Titel der Masterthesis

Können osteopathische Behandlungen nach der „Black Box Methode“ das subjektive Wohlbefinden – also das Zustandsbild des Patienten mit einfachen Migräne – bei gleich bleibender Medikation verbessern?

Master Thesis zur Erlangung des Grades
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von **Maria Spannbauer**

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Korrektur gelesen von Christine Lang (Native Speaker)

Eidesstattliche Erklärung

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Abstract

Maria Spannbauer, Mai 2008

Can osteopathic treatments improve the overall picture of the patient with a common migraine, specifically the subjective well-being using the „Black Box Method", with consistent intake of medication?

In the civilised world **migraine is a common disease**. Migraine affects people of all races, ages, cultures, personality types, occupations and income levels. Migraine is an important target for treatment because it is not only common, it is **disabling** and **costly** and it has major **comorbidities**. On the one hand this is an economic burden due to missed work, absence or reduced efficiency and, on the other hand, each individual suffers from **reduced quality of life**. Therefore it is the major target of my study to proof the efficacy of osteopathy in the treatment of common migraine.

Migraine forms a clinical picture which is influenced and released by multiple factors (**trigger factors**). Osteopathic treatment takes the whole person into consideration and seeks to recognise multiple influences and connections and to develop from them an individual "red thread" throughout the therapy.

If improvement of the symptoms succeeds in such a multifaceted and complicated illness like migraine through osteopathic treatment, this would be further proof of the need for individual treatment plans and it would be another success for the general idea of osteopathy.

Twenty-six patients took part in my study. With the help of the MIDAS questionnaire – which was developed especially for the verification of the migraine – I evaluated the state of the patient three times: three months before beginning therapy, after ten treatments and three months after ending the therapy. The patients thereby operated as their own control group (within subject design) and I could document long-term results. After taking an exact anamnesis with every patient, and have carried out a thorough examination, an individual treatment plan was made. According to the needs of my patient – and using the "Black Box Method", I chose structural, visceral and cranio sacral therapy approaches.

The considerable improvement in the conditions of the patients in their own opinions is a very rewarding outcome of this study.

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1. Introduction

In the civilised world migraine is a common disease. Because of this, physicians and scientists are engaged with this topic continuously. In current research this topic has important significance throughout the world. In the following statements the current standard of knowledge in research for migraine is reflected.

Migraine is a common medical disorder that occurs everywhere (Lipton, 2001). Migraine characteristics are associated with impaired functioning and quality of life (Holroyd, 2007). Detailed requirements for the onset of migraine are not yet clear. (Luthringshausen, 2006). Migraine affects people of all races, ages, cultures, personality types, occupations and income levels (w-h-a, 12/2006). Lipton confirms that migraine is common, disabling and costly and, because it has major comorbidities, migraine is an important target for treatment (2007). The International Headache Society differentiates more than 200 different headache characteristics. Diener (2006) says that 90% of all headaches lead back to two different headache characteristics: “migraine and tension-type headache”.

In Austria, too, this illness is widespread, as seen in a census done by Statistik Austria in 2006/2007 where questions were asked about the health of Austrian people. One outcome was that every fourth female Austrian and every ninth male Austrian suffers from migraine. Not only the person concerned is reduced in quality of life – which is explained later - it strains the national economy of Austria. Employees affected by migraine perform less productively for their companies during an attack and / or are away sick for one or several days. Therefore, the general public is interested in reducing the severity and frequency of migraine attacks.

On one hand this is an economic burden due to missed work, absence or reduced efficiency and, on the other hand, each individual suffers from a reduced quality of life. In my practice I often have patients with different types of headache.

Since 1987 I am free-lancing in my practice and I frequently encounter patients suffering from migraine. Since the beginning of my training to become an osteopath I have been engaged with this topic even more. I am convinced that Osteopathy offers a multifaceted approach for the treatment of migraine. Because of this it was self-evident to immerse myself into this topic even more and to focus my Masterthesis on the treatment of migraine from an osteopathic point of view.

I have given my work the following structure:
I start my work with the definition and the different types of migraine because they are essential for the anamnesis of the illness and the approaches to treatment. Equally important is the overview regarding the pathogenesis of migraine and its relevance for the general public. In the following two chapters I discuss the trigger factors and phases of a migraine attack. Interesting are the interconnections of migraine to other illnesses, which I examine in the chapter Comorbidity. The chapter Relevance for the Patient shows the reduced quality of life due to migraine attacks. Chapter 9 highlights multifaceted approaches to treatment that Osteopathy offers. Chapters 10 und 11 document my personal approach in this study. For my investigation, I used a special method to gain reliable results from my treatment out of a homogenous group of patients which are discussed in the statistics afterwards. Finally possible improvements are shown in the concluding discussion.

2. Definition

Diener (2006) says that, from a pathophysiologic point of view, headaches can be classified into **idiopathic** or **symptomatic headaches**.

Idiopathic headaches are migraine without aura and migraine with aura, tension-type headache and cervicogenic headache. It is the opinion of several investigators that organic reasons are assumed (see chapter 3.7.2). It is also common standard of knowledge that **symptomatic headaches** can appear in connection with the intake of special substances or in connection with different diseases such as: traumata, cerebral bleeding, cerebral ischemia, tumors, changes of pressure in the brain, inflammation, hypertensive crises und glaucoma. Often there are lesions in structures.

My thesis is concerned only with migraines without aura. Because of the homogenous nature of my study group, I have eliminated all other forms of migraine (10.6. exclusion –criteria).

The International Headache Society (HIS) defines **migraine without aura** as a recurring headache disease with the following diagnostic criteria:

At least 5 attacks have to occur, which fulfil the criteria B – D:

B. headache attacks which (without treatment or unsuccessfully treated) last for 4 – 72 hours

C. The headache has at least two of the following characteristics

1. unilateral localisation
2. pulsating character
3. middle or high intensity of pain
4. increased pain caused by physical routine activities (for example walking or climbing stairs) or which leads to avoidance of these activities

D. During the headache there is at least one of the following:

1. nausea and / or vomiting
2. photophobia and phonophobia (hypersensitivity against light and noise)

Connections to an illness other than migraine must be eliminated.

Diener (2002) states the frequency of typical accompanying symptoms of migraine in the following percentages:
nausea 80%, vomiting 40 – 50%, photophobia 90%, sensitivity against noise 75% and sensitivity to smells 40%.
Evers (2006) states that sensitivity to smells has a high specifity for migraine.
In one third of all attacks and patients the localisation of the headache is unilateral. The headache can also change sides from attack to attack as well as within an attack. Very often pain starts in the neck and reradiates later on into the head and temple region.
Migraine headache develops mostly within 15 minutes to up to 2 hours. If the migraine attack begins during sleep, the headache may be at full intensity upon waking (Diener, 2002). The typical age at first onset of migraine in women is between the 12th and 16th year, in men between the 16th and 20th year. Migraine occurance between women and men is characteristically seen in the ratio of 3:1 (Diener, 2002).
The above mentioned forms of appearance are consistently certified by other scientists. For example by Lipton and Bigal (2007). Conclusive set of studies made by Lipton and Bigal showed that the three best predictors specifically for migraine were nausea, disability and photophobia. They also developed the Migraine Disability ASssessment (MIDAS) to improve the recognition and measure of migraine disability. The MIDAS tool stratified migraineurs into 1 of 5 grades – 0 to IV with IV being a severely disabling migraine. This should lead to a better adapted treatment due to the different severity codes of migraine. Because of this it should also be possible to

help to avoid migraine progression. Their work has informed my study and I have used their MIDAS questionnaire.

2.1. Genetics:

Evers (2006) brings up the discussion whether or not a modified cortical processing of impulses could play a role in affected people. His statement is, that in everyday life, people with migraine have an increased alertness to different simultaneously presented impulses. The question is whether or not this could be a benefit in evolutionary selection because migraine is known to be a genetic disease, but definitely also has disadvantages in selection (Evers, 2006).

Diener (2002) also states in his book, that there is evidence that migraine is a genetically determined disease. Studies on twins, as well as molecular biologic result, support this opinion. Diener (2006), Evers (2006) and Keidel (2007) agree that the probability of migraine is twice as high in identical twins than in fraternal twins. Furthermore all three auhors attest that in familiary hemiplegic migraine (FHM) – in which a complete hemiplegia appears during aura – a gene defect on chromosome 19 can be held responsible. Evers und Keidel agree that this gene mutation can be found in some families with migraine without aura where it plays a causal role (Evers, 2006).

2.2. Signs of Alarm:

Warning signs which identify symptoms include severe changes or illness. Here an exact medical clarification is necessary. For this purpose continuative diagnostic procedures such as cranial computertomographie (CT), cranial magnetresonanztomographie (MRT), angiographie, sonographie, laboratory investigations and others are used. In addition one should seek medical advice from other medical specialists such as otologists, internists, medical specialist for the eyes and/or a dentist.

There is demand for clarification, when:

- Headaches appear suddenly, for the first time and are very severe
- Headaches increase in severity
- Headaches appear in young children or in advanced age
- Headache, with systemic or neurologic ephenomenon which point to other diseases.

Diener (2006), Keidel (2007) and Evers (2006) agree that for 95% of patients with headaches a detailed anamnesis combined with an exact clinical and neurological examination is needed for an adequate diagnosis of migraine.

3. Pathogenesis of Migraine

3.1. History:

Evers (2006) reports that the earliest hints to migraine are found in about 3000 B.C. in mesopotamic verses which describe a combination of eye- and head-illnesses. Keidel (2007) argues that the earliest documents of head illnesses go back to the 6th century B.C.. By means of magic, mystic and prehistoric religious ideas, headache was seen as the creation of malicious spirits. This interpretation can be gathered from sumeric, babylonic and assyric boards. Probably to release the evil spirits out of the head and brain, skull-trepanations (cavities that were sculptured in the skull) were performed. Interventions like this were even done occasionally in the time of the Renaissance.

In about 180 A.C. Galen had the opinion that an excess supply of juices, mainly of yellow gall, was the reason for migraine. These antiquated theories were taken over by medical schools into the Middle Ages (Evers, 2006).

As an example for a psychogenic theory of migraine, Evers (2006) mentions the familiar nun and mystic Hildegard von Bingen (1098 to 1179). Her visions can be interpreted as migraine attacks because the visual hallucinations resemble a migraine aura. But he also states that, in the case of Hildegard von Bingen, divine inspiration and a strong suggestibility are preconditions for her visions. The interpretation of Evers conforms with the opinion of Sacks (1998). He maintains that Hildegard von Bingen had visions beginning from her earliest childhood until the end of her life. After accurate study of the written descriptions and her pictures, he comes to the conclusion that they were caused by aura visions of migraine.

In the 18th and 19th century the neurogenic theory of migraine onset arose. The malfunctioning neuronal activity in the cortex was discovered (Evers, 2006).

In the last decades mechanisms of pain development have often been studied. Lampl (2006) attests that the hypothesis regarding the contractility of the cranial vascular system at the centre of the discussion since the end of the 1930s. For a long time everyone believed in the vascular theory of migraine onset. This theory

meant that migraine comes along with vasodilatation (widening of blood vessels) in the concerned half of the head which irritates the meningen and therefore leads to headaches (Evers, 2006).

Lampl (2006) attested that only modern picture taking-techniques in the 90s made it possible to understand the central mechanisms of pain development in migraine. Probably a temporary drop out or reduced activity of a centre lying in the brainstem is responsible for the onset of migraine. Evers (2006) attests, too, that picture taking methods show that the initial changes within a migraine attack are localised in the brainstem.

3.2.”Migräne Generators“

Evers (2006) said that Positrons-Emissions-Tomography (PET) and functional MRT–studies could verify a region in the brainstem and in the middle of the brain which, during a migraine attack, had an clearly increased metabolism and, because of this, could be called “migraine generators“. This increased metabolism remained even if the migraine pain had been treated sufficiently.

3.3. Functional disorder in the brainstem

Evers (2006) says that it is positive that even in a long-lasting migraine, no structural changes in the brainstem can be verified. In his opinion the “migraine generators“ seem to be a functional disorder and not an anatomic – morphologic disorder.
In opposition to this statement Kriut (2004) says that clinical studies have suggested an increased prevalence of cerebral infarction and white matter lesions in migraine patients. To find out whether these lesions are prevalent in the general migraine population, he performed a study of a population-based sample of Dutch adults. The results were that no participants reported a history of stroke or transient ischemic attack or had relevant abnormalities during standard neurological examinations. He found no significant difference between patients with migraine and controls in overall infarct prevalence. “However in the cerebellar region of the posterior circulatory territory, patients with migraine had a higher prevalence of infarct than controls”.

Laegeforen (2007) says that patients with migraine probably have increased cortical excitability. He supposes that attacks may be initiated by the neurophysiologic phenomenon ”cortical spreading depression” and that this may again lead to meningeal inflammation and irritation of pain-sensitive fibres in the trigeminal nerve. An episodic dysfunction of the brain stem plays an important role - either as a primary generator of or secondary contributor to - migraine attacks.

3.4. Trigeminovasculary system

Current studies – for example from Evers (2006) - verify an allodynia in migraine affected people which means a painful feeling of normally not painful impulses in dermatoms (areas in the skin) which are innervated by the N. trigeminus. This allodynia can appear hours before the onset of migraine attack. This could be a sign of a central facilitations or sensitisation of N. trigeminus. The activation of the “migraine generators“in the brainstem can lead to a hypersensitivity of trigeminal neurons.

3.5. Vasoactiv neurotransmitter

Because of the activation of trigeminal neurons **vasoactive neurotransmitter** are released from trigeminal peripheral endings of the nerves in the meningen (skin in the brain) and the meningeal vascular system.

The neurotransmitters serotonin and Calicitonin-gene-related-peptide (CRGP) play by far, **the most important role in pathogeneses of migraine.** These neurotransmitters lead to vasodilatation of cerebral and dural vessels. In a following reaction, an aseptic inflammation develops. Picture taking methods such as sonography show that the dimension and even the side of widened vessels do not correlate with the pain of migraine. Even the effectiveness of pharmaceutical therapy of a migraine attack does not show a connection with the changes of cross-section vessels. This could be a sign that the widening of the vessels– different to what was believed in former times – is not the critical mechanism for the development of migraine headache pain. What is now standard knowledge is that the irritation of the nociceptors (receptors for pain) caused by neurogenic inflammation is the releasing factor for onset of migraine pain (Evers, 2006).

Serotonin

This neurotransmitter affects numerous receptors, two of which are specific ones which are found primarily in the brain and in intracranial (in the head) meninges.

The 5-HT 1B- receptor causes a constriction of meningeal vessels. The 5-HT 1D-receptor causes a reduction of activating trigeminale neurons in the brainstem and therefore an inhibition of pain sensitization. One can demonstrate that, at the beginning of a migraine attack, serotonin is released massively and, during a migraine attack, a deficit of serotonin exists (Evers, 2006).

3.6. Modern medicine such as triptans

Triptans have the ability to selectively activate the 5-HT 1B- and 5-HT 1D-receptors. The following models of explanation are proved experimentally. On one hand they lead to a specific constriction of vessels in the meningeal supply area and therefore reduce the dumping of neuropeptids. On the other hand, the effect of these medicines is based on the inhibition of neurogenic inflammation (trigemino-vasculary system) and on a favourable influence on the central development of pain (Diener, 2006). Evers (2006) and Lampl (2006) confirm this specific effect on migraine.

Based on this specific effect, triptans are ineffective for other forms of headache (Diener , 2006).

In addition to these pharmaceuticals, which affect these 2 well-known serotonin-receptors, there are still other efficient drugs for migraine prevention. Ramadan (2007) says that a variety of drugs from diverse pharmacological classes are in use for migraine prevention; for example, ß-adrenergic blockers, anticonvulsans, tricyclic antidepressants and serotonin receptor antagonists. The mechanisms of migraine preventive drugs are multiple, but it is postulated that they converge on two targets: 1) inhibition of cortical excitation; 2) restoring nociceptive dysmodulation. Modulators of the serotonergic and adrenergic system and cholinergic enhancing drugs may restore descending nociceptive inhibition and play a role in migraine prevention.

3.6.1. Contraindications

You have to bear in mind that there are contraindications for triptans. They are not allowed to be taken if there is any cardiovascular illness (Diener, 2006). Selim (2007) lists the following medical problems as contraindications for triptans: coronary heart disease, myocardial infarct in anamnesis as well as cerebral vascular processes or

status post apoplectic stroke and ineffectively treated hypertonic, gravidity and lactation.

3.6.2. Preventative medication is needed when there are:

- More then 3 migraine attacks per month
- Long-lasting attacks (>48 hours.
- Very severe and complicated attacks (neurologic deficits >1 week)
- Long-lasting auras
- Intolerable side effects of migraine medicine
- Increasing frequency of attacks and if the patient needs pain or migraine medicine more than 10 times a month

Menstrual migraine is in the group of especially long-lasting and severe migraine attacks. About 10% of women are affected by this form of migraine in which the symptoms are closely connected with menstruation (Diener, 2006). The use of preventive medication shows a reduction of frequency, severety and duration of migraine attacks and the prevention of the medicinally induced headache. If there is a reduction of attack frequency of at least 50%, Diener (2006) says the prevention of migraine is effective.

3.7. Anatomical coherence / chains of reaction

Most physicians associate migraine attacks with anatomical connections between the upper cervical spine, certain brain nerve nuclei and the sympathetic nerve system.

3.7.1. Coherence between upper cervical spine, cerebral nerve nuclei and the sympathetic chain

Strackharn (2003) deduces the onset of migraine from the following anatomical context and he agrees in this with Bogduk (1995):

The trigeminocervical nucleus is the region that receives afferents from the trigeminal nerve and from the upper three cervical spinal nerves, together with additional fibres from the N.facialis, the N. glossopharingeus and the N. vagus. “The significance of the trigeminocervical nucleus is that it is the essential nociceptive nucleus of the head, throat and upper neck” (Bogduk, 1995, page 436). Because of the overlapping pattern of ramification of primary afferent fibres, fibres from different peripheral

nerves end on the second-order neurons in the trigeminocervical nucleus. **This convergence has been demonstrated physiologically because neurons in the C1 and C2 segments respond to the stimulation of afferents in both the upper cervical spinal nerves and the trigeminal nerve**. This convergence creates the basis of referred pain in the head und upper neck. Referred pain caused by cervical stimulation is most commonly, perceived in the occipital and fronto-orbital regions of the head. Less commonly it is felt in fields innervated by the maxillary and mandibular divisions of the trigeminal nerve. In regard to this, Bogduk and Strackharn have the same opinion.

Bogduk (1995) and Strackharn (2003) are sure that the spinal nerves from C1 – C3 divide into ventral and dorsal rami. Their ventral rami join with that of C4 and together form the cervical plexus. From this plexus muscular branches are distributed to the prevertebral muscles such as the longus capitis and cervicis, rectus capitis anterior and lateralis and the sternocleidomastoideus and trapezius. The C1 – 3 spinal nerves form at their origin recurring meningeal branches, the so-called sinuvertebral nerves. The sinuvertebral nerves of C1 – 3 are joined in the posterior cranial fossa by meningeal branches of N vagus and N hypoglossus. “Although arising from cranial nerves these branches are cervical in origin having gained the cranial nerves outside the skull where they communicate with the cervical plexus”.

Strackharn (2003) continues that only these three anterior branches of nerves from the neck have connections to the ganglion cervicale superius. This ganglion is the most cranial part of the sympathetic chain. It is responsible for the arterial blood circulation of the face, the brain and the immediate surrounding of the neck. Additionally Strackharn (2003) emphasises the central significance of the upper thoracic spine from up to the 5th thoracic vertebra to the ganglion cervical superius. He characterises congenital or acquired false position of the spine or the ribs in the upper thoracic spine as an accelerator for migraine attacks. We do not know the opinion of Bogduk relating to the thoracic spine. Today’s theories add to these results.

To reveal connections between headache and changes in the neck, Strackharn arranged a study with 278 patients with chronic headache.

The conclusion of this study is that migraine that has a connection with acute loss of movement in the neck and pain on the one half of the head may relate to enforced side bending of the head (Strackharn, 2003).

Strackharn builds a context over the following chains of muscles which are activated by ongoing stress:

- The M. trapezius pulls the shoulder up with its upper part.
- The N. accessorius, which innervates the M. trapezius, is closely connected with the N.vagus. These nuclei of the nerves sometimes arise from each other.
- Strackharn calls the N. vagus a sort of “stress nerve” (2003, page 70).
- Because of this close connection of the two nerve nuclei, the N. accessorius reacts to stress and holds the shoulder in this raised position.
- The N. accessorius innervates two muscles, the M. trapezius and the M. sternocleidomastoideus, which brings the head to an asymmetric position.
- Both of these muscles get their sensitive nerve fibres from the upper three nerves of the neck. Because of ongoing tension in the muscles, pain in the muscles does develop. This pain is referred to the spinal cord which intensifies the tension.
- In the last chain of the chain-reaction, the M. levator scapulae (which is innervated by the branches of C 2. to C5) pulls the first vertebra into a false position of rotation and maintains this position.
- This false position of rotation prepares the ground for the acute neck and is the reason for the onset of migraine (Strackharn, 2003).

Strackharn (2003) bases the onset of migraine on two facts: on the one hand, stress and the thereby activated muscles, and on the other hand, the false position and /or the incorrect posture of the thoracic spine. The anatomical facts do not allow for any doubt as to the development of this chain reaction.

In addition to this he deduces - out of the anatomical complexity of the upper cervical spine (with the above-mentioned nerves of the brain) - the efficiency of his so-called “cervical selective block of receptors“ with a thinned local anaesthetic as a efficient therapeutic treatment for migraine, which he performs in his clinic.

This connection described by Strackharn (2003) and Bogduk (1995) **is contradicted by Evers** (2006) **empathically**. He writes that tension of the neck muscles is not a trigger for migraine attacks. In his opinion tension of the neck is an expression of a

simultaneous activation of nerves which innervate the muscles of the neck and throat.

The controversial discussion goes on. Keidel (2007) maintains that one can often observe a combination of types of headaches. The person concerned suffers from migraine as well as from tension-type headache. He adds that having a migraine does not exclude one from having a cervicogenic headache, too. His opinion is that a cervicogenic headache is combined with migraine or tension-type headache in up to 15% of cases-

For a better understanding here is a comparison of these 3 types of headache from Keidel (2007):

	Migraine	Tension-type headache	Cervicogenic Headache
Localisation	Unilateral	Whole head	Unilateral
Starting point	Front-temple region		Starting at neck
Side	Change of side possible		Constant, on one side
Intensity	Servere	Light-middle	Changeable
Character	Pulsating	Dull-oppressive	Dull-vesicant
Duration	Days	Hours	Hours-days

As I discuss in chapter 9, the different opinions of experts may lead to different approaches for treatment.

3.7.3. Central Sensitization Hypothesis

Dodick and Silberstein (2006) explain in their article that the most recently articulated theory of migraine is the **central sensitization hypothesis**. This hypothesis proposes that altered processing of sensory input in the brainstem, principally the trigeminal nucleus caudalis, could account for many of the temporal and symptomatic features of migraine. This theory could as well explain its poor response to triptan therapy when such treatment is initiated up to an hour after the onset of pain. Preclinical as well as clinical data support the central sensitization theory. Cutaneous allodynia-pain arising from harmless stimulation of the skin, such as hair brushing, is an easily identifiable marker of central sensitization. The presence or absence of cutaneous allodynia can be integrated into the routine clinical assessment of

migraine. Both authors are sure that future basic and clinical research on central sensitization is likely to be of ongoing importance to the field.
Keidel (2007), too, writes that the release of a vasoactive messenger could be a consequence of activation of the trigeminal nerves. The release within a migraine attack can lead to a central facilitation or sensitization. At the endings of the nerves around the blood vessels of the brain and the meninges, proteins are released. They cause a dilatation of the vessels, in particular the small blood vessels of the meniges. As a consequence liquid from the blood escapes through the septum of the vessels into the area surrounding the vessels, named plasma extravasation. Due to the release of particles of inflammation such as histamin, serotonin and prostaglandin, a neurogenic-caused inflammation develops. Probably, because of a functional deficit of a circumscribed area of the brainstem, a reduced inhibition of pain is caused and leads to increased pain sensitivity.

3.7.4. Immaturity of the intestine/allergies as activators of migraine

Carreiro, a lecturer on Vienna School of Osteopathy working at the University of Maine, told us that she, too, works with migraine patients. She assumes, due to her anamnesis-questionnaire, that patients with migraine have more often abdominal colic in the first months after birth. She posed the question whether or not immaturity of the intestines could be one of the reasons for the onset of migraine.

Strackharn picks up exactly this question in his book. He emphasises **allergies** which play a role in the intestines and gain momentum from this origin. He arranges an "intracellulary analysis of mineral material". He confirms that you can often find a malfunctioning metabolism of mineral material. Often this is combined with a change of the bacterial count in the intestines. This malfunctioning mineral material does not only cause problems to the intestines but becomes noticeable in the irritability of the nerves and in the neurotransmission. In his observations there is often a displacement of potassium/sodium in the intracellular analysis of mineral material in patients suffering from migraine. Often potassium is reduced and sodium is increased. Because of this imbalance, the irritability of nerves and muscles is increased.

His conclusion is that:

- Chronic inflammation in the intestines have an influence on the development of migraine
- It makes sense to reconsider accepted customs of nutrition if the frequency and duration of attacks increase
- If the usual drugs for elimination of migraine attacks are useless or are only slightly effective, this, too, could be caused by false intestinal flora

3.7.5. Neurogenic inflammation

Another reason for the repeated occurrence of migraine is **neurogenic inflammation**. Chronically irritated pain receptors release neuropeptides which cause neurogenic inflammation. This inflammation leads to an acidity of the surrounding area and to an increase of irritability of the receptors. This then leads to an enlargement of the neurogenic inflammation and, therefore, causes an important increase in sensitivity to pain. This means, the longer chronic pain lasts, the more sensitivity to pain a person develops (Strackharn, 2003).

3.7.6. Onset of pain

In the article of N. Bogduk you can read that there are three basic mechanisms by which pain may be generated:

Nociceptive pain is caused by some form of pathology or disturbance in the periphery that can activate nerve endings. In the context of headache the mechanical irritation that makes pain is strain of the dura mater. Chemical stimulation because of inflammation or caused by the liberation of potassium ions (potassium –Ionic) from injured cells can be the cause of nociceptive pain.

Neurogenic pain arises when the axons or cell bodies of a peripheral nerve are stimulated. The lesion that causes neurogenic pain does not lay in the peripheral territory supplied by the nerve, but may be as far proximal as the roots of the nerve. The archetypical neurogenic headache is trigeminal neuralgia. Most often it is the result of irritation by an aberrant nearby vessel. The characteristic clinical features are: the patient suffers repeated staba of lancinating pain in the forehead, typically triggered by touching a particular spot on the surface of the face or mouth. This lancinating quality of pain is characteristic of neuralgia.

Central pain is a mysterious phenomenon. The classical models were framed in terms of dilatation of cranial vessels. The distended vessels were presumed to be the source of the pain. The pain evoked is nonetheless perceived in the territory of the nerves that relay to the pathway involved. Yet there is no pathology in the periphery to explain the pain. Another model is dysmodulation, in which the descending inhibitory pathways that control pain perception are somehow themselves inhibited. The result is an illusion of pain, but pain that is real in terms of the suffering it produces (Bogduk, 1995).

3.7.7. Vegetative attendant symptoms of migraine

Additional to the most common vegetative attendant symptoms in a migraine such as nausea and/or vomiting, as well as sensitivity against light and/or against noise migraine without aura may have a long list of possible **vegetative symptoms (**Evers, 2006). Among them are:

- Wet eyes, because of increased lacrimation
- Itching and burning in affected eye
- Increased salivation
- Running nose
- Abdominal symptoms such as painful colics
- Fever, shivers, trembling or sweating
- Organic hypersensibility against touching
- Changes of affect such as disgruntlement and irritable hyperactivity are common in the early phases of attack (Sacks, 1998). At the maximum point of attack, these symptoms can lead to drowsiness, dizziness and feelings of weakness including apathy, lethargy, somnolence and depression.

Keidel (2007) mentions in his book that vegetative attendant disorders in children and youngsters come to the fore and can strain more than the headache itself. When treating a migraine attack vegetative attendant disorders may not be neglected.

Gupta (2006) reports that cranial autonomic symptoms were present in 73.1% of subjects and, commonly, they were ipsilateral to headache. Patients with autonomic symptoms had a longer duration of illness and longer headache episodes. In addition, he found out that sleep was ineffective in relieving their headache.

4. Trigger Factors

In addition to definition and pathogeny of migraine, a special main focus of my investigation is the question of causes of migraine because these are for every single patient of enormous significance. These causes are called trigger factors.

In persons with a genetically determined inner disposition, **internal as well as external trigger factors** can activate a migraine attack. An example of an internal activator is psycho-emotional stress; an external activator of a migraine could be a special smell. Nevertheless Evers (2006) says that **these trigger factors are not causally involved in the development of a migraine.**

However, he states, nevertheless in his book (2006) a list of trigger factors and he makes a distinction between epidemiologic-statistic verified triggers and triggers verified in experimental studies. The first are, for example, changes of weather (especially temperature inversions), psychosocial burden, irregular meals (especially a deficit in carbohydrates or the absence of carbohydrates fasting), changes in daily rhythms, certain foods (such as, for example, French red wine) and flavour enhancers (glutamat). Nitric compounds, histamine, changes in sex hormones, in which the premenstrual reduction of oestrogen is the strongest trigger, French red wine and glutamat („Chinarestaurant-syndrom“) are verified triggers according to the experimental studies.

Research done by Diener (2002) confirms that, in perhaps 10% of women suffering from migraine, a pure menstruation-associated migraine is evident. He agrees with the statement that pathophysiological (the sudden reduction of the level of hormones) is responsible for migraine.

In addition, Keidel (2007) also speaks of migraine not only appearing during menstruation, but also during ovulation, because at that time, hormone levels also change. In young child-bearing women who use estrogen contraceptives. In older patients taking hormone after menopause, the frequency of the attacks can increase. Because, after the birth of a child, estrogen levels drop again, the frequency of migraine attacks can increase during lactation.

In his published book in 2006 Diener writes that, in addition to alcohol, smoking and caffeine-withdrawal, weather and shifting of time, as well as flickering light, noise, smells, stress, stress release, speculation of fear and hunger, too, are trigger factors.

Evers states in his publication from 2006 that there are some triggers which one cannot prove academically. These include special sorts of cheese, chocolate, nuts,

citrus fruits and preventing agents in foods. Keidel (2007) also occupied himself with this subject intensely, including specific drugs such as nitro-glycerine-spray, as used in angina pectoris which can cause a migraine attack. If incorrectly implicated and too often used, migraine medicine such as ergotamine or triptans can cause a migraine attack as well.

4.1. Exposure to trigger factors

Diener (2002) ascertains that most female and male patients have trigger-factors. These factors should be identified and investigated to see if influencing them or avoiding them produces positive results. In his opinion, possible factors of influence are:

- Retaining sleeping rhythms even during the weekend, if changes of daily rhythms are a releasing factor for migraine
- Reorganization of daily routines to avoid stress, if psycho-emotional stress is identified as a trigger
- Retaining consumtion of caffeine on weekends, if withdrawal of caffeine causes migraine attacks
- Avoidance of special foods only if they definitely are identified as trigger factors
- Avoidance of alcohol only if it is individually definitely identified as a migraine trigger

Selim (2007) concludes that regular meals should be taken, if phases of hunger and, due to this, changes in carbohydrate metabolism are a reason for the onset of migraine. For Selim the avoidance of trigger factors is one of four pillars in migraine therapy. The second pillar in therapy is the stabilisation of handling impulses in the brain. In his opinion the brain of patients suffering from migraine cannot get used to permanent changes as well as to a sudden overflow of such impulses. Therefore the patient has to learn for himself to get used to these impulses. The patient has not only to know about these triggers, but he also has to develop strategies to avoid these triggers or at least to reduce them effectively. In Selim’s opinion the “psycho-education“has an important influence on the controllability of migraine. As the fourth pillar of migraine therapy, Selim names the inhibition of excessive activity of neurotransmitters in the central nervous system and the blockage of neurogenic inflammation.

As an effective relaxation technique he mentions the progressive muscle relaxation of Jacobson, yoga, meditation, autogenic training, self-hypnosis and auto-suggestion. He recommends endurance sports such as jogging, bicycling and swimming, as well as, walking or Nordic Walking and dancing as beneficial for migraine patients.

Selim ascertains that with his patients, relaxation training as well as perseverance sports help to diminish stress and to harmonise internal balance. This effect was ascertained purely empirically. The model assumes that, with regular perseverance sport, the preliminary stages of stress hormones are diminished and in addition endorphin is produced. Because of this, Diener (2002), too, recommends endurance sports to reduce the frequency and severity of migraine attacks. In contrast martial arts are not recommendable, because, in his opinion, it is possible that they are able to induce migraine attacks**.**

Under the topic psycho-education, Diener (2006) suggests that patients with an episodic or highly frequent migraine (three or more attacks/month) should attend a psychologic therapy such as behavioral therapy. For these therapies, reliable studies are available. These behavioral therapies should be used alternatively or in combination with medicinal treatment.

- One of the most important methods is EMG-biofeedback-therapy (constriction of the A. temporalis superficialis, relaxation of M. frontalis and the m. temporalis) and progressive muscle relaxation (PMR). Both possibilities demand much more activity from the patients.
- The cognitive-behavioral pain-accomplishment-training is also used. These methods target the strengthening of self-control for the minimizing of impairment or to increase pain-reduction (Diener, 2006).

Nash and Thebarge (2006) confirm that cognitive-behavioral therapy is a treatment that includes a specific stress management component along with some form of physiological self-regulation element.

Part of the approach can be a focus on modifying health behaviours, such as caffeine reduction and sleep hygiene, which both are known headache precipitants (Bigal, Lipton, 2006).

In clinical practice guidelines for migraine, cognitive-behavioral therapy, EMG biofeedback, relaxation training and thermal biofeedback combined with relaxation training are empirically supported treatments (Grath, 2006).

Ciancarelli (2007) says in the conclusion of his study that biofeedback training sessions are an appropriate therapeutic tool to reduce the vulnerability of chronic migraine sufferers to oxidative stress. He explains that that central sensitization is influenced by excitatory amino acids such as glutamate and by nitric oxide (NO) and might be maintained by neuropeptides such as calcitonin gene-related peptide (CGRP)

Because of biofeedback training a specific radical-scavenging enzyme (superoxide dismutase=SOD) showed an increased activity and NO bioavailability increased.

This was associated with a decrease in peroxides levels.

In my study, it appeared that, for my patient group, stress is an especially serious trigger factor – and one which is very difficult to influence. Therefore I dedicate to the subject Stress another chapter.

4.2. Stress as a trigger factor:

Interesting form me was the large number of scientific statements on the subject of stress in the varied facets of literature. The most interesting, I have stated here.

In a prospective cohort study Mäki examined whether work stress, as indicated by the job strain model, and the effort-reward imbalance model, predicts new-onset migraine among 19.469 female employees with no history of migraine at study entry.

In a follow-up two years later, 1281 new cases of migraine were detected. The proportion of new migraine cases attributable to high effort-reward imbalance was 6,2%. Mäki said that, if the observed association is causal, his findings suggest that high effort-reward imbalance might function as a modifiable risk factor for new- onset migraine (Cephalalgia, 2007).

Nash (2006) explains that life stress is a psychosocial factor that is generally acknowledged to be a central contributor to primary headache. Psychological stress is conceptualized as an imbalance between perceived demands and perceived resources resulting in demand on the biological system. There are multifaceted ways that stress and headache relate.

A stressor is any perceived challenge or threat, whether objectively verified or not, to normal functioning. The stress response is the body’s activation of physiological systems, namely the hypothalamic-pituitary-adrenal axis (HPA), to protect and restore functioning. The HPA axis is ultimately responsible for controlling virtually all the hormones, nervous system activity, and energy expenditure in the human body,

as well as modulation of the immune system. In response to a stressor, ß-endorphine, cortisol, and other related hormones are released.

Allostatic overload can result from chronic stress exposure, poor adaptation to repeated stressors, and inability to shut off allostatic response after a stressor is terminated. Allostatic overload can predispose the individual to disease.

Nash and Thebarge are sure that the pathophysiologic consequences can also be complicated by behaviours such as the consumption of tobacco, alcohol and drugs, dietary choices, sleep schedule, exercise, and adherence to medical regimes. These behaviours are a reflection of the ways people respond to life challenges. The vulnerability within the individual can be the reason of the specific impact of allostatic load.

Lazarus (1993) is credited with clarifying the factors that account for the individual variation that occurs in stress responses. He emphasized the importance of appraisal in determining the stress response. The availability of coping resources (personal, social, and material) along with the ability to cope effectively, help to determine the magnitude of the stress response.

Nash (Headache 2006) determines that ”stress” can (a) be a predisposing factor that contributes to headache disorder onset, (b) accelerate the progression of the headache disorder into a chronic condition, and (c) precipitate and exacerbate individual headache episodes.”

Stress is characterized by excessive worry and an anxious emotional over-reactivity to environmental demands. These stress-related factors are related to migraine independent of history of anxiety and depression, sex, headache history, or maternal headache status. Whether these stress-related factors are a precipitant of headaches or an early correlate with shared pathophysiology (eg. serotonergic dysfunction) is unclear (Waldie, Poulton, 2002).

“Stress is often cited as one of the most frequent aggravating factors in headache” (Kaynak, 2004; Rasmussen, 1993; Spierings, 2001).

Headache attacks were preceded by an increase in the incidence of stressfulness of daily problems, with increases in tenseness, irritability, and fatigue occurring for one or more days prior (Drummond, Passchier, 2006).

Huber (2003) confirms that sensitivity to stress has been found to be related to increased headache duration.

Bigal (2006) confirms in his study that stressful life events is one of the already identified remediable risk factors for migraine progression.
Dummond (2006) affirms that the impact that stress and emotional factors has on individual episodes may be at both the peripheral and central levels. “At the peripheral level stress may provoke perivascular inflammation and pericranial muscle tenderness”.
Olesen (2006) says that, at the central level, stress may influence supraspinal control of neurons of the trigeminal nucleus caudalis and this leads to increased excitability at the spinal/trigeminal level.
Nash (2006) supposes that “it is possible, although not empirically established, that the presence of stress lowers the threshold for hormonal fluctuations to have a greater impact on headache”.
Schoonman (2006) is in opposition to all of these authors because he says that, although stress-sensitive patients - in contrast to non stress-sensitive patients - may perceive more stress in days before an impending migraine attack, he failed to detect any objective evidence for a biological stress response before or during migraine attacks.

5. Development of a Migraine Attack

The course of the migraine recurs with most patients according to a certain pattern. Diener (2006) differentiates four phases of a migraine attack, which do not always appear in the same intensity:

- **1.Phases: prodromi**
- **2.Phases: Aura**
- **3.Phases: headache**
- **4.Phases: back-formation**

The first phase of a migraine attack is called the prodromal phase. Hours to days before headache appears – as a so-called forerunner – different disorders may appear. In this beginning stage of migraine there is often an increased swelling of the stomach area with missing borborygmi and obstipation. Also fluid-retension in the body and thirst may be present. Following further possible disorders may appear in the prodromal-phase:

- Mood variations

- Inner concern
- Decreased concentration
- Changes in appetite
- Shivering
- Oedemas
- Sleep disorders

Some patients simply feel that a migraine will be coming soon.

The second phase of migraine may be distinguished by an **aura -** different forms of hallucinations are seen. The manifestations of migraine-aura are of extraordinary plurality according to Sacks (1998). For example simple or complex hallucinations such as paraesthesia may begin in the hand or in the foot and may ascend to the head. Patients often report visual disturbances such as limitations in the visual field, flickering or flashes of light. Also, temporary hemiparesen (hemiplegia) are observed. Intensive affective toned conditions, deficits and disorganisation of speech and ideation, distortion of spacial-temporal perception and conditions as if in trance, as well as deliriousness, can occur as manifestations of aura. Aura seldom longer lasts than half an hour. Aura is experienced before or at the same time as headache appears (Diener, 2006).
I mention the aura here because aura may occur in the group of my patients who have the diagnosis “migraine without aura” sometimes within a very severe migraine attack.
Diener (2002) says the following in his book:

- The most common form of migraine is migraine without an aura. It affects about 70% of migraine-patients. It is called simple migraine.
- About 10% of patients suffer from migraine with an aura, the so called classic migraine.
- Approximately 20% have a migraine which is sometimes accompanied by an aura.
- Other forms of migraine are very seldom and represent less than 1% of cases.

The third phase of a migraine attack is the **headache**. Often they start in the morning. After ¼ - 2 hours they develop their maximum strength and last for 4 –

72 hours. 2/3 of patients sense the pain on one side of the head, whereas the side of headache may change even within an attack. It is typically for somatical exposure to aggravate the pain. In addition many patients are sensitive to light, noise and smells and seek quiet. Nausea, vomiting, polyurie and Diarrhoe may appear as side-effects. Often the last phase of headache is sleep.

In the fourth phase of migraine attack, the **back-formation,** common symptoms appear which are complementary to the prodromi – the first phase of migraine. If, for example, in the beginning one feels hunger, now loss of appetite may appear (Diener, 2006).

Accompanying these phenomena which refer only to migraine studies, evidence shows that migraine patients are inclined to have other illnesses.

6. Comorbidity

As the following citations show, depression and anxiety often appear together with migraine.

In the article “Ten Lessons on the Epidemiology of Migraine” (2007) Lipton and Bigal are sure that migraine is comorbid with a number of other disorders. “By definition, comorbidity refers to the occurrence of two medical disorders in the same individual at a frequency greater than chance” (Lipton and Bigal, Headache 2007; 47, page 4). The most common comorbidities of migraine are depression and anxiety disorder, but bipolar disease is also highly comorbid with migraine from a clinical perspective.

Hamelsky (2006) confirms this theory. She says that a diagnosis of one of these psychiatric disorders should increase vigilance for migraine. “Treatment plans for migraine should be mindful of the comorbid condition”. If the association is bidirectional (if each disorder predisposes the other) the association may arise from an underlying vulnerability.

Conclusions of her studies were that migraine is consistently associated with several psychiatric disorders. These disorders include depression, anxiety and

bipolar disorders. Hamelsky says that it is important to maintain diagnostic vigilance for comorbid conditions and to take both disorders in account in formulating a treatment plan.

Hamel (2007) reports that, similar to migraine, depression is also considered to be a disorder of low brain serotonergic activity, and epidemiological studies have reported comorbidity of migraine with depression. Pharmacologically controlled depressed patients submitted to a rapid tryptophan depleting diet experienced a depressive relapse together with symptoms reminiscent of migraine, such as increased nausea or vomiting, drowsiness and, in some cases, headache. He confirms that further work will be needed for a better evaluation of the clinical outcome or therapeutic benefits of such interactions of the triptans with the serotonergic system.

Yet Breslau (2003) comes to the conclusion that major depression increased the risk for migraine, and migraine increased the risk for major depression.

Investigations of Drummond and Scher have proved that migraine patients suffer more often than others with motion sickness and other pain syndromes.

Drummond (2004) arranged a fascinating study which connects motion sickness and migraine. He found out that symptoms of motion sickness provoked by optokinetic stimulation were greater in those with migraine than in controls. Painful stimulation of the temple intensified nausea and headache during optokinetic stimulation. Since nausea also intensifies facial pain during motion sickness, nausea and headache may reinforce each other in a vicious circle “This suggests the involvement of trigeminal affects and is in keeping with the trigeminovascular theory of migraine pathogenesis.” (Drummond, 2004, page 399)

Scher (2006) investigated in her study the comorbidity of headache with other pain syndromes. She came to the conclusion that, in both children and adults, those with migraine or frequent headache are at increased risk of co-occurring non-headache pain as compared to those without headache, with the best data related to musculoskeletal pain or arthritis. ”The likelihood of comorbid pain may be related to the frequency or severity of either condition” (Scher,

2006, page1421): She says that the presence of multiple pain conditions is a negative prognostic factor for pain recovery.

Kurth (2006) reported in a large, prospective group of women, active migraine with aura was associated with an increased risk of major cardiovascular diseases (CVD), myocardial infarction, ischemic stroke, and death due to ischemic CVD, as well as with coronary revascularization and angina. Active migraine without aura was not associated with increased risk of any CVD event.

6.1. Modifiable risk factors for migraine progression

Interestingly, tension-type headache and trigeminal autonomic cephalalgias also have clinically progressive forms.

As a study from – below cited – scientists' shows, migraine is sometimes cited as a clinically progressive disorder. Medication overuse, attack frequency, obesity, low socioeconomic status, stressful life events, head injury and snoring also were all statistically significant factors associated with the development of chronic daily headache (CDH). This study also identified both dietary and medicinal caffeine ingestion as risk factors for the development of CDH (Bigal , Lipton, 2006; Bigal 2002, Cady, 2005; Chakravarty, 2003; Fanciullacci, 2005; Silberstein, 2006).

Lipton and Bigal (2006) found that body mass index (BMI) was a very powerful predictor of illness progression. They examined BMI because snoring was also a strong predictor. They were concerned that snoring, a marker for sleep apnea, might be associated with obesity in some subjects. Relative to normal-weighted controls, overweight individuals were twice as likely to develop CDH, and obese individuals were 5 times more likely to develop CDH.

Therefore, preventing migraine progression is an important clinical goal.

Some of the risk factors are modifiable. These modifiable risk factors are ones that patients and physicians can change. For example reducing attack frequency can be done with preventive medication or behavioural

interventions. To encourage weight loss, obese patients may reduce the risk of developing chronic daily headache (Lipton & Bigal 2007).
Chronic migraine is characterized by headache, depression or anxiety and sleep disturbance (Ramadan, 2007).

6.2. Migraine pain and other associated symptoms

Kelman and Tanis published a retrospective study in Cephalalgia 2006. They found out that headache intensity correlated with nausea, vomiting, photophobia, phonophobia, dizziness, running of the nose / tearing of the eyes and osmophobia. The duration of headache correlated only with osmophobia and taste abnormality

This varied clinical picture of the migraine with ist numerous facets has, of course, numerous implications for the general public, as well as for the affected patient.

7. Relevance for the General Public

Because migraine is well examined in Europe and North America and frequent appearance is documented, it is essential to investigate which effects this has on the economic and social status of a country.

7.1. Incidence of migraine

In statistics concerning migraine provided by the European Headache Federation, one can read that the percentage of people with this disorder (prevalence) is about 8 to 14 percent of people in developed countries, with equal or somewhat lower prevalence in other areas (World Headache Alliance, 2007).
Diener (2006) also confirms that in all Western industrial countries and in the United States the incidence of migraine is about 6-8% in men and 12-14% in women.
The frequency summit shows slightly divergent results with regard to the age – with different authors – however, is always in the area of active working years of the affected person.
Migraine is an extraordinarily common disorder. A study mentioned in Headache (Lipton, 2007) tells that migraine affects roughly 18% of American woman and 6% of

American men or nearly 30 million Americans. Migraine is most common between the ages of 25 and 55, during the peak productive years. Migraineurs experience high levels of pain and substantial disability. Therefore less then 10% of migraineurs report that they are able to work or function normally during their headaches.
Diener (2006) confirms that, with respect to incidence and severity of attacks, migraine has reached its maximum and peak of frequency between the 35th and 45th year of life. Afterwards migraine gets better in men as well as in women. In his book (2002) the author points out that there are female, as well as male patients, in whom, even after the 65th year of life, further regular severe migraine attacks may appear. The incidence of migraine beyond puberty in women is higher than in men. On one hand, this has genetic reasons and, on the other hand, hormonal reasons.

In a census done by Statistik Austria in 2006/2007 the fact emerged that there are big gender-related differences in the appearance of migraine or frequent headaches. 26% of women and 11% of men (every forth woman but only every ninth man) suffers from these health problems. In Austria, too, the age-group of 45- to 59-year-olds are affected most (women: 30%, men: 13%).
“WHO ranks migraine as one of the top twenty causes of years of healthy life lost to disability” (Tfelt-Hansen, 2004, Lifting the burden, downloaded on 01.12.07, page 1).
The result of this data is that patients with migraine have to be away from work sick during an attack and therefore burden the national economy.

7.2. Migraine costs

According to population–based epidemiological studies using International Headache Society diagnostic criteria, the prevalence of migraine in developed countries is about 8 to 14 %. Migraine is a chronic episodic disorder that affects people during their working lives. Indirect costs associated with reduced productivity, disrupted work or absence from work are very very high in comparison to direct costs (Lipton, 1994).
Investigation of different states show the financial burden caused by migraine patients.
The costs in the U.S.A. are extraordinarily high. In his article (Arch Intern Med. 1999) Hu XH estimated that, in 1998, the lost productivity costs of migraine in the United States were approximately $ 13 billion per year.

A French article tells us about the direct annual health care costs of migraine in France. They were assessed over 10 years ago. The direct costs were determined for physician consultations, hospitalisation medication use and diagnostic/laboratory tests. Information on absenteeism and lost productivity was derived from the Migraine Disability Assessment Score (MIDAS) questionnaire. The prevalence of migraine was determined to be 17%. “Total annual direct health care costs were estimated to be Euros 128 per individual with migraine in 1999, corresponding to Euros 1044 million when extrapolated to all individuals experiencing migraine and aged 15 or more years” (Pradalier,2004)

Very detailed data exist from Germany from the year 2005 with a population figure of about 82 million. Evers (2006) refers to this data and classifies the economic follow-up-costs into direct, indirect and tertiary costs.
The following costs are approximations based on a census of the institutes for health economy done in Munich:
To the direct costs which emerge from treatment and prevention of headaches, one can count:
- Costs of medically prescribed medicine for migraine at about € 67 Mio.
- Costs of over-the-counter purchased medicine for migraine at about € 92–490 Mio. ,
- Costs of stationary treatment due to migraine at about 26 Mio. € and
- Costs of ambulant treatment due to migraine at about 40–150 Mio. €.

To the indirect annual costs, one can count days absent from work with 1–3,9 Billion €, as well as reduced productivity at work, at a cost factor of about 0,7-2,9 Billion €. These approximations were done based on a rate of prevalence of migraine of 4-16%.

To the tertiary costs, one can count follow-up-costs which are caused by a false headache therapy or by invalidity (for example, costs of dialysis of patients due to abusus of pain medicine) at about 307 Mio.€.

These figures show that pain improvement in migraine patients would have a considerable economic benefit for the general public (Evers, 2006).

8. Relevance for the Patient

One criteria of migraine is – in the definition by International Headache Society –an increase of headache by physical activities of daily routine.

Because of this migraine is heavily reducing the quality of life in suffering people. In a representative study with 1810 migraine-patients they where asked about reduction of quality of life in different parts of daily life. It was documented

- By 46% of patients a definite impact on their working place or in school in the past 6 month in form of average 3 days of absence, as well as
- Moaned about an impact on personal relationship. In this topic 62% of patients indicated that within the past 6 month because of migraine in average they could spend 2,9 days less time with their family or with friends. Even the
- Behaviour in leisure time of migraine-patients was affected negatively due to their illness. 67% documented that, due to migraine, in the past 6 month they were unable to enjoy relaxation and free-time-activities (average frequency = 3,4). Not at least the
- Mental well-being is affected, because 44% worry about getting a migraine and about 86% of effected patients believe that their life would be better without migraine.(Evers,2006)

These date show that the impact on quality of life of affected persons can be very severe. One can expect that an improvement of troubles caused by migraine leads to a progression of life-quality could also reduce secondary illnesses such as depression and anxiety.

9. Relevance for the Osteopathy/Approaches to Treatment

Out of triage of scientific literature I came to the conclusion that trigger factors, as well as, specifity of migraine severity is very individual in each patient. Therefore it seems important to me to adjust osteopathic therapy individually to the patient. Osteopathy offers different approaches to treatment. These approaches are shown in the following parts.

The following conclusions have a character as models. At present they cannot be verified scientifically.

9.1.Anatomic reasons

A very common approach in osteopathy is, to detect disturbances in the musculoskeletal system and, if possible, to correct them.

In terms of biomechanics they often talk about different shock absorbers in the body. Klein and Sommerfeld (2004) report that in every step shocks, resulting from touching the ground with your foot, evolve. These shocks are at first affecting the foot, in further consequence they effect the whole lower extremity, the pelvis, the spine up to the skull and therefore the brain. The target of these shock absorbers is the protection of central nervous system and of important organs. To understand - in terms of biomechanics - these connections, shock absorbing mechanisms have to be adducted as model of explanation.

The mission of a shock absorbing system is, to reduce the power that occurs in an impulse, as gentle as possible. If a shock absorbing system drops out partly, the remaining mechanisms have to compensate. This leads to dysfunctions, inappropriate attitude and at least to overload.

As walking is an every day activity, minor dysfunctions can lead to reduced shock absorption, up to the upper neck. If there is a connection between an upper neck dysfunction and migraine – as explained in **3.7.anatomic context** – there has to be an anatomical connection, deduced from dysfunctions of the whole skeleton down to the foot. Therefore within the physical examination of your patient and the evolving treatment it is very important, to find dysfunction in joint-play even upraising from the foot, to correct the transmission of dysfunctions of muscle-chains and to remove a reduced effectivity of the whole shock absorbing mechanism.

Lelievre gives one example of an anatomic context between headache and disturbances in muscle balance of the anterior throat. In his Masterthesis about: Are Osteopathic Lesions of the Anterior Throat Related with Headaches, Lelievre tells in his conclusion “we can say that there is a relationship between the anterior throat lesions and headache”.

Due to the anatomic context an important structural approach for osteopathic treatment arises. After detecting the disturbance the following steps are taken:

- Improvement of joint movement
- Harmonisation of muscle function and muscle tension
- Balancing effect on the autonomic nervous system throughout treatment of the spine

The goal of the anatomic-structural treatment approach is the trouble-free function of the whole musculoskeletal system.

9.2. Visceral reasons

Another access of an osteopath is having an effect on internal organs. An undisturbed gliding of all layers of tissues is necessary for good function.Malfunctioning can release chain reactions which irritate other internal organs, as well as irritates activity in musculoskeletal system. As an example, how important the well functioning of internal organs to the whole organism is, I want to mention the digestion. Dysfunctions of digestion may have multiple ways to affect the whole body in its health.

As documented in chapter **3.7.4.immaturity of intestine / allergies as activator of migraine**, changes in the bacterial count in the intestines often occur combined with a malfunctioning metabolism of mineral material. This malfunctioning mineral material becomes noticeable in the irritability and in the neurotransmission of nerves (Strackham, 2006).

You can assume that improvements of digestion support the whole body in gaining and restoring health in all parts and therefore be effective against development of migraine.

The cited digestion example may be extended to the functioning of all other inner organs. The visceral approach to therapy is an important part of osteopathic treatment. Possibilities are:

- Gliding improvement of the organs

- Relaxation of tension in scars

The optimal functioning of all inner organs is the goal of the visceral treatment approach. This would underline the meaningfulness of visceral therapy in migraine.

9.3.Fascia

Nearly similar as with the inner organs, is the approach by fascia.

All muscles, inner organs, vasculary and neural connections are surrounded by fascia. These facia allow a frictionless sliding of different layers of textures against each other.

Attlee (2004) describes the following in his work: He emphasized the meaning of fascia as a sheath, that interconnects throughout the body and is”continuous from top to toe”. The Fascia ensures a smooth frictionless movement of all organs and therefore enables the free flow of fluids through the body along Fascia. Because of this, the integrity and free mobility of the Fascia is essential to a proper function of the whole body (Attlee).

Osteopathic treatment of fascia is establishing an additional possibility of removing disturbances. Adequate methods are:

- Elimination of abnormal tension in the gliding tissues
- Slowing down of irritated nerves connections.

It is the goal to provide a good function of all tissues in the body.

9.4. Cranio-Sacrale Therapy

Cranio-sacral therapy is another important treatment approach in osteopathy.

There are case studies that cranio-sacral therapy can contribute to an improvement of well-being of migraine-patients (Nüsslein, personal experience).

The cranio-sacral-therapy is a part of osteopathic treatment approaches. In this approach Osteopathy assumes a model-imagination that the so called Primary Respiratory mechanism (PRM), a wave motion, is the so-called breath of life. Everybody needs a good expression of PRM to stay healthy. An experienced person is able to feel the PRM in every part of the body. Nüsslein also has the experience that an improvement of the PRM, may produce a very deep relaxation of all textures. The harmonisation of the movement of the skull-bones is able to improve the circulation of liquor in the brain and in the spine, as well as improve blood circulation

and venous drainage in the inner part of the skull. One can assume that this could positively influence the situation of second messengers in the brain. Furthermore the PRM is able to initiate a special well harmonising between skull and pelvis throughout the central organ spine. This PMR can be used for balancing tensions and to harmonise of all functions in the body, in structural as well as in visceral fields. (Nüsslein)

Sutherland described the Cranio-Sacral-System as PRM with the implication, that fluent Cranio-Sacral function is the fundamental basis underlying health.

In cranio-sacral therapy one tries to:

- Release deep lying tension
- To harmonise all tissues
- To activate the self-healing-mechanism
- To eventually handle deep lying psychic traumata

The goal of cranio-sacral therapy is to re-establish harmony between body, spirt and mind.

9.5. Autonomic nervous system

Due to the fact that the autonomic nervous system influences all parts of the body, a disharmony is affecting the situation of the patient negatively. Osteopathy therefore tries to harmonise this system.

The autonomic nervous system, especially the n. vagus as an important part of the parasympathetic nervous system, is also irritated in a migraine-patient. In Chapter 3.7. anatomic context specially Bogduk (1995) explained detailed connections between different brain-nerves – N. vagus is a member of – and the upper cervical spine.

Also an increased activity of sympathetic nervous for example caused by psycho-emotional stress on the working place or in the family, may be a trigger for a migraine-attack (Evers, 2006).

Nash (2006) says, that psychological stress is generally acknowledged to be a central contributor to primary headache. Chronic activation of the stress response eventually can predispose an individual to disease.

Within my treated migraine-patients impressively I fond a connection between an increased sense of duty and the acceptance of a high grade of responsibility for

family and job. Also a high grade of accuracy and ambition play an important roll within my migraine-patients.

Raphael van Asche presumes that deep lying psychic traumata, too, which are not worked up yet, may increase the emotional stress and therefore be a precursor for a migraine. In these cases techniques like somato-emotional release and attendant cranio-sacral therapy could be helpful, if the patient appeals to his psychic traumata during a treatment. This model of somato-emotional release is not proven scientifically.

It is evident, that psycho-emotional states as nervous tension, anger, urgency or frustration are reflected in the body. It is possible, that the muscular tension as well as the blood pressure increases and therefore these emotions may lead to less efficient functioning of the body (Attlee).

The influence on this autonomic nervous system is possible in osteopathy throughout different approaches:

- Through the structural treatment approach, for example, the N. vagus may be positively influenced through harmonising the junction between the bases of the skull to the first cervical spine. The sympathetic nervous system may be positively influenced by the thoracic spine.
- Through the cranio-sacral treatment approach harmony may be re-established in the autonomic nervous system.

The goal of osteopathic treatment of the autonomic nervous system is to establish balance between the sympathetic nervous system and the parasympathetic nervous system.

9.6. Realistic expectations of possible treatment success

Point 9.1. to point 9.5.show the variety of osteopathic treatment approaches due to the fact that osteopathy examines a person as a whole. Because of this a detailed anamnesis is taken, as well as general conversation, to give information required, and mentoring is especially necessary and important in migraine treatment.

In his book Diener (2002) gives his medical colleges the hint for practical expirience that migraine-patients have to learn that a **healing of migraine is not possible**. They have to learn to live with it. He writes that neither the medicamentous prophylaxis for migraine nor the non medicamentous treating possibilities are able to heal migraine. Diener admits that it is possible throughout a consequent treatment to

influence the frequency and severity of migraine-attacks. This awareness could relieve the contact between medical practitioner and patient and could also increase the compliance of the patient. He is committed that the combination of a medicamentous treatment with a non-medicamentous method could promise the highest success rate.

In terms of improvement of life-quality of migraine-patients it is important to optimize as well the medicamentous supply of the patient as well as throughout an individual therapy and mentoring find the best combination for the patient to ease the set of problems of migraine. Important are combined efforts of practitioner, Osteopath and patient.

In an osteopathic treatment you have to bear in mind two things:

The patient has - on the one hand - to get extensive encouragement on a structural, visceral and cranio-sacral basis throughout the therapist.

As Struthers showed in a retrospective study about “Osteopathic Treatment for Cervicogenic Headaches and its Effectiveness”. She found out, that “the most successful technique was structural technique combined in combination with articulatory and cranial techniques” (2004, page V).

On the other hand, the patient has to learn throughout a specific mentoring, to agree to the individual needs of his body, to unburden his own body. Out of this it should be possible, to ease the frequency and severity of migraine-attacks.

Attlee says that an effective osteopathic treatment of migraine patients has to involve the coordinated integration at all levels of involvement. Very important for the treatment is to have an appropriate empathetic relationship with the patient. Through this empathetic feeling, one will have the possibility to affect the patient more profoundly.

10. Methodic

10.1.Pool of patients

On one hand patients, took part in my study, who were assigned to my practice from general practitioners, from neurologists, from internists and from orthopaedists with the diagnosis of migraine.

On the other hand I hung up a poster in the practice of a general practitioner concerning the performance of my study. Interested parties who were prepared to take place in my study, formed the second part of my pool of patients.

10.2. Anamnesis of migraine

Using a questionnaire which was edited by International Headache Society, I collected data regarding the present state of health of my patients, as well as the condition and course of disease within the last three months.

This MIDAS – questionnaire was developed by headache specialists. MIDAS is the short name for “Migraine Disability Assessment“. It helps to gather the influences of headaches on the quality of life of the suffering person the previous three months. There is a MIDAS questionnaire at the end of my scriptum

If the migraine existed for three months and a migraine diary existed, the process of migraine in this patient was made a note of.

In the following, I will talk about female patients because the predominant part of my patients was female.

10.3. General anamnesis/inspection

I started with the investigation of an accurate anamnesis which is typical in osteopathy, to gather as many effects as possible out of the past which may have an influence on the onset of migraine.

After this, a detailed examination of the body in standing-, sitting-, back-lying-position - and, if necessary, in side-lying-positions also followed.

All patients who had X-rays of the spine or from parts of the spine were asked to bring these with them to the first appointment. Some patients who had no x-rays but special asymmetries of the spine, caught my attention, I asked, after consultation with their attending physicians, to prepare an x-ray of the spine in a standing position.

If there was no chronicle regarding process, severity, frequency of attacks and attendant symptoms of migraine, I asked the patient to keep records. After three months of waiting time, caused by the large interest in the therapies, I was able to make new appointments to start with the therapy.

10.4. Approach for treatment

Having planned my approach to therapy, based on the to anamnesis and examination, I started my treatment.

Because the needs of my individual patients were very different, as I presumed, I - due to the “Black Box Method” –adjusted structural, visceral, as well as cranio-sacral treating approaches to the individual set of problems.

Depending on availability of my patients, as well as rapidity of improvement, I treated my patients in shorter or longer intervals, each for ten times.

Concluding, in collaboration with the patients, I collected the actual disposition and the degree of accomplished improvement. Three months after finishing the therapy, I asked each patient to come to my practice to go through the MIDAS–questionnaire together again. If this was not possible, I sent her the questionnaire to investigate the actual disposition. It was important for me to document the long-term results of the treatment.

10.5. Inclusion-criteria

- Grown-up patients with migraine without aura

10.6. Exclusion–criteria

- Migraine with aura
- Special forms of migraine such as cluster-headache, migraine accompagne with hemiplegy-like neurologic deficits
- Patients with tumors
- Patients with acute infections
- Patients with fresh traumata
- Children

11. Analysis/Statistics

Descriptive statistics

Description of sample

A total of 30 persons were treated and interviewed from December 2006 to April 2008. In the following, common characters of the sample are described.

Description of the sample due to gender

The predominant majority of interviewees are female patients with 86.7% (26 of 30 interviewees).

Gender

		Frequency	Percentage	Legal Percentage	Cumulative Percentage
legal	f	26	86.7	86.7	86.7
	m	4	13.3	13.3	100.0
	total	30	100.0	100.0	

Description of the sample due to age-composition

The age range within the random check is relatively high at the age of 44 years. The youngest patient is 18 years old, the eldest patient, 62. The average age lies with 42 years; about 50% of patients are between 40 and 50 years old.

Description of the sample due to sporty activity

Since regularly exercise or sport a positive influence on migraine is presumed, patients were asked about their sporty activity. The rate is well-balanced: 46.7% stated participation regularly in sports.

Does the patient operate regularly in sports?

		Frequency	Percentage	Legal Percentage	Cumulative Percentage
legal	Not regularly	16	53.3	53.3	53.3
	Regularly	14	46.7	46.7	100.0
	Total	30	100.0	100.0	

Description of the sample due to migraine attacks in the family

Because in migraine without aura a genetic disposition is presumed, too, I asked about migraine in the family of origin. In 73.3% of patients further migraine-attacks are known within family.

Migraine within the family

		Frequency	Percentage	Legal Percentage	Cumulative Percentage
legal	no	8	26.7	26.7	26.7
	yes	22	73.3	73.3	100.0
	total	30	100.0	100.0	

Analysis of trigger factors

The patients were asked to list, which trigger factors release a migraine in their experiences. The most common factors were stress (93.3% of interviewees), anger (56.7%), the weather (46.7%) and light (43.3%).

		no	yes
Trigger-factor stress	Number	2	28
	%	6.7%	93.3%
Trigger-factor anger	Number	21	9
	%	70.0%	30.0%
Trigger-factor deficit of sleep	Number	13	17
	%	43.3%	56.7%
Trigger-factor foods	Number	22	8
	%	73.3%	26.7%
Trigger-factor alcohol	Number	19	11
	%	63.3%	36.7%
Trigger-factor histamin	Number	26	4
	%	86.7%	13.3%
Trigger-factor smells	Number	18	12
	%	60.0%	40.0%
Trigger-factor weather	Number	16	14
	%	53.3%	46.7%
Trigger-factor noise	Number	22	8
	%	73.3%	26.7%
Trigger-factor light	Number	17	13
	%	56.7%	43.3%
Trigger-factor hormons	Number	21	9
	%	70.0%	30.0%

Spine-diseases

Strackharn (2003) and Bogduk (1995) see a connection between changes in the cervical spine and migraine development. Strackharn names scoliosis in part of the thoracic spine as an accelerator of attacks. Due to this I have collected available spine-diseases in the patients. 90% of interviewees named problems in the cervical spine area, 53.3% have problems in the lumbar spine. 46.7% suffer from scoliosis.

	Problems in cervical spine		Problems in thoracic spine		Problems in lumbar spine		Scoliosis	
	Number	%	Number	%	Number	%	Number	%
no	3	10.0%	24	80.0%	14	46.7%	16	53.3%
yes	27	90.0%	6	20.0%	16	53.3%	14	46.7%

Additional diseases

In „Ten lessons on the epidemiology of migraine“ from Lipton and Bigal (2007) it emerges that migraine-patients suffer more often from depression, so I asked about depressive diseases within my patients. Carreiro (2007) posed the question, whether a certain immaturity of intestine could be one more reason for development of migraine. Due to this, I have also asked my patients about distinctive disorders in the intestines in the form of “Colon irritabile“.

43.3% of patients suffer from depression, only 13.3% said that they have allergies, 33.3% said that they suffer from „Colon irritations“

	Additional disease depression		Additional disease allergy		Additional disease Colon irritabile	
	Number	%	Number	%	Number	%
No	17	56.7%	26	86.7%	20	66.7%
yes	13	43.3%	4	13.3%	10	33.3%

Accompaniment

The question about accompaniments of migraine disease resulted that all affected patients (100%) suffer from a loss of power during a migraine-attack. 93.3% reported to be struck by nausea, in 83.3% hypersensitivity against light was reported, in 73.3% hypersensitivity against noise and in 70% hypersensitivity to smells was reported.

		no	yes
Nausea	Number	2	28
	%	6.7%	93.3%
Nausea and vomiting	Number	21	9
	%	70.0%	30.0%
Hypersensitivity against light	Number	5	25
	%	16.7%	83.3%
Hypersensitivity against noise	Number	8	22
	%	26.7%	73.3%
Hypersensitivity to smells	Number	9	21
	%	30.0%	70.0%
Ravenous appetite	Number	26	4
	%	86.7%	13.3%
Loss of appetite	Number	17	13
	%	56.7%	43.3%
Loss of power	Number		30
	%		100.0%

Description of pain

To paint a more precise picture of arising pain, patients were requested to escribe the localisation and character of pain.

Localisation of pain

Most commonly the pain is in the area of the eyes (in 76.7% of interviewees), followed by temporal area(73.3%) and occipital area(70%). Only 10.3% suffer from pain in the area of the nose.

		no	yes
Localisation of pain eye	Number	7	23
	%	23.3%	76.7%
Localisation of pain frontal	Number	16	14
	%	53.3%	46.7%
Localisation of pain temporal	Number	8	22
	%	26.7%	73.3%
Localisation of pain parietal	Number	24	6
	%	80.0%	20.0%
Localisation of pain occipital	Number	9	21
	%	30.0%	70.0%
Localisation of pain nose	Number	26	3
	%	89.7%	10.3%

Character of pain

About three-quarter of patients (76.7%) describe their pain as pulsative/beating. Stinging and respectively dull/oppressive pain appears in 53.3% as the case may be in 46.7% of interviewees. Much more seldomly pain which is referred into the anterior muscles of the neck.

	Character of pain pulsative/beating		Character of pain dull/oppressive		Character of pain stinging		Pain referred into the anterior muscles of the neck	
	Number	%	Number	%	Number	%	Number	%
no	7	23.3%	16	53.3%	14	46.7%	28	93.3%
yes	23	76.7%	14	46.7%	16	53.3%	2	6.7%

Other factors

Bigal and Lipton (2006) report that intensive obesity could be a factor, through which migraine can progress into a chronic and more intensive form. Therefore I have asked about irregularities in eating behaviour.

Because it is presumed that all diseases which convert the metabolism activity inauspiciously, have an influence on process of migraine, I asked my patients about this topic. One patient had an acute pankreatitis left behind.

At a certain point of severity of migraine, patients are recommended to take special medication as a prevention of migraine. Therefore, I wanted to know how many of my patients actually do this.

		no	yes
Preventive medicine	Number	23	7
	%	76.7%	23.3%
Dysregulation in eating behavior, severe obesity	Number	27	3
	%	90.0%	10.0%
Eating behavior severe anorexia	Number	25	5
	%	83.3%	16.7%
Dysregulation in the thyroid gland	Number	26	4
	%	86.7%	13.3%
Acute pancreatitis	Number	29	1
	%	96.7%	3.3%

Analysis of MIDAS-questionnaire

The MIDAS-questionnaire is a valid test which was developed by the International Headache Society to collect the disabilities caused by migraine.

In the following, several questions with regard to changes over time and conclusions regarding success of therapy are analysed.

Question 1

Descriptive statistics

Before starting the treatment, patients could not go to their job because of headaches – on average for 3,35 days within 3 months. About 50% of interviewees were not able to work for 2 to 5 days. Maximum values before starting treatment was at 12 days and was reduced - due to treatment - to 6 days with the average value declining to 1.69 days after finishing therapy.

I don't dare to assume that this considerable reduction be regarded as success of treatment.

I tried trough mentoring to encourage my patients to take personal responsibility for their illness patterns. For example, through changes in daily activities to include phases of relaxation to reduce stress. Regular control of the positioning of the body, for example, while working on the computer, could help to balance tension in the muscles. In addition to this I have tried to improve the cooperation with the attending physicians, for example, to refer back to the neurologist to improve medication.

It is my belief that, only by exerting an influence on as many factors as possible, a clear and persistent improvement of the set of problems related to migraine can be achieved.

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The standard deviation (which is the spreading of several indicated values around the average value) reduced continuously from 3.046 before treatment to 2.015 to 1.784 in the 3 months after therapy completion.

Descriptive statistics

	N	Mean	Standard Deviation	Minimum	Maximum
MIDAS questionnaire question 1, 3 months before starting treatment, in days	26	3.35	3.046	0	12
MIDAS questionnaire, question 1, after finishing treatment, in days	26	1.69	2.015	0	6
MIDAS questionnaire, question 1, 3 months later, in days	26	1.31	1.784	0	6

Whereas, before starting treatment only 26,9% of patients reported no days at work missed due to migraine pain, this percentage increased after finishing treatment to 40.7% and, in the following 3 months, to 48.1%.

Statements about the therapy success

To check statements related to the therapy success to see whether the decline really shows therapy success. The following examines statistical significance. An accidental reduction of days missed and random checks on the differences in the three appointments were checked for significance.

In addition, a test was applied on the first step of normal distribution. Because it turned out that the random check shows no normal distribution (p=.200) before the beginning of treatment, a non-parametric procedure is applied for comparison of the three random checks (Friedmann test for k dependent random checks).

Tests on normal distribution

	Kolmogorov-Smirnov(a)			Shapiro-Wilk		
	Statistics	df	Signifi cance	Statistics	df	Signifi cance
MIDAS questionnaire question 1, 3 months before starting treatment, in days	.136	26	.200(*)	.901	26	.016
MIDAS questionnaire question 1, after finishing therapy, in days	.223	26	.002	.794	26	.000
MIDAS questionnaire question 1, 3 months afterwards, in days	.268	26	.000	.756	26	.000

* This is a lower border of the real significance.
a significance correction to Lilliefors

The comparison of the middle values in the course of the Friedmann test shows that at least two of the random checks differ highly significantly to each other (p= .000), which means they cannot be gained from an accidental development.

Values

	Middle value
MIDAS questionnaire question 1, 3 months before starting treatment, in days	2.73
MIDAS questionnaire question 1,after finishing therapy, in days	1.79
MIDAS questionnaire question 1, 3 months afterwards, in days	1.48

Statistic for test(a)

N	26
Chi-Quadrat	35.292
df	2
Asymptotic significance	.000

a Friedman-test

The other investigation, in pairs, by means of the Wilcoxon-test shows that, between all of the three random checks, statistically highly significant differences exist (p= .000 or. p= .008).

Statistic for test(b)

	MIDAS questionnaire question 1, after finishing therapy, in days - MIDAS questionnaire question 1, 3 months before starting treatment, in days	MIDAS questionnaire question 1, 3 months afterwards, in days - MIDAS questionnaire question 1, 3 months before starting treatment, in days	MIDAS questionnaire question 1, 3 months afterwards, in days - MIDAS questionnaire question 1, after finishing therapy, in days
Z	-3.974(a)	-3.880(a)	-2.640(a)
Asymptotic significance (2-tailed)	.000	.000	.008

a Based on positive ranks.

b Wilcoxon-test

Question 2

Descriptive statistics

In the last three months before therapy began, the average of the number of days on which the ability of patients was reduced to half or more at their working place, was at 8.72 days. A maximum of 35 days was mentioned. This data, too, was reduced clearly during course of the period of examination: the average was at 4.36 after finishing therapy and kept on sinking in the three months following, to 3.6 days. The maximum reduction was 10 days. Standard deviation (scattering of data around the average) too, was reduced from 7.080 (before starting treatment) to 3.062(after finishing therapy) to 2.646 in the three months following.

Descriptive statistics

	N	Mean	Standard Deviation	Minimum	Maximum
MIDAS questionnaire question 2, 3 months before starting treatment, in days	25	8.72	7.080	0	35
MIDAS questionnaire question 2, after finishing therapy, in days	25	4.36	3.026	0	12
MIDAS questionnaire question 2, 3 months afterwards, in days	25	3.60	2.646	0	10

Statements about therapy success

Again it is checked whether this reduction shows statistical significance, or whether it concerns merely an accidental development. The test at normal distribution shows that the random checks show after therapy end and afterwards no normal distribution (p= .200) which is why, again, the Friedman-test as a non-parametric testing method.

Tests on normal distribution

	Kolmogorov-Smirnov(a)			Shapiro-Wilk		
	Statistics	df	Signifi cance	Statistics	df	Signifi cance
MIDAS questionnaire question 2, 3 months before starting treatment, in days	.228	25	.002	.797	25	.000
MIDAS questionnaire question 2, after finishing therapy, in days	.136	25	.200(*)	.937	25	.124
MIDAS questionnaire question 2, 3 months afterwards, in days	.117	25	.200(*)	.948	25	.228

* This is a lower border of real significance.
a Significance correction to Lilliefors

The comparison of the middle values proved again that, at least between two of three random checks, a statistically highly significant difference exists (p=.000), and coincidence is excluded.

Rank

	Mean Rank
MIDAS questionnaire question 2, 3 months before starting treatment, in days	2.92
MIDAS questionnaire question 2, after finishing therapy, in days	1.68
MIDAS questionnaire question 2, 3 months afterwards, in days	1.40

Statistics for test(a)

N	25
Chi-Quadrat	43,053
df	2
Asymptotic significance	,000

a Friedman-test

The other investigation in pairs by means of the Wilcoxon-test shows that, between all three of the random checks, somewhat highly significant differences exist (p= .000 or. p= .014).

Statistics for test(b)

	MIDAS questionnaire question 2, after finishing therapy, in days - MIDAS questionnaire question 2, 3 months before starting treatment, in days	MIDAS questionnaire question 2, 3 months afterwards, in days - MIDAS questionnaire question 2, 3 months before starting treatment, in days	MIDAS questionnaire question 2, 3 months afterwards, in days - MIDAS questionnaire question 2, after finishing therapy, in days
Z	-4.214(a)	-4.296(a)	-2.456(a)
Asymptotic significance (2-tailed)	.000	.000	.014

a Based on positive ranks
b Wilcoxon-test

Question 3

Descriptive statistics

The patients could do no housework before the beginning of treatment on average on 1.28 days on account of the headaches during the last three months. The maximum value lay at 19 days. After the end of the therapy, the average lay at 0.97, the maximum at 12 days. Also, during 3 months after the therapy, the values sank further to 0.38 (average) and 4 (maximum). The random checks became more compact in itself as seen in the change of the standard divergence points: before the beginning of treatment, the values scattered on average at about 3.693 days round the average, after the end of the therapy, at about 2.556 days, and during three months afterwards, at about 1.049 days.

Descriptive statistics

	N	Mean	Standard Deviation	Minimum	Maximum
MIDAS questionnaire question 3, 3 before starting treatment, in days	29	1.28	3.693	0	19
MIDAS questionnaire question 3, after finishing therapy, in days	29	.97	2.556	0	12
MIDAS questionnaire question 3, 3 months afterwards, in days	29	.38	1.049	0	4

Statements about the therapy success

To be able to make conclusions on the therapy success, the random checks are checked first because of normal distribution. All three random checks show a normal distribution (p= .000), which is why the t-test is applied with combined random checks as parametric procedure to the other examination of the statistical significance.

Tests on normal distribution

	Kolmogorov-Smirnov(a)			Shapiro-Wilk		
	Statistics	df	Signifi cance	Statistics	df	Significanc e
MIDAS questionnaire question 3, 3 months before starting treatment, in days	.394	29	.000	.393	29	.000
MIDAS questionnaire question 3, after finishing therapy, in days	.440	29	.000	.449	29	.000
MIDAS questionnaire question 3, 3 months afterwards, in days	.503	29	.000	.412	29	.000

a Significance correction to Lilliefors

The comparison of the averages of the three random checks in the course of the t-test occurs in pairs. Therefore, every measurement for similarities and differences.

Paired Samples Test

		Paired Differences					T	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	MIDAS questionnaire question 3, 3 months before starting treatment, in days - MIDAS questionnaire question 3, after finishing therapy, in days	.310	1.713	.318	-.341	.962	.975	28	.338
Pair 2	MIDAS questionnaire question 3, 3 months before starting treatment, in days - MIDAS questionnaire question 3, 3 months afterwards, in days	.897	3.559	.661	-.457	2.250	1.357	28	.186
Pair 3	MIDAS questionnaire question 3, after finishing therapy, in days - MIDAS questionnaire question 3, 3 months afterwards, in days	.586	2.338	.434	-.303	1.475	1.350	28	.188

The results of the t-test show that among no pairs of the three random checks does a significant difference exist (p = .338 or. p = .186 or. p = .188). This means that,

coincidence in the development of the values is more than three measurements away.

Question 4

Descriptive statistics

Before starting treatment (within the last 3 months patients documented), restrictions in productivity in housekeeping to the half or more was due to headache. This average was reduced clearly by the time of treatment finish to 2.97 days. Within the following 3 months it sank a little to 2.52 days. Maximum data at begin was 42 days and sank to 34 days after treatment finish and to 27 days in the following 3 months. Standard deviation, too, reduced in the course of the examination period.

Descriptive statistics

	N	Minimum	Maximum	Mean	Standard Deviation
MIDAS questionnaire question 4, 3 months before starting treatment, in days	29	0	42	6.38	10.428
MIDAS questionnaire question 4, after finishing therapy, in days	29	0	34	2.97	6.434
MIDAS questionnaire question 4, 3 months afterwards, in days	29	0	27	2.52	5.221
Valid N (listwise)	29				

Statements about the therapy success

To be able to make declarations about the therapy success the random checks are checked first for normal distribution and are analysed on account of the unequivocal results (p= .000) by means of the t-test for combined random checks on equality of the averages there.

Tests on normal distribution

	Kolmogorov-Smirnov(a)			Shapiro-Wilk		
	Statistics	df	Signifi cance	Statistics	df	Signifi cance
MIDAS questionnaire question 4, 3 months before starting treatment, in days	.270	29	.000	.626	29	.000
MIDAS questionnaire question 4,after finishing therapy, in days	.322	29	.000	.464	29	.000
MIDAS questionnaire question 4, 3 months afterwards, in days	.315	29	.000	.494	29	.000

a Significance correction to Lilliefors

The comparison of the averages of the three random checks occurs again in pairs. Therefore, every measurement is compared for both similarities and differences.

Paired Samples Test

		Paired Differences					T	df	Sig. (2-railed)
		Mean	Std.Deviation	Std. Error Mean	95% Confidence Intervall of the Difference Upperr	Low er			
Pair 1	MIDAS questionnaire question 4, 3 months before starting treatment, in days - MIDAS questionnaire question 4,after finishing therapy, in days	3.414	7.557	1.403	.539	6.28 8	2.433	28	.022
Pair 2	MIDASquestionnair e question 4, 3 months before starting treatment, in days - MIDAS questionnaire question 4, 3 months afterwards, in days	3.862	7.749	1.439	.914	6.81 0	2.684	28	.012
Pair 3	MIDAS questionnaire question 4, after finishing therapy, in days - MIDAS questionnaire question 4, 3 months afterwards, in days	.448	1.404	.261	-.086	.982	1.720	28	.097

The results show clearly that, between the random check before the beginning of treatment (measurement 1) and the data after end of the therapy (measurement 2) a

statistically significant difference (p =.022), also between measurement 1 and measurement 3 (1-3 months ago). No accidental development can be mentioned. Indeed, the values have not changed between measurement 2 and measurement 3 any more significantly (p =.097).

Question 5

Descriptive statistics

During the last 3 month before treatment started, social or leisure activities because of their headache for an average of 3.73 days patients could not take part in family.. The average sank after finishing therapy to 2.3 days and in further succession to 1.43 days. Maximum data is conspicuous and standard deviation does not reduce between the first two measurements but rather increases clearly. This tendency, however, is mainly caused by one singular case with an abrupt rise in restricted days.

Descriptive statistics

	N	Minimum	Maximum	Mean	Standard Deviation
MIDAS questionnaire question 5, 3 months before stating treatment, in days	30	0	20	3.73	3.704
MIDAS questionnaire question 5, after finishing therapy, in days	30	0	34	2.30	6.154
MIDAS questionnaire question 5, 3 months afterwards, in days	30	0	15	1.43	2.825
Valid N (listwise)	30				

Statements about the therapy success

To analyse statements about the therapy success around the development of the random checks for the investigation period concerning statistical significance, a test on normal distribution is carried out first. This proves that the random checks are normally-distributed by all measurements (p =.000), which is why, again, the t test is used for combined random checks for comparison of the averages.

Tests on normal distribution

	Kolmogorov-Smirnov(a)			Shapiro-Wilk		
	Statistics	df	Signifi cance	Statistics	df	Signifi cance
MIDAS questionnaire question 5, 3 months before starting treatment, in days	.233	30	.000	.675	30	.000
MIDAS questionnaire question 5, after finishing therapy, in days	.354	30	.000	.349	30	.000
MIDAS questionnaire question 5, 3 months afterwards, in days	.328	30	.000	.487	30	.000

a Significance correction to Lilliefors

The comparison of the averages of the three random checks occurs again in pairs. Therefore, every measurement is compared for both similarity and differences.

Paired Samples Test

		Paired Differences					T	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Upper	Lower			
Pair 1	MIDAS questionnaire question 5, 3 months before starting treatment, in days - MIDAS questionnaire question 5, after finishing therapy, in days	1.433	3.626	.662	.079	2.787	2.165	29	.039
Pair 2	MIDAS questionnaire question 5, 3 months before starting treatment, in days - MIDAS questionnaire question 5, 3 months afterwards, in days	2.300	2.103	.384	1.515	3.085	5.989	29	.000
Pair 3	MIDAS questionnaire question 5,after finishing therapy, in days - MIDAS questionnaire question 5, 3 month afterwards, in days	.867	3.491	.637	-.437	2.170	1.360	29	.184

The comparison of sample average data due to t-test shows that, in between data before treatment started (measurement 1) and data after finishing therapy

(measurement 2), there was a significant difference (p=.039). Likewise, in between measurement 1 and data 3 months after finishing treatment (p=.000). However, here, too, data in between measurement 2 and 3 have not changed significantly (p=.184).

Summative value of questions 1 – 5

The values from the questions 1-5 were added – in three measurements - and from that three new variables were provided. On account of partially missing values, 5 cases had to be excluded with this procedure by the first measurement and 4 cases in the second and the third measurement which would have otherwise falsified the results. The newly calculated variables are the sum of the values from questions 1-5 before the beginning of treatment (n=25), the sum of the values after end of the therapy (n=26) and the sum of the values 1-3 months ago (n=26).

Descriptive statistics

The average of the sum was at 20.88 days before starting the therapy and was cut in half to 10.04 days after finishing treatment. Within the following 3 months, it reduced again to 7.88 days. Even the maximum data in between the first two measurements sank considerably from 78 to 24 days. In between measurement 2 and 3, it sank only about 2 days to 22 days. The comparison (of middle grade) shows that, at least between 2 out of 3 samples a statistically high significant difference exists (p=.000), and coincidence is excluded.

Descriptive statistics

	N	Minimum	Maximum	Mean	Standard Deviation
Summative values of questions 1-5, 3 months before starting treatment, in days	25	6	78	20.88	15.265
Summative values of questions 1-5, after finishing therapy, in days	26	0	24	10.04	6.527
Summative values of questions 1-5, 3 months afterwards, in days	26	0	22	7.88	5.595
Valid N (listwise)	25				

Statements about the therapy success

The random checks concerning a normal distribution and the Kolmogorov-Smirnov test show that only at the measurement before the beginning of treatment does a

normal distribution rules. Hence, the Friedman test is used for comparison of the random checks as a non-parametric test for more than two random checks.

Tests on normal distribution

	Kolmogorov-Smirnov(a)			Shapiro-Wilk		
	Statistics	df	Signifi cance	Statistics	df	Significanc e
Summative value of questions 1-5, 3 months before starting treatment, in days	.219	25	.003	.755	25	.000
Summative value of questions 1-5, after finishing therapy, in days	.102	25	.200(*)	.959	25	.403
Summative value of questions 1-5, 3 months afterwards, in days	.123	25	.200(*)	.946	25	.205

* This is a lower border of the real siginficance
a Significance correction to Lilliefors

The comparison of the middle values proved that, at least between two of three random checks, a statistically highly significant difference exists (p =,000), and coincidence is excluded.

Rank

	Mean Rank
Summative values of questions 1-5, 3 months before starting treatment, in days	3.00
Summative values of questions 1-5, after finishing therapy, in days	1.76
SUM QUESTION 1-5, 3 months afterwards, in days	1.24

Statistics for test(a)

N	25
Chi-Quadrat	45.422
df	2
Asymptotic significance	.000

a Friedman-test

The other investigation in pairs with the help of the Wilcoxon test shows that, between all of the three random checks a statistically highly significant difference exists (p =.000 or p =.001).

Statistics for test(b)

	Summative values of questions 1-5, after finishing therapy, in days – Summative values of questions 1-5, 3 months before starting treatment, in days	Summative value of questions 1-5, 3 months afterwards, in days – Summative value of questions 1-5, 3 months before starting treatment, in days	Summative value of questions 1-5, 3 months afterwards Summative value of questions 1-5, after finishing therapy, in days
Z	-4.377(a)	-4.383(a)	-3.210(a)
Asymptotic significance(2-tailed)	.000	.000	.001

a Basiert auf positiven Rängen.
b Wilcoxon-Test

Question A

Descriptive statistics

Before starting treatment patients were suffering from headache at an average of 23.63 days within 3 months. The maximum was 70 days. Within the course of the period of examination average data reduced to 15.27 days after finishing therapy and to 10.83 within the 3 month following. Conspicuous again is that the maximum and the standard deviation increase in between the first 2 measurements, which again could be ascribed to 2 isolated cases.

Descriptive statistics

	N	Mean	Standard Deviation	Minimum	Maximum
MIDAS questionnaire question A, 3 months before starting treatment, in days	30	23.63	13.402	6	70
MIDAS questionnaire question A, after finishing therapy, in days	30	15.27	16.324	0	90
MIDAS questionnaire question A, 3 months afterwards, in days	30	10.83	7.321	0	30

Statements about the therapy success

Statements about the therapy success at three random checks are checked in the following for normal distribution to be able to select the suitable test procedure for comparison of the values. Because the random check is not normally-distributed by

the first measurement (p =.200), the Friedman test is used as a non-parametric procedure.

Tests on normal distribution

	Kolmogorov-Smirnov(a)			Shapiro-Wilk		
	Statistics	df	Signifi cance	Statistics	df	Signifi cance
MIDAS questionnaire question A, 3 months before starting treatment, in days	.097	30	.200(*)	.899	30	.008
MIDAS questionnaire question A, after finishing therapy, in days	.240	30	.000	.632	30	.000
MIDAS questionnaire question A, 3 months afterwards, in days	.170	30	.027	.911	30	.016

* This is a lower border of the real significance.
a Significance correction to Lilliefors

The comparison of the middle values proved that, at least between two of three random checks, a statistically highly significant difference exists (p =.000), and coincidence is excluded.

Rank

	Mean Rank
MIDAS questionnaire question A, 3 months before starting treatment, in days	2.93
MIDAS questionnaire question A, after finishing therapy, in days	1.93
MIDAS questionnaire question A, 3 months afterwards, in days	1,13

Statistics for test(a)

N	30
Chi-Quadrat	52.286
df	2
Asymptotic significance	.000

a Friedman-test

The other investigation in pairs by means of the Wilcoxon test shows that, between all of the three random checks, statistically highly significant differences exist (p =.000).

Statistics for test(b)

	MIDAS questionnaire question A, after finishing therapy, in days – MIDAS questionnaire question A, 3 months before starting treatment, in days	MIDAS questionnaire question A, 3 months afterwards, in days - MIDAS questionnaire question A, 3 months before starting treatment, in days	MIDAS questionnaire question A, 3 months afterwards, in days - MIDAS questionnaire question A, after finishing therapy, in days
Z	-4.015(a)	-4.787(a)	-4.128(a)
Asymptotic significance (2-tailed)	.000	.000	.000

a Based on positive ranks.
b Wilcoxon-test

Question B - migraine

Descriptive statistics

The patients were asked to value her migraine headaches on a scale from 1-10. Before the beginning of the treatment, the average lay at 8.43, after end of the therapy at 7.70 and in 3 months afterwards at 7.67. In addition, the dispersion of the values is relatively low (see standard divergence).

Descriptive statistics

	N	Minimum	Maximum	Mean	Standard Deviation
MIDAS questionnaire question B migraine, 3 months before starting treatment, strength from 0-10	30	5	10	8.43	1.104
MIDAS questionnaire question B migraine, after finishing therapy, strength from 0-10	30	4	10	7.70	1.418
MIDAS questionnaire question B migraine, 3 months afterwards, strength from 0-10	30	4	10	7.67	1.398
Valid N (listwise)	30				

Statements about the therapy success

To be able to make conclusive statements about the therapy success, three random checks are checked for normal distribution again to be able to select the suitable test procedure. All three random checks are normal distributed (p =.000) which is why the t test for combined random checks for comparison of the averages is applied.

Tests on normal distribution

	Kolmogorov-Smirnov(a)			Shapiro-Wilk		
	Statistics	df	Signifi cance	Statistics	df	Signifi cance
MIDAS questionnaire question B migraine, 3 months before starting treatment, strength from 0-10	.263	30	.000	.838	30	.000
MIDAS questionnaire question B migraine, after finishing therapy, strength from 0-10	.284	30	.000	.878	30	.002
MIDAS questionnaire question B migraine, 3 months afterwards, strength from 0-10	.294	30	.000	.833	30	.000

a Significance correction to Lilliefors

The comparison of the averages of three random checks occurs again in pairs.

Therefore, every measurement is compared to similarity and differences.

Paired Samples Test

		Paired Differences					T	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Upper	Lower			
Pair 1	MIDAS questionnaire question B Migraine, 3 months before strating treatment, Strength from 0-10 – MIDAS questionnaire question B Migraine, after finishing therapy, Strength from 0-10	.733	.868	.159	.409	1.058	4.626	29	.000
Pair 2	MIDAS questionnaire question B Migraine, 3 months before starting treatment, Strength from 0-10 - MIDAS questionnaire question B Migraine, 3 months afterwards, Strength from 0-10	.767	.935	.171	.417	1.116	4.490	29	.000
Pair 3	MIDAS questionnaire question B Migraine, after finishing therapy, Strength from 0-10 - MIDAS questionnaire question B Migraine, 3 months afterwards, Strength from 0-10	.033	.765	.140	-.252	.319	.239	29	.813

The comparison of average data (due to T-Test results in between the data) before starting treatment and after finishing therapy, as well as, in the three months afterwards shows in each case a significantly high difference exists (p=.000). In between the measurement 2 and 3, the sample has not changed significantly (p=.813).

Question B - headache

Descriptive statistics

Patients additionally were asked to evaluate their headache in a spectrum from 1 – 10. The average before starting the treatment was at 5.08, after finishing therapy at 4.20 and in the three following months at 3.84. The scattering of data is relatively small (see standard deviation).

The calculation shows that, between all of three samples there are partially high significant differences, due to this, there was no coincidence in the course of period of examination.

Descriptive statistics

	N	Minimum	Maximum	Mean	Standard Deviation
MIDAS questionnaire question B headache, 3 months before starting treatment, strength from 0-10	25	3	7	5.08	1.115
MIDAS questionnaire question B headache, after finishing therapy, strength from 0-10	25	2	6	4.20	.913
MIDAS questionnaire question B headache, 3 months afterwards, strength from 0-10	25	2	6	3.84	.898
Valid N (listwise)	25				

Statements about the therapy success

The test to Kolmogorov-Smirnov shows that all three random checks are normal distributed (p =. 001 or p =.005 or p =.002), which is why the t test for combined random checks for comparison of the averages is applied.

Tests on normal distribution

	Kolmogorov-Smirnov(a)			Shapiro-Wilk		
	Statistics	df	Signifi cance	Statistics	df	Signifi cance
MIDAS questionnaire question B headache, 3 months before starting treatment, strength from 0-10	.231	25	.001	.904	25	.022
MIDAS questionnaire question B headache,after finishing therapy, strength from 0-10	.213	25	.005	.892	25	.012
MIDAS questionnaire question B headache, 3 months afterwards, strength from 0-10	.229	25	.002	.895	25	.014

a Significance correction to Lilliefors

The comparison of the averages of the three random checks occurs again in pairs. Therefore, every measurement is compared to similarity and differences

The calculation shows that between all three random checks partly highly significant differences exist and coincidence is excluded.

Paired Samples Test

		Paired Differences					T	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Upper	95% Confidence Interval of the Difference Lower			
Pair 1	MIDASquestionnaire question B Kopfschmerz, 3 Monate vor Behandlungsbeginn, Stärke von 0-10 - MIDAS Fragebogen Frage B Kopfschmerz, nach Abschluss der Therapie, Stärke von 0-10	.880	1.054	.211	.445	1.315	4.176	24	.000
Pair 2	MIDAS Fragebogen Frage B Kopfschmerz, 3 Monate vor Behandlungsbeginn, Stärke von 0-10 - MIDAS Fragebogen Frage B Kopfschmerz, 1-3 Monate danach, Stärke von 0-10	1.240	1.052	.210	.806	1.674	5.894	24	.000
Pair 3	MIDAS questionnaire question B headache, after finishing therapy, Strength from 0-10 - MIDAS questionnaire question B headache, 3 months afterwards, Strength from von 0-10	.360	.757	.151	.047	.673	2.377	24	.026

Results of question 1 – 5 in degree according to MIDAS-questionnaire

The values from the question 1-5 were added as described in three measurements and, from it, three new variables were provided. Based on the parameters of MIDAS-questionnaire these parameters were converted in three new variables with the unity in degree:

0-5 points – few or no interference – degree I

6-10 points – low interference – degree II

11-20 points – moderate interference – degree III

21 + - heavy interference – degree VI

Frequencies

The frequency distribution shows that, before starting treatment the degree III with 40%was strongest represented, followed by degree IV with 26.7%. After finishing the

therapy the degree IV appeared only with 6.7% of the patients, also the frequency of degree III moved in favour of the lower degrees.

Degree sum of the question 1-5 before the therapy beginning

		Frequency	Percent	Valid Percent	Accumulated Percent
Valid	II	5	16.7	20.0	20.0
	III	12	40.0	48.0	6.,0
	IV	8	26.7	32.0	100.0
	Total	25	83.3	100.0	
Missing		5	16.7		
Total		30	100.0		

Degree sum of the question 1-5 after end of the therapy

		Frequency	Percent	Valid Percent	Accumulated Percent
Valid	I	6	20.0	23.1	23.1
	II	7	23.3	26.9	50.0
	III	11	36.7	42.3	92.3
	IV	2	6.7	7.7	100.0
	Total	26	86.7	100.0	
Missing		4	13.3		
Total		30	100.0		

Degree sum of the question 1-5 3 months afterwards

		Frequency	Percent	Valid Percent	Accumulated Percent
Valid	I	9	30.0	34.6	34.6
	II	10	33.3	38.5	73.1
	III	6	20.0	23.1	96.2
	IV	1	3.3	3.8	100.0
	Total	26	86.7	100.0	
Missing		4	13.3		
Total		30	100.0		

During three months after finishing therapy the most frequent values appeared at degree II at 33.3%, degree IV appears only with one patient.

Statements regarding the therapy success

Due to ordinal-scaled data, the Friedman-test is used as a non-parametric procedure for analysis of the data.

The comparison of the middle value proved – according to the results of the calculations with the variable sum 1-5 – that at least between two of the three random checks a statistically high significant difference exists (p= .000) and coincidence is excluded.

Rank

	Mean Rank
Degree sum of question 1-5 before starting treatment	2.80
Degree sum of question 1-5 after finishing therapy	1.82
Degree sum of question 1-5 3 months afterwards	1.38

Statistics for test(a)

N	25
Chi-Quadrat	37.211
df	2
Asymptotic Significance	.000

a Friedman-test

The other investigation in pairs with the help of the Wilcoxon-tests shows that, between all of the three random checks, statistically highly significant differences exist (p= .000 or. p= .007), there has been a significant improvement in the course of the time.

Statistics for test(b)

	Degree sum of question 1-5 after finishing therapy – degree sum of question 1-5 before starting treatment	Degree sum of the question 1-5 3 months afterwards – degree sum of the question 1-5 before starting treatment	Degree sum of the question 1-5 3 months afterwards – Degree sum of the question 1-5 after finishing therapy
Z	-3.944(a)	-4.456(a)	-2.714(a)
Asymptotic Significance (2-tailed)	.000	.000	.007

a Based on positive Ranks.
b Wilcoxon-test

12. Discussion:

Quite positive results concerning treatment success appeared**.** On the basis of the MIDAS questionnaire the following is revealed.

Question 1)

During the last 3 months before treatment started, patients were not able to go to work, on average for 3.35 days. 50% of interviewees were not able to go to work for 2 to 5 days. The maximum number of missed days was, for one patient, 12 days which was reduced - due to treatment - to 6 days. The average of missed days sank to 1.69 days after finishing therapy. Further investigation (based on Wilcoxon tests) shows that, between all of the 3 samples, highly significant differences exist.

Question 2)

In the last three months before therapy began the average of the number of days on which the ability of patients was reduced to half or more at their working place, was at 8.72 days. A maximum of 35 days was mentioned. This data, too, was reduced clearly during course of the period of examination: the average was at 4.36 after finishing therapy and kept on sinking in the 3 months following, to 3.6 days. The maximum reduction was 10 days. The comparison (of middle value) proved again that, at least in between two of three random checks, a statistically high significant difference exists.

Question 3)

During last 3 months - due to headache - patients where not able to do housework on average for 1.28 days before starting treatment. Maximum data was 19 days. After therapy finish, the average was 0.97, with a maximum at 12 days. Also in the 3 months after finishing treatment, data continued to reduce to 0.38 (average) and 4 (maximum).

Question 4)

Before starting treatment within the last 3 months patients documented, to be restricted in productivity in housekeeping to the half or more due to headache. This

average was reduced clearly by the time of treatment finish to 2.97 days. Within the following 3 months it sank a little to 2.52 days. Maximum data at begin was 42 days and sank to 34 days after treatment finish and to 27 days in the following 3 months.
The results make clear that, between the random check before the beginning of treatment (measurement 1) and those after end of the therapy (measurement 2) a statistically significant difference (p =.022) exists, also between measurement 1 and measurement 3 (3 months ago). There is no significant accidental development. Indeed, the values have not changed between measurement 2 and measurement 3 any more significantly (p =.097).

Question 5

During the last 3 month before treatment started, at an average of 3.73 days, patients could not take part in family, social or leisure activities because of their headache. The average data sank after finishing therapy to 2.3 days and in further succession to 1.43 days. The comparison of sample average data due to t-test shows that, in data before treatment started (measurement 1) and data after finishing therapy (measurement 2) there was a significant difference (p=.039). Likewise, in measurement 1 and data 3 months after finishing treatment (p=.000). However, here too, data between measurement 2 and 3 has not changed statistically significantly (p=.184).

Sum question 1 - 5

The average of the sum was at 20.88 days before starting the therapy and was cut in half to 10.04 days after finishing treatment. Within the following 3 months it reduced again to 7,88 days. Even the maximum data in between the first two measurements reduced considerably from 78 to 24 days. In between measurement 2 and 3 it sank only from about 2 days to 22 days. The comparison (of the middle values) resulted in, at least between 2 out of 3 samples, a statistically high significant difference (p=.000), a coincidence is excluded.

Question A

Before starting treatment patients were suffering from headache at an average of 23.63 days within 3 months. The maximum was 70 days. Within the course of period

of examination, average data reduced to 15.27 days after finishing therapy and to 10.83 within the 3 month following.
The comparison (of the middle value) resulted in, at least between 2 of 3 samples a statistically high significant difference existed (p=.000), a coincidence is excluded.

Question B - migraine

Patients were asked to evaluate their migraine headache in a spectrum from 1 – 10. Before starting the treatment the average was at 8.43, after finishing therapy at 7.70 and in the 3 months afterwards at 7.67. The scattering of data is relatively small (see at standard deviation).
The comparison of average data (based on T-Test results between the data of measuring) before starting treatment and after finishing therapy, as well as, in the three months afterwards in each case a significantly high difference exists (p=.000). In between the appointment 2 and 3, the sample has not changed significantly (p=.813).

Question B – headache

Patients additionally were asked to evaluate their headache in a spectrum from 1 – 10. The average before starting the treatment was at 5.08, after finishing therapy at 4.20 and in the three following months at 3.84.
The calculation shows that between all of three samples there are partially high significant differences, due to this, there was no coincidence in the course of period of examination.

Sum question 1 – 5 in degree according to MIDAS-questionnaire

The values from the question 1-5 were added as described in three measurements and, from it, three new variables were provided. Based on the parameters of MIDAS-questionnaire these parameters were concerted in three new variables with the unity in degree:
0-5 points – few or no interference – degree I
6-10 points – low interference – degree II
11-20 points – moderate interference – degree III
21 + - heavy interference – degree VI

Frequencies

The frequency distribution shows that before starting of treatment the degree III with 40%was strongest represented, followed by degree IV with 26.7%. After end of the therapy the degree IV appeared only with 6.7% of the patients, also the frequency of degree III moved in favour of the lower degrees.

The other investigation (with the help of Wilcoxon-test) shows that between all of three random checks statistically highly significant differences exist (p= .000 or. p= .007), there has been a significant improvement in the course of the time

13) Summary

The rewarding outcome of this study – documented in chapter 12 Discussion – was the considerable improvement of the conditions of the patients in their own opinions .

The results of my study agree to a high percentage with data from current scientific literature:

1) In my study, 100 % of migraine patients documented a loss of power during the attack.

- The International Headache Society (HIS**)** defines migraine without aura as a recurring headache illness. Increased pain caused by physical routine activities is one of the diagnostic criteria for migraine.
- Diener (2002) says that after ¼ - 2 hours migraine headache develops its maximum strength and lasts for 4 – 72 hours. It is typical for somatic exposure to aggravate the pain. Often the last phase of headache is sleep.
- Lipton (2007) confirms that migraineurs experience high levels of pain and substantial disability. Therefore less then 10% of migraineurs report that they are able to work or function normally during their headaches.

2) In my study, 93 % of patients said that stress is a trigger factor for migraine.

- Nash (2006) explains that life stress is a psychosocial factor that is generally acknowledged to be a central contributor to primary headache.
- “Stress is often cited as one of the most frequent aggravating factors in headache” (Kaynak, 2004; Rasmussen, 1993; Spierings, 2001).

3) 90 % of patients taking part in my study had problems with their cervical spine.

- Strackharn (2003) deduces the onset of migraine from the following anatomical context and he agrees in this with Bogduk (1995):The trigeminocervical nucleus is the region that receives afferents from the trigeminal nerve and from the upper three cervical spinal nerves

4) 46.7% of my migraine patients have a scoliosis

- Strackharn (2003) emphasises the central significance of the upper thoracic spine from up to the 5th thoracic vertebra to the ganglion cervical superius. He characterises congenital or acquired false position of the spine or the ribs in the upper thoracic spine as an accelerator for migraine attacks. Today's theories add to these results.

5) Astonishingly 43 % of my patients suffered from depression.

- Hamel (2007) reports that, similar to migraine, depression is also considered to be a disorder of low brain serotonergic activity, and epidemiological studies have reported comorbidity of migraine with depression

6) The prevailing majority of the patients in my study had, in addition to migraine, tension-type headache or cervicogenic headache where the structural treatment approach also showed good results. The percentage lies well over that of Keidel, however, it confirms the possibility of a combination of different types of headache.

- Keidel (2007) maintains that one can often observe a combination of types of headaches. The person concerned suffers from migraine as well as from tension-type headache. He adds that having a migraine does not exclude one from having a cervicogenic headache, too. His opinion is that a cervicogenic headache is combined with migraine or tension-type headache in up to 15% of cases-

This study and the consequentially gained results approve that there is a plurality of factors an Osteopath should incorporate. It is not enough to filter out medical factors; one should involve and include the individual surrounding influences of each person into treatment and mentoring. It is my belief that, only by exerting an influence on as many factors as possible, a clear and persistent improvement of the set of problems

related to migraine can be achieved. Only then can optimal success and an improvement of life quality of the migraine patient be possible.

Despite the positive developments of my study, some aspects may be certainly be improved. The study could be even more expressive if:

- The number of patients was bigger;
- The ratio of women to men would be 3:1:
- Younger patients would take part:
- Before starting the treatment, an x-ray of all participants would be available

Addition:

Dieser Fragebogen kann Ihnen und Ihrem Arzt helfen,
die Behandlung Ihrer Kopfschmerzen zu verbessern.

Leiden Sie an Kopfschmerzen (Migräne)?

Midas Fragebogen

Anleitung: Bitte beantworten Sie die folgenden Fragen über ALLE Kopfschmerzattacken, die Sie in den letzten drei Monaten hatten. Füllen Sie das Kästchen neben jeder Frage mit der entsprechenden Zahl aus. Schreiben Sie 0, wenn die Antwort negativ ist.

1.	An wie vielen Tagen in den letzten drei Monaten sind Sie wegen Kopfschmerzen nicht zur Arbeit gegangen?	Tage
2.	An wie vielen Tagen war in den letzten drei Monaten Ihre Leistungsfähigkeit am Arbeitsplatz oder in der Schule um die Hälfte oder mehr eingeschränkt? (Zählen Sie die Tage, die Sie bei Frage 1 angaben, NICHT dazu)	Tage
3.	An wie vielen Tagen in den letzten drei Monaten konnten Sie wegen Ihrer Kopfschmerzen keine Hausarbeit verrichten?	Tage
4.	An wie vielen Tagen in den letzten drei Monaten war Ihre Leistungsfähigkeit im Haushalt um die Hälfte oder mehr eingeschränkt? (Zählen Sie die Tage, die Sie bei Frage 3 angaben, NICHT dazu)	Tage
5.	An wie vielen Tagen in den letzten drei Monaten haben Sie an familiären, sozialen oder Freizeitaktivitäten wegen Ihrer Kopfschmerzen nicht teilnehmen können?	Tage

Ergebnis: Tage

A	An wie vielen Tagen hatten Sie in den letzten drei Monaten Kopfschmerzen? (Wenn die Kopfschmerzen länger als einen Tag angehalten haben, zählen Sie jeden Tag)	Tage
B	Wie stark waren diese Kopfschmerzen? Bitte geben Sie die Schmerzintensität auf einer Skala von 0 – 10 an. (0 = keine Schmerzen, 10 = unerträgliche Schmerzen)	

Bitte zählen Sie die Tage der Fragen 1 – 5 zusammen, sobald Sie den Fragebogen vollständig ausgefüllt haben. (Die Fragen A und B bitte NICHT dazuzählen)

Auswertung des MIDAS Fragebogens:

Grad	Definition	Punkte
I	Wenig oder keine Beeinträchtigung	0 – 5
II	Geringe Beeinträchtigung	6 – 10
III	Mäßige Beeinträchtigung	11 – 20
IV	Schwere Beeinträchtigung	21+

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