OSTEOPATHY

IN

IDIOPATHIC PARKINSON SYNDROME

Diploma Thesis

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by

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INTRODUCTION

„Healthy action of brain with its magnetic and electric forces to the vital parts which sustain life, memory and reason depend directly and wholly upon limited freedom of the circulatory system of nerves, blood and cerebral fluid.“ [5]

More than 100 years have passed, since Dr. A.T. Still, the founder of osteopathy wrote down these thoughts in his work „Philosophy of Osteopathy“ and this sentence has lost nothing of its importance and validity in the field of osteopathy until today.

From this point of view, the amplitude of the subjects in the field of neurology-osteopathy which have not yet been studied systematically seems to be more surprising. This is also especially true for Parkinsonism, which is known as one of the most frequently occurring and most important neurological diseases. About four million people are affected worldwide.

A conventional literature research in the Internet (Google, advanced pub-med) with the search terms “Parkinson - osteopathic manipulative treatment“ ended with the result of only two titles referring to this branch of medicine.

In one of them the author describes the positive influence of a standardised osteopathic treatment on gait.[18] The second piece of work studies various osteopathic types of skull lesions of patients with parkinsonsyndrome.[15]

In the course of my internship in a hospital focusing on the treatment of neurological diseases I observed various positive effects, e.g. improved posture, smoother movement, reduced secondary pains, but also an improvement of the general well-being of patients suffering from Parkinson’s disease. These effects which result from an individual osteopathic approach focusing on the individual have motivated me to examine this subject more closely in the frame of a clinical study.

This study deals primarily with the question whether osteopathic treatment leads to an improvement of the motor skills of concerned patients. For methodical reasons, this study is restricted exclusively to the motor aspects of this extremely complex illness.

CLINICAL PART

CLINICAL DESCRIPTION OF PARKINSON'S DISEASE

Definition: In 1817 James Parkinson described the disease as „shaking-palsy".[13] This is a hypokinetic-hypertonic syndrome with the trias: tremor, muscular rigidity, akinesia. From the pathogenetic point of view, the illness can be described as a progressing degeneration of nigrostriatal dopaminergic neurons. In aetiology, the idiopathic Morbus Parkinson can be distinguished from symptomatic forms of the illness.

Epidemiology: Parkinsonism ranks among the most frequently occurring neurological diseases. According to the majority of examinations there is a prevalence (number of the persons afflicted by the disease in relation to the whole population) of 100-200 / 100000. The incidence (annual number of new cases) amounts to approximately 20 / 100000. If we consider similar epidemiological data, the number of patients concerned by Parkinson's disease in Austria is estimated to reach up to 15000, to which 1500 are supposed to add every year.

If we observe the age of the patients at the time of the onset of the illness, we can see that the number of patients reaches its apex between the age of 50 and 70 and that it is relatively widespread between the age of 30 and 90. A comparison of the number of male patients and that of female patients shows a slight majority of male patients.

The frequency of the illness has remained almost constant in recent years, nevertheless an increase of patients is to be expected within the next decades due to the demographic development.

Symptomatology: In general, the clinical symptoms of Parkinson's disease are subdivided into the four cardinal symptoms (tremor, rigor, akinesia, postural instability) and several accompanying symptoms. Although this subdivision is partly relatively arbitrary and the borders between the cardinal symptoms can sometimes not be drawn clearly, the subdivision has worked out well in the clinical practice. In

the early stages of the illness the cardinal symptoms occur almost always on one side. (Hemiparkinsonism).

Tremor: An involuntary rest tremor at a fixed rate of 4-5,5/s is regarded as the best-known and most obvious cardinal symptom. It affects primarily the hands and is then described as pill-rolling tremor. Under the influence of emotional stimuli or stress tremor intensifies significantly and when the patient initiates movements, it may disappear completely or it may also simply become weaker. In the latter case it may impair the concerned patient severely in the form of intentional and postural tremor. Tremor may also affect the arms and legs, axial structures, the head, the jaw and the tongue.

Rigor: Rigor is caused by an increase of the muscular tone. The augmented muscular stiffness becomes apparent as raised, tough and waxy resistance occurring permanently and independently from the speed of movements. Slight fluctuations in tone are described as the so-called cog-wheel rigidity. Rigor intensifies under the influence of emotional pressure and may extend to remote even contralateral muscular groups. Axial rigor leads to difficult turning movements (e.g. changes of position during sleep), neck rigour may make it impossible for the patient to lay his head down on the pillow. In cases of hemiparkinson the asymmetric increase of muscular tone is associated with the development of scoliosis. Rigor causes muscular pain primarily in the shouldergirdle and the neck-region.

Akinesia: For the patient akinesia appears mostly as the worst impairment. On the one hand, it becomes apparent as a reduction or loss of automatic movements (e.g. coordinated reflexes). On the other hand, voluntary movements are slowlier (Bradykinesia) and reduced in amplitude (Hypokinesia). For the patient it seems more difficult to initiate movement (start hesitation). Complex movements are much more severely impeded than simpler movements. Simultaneous movements, repetitive sequential patterns of movement and rhythmic-expressive movements are limited to an extremely high degree. The face loses spontaneous expression (masked facies, Hypomimia).

Based upon a diversified predominance of the cardinal symptoms we are familiar with a rigor-akinesia-dominance type, an equivalence type and a tremor dominance type.

Postural instablility: Rigor and akinesia lead to a posture which is typical of the illness, to disturbances of posture and gait combined with a tendency to stumble but also to impairments of the fine motor skills.

In upright position the patient's trunk is bended ahead, his knees and ellbows are slighty flexed, his ellbows are pronated. The back is kyphotic, the neck is tilted ahead and the head is translated in an anterior position.

For the patient it is difficult to initiate gait. He moves slowlier, his steps are short and narrow and the gait is shuffling. His arms swing little or not at all. His turning movements are staccato and stiff in character. In later stages of the sickness there exists an increasing tendency to walk only on the forefoot. Since the steps become shorter during gait, which leads to propulsion, patients are no longer able to stop the movement abruptly and tilt forwards. („The feet remain at the same place, the upper extremities move straightforward"). In some rare cases these motor dysfunctions may also lead to retropulsion ot lateropulsion.

The extent of this disturbance is subject to diverse situative and psychologic changes. Before narrowings and in front of obstacles blockades of gait may occur rapidly which make it impossible for the patient to continue his movement for seconds or even for minutes ("freezing"). The mechanism may also be caused by virtual obstacles. (e.g. markings on the floor). On the contrary, patients may surmount their starting difficulty if they have to get over an obstacle (propably also only an imagative one). They may also keep their movement smooth if they or some other person gives them a rhythmic command (counting "left-right"). By means of shock a so-called "paradoxe crisis" may be initiated , in the course of which a severe akinesia may be absent for a short period of time. Such fluctuations may lead to doubts about the organic character of the illness among the relatives and the medical staff. The freezing-phenomenon can also be observed in patients which are not treated with medicines and must not be mixed up with the response fluctuations of medications for Parkinson's disease (on-off phenomenon).

The disturbed fine motility becomes significantly obvious in the handwriting and is described as micrographia. In many cases, the first letters of a sentence or a line are

still legible but then they are rapidly becoming smaller and smaller, the writing becomes crude. The disturbed fine motility has also a negative influence on the patient's everyday life as far as activities like dressing and undressing, combing one's hair, razing or buttoning one's jacket and the use of cutlery are concerned, even in this connection the disorder can predominantly be noticed when complex or simultaneous movements are carried out.

The **accompanying symptoms** of Parkinson's disease comprise mainly vegetative and mental disturbances.

Vegetative disturbances: Many patients affected by Parkinsonism complain about a persistent salivation. It is propably caused by reduced involuntary swallowing and the slight tendency to keep one's mouth opened. Then salivation just apparently seems to be increased.

Concerning skin disorders, the seborrhoe (excessive discharge of sebum) is the most significant one leading to the typical seborrhea oleosa. As far as disturbances of the digestive system are concerned, obstipation is certainly the most important one. Dysfunctions of motility also affect the upper region of the digestive organs and occur in the form of heart-burn or gastroesophogal reflux. Disturbances of micturition, sexual functions and reduced libido occur frequently but they are rarely reported spontaneously. Speaking disturbances with a monotone, aphonic voice are caused by a variety of factors, the same is true for the impaired swallowing, which does not occur so often. 80 % of all patients say that they suffer from sleeping disturbances. Generally, these comprise intitial insomnia, reduced amount of sleep, sleep maintenance insomnia and daytime hypersomnolence. In rare cases, episodic hypersomnolence during the day ("sleep-attacks") is also reported by patients.

Mental disorders: When he defined the signs of the illness, James Parkinson excluded that there exist any psychological disorders which can be associated with the disease. He wrote: ".... the senses and intellect being uninjured". Today this view is largely refuted. Nevertheless, the extent and the evaluation of these disturbances is viewed in many different ways. Just to mention an example, the frequence of depressions afflicting parkinsonian patients is said to range between 20 - 90 % and this discrepancy results, among other reasons, from the fact that the depression often

does not reach a severe stage. On the one hand, the disturbance is characterised as endogenous and on the other hand it is considered to be reactive to the restrictions which are necessitated by the disease (reduced mobility and poor possibilities of communication, which are often associated with stigmatization and social reclusion). Depression is different to Bradyphrenia, which becomes apparent in a form which is similar to motor impairment**,** namely in the form of slowness of thinking processes and a lack of flexibility and resolution. Cognitive disturbances which can be investigated in psychological tests affect predominantly elderly patients and in many cases they can not certainly be connected with the main illness.

There are also conflicts among the physicians concerning the question whether patients affected by Parkinsonism suffer from a premorbid personality structure.

Our attention must be drawn to the aspect, that according to their own reports parkinsonian patients suffer from a physical or a mental trauma before or at the onset of the sickness. The importance of these findings is difficult to interpret and they can only in the foreground be considered as the „necessary reason of the illness“ from the patient’s point of view.

At the end of the clinical description I want to point out once more that there exists hardly any other neurological disease with as much different connections between the motor system, the vegetative and the psychogenic system.

STRUCTURE AND FUNCTIONS OF THE BASAL GANGLIA, PATHOPHYSIOLOGY OF PARKNSON SYNDROME

Parkinsonism belongs to the so-called **extrapyramidal diseases** of the motor system caused by a dysfunction of the basal ganglia.

In anatomy, the basal ganglia are those parts of the gray substance (nucleus) of the cerebrum which are situated deeply in the hemispheres unlike the cerebral cortex. Under a more functional aspect, the term basal ganglia also comprises the nucleus regions of the diencephalon and the mesencephalon.

Together with the cortex and the thalamus, the basal ganglia form part of a circuitry which is primarily responsible for motor activities. It was not until recently that evidence material indicated that limbic and prefrontal structures are also involved into this system (which corresponds well with the complex clinical disorders of the disease affecting the basal ganglia).

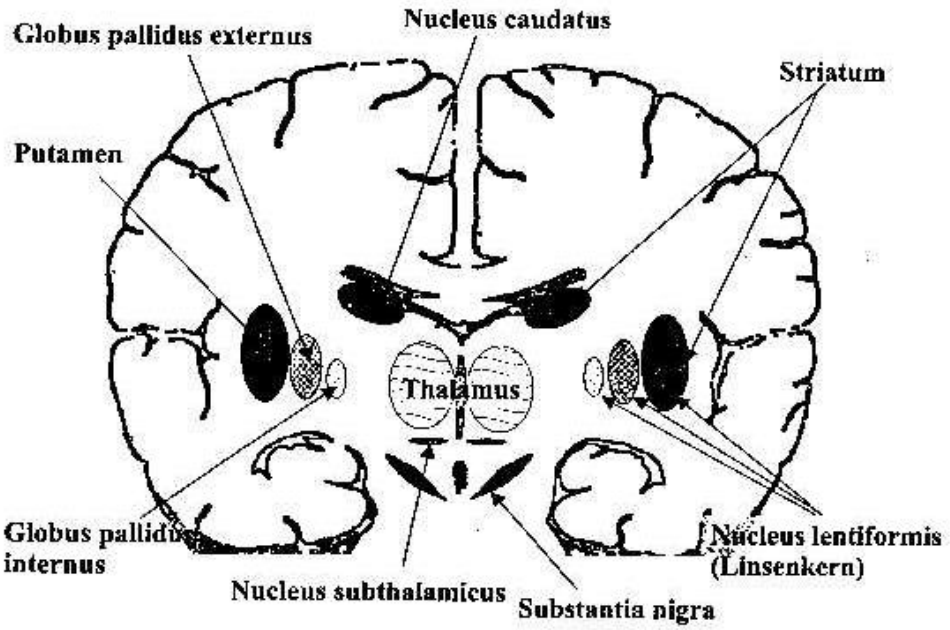
The role which the basal ganglia play for the motor system is not completely clear. The following points shall sum up our present knowledge:

In the **basal ganglia** some type of parallel information processing takes place, which is responsible for the preparation as well as for the performance of motor activities. Movements may be planned in sequential patterns or inhibited in a process which is similar to a catalytic or filter function. This includes also the performance of necessary coordinated movements and postural modifications.

Furthermore, the basal ganglia fulfill a remarkable task when self-induced movements are planned and initiated. In this connection, time-related aspects like premotor activities, expectation or synchronisation of muscular activity also have some influence. The basal ganglia are involved in the acquisition of simple and complex motor skills.

Since they also consist of additional prefrontal and limbic loops they have a crucial impact, especially when feelings are integrated into expressive movements.

Depiction 1 shows a semischematic representation of the position and the several designations of the basal ganglia. Orientation in this nucleus region is aggravated by the fact that anatomical and functional structures are not always equivalent.



Depiction 1: Position and designation of the basal ganglia (taken from: Prosiegl, Klinische Hirnanatomie)

Nucleus caudatus and Putamen: They form a functional unit and act as an input station to the basal ganglia, where the input impulses from the cortex innervate. Anatomically, the two nuclei are separated by the passing “internal capsule“ but they are connected with each other through pontes striae (striae), to which the whole structure owes its designation “striatum“.

Substantia nigra: Dark (=nigra) nucleus region of the brain which is situated in the mesencephalon and consists of two parts functioning in different ways. The pars compacta projects towards the putamen while the pars reticularis projects towards the thalamus and consequently forms part of the output station of the basal ganglia system.

Nucleus subthalamus: Internal nucleus of the basal ganglia loop.

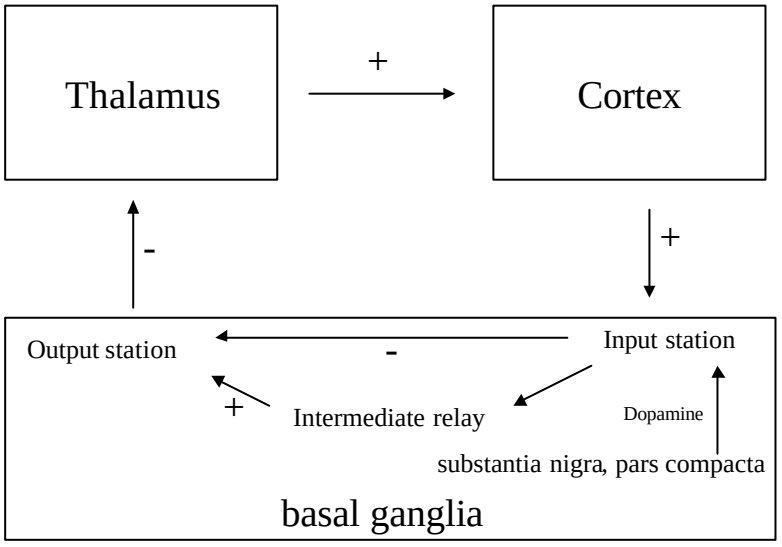
Globus pallidus: A structure situated at the median of the putamen, which is composed of two parts functioning in different ways.

The globus pallidus externus is an internal nucleus of the basal ganglia loop, it receives output impulses from the putamen and sends input impulses to another internal segment (Nucleus subthalamicus) and directly to the output station of the basal ganglia circuitry (Substantia nigra, Pars reticularis and Globus pallidus internus).

The Globus pallidus internus forms part of the output station of the basal ganglia circuitry.

Putamen and Globus pallidus, which form a morphological (but not functional) unit, are together also designated as **Nucleus lentiformis**, which is a consequence of their anatomical proximity.

Depiction. 2 is a very simple representation which gives us a survey of the most important functional connections between the basal ganglia.



Depiction 2: Functional connection between the basal ganglia (modified according to Prosiegl, Klinische Hirnanatomie)

The Striatum forms the **input station** of the basal ganglia. The putamen receives the impulses of the motor cortex in somatotopic order (premotor cortex, supplementary motor area, frontal vision field), the Nucleus caudatus receives them from the

premotor and limbic cortex and from the somatic sensory area of the sensory cortex and the cortical association area.

In the region of the basal ganglia system there is a **"direct pathway**" between the input station and the output station (Globus pallidus internus, Pars reticularis of the substantia nigra), which inhibits the output station. A second, **"indirect pathway"** leads from the input station over interconnected relay nuclei (which comprise the Nucleus subthalamicus and the Globus pallidus externus) to the **output station** where it exerts the influence and augments the activity of the output station.

The respective degree of activity of the output station results immediately from the present „relation between the forces" of these two contending pathways which operate at the same time.

The output station conducts inhibitive efferent impulses to the thalamus, which projects excitatory impulses towards the Cortex.

So, the raised activity of the output station acts like a brake on cortical activity. Consequently, the "direct pathway" is known to have an increasing impact on motor activity, the „indirect pathway" is known to inhibit motor activity.

The influence of **dopaminergic nigrostriatal neurons**, the degeneration of which leads to Parkinson's disease, has a twofold influence as far as this function is concerned: Dopamine has an activating impact on the direct pathway via D1-receptors and inhibits the indirect pathway working through D2-receptors. So, dopamine has a twofold effect on the sequences of motor activities.

As the degeneration of dopaminergic neurons in the substantia nigra plays an important role in pathophysiology of Parkinsonism, I want to describe the metabolism of dopamine and the dopaminergic synapse in every detail:

Dopamine belongs to the catecholamines of the brain where it accumulates primarily in the basal ganglia. About 80 % of the cerebral dopamine can usually be found in the pars compacta of the substantia nigra and in the striatum.

The amino acids phenylalanin and tyrosine enter the neurons from the blood stream where they are hydroxylated into dopa and are then decarboxylated into dopamine. It

is remarkable that dopamine is not able to cross the blood-brain-barrier. Nevertheless, in its preliminary stage dopa is able to cross this barrier. (The medicative treatment using a substitute is based upon this fact.)

In the nerve endings dopamine is stored in vesicles and is released into the synaptic gap due to an activity potential of the neuron. On the one hand, it has an impact on the postsynaptic receptors and on the other hand, it influences the presynaptic autoreceptors for feedback-purposes.

The released dopamine is partly metabolised by monoaminoxydase (MAO) and the katechole-0-methyle transferase (COMT),it is then deactivated.A part of it will again be absorbed by the presynaptic nerve ending without metabolisation.

There are several types of cerebral dopamine receptors, which can be subdivided into the D1-receptors and the D2-receptors. There are at least five different subtypes which have already been identified.

Parkinson’s disease does not only consist of a disturbance of the dopamine metabolism because other transmitter systems are also impaired, even if these impairments are much less significant.

AETIOLOGY, THERAPY AND PROGNOSIS

Aetiology

Even in healthy persons dopaminergic neurons degenerate with increasing age. The dopamine content decreases until to 13 % every 10 years. [14] Parkinson's disease leads to the loss of approximately all of these cells. A clinical symptomatology can not be realised before a percentage between 70 and 80 % of the dopaminergic neurons has degenerated.

The loss of dopaminergic neurons is either the result of damage to presynaptic dopaminergic neurons of the Substantia nigra/Pars compacta or the result of a lesion of postsynaptic neurons in the Putamen. In both cases the effects of dopamine are reduced.

Until today the factors which lead to the degeneration of the nigrostriatal system are unknown. In these cases we speak of **idiopathic Parkinsonism** and this illness causes the prevailing majority of the cases.

On the contrary, there exist **symptomatic forms**, the reasons of which shall be treated in close detail. Our certain knowledge that these symptomatic forms of the sickness exist, is not only crucial for the medical fields of differential diagnosis and differential therapy, the medical profession also hopes to find out the factors which underlie the idiopathic form of the disease.

The **postencephalic type of Parkinsonism** occurred in the course of or 20 years after symtoms indicated a case of Encephalitis lethargica, described by Economo. Encephalitis lethargica was a pandemic disease between 1916 and 1926 and at that time the most widespread reason of Parkinsonism. Since 1967 no new cases were entered into the record.

There is still no answer to the question whether the **vascular** or arteriosclerotic type of Parkinson's disease really exists, but it occurs by far more rarely than it is occasionally assumed.

The influence of the **genetic factors** has been the central issue of discussions but they play a subordinated role.

A **traumatic type** of Parkinson's disease is very seldom and may occur after multiple traumatisms, i.e. as it is typical for boxers.

Types of Parkinsonism, which are caused by **medications** or iatrogenic types have been observed especially after treatment with neuroleptics and in rare cases in which lithium and calciumantagonists were prescribed.

A **toxic type of Parkinsonism** is reported to have occurred after intoxications with mangane, mercury, carbon monoxide, hydrogene sulphide, cyanide, methanol, benzole and various solvents and insecticides. In many cases of these intoxications the extrapyramidal motor dysfunction is accompanied by various other symptoms.

Much attention was drawn on the toxic form of the sickness, since MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyrine) was discovered as the factor causing a clinically relatively „evident and pure" type of Parkinsonism. MPTP is a kindred form of Meperidine (MPPP), which is used as an addictive narcotic drug and may be produced as a by-product when merperidin is synthesized. From the pathologic-anatomic point of view, an almost selective degeneration of the basal ganglia in the Pars compacta of the Substantia nigra can be observed. In medical research, the substance has become important, because it causes the same changes in humans and in a number of primates - especially in monkeys - and because Parkinson's disease can now be induced in experiments.

Since it was discovered that MTPT might lead to Parkinsonism, chronic environmentally-caused influences have increasingly become the central question of discussions.

Therapy

The treatment of Parkinson's disease with medicines aims primarily at the disappearance of the dysfunctions at the transmitter stage.

The substitution-therapy with **L-Dopa** (Levodopa), which is then decarboxylated in the brain and converted into dopamine, is the usual therapy. L-Dopa is always

administered in combination with the peripherically acting Dopa-decarboxylase-inhibitors benserazide and carbidopa. So, the conversion of L-Dopa into dopamine in the body (outside the brain) is prevented, which is a protection against systemic side-effects of Dopamine. The Dopa-Decarboxylase-inhibitors are unable to cross the blood-brain-barrier and they remain ineffective in the central nervous system.

Similarly, **COMT-inhibitors** (Entacapone) prevent early L-Dopa deficiency in the blood. They are only administered together with L-Dopa in order to achieve more equally-balanced effects of Dopamine in the brain.

Dopamine-receptor antagonists (e.g. cabergoline, lisuride, pergolide, pramipexole, ropinirole) act primarily on the D2-receptors and form part of the standard therapy even at the first stage of the illness. There are primarily different half-lives and differing binding properties to subtypes of dopamine-receptors.

The influence of the **NMDA-antagonist** amantadine works via glutamate. It is used in a combined therapy, but primarily as a short-term treatment of akinetic crises.

The **MAO-B-inhibitor** selegiline becomes effective by means of a reduction of the dopamine deficiency. Its use is also very common because of an supposed neuroprotective effect.

Anticholinergic medicines are only used in exceptional cases and then mainly to treat tremor, as they are known to cause a variety of side-effects.

The crucial question concerning the administration of medications for Parkinsonism is to use the appropriate dose and combination of the several substances at the different stages of the illness in each of the individual cases. In this way it shall become possible that the patient's motor skills function properly in order to prevent the frightened response fluctuations and late complications we know from long-term therapy (e.g. dyskinesias).

Prognosis

Idiopathic parkinsonism is a **chronic progressive disease**. How far the illness progresses within a certain period of time, varies strongly in the individual cases. In patients which have achieved a high age before the illness afflicts them, the disease

propably progresses faster. Especially at the time of the onset of the disease it is hardly possible to determine clearly how far the disease is expected to progress in single cases.

Before the times when Parkinsonism was treated with medicines, the average duration of the sickness until death amounted to 9 years. [9] Nevertheless, we do have observations of patients of that time, in which no significant or only a slightly significant progression of the illness had been observed for many years.

Today, the life expectancy of parkinsonian patients has almost normalized and it is propable that the progression of the impairment was reduced.

The sickness may be deadly if secondary complications such as e.g. sequelae after falling down or immobility sequelae (pneumonia, thromboembolia, decubitus ulcer) occur.

OSTEOPATHIC APPROACH

Introduction

Parkinsonism belongs to the neurodegenerative diseases. The reason of the disease is unknown as well as a causative therapy. The medical therapy focuses on the medicative treatment of the symptoms aiming to relieve the disorder at the neurotransmitter stage.

If we consider the pathophysiologic aspects of the disease, metabolic changes which lead to an increased production of toxic metabolic end-products causing cell damage play an important role.

In aetiology of secondary Parkinsonism there are also indications that metabolic and toxic influences as well as post-infectious changes may cause the same disturbance.

In this connection a possible approach to improve the clinical situation by means of osteopathic methods arises.

Osteopathy aims to ameliorate the exchange of fluids between all bodily tissues at the intracellular and at the extracellular stage.

Nutrition, transport of metabolic end-products, energy storage, carrier functions, a.s.o., everything depends on an intact and well-balanced circulation of fluids so that the whole organism may keep up its homeostatic integrity. If blood circulates freely and unrestraint and the bodily tissues and nerves are sufficiently supplied with blood, the tissues are able to work as good as possible. [16]

From **literature** we know only few about osteopathy and Parkinsonism.

In a conventional literature research I found the following two articles::

1. A retrospective study of cranial strain patterns in patients with idiopathic Parkinson´s disease, by Sonia Rivera-Martinez.[15]

The objective of this study was to observe cranial lesion patterns of parkinsonian patients. 30 patients suffering from idiopathic Parkinsonism were tested. Several staff members tried to find out if there exists a higher amount of certain strain patterns in parkinsonian patients in comparison with a control group.

Patients afflicted by Parkinson's disease showed a significantly higher amount of bilateral atlantooccipital and occipitomastoidal compressions. The study does not treat the therapeutic aspects.

2.Standard osteopathic manipulative treatment acutely improves gait performance in Patients with Parkinson´s disease, by Michael R. Wells.[18]

In this study the quantitative effect of an osteopathic treatment on gait was evaluated. 20 parkinsonian patients and 8 probands took part in the study. The effect of the osteopathic treatment was measured among 10 parkinsonian patients, the other 2 groups (10 parkinsonian patients and 8 probands) were control groups.

The study analyses gait on a 40-feet-long distance by means of a computerized two-dimensional saggital gait analyse system. The gait parameters were: length of the steps, rhythm, shoulder speed, speed of the armswing and of the several large joints of the lower extremity. Every patient was treated for 30 minutes and the treatment sequences, which comprised 14 different techniques and technique applications, were the same for each patient. One single osteopathic treatment of the parkinsonian patients resulted in a significant augmentation of the gait parameters in comparison

with the data measured before the treatment. The Parkinonism control group showed the tendency of a slight deterioration.

So, in this study, in which a structural approach was chosen, an acute improvement of gait is described. The said improvement was achieved by means of a fixed treatment plan which was the same for each patient.

Nevertheless, in the field of osteopathy there are various possibilities of approach based on different focuses: structural, cranio-sacral and visceral.

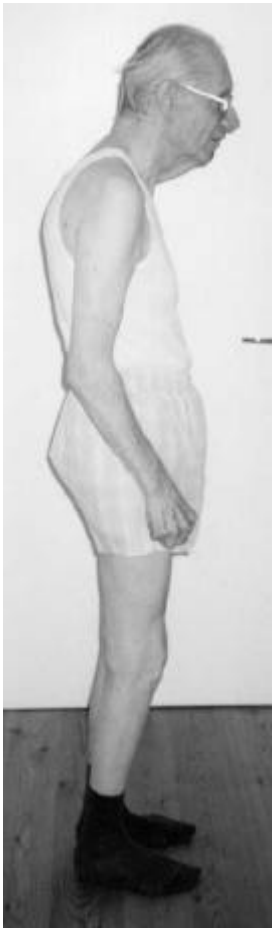
There exist no examinations based on cranio-sacral or visceral focusses.

Osteopathic approach

Based on the results in the literature, in my approach structural lesions also play an important role, but there is the difference that the treatments correspond to patient-related lesion chains which also involve cranial and visceral aspects.

Each patients shows an individual lesion pattern. To demonstrate the kind and the extent of the structural lesions of my patients, I attached a table in the appendix (Table 3), in which the most apparent lesions of every patient are roughly structured and enlisted. From these differences alone it becomes obvious that a standardised treatment does not correspond to the individuality of each patient and to the individual osteopathic approach.

The disturbance of the patient's motor system nevertheless causes a postural pattern so typical for the disease, that the diagnosis can easily be expected (Depict. 3).



Depiction 3: Postural pattern in parkinsonian patients (with friendly permission of the patient)

One of the clear signs of this postural pattern is an unphysiological position of the thorax and the cervical spine. The findings do not only confirm the restricted movement of the thorax and the collar bone, there exist also massive changes of adjacent structures. Among others, they comprise the arterial vessels running to the brain and the veinous vessels. So, the system of the cerebrospinal fluids is indirectly influenced. From the point of view of osteopathy, restrictions of these structures may affect the metabolism and the function of the brain if the circulation of fluids is unbalanced.

In the following chapter the anatomically relevant regions shall be described more closely, taking into account the tight interactions between structural, visceral and craniosacral structures.

Osteopathic considerations concerning the anatomical structures

The thorax and the respiratory muscles are the most important driving force for the vital gas exchange.

The diaphragm separates the thoracic cavity from the abdominal cavity. In flaccid condition which means during the exhalation phase of respiration, the diaphragm bulges towards the breast region. When it contracts during inspiration the curvature flattens. The resulting underpressure induces the two pulmonary wings to extend. In addition it could be proved that this negative pressure fosters the reflux of the veinous blood stream, thus supporting the heart in its function to assure blood circulation. [6]

Chronic diseases with a raised motor activity restriction of the bony thorax cause mechanic disturbances which influence the pulmonary capacity directly and thus restrict the physiological processes in the thorax region.

To demonstrate the numerous connections as well as the approaches of the treatment between the diaphragm and the bony thorax I want to describe the anatomy of the diaphragm exactly:

The diaphragm is made up of a central tendon, the Zentrum tedineum, and a muscular portion. Depending on the origin of the muscular bundles the latter can be divided into a Pars sternalis, a Pars costalis and a Pars lumbalis. Besides from the aorta, different anatomical structures such as V. cava inferior, Ductus thoracicus, Oesophagus, Truncus symphaticus and N. vagus pass the diaphragm.

Later, only those parts of the diaphragm are described in detail which can be closely connected with the Hiatus aorticus, the point where the aorta passes the diaphragm.

The Pars lumbalis of the muscular portion consists of the crus mediale and laterale on the right and on the left side respectively.

The aorta passes the diaphragm between the sinewy fortified Cura medialia at the projection height TH 12.

The Crus mediale dextrum consists of three muscular bundles. The largest one has its origin in the anterior region of the first four lumbar vertebrae and the disci and travels directly to the Centrum tedineum.

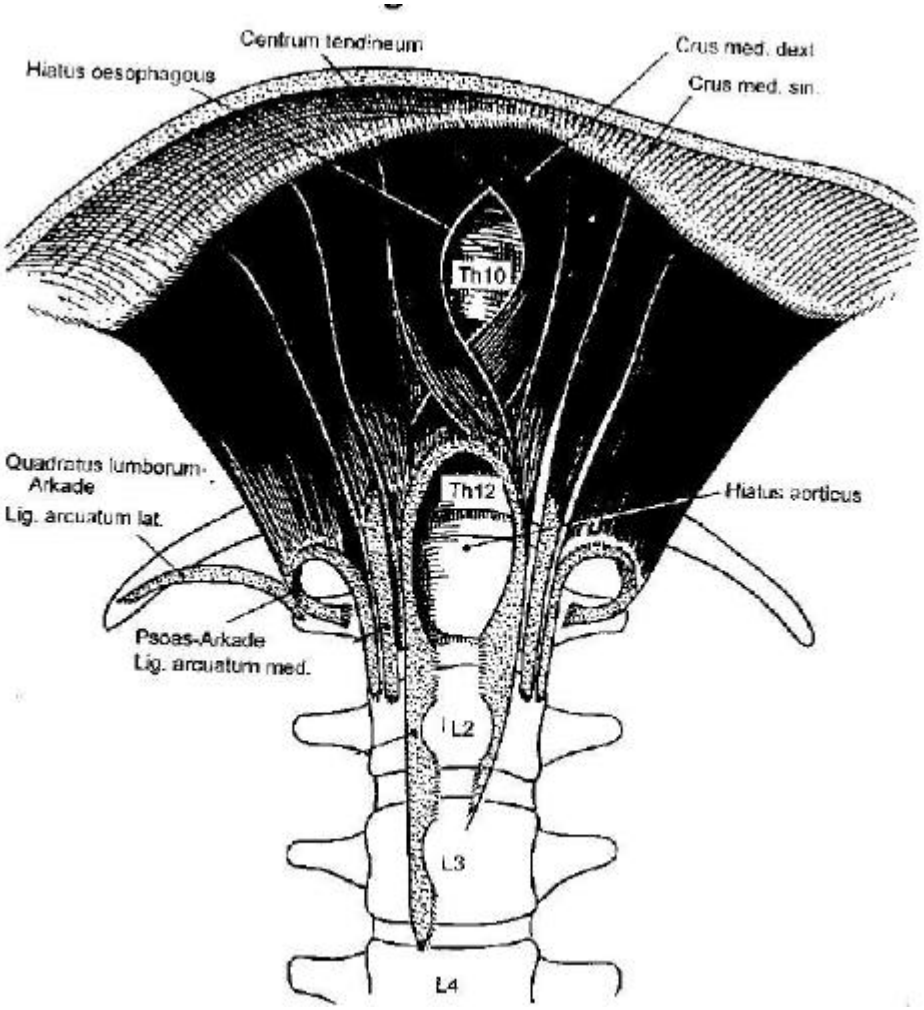
The other two muscular bundles originate in the sinewy peripheric regions of the Hiatus aorticus, cross each other and form the right and the left border of the Hiatus oesophageus.

The Crus mediale sinister starts directly at the first three lumbar vertebrae and forms the basis of the Treitz' muscle (M. suspensorium duodeni).

The right and left Crus laterale start respectively in the two arcades. On the one hand, in the psoasarcade (Lig.arcuatum medianum), which fortifies the Fascia iliaca and travels from the lateral side of L2 to the Processus costalis of L1, on the other hand, in the Quadratusarcade (Lig.acruatum laterale), which is situated between the apex of the Processus costalis and the apex of the twelfth vertebra. So, there exists a direct anatomic link to the pelvis and large muscular groups, such as the M. psoas and the M. quadratus lumborum.

The arcades are directly connected with the posterior side of the kidneys and the subrenal glands.

The Ductus thoracicus passes the diaphragm at the same point as the aorta. Closely below the diaphragm the Truncus coeliacus starts from the aorta, which is surrounded by the Plexus coeliacus. It receives afferent fibers from the two Nn. splanchnici and from the N. vagus.



Depiction 4: Anatomy of the diaphragm (taken from: Ligner, Skript viscerale Osteopathie)

If there occur restrictions, which inhibit the usual movement of the diaphragm, a remarkable number of closely cooperating systems is impaired, especially the aorta and the Ductus thoracicus, the Vena cava inferior, the kidneys, the sympathetic trunk and the N. vagus.

The optimal extension of the **lungs** depends on the mobility of the pleura besides from the activity of the diaphragm. The negative pressure in the pleura gap keeps up the permanent dilatation of the lungs. The change between negative and positive

pressure makes it possible to balance the myofascial tensions and improve the circulation of the fluids, the cardiac performance, ventilation and perfusion by means of the soaking mechanism.

The thorax can also be described as an emotional region. Here tensions may occur, e.g. in the mediastinum due to psychological problems or stress.

The visceral structures are connected with the bony thorax and via these tendons and fascies the balance can be improved at various stages by osteopathic treatment.

From the point of view of osteopathic treatment, the region of the **upper thorax apertures** is a highly relevant zone due to its close anatomical connections between bony structures, soft tissues and vessel system, because blockades of the first rib, for example, lead to severe disturbances of fluid circulation.

The aortic arc arborizes, forms the Truncus brachiocephalicus on its right side and travels behind the first vertebra where it arborizes again and forms the A. caorotis communis dextra and the A. subclavia dextra. The A. carotis communis sinistra stems from the left side of the Trachea and travels in the cranial direction. The Carotides pass the upper thorax aperture, which is bordered on its side and in the front by the interior end of the two first ribs, the upper end of the Manubrium sterni and from the medial end of the clavicula. The posterior border is formed by the first thoracic vertebra.

The upper thoracic aperture is closely connected with superficial structures. The posterior segment of the first rib is covered by four muscle layers:

M.splenius, M.serratus posterior superior, M.rhomboideus und M.trapezius.

In the lateral sphere those muscles, which are connected with the first or the second vertebra, are imporant: M.levator scapulae, M.scalenus posterior and medius, and the first point of the M. Serratus anterior.

In the anterior sphere, the M. scalenus anterior is situated and the N. phrenicus, which itself starts in the C3-C4 region, reposes on it and travels to the thoraco-abdominal diaphragm.

Together with the M. scalenus medius it forms the posterior Scalenus leak, which is

the point where the A subclavia and the Plexus brachialis pass the diaphragm.

In the region of the anterior Scalenus leak the V. subclavia and the collective lymph nodes of the supraclavicular lymph ways are situated, which conduct the lymphs to the Ductus lymphaticus dexter and further into the Ductus thoracicus.

Other structures which may impair the arterial blood stream in this region are muscles which are linked with the upper thorax aperture: the sternal and the clavicular part of the M.sternocleidomastoideus, the M.subclavius, which connects the first rib with the clavicula and the lower hyoid muscles which are surrounded by the Fascia cervicalis media.

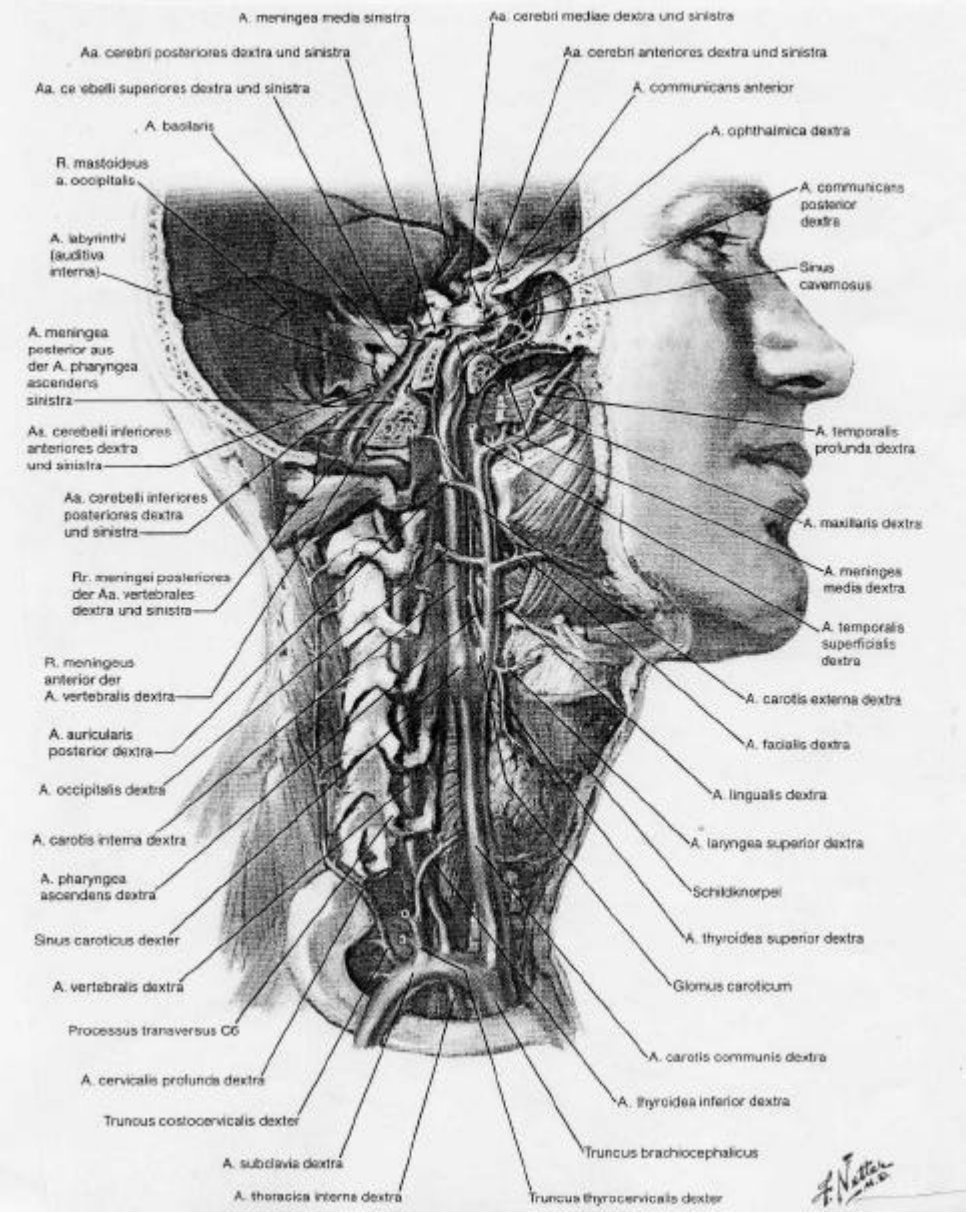
The arterial supply of the cervical spine and the skull

A.carotis communis travels along the neck without separating into branches. A compression against the prominent transverse process of the sixth collar vertebra (Tuberculum caroticum) is possible.

At the height of the fourth collar vertebra it arborizes and forms the A.carotis externa and the A.carotis interna.

A.carotis externa then separates into branches for the neck, the face, the skull and the cranium.

A.carotis interna runs without arborizing to the skull base and supplies the occular cavity, the hypophysis and the brain. The cervical part of the A. carotis intern is covered by the M. stenocleidomastoideus, the parotid gland and the M. digastricus as well as the stylohyoideus.



Depiction 5: Anatomy of the neck (taken from: Netter, Atlas Neuroanatomie)

The A carotis interna passes into the skull via the canalis aorticus of the petrosal bone and runs into the interior skull via the Foramen lacerum at the top of the petrosal bone. The Foramen lacerum is made up of 3 bones: the Os temporale, the Os occipitale and the Os sphenoidale.

The vessel travels in the form of an S-shape, first in the ventral and then in the superodmedial direction in the Sinus cavernosus besides the Os sphenoidale. Then the A. carotis interna passes the Dura mater medial of the Proc. clinoideus.

Now it travels in the intracranial sphere. It travels in pairs to the posterior side and to the side and comes to rest between the N. oculomotorius and the N. opticus. It ends behind the Trigonum olfactorium, immediately caudal of the substantia perforata anterior. At this stage, the A. carotis interna separates into two branches, namely the A. cerebri anterior and A. cerebri media.

A.cerebri media is the strongest intracerebral vessel, supplies approximately two thirds of the brain hemispheres and on its course to the cerebral surface it arborizes into many parallel, thin and wide-ranking branches. They are called Aa. lenticulostriatae or Rami perforantes and they supply the stem ganglia and the capsula interna and externa.

Indirectly, the **A.vertebralis** is meaningful in this connection, because it anastomosizes via the Ramus communicans posterior of the circulus arteriosus Willisii with the Carotis stream region. This connection to the stem ganglia only becomes functionally effective in cases of vessel pathologies in the carotis region.

The Arteria vertebralis originates in the A subclavia and travels between the M. longus colli and the M. scalenus upwards in the anterior direction and rises inside the processi transverse between the first to the sixth cervical vertebra. Via the first arch of the first vertebra it runs to the Membrana atlanto-occipitalis posterior, passes it and travels to the subarachnoid space. From there it passes the Foramen magnum before it runs into the cranial fissure. The Aa. vertebrales on either side unite at the height of the pontomedular transition to form the unpaired A. basilaris. This vertebro-basilar system supplies the infratentorial and the dorsal supratentorial cerebral region.

If a dysfunction, particularly one of the **Synchondrosis sphenobasilaris** occurs, the artery may be narrowed by bones. Magoun describes the connection of the SSB with the vascular supply of the vital zones of the CNS[11]. Dysfunctions of the Os sphenoidale, especially torsions or side-bending rotation, might cause disturbances of the A. cerebri media and the normal movement of the CSF in the subarachnoid space.

The venous drainage of the basal ganglia:

The venous drainage of the basal ganglia is carried out by the V. thalamostriata (V. terminalis) and the V. basilaris (Rosenthal), which belong to the deeper cerebral veins.

The drainage of the deep cerebral veins is exclusively carried out by the sinus rectus.

The **V.thalamostriata** receives affluents from the border region between the frontal and the parietal lobe. It travels supependymal on the ground of the Pars centralis in the sulcus terminalis between the Thalamus and the Nucleus caudatus.

The V.thalamostriata unites with the V. choroidea and the V. septi pellucidi at the Foramen interventrikulare Monroi to form the V.interna cerebri. It travels to the backside above the top of the third ventricle where it unites with the vein of the other side. The V. magna cerebri (Galen) is a short vessel, which travels from this point of unification to the Sinus rectus.

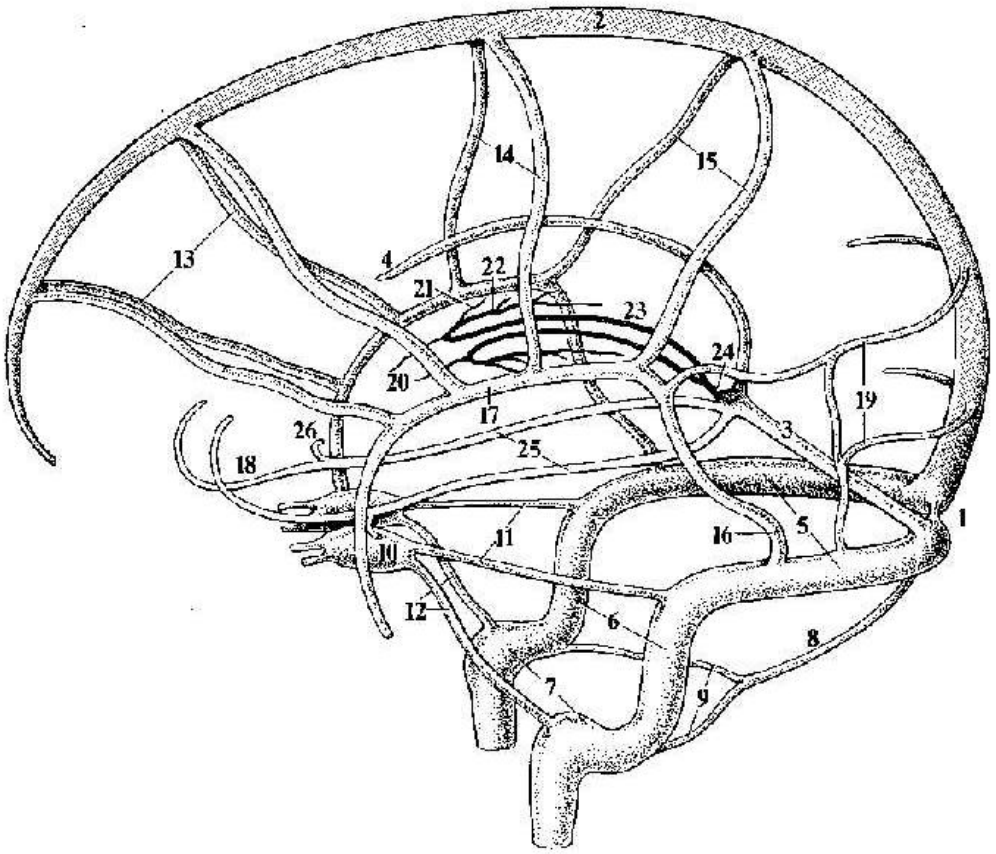


Abb. 3.15 Räumliche Darstellung der Hirnvenen (nach GRAY)

Sinus durae matris
1 Confluens sinuum
2 Sinus sagittalis superior
3 Sinus rectus
4 Sinus sagittalis inferior
5 Sinus transversus
6 Sinus sigmoideus
7 Bulbus superior venae jugularis internae (als Fortsetzung von 6)
8 Sinus occipitalis
9 Sinus marginalis
10 Sinus cavernosus
11 Sinus petrosus superior
12 Sinus petrosus inferior

Venae superficiales cerebri
13 Vv. frontales
14 Vv. parietales
15 V. anastomotica superior (TROLARD)
16 V. anastomotica inferior (LABBÉ)
17 V. media superficialis cerebri
18 V. lobi frontalis cerebri
19 Vv. occipitales

Venae profundae cerebri
20 V. septi pellucidi
21 V. thalamostriata
22 V. choroidea
23 V. interna cerebri
24 V. magna cerebri (GALEN)
25 V. basalis (ROSENTHAL)
26 V. cerebri media profunda

Depiction 6: Drainage of the deeper cerebral veins (taken from Firbas, Neuroanatomie)

The **V.basalis** receives its blood from fronto-basal cerebral tracts and from the insular region via the V. cerebro profunda. It travels around the midbrain in the Cisterna ambiens. It absorbs blood from the midbrain and from the Corpus pineale and then it enters either into the V. magna cerebri (Galen) or directly into the sinus rectus.

The **Sinus rectus** forms part of the venous blood vessels in the region of the hard

meninges.

They are formed of the periostal and the meningeal blade of the Dura mater and absorb blood from the brain, the meninges and the diplopia and are interconnected via emissaries with extracranial veins. The Vv. diplopicae are situated between the Lamina interna and the Lamina externa of the top of the skull. Via the cranial openings the Vv. emissariae connect the diploid veins and the Sinus durae matris with the surface veins of the skull. The Sinus durae matris are not only responsible for the venous drainage of the brain, they also house the Pacchionic Granulations, through which the liquor cerebrospinalis is reabsorbed into the venous system.

95% of the venous blood leave the blood over two Foramina jugulare. A small part is drained through the Plexus basilaris and the Plexus pterygoideus. Every Foramen jugulare is formed by an articulation of the Processus basilaris of the Occiput and the Pars petrosa of the Os temporale. The walls of the venous sinus are formed by the dura mater. The absence of tensions in the dura has always to be viewed in connection with the mobility of the cranial bones.

If there occur tension alterations in the region of the Dura, e.g. by bony lesions, this may influence the diameter of the Sinus.

The venous blood is transported to the exits by membranous activity. Every form of luxation in this region influences the venous reflux of the entire cranium, according to Sutherland. [17]

These tension alterations, which are continued in the entire body, can be explained with the **”Reciprocal tension membrane“.**

The reciprocal tension membrane is a designation for the intercranial and extracranial dura, which form a functional unit. Intracranially, falx and tentorium form the connection to the bones due to their starting points. Extracranially, the dura surrounds the spinal marrow and there exist fastenings at the Foramen magnum at the heights C2, C3 and S2 and other, less strong fastenings.

This system, which is called „craniosacral system“ owes its designation to this connection of the cranium with the sacrum via the Dura mater. Sutherland describes a fixed point (Sutherland Fulcrum) in the field of the Sinus rectus around which the

reciprocal tension membrane is circulating.

The Dura mater, die deeper **Fasciae** and the Periosteum are made up of the same collagen composition. The fibres are woven crosswise and act like a concertina barrier, which allows shape changes with a constant volume.

There is a very strong connection between the skull base and the Fascia pharyngobasilaris, which forms the posterior wall of the respiratory tract. It continues downwards and unites with the M. constrictor, the hyoid bone and the lower neck fascia. For this reason, connections with the fascia of the aortic arcade, the sternum, the breastbone and the diaphragm arise. If problematic zones occur in this region, a shift of the skull base may be caused.

Liquor cerebrospinalis

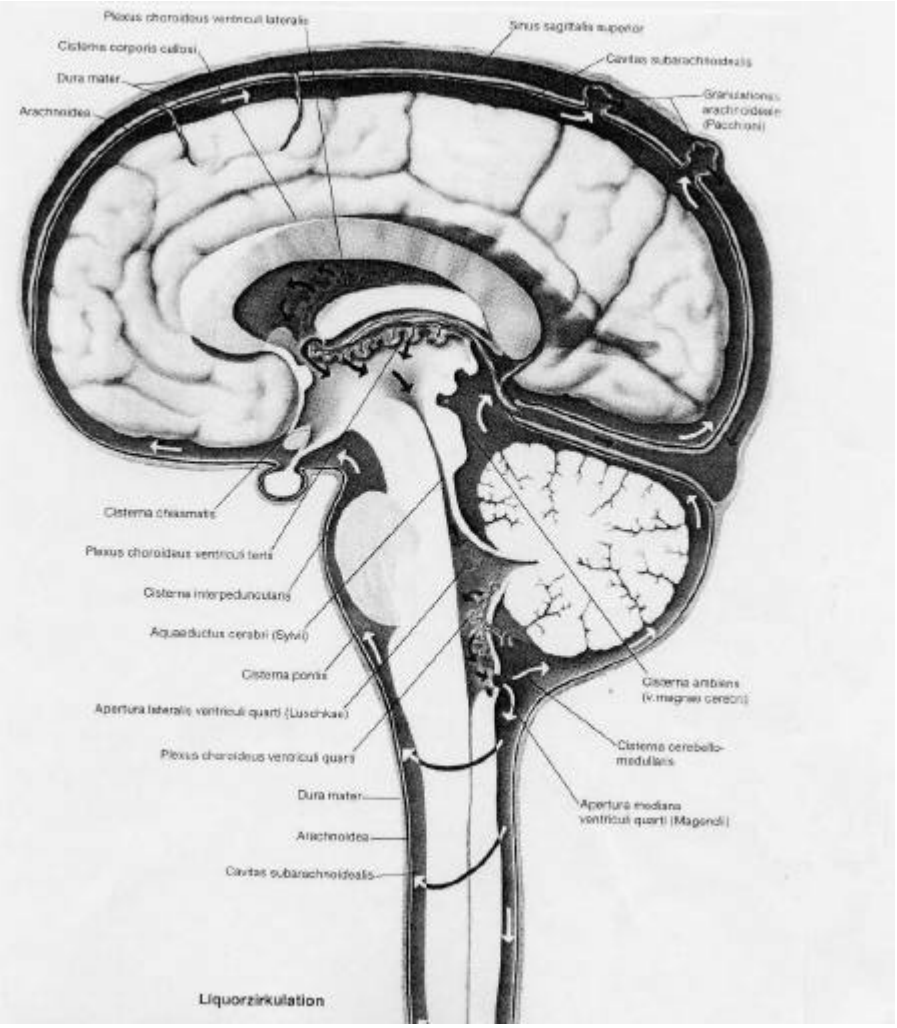
The liquor refills the interior spaces of the brain **(ventricles)** and surrounds the CNS everywhere in the **subarachnoid space**. The inner and outer spaces communicate with each other in the region of the fourth ventricle.

This movement is called **inspiration phase**, which is perceived as contraction and broadening in the human body and expiration phase, which is felt as extension and narrowing.

During the inspiration phase the sinus broaden, during respiration they narrow. This can be described as a pump function in the osteopathic sense. During the inspiration phase the side ventricles are swinging outwards and the third ventricle has a V-shape. In this phase the plexus choroideus at the top of the third ventricle is extended.

During the **exhalation phase** the side ventricles contract and the plexus relaxes (bundling). If this mechanism is disturbed it can be imagined that the chemical balance between blood and liquor is disturbed. If the movement of this mechanism is restored, the question whether the normal physiological exchange between blood and liquor can function again, arises.[17]

The close relationship between the liquor system and the basal ganglia can also be demonstrated at an example from **embryology**:



Depiction 7: Liquor circulation (taken from Netter, Atlas Neuroanatomie)

The cerebral hemispheres develop primarily from neuroepithelium at the beginning of the 5th week. As a large number of neuroblasts is produced in the neuroepithelium a coat layer is formed in the basal section of the hemispheres. This layer enlarges at the middle of the second month and bulges into the lumen of the side ventricle and the lower section of the Foramen Monroi. On the basis of the side ventricles the motor nuclei of the cerebrum form, namely the basal ganglia. As the cerebral hemispheres grow, the basal ganglia at the ground of the side ventricles enlarge. This is the reason of the close relationship between the basal ganglia and the liquor system.

METHODOLOGY

Study design

Patient group:

Patients afflicted by idiopathic Parkinsonism registered in my practice after I had put an ad for the study in the “Journal of the self-help group of parkinsonian patients in Vienna“. A first assessment according to the following criteria for participation and exclusion was made:

Inclusion criteria:

1. Idiopathic Parkinsonism, Rigor-Akinesia dominance-type
2. Ability to walk without any means of support
3. Stable state during a medical therapy for at least 4 weeks without any significant response fluctuations
4. Absence of any treatment modifications during the study

Exclusion criteria:

1. any other illness of the CNS which is associated with some impairment of the motor system
2. on-off phenomenon
3. osteopathic pre-treatment

Based upon the fulfilment of the participation and exclusion criteria, the patients were chosen **at random** to form two patient groups. The patients were neither given any information about the division into patient groups nor about the kind of treatment.

The patients were treated on two dates in an interval of 3 weeks for 60 minutes. The time of the day was the same on these two dates in order to exclude any possible response fluctuations.

At the first date, **Group 1** received an **osteopathic treatment**.

Group 2 was the **control group**. Some unspecific physical training exercises were applied.

At the second date, the methods of treatment of the two groups were interchanged (**cross-over** design), so that in sum each patient received one osteopathic and one unspecific treatment. I chose this design also because I wanted to ensure one osteopathic treatment for each patient. This offer was meant to motivate the patients to participate at the study and it facilitated the search for volunteers. The interval of 3 weeks between the two treatments was chosen relatively long to exclude a long-term effect of the first treatment as good as possible.

Tests

Tests, which are relevant for the medical practice and can easily be applied and repeated, were selected. The results of these tests were not predetermined by personal opinions. The tests were made before and after each treatment to evaluate and to take record of the motor function.

Test 1: Walking of a distance

The patient was told to walk 10 steps straightforward and then the required time and the walking distance were measured. On the basis of these measurements, the average length of one step and the walking speed were calculated. The patients started from an upright, standing position in order to take record of any possible start hesitation. The patients were asked to walk their usual fast walking speed, when they heard the command to start.

Test 2: Alternating pro- and supination movements of the forearms

The time which the patient needed for 10 sequences of alternating pro- and supination movements of the forearms was measured. The starting position was a seating position and the proband's palms lay down on his thighs. After each movement the patient had to contact the backside of his hands or the palms against his thigh.

Control group of healthy persons

A control group consisting of persons of comparable age and gender distribution was recruited according to the random-principle (pedestrians at the market square in Perchtoldsdorf nearby Vienna). This control group was asked to participate in Test 1 (Walking test).

Test material:

For the test of the motor skills I used only a time-keeper, a tape-measure and a starting-line. This demonstrates the feasibility of the tests with simple means.

Statistical target parameters:

The first target parameter for statistical assessment is the comparison of speed of gait and pro-and supination movement of the forearm before and after the therapy of both groups. The results of both groups are compared. In cases of significant speed increases, further examinations shall answer the question whether improvement can be achieved by means of a reduction of the walking time or by means of a larger walking distance. Furthermore, some possible impact of the starting speed on the speed improvement shall be examined. The starting speed of the patient group shall be compared with a control group of healthy individuals to evaluate to what extent the motor skills of the patients are impaired.

Organisation of the treatment

For this study **23 patients** in total were recruited, 4 patients dropped out for organisational reasons or due to infractions of the protocol. 19 patients completed the examination tests and were evaluated. 9 of them were female, 10 were male. Their average age was 66.9 (+/- 7.8) years. The youngest participant at the study was 38 years old, the oldest one 79 years. The average duration of the sickness is 8.8 (+/- 5.5) years.

The **control-group** of healthy persons consisted of 18 persons, 9 of them were women and 9 were men. Their average age is 66.9 (+/- 7.7) years.

Osteopathic access:

Anamnesis:

An anamnesis of the osteopathic symptoms and one of the clinical symptoms of each patient were taken to record. The results of this anamnesis record were entered into a list, which you will find attached in Table 1 and 2 in the appendix of this study.

In the anamnesis sheet you can see data referring to duration of Parkinson's disease, to the first clinically-visible symptom, to the present antiparkinson medication and to the question, if the sickness started on one side of the body.

Additionally, the patients were posed questions to outline a **clinical score**. They were asked to evaluate 13 single symptoms in a scale ranging from 1 to 5 (1-never, 2-almost never, 3-sometimes, 4-often, 5-always). The following single symptoms were evaluated: tremor, rigor, akinesia, on-off phenomenon, depressive mood, bradyphrenia, sleeping disturbances, sleep attacks during the day, hypersalivation, obstipation, bladder disturbance, disturbed temperature regulation, pain. The on-off phenomenon was included in the evaluation sheet because it constitutes an exclusion criterion. All participants of the study answered this question with „never".

The total score number is the median of the several single evaluations and rises with the intensity of the patient's symptoms.

The clinical score has the purpose to assess the seriousness of the sickness of the individual patient on a scale. The average score number was 2.31 (+/- 0.35). The lowest score number was 1.85, the highest was 3.15. The illness appears to afflict all patients with a unique seriousness, in spite of huge age differences and different duration of the suffering. The relatively low average score is a consequence of the participation and exclusion criteria, as only patients with the ability to walk but without on-off phenomenon were allowed to participate.

The general anamnesis constitutes a record of the complaints affecting the inner organs, the operations, the traumata, the allergies, the infects and the events in connection with the onset of the illness.

Examination and treatment:

The **GOT (general osteopathic technic)** was applied as **initial** method for subsequent diagnostics and treatments. The aim was to get a first impression about the elasticity of the tissue, the limbs, the tendons and the muscles. At first, it was a positive influence for the muscular tone and also it served for diagnostic means in subsequent osteopathic approaches. It was also an occasion to strengthen the patient's confidence beneficially. The regulation of the tonus and the simultaneous mobilisation of several joints have always been an important element of the treatments because the access to the joints and to the muscles would be more difficult as a consequence of rigor.

The **subsequent treatment** of the patients focused on several individual problems. In many cases, the C0/C1-region, the collar bone, the thoracic inlet, the dorsal bone, the ribs, the thoracic diaphragm and the pelvis were treated. With regard to the anamnesis, the various problematic regions were put into categories in the course of the treatment.

On the table 3 in the appendix becomes obvious, where **lesions at the structural stage** occurred in each individual patient.

Treatment techniques:

The treatment techniques were attuned to the individual patient. In many cases soft tissue techniques were applied. Thrust techniques were never employed, because on the one hand the average age was rather high and on the other hand, long-maintained compensation mechanisms with vertebral group lesions had occurred.

Persons suffering from Parkinson's disease have a similar outward appearance. So it was much more surprising for me to find a broad spectrum comprising highly-diversified treatment focusses. As a result of the pathologically-raised muscular tonus, certain muscular groups seem to force the skeleton into a particular position, but nevertheless each patient proves to be a very individual problematic case.

Most parkinsonian patients suffer from a scoliosis leading to a torsion of the diaphragm. In the regions of the diaphragm and the pars mediastinalis, craniosacral techniques proved to be helpful. Feeling the primary respiratory mechanism in this

area also constitutes a significant barrier on the energetic stage. To many patients their thorax seems to be a shell.

The decompression of the 12th rib often formed part of the therapy in order to exert some influence on the thoracic diaphragm as well as on the Musculus psoas via the Ligg. arcuata. The pelvis and the hips were preferably treated with myofascial techniques.

In the region C0/C1 lesion patterns in the form of compressions or rotations were found. This led to a significantly-raised tension of the suboccipital membrane.

In this region it was beneficial to relax the suboccipital membrane or musculature with release techniques. Tractions for the entire cervical spine were also often applied.

In the region of the cervical spine and the muscles of the shoulder-girdle, which showed an extremely high muscular tone, fascial stretching techniques, primarily of the cervical fascias were employed.

As a consequence of posture, parkinsonian patients suffer from pressure relations which are unbeneficial for the digestive organs in the stomach region. The visceral side of osteopathy was integrated indirectly in the form of the treatment of the sacral bone, the pelvis, the fascias, the muscles and the diaphragm.

Contents of treatment for the control group:

The patients received physical training exercises. The length of treatment corresponded to that of the group which received osteopathic treatment.

The exercises were adapted to the motor skills of the patient and done from the following starting positions: supine, seating and upright position.

Fix constituents of the treatment were:

- stretching of contracted muscles
- relaxation exercises like e.g. breathing technique exercises
- balance-control exercises
- exercises for weak trunk muscles
- supporting help at position-shifts

RESULTS

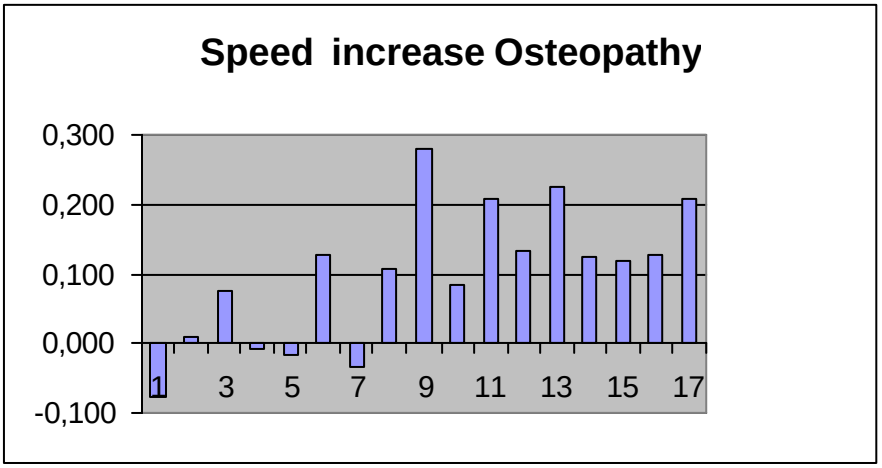
17 out of 19 persons were evaluated in the walking test and in the pro- and supination test. Two patients were dropped due to very unexact time measurements. They happened because the patient did not understand the task clearly or because he carried it out wrongly. In the control group of the healthy persons, the measurements of all 18 participants were included in the statistical evaluation.

Osteopathy group: Walking test

The average speed before the therapy was 0.85 m/sec and after the therapy it was 0.95 m/sec, what corresponds to an improvement of more than 10 %.

On average, the increase in speed amounts to 0.1 m/sec (+/- 0.07 m/sec), 4 patients out of 17 showed a tendency to loose speed, 13 showed a tendency to increase their walking speed. The highest decrease is 0.076 m/sec, the highest increase is 0.28 m/sec.

The statistical evaluation reveals a speed increase of a highly positive significance. (p= 0,001).



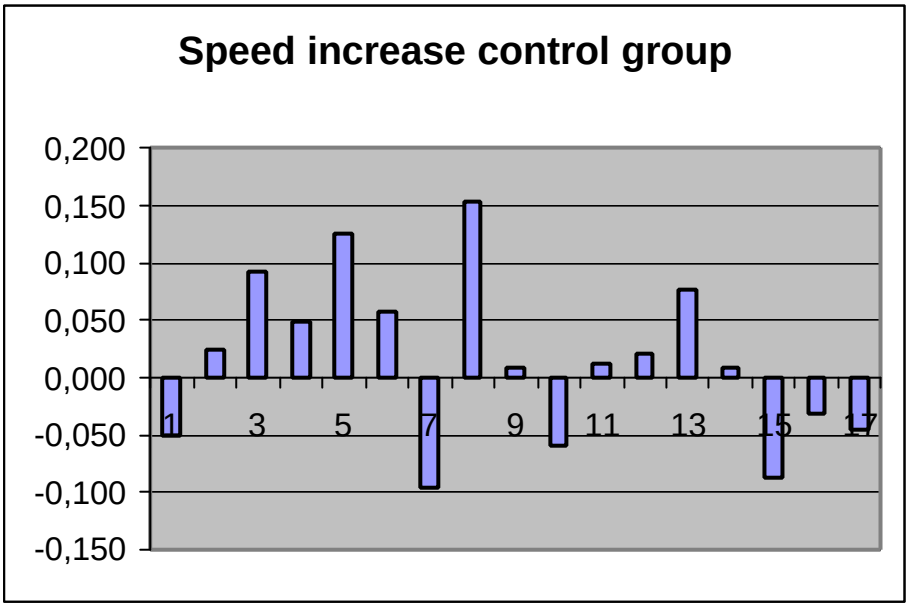
Graph 1: Changes of the walking speed after osteopathic treatment

Control group: Walking test

The average speed before the treatment was 0.88 m/sec, after the treatment it was 0.90 m/sec.

The average increase of 0.02 m/sec with a statistical error of +/- 0.06 m/sec does not diverge significantly from zero in the statistic evaluation (p=0,935).

The number of samples, which are at my disposition, is not sufficient to deduce an increase of speed in the control group. It cannot be excluded that a more representative sample including more patients may prove the desired result, but the achieved results are by far less remarkable than those of the osteopathic treatment - and this is already obvious from the data which are at our disposition.



Graph 2: Changes of walking speed in the control group

Osteopathy group - control group walking test

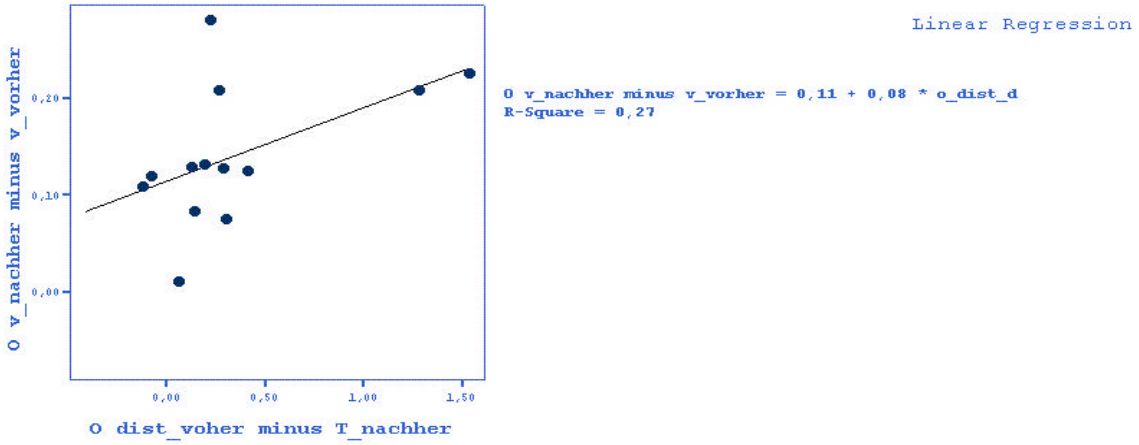
A comparison of speed increase in both groups (speed increase osteopathy group minus speed increase control group) also reveals a significant result. (p=0.01)

Detailed results: walking test, osteopathy group

As the result of the osteopathic group was significantly positive, it will be analysed in closer detail:

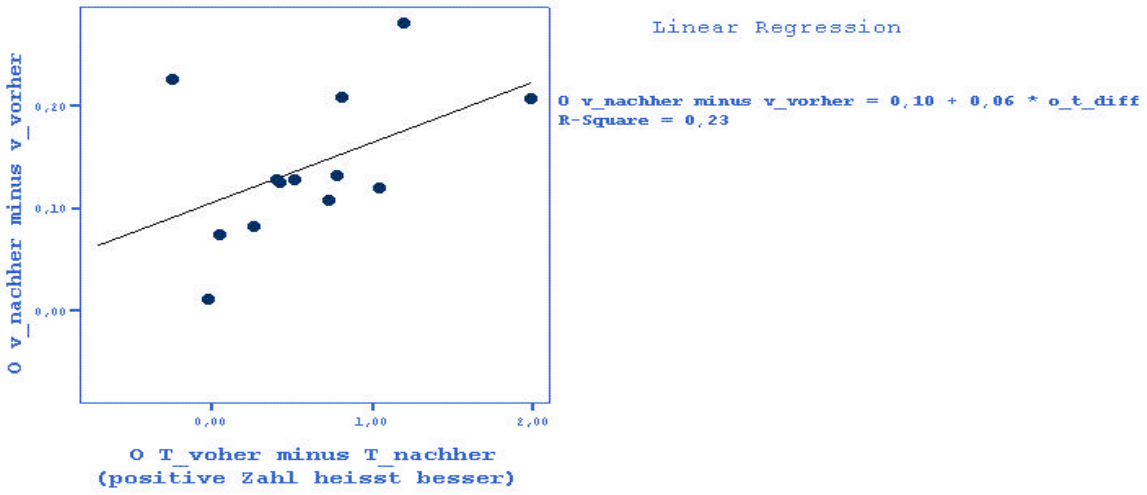
The speed increase is either a consequence of a wider distance (= a larger average length of steps) or of a reduction of time (=patients walk faster).

As far as the question, whether speed increases on the basis of **distance** is concerned, there is no significance, although a tendency can be seen. (p=0.071)



Graph 3: Speed increase on the basis of the distance

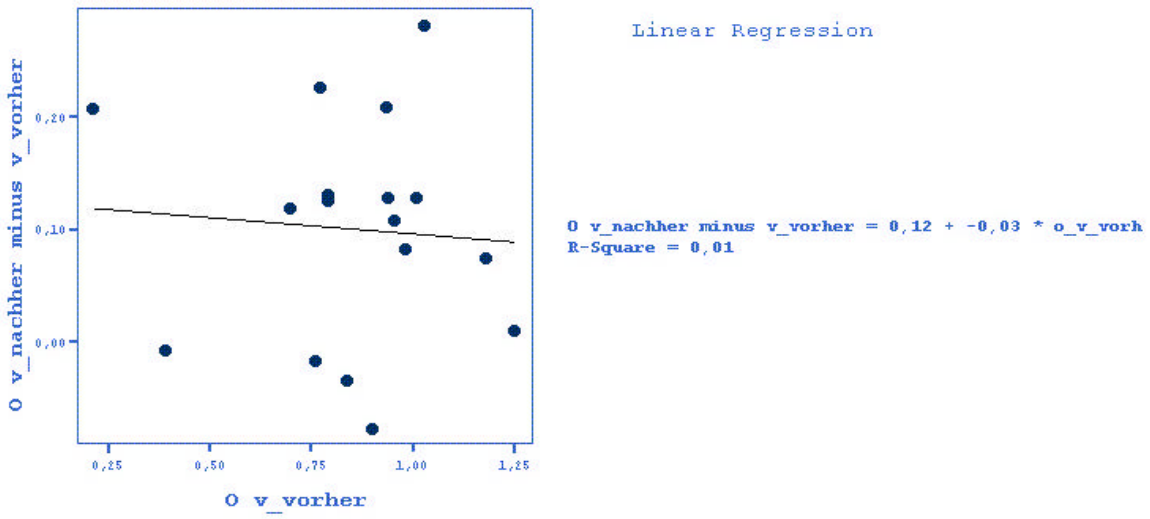
As far as the question, whether speed increases on the basis of **time** limits is concerned, there is no significance (p=0.097)



Graph 4: Speed increase on the basis of time

So we can conclude that speed increase in the osteopathy-group was rather achieved by means of a wider distance than by means of time limits, although no significance for any single parameter can be seen. A significance became only apparent after both parameters (=speed) were combined.

Does the starting speed of the patients influence speed increase? In this connection, no relation can be derived.

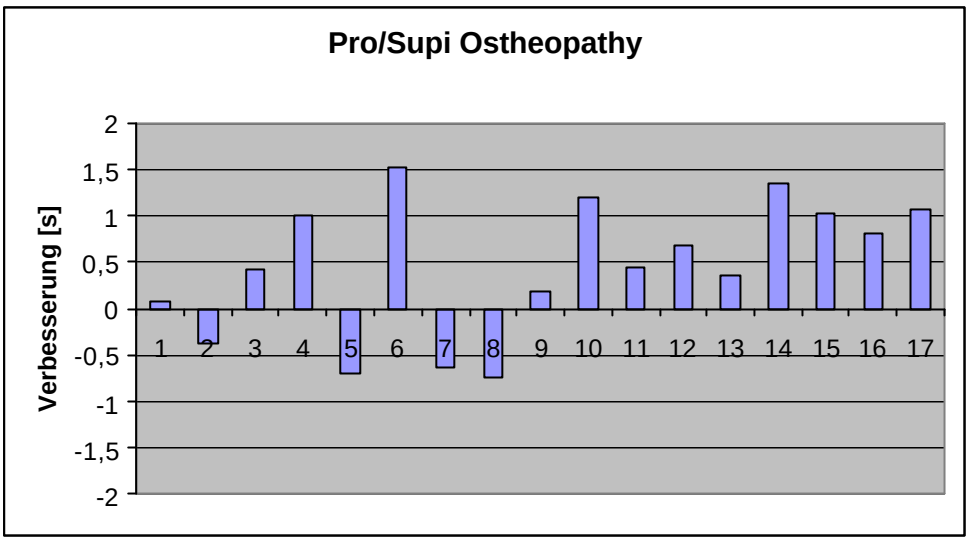


Graph 5: gait improvement, influence of initial speed of gait

So, speed increase in the osteopathic group does not depend on the usual walking speed of the patients before the therapy.

Osteopathy group: Propination- supination of the forearms

The time measured for pro- and supination averaged 9.21 s before the therapy. On average the improvement amounts to 0.45 sec or 5%. 13 out of 17 patients have improved and 5 among them improved by more than 1 sec which corresponds to a percentage of almost 10%. The results of the paired-T test are significant (p=0.021)



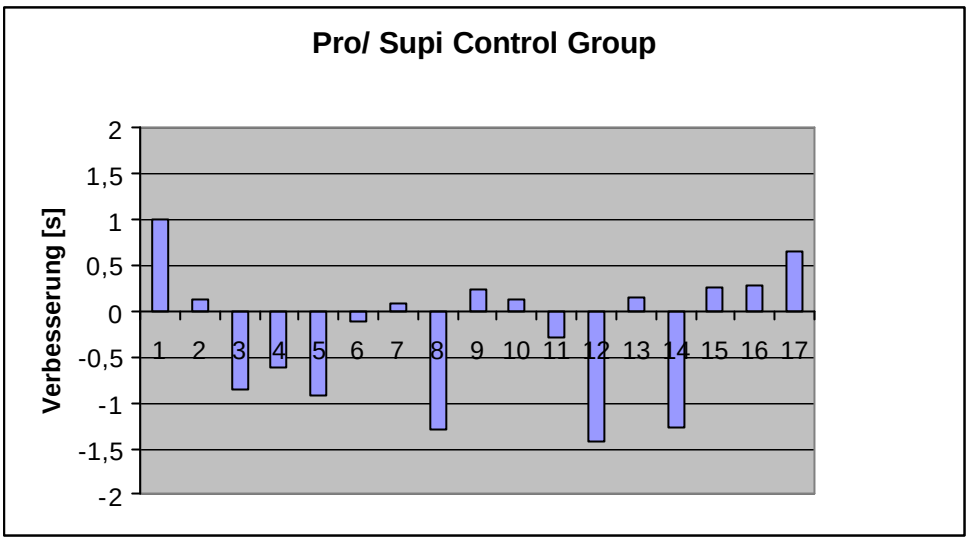
Graph 6: Change of speed alternating movements

There exists no correlation between the improvement and the starting speed, which means that there are quick as well as slow patients who have improved or worsened.

Control group pro-supination:

The time measured for pro- and supination was 9 sec on average in the control group before treatment and 9.23 sec after the treatment. On average, there is a slight loss of time of 0.23 sec., which however is not significant due to the error margin of +/- 0.69 sec. No tendency has become obvious. What is surprising, is that only one patient was able to achieve an improvement of more than 1 sec while 3 patients lost much time.

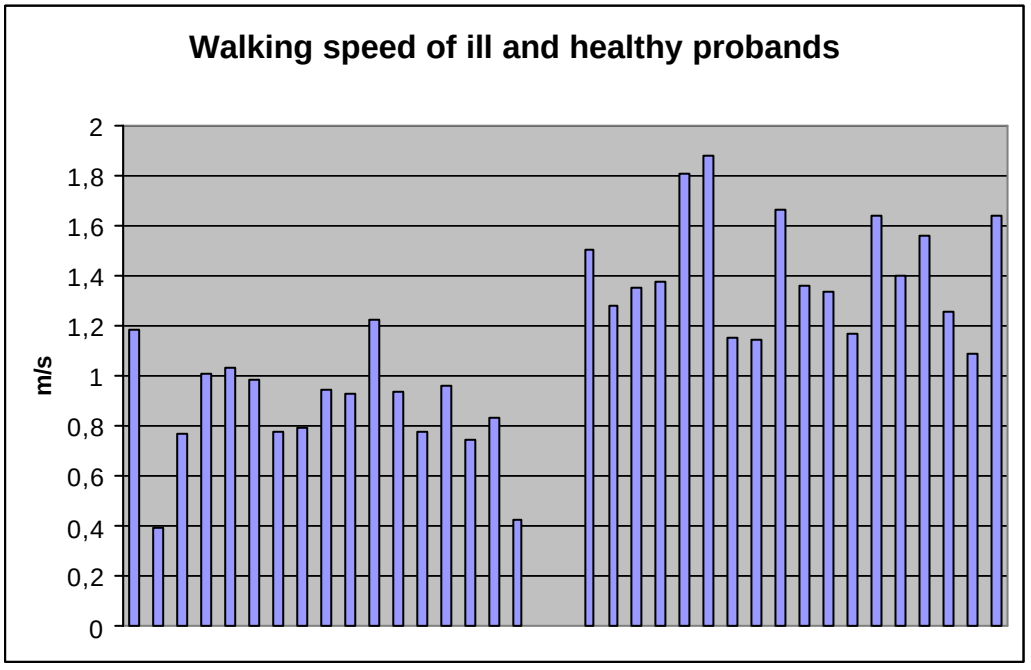
Similar as in the osteopathy group, no relation between starting speed and the improvement of time is obvious.



Graph 7: Change of speed alternating movements control group

Patient group - Comparison group healthy persons

The different walking speeds of the patient group and the control group can be seen in graph 8.



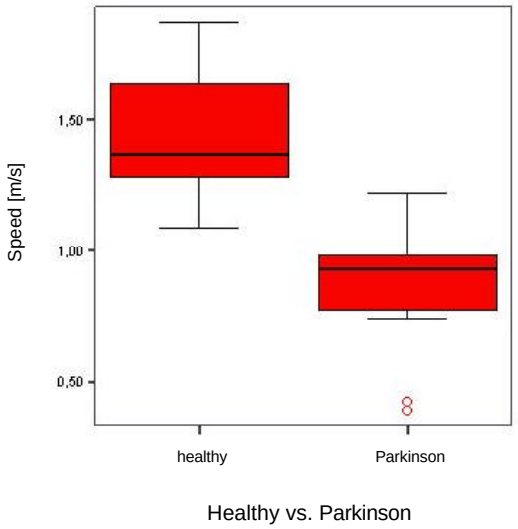
Graph 8: Walking speed ill and healthy probands

On average, parkinsonian patients have a usual walking speed of 0.86 m/s (which corresponds to 3.11 km/h) The healthy control group averages a usual walking speed

of 1.42 m/s (5.11 km/h). Only two parkinsonian patients would have been able to walk as fast as the slowliest ones of the healthy probands.

The T-test and the Mann-Whitney test are significant.

The test result can be seen in graph. 9.



Graph 9: Range of fluctuations of walking speed of ill and healthy persons

DISCUSSION

The primary result of my study is, that the speed of gait of parkinsonian patients can be influenced positively by means of one osteopathic treatment. The patients of my group, all of which suffer from an already progressed Parkinsonism, are significantly impaired in their motor activity. Most of them need help in their everyday lives and all of them suffer from postural instability. The extent of the gait disturbance of the patient group becomes apparent in a comparison with a control group of healthy persons. Here it becomes evident that the patients walk on average 2 km/h slower before the treatment (3.11 km/h the patients compared with an improvement of 5.11 km/h of the healthy persons), which means in other words that patients achieve only about 60 % of the speed of healthy persons.

After on osteopathic treatment patients walk by approximately 10 % faster and achieve as well a statistically significant speed increase (p=0,001) However, the walking speed of healthy persons is also not approximately achieved after the treatment.

In the patient control group no significant effect on the speed of gait was measured. This demonstrates that the treatment does not lead to any relevant (placebo) effect.

If we analyse the result of the gait measurements in detail, there is an interesting aspect. The speed increase is achieved via the factor distance (larger steps) as well as via the factor time (faster steps), but the majority achieved it rather via the distance than via time. So, the patients primarily have a larger frame of action for their single movements which in sum results in an increased length of steps

The degree of the improvement does not depend necessarily on the initial situation. Patients who had relatively few bad initial scores within the patient group benefited as much as those who had relatively good initial scores. So, an effect within the whole range of the members in the patient group, which was formed on the basis of the participation criteria, can be proved.

As far as the walking speed is concerned, I also examined whether some effect can be proved after 3 weeks have passed. The period of 3 weeks between the 2 examinations was chosen arbitrarily by myself, because I found no details about any

long-term effects of an osteopathic treatment in the literature. I chose a rather long interval because of the study-design (cross-over design) and in order to exclude any effects of the first treatment in connection with the initial situation of the second treatment as good as possible.

Any long-term effects on gait can not be detected, neither in the group which received an osteopathic treatment nor in the control group. The parameters initial walking speed before the first treatment and initial walking speed before the second treatment do respectively no differ significantly. 3 weeks after the first treatment there is no effect independently from the factor, how the treatment was applied. The measurements of the second treatment are definitely not influenced by any long-term effects of the first treatment which is relevant for my treatment instructions as mentioned above.

The question how long the effect of an osteopathic treatment on gait lasts can not be deducted from my study but in any case, it is shorter than 3 weeks. (Note: According to individual estimations of my patients, who I have treated for a long time now, the improvement of their motor abilities in everyday life lasts for approximately 4-5 days.)

To demonstrate the changes of the patients motor ability not only punctually at the example gait, another measure parameter was examined. The time required for a sequential combined pro- and supination movement of the forearm was measured. In this connection, the patient group which had received an osteopathic treatment was also able to improve speed significantly ($p=0.021$), which was again not true for the control group. This result proves an effect of the osteopathic treatment in this field as well. With a speed increase of approximately 5 % the is nevertheless not as clear as the result of the gait analysis.

I want to reflect about the possible reasons of the results. The condition that the movement should be carried out exactly, could not be fulfilled properly by some patients because of their restricted motor abilities (especially the supination movement). The measurements of this movement contains more sources of errors or uncertainties in connection with the cooperation of the patients which must be reckoned as a basic restriction for the interpretation of the results.

Another difference to the gait test is the fact that in this test an improvement can only

be achieved in time, because the distance is exactly prescribed in the instructions. (The palms and the backside of both forearms must entirely touch the thigh). As demonstrated in the gait test, the effect was rather achieved via the distance factor than via the time factor, and so only a slight improvement could be expected in this connection.

As far as the kind of movement is concerned, gait and alternating forearm movements have different characteristics. For gait large muscular groups and muscular portions in the proximity of the axis are needed and involuntary movements are very important. On the contrary, in alternating movements rather distal, smaller muscles are involved and the movements are primarily the result of voluntary, skilled muscular activity.

Why are different movement patterns influenced to a different degree by Parkinsonism? The answer to this question is certainly manifold and depends primariliy on the functioning of the basal ganglia, and its automatic control system which is not clearly known to us.

The impairment of the motor activities typical for Parkinsonism is nevertheless in every case caused by the primarily impaired planned movement at the central stage as well as by the secondary phenomenons, which develop as the illness progresses. Among the latter rank for example the abnormal posture or chronic pain.

This point leads us to the question whether osteopathic approaches can be found. From the point of view of theoretic osteopathic considerations, basically both factors can be influenced, the central disturbance and the secondary phenomenons.

To influence secondary damage via a primarily structural approach is plausible. Besides from the attention to wrong posture the pain problem, which further aggravates the everyday life of almost every patient, must be taken into account and integrated in the treatment.

Reflections about the influence on the brain remain more difficult and broadly speculative. Idiopathic Parkinsonism finally leads to increased depletion of dopaminergic neurons in the basal ganglia. The reason of the damage is unknown, a toxic mechanism, which affects the cells is regarded to be responsible for it. It is

possible that an ameliorated circulation of fluids in the brain may improve the conditions for physiological processes and that a progression of cell depletion and the illness may be slowed down.

By means of the techniques of craniosacral osteopathy, there exists the possibility to influence the mobility of the cranial bones. Several osteopathic works demonstrate that the mobility of the cranial bones exerts an influence on the circulation of fluids in the brain.[12, 17]

Magoun points out in particular that Synchondrosis sphenobasilaris (SSB) plays a role in the vascular supply of the vital zones of the CNS. Dysfunctions of the Os sphenoidale, especially torsions or side-bend rotations, may cause a disturbance of the A.cerebri media and the movement of the cerebrospinal fluids in the subarachnoid space[11]. In the study of Rivera-Martinez nevertheless, the presence of the cranial strain patterns in the region of the SSB could not be verified in parkinsonian patients.[15.] This finding renders the meaning of SSB in connection with Parkinsonism less important.

On the contrary, the links between trunk and head appear to be particularly more relevant for this illness. Parkinsonian patients show an especially apparent wrong posture in this region. A factor which favours this is the abnormally raised muscular tonus. Magoun describes 36 muscles which originate in the Os occipitale and in the Os temporale.[11] How a unilateral contraction, e.g. of the M. sternocleidomastoideus may lead to an atlantooccipital compression, was described by Bernhardi.[7]

The clinical meaning of this dysfunction in parkinsonian patients was proved by Fiske who stated that there exists a connection between the cervical flexion and a compensatory anteriorised atlas.[8] This also leads to an atlantooccipital dysfunction. Rivera-Martinez found out in a comparison that there is a higher frequency of bilateral atlantooccipital and occipitomastoidal compressions in parkinsonian patients than in a control-group of the same age.[15] These regions are exactly the points where the arterial blood stream and the venous reflux of the brain pass. If we suggest that the structure has an influence on the function, this region can also be regarded to be a key region concering the functioning of the brain in connection with Parkinsonism.

The descriptions in literature do not give any indications for a defined concept of

treatment. When I planned this study, I also took my experiences with patients from my work in the hospital as a guideline. These experiences indicated that an individual osteopathic approach reveals itself as positive in any case. An individual approach takes account of the structural lesion chains of the individual patient and can be very effective at this stage. To reckon this point, I did not reflect about a fixed treatment plan when I set up the concept of the study. This examination again showed me how different the initial situations of the patients may be in spite of the common diagnosis Parkinsonism. Finally, the individual approach corresponds rather to the practical osteopathic work than a fixed sequential treatment.

I am aware of the fact that the individual approach may be disadvantageous for the evaluation of the results, because not all the treatment sequences can be objectively retraced, reproduced and in this way compared with other results. Nevertheless, the results of the treatment remain plausible and clear even before this background.

Further examinations can be built up on the basis of these results. To make a comparable approach for such examinations possible, I used only such equipment for my tests, to which everybody has an easy access (tape-measure and time-keeper). In this connection it was especially important for me to find a simple but expressive test. To measure the wallking speed appears to be a good instrument to obtain information about the quality of treatment of Parkinsonism.

In my study I restricted myself to the aspect of motor abilities of parkinsonian patients. I was not able to treat other subjects in connection with this much-layered illness and did not examine them systematically. One interesting point has crystallized for me in the course of my research work and this was the psychologic aspect.

At the beginning of the study many patients were doubtful and in this patient group it was difficult to make the body accessible for osteopathic treatment. It is also very remarkable that the majority of my patients connects an extraordinary psychologic burden with the onset of the suffering in the anamnesis. The sternum, which is regarded to be an emotional zone in osteopathy and which seems to be stare shield for the patients was the focus of the treatment. For almost every patient, especially the thoracic diaphragm appeared to be one of the most important zones and it

showed various torsion patterns on the structural as well as on the cranial stage. It would certainly be a major challenge to elaborate this field more closely under the aspect of mutual influences between the psyche and the motor activity.

This question and many others have remained unanswered in the field of Parkinsonism-osteopathy, some have not yet been posed. I want to encourage my colleagues to carry out more studies. For a single person or a small group they can easily be realised without much expenses.

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Appendix

Explanation of Table 1 and Table 2 (Anamnesis)

Clinical score:

The patients were asked the following 13 single symptoms:

Tremor, rigor, akinesia, on-off phenomenon, depressive mood, obstipation, bladder disturbance, disturbed temperature regulation, hypersalivation, sleeping disturbances, sleep attack during the day, bradyphrenia, pain.

When the patients were asked about the symptoms, they were clearly explained to them. The tables contain the subjective judgements of the patients.

The presence of the symptoms was judged on a scale ranking between the scores 1 and 5, on which 1 stands for “never” and 5 for “always”.

The on-off phenomenon was questioned to assure that none of the patients suffers from it because it would have constituted an exclusion criteria. Every patient answered this question with “never“.

The total score number is the median of the sums of the various evaluations and rises the higher, the more serious the patients are concerned by the disease.

Patient No. Age, m/f	Duration of the illness in years	First symptoms, subj.	Antiparkinsonian medication	Beginning of symptoms on one side	Present degree of complaints (acc. to clinical score)
1 78, f	5a	Small steps	Levodopa Entacapone Ropinirole	-	2,38
2 73, f	9a	Tremor	Levodopa Cabergoline Selegiline	Rright	2,77
3 62, f	16a	Tremor Gait detoriation	Levodopa Pergolide Selegiline Amantadine	-	2,70
4 75, m	12	Tremor	Levodopa Entacapone	Right	2,08
5 67, f	3	Small steps	Levodopa Entacapone Ropinirole	-	2,00
6 76, m	10	Micographia Bradyphrenia	Pergolide	-	3,15
7 38, f	3	Tremor Hypokinesia	Pramipexole Selegiline	Left	2,85
8 70, m	16	Tremor Micographia	Levodopa Entacapone Pergolid,eAmantadine	Right	2,92
9 63, f	4	Tremor Rigor	Levodopa Cabergoline Selegiline	Left	3,15
10 60, m	7	Tremor Postural changes	Levodopa Pergolide	Right	2,15
11 69, f	3	Gait detoriation	Pramipexoe Amantadine	-	1,85
12 63, m	17	Micographia Gait detoriation	Levodopa Lisuride Selegiline	Right	2,54
13 50, f	3	Rigor Hypokinesia	Pergolide	Right	2,62
14 76, m	3	Postural changes Gait detoriation	Levodopa Amantadine	-	1,85
15 75, m	5	Tremor	Levodopa Pramipexole	-	2,31
16 61, m	10	Hypokinesia	Levodopa Entacapone Pramipexole Selegiline	Right	1,92
17 72, m	33	Gait detoriation Micrographia	?	Right	2,15
18 64, f	5	Micrographia	Levodopa Ropinirole	Right	2,54
19 79, m	4	Tremor	Levodopa	-	2,31

Table 1

Patient No. Age, m/f	Complaints Inner organs	Operations	Traumata	Allergy	Infects	Events in connection with the onset of the illness
1 78, f	Reflux disease	TEP li CHE AE	Fract. radii.	Dust		TEP Acute psychologic burden
2 73, f	Obstipation Anuria Dyspnoe	Cataract Glaucome AE		Chinine		
3 62, f	Hypertonia Obstipation Incontinence	Uterus		Penicilline	Hepatitis	Acute psychologic burden
4 75, m	Reflux disease Nephrolithiasias Psoriasis	AE TE Nose polypsn		Pollen		Stress
5 67, f	Dyspnoe Incontence	NNH Uterus AE		Sulfonamide	Scarlatina	Acute psychologic burden
6 76, m	Reflux disease Abdominal pain Bladder weakness	CHE Prostata	LWK- Fracture	Penicilline	Hepatitis	
7 38, f	Hypertonia Migraine Asthma	TE	Tibia- Fracture		Meningitis Cystitis rez.	
8 70, m	Hoarseness	Hernia Renal caniculi Toe 1,2 ri.	Fracture of the skull base, max. bone fract.	Pollen Wasps		Stress
9 63, f	Sore throat rez. Dysphagia Obstipation	Struma Uterus CHE				Acute psychologic burden
10 60, m	Dyspnoe Dysphagia Obstipation	TE	Polytrauma (Femur, Coccyx,Ribs)			Acute psychologic burden
11 69, f	Stress incontinence Hypertonia	CHE Uterus, Adnexa AE	Torn tendon ri. knee			
12 63, m	Hypertonia Dyspnoe Incontinence	AE		Sulfonamide		Acute psychological burden
13 50, f	Vocal chord polyp Migraine	Uterus, Bladder plasitic, TE, Hernia umbil.		Scents		Acute psychologic burden
14 76, m	Dyspnoe Hypertonia Obstipation	Knee prothesis li Meniscus re			Hepatitis Malaria	
15 75, m	Hypertonia Hypakusis	Cancer l. intestines Scar hernia	Clavicula li		Hepatitis	
16 61, m	Obstipation		Torn tendon le. knee Tibia fissure.ri			
17 72, m	Reflux disease Ulcus ventriculi	Ossification both shoulders, both elbows	Frozen ears hands			Existential fear
18 64, f	Struma Parox. Tachykardia Renal dysplasia	CHE Uterus		x-ray contrast medium		I
19 79, m	St. p. Hypothyreosis	CHE, AE, Ileus Vitrectomy	Patella fracture, ri., Sacrum-Trauma			

Table 2

Explanation of table 3

The following table gives a broad survey of those body regions, where structural lesions occurred in parkinsonian patients. The table contains only those regions where joint dysfunctions were found. The kind of the dysfunction was not explained in close detail because the table can not be regarded as a description of individual cases. The quoted structures were not subjected to an evaluation, which describes the severity of the lesion. In the course of the osteopathic treatment much importance was attached to the ranking and the sequences of the treatment.

Patient No. Age, m/f	Occiput C0/C1	Sacrum Pelvis	Sacr. bone	Breast bone	Collar bone	Hip joint	Knee joint	UTJ LTJ Foot	Ribs	Thorac. Diaphr.	GHJ SCJ ACJ	Elb. Hands
1 78, f	x		x	x		X			x	x		
2 73, f		x				x				x	x	
3 62, f			x		x			x			x	
4 75, m	x	x		x		x					x	
5 67, f	x		x	x	x	x	X			x		
6 76, m	x		x							x		
7 38, f	x	x	x	x				x	x	x	x	
8 70, m		x	x								x	
9 63, f			x	x	x						x	
10 60, m				x	x	x			x	x		
11 69, f	x			x	x	x					x	
12 63, m			x	x		x		x	x	x		
13 50, f	x			x	x						x	
14 76, m	x		x	x			X			x	x	
15 75, m					x			x	x	x	x	
16 61, m		x		x	x	x					x	x
17 72, m	x	x	x	x	x				x	x	x	
18 64, w	x		x	x					x			
19 79, m	x			x	x		X				x	

Tabelle 3

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