Cranial Osteopathy Treatment in the Neurology

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Introduction

Neurology

Neurology or neuroscience is a medical speciality that deals with disorders of the nervous system. More specifically, it is concerned with the diagnosis and treatment of all diseases that are associated with the central and peripheral nervous systems. A neurologist is a medical doctor specializing in neurology, whose task is to investigate, diagnose and treat neurological disorders. Neurologists may be involved in clinical research and testing, as well as basic and translational research.
During the neurological examination, the neurologist will review the patient's medical history, paying particular attention to the current condition. The patient is then given a neurological examination. The examination checks mental status, cranial nerve function (including vision), strength, coordination, reflexes, and perception. These tests will help to determine whether the problem is neurological and, if so, where it is located. Accurate localization is key because it is the basis for the neurologist's differential diagnosis. Further tests may be necessary to confirm the diagnosis and help determine the appropriate therapy and treatment.

Cranial Osteopathy

Cranial osteopathy is a subfield of osteopathy along with parietal and visceral osteopathy.
In cranial osteopathy, the subtle movements of the cranial bones, sacrum, and associated connective tissues are examined and treated.
The aim of cranial osteopathy is to stimulate the free flow of fluids in the spinal column up to the sacrum and in the craniosacral system as a whole and to restore it in the case of abnormalities.
CSF is necessary for brain development, function, and growth. It transports important nutrients to the brain and nerves.
The theory of cranial osteopathy assumes that there are other rhythms of the body besides the breathing rhythm and pulse, such as the rhythm of cerebrospinal fluid and other fluids.
In the spinal cord and brain, the movement of fluids between the sacrum and the skull is perceived by trained osteopaths as a slight pulsation throughout the body.
The subtle vibrations are transmitted to body fluids and surrounding tissues. The rhythm of these fluid movements can be used as a diagnostic tool. Changes in this rhythm can be related to our hormonal balance, immune system, respiration, and nervous system.

The effects of disruption:
Disturbances of the craniosacral system can manifest themselves, for example, in migraines, back pain, or immune deficiency. Various disorders can also be triggered by dental treatment, stress, stress, or accidents.

Diagnosis and treatment:
Treatments involve very gentle pulling-pushing movements and very gentle holding of various points. As a result, muscles, bones, organs, and nerves are allowed to return to their natural flow.

Cranial osteopathy, like other areas of osteopathy, affects the whole human body. This allows very different areas of the body to be affected simultaneously.

This thesis aims to compare classical neurological examination and treatment methods with the treatment and examination used in osteopathy.

In this thesis, particular attention will be paid to the causes and possible treatments of headaches.

The thesis covers the treatment of 24 patients with osteopathy who had previously been examined by a neurologist for headache or migraine complaints.

Headache and migraine

The earliest attempts to cure migraines were skull carvings from the Middle Stone Age. At that time, neurological disorders such as headaches were believed to be caused by demonic possession and were therefore attributed to possessed evil spirits and demons. The purpose of craniotomy was to remove these psychic entities from the head. Skull carving served various purposes, including religious, spiritual, and medical. Results showed that half of the patients experienced long-term improvement. The procedure was also common among Hungarian immigrants. It was still practiced in the 17th century.

Other approaches were also used in ancient Egypt. The Ebers Papyrus, written around 1550 BC, describes various treatments. One such experiment involved coating the painful area with the remains of a catfish skeleton.

Hippocrates was the first to believe that the aura was a precursor to headaches. He commented that it was caused by vapors accumulating in the stomach and causing headaches. In the second century, Aretaius described the full spectrum of migraine symptoms, which he called heterochromia, including headache, sweating, nausea, and vomiting. Galen initially described the condition as hemicrania, attributing the disease to excessive yellow bile.

The central nervous system associated with migraines was first documented by Thomas Willis in 1664, along with the growth of blood vessels in the brain. At that time, migraine sufferers were treated with mercury compounds, including methylmercury, as well as compounds that lacked the toxic properties of mercury, including wormwood, pansy, and foxglove.

In 1884, William H. Thompson documented the successful extraction of an emulsifying agent from ergot that was effective in migraine. This formed the basis of modern migraine treatment. The substance responsible for the effect, ergotamine, was originally isolated by Arthur Stoll in 1920. The discovery of ergotamine's mechanism of action prompted the creation of new drugs, the tryptans.

Definition

Migraines are a specific type of headache, considered by many to be synonymous with severe, long-lasting headaches. A migraine aura, consisting of visual disturbances and flickering, zigzagging lights, often precedes the headache, which is usually unilateral. Movement disorders may also occur.

Because of its hemiplegic nature, the Greeks called it hemicrania, from which the Old French migraine derives, through the Latin form hemicrania.

This neurological disease affects 10% of the population. It is three times more common in women than in men. The pain is typically recurrent, throbbing, one-sided, and comes in attacks. It may be accompanied by nausea, vomiting, and sensitivity to light and sound.

Epidemiology

According to the Global Burden of Disease (GBD) study, headache is one of the most common and disabling conditions worldwide. The GBD is based on epidemiological studies (published and unpublished) that show significant differences in both methodology and prevalence estimates. Our first objective was to update the literature on headache epidemiological studies and to summarize global prevalence estimates for all headache types, migraine, tension-type headache (TTH), and headache occurring more than 15 days per month (H15+), and to compare these with the GBD. The time course is explored. Trends and geographical differences. Our second objective was to analyze how methodological factors influence prevalence estimates.

Migraines can occur in anyone, regardless of age or gender. A family history of migraine has also identified a certain hereditary predisposition that makes it more common. The prevalence is 8-15% of the total population, with the highest prevalence in women aged 20-50, in this group it is 20-25%.

Most patients are between 25 and 45 years of age, but the disease can start in childhood. The prevalence increases to 20% of all headaches by adolescence. There is no difference in prevalence between the sexes in children, only becoming more common in women after sexual maturation. Men are more likely to suffer from atypical migraine and are therefore less likely to be diagnosed with the condition. Because of its prevalence, its economic importance should not be underestimated, both in terms of the cost of treatment and the time lost in terms of lost work time and lost productivity.

Pathogenesis

Migraine is a primary cerebral disease resulting from alterations in the modulation of normal sensory stimuli. It was previously thought that migraine aura results from cerebral vasoconstriction and that headache is associated with cerebral and meningeal vasodilatation. It is now believed that migraine aura is a consequence of cortical depression and not vasoconstriction; ischemia is very rare if it occurs at all. The headache begins when cerebral circulation is reduced, but not to ischaemic levels, and is not the result of reflex vasodilation. Woods performed a PET study where a migraine patient showed propagated hypoperfusion during the pain phase of a migraine attack. A PET study of migraine patients by Denuelle also showed cortical hypoperfusion during the pain phase of migraine.

The aura is triggered in the hyperexcitable cortex. The visual aura-enhancing spectrum correlates with a cortical event of 2-3 mm/min, which is similar in nature to cortical spreading depression. The cortical phenomenon was first described by the AAP LEAO Institute as an intense depolarization of neuronal and glial membranes accompanied by a sharp drop in ion gradients and loss of membrane resistance.

The headache and associated neurovascular changes originate from the trigeminal vascular system. The headache is likely to be due to the activation of meningeal and vascular nociceptors, with associated changes in central pain modulation. Trigeminal sensory neurons contain neuropeptides called P-matter, calcitonin gene-related peptide, and neurokinin A, as well as glutamate. Trigeminal activation is associated with vasoactive neuropeptide release from nerve terminals. These mediators cause mast cell activator, nerve endings sensitization, fluid extravasation into the perivascular space (plasma protein extravasation) around the dura vasculature, and platelet activation (neurogenic inflammation). The latter sensitizes peripheral nociceptors (peripheral sensitization).

Neurons' responsiveness to painful - and non-painful - stimuli is increased. The receptive field expands, and pain is felt by the head over much of the skull. This results in hyperalgesia (increased sensitivity to pain) and cutaneous allodynia (sensation of pain in the skin where no previous stimulus has caused pain). Normal rhythmic pulsations of the meninges (innervated by peripheral trigeminal neurons) are also interpreted as pain.

Neuropeptides that are thought to play a role in the pathogenesis of migraine include:

Serotonin is released from the serotonergic nuclei of the brainstem and is thought to play a role in migraine. The exact mechanism, however, remains controversial. Serotonin levels may decrease during a migraine attack, leading to a deficiency of the serotonin pain-relieving system. This reduction may help activate the trigeminal nerve system, thereby exacerbating migraine

symptoms. Serotonin may act through direct action on cortical vessels, central analgesic pathways, or cortical projections from the serotonergic nuclei of the brainstem.

Calcitonin gene-related peptide: CGRP is abundant in the neurons of the trigeminal ganglion and is released from peripheral and central nerve endings. CGRP is secreted by the trigeminal ganglion. When released from peripheral nerve endings, CGRP stimulates the increased synthesis of nitric oxide and subsequent sensitization of the trigeminal nerve. CGRP acts as a potent vasodilator of cerebral and dural blood vessels, causing neurogenic inflammation and mediating the transmission of pain from the trigeminal vessels to the central nervous system.

PACAP may also play an important role in the mediation of migraine attacks, as its concentration increases during attacks and infusion of PACAP may induce migraine in susceptible patients.

Classification

Migraines are the primary cause of headaches. This means that there is no obvious cause for the headache, such as a brain tumor, brain injury, or inflammation. Migraine and related conditions are classified by the International Headache Society (IHS) primarily in terms of the aura.

Migraine without aura
Migraine without aura, also known as common migraine, is the most common form of migraine, accounting for 80-85% of migraine. It occurs when there is a history of at least five headache attacks that meet at least two of the four main criteria:

One-sidedness, possible side change
Medium or high-intensity
Crippled nature
Increased by exercise
In addition, there must be at least one other vegetative symptom, nausea, light or sound phobia.

In the absence of aura, restlessness, agitation, and mood swings may predict a seizure an hour or two before. In two-thirds of cases, the pain is unilateral, throbbing, increases with movement, and can last from four hours to three days without treatment. It may be accompanied by nausea and vomiting (80%), sensitivity to light (60%), sound (50%) and smell (< 30%).In children, the attacks may be shorter and the pain may be bilateral. In women, migraines without aura are often related to the menstrual cycle. Newer classifications therefore distinguish between menstrual migraine, non-menstrual migraine, and menstrual migraine.

Migraine with aura, also called classic migraine, is accompanied by a variety of neurological symptoms:

Visual disturbances
Visual field loss
Scotomas
Flashes of light
Colored, shiny, zigzagging lines
Micropsia, macropsia (perceiving oneself or the surroundings as being of a different size)
Disturbances in skin sensation
Loss of sensation
Numbness
Speech disorders
Movement disorders, paralysis

The average duration of symptoms is 20-30 minutes, but can sometimes exceed an hour. During or within an hour of the aura symptoms, the headache phase begins, which may be accompanied by nausea, vomiting, and sensitivity to light, sound, and smells. This phase may or may not occur. According to the IHS, migraine with aura occurs when: Temporary visual, sensory, and speech disturbances symptoms appear or disappear within 5-60 minutes.

Classic aura of migraine
The most typical form of migraine aura is the classic migraine aura. In many patients, this is followed by a non-migraine headache without the typical migraine symptoms.

Classic aura, without headache
This migraine aura without headache occurs in a minority of patients. It occurs mainly in men. Over time, with age, the attacks may develop into headaches.

Familial hemiplegic migraine
In addition to the above symptoms, in rare cases, temporary hemiplegia may also accompany migraine symptoms. This symptom is more common in some families. It may also be accompanied by coma, fever, and confusion. Familial accumulation is identified when first- or second-degree relatives also suffer from migraines. Therefore, inherited mutations on chromosomes 1, 2, and 19 are responsible for the familial accumulation. There are three types: type I (FMH1), type II (FMH2), and type III (FMH3).

Sporadic hemiplegic migraine
Symptoms of sporadic hemiplegic migraine are similar to those of cumulative hemiplegic migraine, but there is no family history of these symptoms. No genes are

associated with this form. Its prevalence is comparable to that of familial cumulative hemiplegic migraine. This type is more common in men.

Basal migraine

Basal migraine occurs in young people. Its typical symptoms may differ from other migraines with aura and include dizziness, tinnitus, hearing and speech disturbances, binocular diplopia and other visual disturbances, ataxia, confusion, or bilateral paresthesias. In some cases, there is a complete loss of movement that lasts for 2–30 minutes, reminiscent of locked-in syndrome. Only the eyes can move vertically (Bickerstaff syndrome). The aura and headache are bilateral.

Retinal migraine

Retinal migraine is identified by unilateral visual disturbances that occur with or within an hour of the onset of a migraine.

Vestibular migraine

Dizziness with head movement is seen primarily in migraine patients, especially those with aura. Dizziness is more likely to occur with head movement during the absence of seizures, but may also occur when watching moving objects or even when watching television, even when the head or eyes are not moving. Symptoms can range from simple unsteadiness to severe dizziness that lasts for days. It can be accompanied by tinnitus and ringing in the ears, but dizziness episodes do not necessarily have to be accompanied by headaches. Because severe dizziness can be caused by other conditions that are often more serious or untreatable, vestibular migraine can be diagnosed only after these conditions have been ruled out.

Sign and Symptom

Prodrome
Some patients feel the onset of an attack a day earlier, mainly because mood changes can alert them to an impending attack. This is known in the literature as prodrome. Typical symptoms include lethargy, unusual fatigue or a marked and unexplained feeling of excitement and euphoria, increased appetite, and craving for carbohydrate-rich foods and chocolate.

Aura phase
Around a quarter of seizures are caused by a visual disturbance called migraine, in which part of the visual field appears only as a black or flickering silver spot, or as flashing spots or bright, possibly colored zigzag lines, partial loss of vision, loss of field vision, tunnel vision, spherical or star-shaped appearance. These symptoms develop gradually and usually disappear within half an hour. Some artists are inspired by these visual phenomena.

Less commonly, there may also be numbness of the limbs, weakness, difficulty speaking, or dizziness. In migraine, these are also called auras. Some people have aura when an epileptic seizure occurs, sometimes only headaches without aura, and sometimes only aura symptoms. The headache occurs within an hour of the aura symptoms disappearing.

Migraine aura is characterized by migration or transformation of sensory and visual dysfunction. Migration of the dysfunction causing the symptoms can also be observed in the brain by special tests. Its slow onset and disappearance and its dynamic changes make it easily distinguishable from other neurological disorders such as epileptic seizures. The aura does not damage brain tissue and does not last more than an hour.

The headache stage
The pain caused by aura symptoms can range from mild to almost unbearable and is often throbbing and spasmodic. In 70% of cases, it is unilateral, affecting mainly the forehead, the back of the head, and around the eyes. It is almost always accompanied by nausea (80%), but at least by loss of appetite (more than 80%), sometimes vomiting (40-50%), and increased sensitivity to external stimuli (light 60%, sounds 50%, smells 50%-10%). Movement can aggravate the pain. Duration varies from one hour to three days. In children, the attacks are short-lived and often occur on both sides of the forehead and temple. They are more prone to noise sensitivity, dizziness, and imbalance. Some forms of migraine do not have a headache phase.
During a seizure, most patients can barely carry out their daily activities and are best able to lie down in a dark, quiet, calm room where no one disturbs them.

Postdrome phase
The headache may be followed by several days of fatigue, muscle warmth, and muscle aches. Sufferers may feel "run down", but some relax and recharge after an episode.

Causes

The exact cause of the disease is unclear, but the suspected disorder is caused by spasmodic contraction and dilation of the brain's blood vessels and a wave-like brain dysfunction that spreads from the back to the front.

As its prevalence in industrialized countries has doubled or tripled in the last four decades, environmental and lifestyle factors are thought to play a role. In sensitive people, certain substances and situations can trigger seizures. These include hormonal changes, stress, sleep, and food. These factors vary from person to person and can be detected by keeping a headache diary.

The most common triggers of epileptic seizures are stress, disturbed lifestyle, insufficient or excessive sleep, and the environment. In some patients, seizures occur after periods of stress, for example at weekends. The most common environmental stimuli that trigger seizures are weather and odors.

In women, even hormonal changes linked to the menstrual cycle are among the more common causes of seizures. Half of women with migraines have this problem. The most vulnerable period is the late luteal phase or the break from taking the contraceptive pill.
Two-thirds of migraine sufferers think there is a link between eating certain foods or taking recreational drugs and migraine attacks. The most common of these is alcohol. Seizures are also often triggered by foods containing glutamate, tyramine, histamine, and serotonin, and stimulants such as red wine, chocolate, and cheese. Coffee is also often blamed.

Many patients specifically crave certain foods or recreational drugs in the earliest stages of a migraine attack, the day before the onset of aura and headache (the prodromal stage), and mistakenly attribute the attack to them. A causal link between intense cravings for certain foods and the onset of migraine has not yet been established. Certain drugs, in particular nitric oxide-releasing vasodilators, can also trigger seizures.
MSG is found in many foods and is mainly used in Asian (Chinese, Japanese) foods.

Genetic causes
As some types of migraine run in families, it is assumed that certain mutations play a role in their development. Defects in three different genes have been identified as the genetic cause of familial hemiplegic migraine.

Type I familial hemiplegic migraine is caused by a defect in the CACNA1A gene on chromosome 19. This gene encodes the calcium channel Cav1.1.
Type II familial hemiplegic migraine is caused by a defect in the ATP1A2 gene on chromosome 1. This gene encodes sodium-potassium ATPase.
Type III familial hemiplegic migraine is caused by defects in the SCN1A gene on chromosome 2, which encodes the sodium channel.
Defects in some genes are also associated with epilepsy.

Migraine with aura is more common in patients whose atrial wall maintains oval openings, so it is assumed that both are caused by the same genetic defect. Similar hypotheses have been made about the relationship between migraine and depression.

Differential diagnosis

To correctly diagnose migraine headache, it must be differentiated from other types of headaches and neurological problems. Differential diagnosis is the process of studying different symptoms and factors to see if they fit with other disorders. This complex process ensures that patients receive the most effective and appropriate treatment for their condition.

The diagnostic features of migraine patients include:

Chronic paroxysmal hemicrania
Dissociative syndromes
Meningitis
Cluster headache
Cluster migraine headache
Tension-type headaches
Encephalitis
Subarachnoid/intracranial hemorrhage
Temporal/giant cell arteritis
Tension-type headaches typically present as bilateral pain lasting from 30 minutes to 7 days. Patients often experience pressure or tightness but typically remain active without associated symptoms.

Cluster headache is usually associated with unilateral sudden onset pain around the eye or temple. These headaches intensify within minutes and become intolerable and constant, lasting up to 3 hours. Other associated symptoms include watering (tearing) and redness of the eyes, nasal congestion (runny nose), pallor, sweating, drooping of the eyelids and pupil on the affected side. These symptoms are collectively known as Horner's syndrome. Cluster headaches are often caused by alcohol consumption.

Diagnostics

The investigation of migraines usually starts with a medical consultation and diagnostic tests. Below are some important steps in the investigation of migraine:
Medical history: the doctor interviews the patient about his or her medical history, including the type, frequency, intensity, symptoms, and triggers of migraine episodes.
Physical examination.
Laboratory tests: In rare cases, laboratory tests may be performed to assess the underlying causes of migraine, such as hormone levels or the functioning of physiological systems.
Imaging tests: In rare cases, imaging tests such as CT or MRI scans of the head may be used to rule out other neurological problems in migraine, including brain tumors or vascular accidents.
Making a migraine diary: Patients are advised to keep a migraine diary in which they record the frequency, intensity, triggers, and treatment of their headaches. This can help the doctor to understand the migraine and choose the appropriate treatment.
Migraines have no consequences, but in the case of aura migraines - when vision, speech, and movement problems occur - the risk of stroke may increase, but this depends on the severity and frequency of the migraine, so it is always advisable to see a specialist if you have a migraine!

Treating migraines

Migraines can very rarely be relieved by conventional treatments with non-steroidal anti-inflammatory drugs. The primary cause of pain in migraine is not inflammation, but abnormal activation of the nervous system, the pain-sensing system. For this reason, the complaint cannot be resolved with specific drugs specifically designed to relieve migraines, which are typically triptans. These should be accessed as early as possible, at the onset of the attack, because the release of toxic substances in the brain that cause the complaint can only be prevented by prophylaxis.
If there are four or fewer attacks a month, then seizure management is appropriate, which means that non-steroidal anti-inflammatory drugs can be taken alongside triptan-based drugs. No special effort is typically needed to relieve nausea and vomiting; specific migraine medication taken in time will usually prevent these symptoms too. If the onset of migraine is so rapid that oral medication is not effective, a nasal spray may be used or the patient may self-administer the medication. In addition to medication, it is also important to pay attention to lifestyle to help avoid the causes of migraines. This is important because you cannot take medication indefinitely.
Antibody therapy is now used to treat migraines. Its effect is based on the idea that CGRP released by migraine binds to a receptor in the brain, but if the receptors are blocked, the CGRP cannot bind to anything and migraine cannot develop. This treatment should be used once a month.

Osteopathic neurological examination

Many practicing neurologists do not do all the tests and do not follow the same sequence.
Based on the clinical question, the anamnestic data, and the patient's complaints, a multifaceted examination can be planned.
This is because a complete, detailed patient examination is not always appropriate for all patients.
In this section, I describe the steps of the neurological examination.
Whose practice may be relevant to practicing physicians and osteopathic students. A number of factors contribute to this.
There is a diagnostic component that is relevant to a particular topic, but commonplace.
Rare in real life. Only specific aspects of these specific procedures are discussed. I think it is medically significant.
These tests provide a broader insight into the patient's neurological condition than focusing only on migraine tests. In the case of migraine and headaches, a neurological examination with an osteopathic approach may be helpful. It is important to examine the exit points of the brain nerve as they can also cause headaches if compressed.

Basic steps of the neurological physical examination
Taking a comprehensive and detailed history. Complete and detailed documentation of events.
How did the complaints arise? Acute?Subacute?Chronic?Progressive?Paroxysmal?
Formulation of a research hypothesis: in which area is the damage located?
In the central nervous system? In the periphery? Both?
Which organism might it consist of? Brain? Brainstem? Spinal cord? Root? Plexus? Nerve? Muscle-Neuron?Muscle?
What can cause the lesion? Vascular? Tumoral?Infectious?Neurodegenerative?Toxic?Functional?
Does the physical exam support the theory?
Differential diagnosis. Limitation of possible diagnoses.
What additional tests or instruments are needed to support the theory?
Is it possible to perform any imaging or physical examination?
If physical examination, instrumentation, or imaging does not support the diagnosis.
Hypothesis, attempt another hypothesis.
In some cases, just observing the patient, and recording changes in symptoms.
The disease may be characterized by a longitudinal progression.
Determining the final diagnosis.
Is the severity of the disease taken into account?
What procedure is possible?
What is the diagnosis of the disease?

Neurological examination is unknown to the majority of patients. For an effective partnership we recommend that you always discuss with the patient what you will do and what you expect from them. It is worth considering targeted and flexible examinations for the convenience of the patient. Let's do it. If, for example, the patient is in a lying position, we will assess the lying examinations first. If the patient is in an active position, the patient should be examined in the

first instance for any conditions (e.g. signs of meningeal stimulation (e.g. lower limb coordination tests).

Inspection
Like the physical examination in internal medicine, the neurological examination is the first and most important component of the examination.
The component is observing. By observing it, we can immediately determine the patient's overall health. Skin color may be suspected before the diagnosis of an internal medicine disease (e.g. jaundice due to liver disease, cyanosis, respiratory failure, and pale skin may indicate possible shock). After the loss of consciousness or trauma, the aim is to localize signs of external damage and lateral tongue sting. The ability to judge is essential.
In addition, the patient's movement pattern is observed, which facilitates a more accurate assessment of subsequent muscle loss. Also, observe the patient's gait. How fast? On a wider basis? There are ambiguous signs? Joint movement of the arms? Observe the patient's expressions, actions, and words.

Record anamnestic data
Taking anamnesis includes tracing previous illnesses and treatments. In many patients
other important factors must be consider: hetero-anamnesis is also necessary (for example, in cases where seizures are triggered by brain disorders, dementia, increased or decreased severity.
Family history is crucial in the diagnosis of hereditary diseases. Social
Social history (addiction, diet, living conditions) increases the likelihood of developing certain diseases.

Touch
In degenerative diseases of the spine and scoliosis, it is important to look at the spinal column, the palpation of the paravertebral muscles, and palpation along the entire length of the spine. For example in dystonia, palpation of the paravertebral muscles helps to identify the abnormally functioning muscle group identification of the affected muscle group. Also, palpation can be used to examine the trophic properties of muscles.

Symptoms of meningeal agitation

Neck strap
Cervical decompression should only be considered in the event of cervical spine injury, fracture, or joint instability. The likelihood of this scenario is discarded. Otherwise, it could lead to severe myelopathy! During the examination, the patient should be asked to lie horizontally and release tension. Remove the pillows under the patient's head. Place the palm of the hand under the patient's head and gently lift it. There is usually no problem lifting the patient's head.
In positive cases, there is resistance to tilting the head forward while the patient acknowledges the pain.
If the patient reports only a headache but no stiffness of the back of the head, the test is also should be considered negative.

If head movement is also consistent with steady tension, then we are talking about cervical rigidity, which can also be present in Parkinsonism, dementia, and irritability.
Lhermitte's signature.
Rapid stretching of the patient's head towards the spine produces an "electric" sensation, causing paresthesia. Often multiple sclerosis, neck tumors, and Arnold-Chiari syndrome are also affected. It can occur with developmental abnormalities. It also lacks signs of meningeal excitability.

Kernig signal
There are two ways to test:
The patient lies horizontally, with the hip and knee joints flexed while the patient's feet and knee joint are in the knee joint position. Both lower limbs are examined separately. The patient's leg is straightened while the hip remains immobile. It is considered positive if, on extension, resistance is noted with non-root pain.
The Kernig sign can be tested in a similar way to the Lasegue sign. The patient lies horizontally with the hip and knee joint extended. The lower limbs are lifted passively one at a time so that in such a way that the knee remains extended. If positive, the patient complains of non-root pain or bends the knee to a certain degree. In contrast, in a positive Lasegue's sign, the pain is radicular and typically occurs on one side only and there is no knee flexion.

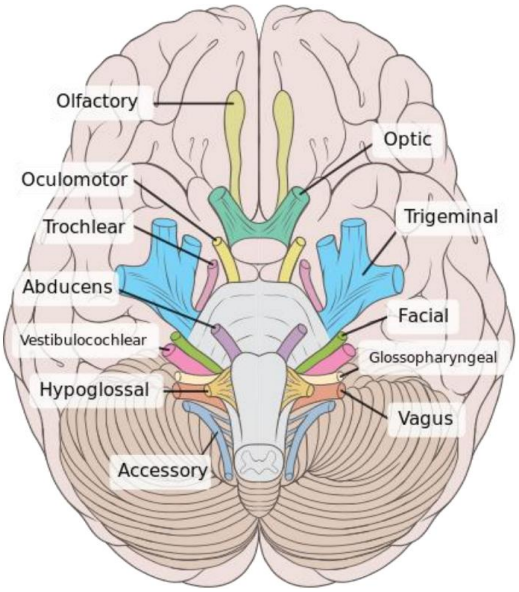
Brudzinski signal
The patient is placed horizontally for the test. As the head is bent forward, the neck flexion is increased, resulting in may be accompanied by flexion of the lower limbs at the knees and hips.

Evaluation of meningeal excitement signals

The presence of meningeal signs of agitation is most often associated with meningitis. Low cell count meningitis or immunodeficient patients, meningeal signs often remain negative
even in microbiologically confirmed infections. So meningitis is the presence of meningeal excitatory signs and the absence of meningeal signs. If meningitis is suspected clinically, a CSF examination should be performed even if the meningeal signs are not positive.

In addition to meningitis, they may also be positive in the following conditions:
Subarachnoid haemorrhage
Cerebrospinal fluid hypotension (e.g. after lumbar puncture)
Tonsillar impaction
Cervical tumors or metastases
Meningismus (systemic infection but CSF remains cell-free).

Brain nerves



Brain nerves I and II are not considered to be true brain nerves, they do not originate from the brain stem. Exit of brain nerves III and IV: mesencephalon(midbrain).
V-VIII exit from the pons.
IX-XII exit the brain nerves: the medulla oblongata (medulla oblongata)
There are purely sensory (I, II, VIII), purely motor (III, IV, VI, XI, XII), and mixed (V, VII, IX, X) cranial nerves.
Only parasympathetic fibers exit the brainstem (III, VII, IX, and X). Sympathetic fibers are of spinal origin and travel through the carotid branch system in the vascular wall to different parts of the head.

Localization of brainstem lesions

Inferior motoneuron lesion, peripheral lesion = lesion of the motor nucleus, or its axon, or the neuromuscular junction, or the muscles supplied by the cranial nerves.
The central lesion, supranuclear lesion = Damage to structures above the motor nucleus.
A lesion located in the brainstem (e.g. lacunar stroke, cavernoma, small tumor) can also cause peripheral damage if it also affects the motor nucleus of a nerve (nuclear lesion). In other words, lesions with a certain localization in the brainstem (i.e. in the central nervous system) can also cause a lesion that is clinically peripheral if it also affects the motor nucleus of the affected nerve.
Brain nerve damage is not caused by spinal cord damage, i.e. in the case of brain nerve damage, the lesion may be in the cerebellum, brainstem, brain nerve, or neuromuscular junction, possibly originating in the cerebellum and affecting surrounding structures.
Lateral brain nerve damage and motor or sensory damage to the motor or sensory limbs of the brain on the lateral side of the brain may usually indicate supranuclear damage.

Lateral brain nerve damage and motor or sensory damage affecting the left limbs may usually indicate brainstem level damage (brainstem alternating syndromes or hemiplegia alternates).
Hemiplegic V-VIII nerve damage and motor or sensory damage to the contralateral limbs may usually indicate pons or cerebellum-little bridge angular damage.
Hemiplegic IX-XI nerve damage without motor or sensory damage to the contralateral limbs may indicate hemiplegic foramen jugulare damage (e.g. thrombosis, tumor).
Hemiplegia III-VI with motor or sensory damage to the contralateral limbs without sensory or sensory impairment, may indicate a hemilateral sinus cavernosus lesion (e.g. thrombosis, tumour, inflammation).
Compression in the area of the brain nerves or other nerve damage may cause migraine symptoms.

Basics of Carnio-Sacral Osteopathy

The "craniosacral system" is defined by five factors, which in their common function constitute the primary respiratory mechanism (PRM):

1. mobility of the bones of the skull

2. involuntary movement of the sacrum between the two iliac blades

3. mobility of the intra- and extracranial membrane

4. fluctuation of the liquor

5. specific movement (motility) of the brain and spinal cord

The primary respiratory mechanism = PRM

The term "primary" means that this mechanism is directly related to the internal tissue respiration of the central nervous system, pulmonary respiration, and other bodily functions. The PRM already exists intrauterine, i.e. before the onset of pulmonary respiration.

"Respiratory" refers to a rhythmic, breathing-like course in which metabolic processes occur. These metabolic processes initially have an intracranial effect and then affect the entire body via the nervous system and the cerebrospinal fluid. In contrast to pulmonary respiration, rhythmic draining movements are involuntary and autonomic processes. There are different theses on how this pulsating movement is generated.

The term "mechanism" refers to a phenomenon that consists of different parts that together form a whole, namely the motor of the craniosacral rhythm. The term CRI (cranial rhythmic impulse) is a synonym for PRM. It comes from the French term MRP (mécanisme respiratory primaire), which means the same. The rhythmic pulsating movement, which is felt in the skull and throughout the body, is also called craniosacral inspiratory and expiratory movement (expansion and retraction movement). The frequency of the rhythm is 8-12/min in adults and 12-14/min in children.

Mobility of the skull bones

While infants have 45 bones in the skull, the adult skull consists of 22 bones (28 including the accessory bones). We can speak of about 100 connections through the various cranial sutures (sutures). The living skull, like other bones, has a good blood supply and is flexible. This allows the skull to change shape.

The sutures provide the possibility of fine movements and respond to compression and decompression. The skull bones are capable of three-dimensional movement along the shape and course of the sutures. The starting point and center of the movements of the skull bones is the skull base, as we call the sphenobasilar synchondrosis, SSB for short.

Involuntary movement of the sacrum between the iliac blades

The dura mater encephalis, which forms the inner shell of the skull, exits through the foramen magnum and passes into the dura mater spinalis. The dura mater spinalis is connected to the foramen magnum of the occipital bone and to the sacrum at the level of S1 and especially S2 within the spinal canal. The movements of the cranial bones are thus transmitted to the sacrum through the anatomical connection (see the section on the dura mater spinalis).

Mobility of the intra- and extracranial membrane

The force generated by the motility of the nervous system and the fluctuation of the cerebrospinal fluid requires structures that are able to absorb and transmit it. These structures are found in the intra- and extracranial membrane system, which is formed by the dura mater.

The membranes consist of resistant, inelastic connective tissue. They control and limit the involuntary movements of the skull and sacrum. Sutherland called this membrane system a "reciprocal tension membrane" (RTM), which refers to the evenness of the force transmission. The tension developed at one location on the membrane triggers a reaction at another location on the membrane. (see the reciprocal tension membrane chapter)

Fluctuations of the cerebrospinal fluid

The cerebrospinal fluid (CSF) is secreted oncotically in the walls of the arterial capillaries of the CSF is rich in nutrients, and is finally reabsorbed back into the blood and lymphatic circulation by the venous CSF capillaries and lymphatic vessels.

The rhythmic production and resorption phases of the CSF create fluctuating motion.
The extension of the cerebrospinal fluid is not limited to the brain and spinal cord, but also extends to the continuation of the membrane, through the exit sites of the spinal nerves along the nerve sheaths towards the periphery. With the help of microtubules, the LCS then communicates with the extracellular space, the lymphatic system, and the fascial system.

The specific movement (motility) of the brain and spinal cord

Like all organs, the brain also has its own movement. This seems to be a developmental physiological condition. The hemispheres of the cerebrum form a ram's horn shape during embryonic development. The motility of the brain is a rhythmic up-and-down movement in this ram's horn shape. In addition to motility, other rhythms can also be felt on the skull, such as the heartbeat or pulmonary respiration.

Summary:
For optimal function of the PRM, all of the above factors must be in harmony with each other. Although there is no definitive scientific evidence to date, the following functional sequence is assumed: Vasomotor waves of the cerebrospinal fluid are transmitted to the meninges.
This transmits the pulsation to the sutures of the cranial bones and towards the sacrum, where the biomechanics of the cranial bones and the sacrum lead to a completely specific movement and result in a deformation within the bone. It is transmitted through the membranes to the exit points of the spinal nerves and the craniosacral rhythm extends to the extracellular space, the lymphatic system, the fascial system, and thus to the entire body.
The expression of the PRM is therefore the craniosacral rhythm.
If one or more parts of this function are damaged, the craniosacral rhythm is also disturbed.
The different structures can be examined and normalized with techniques specific to the dysfunction. The PRM coordinates and regulates the complex relationships throughout the body, resulting in a close and reciprocal relationship between the different body structures, sections, and systems.

Cranial bones, Craniometric points, and sutures

Cranial bones
There are 22 (23) bones in the skull, in addition to the accessory bones and the hyoid bone.
Neurocranium (brain skull)

1. Frontal bone unpaired (embryologically paired)

2. Temporal bone - paired

3. Parietal bone paired

4. Sphenoid bone - unpaired

5. Occipital bone - unpaired

Viscerocranium (facial skull)

1. Ethmoid bone - unpaired

2. Vomer bone unpaired

3. Palatine bone - paired

4. Zygomatic bone - paired

5. Nasal bone paired

6. Lacrimal bone - paired

7. Concha nasalis inf. - paired

8. Maxilla paired

9. Mandible- unpaired

The hyoid bone (unpaired) is classified as viscerocranium. For functional reasons, osteopathy can be further classified as:

Central line bones

Occipital bone

Sphenoid bone

Ethmoid bone

Vomer bone

Sacrum bone

All other parts of the skull are classified as peripheral bones.

Craniometric points

Craniometric points are used in medicine as cornerstones for measuring the skull. In cranial osteopathy, they help to orientate the skull and help to distinguish between different parts of the skull.

1. Nasion: in the middle of the frontonasal suture

2. Glabella: the area between the eyebrows

3. Ophryon: above the glabella

4. Bregma: the junction of the coronal and sagittal sutures 5. Vertex: the highest point of the skull

6. Inion: the external occipital protuberance

7. Opistion: the middle of the posterior arch of the foramen magnum

8. Gnathion: the lowest midpoint of the chin

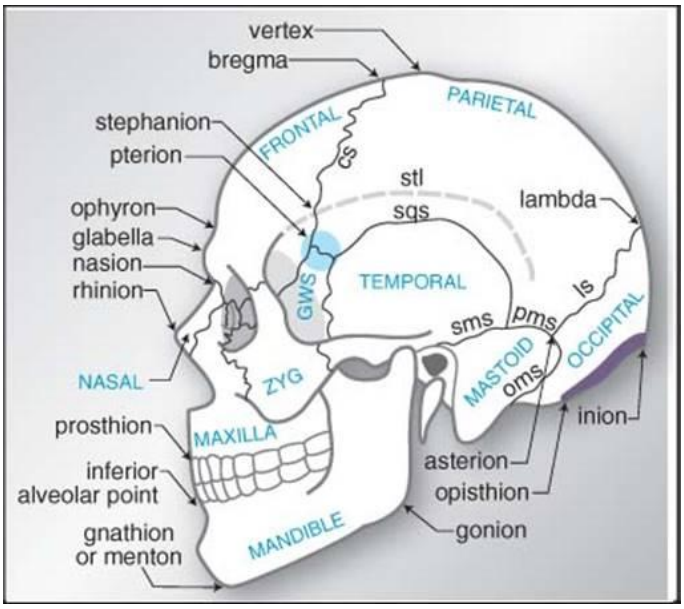
9. Gonion: the angle between the horizontal and vertical mandibular sutures

10. Lambda: the junction of the lambdoid suture and the sagittal suture

11. Pterion: the junction of the frontal, parietal, sphenoidal, and temporal bones

12. Asterion: the junction of the occipital, parietal, and temporal bones

13. Obelion: a well behind the vertex



Sutures Anatomy/Physiology

Cranial sutures are synarthroses, fibrous connections that connect the parts of the skull. Calcification of the cranial sutures begins in childhood. The flexibility of the sutures can also be detected in old age. The quality and quantity of the mobility of the sutures are determined by the bony structure of the suture.
Fibrous connective tissue is due to the flexible bridge formation between the individual bones, as well as a network of blood vessels and nerve endings as an expression of functional mobility. Before the age of 6, there are no sutures, the bones are located directly next to each other with their smooth surfaces.

Sutures of the external skull

From the side:

Sut.coronalis

Sut.parieto-squamosa

Sut.parieto-mastoidea

Sut.lambdoidea

Sut.occipito-mastoidea

Sut.spheno-squamosa

Sut.spheno-parietal

Sut.spheno-frontal

Sut.temporo-zygomatic

Sut.zygomatico-maxillary

Sut.fronto-nasal

Sut.fronto-maxillary

From the cranial side:

Sut.metopic

Sut.coronal

Sut.sagittal

Sut.lambdoidea

Due to its location, the frontal bone has two bone nuclei, the ossification of which is completed by the 3rd year of life. There are cases when the suture does not (completely) ossify.
An increased deformability of the two bone parts remains throughout life. Craniosacral therapy takes this into account and examines the frontal bone as a paired bone (therefore 23 bones).

Sutures of the skull base

Sut.petro-basilar

Sut.petro-jugular

Sut.spheno-petrosa

Synchondrosis spheno-basilar

All other sutural connections can be found in the description of the individual bone part.

Classification of cranial sutures

1. Sutura squamosa: squamous suture; adjacent bones overlap, grooves in the connection guide the direction of movement. The inward-facing edge covers the outward-facing edge.
e.g.: temporale covers the parietal in the case of sut.parieto-squamosa

2. Sut.lumbrosa = Sut. squamoserrata: In addition to the connection of the bones with each other, this form is accompanied by the overlapping of the bones along the oblique edge.
e.g.: Sut. coronalis and Sut. lambdoidea

3. Sut.dentata = dentate suture: e.g.: Sut.sagittal

4. Sut.plana: simple connection of rough bone edges
e.g.: between the maxillae on the palate and in the case of both ossa palatine (sut.intermaxillare, interpalatina)

5. Synchondrosis; not a suture in the true sense, but a bony-cartilaginous connection, which occurs on the skull only in the area of the SSB

6. Gomphosis: spike and socket connection (like the hub of a wheel) e.g.: Sut. petrobasilaris

7. Sut.serrata: sawtooth connection e.g.: Sut.temporo-zygomatica, Sut.fronto-zygomatica

8. Schindylesis: groove and feather connection, e.g.: between the sphenoideum and the vomer

Special features:
Os suturarum: surrounding bones at the sut.sagittalis and sut.lambdoid
Os incae: accessory bone at the occiput (also called os interparietale)

Orientation of sutures

The orientation of sutures depends on the overlap of the corresponding cranial bones. The suture surface of the overlying cranial bone faces inward, and the suture surface of the underlying cranial bone faces outward.

Pivot point:

In some sutures, the orientation of the suture planes changes, this location is called the pivot point. External sutures with pivot points.

1. Coronal suture
Runs in a semicircular arc between bregma and pterygium
Bregma area: Frontal > Parietal
Lateral part to Pterion: Frontal < Parietal

2. Lambdoid suture
Runs in a semicircular arc between Lambda and Asterion
Lambda area: Occiput > Parietal
Lateral part to Asterion: Occiput < Parietal

3. Sut.parieto-mastoidea
Runs almost horizontally at the end of the pars squamosa in the area of the Asterion
Pars squamosa area: Parietal > Temporal
Asterion area: Parietal <Temporal

4. Sut.occipito-mastoidea

5. Sut.spheno-squamosa

The seven fossae

1. Bregma
Anterior fossae fonticulus anterior = large fossae
Ossification in 1-3 years of age
Separates three bones: frontale/ two parietale

2. Lambda
Posterior fossae fonticulus posterior - small fossae
Ossification in the first three months
Separates three bones: occiput/ two parietale

3/4. Pterion
Anterior/lateral fossae fonticulus sphenoidalis
Ossification in from 3-6 to 9 months
Separates four bones: Frontale/ Parietale/ Sphenoidale/Temporale

5/6. Asterion
Posterior/lateral foramen = fonticulus mastoideus ossification from 9th month to 1.5 years
Separates three bones: Occipitale/ Parietale/Temporale

7. Obelion

Ossification of accessory fontanelle 1.5 years
Overlaps (>= covered, <= will be covered)
Bregma: Frontale > Parietale
Pterion: Frontale < Parietale < Sphenoid < Temporale
Lambda Occiput > Parietale
Asterion: Occiput < Parietale < Temporale

Treating these anatomical structures can reduce the symptoms of headaches. Treatment of craniometric points can have an effect on the central and peripheral nervous system.

Case studies

In the case studies patients diagnosed with migraine who are not on pharmaceutical therapy.

In my naturopathic practice, I have treated 24 patients with migraine.

They were examined and treated using osteopathy medicine methods.

Physical examination

Of the 24 patients with migraine, all had headache complaints every day. They had frequent headache complaints in different places of the skull. I found several cranial dysfunctions on the skull.
Both connective and muscular tissue around the craniometric points were tight. I also examined the cervical segments of C1-C2. Both connective and muscular tissues were tight in the sub-occipital region.Many sutures were blocked.

Treatment

The patients received 10 treatments.

The sessions were 40 minutes long and included 5-10 minutes of discussion about their condition. There were 30 minutes of cranial osteopath. I treated craniometric points and sutures of the skull.

Results

14 out of 24 patients had almost complete relief of migraine complaints.
They report that the migraine reduces by 80-90%.

By 4 patients have improved a lot but still have some migraine complaints.
They report that migraine reduces by 50%.

By 6 patient's migraine complaints have not changed.

Conclusion

My research shows that osteopathic medicine is effective for migraine patients. If the craniometric points are released the headache symptoms can decrease. The exit points of the cranial nerves from the skull also proved to be an important area. Treatment of these areas may be important in migraine patients. Further research on migraine patients with osteopathic techniques is worthwhile. I believe that with osteopathy medicine we can help people with migraine. Further research on the relationship between the craniometric system and migraine diseases is worthwhile.

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