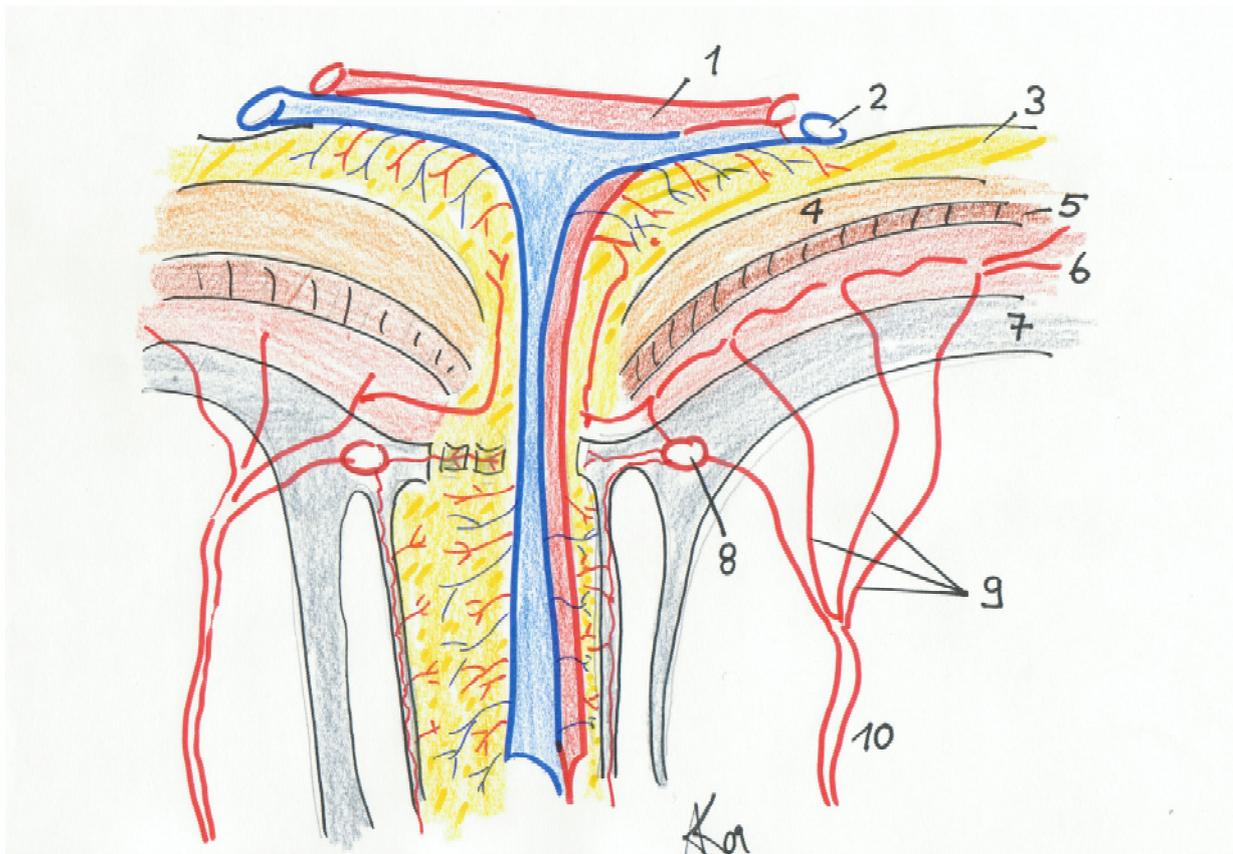


Figure 5.2: Course of the N. Opticus

The optic nerve has several sections. After leaving the retina at the optic disc and passing through the cribriform plate (*Pars intraocularis*), the optic nerve fibers move in an s-forming pattern into the Orbita (*Pars orbitalis*). They pass through the *Canalis opticus* (*Pars intracanalalis*), where they are especially endangered, into the brain cavity (*Pars intracranialis*), and then to the *Chiasma opticum*. *Pars intracanalalis* and *Pars intracranialis* together have a length of about 18 mm. The rear section of the *N. Opticus* is enclosed within the fork tongs of the *A. Carotis interna* and the *A. Cerebri anterior* (Ugi, 2004, 239-246). *Dura* and *Pia mater* as well as *Archnoidea* surround the optic nerve. Yet the subarachnoidal area contains no liquid. Outside the eyeball the optic nerve fibers are wrapped in medullary sheaths (neuroglia) (Sachsenweger, 2004, 302-322).

The optic disk supplies the optic nerve's **blood supply** through the Zinn-Haller vessel corina, outside via vessels of the *Pia mater* and the *A. centralis retinae*. They originate in the *A. Ophthalmica*, enter the optic nerve behind the *Bulbus* and provide it with blood (Ugi, 2004. Sachsenweger, 2004, 49-53).



**Figure 5.3:** The optic nerve's blood supply

- |                                   |                                     |
|-----------------------------------|-------------------------------------|
| 1. A. centralis retinae           | 7. Dura                             |
| 2. V. centralis retinae           | 8. Zinni-Haller- vessel corina      |
| 3. Layer of nerve fiber           | 9. Aa. Cilliares posteriores breves |
| 4. Retina                         | 10. A. ciliaris posterior           |
| 5. Retina's pigmentary epithelium | 11. Arachnoidea                     |
| 6. Chorioidea                     | 12. Pia Mater                       |

## 5.2 PHYSIOLOGY FOR EMERGENCE OF INTRAOCULAR PRESSURE

Intraocular pressure (like air in a balloon) provides the eyeball's symmetrical form, which exists inside as a gel-like masse (vitreous humor). A liquid (aqueous humor) rinses the space between the lens, iris, and cornea. It helps care for these organ parts. Vitreous humor is formed from the ciliary body's epithelium and released at the rear of the eye chamber. Minute volumes of the aqueous humor amount to 2 mm<sup>3</sup>. It flows through the elastic *Zonula Zinnii* along the front lens capsul as well as the rear surface of the iris and through the pupil to reach the front of the eye chamber, which is about 3-3.5 mm deep. There it typically results in streams of aqueous humor. Part flows up to be warmed by the iris, another part moves below to cool the cornea endothelia. Up to 90% of the liquid released from the front of the chamber occurs at the iridocorneal angle through the *Trabeculum corneosclerale* network. It flows from this point to the ring-formed Schlemm's canal found in the sclera, which nourishes

the aqueous humor veins with aqueous humor, finally leading to the conjunctival vessels. The remaining 10% of the aqueous humor leaves the eye via the uveoscleral discharge into the general venous circulation. It flows through the ciliary muscle, then between the ciliary muscle and sclera, eventually to be released transsclerally. The aqueous humor is constantly reformed and drained off, again through the blood circulatory system. If it cannot flow – or only with difficulty – the intraocular pressure increases, which causes mid-term damage to the optic nerve and the cornea's nerve cells (glaucoma) (Klauss, 2003, 210-238).

The flow of blood within the optic disc plays a more crucial role in emergence of glaucoma ailments than had been assumed until now (Naseman, 2004, 259-270).

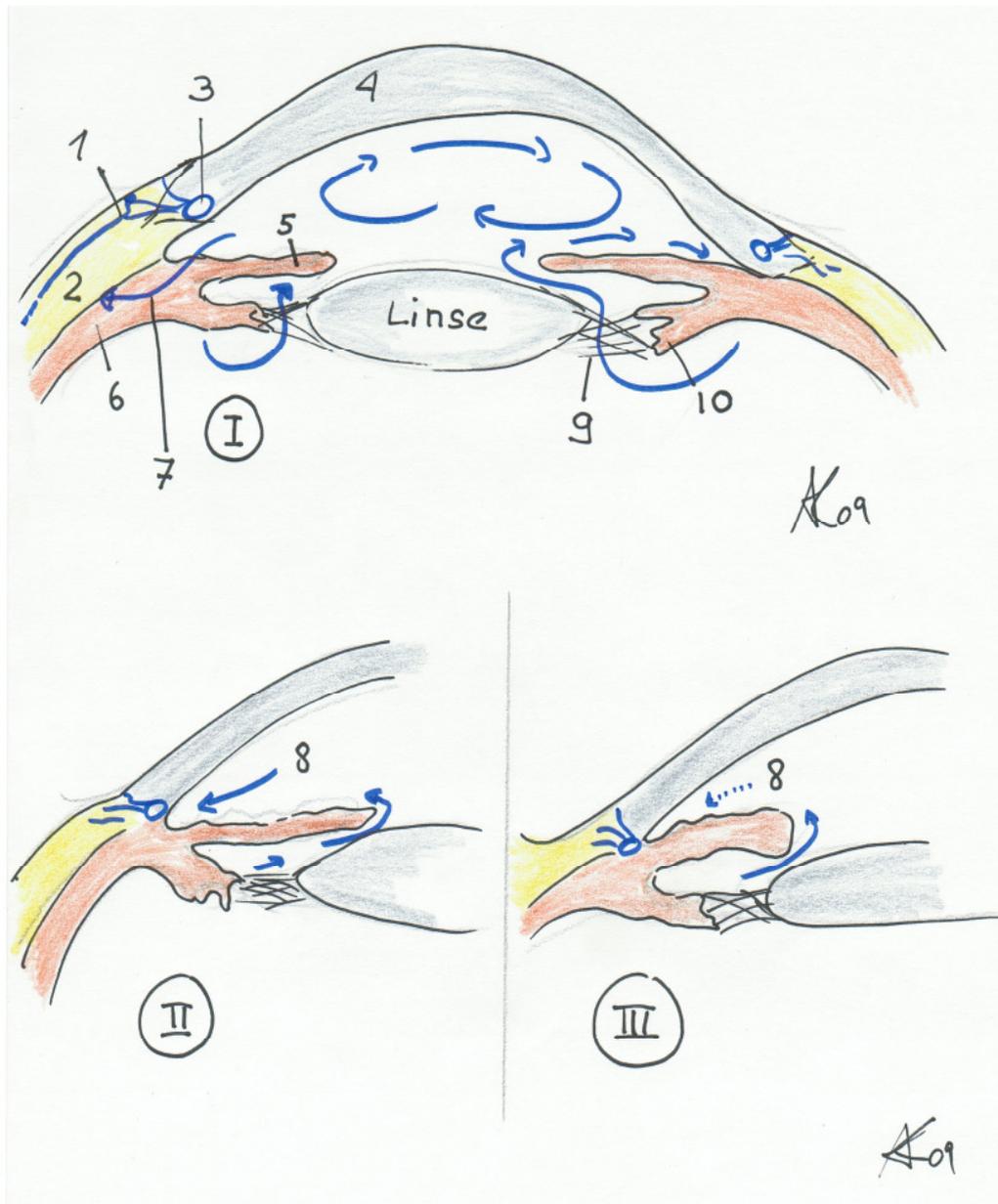


Figure 5.4: Aqueous humor circulation

- |                       |                          |
|-----------------------|--------------------------|
| 1. Aqueous humor vein | 6. M. ciliaris           |
| 2. Sclera             | 7. Uveoscleral discharge |
| 3. Schlemm's canal    | 8. Trabecular discharge  |
| 4. Cornea             | 9. Zonula ciliaris       |
| 5. Iris               | 10. Ciliary body fringe  |

- I. A warm flow of aqueous humor (uveoscleral discharge) surges from the rear to the front of the eye chamber.
- II. Discharge from the front chamber of the eye at a wide iridocorneal angle (trabecular discharge)
- III. Discharge from the front chamber of the eye at a narrow iridocorneal angle (trabecular discharge)

### 5.2.1 ANATOMICAL FACTORS REGARDING THE EYE IN AN OSTEOPATHIC SENSE

A.T. Still pointed out how closely the cranium interacts in connection with all other bodily organs. A great many misinterpretations would occur without the five sensory organs. Hence it is important to analyze precisely any illnesses that affect the cranium. An examination here should determine where and within which system the major problem (primary dysfunction or lesion) occurs (Still, 1889, 25). Still writes: *“Keep the road from the heart to the brain open and in first-class condition for the passage and delivery of pure arterial blood to the head. The free and unobstructed return of the venous blood is just as important.”* (Still, 1910, 106). He refers here not only to the blood circulation that in Still’s case always takes center stage but also to the biomechanics of the skull bones, the cervical spine, and the transition to the dorsal spine (Still, 1910, 105-107). This Still theory is verified today in many scientific articles in manual medicine such as Diehl *et al.*, 1991. In order to treat the eye, one must understand its multiple anatomic links. In an osteopathic sense the eye has an enormous number of complex anatomic connections. If one considers that the *Dura mater*, *Pia mater*, and *Arachnoidea* surround the optic nerve, one can better judge the link with the brain’s subarachnoidal sphere. Increased cerebral spinal pressure on the optic nerve and optic disk trigger swelling of the optic disk in the form of an engorged papilla (Sachsenweger, 2004, 302-310). The *Sinus cavernosus* connects the eye to the venous discharge system. There are many techniques available in osteopathy with which one can treat and influence the cranial region (Liem, 2003; Blum, 1988; Lippincott, 1958)

The sympathetic and parasympathetic nerve system nourishes the eye. The sympathetic portion can in turn be influenced with osteopathic techniques according to Cloet (Cloet, 1995, 135-144) via the cervical spine and dorsal spine. The linkages mentioned above invite the osteopath to consider and treat a complex (Eder, 1991; Korr, 1978).

### 5.3 GLAUCOMA

Glaucoma (green cataract) is understood to be a pathological state of tension that can lead to tissue damage, especially at the optic nerve. In the process, the intraocular pressure can be absolutely or relatively too high in comparison to the blood pressure in the *A. Ophthalmica* or retina and choroid membrane circulation. The

major causes lie in an increase of intraocular pressure (mechanical factor) and/or in a circulatory disturbance within the optic disc (vascular factor) (Sachsenweger, 2004, 210-238).

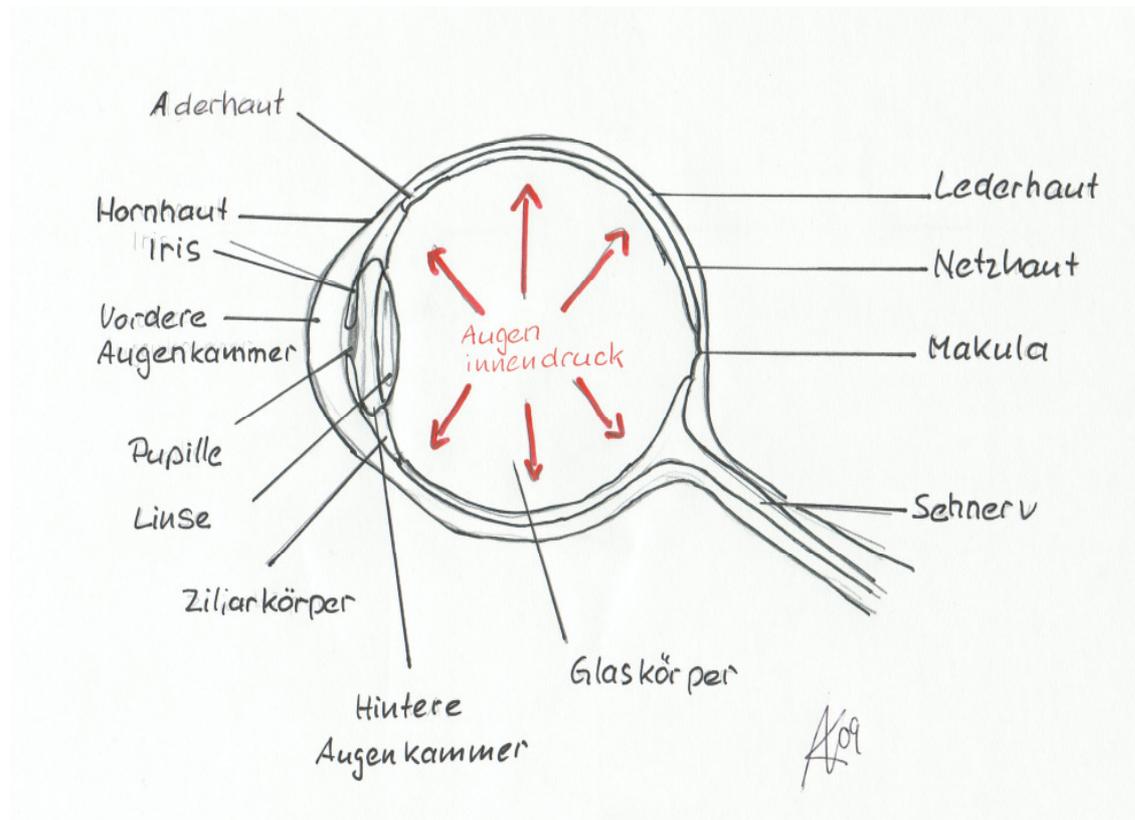


Figure 5.5: Intraocular pressure

### 5.3.1 Causes and origin of Glaucoma

Increased intraocular pressure plays a crucial role in most cases in which glaucoma emerges. Causes can be too high or too low blood pressure, circulatory disturbance at the optic nerve, diabetes mellitus, or also cortisone treatment (Klemm, 2008).

The following glaucoma ailments are known: narrow-angle glaucoma, acute glaucoma, primary congenital glaucoma, and secondary glaucoma.

Emergence of glaucoma is usually diagnosed by increased intraocular pressure. But it must also be noted that intraocular pressure is individual with each human being. People who have glaucoma patients among their closest relatives are particularly vulnerable to developing this disease. Other risk factors are, for example, the ethnic origin of a patient, whereby it has been determined that Africans carry a higher risk of glaucoma sickness than central Europeans. Japanese, on the other hand, develop normal pressure glaucoma more often. No explanation has been found for this to date (Hoffmann, 2008).

The upper **normal limits** of intraocular pressure lies at about 21 mm Hg. Even if the intraocular pressure lies in the normal range, glaucoma can be present or develop. In these cases the intraocular pressure does indeed lie within the normal range (10-14 mm Hg), but it is too high for the eye affected and causes related damage. On the other hand, increased pressure must not automatically mean that one has or will develop glaucoma. The classical model of glaucoma pathogenesis stems from an increase in intraocular pressure as the major causes. Yet it has been shown that 30% of patients (Pfeiffer, 2005) with progressive changes typical for glaucoma in the optic disc and field of vision show no increased intraocular pressure in the inner eye (Klemm, 2008). Studies at the University Clinic in Hamburg point out that a decreased perfusion in the optic disc area could be responsible for progression of glaucoma (Matthiesen *et al.*, 2004).

**People with false regulations of the vessels** (or vascular disregulation) react to stimulations such as cold and emotional stress more intensively with vascular cramps (vasospasms). These symptoms are also categorized under the term „vasospastic syndrome“. This is not an illness by definition but describes a vascular behavioral pattern that can arise in extreme situations. It can be shown that the reduction of blood circulation in the eye that falls within the framework of vasospastic syndrome represents a certain risk factor for various eye diseases. Vasospastic syndrome is present in half the patients who show normal intraocular pressure but still have glaucoma damage. Though many people suffer from this, there is still little verified scientific data on this syndrome. There is still no therapy as such (Schalnus, 2004). This disregulation, for example, is also aroused by nicotine consumption and other stimulants. Nicotine leads to a reduction in blood circulation within the optic nerve (Sachsenweger, 2004, 302-322). The same patients also often complain of cold hands and feet. **Family medical history** also plays a large role here.

Long and continuous **cortisone treatment** (either local or to the entire body) can lead to an increase in intraocular pressure. For this reason, such patients' intraocular pressure must be checked regularly (Leydhecker, 1984).

A generalized fall in blood pressure can lead to a reduction in perfusive pressure and blood flow in the optic disc. Low blood pressure can cause glaucomatose damage as a result of increased intraocular pressure (Nasemann, 2004) (Thomazo, 2009).

**Diabetes patients** form another risk group, because diabetes can lead to the most dire eye damage. In case of diabetes patients, new, wafer-thin, and vulnerable blood

vessels of low quality emerge in the advanced state. If they arise in the frontal eye sections and grow into the iridocorneal angle, they hinder discharge of aqueous humor. This pathological change can cause a secondary glaucoma. It can result in another increase in intraocular pressure (Katz, and Sommer, 1988). After diabetic retinopathy, retinal vessel obstructions are the second most common vascular retina ailment (Faulbom, 2006).

According to a very detailed study (Schalnus, 2004), **high blood pressure**, diabetes, hypercholesterolemia, and increased blood viscosity are the most frequent causes of increased intraocular pressure. Changes in findings on arterial hypertension develop in proportion to the duration and intensity of hypertension. The same study mentions another direct correlation to renal ailments (Kremmer, 2004).

Patients belong to **risk groups** that regularly receive drops containing cortisone or other medicines containing cortisone as well as people with eye injuries and blood flow pathologies or those who also have diabetes (Katz, and Sommer, 1988).

**Regular checkups** can define individual eye pressure. A possible upward deviation in pressure value can be a sign of potential disease. Intraocular pressure should be checked at least every 2-3 years for each person from age 40. In case of risk patients, more frequent checkups are recommended (Klemm, 2008).

## 5.4 PRIMARY GLAUCOMA

Primary glaucomas are divided into *Glaucoma chronicum simplex*, primary open angle glaucoma and chronic narrow angle glaucoma

### 5.4.1 Glaucoma chronicum simplex

*Glaucoma chronicum simplex* is characterized by years of moderate increase in pressure without other symptoms but with progressive atrophy of the optic nerve as well as functional loss unnoticed over a prolonged time (Ugi, 2004, 210-238).

By far the most common form of glaucoma is primary open-angle glaucoma (green cataract), which affects older people most often. The frequency of disease is at least eight times as high among those between ages 70 to 80 as among the age 30-to-40 group. Men and women are equally susceptible to glaucoma development during their younger years. Women are affected more often than men by green cataracts (Rüther, 2007).

**Aetiology and pathogenesis:**

The cause lies in a usually bilateral dysregulation of blood circulation in the optic disc related to aging. But the onset of unilateral dysregulation may often be present.

On one hand, the dysregulation is caused by an obstruction of aqueous humor discharge resulting in an increase in intraocular pressure, a rise in resistance to discharge, and a reduction in ability to discharge aqueous humor. Finally it affects sclerosis of the trabecular structure. On the other hand, it prompts a restriction of blood circulation in the optic disc in case of a normally wide and open iridocorneal angle (Silbernagel, 1991).

Since the intraocular pressure is too high in comparison with the optic disc blood circulation, it results over the course of years in irreversible glaucomatic atrophy of the optic nerve, caused by a slow but progressive loss of axons within the retinal ganglion cells. The consequence is a diminished field of vision that usually is only noticed when the visual core is affected (Ugi, 2004, 302-322).

A primary open-angle glaucoma shows no symptoms for a long time, and the course of the illness is usually free of pain. The initial symptoms for many patients appear first as large colored rings. This is caused by epithelial swelling, water accumulation in the cornea's outer layer. During the course of the disease, it results in defects within the field of vision ranging up to total blindness. In Germany alone some 500,000 people suffer from a manifest illness, and 30,000 of them have become totally blind. Up to 90% of all glaucoma patients are afflicted with open-angle glaucoma (Sachsenweger, 2004, 210-238).

**Diagnostics:** In case of an eye otherwise normal and free of irritation with full visual sharpness and a normally deep front chamber, at first only an increase in pressure of between 20 and 30 mm Hg points to a potential glaucoma. In case of an increased intraocular pressure above 26 mm Hg with visual loss, a conspicuous ophthalmoscopy (loss at the cornea's region's nerve-fibre level) and a positive family medical history the diagnosis becomes clear. Despite all the findings, which must always be seen within a context, judgments and diagnosis must be linked to great experience (Thomazo, 2009).

When **treating** primary open-angle glaucoma, lowering the intraocular pressure takes first priority. Since it concerns a chronic illness with a slow course, the disease's progress can be influenced and inhibited through medicinal therapy. Medicines are trickled into the eye as eye drops. If the medicinal treatment does not

suffice, laser therapy can be initiated. If even this does not lead to the desired improvement, an operation under local anesthesia can be carried out. In case of this operation, an artificial discharge is created for the aqueous humor.

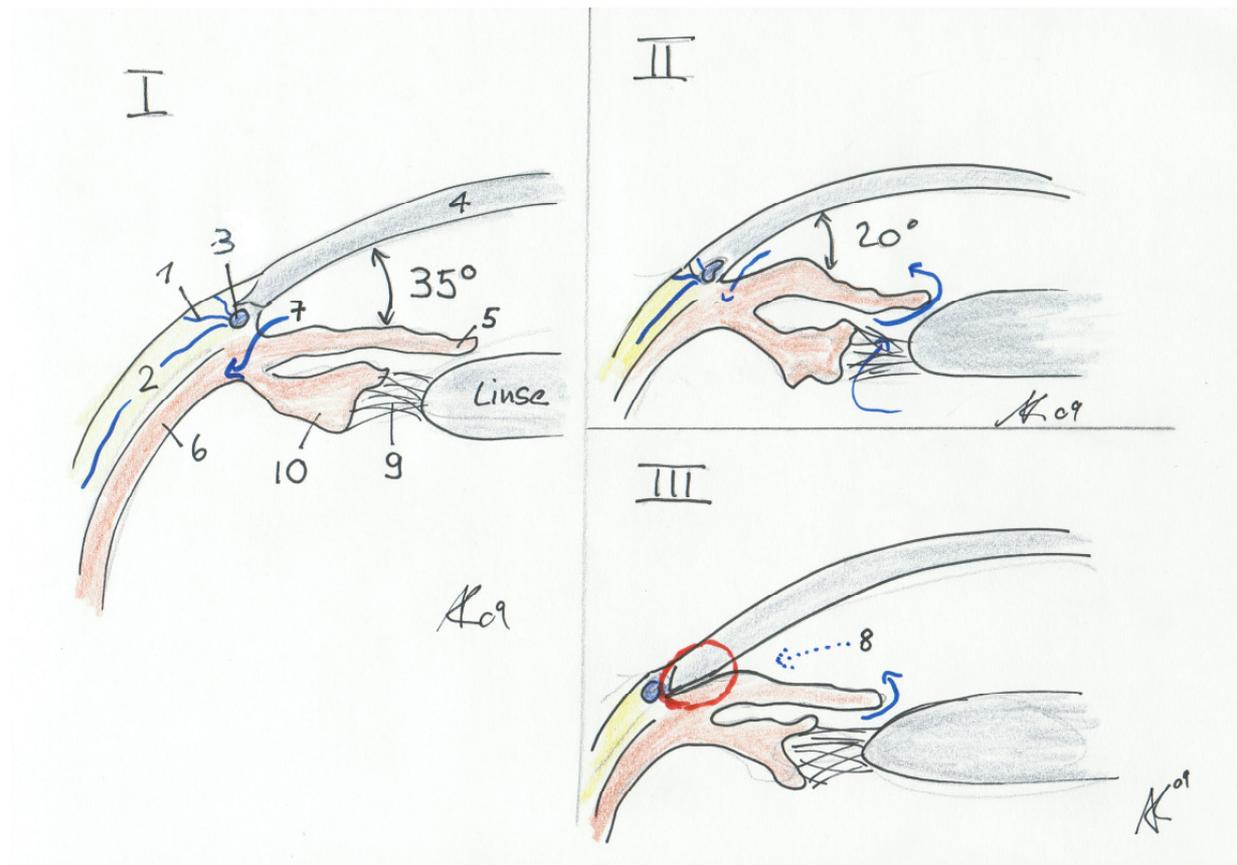
**Risk factors: family medical history** plays a very important role here. Moreover, smoking should be avoided, since the nicotine leads to a reduced blood circulation in the optic nerve. Emotional stress should be avoided. Taking cortisone preparations over a long period can also lead to an increased risk (Leydhecker, 1984). A generalized lowering of blood pressure can lead to reduction in perfusion pressure and less blood flow in the optic disc. Lower blood pressure can result in increased intraocular pressure causing glaucomatic damage (Sachsenweger, 2004, 412-417).

#### 5.4.2 Acute glaucoma

A glaucoma attack is an acute, high-grade, and mostly one-sided increase in intraocular pressure with intense pain and very swift loss of visual function. Acute glaucoma can reach a sudden increase in pressure of 80 mm Hg. Severe headaches and stomachaches as well as vomiting are frequent secondary symptoms of acute glaucoma (vagus nerve stimulation). Patients also describe a rainbow colored ring of light that occurs due to corneal edema. The *Bulbus* feels as hard as stone in case of palpation through the usually swollen upper lid. The eye becomes intensely red due to the blockage, while the pupil is highly dilated and fixed. Acute glaucoma should be subjected to medicinal and operative treatment immediately. In these acute cases, every hour counts in order to save or maintain the eyesight. If an acute glaucoma is not treated immediately, it can lead to complete blindness (Kanski, 1996).

#### 5.4.3 Chronic narrow-angle glaucoma

This form is characterized by high-grade increase in intraocular pressure and complaints as well as very swiftly appearing optic nerve atrophy. Characteristics for this illness are periodically appearing rises in pressure with peak values of up to 40-60 mm Hg and pressure-free intervals that can often last for months (Sachsenweger, 2004, 210-238). A discharge obstruction of aqueous humor is usually present on both sides due to a narrow iridocorneal angle. Caused by temporary formation of a cornea edema, patients complain of moderate eyeaches and headaches as well as perceptions such as nephelopia and color rings (Sachsenweger, 2004, 210-238). Here it concerns phases between the glaucoma attacks.



**Figure 5.6:** Three different iridocorneal angles

- |                       |                          |
|-----------------------|--------------------------|
| 1. Aqueous humor vein | 6. M. ciliaris           |
| 2. Sclera             | 7. Uveoscleral discharge |
| 3. Schlemm's canal    | 8. Trabecular discharge  |
| 4. Cornea             | 9. Zonula ciliaris       |
| 5. Iris               | 10. Ciliary body fringe  |

I. Wide open iridocorneal angle of  $35^\circ$

II. Reduced iridocorneal angle of  $20^\circ$

III. Angle blockage glaucoma; regular aqueous humor discharge no longer possible

#### 5.4.4 Low pressure glaucoma

This form of glaucoma reveals normal intraocular-pressure relationships and an open iridocorneal angle. Between 10 and 15 percent of glaucoma patients suffer from low-pressure glaucoma. As a result the optic disc area is "indented". In these cases, too low blood pressure leads to the sensory cells in the eye being supplied with insufficient oxygen and nutrition. It can be difficult to differentiate between low-pressure glaucoma and primary open-angle glaucoma. Vascular factors are responsible. The perfusion pressure in the optic disc is too slight to ensure sufficient

papillae blood circulation. Papillae circulation disturbance leads to glaucomatic optical atrophy. This form of glaucoma describes an increasing disturbance of the field of vision with progressive excavation of the papillae at “normal” intraocular pressure values and otherwise unsurprising eye findings. It can be difficult to differentiate between low-pressure glaucoma and primary open-angle glaucoma. It is difficult to distinguish *Glaucoma Chronicum simplex* from optical atrophy of a nonglaucoma gene (Sachsenweger, 2004, 210-238).

## 5.5 TREATMENT OF VARIOUS TYPES OF GLAUCOMA

Narrow-angle glaucoma appears independent of age. To treat it, one first lowers the intraocular pressure with medication. Extended therapy occurs by means of an operation that reestablishes the balance between aqueous humor production and discharge. In case of an inborn glaucoma, this therapy must be applied fundamentally.

The most common form of green cataract, the open-angle glaucoma, which appears increasingly after the 40<sup>th</sup> year of life, is maintained at first through medicinal therapy. Good results can be achieved here. It involves normalizing intraocular pressure through eyedrops that must be given very precisely 2-3 times a day. Maintaining the exact time for the eyedrops prevents further damage to the optic nerve, since the drops either reduce formation of aqueous humor or can improve the trabecel's resistance to discharge. Eyedrop therapy must be carried out regularly throughout one's life (Kanski, 1996).

Medicines such as Timolol®, Porpanolol®, Arutimol®, Chibrotimoptol®, and Betaman® – all of them considered betablockers – are **used to reduce** aqueous humor formation. D\_Epifrin® is an adrenaline derivative of similar composition. In Switzerland the medicine Trusopt® is often used as a carbohyrase inhibitor. The combination drug is called Cosopt® (Pfeiffer, 2005).

A medicine to reduce aqueous humor and improve its discharge is a prostaglandin agonist named Xalatan®.

If the target is improved blood circulation in the optic nerve sphere as well as the two factors mentioned above, the preparation Alphagan®, an alphagan agonist, is recommended. If the drops do not suffice to lower intraocular pressure and the limited field of vision contracts further, an operation may be required. This will occur by means of a laser or surgery (Pfeiffer, 2005) ([www.med.uni-jena.de](http://www.med.uni-jena.de))

## 5.6 OSTEOPATHIC RELATIONSHIPS TO EMERGING GLAUCOMAS

The body is a vital organism and is found to depend upon its own mechanisms. It is assumed that the basis for all treatments of these mechanisms lies in their origin within the nervous system. It stands connected to the sympathetic nervous system that exercises ultimate control over all organic processes. If any sort of problem arises in the spinal nervous system, it affects the visceral system in a reflex manner (Wernham, ca. 1950, 126-132).

Osteopathic techniques now enable active mobilization of the body. It helps in this regard to imagine the body as a machine that must be kept in good working order (Wernham, 1950, 48-51).

In order to understand how the mechanism works in withstanding emergence of glaucoma, the aspects mentioned must be considered. Many of the causes may lie in the mechanical sphere. Osteopathy has many treatment options that promise success here.

Other important osteopathic treatment methods include indirect involvement to improve the circulation and discharge of lymphs and veins. Moreover, one must treat the cranial area above all to relieve skull bones and the related facial nerves (Mc. Conell, 1899, 570-572).

Furthermore, the orthosympathetic nervous system with its gangliae should be checked for mobility. Many basic disorders of the glaucoma originate in the autonomous metabolic system. Osteopathy underscores the principle to keep the body always “flowing”. Circulating fluids, such as blood and lymphs, should always be maintained in balance with one another. The rule applies here that no bottleneck situation can emerge. In the case of glaucoma – in order to interweave the issue of my master’s thesis here – it primarily concerns such a situation in the front of the eye chamber. The related question is the cause of a bottleneck as well as the possibility of resolving this mechanical or metabolic issue (Still, 1902e, 68, Stark, 2004, 136).

An osteopathic treatment can improve blood circulation by normalizing it. This results in the blood being resupplied with oxygen. As a consequence, the liver regains and increases its metabolic power, which in turns supports detoxification. This indirectly relieves the functions of the glands, the kidneys, the skin, etc. Improved metabolism results in improvement of the entire organism, which positively affects bottleneck situations that have arisen in the body (Wernham, 1950, 126-132).

Still's thoughts on the *fasciae* were intensive and far-reaching. Jane Stark's book on Still's *fasciae* concepts explains an osteopathic connection with intraocular pressure and describes it in great detail. The *fasciae* were for Still not simply connective tissue that held "everything" together. His assertions went much further.

Still's assertions are based on **three** statements. The first described: "*networks out of nerves, cells, and tubes that lead to and from the fasciae.*" These, he held, "*are connected and doubtlessly filled with millions of nerve centres and fibres that continuously secrete vital and decomposable liquids inside and outside*" (Still, 1902e, 60, Stark 204, 267). He explained further that: "*all liquids must be guided through the body by means of arteries, veins, lymph tracts, and tunnels that discharge as well as secrete*" (Still, 1902e, 283, Stark 267). His third statement: "*All fasciae must be free in all parts of the body, so that they can receive and discharge all fluids, particularly liquids that remain for an abnormally long time*" (Still, 1899g, 166; Stark 2004, 220, Still 1092, 50). Still believed that an unnatural accumulation of fluids would create a vicious cycle in the sensory nervous system (Still, 1899g, 220, 1902e, 50). He meant by this that if swelling occurs, danger lurks that it would be maintained by feedback mechanisms (Stark, 2004, 204).

Still also mentioned the importance of liquid nutrition in this context. Another of his thoughts noted the vital role that cerebrospinal fluids play in irrigating the entire body (Stark, 2004, 267, Still 1908a, 305).

The study by Gerste *et al.*, 2008 pointed to infection of the cerebrospinal fluid and emergence of glaucoma, verifying it scientifically. Thus Still's assumption was confirmed (Still, 1902, 44f).

Jean Stark collected more discoveries of Still's understanding of body fluids. "*Still held,*" she wrote, "*that starvation (lack of nutrition caused by pressure and swelling) affected movement blockages in the nerves. This starvation cannot be of long duration. The harmful effect on nerves that are not nourished by the arteries promptly leads to their failure.*" (Still 1902e, 50)

The secreting systems (and to a lesser extent the veins) partially allow free flow of blood and lymph secretions. A feedback loop begins here once again in this manner (Stark, 2004). This interpretation of the swelling and thoughts on fluids has a direct connection with the problem of glaucoma illness described in this study and the related increase of intraocular pressure (Still, 1902, 224-265f).

Viola Frymann points out the importance of the venous system in her book (Frymann, 1998, 439-440). She explains as well how vital it is that the precise amount of blood enriched with oxygen is transported to the nerves. Fryman's emphasis lies in the essential fact that she finds extremely crucial: the feedback of blood lacking or deficient in oxygen has on the venous system. She notes that an imbalance in interstitial fluid can result in tissue problems in equalizing pressure. These pressure balances can only be assured if no congestion occurs in the body. In case of acute chronic infections, for example, the healing process only begins when venous blockages are removed (Frymann, 1998, 219-224).

## **6 PROBLEM FORMULATION**

A **flow diagram** was created to show the organizational and study flow. All the study's developments are clearly visible and categorized in this flow diagram.

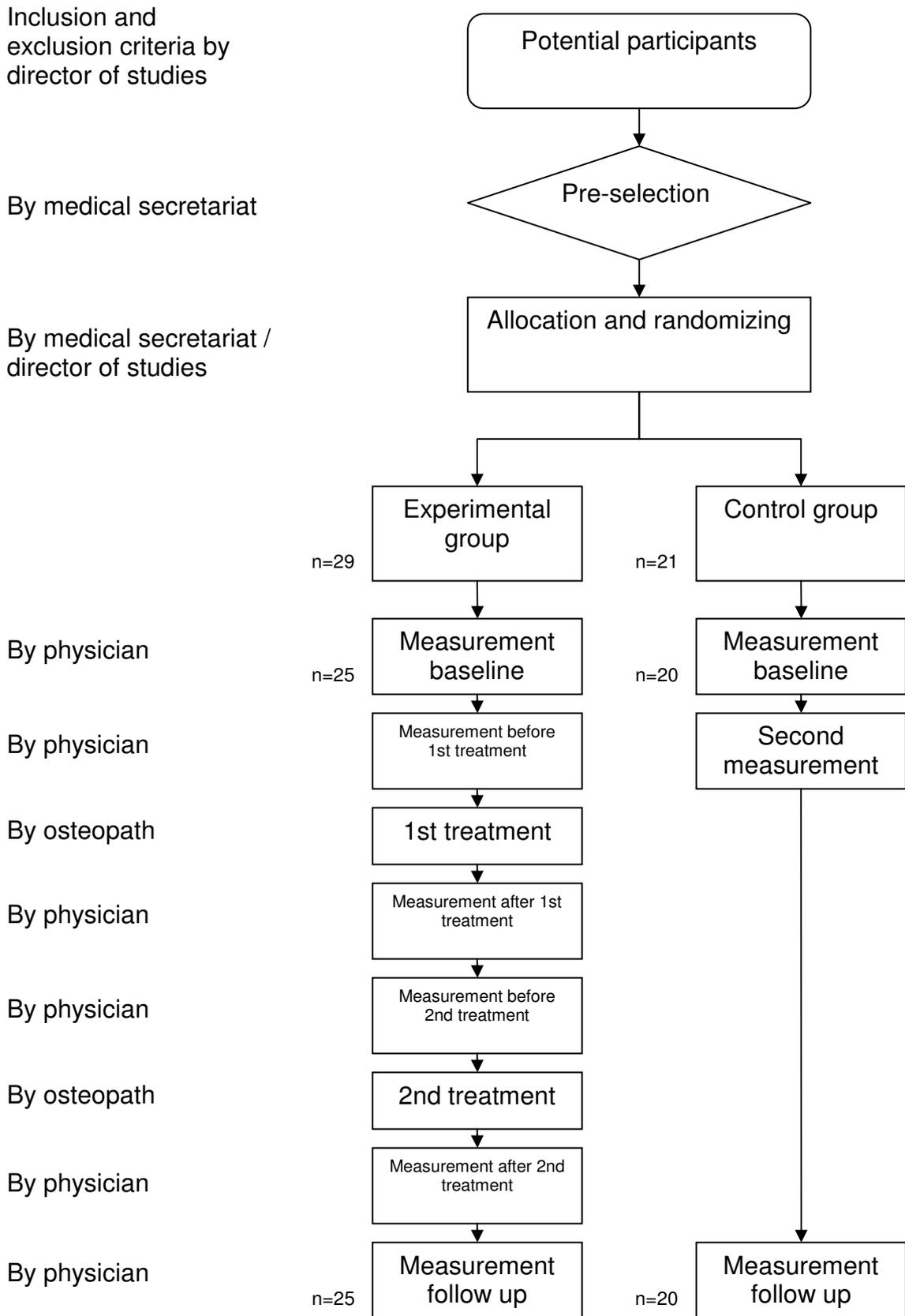


Figure 6.1: Flow diagram of sample construction

## 6.1 ISSUE FORMULATION

Is it possible to influence medicinally stabilized intraocular pressure of patients undergoing treatment with an additional approach through osteopathic treatment?

## 6.2 HYPOTHESIS

H<sub>0</sub> No changes in intraocular pressure occur through osteopathic treatments.

H<sub>1</sub> It is possible to lower intraocular pressure with an osteopathic treatment approach.

## 6.3 METHODOLOGY

This study concerns a random sample that should demonstrate the effectiveness of osteopathic treatment in case of increased intraocular pressure. The effect of treatment is recorded by measuring the intraocular pressure with a tonometer (Gold standard, see 6.7).

- **Blind procedure:** The single-blind procedure was used. Patients did not know in which group they were assigned.
- **Randomization:** A randomization list was compiled in advance. Assignment of randomization numbers to patients occurred through a person independent of the study.
- **Group division:** Patients were divided into treatment group A (experimental group) and treatment group B (control group) based on the randomization list.

## 6.4 INCLUSION CRITERIA

- Patients who had a significantly increased eye pressure of more than 19-21 mm Hg or recorded it after the first measurement
- No other eye diseases could be determined
- Patients for whom the intraocular pressure is stable with medication or lies within the same mm Hg sphere
- No other cranial pathology was determined.
- Patients with stable blood pressure

## 6.5 EXCLUSION CRITERIA

- Acute increase in intraocular pressure
- Acute and chronic infections in the eye region
- Other preexisting eye diseases that affect the eye and influence intraocular pressure
- Known tumor diseases in the head area

A group size of 50 test subjects was envisioned. Some 25 of these subjects were treated osteopathically. The other 20 subjects entered the control group but were not treated.

## 6.6 PLANNED STUDY SEQUENCE

The patients' intraocular pressure was measured six times by the physician using the Goldmann Tonometer. Those patients who showed an increased intraocular pressure were informed of the study's inclusion and exclusion criteria and – if they corresponded to the criteria – recruited. If the patients took part in the study, first measurement data were noted in keeping with inclusion and exclusion criteria. After this first measurement, patients were registered and entered on the randomization list.

### 6.6.1 Experimental group

The second measurement took place at the doctor's office before the first osteopathic treatment. The third measurement occurred immediately after the first osteopathic treatment and on the same day. It was noted that the patients maintained the same time interval between measurements before and after treatments. Then a two-week break in treatments occurred. After two weeks the patient was again measured before and after treatment in the same rhythm as at the first treatment. (These were the fourth and fifth measurement check-ups). Another two-week break occurred after the fifth measurement. Then the final measurement was carried out with both the control group and the experimental group.

### 6.6.2 Control group

Three measurements of the control group were run to compare with the experimental group. The first measurement corresponded to that carried out for inclusion and

exclusion criteria at recruitment. Measurements of the control group followed the normal course of time for regular control measurements taken routinely for these patients in the ophthalmologist's office. The control group was not treated, but three measurement dates from the routine plan served as comparisons for the experimental group.

### 6.6.3 Measurement parameters; FS 36; blood pressure

Two other important measurement parameters were blood pressure measurement and the questionnaire concerning the family medical history regarding eye diseases.

- Blood pressure measurements before and after each treatment (four measurements)
- Questionnaire FS 36: This was distributed before and after the first treatment and before the second treatment (two questionnaires).

## 6.7 METHOD OF MEASUREMENT

Measuring intraocular pressure occurs with the help of a tonometer. A common and reliable procedure today is measuring intraocular pressure according to Goldmann. For this purpose, the patient sits at the slit lamp. A local anesthetic, which also contains a yellow dyestuff (fluorocein®), is dropped into the eye to be measured. A disinfected measuring head is then placed on the eye. It has a frontal surface of about 3 mm in diameter. The physician can read the pressure required to flatten the corresponding surface on the cornea. This pressure corresponds to the intraocular pressure. The applanation tonometry using the Goldmann device possesses sufficient precision and is usually simple for an experienced examiner to carry out (Rambold, 2005). The disadvantage is that transmission of disease germs (*e.g.*, *Tunica conjunctiva* infection) by touching the eye cannot be ruled out (Gasser, 2004).

**Goldmann Tonometer.** The Goldmann tonometer was introduced in 1957 as the first corneal applanation tonometer with adjustable power. The surface to be flattened has a diameter of 3.06 mm. The applanation's volume amounts to 0.05 µl. The rise of up to 3% intraocular pressure caused by IOP tonometry can be disregarded. Therefore, a correction corresponding to the individual ocular rigidity is not required. The precise intraocular pressure can be read directly in mm Hg (Bucci, 1997).

At present the Goldmann applanation tonometer is the most reliable device and is used globally more than any other. It is used as the standard for evaluating other tonometers (Plagwitz, 2005). Possible limitations and sources of failure are:

- A seated position is always required for measurement.
- Ocular pulsations cause excursions of the tear menisci.
- The breadth of the dyed tear meniscuses influences the IOP reading.
- A thick cornea or an irregular cornea surface complicates measurement or leads to incorrect values.
- Repeated measurements or extended contact between cornea and tonometer can damage the cornea epithelium and influence a precise reading of the pressure value.
- As with the Schiøtz tonometer, the tonometer head must be sterilized before each measurement.

## 6.8 WORKING BASIS

Recording the findings and treatment occurred according to a method of the Lien Mecanique Osteopathique (MLO), developed by Paul Chauffour and Eric Prat (2003, 33 ff). Chauffour developed the concept in 1978. J.M. Guillot, M.D., first introduced the method publicly in 1984. It involved a very precise general finding and an equally precise diagnostic method. A precise and very structurally oriented diagnosis took center stage for the founders of the LMO method. Thus evaluation of findings demands most of the treatment time. Recording the findings always begins holistically. Ultimately the therapist completes treatment with a recoil at the smallest structure level. This corresponds to the osteopathic conceptual model, which covers the intellectual property of A.T. Still. As he put it: *“Find it, fix it, and leave it alone.”* By *“leave it alone”*, he meant the time after treatment. Here nature must go to work and mobilize its powers of self-healing.

Eight units are categorized as follows: 1. occiput – spinal column basin and extremities including joints and interosseous lesions, 2. bones, 3 energy lines, and joint diastase), 4. skull, 5. inner organs, 6. arteries, 7 the neurovegetative and peripheral nervous system, 8. skin. At the finding, which takes 80% of consultation time, all functional units are tested, compared with each other, and evaluated. One dominant is determined in each unit, which is then compared to the dominances of the other functioning units. One strives to find the “primary lesion“ or primary

dysfunction. Quite clearly one seeks to filter the dominant restriction of one of the eight functional units within the primary dysfunctions of the individual units and to determine it.

The LMO's therapeutic approach is based on a schematic and innovative protocol and a very secure recoil treatment technique. The structure identified as dominant is treated by means of recoil. The concept mainly foresees treating the area where the obvious pain or restricted movement really lies. Here the therapist strives to follow osteopathic thinking and to keep the spirit free in order to identify hidden causes, primary dysfunctions, and the root problem.

Quoting from Paul Chauffour: *"The recoil is like a breath of wind that stretches out over the structure."* And he writes further: *"The recoil is a technique that can be carried out on the entire body – on joints, organs, or the skull. Thus a vibration is produced on the tissue resistance to be treated in which the skin is subjected to a very rapid suppression at the point affected. The amplitude of this movement is next to nothing"*.

The LMO concept calls for an emergency treatment directly at the affected structure. But this is not necessarily practiced in chronic cases. However, it should be mentioned that, after treating the primary dominance – or the primary lesions of the other seven individual units – should be examined (if they existed) to see if they still remain. If this is the case, this structure (dominance/lesions) is treated. This is called an inhibition test or also a comparison test. These tests are repeated after each recoil technique is carried out. They serve to check the treatment and to monitor if anything has changed and improved. The treatment's protocols identify individual lesions in all units. This supports later examination at a following treatment and serves as a monitoring step. The treatment of the structure identified as the dominant one is carried out by means of recoiling.

The method is continuously adapted to the latest findings of today's osteopathic medicine. The method is explained in greater detail in the German *Zeitschrift für Osteopathie* (Somody-Neplaz, 2007), which points out how important the treatment and recognition of dysfunctions is within the arterial system.

The basic thinking of Andrew Taylor Still is considered in this method, and his basic principles serve as its binding thread (Somody-Neplaz, 2007).

## 6.9 CARRYING OUT THE STUDY

The randomizing and single-blind procedure could be maintained as planned. Treatments of the experimental group occurred without complications. The course of treatment could be carried out for all patients as planned.

## 7 RESULTS

### 7.1 DESCRIPTION OF THE TEST GROUPS

Examinations of the control group determined if possible changes in intraocular pressure in the experimental group could be traced to circumstances also present in the control group. This included checking the comparability of the groups in relevant characteristics – especially age and gender.

Osteopathic treatments in the experimental group were carried out at two meetings of 60 minutes apiece within half a year. Hence individual time intervals between the first and second treatment were nearly constant (92% between 13- to 15-day intervals).

Due to dropouts of four test subjects, their measurement values had to be removed from the experimental group, so this group still consisted of 25 people. The values of one person in the control group were ultimately disqualified, so a total of 20 people provided complete measurement values.

	Experimental group	Control group	Statistic	p	Significance
Number	25	20			
Age (in years)	69.8	68.6	t=0.344	0.721	n.s.
Gender (proportion female)	36%	30%	$\chi^2=0.180$	0.671	n.s.
Initial ocular pressure	17.5	17.9	t=-0.485	0.631	n.s.

*Table 7.1: Description of the sample checks*

As can be seen in Table 7.1, differences between the two groups in the spot checks were not statistically significant. Both the average age and distribution of gender as well as initial intraocular pressure were comparable.

### Medicines and illnesses

By far the largest portion of the experimental group (76%) already had increased intraocular pressure for five years or longer. In most of these cases (89.5%) it had been treated from the start. All patients in the experimental group were being treated medicinally due to their increased intraocular pressure, whereby many patients in the experimental group (60%) took additional medications beside those to treat the intraocular pressure. Those ailments treated with other medications involved very

different types of illness. For example, eight study subjects suffered from heart circulatory problems, five indicated renal illnesses, and two ailments in the thyroid gland area. None of the study subjects suffered from diabetes types I or II. Thus the number of medications taken had no significant statistical correlations related to the initial intraocular pressure before the first treatment.

### **Smoker status**

Unfortunately smoker status was only recorded for the experimental group but can provide interesting information for description of the spot checks. Two of the 16 male participants in the study's experimental group are smokers (12.5%). All nine women who took part in the study are nonsmokers (0%). Compared to the average portion of smokers in the Swiss population (23.6% among males and 21.1% among females) (IUMSP, 2005), this results in an obviously lower but statistically insignificant smoker portion among both male and female study subjects. The percentage of smokers in Switzerland (21.1% among women and 23.6% among men) differs by only 2.5% and is thus insignificant. The smoking portion of men and women combined resulted in an average value of 22.3%. This portion offers only a marginally significant statistical deviation from the average ( $p=0.0599$ ). Thus there is no statistical relevance between smoker status and the onset of intraocular pressure. The same applies for systolic and diastolic blood pressure.

Participants of the experimental group were also asked about their **family medical history** regarding glaucoma illnesses. Eight of the 25 study subjects (32%) indicated a family predisposition. The remaining 17 persons either had no such family history or it was unknown.

## **7.2 CHANGE IN INTRAOCULAR PRESSURE**

To answer the major question – if intraocular pressure can be changed through osteopathic measures – the intraocular pressure measured is first presented in a table.

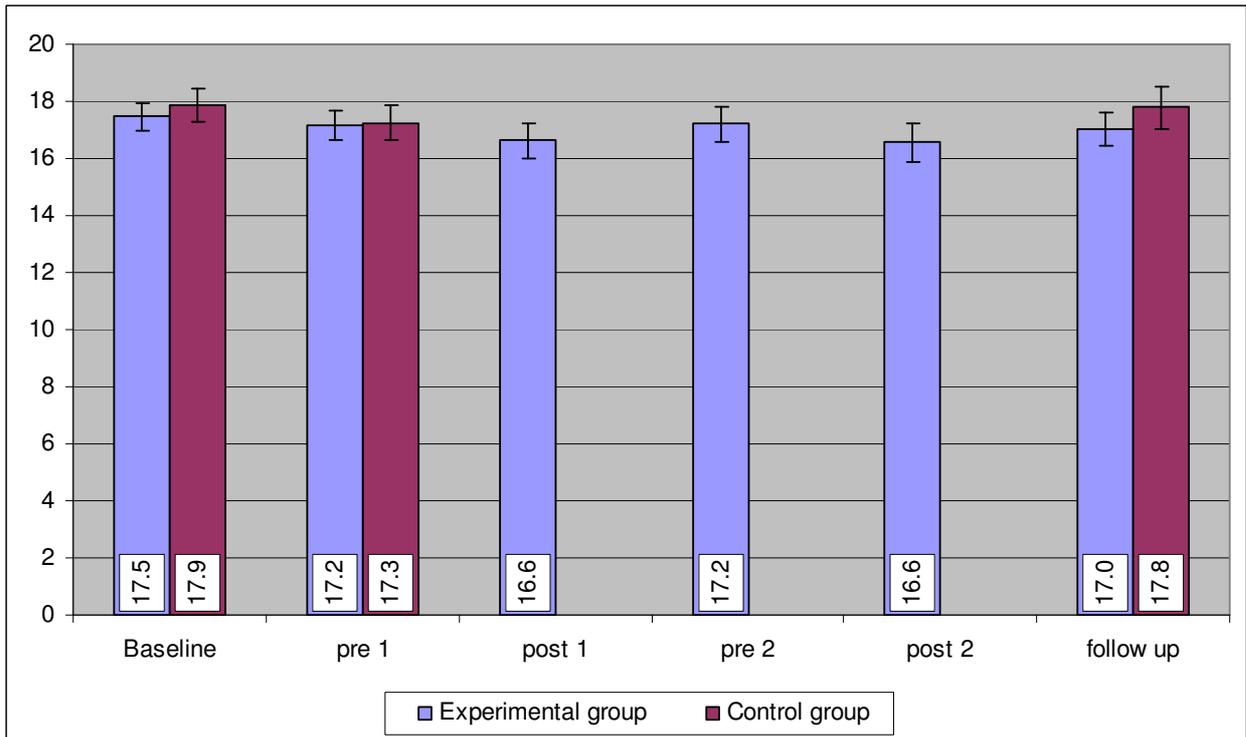
	Experimental group			Control group		
	left	right	average	left	right	average
Baseline	17.52	17.44	17.48	17.80	18.00	17.90
pre 1st treatment	17.00	17.36	17.18	17.25	17.32	17.28
post 1st treatment	16.20	17.04	16.62			
pre 2nd treatment	16.84	17.56	17.20			
post 2nd treatment	16.36	16.76	16.56			
Follow up	16.72	17.36	17.04	17.80	17.84	17.82
Change after 1st treatment	-4.7%	-1.8%	-3.3%			
Change after 2nd treatment	-2.9%	-4.6%	-3.7%			
Change follow up to Baseline	-4.6%	-0.5%	-2.5%	0.0%	-0.9%	-0.4%

*Table 7.2: Intraocular pressure for each eye, averaged for each measurement and differentiated by group*

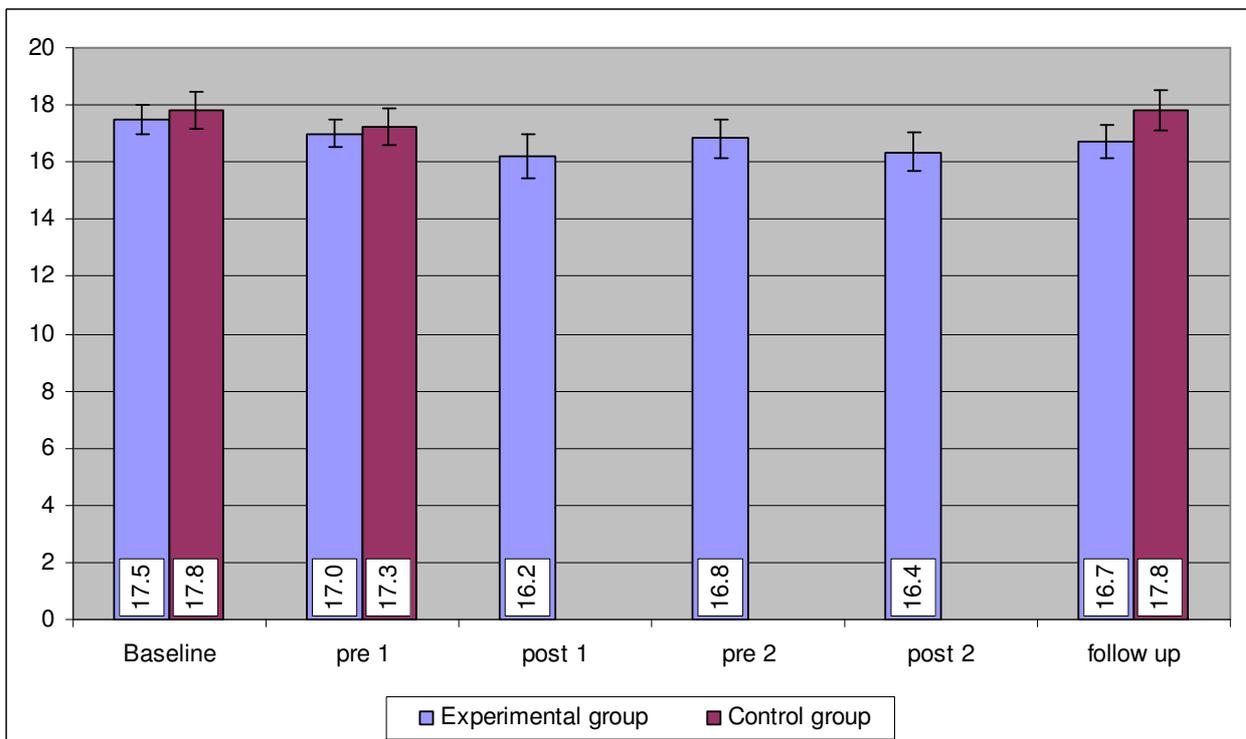
### **Development of intraocular pressure**

A decrease in mean intraocular pressure of 2.5% occurred in the experimental group throughout the entire observation period. At the same time, the eyes were affected to a varying degree: while the right eye showed a lesser decrease of intraocular pressure of 0.5%, pressure on the left eye decreased by 4.6%. It is also obvious that the intraocular pressure decreased directly after treatments, while it had risen again before the second treatment. The mean level of change in the control group's intraocular pressure for both eyes averaged lower than the experimental group: a decrease in average intraocular pressure of 0.4% is indicated here.

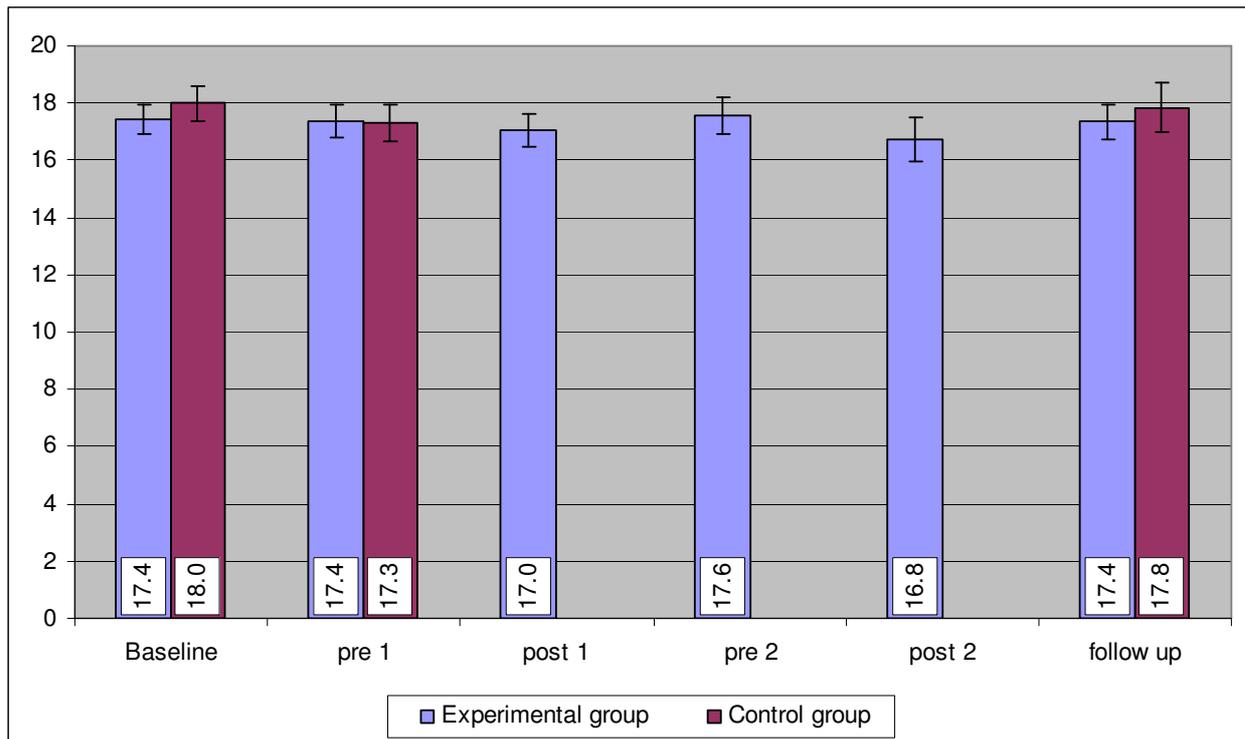
Tables 7.1 through 7.3 indicate the course of intraocular pressure over the period monitored.



**Figure 7.1:** Course of mean intraocular pressure values of both groups from original measurement until final measurement



**Figure 7.2:** Course of mean intraocular pressure value for both groups from original measurement until final measurement, **left eye**



**Figure 7.3:** Course of mean intraocular pressure value in both groups from original measurement until final measurement, **right eye**

Calculation of mean intraocular pressure values in experimental and control groups comparing influence of treatments

	mean ocular pressure pre (t1)	mean ocular pressure post (t2)	Difference	Statistic	p	Significance
Treatment 1	17.18	16.62	-0.56	1.37	0.18	n.s.
Treatment 2	17.20	16.56	-0.64	2.76	0.011	*
Control group t1 t2	17.25	17.78	0.53	-0.99	0.34	n.s.

**Table 7.3:** Mean value calculation of all groups

It follows from Table 7.3 that the mean intraocular pressure after treatments appears to decrease. The comparison with the control group shows that no comparable change appears to occur there.

Accordingly, the **second treatment** accompanies a marked not significant decrease in intraocular pressure, while the first treatment was still unable to achieve a statistically significant effect. Differences between the two measurements in the control group are not statistically significant.

One participant in the control group no longer had a right eye; therefore, the test subject's value could not contribute to calculation of a correlation between left and right. Thus *n* here is only 19.

	Correlation ocular pressure left & right eye	proportion variance not determined ( $1-r^2$ )
CG, baseline	0.821	32.6%
CG, 2nd measurement	0.916	16.1%
CG, 3rd measurement	0.834	30.4%
EG, baseline	0.762	41.9%
EG, pre 1st treatment	0.766	41.3%
EG, post 1st treatment	0.647	58.1%
EG, pre 2nd treatment	0.74	45.2%
EG, post 2nd treatment	0.729	46.9%
EG, follow up	0.841	29.3%

(KG = control group) (EG = experimental group)

*Table 7.4: Determination of common variance for intraocular pressure in the right and left eye*

Since the correlations in five of six pair measurements in the experimental group lie beneath 0.80, it did not seem sufficiently justified to summarize a mean value for the measurements. Therefore, the changes for each eye are covered separately.

Use of several measuring time points and two groups randomized in different ways suggests the need for a variance-analysis examination plan. The results of the three-factor variance analysis with repeated measurements based on two factors (measurement time points and left/right eye sides) are presented in the following. At the same time, evaluation and presentation of the second order's interaction was waived, since it was not expected that the side of the eye had a variable influence on the course of intraocular pressure.

Source		Sum of Squares Type III	df	Mean Squares	F	Significance
MZP	Sphericity assumed	9,409	2	4,705	,814	,446
	Greenhouse-Geisser	9,409	1,967	4,784	,814	,445
	Huynh-Feldt	9,409	2,000	4,705	,814	,446
	lower bound	9,409	1,000	9,409	,814	,372
MZP * GRUPPE_N	Sphericity assumed	5,182	2	2,591	,449	,640
	Greenhouse-Geisser	5,182	1,967	2,634	,449	,637
	Huynh-Feldt	5,182	2,000	2,591	,449	,640
	lower bound	5,182	1,000	5,182	,449	,507
Error(MZP)	Sphericity assumed	485,280	84	5,777		
	Greenhouse-Geisser	485,280	82,615	5,874		
	Huynh-Feldt	485,280	84,000	5,777		
	lower bound	485,280	42,000	11,554		
SEITE	Sphericity assumed	1,702	1	1,702	,733	,397
	Greenhouse-Geisser	1,702	1,000	1,702	,733	,397
	Huynh-Feldt	1,702	1,000	1,702	,733	,397
	lower bound	1,702	1,000	1,702	,733	,397
SEITE * GRUPPE_N	Sphericity assumed	1,354	1	1,354	,583	,449
	Greenhouse-Geisser	1,354	1,000	1,354	,583	,449
	Huynh-Feldt	1,354	1,000	1,354	,583	,449
	lower bound	1,354	1,000	1,354	,583	,449
Error(SEITE)	Sphericity assumed	97,465	42	2,321		
	Greenhouse-Geisser	97,465	42,000	2,321		
	Huynh-Feldt	97,465	42,000	2,321		
	lower bound	97,465	42,000	2,321		

*Table 7.5 Results of the decomposed variances (inner subject factors) for three factors with repeated measurements covering two factors (measurement time points and left or right sides of the eye)*

Source	Sum of Squares Type III	df	Mean Squares	F	Significance
Intercept	79092,250	1	79092,250	2303,742	,000
GROUP_N	14,750	1	14,750	,430	,516
Error	1441,947	42	34,332		

*Table 7.6 Results of decomposed variances (between subject factor GROUP\_N: group membership) in the examination groups (both eyes)*

The result tables show no significant changes in intraocular pressure over group's three measuring time points. Nor did group membership have any influence on intraocular pressure or its course. In order to acquire a more detailed statement on changes directly after treatments that were not recorded in the control group, another dual factor variance analysis was carried out with pure measurement repetition factors (measurement time points and side of eye), for which six measuring time

points can now be used in comparison with the three from the three-factor variance analysis.

Source		Sum of Squares Type III	df	Mean Squares	F	Significance
MZP	Sphericity assumed	32,067	5	6,413	1,022	,408
	Greenhouse-Geisser	32,067	3,255	9,852	1,022	,391
	Huynh-Feldt	32,067	3,826	8,381	1,022	,398
	lower bound	32,067	1,000	32,067	1,022	,322
Error(MZP)	Sphericity assumed	752,767	120	6,273		
	Greenhouse-Geisser	752,767	78,116	9,637		
	Huynh-Feldt	752,767	91,824	8,198		
	lower bound	752,767	24,000	31,365		
SEITE	Sphericity assumed	17,280	1	17,280	2,362	,137
	Greenhouse-Geisser	17,280	1,000	17,280	2,362	,137
	Huynh-Feldt	17,280	1,000	17,280	2,362	,137
	lower bound	17,280	1,000	17,280	2,362	,137
Error(SEITE)	Sphericity assumed	175,553	24	7,315		
	Greenhouse-Geisser	175,553	24,000	7,315		
	Huynh-Feldt	175,553	24,000	7,315		
	lower bound	175,553	24,000	7,315		

*Table 7.7: Results of the variance decomposition with six measuring repetitions in the experimental group (MZP: measuring point factor, SEITE: side-of-eye factor, error in each case: degree of variation in value that could not be explained by the respective factors)*

Consideration of six measurement time points also shows no clear picture of change in intraocular pressure.

The degree of change in intraocular pressure at the **first treatment** lacks a statistically significant correlation with the degree of change in intraocular pressure at the second treatment ( $r=0.256$ ,  $p=0.217$ ). Test subjects whose changes in intraocular pressure moved in a positive direction at the second treatment were not necessarily the same as those for whom this effect occurred at the first treatment (or indicated it to the same degree).

In order to provide a better overview of individual changes in intraocular pressure within the framework of both treatments, individual differences are presented in terms of the intraocular pressure measured. For this purpose the table represents the mean value of intraocular pressure between left and right eyes and each eye separately. The intraocular pressure before treatment is indicated on the axis in each case, as are the ordinates of intraocular pressure after treatment. The points entered are the value pairings of patients examined. If a test subject had the same intraocular pressure after treatment as before, the value pairing lies accordingly at the diagonal

indicated. An increase in intraocular pressure results in placement of the point above the diagonal, a reduction places the point below it.

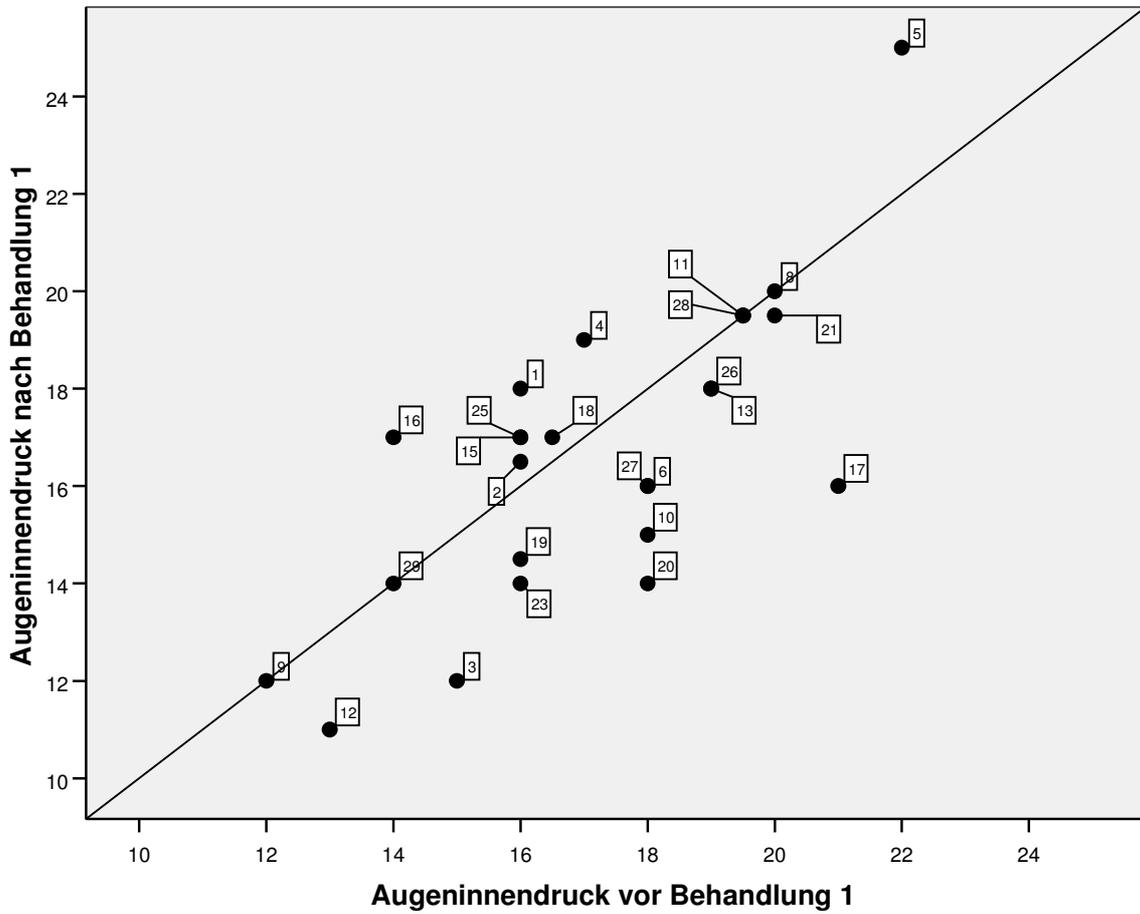


Figure 7.4: Individual median intraocular pressure **before** and **after** the first treatment

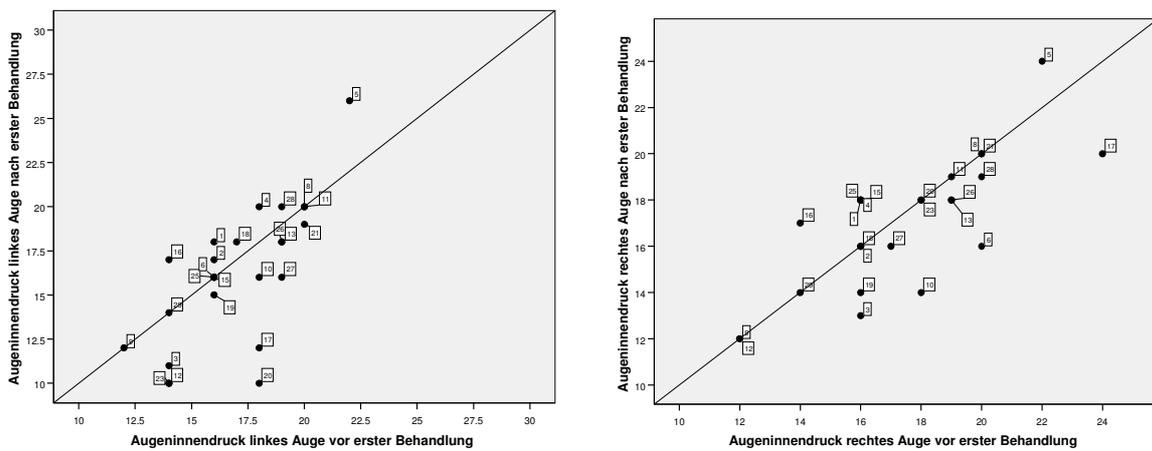


Fig. 7.5 a, b: Individual **left** and **right** intraocular pressure **before** and **after** the first treatment

Eight study subjects (32%) averaged a higher intraocular pressure after the first treatment than before it, five maintained the same intraocular pressure (20%), and 12 achieved a reduced intraocular pressure (48%).

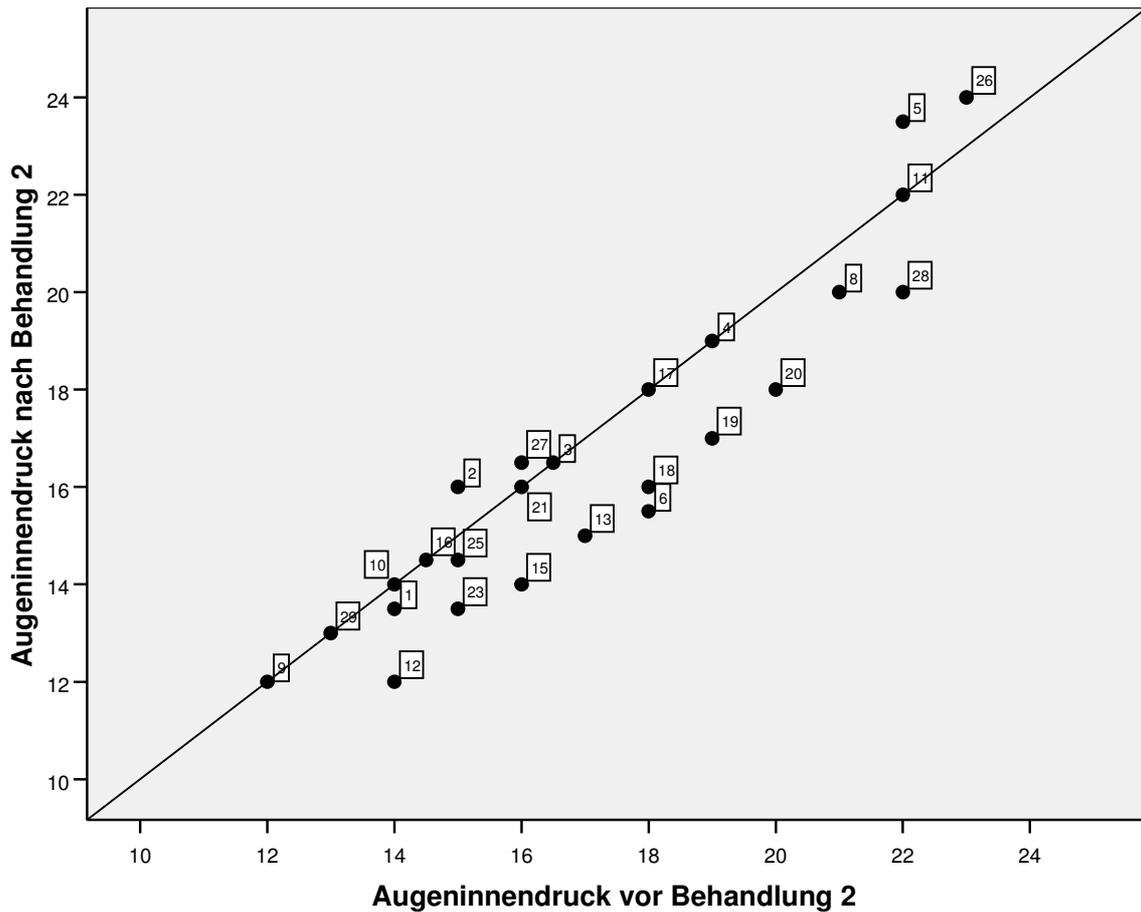


Figure 7.6: individual median intraocular pressure **before** and **after** the second treatment

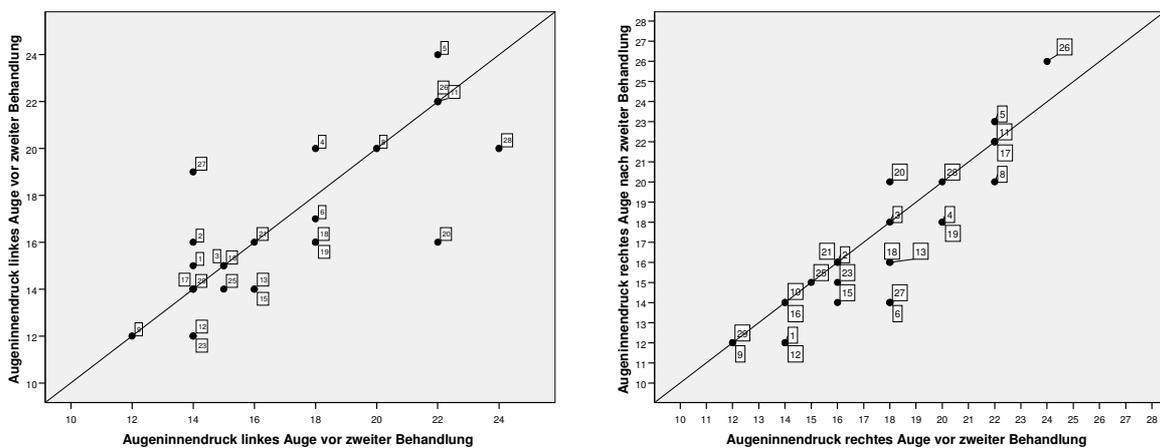


Fig. 7.7 a, b: individual **left** and **right** intraocular pressure **before** and **after** the second treatment

After the **second therapy** unit's treatment, four test subjects (16%) indicated an average higher intraocular pressure than before treatment. The intraocular pressure of nine others remained the same (36%), while 12 subjects achieved a lower intraocular pressure (48%).

The number of other medicines taken did not correlate to a significant degree statistically with the change in intraocular pressure after treatment. Nor was there a statistically relevant correlation between smoker status and change in intraocular pressure after the two treatments.

### **7.3 CHANCES IN INTRAOCULAR PRESSURE IN RELATION TO THE BODY REGION TREATED**

These calculations below were made due to the primary dominances found. These calculations provided only a conditional answer to the hypothesis offered in this study. This raises the question if intraocular pressure can be influenced by osteopathic interventions. Based on the results found, an answer was sought to the special arrangement of dominants found. A further argument was to confirm results found in previous osteopathic studies.

A characteristic of the lymph system is the asymmetrical distribution of outflow routes in the right and left halves of the body. A quarter of the lymph flows to the right and three quarters to the left (see Figure 7.8, which presents the asymmetrical discharge paths of the right and left halves of the body). The lymph outflow paths of the left half of the body have a more complex outflow route than those of the right (Kubik, 1986). Prof. Földi, M.D., divided the human body into regions and described them according to this anatomic basis for the lymph system's outflow routes. This division is used worldwide in the classical lymph drainage treatment for lymph edemas in the conventional sense (Földi, 1999, 13-19).

An outflow blockage of the lymph in the body could also cause an increase in intraocular pressure, since the fluid systems (venous, lymphatic, and arterial) interact with each other. The right side of the head also has less drainage possibilities than the left. *"One could assume,"* Dr. Sutherland says, *"that one cause for glaucoma is an accumulation of fluids that forms blockages along the intracranial membrane wall of the Sinus Cavernosus or the Sinus Petrosus. Possibly a cranial lesion is the cause for a membranous restriction that hinders the venous discharge"* (Sutherland, 1997, 171). Based on these reflections, varying behavior of intraocular pressure could

explain primary dysfunction in the upper right quadrant in contrast to the rest of the body.

This study divided the body into two regions in order to examine the varying effectiveness of treatment by body region in connection with right- vs. left-eye sight. Therefore, the analysis divided patients from the experimental group into two subgroups at each treatment time point. The first subgroup was comprised of patients who showed the primary dysfunction in the upper-right quadrant (right of the median line and above the umbilical line as well as the right half of the face and the right arm). The second subgroup consisted of patients who did not fulfill this criterion or whose primary dysfunction fell outside the upper-right quadrant.

Primary dysfunctions of the individual functional units (Lien LMO, 2007) were recorded in a table based on a sufficiently granular coordinate system. This was transformed once more into two body regions.

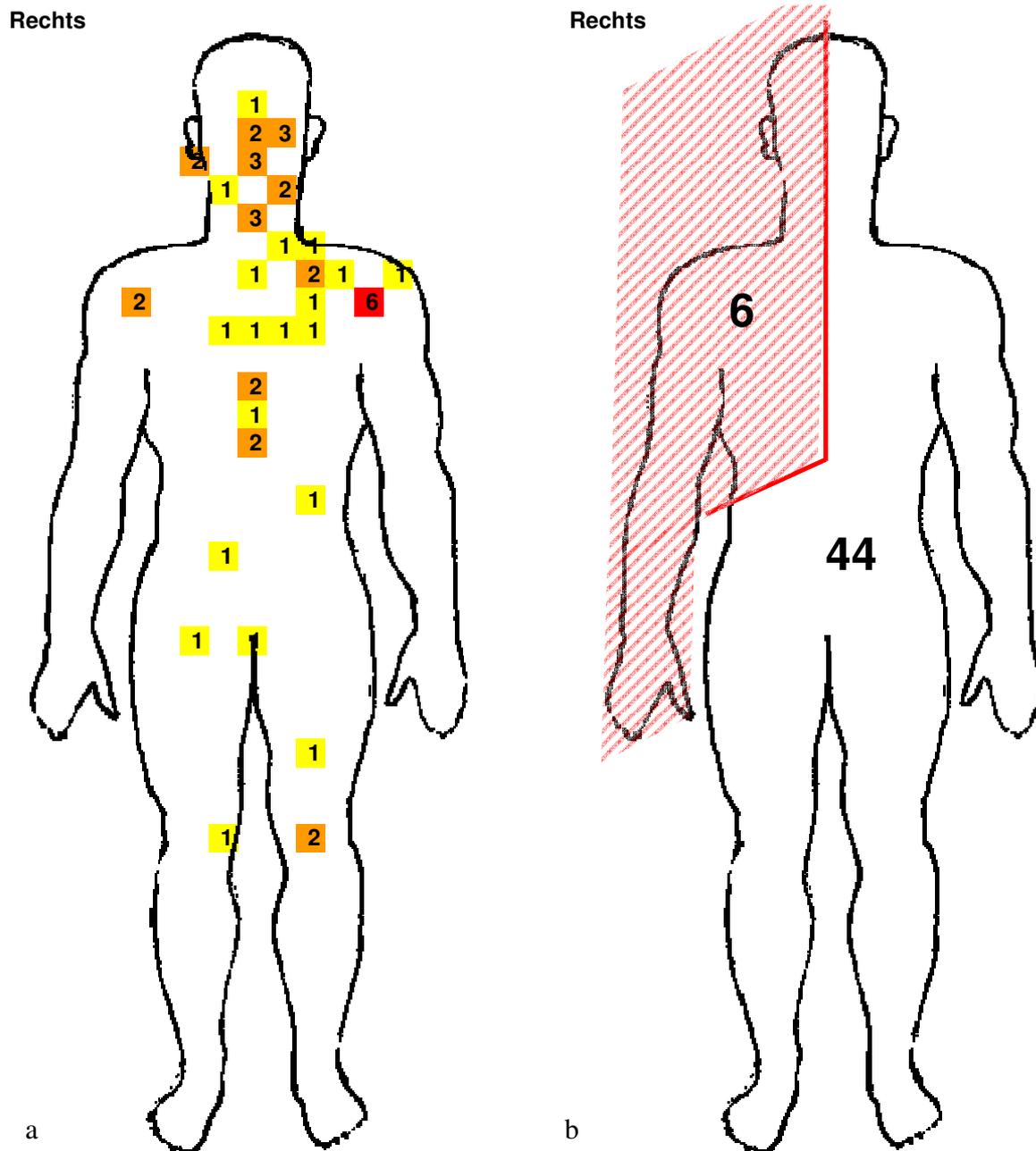


Figure 7.8: Right and left halves of the body

Illustration 7.8a notes all primary dysfunctions that were treated in the course of the two treatments. The primary dysfunction at the first treatment is not necessarily the same as that of the second treatment. Actually the primary dysfunction at the second treatment differed for each patient from the primary dysfunction at the first treatment. However, precisely this focus on individual dysfunctions and carrying out individual treatment approaches distinguishes osteopathic concepts.

Illustration 7.8a shows primary dysfunctions found only once in yellow, while those found twice to five times appear in orange and those found six times in red.

Illustration 7.8b shows the division of the asymmetrical lymph outflow in regard to the right and left halves of the body (Földi, 1986). The part in red hatch-cut shows the lymph flow of the body's upper-right quadrant. The rest of the lymph outflow – put briefly – flows over the left side. The number 6 stands for the primary dysfunctions found and treated in this quadrant, while the number 44 indicates the remainder of the primary dysfunctions treated.

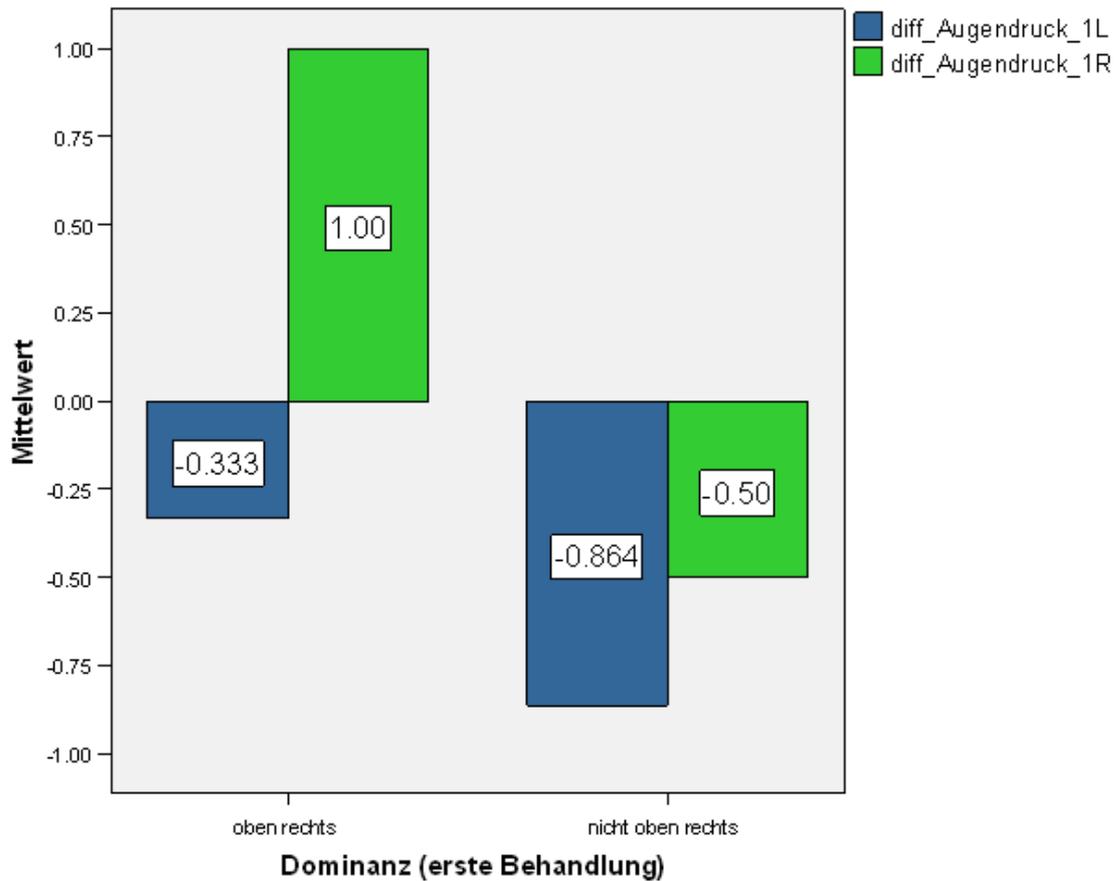
	Group of dominance localisation	N	Mean	Standard deviation	Standard error of Mean
Difference (post – pre) ocular pressure, left eye	Located above right	3	-.3333	2.51661	1.45297
	Not located above right	22	-.8636	2.79958	.59687
Difference (post – pre) ocular pressure, right eye	Located above right	3	1.0000	1.73205	1.00000
	Not located above right	22	-.5000	1.94569	.41482

*Table 7.8: Difference in intraocular pressure in the right and left eyes before and after the first treatment in the experimental group differentiated by localization of dominance*

		Levene-Test of equality of variances		T-Test for equality of means						
		F	Significance	T	df	Sig. (2-sided)	Mean difference	Standard error of the difference	95% Confidence interval of difference	
								lower	upper	
Difference of ocular pressure, left eye	Variances equal	.051	.822	.310	23	.759	.53030	1.70858	-3.00417	4.06478
	Variances not equal			.338	2.725	.760	.53030	1.57079	-4.76704	5.82764
Difference of ocular pressure, right eye	Variances equal	.027	.872	1.264	23	.219	1.50000	1.18664	-.95474	3.95474
	Variances not equal			1.386	2.740	.268	1.50000	1.08263	-2.13811	5.13811

*Table 7.9: T test on variation of difference in intraocular pressure for the right and left eyes before and after the first treatment in the experimental group between the groups of dominance localization*

Descriptive statistics and test of significance for the difference of intraocular pressure in the left and right eyes at the first treatment for patients with primary dysfunction outside the upper right quadrant



*Figure 7.9: Difference of intraocular pressure in the right and left eyes before and after the first treatment in the experimental group differentiated by localization of dominance*

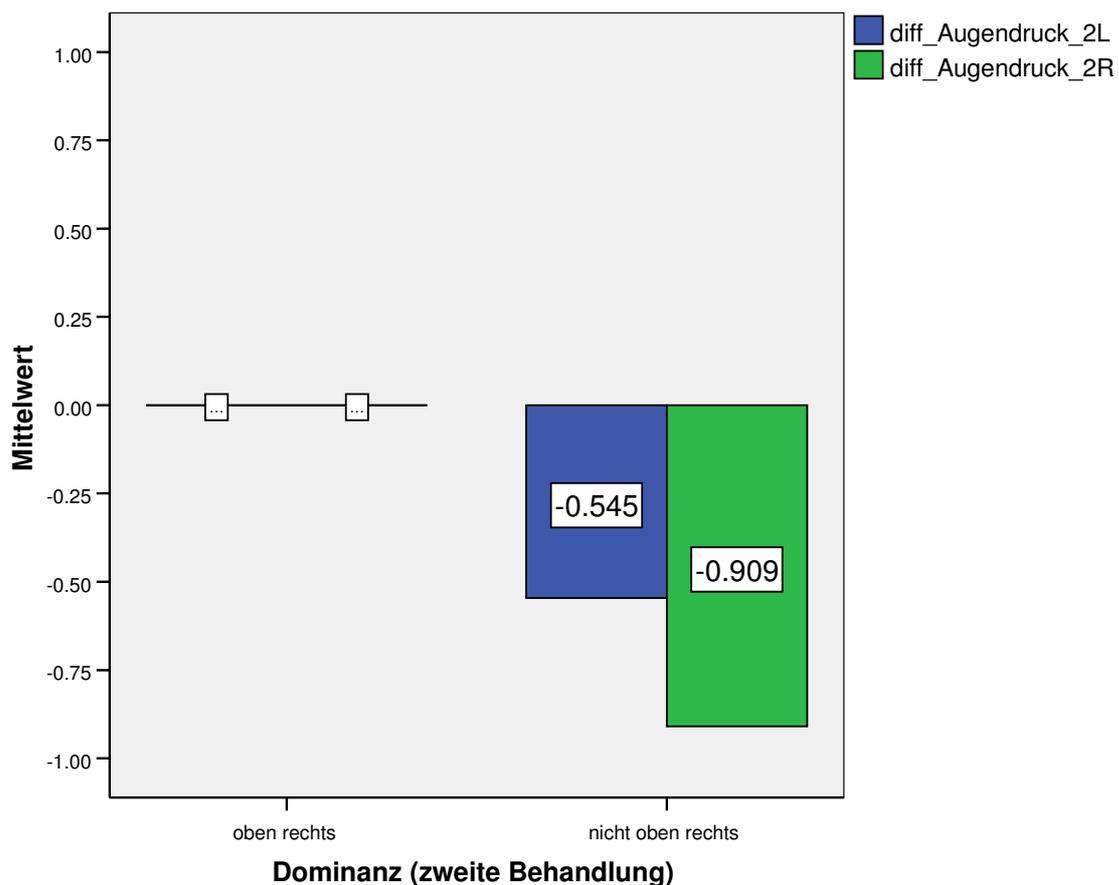
Difference in intraocular pressure through the **second treatment** (differentiated by localization of the primary dysfunction and side of the eye).

	Localisation of dominance	Mean	N	Standard deviation
Difference of ocular pressure, left eye	Located above right	.0000	3	.00000
	Not located above right	-.5455	22	2.28301
	total	-.4800	25	2.14321
Difference of ocular pressure, right eye	Located above right	.0000	3	.00000
	Not located above right	-.9091	22	1.63034
	total	-.8000	25	1.55456

*Table 7.10: Difference in intraocular pressure of the right and left eyes before and after the second treatment in the experimental group by localization of dominance*

		Levene-Test of equality of variances			T-Test for equality of means					
		F	Significance	T	df	Sig. (2-side d)	Mean difference	Standard error of the difference	95% Confidence interval of difference	
									lower	upper
Difference of ocular pressure, left eye	Variences equal	3.678	.068	.406	23	.688	.54545	1.34261	-2.23195	3.32286
	Variences not equal			1.121	21.000	.275	.54545	.48674	-.46677	1.55768
Difference of ocular pressure, right eye	Variences equal	7.573	.011	.948	23	.353	.90909	.95879	-1.07431	2.89249
	Variences not equal			2.615	21.000	.016	.90909	.34759	.18624	1.63194

**Table 7.11:** T test on variance of difference in intraocular pressure in the right and left eyes before and after the first treatment in the experimental group between the groups of dominance localization



**Figure 7.10:** Difference in intraocular pressure in the right and left eyes before and after the second treatment in the experimental group differentiated by localization of dominance

Descriptive statistics and tests on significance for difference in intraocular pressure for the left and right eyes at the second treatment of patients with primary dysfunction outside the upper-right quadrant

Examining the influence of localizing primary dysfunctions found that those test subjects whose primary dysfunction lay outside the upper-right quadrant at the second treatment achieved a significantly greater reduction in intraocular pressure on the right eye than those whose primary dysfunction lay within the upper-right quadrant.

Indeed the **second treatment** managed to achieve an average effect on intraocular pressure (see above, 7.9). A differentiation of primary dysfunctions treated after localization in the upper-right quadrant can provide further information. Unfortunately the scope of the spot check sampling for patients with treatments in the upper-right quadrant was very small (n=3 at the first treatment, n=4 at the second treatment), so that the mean differences found are rather insignificant. Nevertheless, all differences suggest that patients treated whose primary dysfunction lay outside the upper-right quadrant showed a greater reduction in intraocular pressure than patients with primary dysfunctions in the upper-right quadrant. This applies to the examination of both the left and right eyes.

#### 7.4 INFLUENCE OF TREATMENT IN CASE OF PRIMARY DOMINANCE OF THE EYE

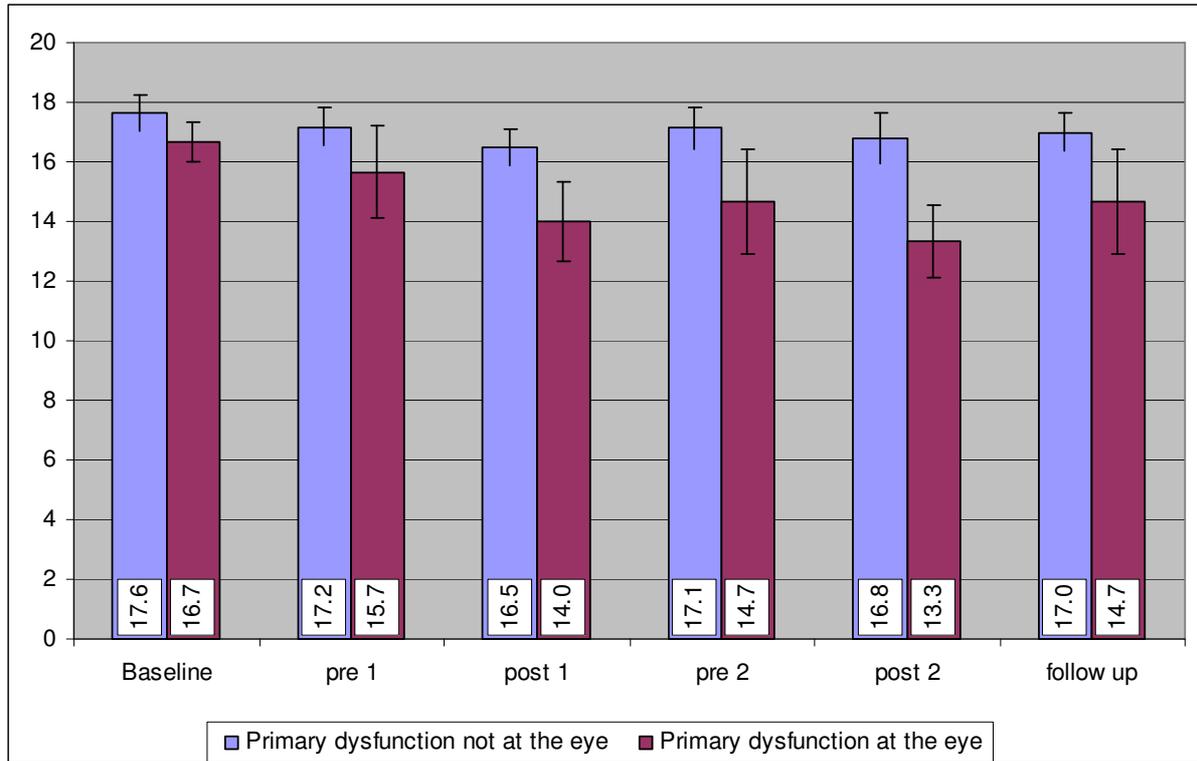
In order to analyze if direct localization of the primary dysfunction at the eye (referred to as “Orbita“) had a greater influence on the course of intraocular pressure, those test subjects were identified who either fulfilled this condition at the first or second treatment. However, the subgroup of the experimental group formed in this manner contained only three test subjects for whom possible differences in the course of the intraocular pressure could at best support a hypothesis but not allow far-reaching findings.

	Baseline	pre 1	post 1	pre 2	post 2	follow up
Primary dysfunction not at the eye	17.64	17.18	16.50	17.14	16.77	17.00
Primary dysfunction at the eye	16.67	15.67	14.00	14.67	13.33	14.67

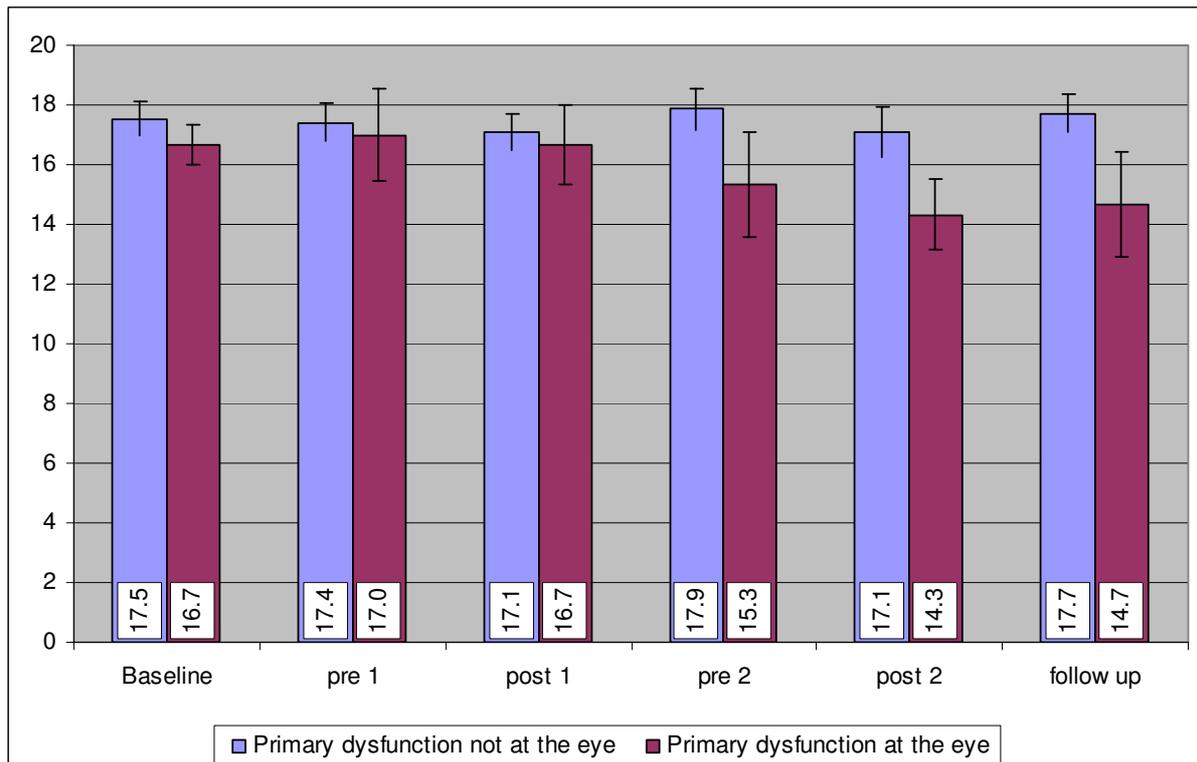
*Table 7.12: Intraocular pressure depending on localization of the primary dysfunction at the eye (left eye)*

	Baseline	pre 1	post 1	pre 2	post 2	follow up
Primary dysfunction not at the eye	17.55	17.41	17.09	17.86	17.09	17.73
Primary dysfunction at the eye	16.67	17.00	16.67	15.33	14.33	14.67

**Table 7.13:** Intraocular pressure depending on localization of the primary dysfunction at the eye (right eye)



**Figure 7.11:** Intraocular pressure depending on localization of the primary dysfunction at the eye (left eye)



*Figure 7.12: Intraocular pressure depending on localization of the primary dysfunction at the eye (right eye)*

Accordingly, osteopathic treatment of test subjects with a primary dysfunction at the **right eye** resulted in a reduction of intraocular pressure from 16.7 mm Hg at the outset to 14.7 mm Hg in the follow-up. This corresponds to a reduction of 12%. On the other hand, test subjects who had no primary eye dysfunction, a reduction of intraocular pressure of 3.6% was recorded (from 17.6 mm Hg before the treatment to 17.0 at the follow-up).

A similar change was observed in the **left eye**: The intraocular pressure among patients with a dysfunction in that eye also amounted here to 12% (14.7 mm Hg at the follow-up compared to 16.7 before treatment), while test subjects with other localizations of the primary dysfunctions even recorded a slight rise in intraocular pressure here of about 1% (17.7 mm Hg compared to 17.5 mm Hg).

A variance-analysis design with a repeated measurement was used to examine the significance of the overall mean value differences.

Source		Sum of Squares Type III	df	Mean Squares	F	Significance
MZP	Sphericity assumed	40,737	5	8,147	1,285	,275
	Greenhouse-Geisser	40,737	3,263	12,486	1,285	,286
	Huynh-Feldt	40,737	4,031	10,105	1,285	,282
	Untergrenze	40,737	1,000	40,737	1,285	,269
MZP * AUGE	Sphericity assumed	23,470	5	4,694	,740	,595
	Greenhouse-Geisser	23,470	3,263	7,193	,740	,542
	Huynh-Feldt	23,470	4,031	5,822	,740	,568
	Untergrenze	23,470	1,000	23,470	,740	,398
Error(MZP)	Sphericity assumed	729,297	115	6,342		
	Greenhouse-Geisser	729,297	75,041	9,719		
	Huynh-Feldt	729,297	92,719	7,866		
	Untergrenze	729,297	23,000	31,709		
SEITE	Sphericity assumed	14,673	1	14,673	1,947	,176
	Greenhouse-Geisser	14,673	1,000	14,673	1,947	,176
	Huynh-Feldt	14,673	1,000	14,673	1,947	,176
	Untergrenze	14,673	1,000	14,673	1,947	,176
SEITE * AUGE	Sphericity assumed	2,206	1	2,206	,293	,594
	Greenhouse-Geisser	2,206	1,000	2,206	,293	,594
	Huynh-Feldt	2,206	1,000	2,206	,293	,594
	Untergrenze	2,206	1,000	2,206	,293	,594
Error(SEITE)	Sphericity assumed	173,347	23	7,537		
	Greenhouse-Geisser	173,347	23,000	7,537		
	Huynh-Feldt	173,347	23,000	7,537		
	Untergrenze	173,347	23,000	7,537		

*Table 7.14: Variance analysis for inner subject factors*

## 7.5 RESULTS FOR SF 36 QUESTIONNAIRE

Data on bodily and emotional ability to function were taken directly before the first and second treatments to supplement the analyses of intraocular pressure and osteopathic treatments. SF questionnaire 36 by Bullinger was used here (Bullinger, and Kirchberg 1998). Accordingly, an influence on this scale can only have occurred at the first treatment (not the second one). However, these inquiries were only carried out on the experimental group, not the control group. Thus a conclusion on a causal correlation for change due to treatment is not justified. Nevertheless, the findings from this questionnaire might provide significant results.

	pre 1st treatment	pre 2nd treatment	Significance
SF-36 physical functioning (0-100)	84.8	84.4	n.s.
SF-36 physical role function (0-100)	74.0	88.0	p<0.05
SF-36 physical pain (0-100)	79.2	78.4	n.s.
SF-36 health perception (0-100)	73.4	71.7	n.s.
SF-36 vitality (0-100)	72.2	72.6	n.s.
SF-36 social functioning (0-100)	93.0	90.5	n.s.
SF-36 emotional role function (0-100)	90.7	89.3	n.s.
SF-36 psychic wellbeing (0-100)	84.0	84.3	n.s.

*Table 7.15: Changes in scales of the SF36*

It follows from the table that only the bodily role function has improved significantly (one-sided) from the first to the second measuring point. Bodily role function means the self-estimated ability to perform as, for example, “be active longer than usual, performed less, etc.” (Bullinger and Kirchberg, 1998). The other seven subscales of the SF36 show no significant changes.

## 8 DISCUSSION

The discussion is divided into five sections.

### 8.1 HYPOTHESIS EVALUATION

The purpose of the study was to determine the effectiveness of osteopathic treatments for patients with increased intraocular pressure experimentally. In comparison to existing studies that used at least identical techniques among all patients treated (Esser, 2002; Bilgeri, 2006), should primary dysfunctions detected here individually be treated individually with individual techniques? The call for a scientific approach should bear in mind that both an experimental and control group were used. This approach had not been selected in one of the studies cited (Esser, 2002), so that this study possessed only conditional significance. The hypothesis that it is possible to reduce intraocular pressure with an osteopathic treatment approach has only been confirmed with reservations.

### 8.2 DISCUSSION OF METHOD

Various studies (Esser, 2002; Bilgeri, 2006) have pointed out that undifferentiated treatments carried out with identical techniques and sequences contradict

osteopathic thinking, and therefore no systematic investigation of the effectiveness of such osteopathic treatment can be applied in a sweeping manner. Therefore, they recommend osteopathic intervention or use of an individually adapted treatment. On this basis I have decided in my study to orient treatments individually to patients and their current problems. The Lien Mécanique Osteopathique (LMO) method was used to investigate the primary dysfunctions (Chauffard, 1978). This method has the advantage of allowing all patients to be examined and treated according to the same plan and pattern. A study on the reliability of this method is being carried out at present at the Vienna School of Osteopathy (WSO). The disadvantage of this method is that a finding of possible effectiveness in using individual treatments with individual techniques on individual structures cannot be assigned a clear causal relationship to a specific technique. Thus the treatment as an entity remains unfathomable and is difficult to justify as empirical science. Osteopathic treatment as a subject of scientific inquiry will therefore also face a great many questions in the future that will hardly be scientifically comprehensible through statistics. Nevertheless the attempt is made in this study to prove the effectiveness of individual osteopathic treatment approaches based on scientific criteria.

The student subjects were recruited from a patient pool that had been known by the physician for a long time. Hence one could assume that the measurement data indicate a certain level of reliability.

### **8.3 DEVELOPMENT OF INTRAOCULAR PRESSURE VALUES FOR THE EXPERIMENTAL GROUP**

Intraocular pressure values have developed similarly to those in the studies of Esser (2002) and Bilgeri (2006). My measurements too showed a certain influence on average intraocular pressure, at most right after treatments. A while after the treatment (that is, directly after the second treatment or at the follow-up) a slight reduction in intraocular pressure indeed showed up in comparison to the start-up value, though no statistical relevance could be proved. Similar to Esser's findings, a clear difference could be noticed between eye pressure measurements of the left and right eyes. I was able to confirm in my study that the intraocular pressure of the left eye is more easily influenced than that of the right.

It should be mentioned here that both eyes behave differently depending on treatment and the region of the body treated. I knew from my earlier D.O. work about

the lymph system's peculiarity. The first results of this study provided my question to what degree the lymph system and intraocular pressure could correlate. Already during my literature research on other studies the opportunity arose to note the varying reactions of the right and left eyes. Since I had registered all primary dysfunctions in a precise scheme, a statistical inquiry based on this data could now be carried out. The anatomical peculiarity of the lymph system could now be considered here.

The thought to arrange the primary dysfunctions precisely in a system of coordinates and quadrants appropriate for the lymph system helped here crucially in making calculations. The sum of all primary dysfunctions in the upper-right quadrant numbered six, while it amounted to 44 in the rest of the body. One can only make note of this fact, since it has no scientific relevance.

Nonetheless, I have tried to bring the number of primary dysfunctions of the upper-right quadrant into a correlation with the intraocular pressure on the same side. It is in fact true that lesions in the upper-right quadrant and the intraocular pressure in the right eye cannot be influenced as well and the intraocular pressure decreases less here. It seems to behave in such a manner that the primary lesions in the upper-right quadrant do not allow themselves to be influenced as much in connection with intraocular pressure. The question also arises if one may produce a correlation here at all. Other studies in this direction would surely confirm or reject my hypothesis or theory.

Osteopathic literature refers again and again to the correlation of anatomic facts. The reason why the left and right halves of the body have a different route for lymph outflow paths has not been precisely explained to this day. This makes it all the more exciting to ask over and over "Why is this so?" The question why the right side or the right eye seems more difficult to influence remains unanswered. It's exciting and inspires other observations. The fundamental thought of osteopathy – "Everything must continue to flow" – has a deep physiological meaning that again clearly steps to center stage in the glaucoma study.

The articles and essays of Still, Frymann, and also Millard remind us over and over how important the exchange of body construction materials is at the cellular level. If the environment is impaired – *e.g.*, by a lymph edema – its chance of survival decreases (Still, 1889). Every little cell needs information, be it neuro-hormonal or

mental. Only in this way does auto-regulation of the cells emerge – both neuro-hormonal but also mental (Glenhofen, 1997).

The study must be carried out over a long time period and with more test subjects in the control group as well as in the experimental group. Thus the experimental group would be larger. One could compare two or three different experimental groups with each other. It would then be possible to evaluate the methods and techniques in a direct comparison.

I would recommend that the LMO method be involved again in the study. I would also involve the lymph system and would obviously treat the regions affected directly. Hence the direct comparison to the individual groups treated differently might be clearer.

#### **8.4 DISCUSSION ON WORKING FUNDAMENTALS**

The LMO's reporting on findings and method of treatment shows a measure of validity and can be upheld under certain circumstances. It is important here to harmonize the test well and perhaps to carry out the study with several therapists at different locations at the same time. It would be interesting to see how the influence of the right side and its intraocular pressure would behave in regard to the anatomic peculiarity of the lymph systems in the upper-right quadrant. Here the question also arises if, after treating the primary dysfunction, one should still treat the affected eye as well in a special way. This does not correspond directly to the LMO concept but should be considered an option at the moment. A possible answer is the result that can be seen in Item 6.4. The eye was found to be directly affected in only three primary dysfunctions. A direct intervention with a LMO technique seems to have an influence here. This raises the question for me if it is still not important to question the concept. If an obvious problem which can be verified (intraocular pressure and measurement results) were not treated directly, would the LMO abuse the ethical components that belong to treatment. Finally I would gladly extend the LMO concept by one unit. I think here especially of the lymph system that to this date is not foreseen in the concept as such. Ignoring the lymph system is a great loss in this still exciting concept.

## **8.5 SF-36 QUESTIONNAIRE**

The SF-36 was distributed twice and showed only one parameter that had improved significantly. Here a third questionnaire before the final measurement might have been able to deliver even more results.

The control group should also have been provided the SF-36 questionnaire. The study must be carried out over a longer period with more test subjects in the control group as well as in the experimental group.

## **8.6 PERSONAL THOUGHTS**

This study has brought me a giant step nearer statistics and scientific thought. I have also been pushed to the limit. Here I would like to mention particularly work on the computer. At the beginning of the study it demanded a great deal of me.

Just as after the D.O. work, my hunger and curiosity for a correlation between the anatomy and physiology has grown still greater. This is a plea, and osteopathy should heed it: It should finally focus its eyes on the lymph system – or at least on its direct work on involvement with patients. During the entire study this thought pursued me. In all the literature, physiology books, osteopathy books point out the importance of blood circulation and the nutrition related to it. The lymph system is constantly mentioned, but it is treated directly only seldom or taught as an important element in the schools. It is amazing to have to determine that many osteopaths disregard this fact.

If I had to repeat this study again today, I would change the following points; first I would demand more time, possibly adding a semester.

Thus the experimental group could be enlarged, so one could evaluation 2-3 different methods and techniques directly in the comparison with one another.

## **9 CONCLUSION**

Glaucoma illnesses will increase 34% by 2030 (Michelson, 2008). By now the possibility exists to treat this illness with medications and laser therapy. This study was undertaken to influence intraocular pressure by osteopathic intervention. The ulterior motive here was to permit the patient to receive an additional osteopathic treatment that should result in possible reduction of medications taken.

A single-blind, randomized, clinical study was undertaken with the goal and hypothesis that one could influence intraocular pressure with osteopathic intervention, but the hypothesis was not confirmed satisfactorily.

The data registry and treatment occurred according to the Lien Mécanique Osteopathique (LMO) method developed by Paul Chauffour and Eric Prat (2003, 38ff). Chauffour developed the concept in 1978. This method served as the working basis.

Some 25 people were recruited for an experimental group and 20 others for a control group, each receiving two osteopathic treatments. The experimental group was measured six times, the control group three times during the same time period.

The amount of change in intraocular pressure between the first and second treatments was not statistically significant ( $r=0.256$ ,  $p=0.217$ ). The test subjects who reacted positively with changed intraocular pressure at the second treatment were not necessarily the same (or represented in the same numbers) as those who also indicated this effect at the first treatment.

At the **second unit of therapy** the treatment resulted in a higher average intraocular pressure than before the treatment for four test subjects (16%), while nine subjects maintained the same intraocular pressure (36%) and 12 others achieved a lower intraocular pressure (48%).

Over the entire observation period the experimental group showed an average 2.5% lowering of mean intraocular pressure. Yet the eyes differed sharply in their involvement: while the right eye recorded a slight decline in intraocular pressure of 0.5%, the left eye decreased by 4.6%. It is also obvious that the intraocular pressure decreased directly after the treatments, while it had risen again before the second treatment. The average level of change in intraocular pressure over both eyes achieved a lower mean within the control group than in the experimental group: a decrease in mean intraocular pressure of 0.4% is recorded here.

**Osteopathic intervention** has a positive influence on intraocular pressure. Clear tendencies can be read from the measurement data in this study. It has the character of a pilot study and should be pursued further in any case with an enlarged experimental group and an equally large control group from which one can and should acquire more data.

## 10 LIST OF ILLUSTRATIONS, TABLES, AND FIGURES

The illustrations and tables are arranged by chapters and numbered sequentially.

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- Figure 5.2      Course of the N. Optikus
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## 12 APPENDIX

### 12.1 HISTORY OF OPHTHALMOLOGY

Ophthalmology seems to have been practiced in Egypt since the ancient empire, and the knowledge of the epoch was imparted over centuries. Only the Mesopotamians could look back on an equally long past in this specialty.

The first writing on healing eye diseases appeared in the 3<sup>rd</sup> century BC and also contained magic practices. Egyptian ophthalmology is fortunately well preserved, due to precise sources well known. However, medical texts on papyrus are of utmost importance.

One found surgical instruments in graves only rarely. For instance, one recently discovered a small bottle in a grave near the pyramids containing an eye preparation that had been preserved. The inscription, „good antimony, good for vision, removes the infection, and relieves pain.“ In ancient Egypt, eye preparations were applied in liquid or viscous form as well as as salves or lotions. Many of these substances could still be analyzed, thanks to residues in the vials. All sorts of medicinal plants were added: *e.g.*, safran, rose, myrrh, incense, and acacia. Goosefat and honey found use as binders, and so did bile and blood as well as animal excrement and urine. Copper sulfate was used to combat conjunctivitis. Lead salt and brine as well as *Lapis lazuli* powder were applied for make-up. Many of these formulas in the history of ophthalmology were used for centuries. The formulas passed from the Egyptians to the Greeks, from there to the Romans and Gauls before they came to the Arabs and thus to the ophthalmologists of the Middle Ages. Astoundingly, many of these medical elements still find applications today.

Physiology was widely developed. Hence one paragraph was written on papyrus by Elber: “Vessels of the heart are in all limbs, and there are four vessels in the temples that lead blood to both eyes and then produce all fluids that moisten the eyes. The openings in the nose are vessels that transmit to the eye sockets.“ From today’s viewpoint, these findings are hardly worth mentioning, but they were pioneer work for the early history of medicine

At the time, the Egyptians laid the cornerstone of today’s ophthalmology (Ophthalmology by Marc-Adien Dollfus).

### 12.1.1 Hippocrates

Hippocrates described a conjunctivitis epidemic in his book "*Folk's Diseases*". Here he also described the connection between a patient's general condition and the risk of falling ill with conjunctivitis. In case of a generally poor condition, the probability of falling ill with conjunctivitis increases (Deichgräber, 1933).

### 12.1.2 The Romans

The Romans promoted surgical interventions and developed them further. One Roman named Celsus described his surgical interventions with great precision and thus preserved them for posterity. He also describes surgical instruments such as the use of forceps. Influenced by the school of Alexandria, he was the first to carry out sections in Rome. He pointed out that the brain's nerves might be inhibited voluntarily and sensitively.

## 12.2 THE MIDDELAGE

- There was almost no development from the 9<sup>th</sup> to the 14<sup>th</sup> century in the ophthalmology sector. Indeed the church recognized the need for medical studies, but it pointed out repeatedly that folk medicine was linked with magic.
- Use of glasses was mentioned for the first time in the 13<sup>th</sup> century, which marked the onset of magnifying glasses.
- The optician was mentioned for the first time in the 15<sup>th</sup> century. The first corrections contained convex glasses to treat age-related farsightedness.
- Only in the 19<sup>th</sup> and 20<sup>th</sup> century did one differentiate between the various corrections.
- The ophthalmologist John Taylor (1703-1772) described for the first time the phenomena of the "supersaturated eyeball, which immediately destroys the eyesight". The hypertension was treated by biopsy using a trocar (a cannula, which the surgeon can penetrate a body cavity). This served to remove liquid from the eye. Over the course of years, this knowledge lost its importance and was nearly forgotten.
- Only in 1818 did a man named Demours return to the topic. From this point on, ophthalmology developed swiftly.
- Anel (1679-1730) developed a method to catheterize the tear ducts by flexible probes that are still in use today.

- The first lens extraction took place in 1752.
- The first chair for ophthalmology was created in Paris in 1880.
- The first Braille alphabet was developed in 1809, and the first school for the blind opened the same year.
- Use of cocaine as a local anaesthetic was first mentioned in 1884.
- Discovery of penicillin and antibiotics in 1944 made many diseases curable.

## 13 PATIENTS' INFORMATION SHEET

### Patient Information

The study focuses on patients with increased intraocular pressure.

Thank you very much for taking a few minutes to read this document carefully.

Osteopathy is a manual diagnosis and a treatment concept that traces back to the American physician, Andrew Taylor Still (1828-1917). According to Dr. Still, disturbances and restrictions in moving the joints can release symptoms at other regions of the body. Specific manual gripping techniques make it possible to relieve blockages and thus related disturbances. A principle of osteotherapy is a comprehensive inquiry into symptoms and the course of a disease. The osteopath always seeks to find the major dysfunction or disturbance. Another important principle of osteopathy is a broad training in anatomy as well as that found in pathology.

Osteopathy remains an unknown method of treatment in Switzerland. Elsewhere in Europe and the United States it has been practiced for 80 years. In order to offer more conclusive evidence about osteopathy and its effectiveness, it is necessary to carry out studies. The University of Vienna in Krems and the Vienna School for Osteopathy have enabled me to carry out this study.

Based on your increased intraocular pressure, you were selected by M.T. Kammann, M.D., to take part in this study. The total duration of the study amounts to about four months. During this period your intraocular pressure will be measured six times. In the time between measurements you will be treated twice osteopathically. The first time Dr. Kammann will examine you during a consultation. The other intraocular measurements will take place shortly before and after the osteopathic treatment. The final intraocular pressure measurement will occur four weeks after the last osteopathic treatment. All intraocular pressure measurements will take place in the practice of Dr. Kammann. The measurements and osteopathic treatments will require two hours on the same afternoon.

- During this period, please take the medicines exactly as the physician prescribed.
- Please inform us immediately if you have unexplainable complaints. Osteopathy treats the entire body and can produce and cause symptoms in other regions of the body.
- Please do not begin a diet during this period.
- Please inform us if you begin other forms of therapy during the study period (acupuncture / craniosacral therapy, physiotherapy, etc.).

The measurements as well as the treatments are free.

If you are interested and in agreement with the framework conditions, I ask you to sign the declaration of agreement and give it to Ms van de Kraats at the first treatment.

Measurements and osteopathic treatments

Please register for the intraocular pressure measurements at:

Marc Kammann, M.D.  
Bahnhofstrasse 3  
7270 Davos-Platz  
Tel. 081 410 60 70

Register for the osteopathic treatments at:

A. van de Kraats  
Gesundheitszentrum Grischuna.  
Bahnhofstrasse 1  
7272 Davos-Platz  
Tel.081/413 22 55  
e-mail: info@praxis-grischuna.ch

Please mention at registration that you are taking part in the study.

**14 PATIENT LINEAGE SHEET****Patient's lineage sheet:**

Name:

Age

Address:

Family medical history:

Related illnesses:

Randomizing number:

Profession:

Hobbies:

Sports:

- Heart/circulation:
- Blood pressure:                      high                      low
- Arteriovenous complaints
- Lymphovenous complaints
- Diabetes II (insulin resistance)                      Diabetes I
- Thyroid illnesses
- Other organs
- Neurovegetative complaints
- Operations
- Fractures

## 15 DECLARATION OF AGREEMENT

# Declaration of Agreement

I declare myself prepared as a patient to take part in Angelika van de Kraats' study „*Can increased intraocular pressure be influenced by osteopathic treatments?*“ I agree that:

- M. T. Kammann, M.D., at the beginning of the study, may carry out intraocular pressure measurements on me before and after each treatment as well as at the end of the study period.
- Ms A. van de Kraats treats me twice osteopathically in her practice.

This will occur under the following conditions:

- All my personal data will only be used for the study mentioned above and will only appear there in anonymous form. Personal protection will be guaranteed.
- Treatments that take place within the framework of the pilot study will be carried out free of charge.
- The eye measurements by Dr. Kammann are also to be administered free of charge.

I confirm herewith that I have read the Declaration of Agreement carefully and am in complete agreement with all points covered.

Location, date, and signature:

**16 RANDOMIZING LIST**

## Randomizing List for Glaucoma Study

(Please enter names in chronological sequence. Thank you!)

No.	Group	Name
1	C	
2	E	
3	C	
4	E	
5	E	
6	E	
7	C	
8	E	
9	C	
10	C	
11	E	
12	E	
13	C	
14	C	
15	E	
16	C	
17	E	
18	C	
19	C	
20	E	

## 17 PHYSICIAN'S TIMETABLE OF APPOINTMENTS FOR INTRAOCULAR VALUES

Timetable of appointments for intraocular values

Name:	Randomized No.
-------	----------------

Startup measurement		Intraocular value mm Hg		
Date:		R:  L:		

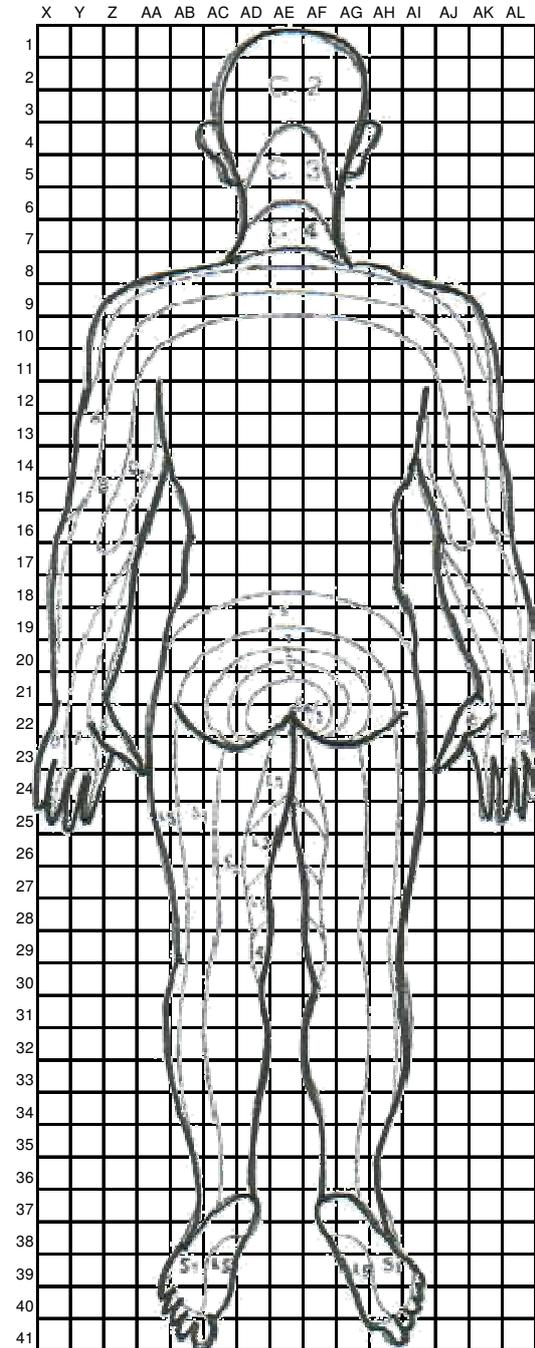
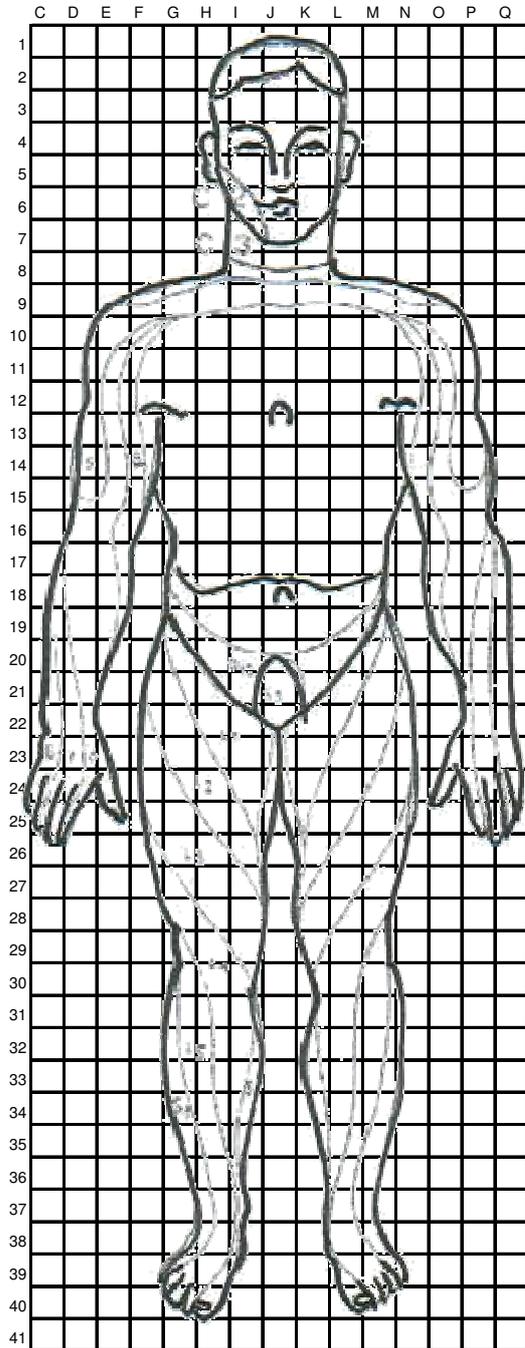
First measurement before first treatment			Second measurement after first treatment	
Date:		R:  L:		R:  L:

First measurement before second treatment			Second measurement after second treatment	
Date:		R:  L:		R:  L:

Measurement after break in treatment:				
Date:		R:  L:		

Possible measurement:				
Date:		R:  L:		

### 18 COORDINAT SYSTEM OF DOMINANCES AND PRIMARY LESIONS



## 19 LIST OF THE TREATED DOMINANCES AND REGISTERED LESIONS

Dominances 1 (registered at 1st examination) and dominances 2 (registered at 2nd examination), these dominances were treated by recoil.

<b>Nr.</b>	<b>Dominanz 1</b>	<b>Koordinaten</b>	<b>Dominanz 2</b>	<b>Koordinaten</b>
1	Atlas	6 AE	Ethmoid	5 J
2	Mastoid li	K 7	Schulter li	11 N
3	Atlas	6 AE	Maxilla re	7 I
4	Mastoid re	6 H	Sternum	12 J
5	Knie med re	30 I	HWS 3/4	8 AE
6	Proc. Xypho.	15 J	A. Vertebralis li	18 L
7	Tuber re	23 AG	Schulter li	11 N
8	Clavicula li	10 M	Schulter li	11N
9	Ethmoid	5 J	HWS 5/6	8 AG
10	4/5 Rippe Ant. Li	12 L	Schulter re	11 F
11	Schulter li	11 N	Fillum	23 AE
12	Knie re	30 L	Orbita li	5 K
13	Mastoid re	6 H	Schulter li	11 M
14	HWS 5/6	8 AE	Lunge re	12 I
15	Diaphragma	15 J	Schulter li	11 N
16	Lungenspitze li	9 AC	HWS 5/6	8 AE
17	A. Vertebrali li	K 9	Femur li	27 L
18	Os Frontale	4 J	Thoracal 1/2	10 AE
19	Clavicula li	10 L	Bronchien li	11 L
20	Herz	12 K	Orbita li	5 K
21	Acromion li	10 O	Knie li	30 L
22	Clavicula li	10 L	Sigmoid	20 I
23	Schulter re	11 F	Mastoid li	7 K
24	Thoracal 12	16 AF	Proc. Xyph	14 J
25	Orbita li	K 5	Thoracal 12	16 AE

Primary lesions at 1st or 2nd examination, not treated

<b>Nr.</b>	<b>prim Läsion 1</b>	<b>Koordinaten</b>	<b>prim Läsion 2</b>	<b>Koordinaten</b>
1	Th 12	16 AE	Isg re	20 AF
2	Rippen re	16 G	Hüfte li	22 M
3	12 Rippen re dorsal	16 AG	Tuber li	23 AC
4	Zygoma re	6 J	Tuberos. Tibia re	32 H
5	Orbita li	5 K	A. Vertebral re	8 H
6	ISG li	21 AC	Hüfte li	22 M
7	Isg re	21 AE	Sternum	12 J
8	Sternum	12 J	Hand Gelenk re	23 D
9	Zygoma li	5 L	Art. Vertebralis li	9 K
10	ISG LI	21 AC	Hand Gelenk re	24 D
11	Parotis	8 I	Sternum	13 K
12	Augapfel	5 K	L5/S1	20 AE
13	Proc. Xyph	14 J	Darm pars .des.	17 L

14	Sternum	12 J	Proc. Xyph	14 J
15	Sternum	12 J	Clavicula li	10 L
16	Schulterblatt li	11 AE	Proc. Xyph	14 J
17	Proc. Xyph	14 J	Leistenband li	21 L
18	Sternum	11 J	Proc. Xyph	14 J
19	ISG re	21 AC	Acromion re	10 E
20	Hand Gelenk	24 E	Lig. Sacrotub. Li	23 AF
21	Schulter li	11 N	Hüfte re	22 G
22	OSG li	39 L	Orbita li	5 K
23	Rippen ant. Re	13 H	Knie li	30 K
24	Ethmoid	5 J	Rippen ant. Li 12	16 AG
25	ISG re	21 AF	Niere re	17 AG

## Primary lesions at 1st or 2nd examination, not treated

<b>Nr.</b>	<b>prim Läsion 3</b>	<b>Koordinaten</b>	<b>prim Läsion 4</b>	<b>Koordinaten</b>
1	A. Vertebralis re	8 L	Knie re	30 AF
2	Knie re	30 H	Spina Scapula re	10 AG
3	Sternum	12 J	Prostat	22 J
4	Metatarsus	40 M		
5	Acromion li	10 O	Lig. Coll. Phren.	16 M
6	Leber	15 I	Bregma	2 J
7	Zäkum	20 I	Th 12	16 AE
8	Insisura Jug.	9 J	Hüfte li	22 M
9	Magen	15 K	Knie li med	30 K
10	Knie re	31 I	Maxilla re	7 J
11	Hüfte re	22 G	Trachea	9 J
12	Clavicula re	10 H		
13	Knie lat re	31 G	Th 12	16 AG
14	Ellbogen lat. Re	18 E	Diaphragma	15 J
15	Tibia li	32 L	Orbita li	5 K
16	Sternum	12 J	Aortabogen li	11K
17	Knie lat re	27 L		
18	Knie re	32 H	Maxilla re	7 I
19	Os Frontale	4 J	Prostat	22 J
20	ISG re	20 AF	Knie med re	31 I
21	Rippen ant. 5/6	13 I	OSG li	39 L
22	L5/S1	20 AE	Spina Scapula	11 AC
23	Knie re med.	30 AF		
24	Sacrum	22 AE	Spina Scapula re	11 AG
25	Lunge re	13 H	Knie li	30 L

## Primary lesions at 1st or 2nd examination, not treated

<b>Nr.</b>	<b>prim Läsion 5</b>	<b>Koordinaten</b>	<b>prim Läsion 6</b>	<b>Koordinaten</b>
1	Radius dist. Re	23 C		
2				
3	Galle	16 I	Prostat	22 J
4				
5	Trochanter li	22 N	Fillum	23 AE

6				
7	Maxilla re	7 I		
8	OSG li	39 L	Os Frontale	5 K
9	ISG re	21 AF	Leistenband	22 I
10	Sigmoid	20 L	Os Frontale	5 K
11	Maxilla re	7 I	Zygoma	5 I
12				
13	grosser Zehe li	41 L		
14				
15	Tibia re	36 H		
16	Mastoid li	7 K	Tuber li	23 AC
17				
18	Clavicula li	10 L	TH 2/3	11 AE
19	Art. Vertebralis li	9 L	Kniegelenk lat re	31 G
20	Mastoid re	6 L	Beckenkam	20 M
21				
22				
23				
24	Hüfte	21 AG	Tibia med re	32 J
25				

Primary lesions at 1st or 2nd examination, not treated

<b>Nr.</b>	<b>prim Läsion 7</b>	<b>Koordinaten</b>
1		
2		
3		
4		
5		
6		
7		
8		
9		
10		
11		
12		
13		
14		
15		
16	Ethmoid	8 AE
17		
18		
19		
20		
21		
22		
23		
24	Clavicula li	10 L
25		

## 20 ACKNOWLEDGEMENTS

This work would never have been completed without the assistance of the following people:

Marc Tell Kammann, M.D., and his super team, without whose vital input the study would have been totally impossible;

My mother, Johanna van de Kraats, who accompanied and supported me during the past year in all aspects of my life;

Stefan Lippitsch, who energetically introduced me to all the secrets of statistics and accompanied me with unlimited patience in their evaluations;

Helga Haas, who carried out the editing and corrections work, transforming my midnight text into scholarly German;

Lyn and Hanni Shepard for the time spent in the English translation;

Ulla Röhrig, who prevented a total PC crash with all my data, rescuing and saving it;

My little cat Gini, who spent many hours in the office with me and shortened the lonely time at the PC;

All my friends - Claudia Schawalder, Vladimir Pillman, Hanne-Garsdal Bosshard-Jespersen, Ruth Peter, Andrea and Lilli Clavadetscher, Hanspeter and Monika Kirchhofer, Ruth de Falco, and Isabelle Daigl - who cooked, accompanied, and guided me;

Claudia and Felix Hafen-Bardella, whom I want to thank for their motivation and critical commentaries.