Donau-Universität Krems

The Influence of Osteopathic Treatment on Postpuncture Complaints

Master – Thesis

to obtain the degree

Master of Science of Osteopathy

submitted by

Alexandra Stephanides

November 2006

Translation: Mag. Barbara Schnürch

Declaration of Academic Honesty

I hereby declare to have written this Student Research Project on my own, having used only the listed resources and tools.

_____ _____

Location, Date Signature of Student

Acknowledgements

I would like to thank Professor Franz Reisecker, who made it possible for me to carry out the study at the hospital of the “Brothers of Mercy” in Graz-Eggenberg.
In addition I would like to express my gratitude towards the senior and assistant physicians at the neurology department for referring numerous patients to me.

I am also very grateful to my parents who have supported me throughout my training as an osteopath.

Robert Felsberger was very helpful in the statistic analysis of the data and in technical emergencies, for which I am very grateful.

Finally, I would like to thank my good friend Monika Zimmer for her support and help during the work on this paper.

Abstract

The aim of the study was to evaluate the influence of osteopathic treatment on postpuncture complaints and how the treatment can possibly shorten their self-limitation period.
Due to time constraints it was not possible to include more than 21 patients in the study, which means that no claim of statistic significance could be made also due to the fact that the patients' statements concerning the intensity of the perceived pain were very varied. However, a clear trend in the two groups could be observed.

Within five days a relative mean reduction of the headaches intensity by 56% could be observed in the TG, while in the CG the pain's intensity was reduced by 29%.
In the TG the relative mean change in the time until the onset of the headaches after adopting an upright position was 13% higher than in the CG.
The intensity of the relative mean nausea in the TG on the fifth day ranged 64% below the initial value, in the CG it increased by 5% with regard to the first day.

These clear trends illustrate that an osteopathic treatment of patients with postpuncture complaints makes sense.

List of Contents

List of Figures

List of Tables

1. Introduction

I work in a hospital where lumbar punctures for diagnostic reasons are regularly on the agenda.
It is not uncommon that therapy sessions have to be cancelled because the patients develop headaches, often in connection with nausea, after such a lumbar puncture, which are so severe that the patients cannot even get out of their bed. Usually the complaints last for several days, where the patients obviously suffer, are limited in their mobility and loose important treatment time. Often the date on which the patients were supposed to be discharged from hospital has to be postponed until the postpuncture complaints subsided.
The standard medical intervention consists in recommending bed rest, to drink sufficiant quantities and the administering of pain killers.
It can be proven that postpuncture headaches are self-limiting, i.e. in most cases they improve except for a few exceptional cases without any therapeutic intervention.

Since a lumbar puncture represents an intervention in the cranio-sacral system and has an effect on the whole organism, my hypothesis is that an osteopathic treatment, especially cranio-sacral techniques, can shorten the period until the complaints disappear.
To verify my hypothesis I have carried out a clinical study with 21 test persons. The patients in both groups received allopathic care. The patients in the treatment group received two additional osteopathic treatments.
The complaints were evaluated by means of questionnaires.

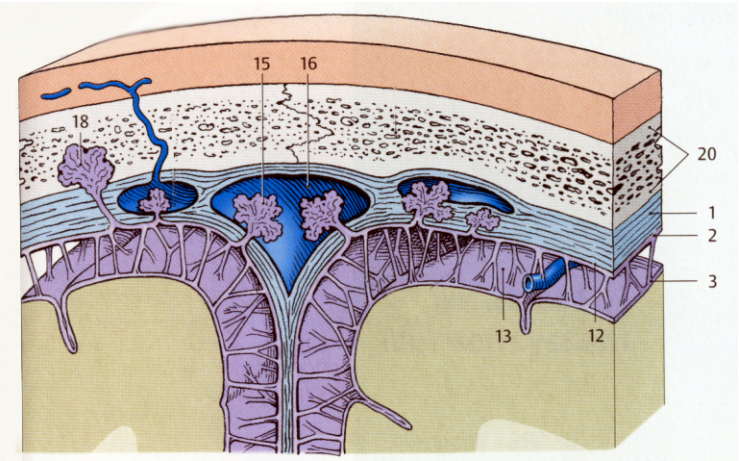
2. Anatomical and physiological considerations

Headaches can be caused by irritations of structures in the cranium due to various pathophysiological processes. The Dura mater in the region of the base of the skull, the Tentorium cerebellli and certain vessels, arteries and sinuses are particularly pain-susceptive structures.
A major role in the clinical picture of “postpuncture headaches” can be attributed to the production and re-absorption of cerebrospinal fluid, the venous drainage and the influence of the vegetative nervous system. [9].

2.1. Cranial and spinal meninges [14, 25, 33]

2.1.1. Cranial meninges

The brain is enveloped by meninges, which are called Dura mater (most external layer), Arachnoidea (intermedial layer) and Pia mater (most internal layer) (Fig. 1).



1 Dura mater
2 Arachnoidea
3 Pia mater
15 Pacchioni-Granulationes
16 Sinus sagittalis superior
20 Os frontale

Figure 1 *Cranial meninges (Kahle 2001, p. 289)*

The **Dura mater** consists of two *layers*:

- Dura periostale: it directly covers the cranial bones and is loosely attached to them in the area of the vault but quite firmly attached to the bones in the area of the cranial base and openings.

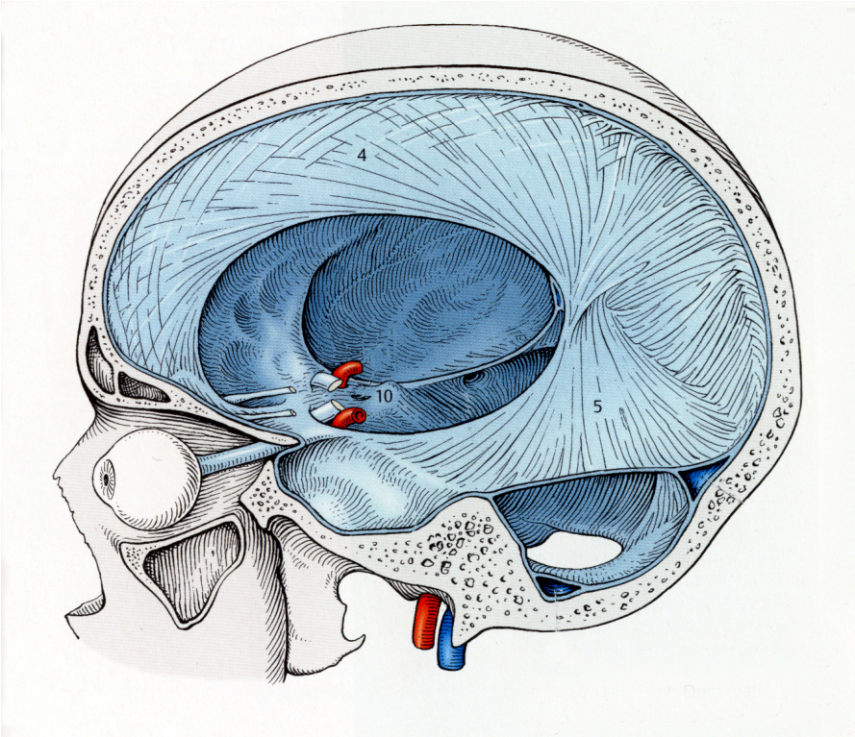
- Dura meninegeale: is closely connected to the external layer of the dura, but becomes detached at some sites to form the venous sinuses and the *septums of the brain* (Fig.2). These are:

  - The Falx cerebri which separates the two hemispheres of the cerebrum. Its anterior pole of attachment is the Crista galli of the ethmoid. It runs along the Crista frontalis, the margins of the superior sagittal sinus, the Crista parietalis and the Sulcus sagittalis occipitalis to the Protuberantia occipitalis interna.
    Its fixed border on the cranial vault forms the superior sagittal sinus; its lower loose border forms the inferior sagittal sinus.

- The Tentorium covers the cerebellum like a tent and separates it from the cerebrum. Where the two dural layers separate the straight sinus is formed. Posteriorly the Tentorium is attached to the Protuberantia occipitalis interna, from which the Sulcus sinus transversus runs laterally on both sides. The Tentorium continues over the Sutura parietomastoidea and is firmly attached to the posterior angles of the Os parietale, and on the Processus mastoideus temporalis. From the latter it runs along the Margo superior partis petrosae, where it forms the Sinus petrosus superior.
  The external border of the Tentorium is attached ventrally on the Procc. clinoidei posteriores, the internal border crosses over the external border and attaches to the Procc. clinoidei anteriores.

- The Falx cerebelli separates the two halves of the cerebellum and has its origin at the undersurface of the Tentorium. It is attached to the Protuberantia occipitalis interna and to the Crista occipitalis interna. The Falx cerebelli participates in the formation of a fibrous ring and continues in the spinal canal in the form of the Dura mater spinalis.

- The Diaphragma sellae covers the Sella turcica and fuses with the Dura mater at its lateral borders. It surrounds the pituitary gland and also has a connection with the anterior poles of attachment of the Tentorium.



4 Falx cerebri
5 Tentorium cerebelli
10 Diaphragma sellae

Figure 2 *Septums of the brain (Kahle 2001, p. 289)*

The septums of the brain have above all a mechanical function. They offer resistance against mechanical impacts, protect the brain and stabilize its position.

After leaving the base of the skull the Dura meningeale changes into the epineurium of the cranial nerves.

Like the Dura mater also the **Arachnoidea** consists of two layers. The external layer has close contact to the Dura without being fused with it; instead it forms the subdural space. Veins and nerves are located in this subdural space.
The inner layer forms numerous small trabercles.
Via the pacchionian granulations the Arachnoidea has a connection to the diploic veins in the cranial bones and via the Granulationes arachnoideae (endothelium) it is connected especially with the superior sagittal sinus and thus the blood circulation.

The **Pia mater** is the innermost meningeal layer which contains vessels and consists of a thin layer of connective tissue with a high share of elastic fibres, which allows that it can match and follow the convolutions of the brain.

The subarachnoidal space lies between the Pia mater and the Arachnoidea. The two meningeal layers are connected by trabercles and septums. The subarachnoidal space is called external CSF (cerebrospinal fluid) space; it also forms the cisterns of the cranium. The bridging veins emanating from it open out into the superior sagittal sinus.
In addition the Pia mater forms the choroid plexuses in the ventricles.

The **Innervations of the cranial meninges** (Fig.3) is guaranteed by several meningeal nerves. The trigeminal nerve supplies the dural layers in the superior regions of the cranium, while the meninges in the inferior regions of the cranium are innervated by the vagus and the spinal nerves 1-3. All meningeal nerves come from the superior cervical ganglion, whose postganglionic fibres follow the Pl. caroticus internus, the Pl. maxillaris and the A. meningea media.

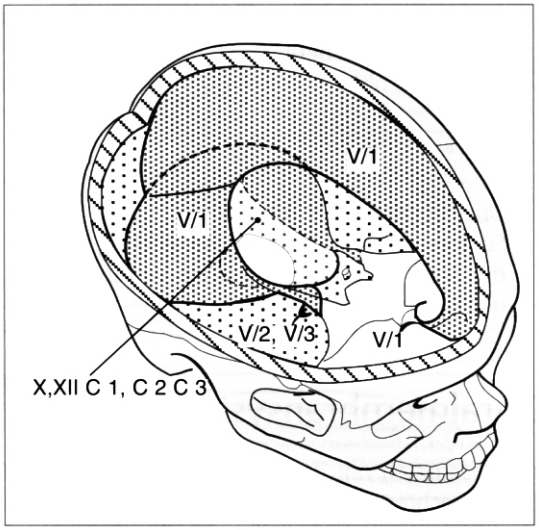


Figure 3 *Innervations of the cranial meninges (Liem 1998, p. 183)*

The parasympathetic supply of the meninges is guaranteed by the N. petrosus major (VII), and the N. vagus as well as the N. glossopharyngeus.

The **arterial supply** of the cranial meninges comes from branches of the A. carotis interna and externa.
The anterior cranial fossa is supplied by the A. meningea anterior, a branch of the A. ethmoidalis anterior.
The middle cranial fossa receives blood from the A. meningea media, a branch of the A. maxillaris.
The posterior cranial fossa is fed by the A. meningea posterior, which directly branches off the A. carotis externa.

Venous blood drains via the Vv. meningeae into the pterygoid plexus and further into the V. maxillaris and the V. ophtalmica inferior before it reaches the cavernous sinus and the jugular vein. The meningeal veins can also directly drain into the jugular vein.

2.1.2. Spinal meninges

Caudally to the Foramen magnum the intracranial dura mater (especially the Falx cerebelli) continues in the region of the spinal canal in the form of the Dura mater spinalis. In the spinal canal the dura is attached posteriorly at the vertebral bodies of C_1 (not always), C_2, C_3, and S2 and is attached to the Os coccygis. Caudal to $S_{2/3}$ the Dura mater spinalis narrows to form the Filum terminale; it goes through the Hiatus sacralis to fuse posteriorly with the periosteum of the Os coccygis.

From the most external to the most internal structure the spinal canal consists of the following layers or cavities (Fig. 4):

- Dura mater spinalis: It consists of a thin external layer, the periosteum of the spinal canal, and a thick internal layer, which forms the sac of the external CSF space.
  The *epidural space* can be found between the two layers. It contains adipose tissue and the Plexus venosus vertebralis internus.
  In the region of the spinal nerves the Dura mater spinalis forms the *Recessus durae matris spinalis*. After the nerves exit through the intervertebral foramen the spinal dura is continuous with their epineurium.

- Subdural space: It is a capillary gap, which physiologically is no space (the space only becomes evident in the case of a subdural haemorrhage).

- Arachnoidea: It accompanies the spinal nerves to the spinal ganglion where it is continuous with the perineurium.

- Subarachnoidal space: contains the cerebrospinal fluid.

- Pia mater: Directly covers the spinal cord and contains nerves and vessels.
  Between the anterior and posterior horn the *Ligg. denticulata* (thin plates of connective tissue) emanate from the Pia mater in the region from the cervical spinal cord to the middle lumbar spinal cord. They continue laterally to the Dura mater and provide a free floating suspension of the spinal cord in the dural sac as well as its stabilization in the frontal plane.

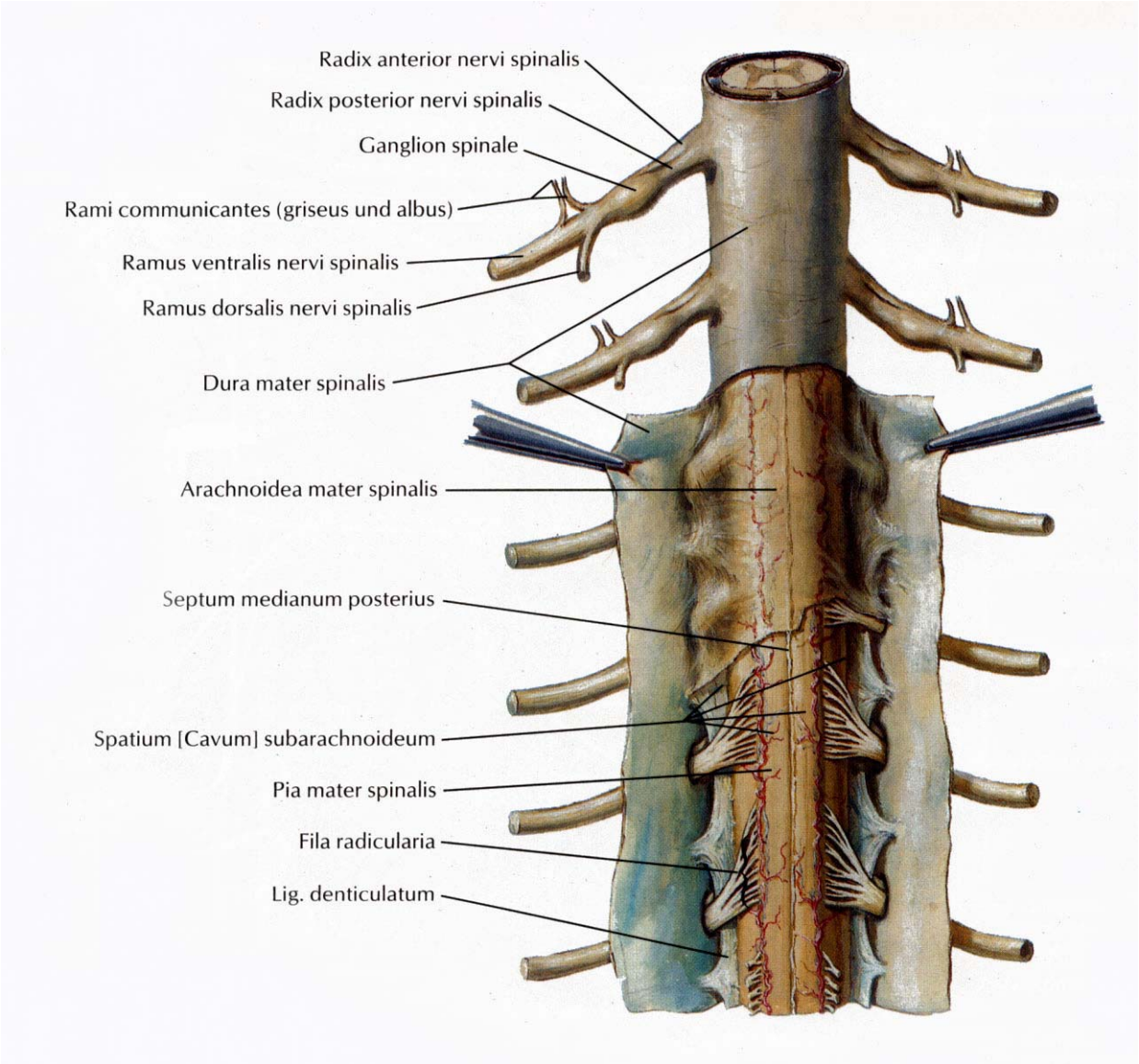


Figure 4 *Layers of the spinal meninges (Netter 2000, p. 155)*

Due to the fact that the spinal cord is relatively shorter in comparison to the spinal canal the Cisterna lumbalis is formed caudally to the Conus medullaris (at the level of L_2). It is filled with CSF and reaches down to the end of the dural sac at the level of $S_{2/3}$.

The **Innervations of the spinal meninges** is guaranteed by the R. meningeus of the spinal nerves, the nervous plexus of the Lig. longitudinale posterius and perivascular nervous plexuses of the radicular arteries.

The **arterial supply** of the spinal meninges is effected by the Aa. spinales posteriores (from the A. inferior posterior cerebelli or the A. vertebralis), which again form Rami spinales, and the A. spinalis anterior (from the A. vertebralis).

The **venous drainage** occurs via the Pl. venosi vertebrales interni anteriores and posteriores in the epidural space. They open into the Vv. lumbales ascendentes, the V. azygos and V. hemiacygos.

2.2. The CSF system [14, 25, 33]

2.2.1. CSF spaces

The cavities filled with cerebrospinal fluid (CSF) can be divided into:

- intracranial internal CSF spaces (ventricles)
- intracranial external CSF spaces (cisterns) and
- extracranial CSF spaces (subarachnoidal space of the spine).

MARTINS, WILEY and MEYERS [38] describe the dural sac as elastic structure which contracts or even collapses when the volume of the CSF is reduced. It forms a reservoir for the CSF and plays a major role in the dynamics of changes in CSF pressure.

2.2.2. Cerebrospinal fluid (CSF)

The CSF is a clear liquid with a low content of protein and cells, consisting of 99 percent water.

It is responsible for

- Nutrient supply of the CNS
- Hydraulic protection of the CNS and the spinal cord
- Absorption of waste products of the neural metabolism
- Carrier of hormones and their preliminary products
- Immune defence and the
- Reduction of the weight of the brain.

For STILL [53] and SUTHERLAND [58] the CSF was very important for the physiological-chemical balance of the whole organism.

2.2.3. CSF production

The production of CSF occurs mainly in the choroid plexus, a network of blood villi, which originate from the Pia mater and cover the walls of the ventricles. The capillary loops can be found

- in the lateral ventricles at the floor of the Pars centralis and the lateral walls of the Cornu inferiores
- in the roof of the 3rd ventricle and
- in the roof and to a small extent also in the floor of the 4th ventricle.

By filtering plasma from blood the external layer of the choroid plexus produces an ultrafiltrate. Through various active metabolic processes the inner layer (astro and oligodendroglia) produces and secretes CSF from this ultrafiltrate.

How much CSF is produced is controlled by the autonomous nervous system. Orthosympathetic fibres, which come from the superior cervical ganglion and provide the sympathetic and parasympathetic innervations of blood vessels as well as the epithelium of the choroid plexus, have an influence on the production of CSF.
This means that an increased sympathetic tone entails a 30 percent reduction in the production of CSF, while an increased parasympathetic tone leads to a 100 percent increase in the production of CSF.

The choroid plexus is also the site of the blood-brain-barrier. The choroid plexus is connected with the capillaries by “tight junctions” of the endothelium (“leaky” epithelium). There is a permeability barrier for water-soluble substances; the Membrana limitans gliae perivascularis prevents the entry of larger molecules, which guarantees a strong selection of the blood in the secretion of CSF. On the other hand, the barrier which controls the passage of the CSF into the blood is quite permeable.

The blood supply of the choroid plexus is assured by the A. carotis interna and the A. cerebri posterior.

To a small extent CSF is also secreted at the capillaries of the Pia mater in the cranial and spinal subarachnoid space. Also at these sites blood plasma is the base for the CSF production, which is effected through diffusion and filtration by the cell membranes.

Every day about 500 ml of CSF are produced. This means that the overall volume of about 150 ml CSF (approximately 20 ml in the subarachnoid space of the spinal cord) is exchanged three times per day.

2.2.4. Re-absorption of CSF

Most of the CSF is re-absorbed into the blood circulation by the pacchionian granulations. They are arachnoidal villi filled with CSF and opening into the lumen of the venous sinuses (esp. the superior sagittal sinus) and the diploic veins of the cranial vault [14].
The re-absorption of the CSF at the arachnoidal villi is pressure-dependent. The higher the pressure, the more CSF is re-absorbed. This mechanism also regulates the pressure of the CSF [33].

In addition, CSF is also re-absorbed at the walls of the capillary vessels of the Pia mater, which open into the peridural veins, and the dural pouches of the cranial and spinal nerves, which communicate with the lymphatic system [33]. The CSF can reach all extracellular spaces through intratubular circulation in the collagen of the fascias [1]. SPERANSKY (1943) could prove the connection of the CSF space of the spinal cord with the lymph nodes of the thorax, abdomen and pelvis by means of experiments with ink [51].
About 10 percent of the CSF is re-absorbed at the choroid plexus [33]
The existence of re-absorption sites other than the pacchionian granulations is corroborated by the fact that the pacchionian granulations are not yet formed at the moment of birth. In older age the granulations are more numerous, richer in fibres and larger. Maybe this is the body's answer to the decreasing quality of collagen in older age [4].

2.2.5. CSF circulation [33]

From its production sites in the lateral ventricles, the choroid plexus and the capillaries of the Pia mater the CSF flows into the third ventricle via the Foramina interventricularia (Monroi) and further into the fourth ventricle via the Aquaeductus mesencephali.

Via the Aperturae laterales and mediales (Luschka and Magendie) the CSF reaches the external CSF space and the subarachnoidal space of the cranium and spine.

This circulation is also possible in the opposite direction, i.e. from the extracranial CSF spaces back towards the lateral ventricles (Fig. 5).

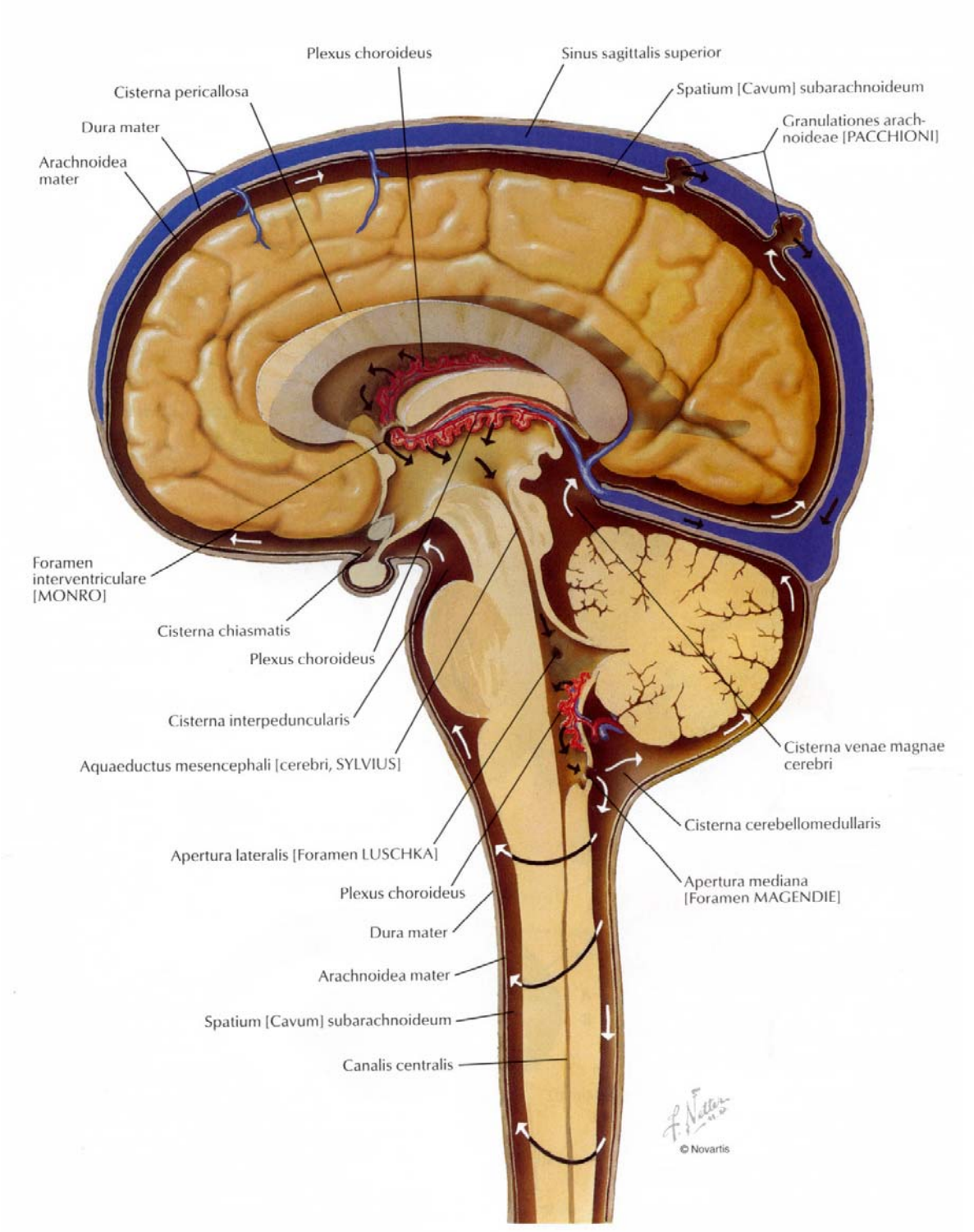


Figure 5 *CSF circulation (Netter 2000, p. 103)*

2.3. Venous system [33]

The pathways for the drainage of venous blood in the cranium are formed by the two layers of the dural membrane, with the Dura periostale forming the roof and the Dura meningeale the walls.
The special features of theses atypical venous pathways are that they do not have any valves, no tone of surrounding muscles and non-elastic walls.

95 percent of all the venous blood in the cranium (brain and meninges) is transported through these pathways.

In a vertical, upright position of the body the venous drainage is supported by the forces of gravity, the arteriovenous pressure and by the Primary Respiration Mechanism (PRM); when the body is in a horizontal position the PRM and the reciprocal tension of the dura support the drainage.

The superficial venous system is fed by the veins of the surface of the brain. The so-called bridging veins run through the subarachnoid space to drain into the sinus system, esp. the superior sagittal sinus.

The profound venous system is formed by the superior sagittal sinus, the occipital sinus and the straight sinus (receives blood from the inferior sagittal sinus and the Vena magna), which all open into the confluens of sinuses.
Further, the cavernous sinus drains into the superior and inferior petrosal sinuses, the latter of which also receives blood from the basilar plexus.
The confluens of sinuses opens into the transverse sinus and the sigmoid sinus, which also receives blood from the superior petrosal sinus.
After passing the Bulbus superior venae jugularis the blood reaches the jugular vein which also receives blood directly from the inferior petrosal sinus (Fig. 6).

If there is a need to compensate the venous blood can also drain from the sigmoid and occipital sinuses into the internal and external vertebral plexuses [14]. The cavernous sinus and the ophthalmic veins have a connection with the facial veins.
The blood can also drain via the Foramen ovale to the Plexus pterygoideus and further via veins in the Canalis caroticus [33].

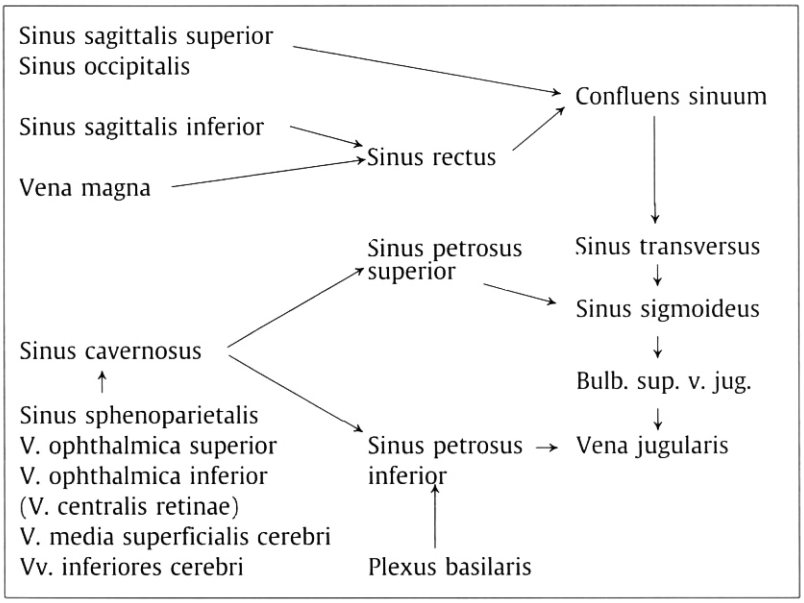


Figure 6 *Drainage of venous blood in the cranium (Liem 2000, p. 209)*

2.4. Autonomous nervous system [25]

As already mentioned above, the production of CSF is controlled by the vegetative autonomous nervous system. An increased parasympathetic activity provokes an increased production of CSF.

The central parasympathetic system is located on the one hand in the brainstem, where the nerve cells form the Ncl. Edinger-Westphal, the Nuclei salivatorii and the Ncl. dorsalis n. vagi. On the other hand the sacral cord contains parasympathetic nerve cells (Ncl. intermediolateralis and Ncl. intermediomedialis). This means that the parasympathetic neural nuclei are located in the cranium as well as in the sacrum.

Besides an increase in CSF production an increased parasympathetic activity also provokes an increase of intestinal motility and secretion, it promotes the defecation and elimination processes and it slows down the heart rate and respiration. Overall these are activities which serve the body's regeneration and the building up of physical reserves.

The main nerve of the parasympathetic nervous system is the N. vagus, which passes through the thoracic inlet together with the large cervical vessels. After its division it expands like a plexus in the thoracic and abdominal regions.

From the sacral cord the parasympathetic fibres pass through the third and fourth sacral root to the N. pudendus and further to the Nn. pelvici and the Pl. hypogastricus inferius, which supplies bladder, rectum and genitalia.

These anatomical facts provide the base for the hypothesis that postpunctural complaints may be influenced by osteopathic techniques, because the pathophysiological factors include structural lesions and abnormal tension in the regions of the cranium and sacrum as well as the pelvic floor and the thoracic inlet.

2.5. Pathophysiology of headaches

As already mentioned, the pain-sensitive structures within the cranium include the Dura mater, the intracranial sinuses, arteries and veins. They react to pressure or traction, which cause an afferent input of the N. trigeminus and N. vagus [33].

2.5.1. N. trigeminus [16, 25, 33]

The Radix sensoria of the N. trigeminus leaves the Pons and runs anteriorly through the Pars petrosa ossis temporalis, where the trigeminal ganglion is located in a dural pouch, the Cavum trigeminale. There the nerve splits into its three main branches: the N. ophtalmicus, the N. maxillaris and the N. mandibularis.

All three branches of the N. trigeminus are involved in the perception of pain of the meninges.

The Nervus ophtalmicus with :

- the Rami meningei of the Nn. ethmoidales (V1) and the R. meningeus n. ophtalmici especially for the Falx cerebri and
- the Ramus tentorii nervi ophtalmici. Right after the Ganglion trigeminale it truns round and passes back into the cranial cavity where it innervates the Tentorium and the dura of the posterior cranial fossa.

The Nervus maxillaris with:

- the Ramus meningeus nervi maxillaris and
- the Ramus meningeus medius nervi maxillaris. Before the N. maxillaris passes through the Foramen rotundum the Ramus meningeus medius nervi maxillaris branches off and runs to the Dura mater of the lateral wall of the cranium.

The Nervus mandibularis with:

- the Ramus meningeus nervi mandibularis. After the N. mandibularis passed through the Foramen ovale the Ramus meningeus nervi mandibularis branches off and runs though the Foramen spinosum back into the cranial fossa.

Primary afferences of the fifth cranial nerve from the Dura mater and the vessels end in synapses in specific nuclei of the thalamus. Fibres of prothopathic sensation (strong pressure, pain, temperature) terminate in particular in the Nucleus spinalis n. trigemini. There they are redirected to the third neuron which relays the input to the limbic system (perception as unpleasant experience) and to the cortex (interpretation of pain).
Certain fibres of the Nucleus spinalis run as Tractus spinalis to the upper cervical cord, where they supply the skin of the face in somatotopic order.

2.5.2. N. vagus [25]

The Ramus meningeus nervi vagi passes through the jugular foramen back into the cranium, where it provides the sensory supply of the Dura mater of the posterior cranial fossa.
These exteroceptive fibres originate in the Ganglion superius and – together with the spinal root of the N. trigeminus – run down to the Nucleus spinalis n. trigemini, where they end.

2.5.3. Trigeminocervical pain complex

The upper cervical cord contains prolongations of the meningeal pain-sensitive fibres via the Tractus spinalis n. trigemini, but also from the supply area of the upper cervical spinal roots, esp. the N. occipitalis major (C_2), which supplies the skin and muscles of the back of the head [6, 50] (Fig. 7).

This means that at the level of the spinal cord primary afferent neurons of the spinal nerves of C_{1-3} converge in joint secondary neurons with primary afferences of the back of the head and trigeminal afferences [19].
A stimulation of the Dura mater can provoke a referred pain and hyperalgisia in the region of the neck [5] and a secondary but pronounced restriction in the mobility of the cervical spine [9].

If the proprioceptive input is strong enough, the coupling of menigeal and cervical afferences in the upper cervical cord can cause a central sensitization and a mutual maintenance or increase of the complaints in the regions of the head and cervical spine [6, 50].

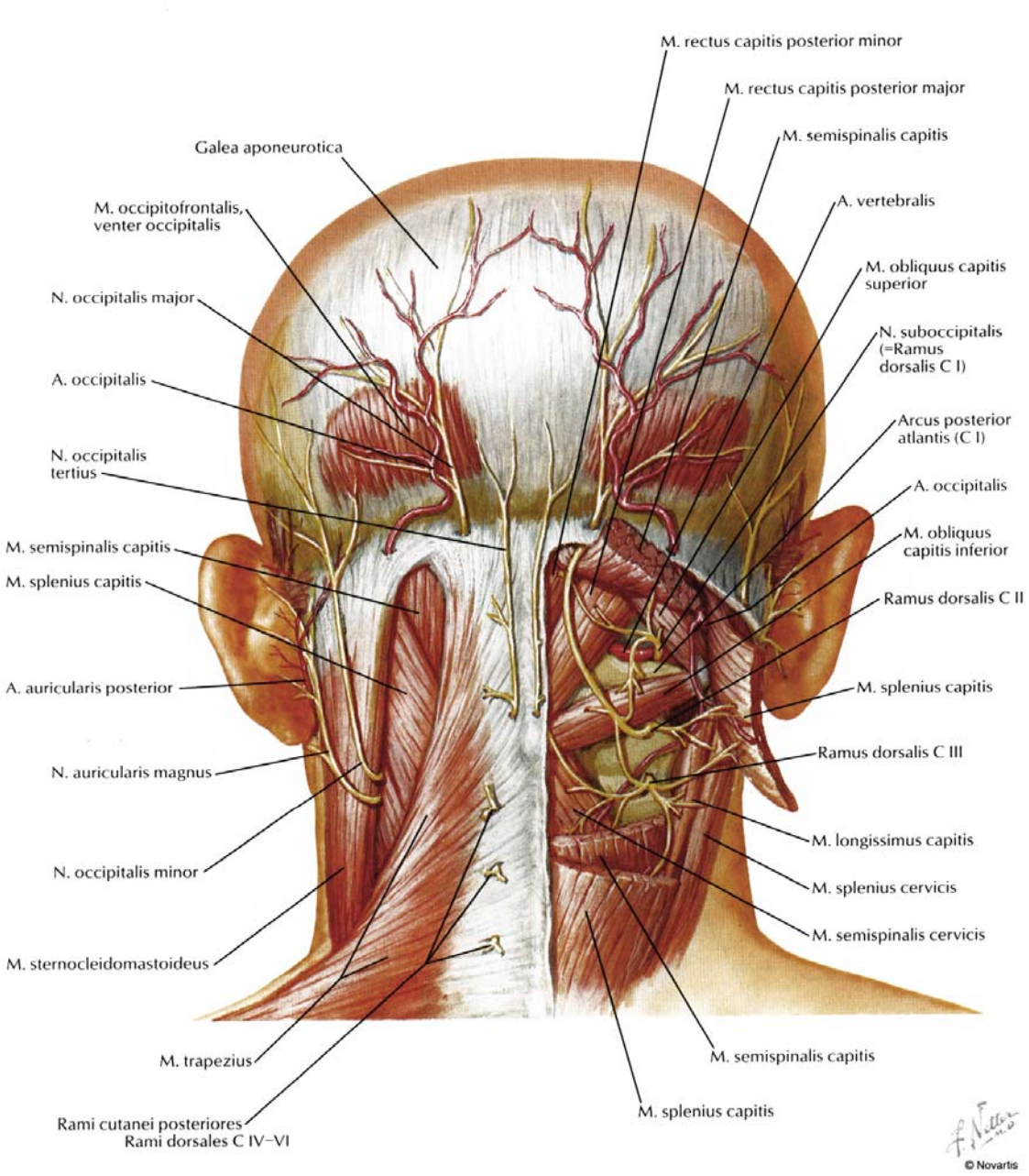


Figure 7 *Suboccipital muscles and innervations (Netter 2000, p. 164)*

A direct link between afferent inputs form the meninges and the spinal supply areas of the nerve roots $C_{1\text{-}3}$ results from the fact that the first three spinal nerves are also the origin of the Rr. meningei, which supply the Clivus [33].

The irritation of trigeminal nerve endings above the Tentorium can provoke frontal headaches or pain in the first trigeminal and cervical dermatoms. An irritation below to the Tentorium can cause occipital headaches [6, 50].

2.6. Diagnostic lumbar puncture (LP)

In a medical context we talk about a lumbar puncture (LP) in cases where the Dura mater is pierced to reach the epidural space.

The first LP was carried out by QUINCKE in 1891 [45]. He was the first to determine the amount of protein and the number of cells in the CSF and to propose to measure the CSF pressure [59].

Today the "Practice Parameters of the American Academy of Neurology" are considered as the standardized guidelines for diagnostic lumbar punctures [13].

2.6.1. Indications [32]

On the one hand punctures of the epidural space are carried out in the course of epidural anaesthesias and to measure the CSF pressure or as a therapeutic intervention in cases of hydrocephalus, intraventricular or subarachnoidal haemorrhages.

On the other hand the puncture of the subarachnoidal space can also serve diagnostic purposes to obtain CSF for clearly identifying inflammatory, septic or infectious diseases of the CNS.

A lumbar puncture is <u>contraindicated</u> in cases of

- focal neurological symptoms
- signs pointing towards an increased intracranial pressure (risk to pinch cerebral tissue in case of lumbar relief)
- superficial or deep inflammatory reactions of the skin or hypodermis or an inflammation of the muscles at the site intended for the puncture
- existing disorders in blood coagulation or therapeutically induced anticoagulation.

2.6.2. Instruments

Several versions of puncture needles can be used for the procedure of an LP. Basically we differentiate between traumatic and atraumatic needles (Fig. 8).

An example for a traumatic needle would be the Quincke needle, which has a bevelled cutting-tip. It severs the dural fibres and leaves an elliptical hole (Fig. 9, needle a).
The Sprotte needle would be an example for an atraumatic needle. It has a blunt end with an ogival form (pencil point tip) and also differs from the Quincke needle through the larger size of its oval aperture proximal to the tip of the needle. In addition, it can be introduced right to the Dura mater with a gauge introducer (contains the mandarin cannula); afterwards a thinner needle can penetrate the dura. Due to the blunt end atraumatic needles do not sever the dural fibres but gently separate them (Fig. 9, needle b). After the cannula is removed the fibres should close again [59].

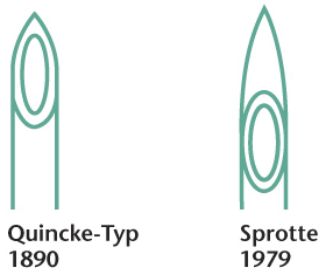


Figure 8 *Puncture needles (PAJUNK© Spezial-SPROTTE©- Kanüle. (2005) www.pajunk.com)*

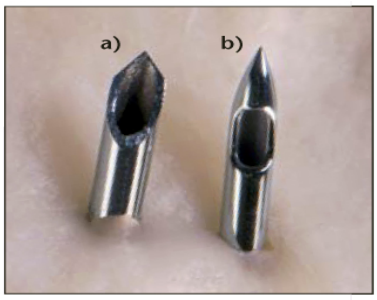


Figure 9 *Punctue needles (PAJUNK© Spezial-SPROTTE©- Kanüle. (2005) www.pajunk.com)*

Puncture needles can also be differentiated according to their diameter.
Needles which have a smaller diameter reduce the risk of postpuncture headaches but they prolong the process of the LP because of a lower flow rate [13]. In addition they are more difficult to handle for the practitioner [60].
The diameter of the needles is indicated in “gauge”, which is the unit of measurement for the external diameter of cannulas: the higher the gauge-number, the smaller the diameter of the cannula [46].

2.6.3. Execution of the procedure

Before a lumbar puncture is carried out all patients are informed about the possible risks and have to declare their informed consent by signing a declaration of consent.

Generally, an LP can be done in a side-lying or sitting position.
All patients participating in this study underwent the procedure in a sitting position. The patient sits on the treatment table with the feet supported and the forearms propped up on his/her knees, so that the lumbar lordosis is eliminated as much as possible.

The doctor who will carry out the procedure palpates L_4 above the iliac crest and marks the intervertebral space below with his/her finger nail. Afterwards the puncture site is disinfected.

With sterile gloves the doctor takes a sterile needle out of its packaging. He/she stabs the spinal needle with mandarin though the skin into the subcutaneous tissue. The cut of the needle should be oriented parallel to the longitudinal axis of the spine and the direction of the dural fibres, i.e. the aperture of the needle faces laterally and the puncture canal runs slightly cranially. First the needle goes through subcutaneous adipose tissue to then penetrate the Lig. flavum. When the Dura mater is pierced the practitioner can feel a loss of resistance when he/she pushes the needle in further. Now the tip of the needle is in the subarachnoid space (Fig. 10, Fig. 11).

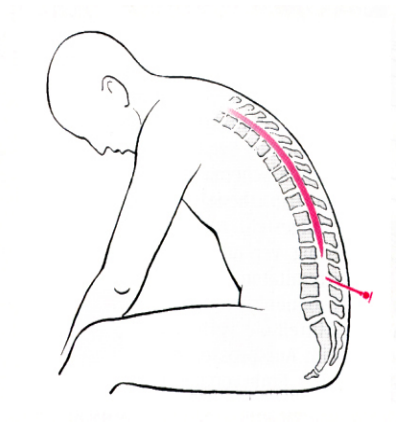


Figure 10 *Lumbar puncture (Kahle 2001, p. 65)*

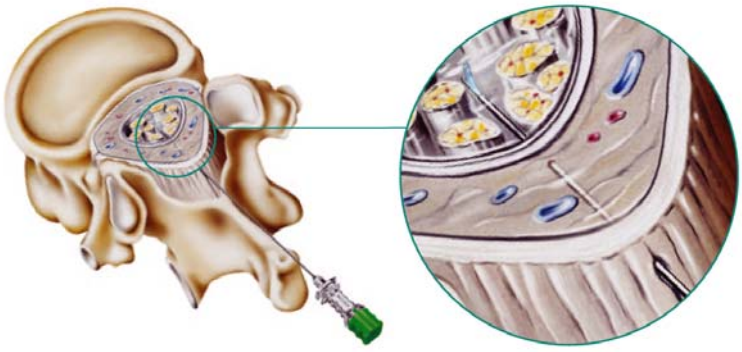


Figure 11 *Lumbar puncture (PAJUNK© SPROTTE©- Kanüle. (2004) www.pajunk.com)*

If the positioning is right, CSF will spontaneously drip from the puncture cannula after the removal of the mandarin. If enough CSF (usually about 10 ml) is obtained, the practitioner re-introduces the mandarin and removes the spinal needle.
After executing a slight pressure on the puncture site, a sterile band aid is placed over the site.

2.6.4. Aftercare

In the hospital where I carried out my study the patients were advised to lie flat on their stomach for half an hour after the procedure, to drink enough liquid and to observe bed rest for the rest of the day if possible.

2.7. Presentation of the clinical picture

2.7.1. Definition

According to the International Headache Society the postpuncture headaches have the code 7.2.1.

For the first time the postpuncture headache was described by BIER in 1898. He administered himself and eight other persons an injection of cocaine into the subarachnoid space. Four of the test persons described a postpuncture headache [59].

The headache occurring after a lumbar puncture is defined as a bilateral, holocephalic headache, which occurs within less than seven days after the LP.
It is an orthostatic headache, i.e. it begins or becomes worse in less than 15 minutes after changing into an upright position and disappears or becomes better in less than 30 minutes after lying down [9].

2.7.2. Incidence

Whether postpuncture headaches occur is highly dependent on the kind and diameter of the puncture needle but also on the skills of the practitioner. Possibly the age, gender and build of the patient play a role as well.

In recent studies the incidence of the occurrence of postpuncture headaches is indicated with 10-30 % [31], 5-20 % [9] and 0-20 % [59].

2.7.3. Epidemiology

Opinions and studies vary as to which factors predispose the occurrence of postpunctural headaches.

The highest incidence of complaints after an LP can be observed in women between 18 and 30 years of age, with a low BMI and already recidivating or chronic headaches before the procedure [13].

While surveys by BÜTTNER et al [7] and KREUSCHNER and SANDMANN [28] show that women are more likely to be affected than men, studies by RASMUSSEN et al [48], QUAYNOR et al [47] and HAFER et al [21] did not observe a gender-specific difference in the occurrence of postpuncture headaches.

Different study results are also available with regard to age. RASMUSSEN et al [48] and STONE and DI FAZIO et al [54] describe a declining rate of postpuncture complaints with increasing age. BÜTTNER et al [7] found in their study no distinct dependence of the headaches on the age. Also QUAYNOR et al [47] and FRENKEL et al [18] do not mention a dependence of the occurrence of postpunctural headaches on the age in their study results.

Patients with a low BMI show a higher incidence of postpuncture headaches [31] because in very adipose patients the fatty tissue exerts an external pressure on the puncture site and thus possibly provokes an increased CSF production [9].

2.7.4. Pathogenesis

After a lumbar puncture both the outflow of CSF and the prolonged CSF leakage through the persisting dura defect [26] reduce the CSF volume. In 1918 MAC ROBERT described for the first time that this leaking of CSF is a decisive factor in triggering a postpuncture headache [36].

In the region of the lumbar spine the Dura mater consists of several layers, which have a variable thickness of 0.5 to 2 mm at different levels [15, 29] in every individual. If the dura is perforated at a thinner site, the incidence of CSF leakage after an LP is higher [15].

The occurrence of a postpuncture headache depends in particular from the amount of CSF that leaks through the original puncture hole, because there is no direct link between the amount of CSF that is obtained during the puncture and the occurrence of postpuncture complaints [10]. I have also observed that after an LP postpunctural headaches may occur even though no CSF was gathered.

The CSF leakage causes a hypovolemia [61] of the CSF since the amount of CSF that was sampled and the amount that leaked through the puncture hole cannot be replaced at the same time.
Within the framework of a survey MOKRI et al (1997) [41] found a lowered CSF pressure only in 46 percent of the patients with postpuncture headaches. In 1999 they described the state as "hypovolemia" rather than something that had to do with the lowered pressure.
However, DIENER et al (2006) [9] und GRANT et al (1991) [20], disagree with this theory labelling the cause of the problem as "Low Cerebrospinal Fluid Pressure Syndrome".

Due to the reduced CSF volume the brain cannot be most effectively supported by the CSF-filled cisterns ("CSF cushions"). In the upright position its weight thus increases [2, 55]. The effect is reinforced through the fact that in the upright position the intracranial pressure decreases through the drainage of venous blood and the relocation of intracranial CSF towards the region of the spine from 5 – 15 mmHg in the recumbent position [14,27] to 0 – -6 mmHg in standing [27]. At the same time the CSF pressure in the lumbar region increases from 5 - 15 mmHg to up to 40 mmHg [27, 59], which again promotes the leaking of CSF through the puncture site.
Due to the decreasing intracranial pressure in the cisterns in standing and the reduced support of the brain, the brain has the tendency to sag. This leads to a stretching of pain-sensitive structures, like vessels, nerves and meninges, esp. the Falx cerebri and the Tentorium [2, 55].

MRI pictures show several changes in cases of postpunctural headaches.
In 85 percent of the cases a dilatation of the anterior vertebral venous plexus and the cervical epidural venous plexus can be observed, esp. at the level of $C_{1/2}$ [6, 17]. The reason for this could be the compensation of the intracranial and intraspinal volume.

The Monoe-Kellie Doctrine describes that the body contains a constant sum of the volumes of the brain, CSF and intracranial blood. The volume of the brain amounts to 80 percent, that of the CSF to 10 percent and that of the blood also to 10 percent of the overall intracranial volume. When the CSF volume decreases the volume in the blood vessels increases [40, 55]. This dilatation also has an influence on the nociceptors in the Dura mater which can be a reason for the headaches.

The MRI also shows a caudal shift of the cerebellum in the posterior cranial fossa, which again could impair the venous drainage and lead to intracranial congestions [44]. This would provoke a stretching of pain sensitive structures through traction and stasis of the venous blood.
However, there are also surveys where MRIs of patients with postpuncture headaches did not show a caudal shift of the brain mass [61].

2.7.5. Clinic

The postpuncture headaches are described as dull and pulling, profound, but sometimes also as pulsating pain or feeling of pressure [23].
Most of the times it is located in the frontal and/or occipital regions [10].

Movements of the head and coughing make the pain worse [59].

Accompanying symptoms can occur in the form of stiffness and pain in the neck, nausea, retching, dizziness and gait insecurities, tinnitus or reduced hearing and impaired vision (diplopic images) [39, 59].

Most of the affected patients describe the pain as very severe, so that they are not able to cope with everyday life or activities of any kind.

In 90 percent of the cases the postpunctural headache occurs within 24 – 48 hours [9] or within 72 hours [30, 34] after the LP.

LYBECKER et al [34] indicate that the period of self-limitation in 80 percent of the cases is about 5 days. Other authors report that the headache disappears after 6 days in 85 percent of the cases [59], or after up to 14 days in 100 percent of the cases [9]. VANDAM and DRIPPS [60] specify the figures of spontaneous remission with 72 percent within

7 days and 87 percent within 6 months. COSTIGAN and SPRIGGE [8] and MACARTHUR et al [37] mention that the period of self-limitation of the postpunctural headaches in 85 percent lies within the period of 6 weeks. In the most severe documented case of postpunctural headaches the pain persisted for a period of 8 years [37].

In addition, it seems that an early onset of the complaints worsens the prognosis [15].

2.7.6. Prophylaxis

Atraumatic needles (since they only separate the dural fibres) [42] and needles with a small diameter (smaller perforation hole) seem to clearly reduce the incidence of postpuncture headaches [13, 57]. REINA et al [49] talk about the theory that the use of atraumatic needles can overstretch the dural fibres and cause a tearing of the fibres, which can lead to a faster closing of the perforation hole due to the inflammatory reaction.
Also the re-introduction of the mandarin seems to reduce the amount of CSF which continues to leak through [56].
When traumatic needles are used the cut should be directed parallel to the orientation of the fibres of the most superficial layer of the Dura mater [13, 35].
In addition, the needle should be introduced in an acute angle to the dural sac. This guarantees that the perforation holes of the Dura mater and the Arachnoidea are at different levels, and that a region of intact tissue lies between the two puncture holes [15].

No significant results are available concerning the bed rest in prone or supine position immediately after the LP. It seems that this measure only prolongs the latency period until the complaints occur, but does not reduce the incidence [12, 22, 52].
Concerning the effectiveness of increased intake of liquids or abdominal bandages to increase the pressure on the puncture hole in the Dura mater no clear statement can be made [11].

2.7.7. Diagnosis

The diagnosis “postpuncture headache” results from the clinic (case history and findings) after an LP.
In the case of persisting headaches an MRI can help to establish a diagnosis.

2.7.8. Conventional therapy

Besides the advice to observe bed rest and to drink sufficient quantities, conventional interventions mainly consist in the administration of pain medication in the form of infusions or tablets.
If required the patients were given common pain killers like Parkemed, Thomapyrin or Novalgin as well as hypertonic saline solution in the form of an infusion.

2.8. Osteopathic approach

In addition to the conventional therapy osteopathic treatment, especially cranio-sacral osteopathy is a good option to influence the effects of an LP and the related intervention in the cranio-sacral system. Through the treatment of the meninges and the autonomous nervous system, as well as by means of improving the CSF production and the venous drainage in the cranium it is possible to directly influence the causes of postpunctural headaches.

2.8.1. Basic osteopathic principles

Alternatively to conventional therapy there is the possibility to support patients suffering from postpuncture headache with osteopathic treatment.

According to the osteopathic considerations the treatment of the complaints after a lumbar puncture seems to be reasonable.
Especially cranio-sacral osteopathy can intervene in the pathomechanism of the disease pattern where the true causes of the complaints are to be searched. Therefore, there is not only the possibility to ease or shorten the acute complaints, but also to reduce long-term consequences of a trauma to the meninges and thus the PRM, which has an influence on the whole body.

If we consider the osteopathic principles, i.e. the “body is a unit”, the “self-healing mechanism”, and the “mutual influence of structure and function”, the “law of the artery” and the “individual treatment of a patient” we find many starting points for an osteopathic treatment for the complaints of postpuncture headaches. [33].

If the clinical picture is fully developed in all its complexity, the body may be over-stressed by additional external stimuli. Since the headaches limit themselves after a certain period of time, the body is obviously able to solve the problem without external help due to its self-healing forces, the recommended bed rest and the intake of a sufficient quantity of liquids. But all surveys carried out so far only looked at the improvement in the acute complaints. So far there are no studies concerning long-term consequences of an LP.

I posed myself the question whether it would pay off to treat the patient on the first day under pressure of time constraints or whether the treatment would be as effective if not more effective if it was carried out at another moment in time (e.g. *before* a planned LP).

There are reports of cases where the patients months or even years after the LP still suffered from headaches [37, 60], where the body obviously was not able to cope with the lesions caused by the LP without external support.

The present study wants to clarify whether it is reasonable to give osteopathic treatment to patients in the acute phase often under the pressure of time constraints of the practitioner and sometimes despite the discomfort of the patient (many patients refuse treatment in the acute phase).

2.8.2. Osteopathic considerations

From an osteopathic point of view a lumbar puncture is a direct intervention in the cranio-sacral system and thus has an effect on the whole organism.
Whether the LP is carried out with a traumatic or an atraumatic needle, the needle leaves a scar in the tissue and influences the mobility and motility of the meninges, the cranium, the sacrum and all tissues related to them. Further, the restriction of motility leads to an impairment of the Primary Respiratory Mechanism (PRM) and the self-healing mechanism of the body.
Especially in cases of postpunctural headaches where not enough CSF is produced to compensate for the CSF lost through the puncture a bad motility of the cranio-sacral system and the PRM, as well as impaired self-healing forces of the body can be observed, which in turn can be a reason for a reduced production of CSF.

If the cisterns are not sufficiently filled with CSF and the brain can thus not be supported so well, the brain tissue sags which causes a drag on the cranial membranes, vessels,

cranial and upper spinal nerves. This drag can possibly be reinforced by a scar in the region of the puncture site (traction due to scar tissue formation). This drag also affects the Arachnoidea, which forms the pacchionian granulations as described above, as well as the Pia mater, which contains the choroid plexuses. Therefore, a direct effect on the CSF production and re-absorption is absolutely imaginable.

A lack in the parasympathetic tone due to stress and possible fear of the LP can cause altered tensions in the cranial and sacral regions and the surrounding structures (localisation of the parasympathetic system), which also reduces the production of CSF by up to 30 percent [33].

According to the Monroe-Kellie-Doctrine the intracranial volume remains constant [40, 55]. An intracranial venous congestion, which on the one hand is provoked by the body to compensate for the reduced CSF volume, and on the other hand is caused by tensions in the Dura mater and the impaired venous drainage from the cranium, can lead the body to believe that it is not urgent to produce CSF. But the stretching of the sinuses by the venous congestion, as well as the drag on the meninges, vessels and nerves can cause pain, which in turn increases the activity of the sympathetic system and the tension of the muscles in the region of the neck and shoulder girdle.

All these factors contribute to a vicious circle: due to the drag on the meninges, a lack of parasympathetic tone and the constant intracranial volume (because of the venous congestion) too little CSF is produced to make up for the loss. The lack of CSF in turn leads to intracranial venous congestions, drag on the intracranial membranes (because the brain is not enough supported) and pain stimuli, which again activate the sympathetic system.

Finally, there are important connections of the cranio-sacral system with the fascial system and thus with the diaphragms, to which abnormal dural tensions are transmitted [33]. The diaphragms can also impair the venous and arterial blood flow through a hypertonic state or reduced mobility. They influence the mobility and tension of the extremities, viscera and the related body functions like respiration or digestion which are even more impaired by the necessary bed rest.

Structural lesions are also linked to the clinical picture of “postpuncture headaches”. The sites of attachment of the Dura mater at $C_{2/3}$, S_2 and the Os coccygis can influence the bony structures, their surrounding and related structures. Via the trigeminocervical

complex there is a direct link between the cranial nerves, the upper cervical region and the suboccipital musculature. All the structural restrictions of mobility and changes in tension have in turn an effect on the cranio-sacral system.

These osteopathic considerations determined the emphasis in the treatment of patients suffering from postpuncture headaches for the purpose of this study.

2.8.3. Applied techniques

The treatment of the patients was limited to the supine position, because the patients could not adopt another position for a longer period of time due to their pain symptoms.

My primary aim was to help the body to step up the production of CSF to compensate for the loss of CSF through the LP and the leakage afterwards.
This gave rise to the necessity to relax the meninges, both the intracranial and spinal membranes, especially at the puncture site. Another focus in the treatment was the increase of mobility and motility of fixed structures, where the Dura mater is attached. This includes the cranial bones, the sacrum, the coccyx and the cervical spine.
Also an improvement of the venous drainage from the cranium is necessary to reduce the tension of the Dura mater in the region of the sinuses and to create more space for CSF volume.
The work on the cranium and sacrum also promotes the activation of the parasympathetic system. The patients were asked to observe the bed rest and to undertake relaxing activities in order to promote parasympathetic activity themselves, and to drink sufficient quantities.
I thought the application of a CV4 technique was also reasonable because this technique optimizes the sympathetic tone and the tension in the connective tissues, and it stimulates the flow and production of CSF.
Finally, I also wanted to check and if necessary correct the tension in the diaphragms because they also have a connection with bony structures and can impair the blood flow into the cranium. In addition, the thoracoabdominal diaphragm is linked to the cervical spine via the N. phrenicus and the trigeminocervical complex.

An important issue in the treatment was the relaxation of the short neck muscles because they represent a direct link between the cranial and structural system.

3. Material and method

In the hospital where I carried out the present study diagnostic lumbar punctures are carried out on a regular basis, especially to determine the presence of multiple sclerosis, borrelian infections or polyneuropathia.

3.1. Patient sample

All patients who suffered from postpuncture headaches after a diagnostic lumbar puncture were included in this study.
No restrictions with regard to age, gender or the reason for the LP were applied.

3.1.1. Miscellaneous

I contacted the patients on the day the postpuncture complaints appeared for the first time. Usually this was the first day after the lumbar puncture was carried out.

Every day of a five day period starting on the day of the first appearance of the complaint both the control group as well as the treatment group completed a questionnaire (cf. Annex).
Both groups were looked after with conventional methods, i.e. if they needed analgesic medication they received pain killers, which was recorded in the questionnaire.

In addition, the patients of the treatment group received an osteopathic treatment on the first and third day after the lumbar puncture.

3.1.2. Division of the patients

The patients, which fulfilled the criteria of the study, were randomly attributed to the treatment and the control group.

3.2. Exclusion criteria

Patients with known migraine or diagnosed psychological diseases were excluded form the study.

3.3. Test person profile

A total of 15 women and 6 men, aged between 18 and 84 years, participated in the study, with 10 patients forming the control group and 11 patients forming the treatment group.
The treatment grop included 8 women and 3 men, the control group 7 women and 3 men.

3.4. Description of the questionnaire

For each patient participating in the study a basic questionnaire was established, and completed together with the practitioner. It contained basic questions concerning the person and the execution of the LP.

The patients themselves had to complete five follow-up questionnaires starting on the day when the complaint occurred for the first time. These included a visual rating scale for headaches, nausea and noise in the ear, as well as questions concerning pain medication.

The design of the questionnaires can be found in detail in the Annex.

3.5. Execution of the treatment

The patients were treated in a quiet room.
In general the treatment session lasted 45 minutes. A global examination (cf. below) of each patient was carried out before each treatment. The treatment answered to the individual needs of the patient and the pathomechanism of their disease pattern.
After the treatment the patients observed a 10 minute period of rest.

3.5.1. Global examination

Before the osteopathic treatment all patients underwent a global examination. This examination was carried out with the patient in supine position, because adopting any other position made the headaches and accompanying symptoms worse.

I undertook

- a global listening on the body and on the cranium

and assessed

- the tension and motility of the diaphragms and the intracranial and intraspinal meninges
- the mobility of the cervical spine
- the tone of the neck muscles and the muscles of the shoulder girdle and
- the quality of the PRM.

3.5.2. Treatment

Osteopathic treatments are always tailored to the needs of the individual patient. The treatment is different for every patient, because every organism has specific needs. Thus in every patient with postpuncture complaints I found different lesions to be treated.

But in all patients suffering from postpuncture headaches I had to

- correct the SBS
- apply cranial techniques on the occiput, the temporal bones, the frontal bone, the ethmoid and the sacrum
- release the tension in the intracranial and intraspinal membranes
- release the tension in the diaphragms (the thoracoabdominal diaphragm, the thoracic inlet and the pelvic floor diaphragm)
- increase the mobility of C_{1-3}
- relax the muscles of the neck and shoulder girdle.

In addition, I treated all patients with a CV4-technique.

3.5.3. Aftercare

After the treatment the patients observed a period of 10 minutes rest.
Further they were asked to try to lie down as much as possible, to enjoy relaxing activities (rest, music, sleep) and to drink sufficient quantities of water.

3.6. Analysis

The data of this study were collected by means of questionnaires and the medical records of each patient.

3.6.1. Questionnaire

For a period of 5 days starting with the day on which the postpuncture headaches first appeared, the patients independently completed a questionnaire for each day. The questions concerned

- the period (minutes) which passed until the headaches started after changing into an upright position
- the intensity of the headache after 15 minutes
- the intensity of a possible feeling of nausea after 15 minutes
- the intensity of a possible noise in the ear after 15 minutes and
- the moment when an analgesic medication was administered.

3.6.2. Evaluation of the medical records

The patient's medical records contained information concerning the day of the LP, the reason for the LP and the causes of the complaints, which were present before the LP, as well as information on when and which pain killer was given to the individual patient.

3.6.3. Statistic analysis

A statistic analysis of the data was carried out with the software Microsoft Excel.
The statistic significance was evaluated by means of the t-test. In the analysis $p < 0.05$ was estimated as being statistically significant.

4. Results

After a study period of 7 months, where each patient participated for 5 days in the study, the collected data were analysed.
Due to the time constraints I could not include more than 21 Patients in the study.

4.1. General remarks

Overall 21 patients fulfilled the criteria to be included in the study.
10 of the patients were attributed to the control group (CG), which was only looked after with conventional methods (pain medication).
11 of the patients belonged to the treatment group (TG). Besides the conventional methods theses patients also received an osteopathic treatment on the first and third postpuncture day.

During the study I found out that one test person of the CG suffered from another form of headaches besides the postpuncture headaches. This could be recognized because it took quite long (30 – 60 minutes after changing into the upright position) until the headaches appeared.
The patient in question was excluded from the study.

4.2. Results

In both groups the patients' answers concerning the individual symptoms (headache, nausea and noise in the ear) were very varied.
If we assume a significance level of $p < 0.05$, the results of both groups did not have statistic significance.

However, both groups showed clear trends in their results.

4.2.1. Overall complaints

The analysis of the data of both groups produced different initial values concerning the overall complaint on the first postpuncture day.

In addition to the headaches the frequency of the answers for the symptoms nausea (N) and noise in the ear (NE) was higher in the TG (N: n=8, NE: n=5) than in the control group (N: n=3, NE: n=0) (Fig. 12).

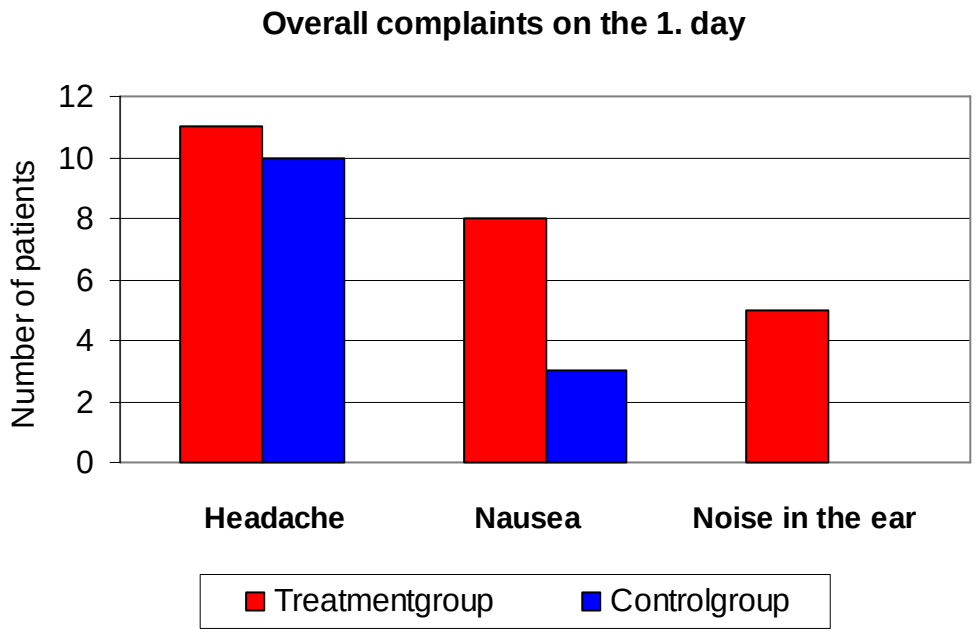


Figure 12 *Overall complaints on the 1st postpuncture day*

4.2.2. Headaches

If we look at the symptom headaches in isolation, the distribution of the initial values for the subjective pain intensity in the two groups on the first day can be illustrated as follows (Tab. 1):

Pain intensity	1	2	3	4	5	6	7	8	9	10
CG	0	1	0	2	1	1	0	2	1	2
TG	0	0	0	1	0	1	2	2	2	3

Tab. 1 *Individual perception of pain intensity on the 1st day (VRS 0-10)*

Fig. 13 shows that the initial values of the subjective pain intensity were higher in the TG than in the CG.

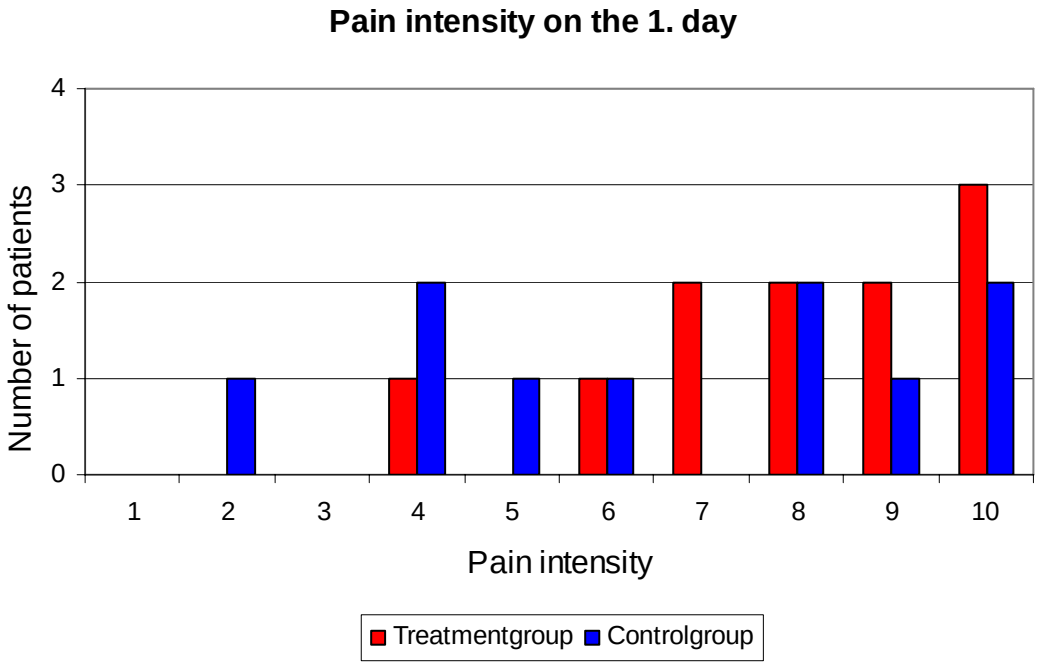


Figure 13 *Distribution of individual perception of pain intensity on the 1st day (VRS 0-10)*

The relative mean value for the intensity of the headaches on the 1st day was 6.6 ± 2.8 (n= 10) in the CG and 8 ± 1.9 (n=11) in the TG.

4.2.3. Changes of the intensity of headaches

Starting from the first indicated value as basic value (= 0%) the changes in the relative mean values of pain intensity in the two groups showed different developments.
While the complaints increased in the CG on the 2nd and 3rd postpuncture day, the pain immediately decreased in the TG (Fig. 14).

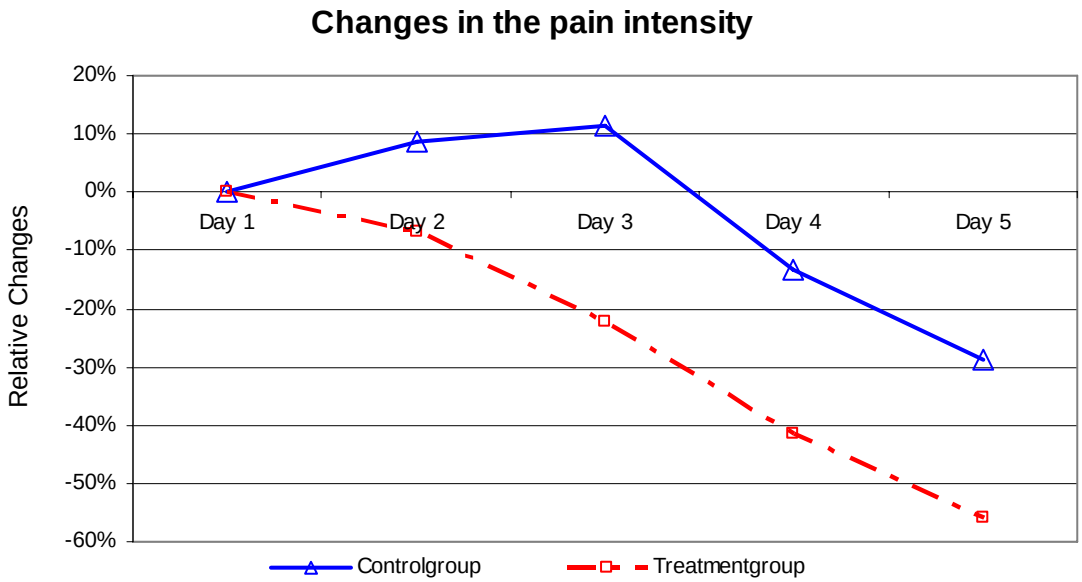


Figure 14 *Changes in the pain intensity*

The relative changes of the headaches on the 1st postpuncture day in comparison with the initial value can be illustrated as follows (Tab. 2):

	Day 2	Day 3	Day 4	Day 5
CG	+ 9%	+ 11%	- 13%	- 29%
TG	-7%	- 22%	- 41%	- 56%

Tab. 2 *Changes of the intensity of headaches on the first day*

In comparison to the first day the relative change in the mean value of the headaches' intensity within five days showed a decrease of 29 percent in the CG and 56 percent in the TG.

If we directly compare the two groups the trend of a stronger decrease in the pain intensity within the 5-day period of the study can be observed in the TG than in the CG (Fig. 15).

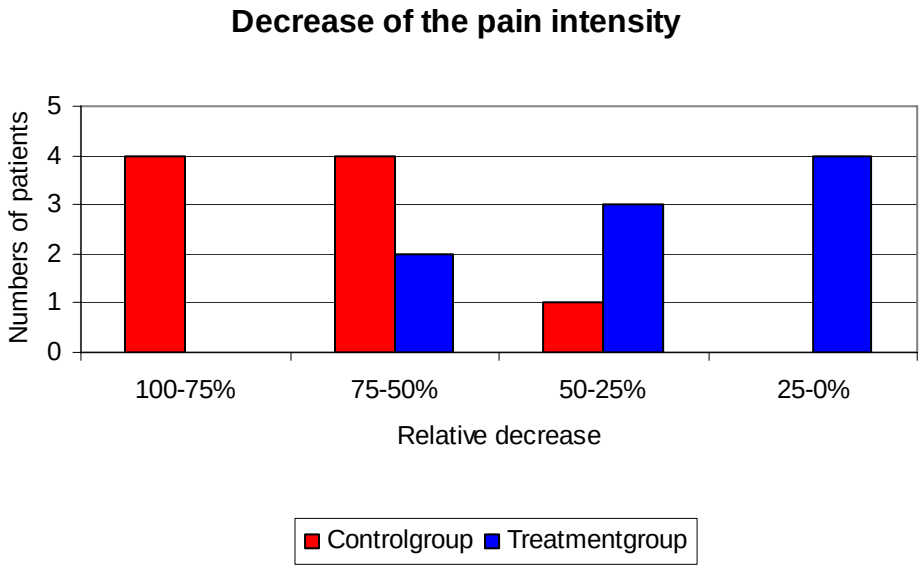


Figure 15 *Decrease of pain intensity on the 5th day*

	-100-75%	- 75-50%	- 50-25%	- 25-0%
CG (n=10)	4	4	1	0
TG (n=11)	0	2	3	4

Tab. 3 *Decrease of pain intensity on the 5th day*

Tab. 3 shows that more patients in the TG than in the CG could be found in the range of a 100-50% improvement with regard to the intensity of the headaches. In the CG the majority of the patients fell in the category of a 50-0% improvement of the intensity of the headaches. One patient of the CG noticed an increase of 50% of the headaches' intensity on the fifth day in comparison with the initial value of the first day.

If we compare 2 age groups (< 40 y, n=5, > 40 y, n=6) of the TG, we can observe different changes of the mean headache intensity (Fig. 16).

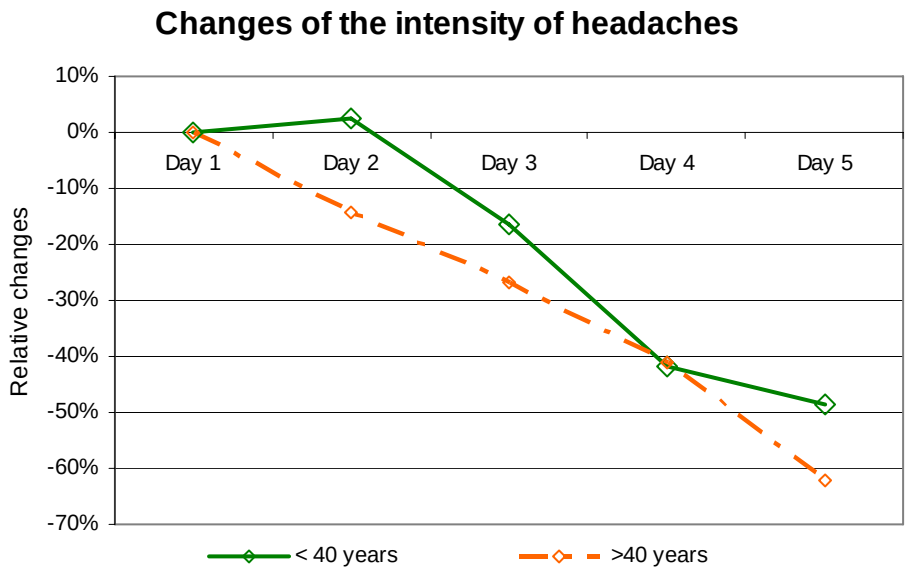


Figure 16 *Changes of the pain intensity in patients of the TG < 40 y /> 40 y*

Patients younger than 40 years still experienced an increase of the subjective pain intensity on the 2nd postpuncture day, while patients older than 40 years experienced an immediate decrease.

On the 5th day a relative mean improvement of the headaches by 49 percent could be observed in the group of patients younger than 40 years, while in the group of patients older than 40 years the pain intensity improved by 62 percent (*Fig.3. 5*).

4.2.4. Time until the onset of pain

The mean values concerning the subjective perception of the time until the onset of the postpuncture headaches were very varied for the first day (initial value). The mean value of the CG ranged around 6.6 ± 5.7 minutes, while the mean value for the TG was 1.8 ± 2.2 minutes.

If we take the first value indicated on the first day as the basic value (=0%), an immediate lengthening of the period until the onset of the pain could be observed in the TG, while in the CG the period started to increase only on the second day Fig. 17).

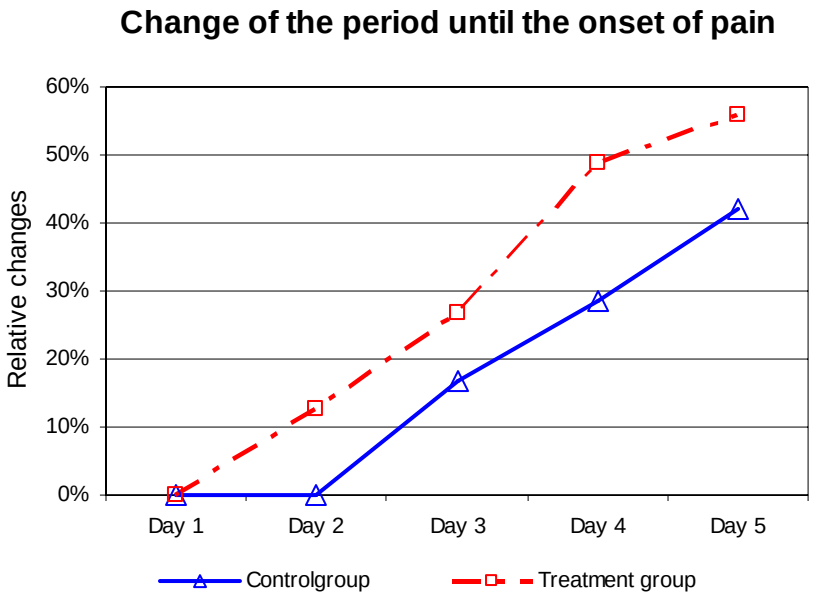


Figure 17 *Change of the period until the onset of pain*

Fig. 17 and Tab. 4 illustrate that the number of minutes which passed until the onset of pain jumped up on the fourth day.

	Day 2	Day 3	Day 4	Day 5
CG	+ 0%	+ 17%	+ 28%	+ 42%
TG	+13%	+ 27%	+ 49%	+56%

Tab. 4 *Change in the time until the onset of pain in comparison to the 1st day*

4.2.5. Nausea

Three patients of the CG (n=10) and 8 patients of the TG (n=11) showed the symptom of nausea.
The mean value for the relative subjective intensity of the nausea was 0.9 ± 1.7 in the CG and 2.9 ± 3.4 in the TG.

As can be seen in Fig. 18 the changes in the CG involved an increase in the frequency and intensity of the nausea until the third day, which can be explained on the one hand by

the intensification of the existing symptom but on the other hand also by the new development of the symptom.

In the TG the patients did not develop the new symptom of nausea and the subjective mean intensity of the existing symptom decreased.

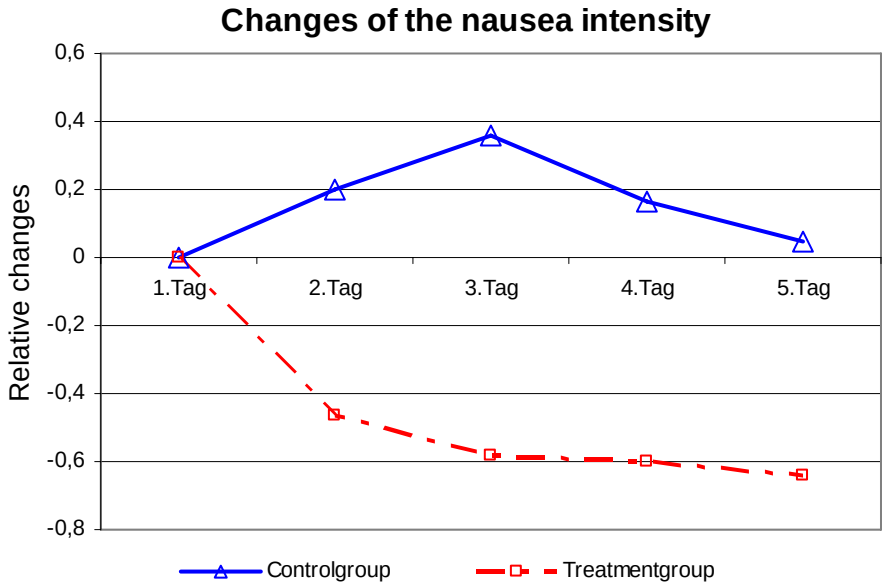


Figure 18 *Change of the intensity of the nausea*

In comparison with the first day no improvement of the intensity of the symptom nausea could be observed until the fifth day in the CG. In the TG the intensity decreased already on the 2nd day.

On the 5th day the intensity of the nausea in the CG changed by + 5% in the CG and by -64% in the TG (Tab. 5).

	Day 2	Day3	Day 4	Day 5
CG	+20%	+36%	+17%	+ 5%
TG	-46%	-58%	-60%	-64%

Tab. 5 *Changes of the symptom nausea*

4.2.6. Noise in the ear

Only one test person in the CG experienced the occurrence of noises in the ear on the 2nd day. In the TG the symptom was much more frequent with a mean value on the first day of 1.4 ± 2.4.

The intensity of the noises in the ear continually decreased in the TG from the first day onwards, with a pronounced drop on the second day. On the 5th day the intensity of the symptom increased slightly from -40% to -39% (Fig. 19).

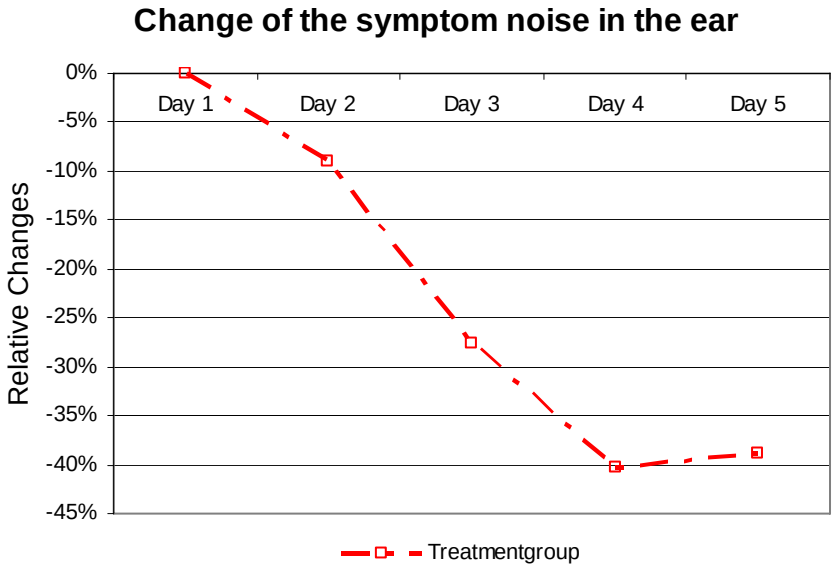


Figure 19 *Change of the symptom noise in the ear*

4.4. Summary of the results

4.4.1. Degree of improvement of the complaints

In both groups an improvement of the complaints could be observed. The degree of improvement followed a clear trend in the two groups.

For all symptoms the initial values of the TG were higher than those of the CG (Fig. 12, p. 34). In the case of the symptom headaches this was due to the intensity which was rated higher in this group; for the symptoms nausea and noise in the ear this was due to the higher incidence and intensity.

The postpuncture headaches could be influenced faster in the TG, which resulted in an immediate improvement of the pain and not – like in the CG – in an increase of the mean intensity of the headaches until the third day. In addition, a relative reduction by -56% could be observed in the TG with regard to the initial value. In the CG the relative reduction was only -29% by day 5 (Fig. 15).

On the fifth day the relative change of the period until the onset of the headaches after adopting an upright position was 13% bigger in the TG than in the CG. The lengthening of this period could be immediately observed in the TG, while in the CG this was the case only from the second day onwards (Fig. 17, p. 37).

In the CG the intensity of the nausea increased further until the third day, while in the TG it was already less on the second day. On the fifth postpuncture day a relative increase in the mean intensity and the frequency of the symptom of nausea could be observed (5%) in the CG with regard to the first day, while in the TG a relative reduction of 64% could be observed (Fig. 18, p. 40).

The symptom noise in the ear could be neglected in the CG since only one test person indicated to suffer from the symptom on one day.
In the TG a relative reduction of 39% of the mean intensity of noises in the ear within the study period could be observed (Fig. 19, p. 41).

5. Discussion

As an introduction I would like to refer to the pathogenesis of postpuncture headaches (cf. also chapter 2.7.4.).

After a lumbar puncture the drainage of CSF and the prolonged leakage of CSF cause a state of hypovolemia of the CSF since the body is not able to immediately compensate for the loss.
Due to the reduced CSF volume the brain tissue is not enough supported by the cisterns, which leads to an overstretching of pain-sensitive structures in the cranium and an impairment of the venous drainage in a caudal direction. From an osteopathic point of view an LP represents an intervention in the cranio-sacral system and thus in the whole body system. Patients suffering from postpuncture headaches show an increased sympathetic tone, a reduced rate and amplitude of the PMR and thus an inadequate production of CSF.
An osteopathic treatment tries to intervene at the origin of this vicious circle and thus has the possibility to influence the postpuncture complaints by working via the PRM to achieve a reduction of the sympathetic tone and a relaxation of pain-sensitive cranial structures and by applying techniques to improve the venous drainage (cf. chapter 2.8.1.).

I did not find any studies on the influence of osteopathic treatment on postpuncture headaches. The treatment approaches applied in the course of the study are based upon my own osteopathic considerations along the lines of the pathophysiology and pathogenesis of postpuncture complaints.

A point of criticism of this study is that the two study groups were quite small with only 10 and 11 test persons respectively. Due to the use of atraumatic puncture needles (cf. chapter 2.6.2.) postpuncture headaches do not occur so often nowadays. The time constraints did not make it possible to prolong the duration of the study beyond the period of 7 months and to include more patients in the study.

It can also be criticized that the data indicated by the patients had a large variance. Both the values indicating the intensity of the complaint on the visual rating scale and the statements concerning the time (minutes) until the onset of the headaches differed quite significantly in all patients. Maybe the choice between 5 possible answers (ranging from „significant improvement“ to „no improvement“ of the complaint) or between 3 different

intervals for the time until the onset of the pain (0-5, 5-10, 10-15 minutes) would have been easier to handle.

Due to the large variance of the data and the small number of test persons the results cannot have statistic significance.
Nevertheless the degree in which the complaints improved shows a clear trend in the two groups.

Postpuncture headaches are self-limiting. LYBECKER et al [34] say that in 80 percent of the patients the complaints disappear within 5 days after their onset. COSTIGAN and SPRIGGE [8] and MACARTHUR et al [37] state that in 85 percent of the cases the complaints limit themselves within 6 weeks.
However, in my study only one patient of the TG did not have headaches anymore after five days. The other twenty patients still suffered from postpuncture headaches, often in combination with nausea and/or noise in the ear.

In my approach I did not want to merely treat the symptoms by administering analgesic medication to bridge the time gap until the self-limitation of the complaint, but to influence the true underlying causes of the problem with osteopathic, especially cranio-sacral techniques.

It cannot be excluded that part of the reduction of the complaint in the patients of the TG can be attributed to the placebo-effect. The osteopathic treatment gives the patients the feeling that they and their complaints are taken seriously, they are gently touched where they say it hurts, and they can talk about their “agony” with the practitioner. In order to clearly exclude this placebo-effect a second control group, receiving a pseudo-treatment, would have been necessary.

Incidentally the patients’ division into the two groups had the result that in the TG the patients’ subjective perception of the complaint on the first day was more severe than in the CG. Especially the symptom headache was clearly attributed higher initial values for the intensity than in the CG (Fig. 13, p. 35).

If we compare the changes in the intensity of the headaches in both groups, a clear trend of a faster improvement in the TG can be observed. An osteopathic intervention on the first day could be responsible for the fact that the headaches’ intensity did decrease already on the second day, while in the CG the complaints still increased on the second

day. After the third day the headaches decreased in both groups. It cannot be said for sure whether the second osteopathic treatment had an influence in this respect. To make a clear statement another TG without a second treatment would have been necessary.
Nevertheless the moments of osteopathic intervention seemed to have been well chosen, for the mean intensity of the complaints of the TG continually decreased.

In the TG a greater reduction of the headaches' intensity could be achieved in the patients older than 40 years than in the patients younger than 40 years.
RASMUSSEN et al [48] and STONE and DI FAZIO et al [54] describe in their study a decreasing rate of postpuncture complaints with increasing age. It can be that in the patients younger than 40 years the complaints can continue to worsen after 24 hours.
A reason fort his could be that "older" patients observe the recommended bed rest and recreation (in conjunction with repose and relaxation) more strictly than "younger" patients.
Concerning the effectiveness of the osteopathic treatment the question arises why patients older than 40 years can be better influenced than younger patients (Fig. 16, p. 38). If we assume that "younger" patients still have more possibilities to compensate lesions and to activate their self-healing mechanisms, while "older" patients need support to activate their self-healing forces and regeneration capacity, this could explain why a practitioner who observes the osteopathic principles is more likely to influence the pain in "older" patients.

Concerning a change in the time until the onset of the headache an immediate improvement and a continuous lengthening of the period until the fifth day could be observed in the TG, with a very pronounced prolongation on the 4th day. In comparison to the results of the TG, in the CG the time gap until onset remained unchanged until the 2nd day, and improved on average by 14% on the fifth day with regard to the initial value (Fig. 17, p. 39).
This could lead to the interpretation that the immediate lengthening of the period until onset of the headache was due to the osteopathic treatment on the first day and that the pronounced improvement on the 4th day was the consequence of the osteopathic treatment on the 3rd day.

Concerning the symptom nausea the patients' indications during the course of the study differed mostly. On the fifth day a mean improvement of the intensity of the nausea of 64% with regard to the initial value could be observed in the TG. In the CG the intensity with regard to the first day increased by 5% (Fig. 18, p. 40).

The vomiting centre is located in the region of the Medulla oblongata and reacts to changes of intracranial pressure. Thus the rapid and significant decrease of the intensity of the symptom in the TG can be an indirect sign that the pressure in the cranium normalized.

It is not possible to compare the intensity and changes of noise in the ear in both groups, because only one patient of the CG indicated the symptom.
Nevertheless, the analysis of the data of the TG showed the clear trend that the intensity of the noises in the ear decreased from the first day onwards.

In sum the analysis of the data confirmed the tendency that the various symptoms decreased in the TG faster and more significantly than in the CG both in their intensity and also in their frequency.
Of course, it would be necessary to carry out a comparative study with a greater number of participants and an optimized questionnaire (or a different kind of evaluation) for the results to be statistically significant.
Further it would be important to pay attention not only to the physical wellbeing of the patients during the 5-day study period but also to consider possible long-term effects.

I also think that it would be wiser and more in line with the principles of osteopathy to prepare an organism for an intervention in its system in order to make it more resistant and able to regenerate so that postpuncture complaints do not occur at all or in a milder form. If an organism is free of severe restrictions and has an optimized rate and amplitude of the PMR, it is able to produce sufficient amounts of CSF and thus less postpuncture complaints should develop in a smaller number of patients. However, due to the fact that I had a limited amount of time available, it was not possible to give prophylactic osteopathic treatments to all patients undergoing a lumbar puncture.

Nevertheless it seems that an osteopathic treatment before the occurrence of postpuncture complaints would pay off, even though the practitioner does not have much time available, because in comparison to the CG the clear tendency of a faster recovery in the TG could be observed.

6. Summary

My personal approach to the topic and the reason why I have chosen to focus on this kind of complaints was my interest in the causative approach of osteopathy to the clinical picture of “postpuncture headaches”.

The chapter “Anatomical and Physiological Foundations” includes on the one hand an explanation of the most important anatomical and physiological considerations, and on the other hand it describes the pathophysiology of headaches and the execution and instruments of a lumbar puncture. Further the clinical picture “postpuncture headaches” as well as their pathogenesis, incidence, epidemiology, clinic and prophylaxis are explained. Finally, it includes a description of an osteopathic approach to the complaint.

The empirical part of the thesis includes the analysis of the questionnaires of the 21 test persons, who filled in a follow-up questionnaire on five consecutive days, starting with the first day on which the postpuncture headaches appeared. The ten patients of the control group (CG) received conventional care according to the guidelines of the hospital (bed rest, increased amount of liquid, pain medication). The eleven patients of the treatment group (TG) received two additional osteopathic treatments on the first and third day of the postpuncture headaches.

Due to the small number of test persons (because of the limited time which was available for carrying out the study) and due to the great variance in the pain evaluation in the questionnaires the results of both groups cannot be considered as statistically significant. However, a clear trend could be observed in the two groups.

Within five days a relative mean reduction of the headaches intensity by 56% could be observed in the TG, while in the CG the pain’s intensity was reduced by 29%.
In the TG the relative mean change in the time until the onset of the headaches after adopting an upright position was 13% higher than in the CG.
The intensity of the relative mean nausea in the TG on the fifth day ranged 64% below the initial value, in the CG it increased by 5% with regard to the first day.

These clear trends illustrate that an osteopathic treatment of patients with postpuncture complaints makes sense.

In a follow-up study it would be reasonable to include long-term effects, to evaluate the physical wellbeing, and therefore the long-term effects of a lumbar puncture on the organism and the influence of osteopathic treatment in this context.

Another osteopathic approach to the disease pattern “postpuncture headaches” would be a prophylactic treatment *before* a lumbar puncture is carried out, to evaluate if there is a lower incidence of the complaints that can be linked with the treatment.

Bibliography

1 Arlot J., D.O., Unveröffentlichtes Skriptum, Wien, 1998

2 Bartsch T, Goadsby PJ., Increased responses in trigeminocervical nociceptive neurons to cervical input after stimulation of the dura mater. Brain 2003;126:1801-1813

3 Bier A. Versuche über Cocainisierung des Rückenmarkes. Dtsch Z Chir, 1899; 51: 361

4 Bolet P., D.O., Unveröffentlichtes Skriptum, Frankreich, St. Etienne, 2002

5 Burstein R, Cutrer MF, Yarnitsky D. The development of cutaneous allodynia during migraine attack. Clinical evidence fort he sequential recruitment of spinal and supraspinal nociceptive neurons in migraine. Brain, 2000; 123 (Pt 8): 1703 – 1709

6 Busch V, Frese A, Bartsch T. Der trigeminocervikale Komplex. Schmerz 2004;18:404 410

7 Büttner J, Wresch KP, Klose R. Bietet eine konisch geformte Kanülenspitze Vorteile bei der Spinalanästhesie. Regional Anästhesie, 1990; 13: 124 – 128

8 Costigan SN, Sprigge JS. Dural puncture: the patients´ perspective. A patient survey of cases at a DGH maternity unit 1983 – 1993. Acta Anaestheiol Scand, 1996; 40: 710 – 14

9 Diener H-C, Schorn CF, Strupp M, Dietrich M, Stolke D, Wanke I. Das Liquorunterdrucksyndrom. Akt Neurol 2006; 33:20-25

10 Dietrich M. Spontanes und postpunktionelles Liquorunterdrucksyndrom. In:Diener H.C. (Hrsg.): Therapie und Verlauf neurologischer Erkrankungen. Stuttgart: Kohlhammer, 2003; 88-94

11 Dietrich M, Brandt T. Incidence of post-lumbar puncture headache is independent of daily fluid intake. Eur Arch Psychiatry Neurol Sci, 1988; 237: 194 – 195

12 Dietrich M, Brandt T. Is obligatory bed rest after lumbar puncture obsolete? Eur Arch Psychiatry Neurol Sci, 1985; 235: 71 – 75

13 Evans RW, Armon C, Frohmann EM, Goodin DS. Assessment: Prevention of post-lumbar puncture headaches. Report of the Therapeutics an Technology Assessment Subcomittee of the American Academy of Neurology. Neurology 2000; 55:909-914

14 Fanghänel J, Pera F, Anderhuber F, Nitsch R. Waldeyer, Anatomie des Menschen. 2003. Verlag de Gruyter

15 Fearon W. Post-Lumbar Puncture Headache. P & S Medical Review März 1994, Vol.1, Nr. 2

16 Feneis H. Anatomisches Bildwörterbuch, 1993, Thieme-Verlag.

17 Forderreuther S, Yousry I, Empl M. et al. Dilated cervical epidural veins and extraarachnoid fluid collection in orthostatic headaches. Neurology 2001; 57: 527-529

18 Frenkel Ch, Altscher T, Groben V, Hörnchen U. Inzidenz von postspinalem Kopfschmerz bei einem jungen Patientenkollektiv. Anaesthesist, 1992; 41:142 – 145

19 Frese A, Schlingen M, Husstedt IW, Evers S. Pathophysiologie und Klinik zervikogener Kopfschmerzen. Schmerz 2003; 17: 125-130

20 Grant R, Condon B, Hart I, Teasdale GM. Changes in intracranial CSF volume after lumbar puncture and their relationship to post-LP headache. J Neurol Neurosurg Psychiatry, 1991; 54: 440 – 2

21 Hafer J, Rupp Daumen, Wollbrück M, Engel J, Hempelmann G. Die Bedeutung von Nadeltyp und Immobilisation für den postspinalen Kopfschmerz. Anästhesist, 1997; 46: 860 – 866

22 Handler CE, Smith FR, Perkin GD, Rose FC. Posture and lumbar puncture headache: a controlled trial in 50 patients. J R Soc Med, 1982; 75: 404 – 7

23 Headache Classification Comittee of the International Headache Society. The international classification of headache disorders, 2nd edn. Cephalalgia 2004; 24:1-160

24 Hoskin MF. Spinal anaesthesia – the current trend towards narrow gauge atraumatic (pencil point) needles. Case reports and review. Anaesth Intens Care, 1998; 26: 96 – 106

25 Kahle W, fortgeführt v. Frotscher M. Taschenatlas der Anatomie. Nervensystem und Sinnesorgane. 2001, 7. Aufl., Thieme Verlag

26 Keener EB. An experimental study of reactions of the dura mater to wounding and loss of substance. J Neurosurg, 1959; 16: 424 – 47

27 Kiefer M, Steudel W-I. Moderne Hirndruckmessung. Grundlagen und Praxis. Der Unfallchirurg, 2002; 105: 578-586

28 Kreuschner H, Sandmann G. Die Minderung des postspinalen Kopfschmerzes durch Verwendung der Witacre-Kanüle. Regional Anaesthesie, 1989; 12: 43 – 45 und 46 – 49

29 Kuczkowski KM., Post-dural puncture headache in the obstetric patient: an old problem. New solutions. Minerva Anestesiol 2004; 70:823-830

30 Kuntz KM, Kokmen E, Stevens JC, Miller P, Offord KP, Ho MM. Post-lumbar puncture headaches: experience in 501 consecutive procedures. Neurology, 1992; 42: 1884 – 1887

31 Lay Christine M. Low Cerebrospinal Fluid Pressure Headache. Current Treatment Options in Neurology 2002; 4:357-363

32 Leitlinien für Diagnostik und Therapie in der Neurologie, 2005, 3. Aufl., Thieme Verlag

33 Liem T, Kraniosakrale Osteopathie. 1998. Hippokrates Verlag

34 Lybecker H, Djernes M, Schmidt JF. Postdural puncture headache (PDPH): onset, duration, severity and associated symptoms. Acta Anaesthesiol Scand, 1995; 39: 605 – 612

35 Lybecker H, Moller JT, May O, Nielsen HK. Incidence and prediction of postdural puncture headache. A prospective study of 1021 spinal anaesthesias. Anaesth Analg, 1990; 70: 389 – 94

36 Mac Robert RG. The cause of lumbar puncture headache. JAMA, 1918; 70: 1350 – 3

37 MacArthur C, Lewis M, Knox EG. Accidental dural puncture in obstetric patients an long term symptoms. Br Med J, 1992; 304: 1279 – 82

38 Martins AN, Wiley JK, Meyers PW. Dynamics of the cerebrospinal fluid and the spinal Dura Mater. J Neurol Neurosurg Psychiatry, 1972; 35: 468 – 473

39 Mokri B. Sydrome of cerebral spinal fluid hypovolemia: clinical and imaging features and outcome. Neurology 2001; 56:1607-1608

40 Mokri B. The Monro-Kellie hypothesis. Applications in CSF volume depletion. Neurology 2001; 56:1746-1748

41 Mokri B. Syndrome of orthostatic headaches and diffuse pachymeningeal gadolinium enhancement. Mayo Clin Proc, 1997; 72: 400

42 Müller B, Adeld K, Reichmann H, Toykak K. Atraumatic needle reduces the incidence of post-lumbar puncture syndrome. J Neurol, 1994; 241: 376 – 380

43 Netter F.H., Atlas der Anatomie des Menschen. 2000, 2. überarbeitete Aufl., Thieme Verlag

44 Pannullo SC, Reich JB, Krol G. et al. MRI changes in intracranial hypotension. Neurology 1993; 43:919-926

45 Pearce JM. Walter Essex Wynter, Quincke and lumbar puncture. J Neurol Neurosurg Psychiatry, 1994; 57: 179

46 Pschyrembel, Klinisches Wörterbuch. 1994. de Gruyter Verlag

47 Quaynor H, Corbey M, Berg P. Spinal Anaesthesia in Day-Care Surgery with a 26-Gauge Needle. British Journal of Anaesthesia, 1990; 65: 766 – 769

48 Rasmussen BS, Blom L, Hansen P, Mikkelsen SS. Postspinal headache in young and elderly patients. Anaesthesia, 1989; 44: 571 – 573

49 Reina MA, de Leon-Casasola OA, Lopez A, De Andres J, Martin S, Mora M. An in vitro study of dural lesions produced by 25-gauge Quincke an Whitacre needles evaluated by scanning electron microscopy. Reg Anesth Pain Med, 2000; 25: 293 – 402

50 Reuter U, May A. Vorraussetzungen für das Symptom Kopfschmerz, Aspekte zur Pathogenese. Schmerz 2004; 18:357-362

51 Speransky AD. Basis fort he theory of medicine. International publishers Inc., 1943

52 Spriggs DA, Burn DJ, French J, Cartlidge NE, Bates D. Is bed rest useful after diagnostic lumbar puncture? Postgrad Med J, 1992; 68: 58 – 3

53 Still AT. Philosophy and mechanical principles of osteopathy. Hudson Kimberly, Kansas, 1902. Reprinted 1986 ba Osteopathic Enterprise, Kirksville, S. 44

54 Stone DJ, DiFazio CA. Postspinal Headache in older patients. Anesth Analg, 1990; 70:222

55 Straube A, Neudert C, Glas M, Brüning R, Padovan CS. Das sogenannte spontane Liquorunterdrucksyndrom. Nervenarzt 2004; 75:1194-1199

56 Strupp M, Brandt T. Should one reinsert the stylet during lumbar puncture? N Engl J Med 1997; 336:1190

57 Strupp M, Schueler O, Straube A, Von Stuckrad-Barre S, Brandt T. „Atraumatic" Sprotte needle reduces the incidence of post-lumbar puncture headaches. Neurology 2001; 57:2310-2312

58 Sutherland WG. The cranial bowl. Free Press Company, Mankato, Minnesota 1939, S. 56 – 57

59 Turnbull DK, Shepherd DB. Post-dural puncture headache: pathogenesis, prevention and treatment. British Journal of Anaesthesia 2003; 91(5): 718-729

60 Vandam LD, Dripps RD. Long term follow up of patients who received 10 098 spinal anesthetics. JAMA, 1956; 161: 586 – 91

61 Yousry I, Förderreuther S, Moriggl B, Holtmannspötter M, Naidich TP, Straube A, Yousry TA. Cervical imaging in Postural Headache: MR signs and Pathophysiological Implications. American Journal of Neuroradiology, 2001; 22:1239-1250

Internetquellen

PAJUNK© SPROTTE©- Kanüle. IntraLong – Die beste Wahl für die kontinuierliche Technik (2004) [WWW Document] URL http://www.pajunk.com/pdf/de/Spezial-Sprotte_dt_RZ.pdf (30.5.2006)

PAJUNK© Spezial-SPROTTE©- Kanüle. Der Fortschritt in der Spinalanästhesie (2005). [WWW Document] URL http://www.pajunk.com/pdf/de/Spinalanaesthesie_D.pdf (30.5.2006)

7. Annex

Basic Questionnaire

Age _____ m / f

Puncture date _____ Time _____

Reason for the puncture

_____

Amount of extracted CSF _____ Practitioner _____

Needle _____

Complaints before the puncture

_____

_____

Following the advice after the puncture

_____

_____

Quantity of water drunk on the 1st day _____

Onset of the complaints _____

Follow-up Questionnaire

Date _____ Time _____

Onset of the complaint after_____ minutes.

After 15 minutes sitting:

Headaches |-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
none very severe

Nausea |-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
none very severe

Noise in the ear |-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
none very severe

Do you need **analgesic medication** ? Yes / No

When ? _____

Controlgroup

Min: Minutes until onset of headache, **HA**: Headache, **N:** Nausea, **NE:** Noise in the ear

			Day 1				Day 2				Day 3				Day 4				Day 5			
	m/f	Age	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE
P1	f	54	10	8	2	0	10	8	2	0	10	8	3	0	10	6	2	0	10	4	1	0
P2	f																					
P3	f	47	5	2	0	0	5	4	1	1	5	5	3	0	5	4	2	0	10	3	2	0
P4	f	32	0	8	0	0	0	7	0	0	5	7	2	0	5	6	2	0	5	5	0	0
P5	m	36	15	6	0	0	15	6	0	0	15	6	0	0	15	3	0	0	15	2	0	0
P6	f	36	1	10	5	0	1	10	6	0	1	10	6	0	5	8	5	0	5	4	1	0
P7	f	28	0	10	2	0	0	10	4	0	1	10	3	0	2	9	2	0	2	9	2	0
P8	f	78	5	9	0	0	5	9	6	0	5	9	5	0	0	2	0	0	10	5	5	0
P9	f	84	5	4	0	0	5	4	0	0	5	3	0	0	10	4	0	0	10	3	0	0
P10	m	45	10	4	0	0	10	4	0	0	15	4	0	0	15	3	0	0	15	3	0	0
P11	m	58	15	5	0	0	15	5	0	0	15	5	0	0	15	5	0	0	15	4	0	0
MV		49,8	6,6	6,6	0,9	0	6,6	6,7	1,9	0,1	7,7	6,7	2,2	0	8,2	5	1,3	0	9,7	4,2	1,1	0
SD		19,01	5,6804	2,7968	1,6633	0	5,6804	2,4518	2,5144	0,3162	5,6184	2,4967	2,201	0	5,5936	2,2608	1,6364	0	4,5717	1,9322	1,5951	0
dMin		21,8	6,6	4,6	0,9	0	6,6	2,7	1,9	0,1	6,7	3,7	2,2	0	8,2	3	1,3	0	7,7	2,2	1,1	0
dMax		34,2	8,4	3,4	4,1	0	8,4	3,3	4,1	0,9	7,3	3,3	3,8	0	6,8	4	3,7	0	5,3	4,8	3,9	0
Min		28	0	2	0	0	0	4	0	0	1	3	0	0	0	2	0	0	2	2	0	0
Max		84	15	10	5	0	15	10	6	1	15	10	6	0	15	9	5	0	15	9	5	0

Standardized

			Day 1				Day 2				Day 3				Day 4				Day 5			
	m/f	Age	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE
P1	f	54	100%	100%	67%	0%	100%	100%	67%	0%	100%	100%	100%	0%	100%	75%	67%	0%	100%	50%	33%	0%
P2	f	55																				
P3	f	47	50%	40%	0%	0%	50%	80%	33%	100%	50%	100%	100%	0%	50%	80%	67%	0%	100%	60%	67%	0%
P4	f	32	0%	100%	0%	0%	0%	88%	0%	0%	100%	88%	100%	0%	100%	75%	100%	0%	100%	63%	0%	0%
P5	m	36	100%	100%	0%	0%	100%	100%	0%	0%	100%	100%	0%	0%	100%	50%	0%	0%	100%	33%	0%	0%
P6	f	36	20%	100%	83%	0%	20%	100%	100%	0%	20%	100%	100%	0%	100%	80%	83%	0%	100%	40%	17%	0%
P7	f	28	0%	100%	50%	0%	0%	100%	100%	0%	50%	100%	75%	0%	100%	90%	50%	0%	100%	90%	50%	0%
P8	f	78	50%	100%	0%	0%	50%	100%	100%	0%	50%	100%	83%	0%	0%	22%	0%	0%	100%	56%	83%	0%
P9	f	84	50%	100%	0%	0%	50%	100%	0%	0%	50%	75%	0%	0%	100%	100%	0%	0%	100%	75%	0%	0%
P10	m	45	67%	100%	0%	0%	67%	100%	0%	0%	100%	100%	0%	0%	100%	75%	0%	0%	100%	75%	0%	0%
P11	m	58	100%	100%	0%	0%	100%	100%	0%	0%	100%	100%	0%	0%	100%	100%	0%	0%	100%	80%	0%	0%
MV		50,27	54%	94%	20%	0%	54%	97%	40%	10%	72%	96%	56%	0%	85%	75%	37%	0%	100%	62%	25%	0%
SD		18,11	39%	19%	33%	0%	39%	7%	47%	32%	31%	8%	49%	0%	34%	23%	41%	0%	0%	18%	32%	0%
dMin		22,27	54%	54%	20%	0%	54%	17%	40%	10%	52%	21%	56%	0%	85%	53%	37%	0%	0%	29%	25%	0%
dMax		33,73	46%	6%	63%	0%	46%	3%	60%	90%	28%	4%	44%	0%	15%	25%	63%	0%	0%	28%	58%	0%

Change

			Day 1				Day 2				Day 3				Day 4				Day 5			
	m/f	Age	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE
P1	f	54	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	33%	0%	0%	-25%	0%	0%	0%	-50%	-33%	0%
P2	f	55	0%				0%				0%				0%				0%			
P3	f	47	0%	0%	0%	0%	0%	100%	33%	0%	0%	150%	100%	0%	0%	100%	67%	0%	50%	50%	67%	0%
P4	f	32	0%	0%	0%	0%	0%	-13%	0%	0%	100%	-13%	100%	0%	100%	-25%	100%	0%	100%	-38%	0%	0%
P5	m	36	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	-50%	0%	0%	0%	-67%	0%	0%
P6	f	36	0%	0%	0%	0%	0%	0%	17%	0%	0%	0%	17%	0%	80%	-20%	0%	0%	80%	-60%	-67%	0%
P7	f	28	0%	0%	0%	0%	0%	0%	50%	0%	50%	0%	25%	0%	100%	-10%	0%	0%	100%	-10%	0%	0%
P8	f	78	0%	0%	0%	0%	0%	0%	100%	0%	0%	0%	83%	0%	-50%	-78%	0%	0%	50%	-44%	83%	0%
P9	f	84	0%	0%	0%	0%	0%	0%	0%	0%	0%	-25%	0%	0%	50%	0%	0%	0%	50%	-25%	0%	0%
P10	m	45	0%	0%	0%	0%	0%	0%	0%	0%	33%	0%	0%	0%	33%	-25%	0%	0%	33%	-25%	0%	0%
P11	m	58	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	-20%	0%	0%
MV		50,27	0%	0%	0%	0%	0%	9%	20%	0%	17%	11%	36%	0%	28%	-13%	17%	0%	42%	-29%	5%	0%
SD		18,11	0%	0%	0%	0%	0%	32%	33%	0%	32%	49%	42%	0%	49%	46%	36%	0%	39%	33%	43%	0%
dMin		22,27	0%	0%	0%	0%	0%	21%	20%	0%	17%	36%	36%	0%	78%	65%	17%	0%	42%	38%	72%	0%
dMax		33,73	0%	0%	0%	0%	0%	91%	80%	0%	83%	139%	64%	0%	72%	113%	83%	0%	58%	79%	78%	0%

Treatmentgroup

Min: Minutes until onset of headache, **HA**: Headache, **N**: Nausea, **NE:** Noise in the ear

			Day 1				Day 2				Day 3				Day 4				Day 5			
	m/f	Age	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE
P1	f	42	0	8	5	5	2	5	1	5	5	3	0	2	10	3	0	0	10	1	0	0
P2	f	55	0,5	9	1	1	0,5	10	0	0	0,5	10	0	0	5	6	0	0	0	2	0	0
P3	m	35	2	7	1	0	5	7	0	0	5	5	0	0	5	4	0	0	5	4	0	0
P4	f	39	1	10	2	0	2	10	0	0	3	7	0	0	4	4	1	0	5	4	0	0
P5	f	18	0	10	2	0	0	10	3	0	0	10	3	0	0	10	1	0	0	10	1	0
P6	f	50	1	7	10	7	1	4	2	7	2	4	0	4	1	4	0	4	2	3	0	5
P7	m	45	0	10	8	0	2	10	4	0	3	10	2	1	3	10	2	0	5	10	2	0
P8	f	22	0	9	3	1	0	9	2	1	0	8	0	0	5	4	0	0	15	2	0	0
P9	m	66	5	6	0	1	5	5	0	1	5	5	0	0	5	4	0	0	5	3	0	0
P10	f	37	5	8	0	0	5	9	0	0	5	7	0	0	15	4	0	0	15	3	0	0
P11	f	57	5	4	0	0	5	4	0	0	10	2	0	0	15	1	0	0	15	0	0	0
MV		42,36	1,7727	8	2,9091	1,3636	2,5	7,5455	1,0909	1,2727	3,5	6,4545	0,4545	0,6364	6,1818	4,9091	0,3636	0,3636	7	3,8182	0,2727	0,4545
SD		14,49	2,1606	1,8974	3,3898	2,3779	2,1095	2,5832	1,446	2,4121	2,958	2,8762	1,0357	1,2863	5,056	2,7732	0,6742	1,206	5,831	3,2808	0,6467	1,5076
dMin		24,36	1,7727	4	2,9091	1,3636	2,5	3,5455	1,0909	1,2727	3,5	4,4545	0,4545	0,6364	6,1818	3,9091	0,3636	0,3636	7	3,8182	0,2727	0,4545
dMax		23,64	3,2273	2	7,0909	5,6364	2,5	2,4545	2,9091	5,7273	6,5	3,5455	2,5455	3,3636	8,8182	5,0909	1,6364	3,6364	8	6,1818	1,7273	4,5455

Standardized

			Day 1				Day 2				Day 3				Day 4				Day 5			
	m/f	Age	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE
P1	f	42	0%	100%	100%	100%	20%	63%	20%	100%	50%	38%	0%	40%	100%	38%	0%	0%	100%	13%	0%	0%
P2	f	55	10%	90%	100%	100%	10%	100%	0%	0%	10%	100%	0%	0%	100%	60%	0%	0%	0%	20%	0%	0%
P3	m	35	40%	100%	100%	0%	100%	100%	0%	0%	100%	71%	0%	0%	100%	57%	0%	0%	100%	57%	0%	0%
P4	f	39	20%	100%	100%	0%	40%	100%	0%	0%	60%	70%	0%	0%	80%	40%	50%	0%	100%	40%	0%	0%
P5	f	18	0%	100%	67%	0%	0%	100%	100%	0%	0%	100%	100%	0%	0%	100%	33%	0%	0%	100%	33%	0%
P6	f	50	50%	100%	100%	100%	50%	57%	20%	100%	100%	57%	0%	57%	50%	57%	0%	57%	100%	43%	0%	71%
P7	m	45	0%	100%	100%	0%	40%	100%	50%	0%	60%	100%	25%	100%	60%	100%	25%	0%	100%	100%	25%	0%
P8	f	22	0%	100%	100%	100%	0%	100%	67%	100%	0%	89%	0%	0%	33%	44%	0%	0%	100%	22%	0%	0%
P9	m	66	100%	100%	0%	100%	100%	83%	0%	100%	100%	83%	0%	0%	100%	67%	0%	0%	100%	50%	0%	0%
P10	f	37	33%	89%	0%	0%	33%	100%	0%	0%	33%	78%	0%	0%	100%	44%	0%	0%	100%	33%	0%	0%
P11	f	57	33%	100%	0%	0%	33%	100%	0%	0%	67%	50%	0%	0%	100%	25%	0%	0%	100%	0%	0%	0%
MV		42,36	26%	98%	70%	45%	39%	91%	23%	36%	53%	76%	11%	18%	75%	57%	10%	5%	82%	43%	5%	6%
SD		14,49	31%	4%	46%	52%	34%	16%	34%	50%	38%	21%	30%	34%	35%	24%	18%	17%	40%	33%	12%	22%
dMin		24,36	26%	9%	70%	45%	39%	34%	23%	36%	53%	39%	11%	18%	75%	32%	10%	5%	82%	43%	5%	6%
dMax		23,64	74%	2%	30%	55%	61%	9%	77%	64%	47%	24%	89%	82%	25%	43%	40%	52%	18%	57%	28%	65%

Change

			Day 1				Day 2				Day 3				Day 4				Day 5			
	m/f	Age	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE	Min	HA	N	NE
P1	f	42	0%	0%	0%	0%	20%	-38%	-80%	0%	50%	-63%	-100%	-60%	100%	-63%	-100%	-100%	100%	-88%	-100%	-100%
P2	f	55	0%	0%	0%	0%	0%	11%	-100%	-100%	0%	11%	-100%	-100%	90%	-33%	-100%	-100%	-10%	-78%	-100%	-100%
P3	m	35	0%	0%	0%	0%	60%	0%	-100%	0%	60%	-29%	-100%	0%	60%	-43%	-100%	0%	60%	-43%	-100%	0%
P4	f	39	0%	0%	0%	0%	20%	0%	-100%	0%	40%	-30%	-100%	0%	60%	-60%	-50%	0%	80%	-60%	-100%	0%
P5	f	18	0%	0%	0%	0%	0%	0%	33%	0%	0%	0%	33%	0%	0%	0%	-33%	0%	0%	0%	-33%	0%
P6	f	50	0%	0%	0%	0%	0%	-43%	-80%	0%	50%	-43%	-100%	-43%	0%	-43%	-100%	-43%	50%	-57%	-100%	-29%
P7	m	45	0%	0%	0%	0%	40%	0%	-50%	0%	60%	0%	-75%	100%	60%	0%	-75%	0%	100%	0%	-75%	0%
P8	f	22	0%	0%	0%	0%	0%	0%	-33%	0%	0%	-11%	-100%	-100%	33%	-56%	-100%	-100%	100%	-78%	-100%	-100%
P9	m	66	0%	0%	0%	0%	0%	-17%	0%	0%	0%	-17%	0%	-100%	0%	-33%	0%	-100%	0%	-50%	0%	-100%
P10	f	37	0%	0%	0%	0%	0%	13%	0%	0%	0%	-13%	0%	0%	67%	-50%	0%	0%	67%	-63%	0%	0%
P11	f	57	0%	0%	0%	0%	0%	0%	0%	0%	33%	-50%	0%	0%	67%	-75%	0%	0%	67%	-100%	0%	0%
MV		42,36	0%	0%	0%	0%	13%	-7%	-46%	-9%	27%	-22%	-58%	-28%	49%	-41%	-60%	-40%	56%	-56%	-64%	-39%
SD		14,49	0%	0%	0%	0%	21%	18%	49%	30%	27%	23%	54%	61%	36%	24%	45%	49%	42%	32%	46%	49%
dMin		24,36	0%	0%	0%	0%	13%	36%	54%	91%	27%	40%	42%	72%	49%	34%	40%	60%	66%	44%	36%	61%
dMax		23,64	0%	0%	0%	0%	47%	19%	80%	9%	33%	33%	92%	128%	51%	41%	60%	40%	44%	56%	64%	39%