Is Osteopathy combined with Hippotherapy a Sensible Therapeutic Measure for the Treatment of Children with Cerebral Palsy?

A Single Case Study

Master Thesis for the Degree of

Master of Science in Osteopathy

Presented to

The **Danube University Krems**

At the **Vienna School of Osteopathy**

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Berndorf, October 2008

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Statutory Declaration

I hereby declare that I have written this master's thesis completely independently.
Any parts taken literally or conceptually from any works by other authors, published or non-published, have been acknowledged as such. All sources and aids used in this thesis have been declared. I declare that this thesis with identical content has never before been presented to any other examination board.

October 2008

Abstract

This osteopathic study examines the effectiveness of osteopathy in a child with cerebral paresis. A single case study has been chosen because the test person, Matthias, is a child who suffers from cerebral palsy. These neurological symptoms can take many different forms. Therefore, it is extremely difficult to make comparisons with several children. Matthias has suffered from tetraplegia since birth. Both his legs and his left arm are affected. Matthias has received physiotherapy from birth onwards, switching later to hippotherapy. This is in effect a physiotherapeutic measure, whereby the three-dimensional movement of the horse is transferred to the rider.

I have chosen a withdrawal design (A-B-A) to permit Matthias to continue with hippotherapy and at the same time to allow me to examine the efficiency of osteopathy during the ongoing, additional hippotherapy. Each phase lasts for five weeks, i.e., the complete study encompassed 15 weeks. In the first and third phases, Matthias had hippotherapy once per week, as usual. In the second phase he was given additional osteopathic treatment.

The hypothesis of this study was confirmed, i.e., that Matthias' flexibility did indeed improve during osteopathic treatment, and that this effect can even be prolonged.

Osteopathy can, therefore, definitely be regarded as a sensible therapeutic method in Matthias' case. This conclusion, however, does not automatically apply to all children with cerebral palsy. Further orthopaedic studies on the clinical picture of cerebral palsy in children would be desirable, as few studies have yet been carried out in this field. It would also be extremely interesting to find a suitable means of measuring the increase in these children's wellbeing and quality of life after osteopathic treatment. Because I used the Smiley Scale, I was unable to distinguish Matthias' true sense of wellbeing, as he always described his condition as being "very good". Possibly, the use of a questionnaire with specific questions aimed at parents and children might provide more accurate information on these children's real feelings of wellbeing.

Key words: osteopathy, hippotherapy, infantile cerebral paresis (palsy), tetraplegia, single case study

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1. Introduction

This study originated from my osteopathic and hippotherapeutic work with children who suffer from infantile cerebral palsy. Having worked for several years at a school for children with special needs, I gained valuable insight into the actual needs these children have. In my own present practice, too, I have many of these children as patients. Working with them has always been, and continues to be, an extremely rewarding experience for me.

Different definitions exist for the concept of “infantile cerebral palsy”. Infantile cerebral palsy is a permanent, but not unalterable, dysfunction of posture and movement as a result of a prenatal, perinatal or postnatal cerebral functional disorder, occurring before the brain has completed its maturation and developing process. (Maurer 2002)

Cerebral palsy, or “CP” for short, is a concept, and not a disease (Carreiro 2004).

Further definitions can be found in Chapter 2.6.

The most common causes of infantile cerebral palsy are perinatal asphyxia and complications arising in connection with premature birth. (Volpe 1995)

Investigations suggest that the insult is intra-uterine in 40-50% of cases, peripartal in 30-40% and postpartal in approximately 10%. (Brown et al. 1974, Low et al. 1989, Volpe 1995) Ceney and Palmer (1997) came to the same conclusion.

The prevalence of cerebral palsy in Europe is 2 -3 in 1000 live births with wide variations in different weight classes. The statistics from a study made in Graz in 2002 dealing exclusively with premature births from intensive care with a birth weight lower than 1500g range between 10-20% (Maurer 2002)

The morphological cause of infantile cerebral paresis is always damage to the first motor neuron. Primarily, and in most cases, (85%) the pyramidal tract (tractus corticospinales) is affected, but extrapyramidal tracts (injury to the basal ganglia) or cerebellum (injury to the tractus cortocopontocerebellaris) can also be affected. Primary or secondary injury to cortical regions can also occur. (Maurer 2002). (See Chapter 2.7)

Which region of the brain is affected depends on the duration of the insult and the gestational age of the foetus, since the sensitivity of certain regions of the brain changes in pregnancy with the development of the vascular patterns in the brain. (Maurer 2002)

In the course of their development, children must learn new strategies for movement, and refine and integrate these strategies, in order to interact with their environment. (Maurer 2002)

This is effected by means of many different forms of therapy, including hippotherapy. Hippotherapy is a well-established, popular form of therapy for children with cerebral palsy. (See Chapter 2.7)

Children with cerebral palsy are usually treated with physiotherapy from birth onwards, as it is imperative for them to learn how to move, and keep secondary damage to a minimum. (Ong, Chong, Jap 2001) Later, mostly when they are of school age, they switch to hippotherapy, because this is more fun for them than indoor physiotherapy. The horse both inspires and motivates the children. (Lisinsky, Stryla 2001)

In my experience, knowledge of osteopathy is not very widespread among these patients. One reason for this may well be the financial aspect (Lisinski, Stryla 2001), since osteopathic treatment is not covered by compulsory health insurance. This fact also motivated me to write this study.

In this thesis, a combination of these two forms of therapy is used to treat a child with cerebral palsy, with the aim of optimising the therapeutic results and/or maintaining the progress achieved over a longer period of time, thereby giving the child a better quality of life.

Because the course and the forms of cerebral palsy in children vary greatly with the individual child (Feldmann-Winter et al. 2002) and because homogeneity as such does not exist, I have decided to adopt a single-subject design. (Logman, Hickmann, Harris, Heriza 2005)

For this reason, the measurement variables must be individually adapted to match the proband. The main problem children with cerebral palsy have is the raised muscle tonus which is expressed in spasticity, as in the case of my proband, Matthias.

In hippotherapy, the muscle tonus can be reduced by means of the regular, rhythmic movements of the horse. (Sterba et al. 2002) After this therapy, more gentle and efficient methods from the wide range of osteopathic treatments can be applied at various levels (structural, visceral, cranial and fascial). (Knox 2005)

Subsequent orthopaedic treatment can then work directly on the joints, since the muscles are then more relaxed, which makes the treatment less painful or unpleasant and hence easier for children to tolerate. Osteopathic experts believe this form of therapy is an excellent method for treating children with cerebral paresis. Statements by Carreiro (2003), Hayden (2000) und Hearsey (1994) confirm this. They state: “Growing brains have an enormous capacity to compensate. Osteopathic treatment can help to remove obstacles in the way of optimum compensation.” In my opinion, therefore, the earlier children receive treatment, the better chance they stand of successful development, since the brain is still capable of learning. In his study, Arbuckle (1955) repeatedly emphasises this fact. With the help of osteopathy, the

children are given more possibilities to adapt to their situation. Frymann (1993), too, worked with children with cerebral palsy. He states: “Cranial articular lesions and resulting membranous imbalances can be reduced successfully with osteopathic treatment.”
These statements are a compelling justification for my thesis, and I shall endeavour to give them a convincing scientific basis.

As yet, osteopathy is not generally included among the classic methods of treatment for children with cerebral palsy. Accepted methods include physiotherapeutic methods such as Bobath, Vojta and hippotherapy, as well as therapeutic concepts such as Castillo-Morales, sensory integration, Petö, Affolter and Frostig. (Maurer 2002) (See chapter 2.6)
Some studies, however, have examined and recorded the positive effects of osteopathy on motor skills and on the general condition of children with cerebral palsy. (See Chapter 2.7)

2. Basics

2.1 Classification of the Branches of Therapeutic Riding

In 1970 the professional association for therapeutic riding in the Federal Republic of Germany – the Authorised Board of Therapeutic Riding (Kuratorium für therapeutisches Reiten – KThR) was founded. The functions of this Board include the establishment and control of training regulations, coordination and organisation of training courses, further education for specialists, exchange of information, contact with the German riding union and professional associations, international exchange of experience, definition of scientific principles as well as cost regulation. (Gehrts 2006)

In order to fully understand hippotherapy, it is important to distinguish the three branches of therapeutic riding. There are three separate branches, which can, however, be inter-connected. These include:

hippotherapy
therapeutic riding and vaulting
riding for the disabled

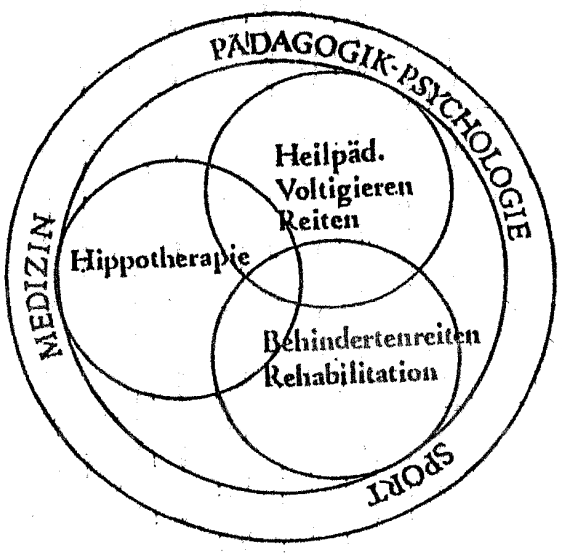


Fig. 1. Overview of therapeutic riding

paedagogics – psychology sport medicine
hippotherapy therapeutic vaulting/riding handicapped riding /rehabilitation

The three branches can be defined in detail as follows:

Hippotherapy is a medically prescribed physiotherapeutic treatment which utilizes the natural movements of the horse. (Wolf, 1996)

A more recent definition describes hippotherapy as follows:
Hippotherapy is a specialised physiotherapeutic treatment that makes use of the horse's unique three-dimensional movement impulses while walking in order to facilitate movement responses in patients sitting on the horse's back. (Strauss 2000, Debuse, Chandler, Gibb 2005)
This form of therapy complements and reinforces standard physiotherapy on a neuro-physiological basis. (Hellwig 2000)
In the process, the three-dimensional movements of the horse's back are put to use from a medical point of view. The patient is stimulated holistically, i.e., physically, emotionally, mentally and socially. This means that the patient's entire body must react to the horse's movements. The patient must adapt to the horse, trust it, and at the same time follow directions from the therapist. This neurophysiological treatment must be medically prescribed, individually adapted and designed for each patient according to the therapeutic plan. (Maurer, 2002)
Hippotherapy is given to people with special needs, with the intention of effecting a positive influence on the locomotor system. (Molnar- Mignon 1997, Tauffkirchen 1997) More on this in Chapter 3.1.!

The concept of "**Therapeutic vaulting/riding**" can be defined as a range of paedagogical, psychological, psychotherapeutic, rehabilitative and socio-integrative treatments with the aid of the horse for children, adolescents and adults who have various disabilities or disorders. (Molnar-Mignon 2000)
Teaching equestrian skills is not significant in this respect. What is important here is encouraging individual progress through the medium of the horse.
Remedial paedagogical therapeutic riding and vaulting, as in the case of hippotherapy, may only be carried out by trained individuals who, as a rule, are pedagogues. The difference between this and hippotherapy is that in this form of therapy, the actual physical disability is not in the foreground. The therapist is primarily working here with mentally challenged children and adolescents, or individuals with learning difficulties or behavioural problems,

disturbances in emotional development or in perception and speech, autistic behaviour or psychological and psychosomatic disorders. (Kapeller 1997, Spurny 1997, Orac 1997)

Handicapped Riding is a sport for handicapped persons of any age who are able to sit unaided on a horse and interact with it. It is a serious form of sport in which tournament status can be attained. To participate in sport means, in effect, despite a physical handicap, to participate in the normality of life and this is what gives the participants great pleasure. While sports groups for the disabled are usually somewhat self-contained, the horse as sports partner helps the disabled rider to overcome his/her physical limitations, and enables him/her to ride on an equal level alongside a group of healthy riders. (Heipert-Hengst 1980, Strauß 2006).
The sensitivity of the horse, which enables it to respond to children with feeble strength, is evident in its performance with individuals with deficiencies. The rider's limited ability to influence the horse's behaviour can be compensated by means of specially designed aids and appliances. (Rieger 1978)
Hippotherapy is often a stepping-stone on the way to practising riding as a sport. If the patient has completed hippotherapy and is physically qualified to ride as a sport, the next step is often equestrianism. (Strauß 2006)
Indications for handicapped riding are:
as a leisure activity, integration of the handicapped or of persons in poor health, personality development, maintenance of and increase in personal fitness, and rehabilitation. (Kapeller 1997, Heipert-Hengst 1980)

2.2 Definition of Hippotherapy

Since my study deals with osteopathy and hippotherapy, the prerequisites - the physical therapist, therapeutic horse and horse instructor - are defined in this chapter.
Only **physical therapists** with special supplementary qualifications are authorised to perform this form of therapy. A **therapeutic horse** and its **instructor** are also requirements.

The physiotherapist must have successfully completed two years of occupational practice or possess a supplementary qualification in neurology, as well as a basic knowledge of riding, before he/she is allowed to take part in a professional training course. The duration of the course is two periods of seven days, covering theory and practice.
At the end of the second part of the course there is a final examination to test theoretical knowledge, analysis of motion, riding, horse knowledge and care, saddling, use of the bridle and practical lungeing and riding. (Molnar-Mignon 2000, Strauß 2006, Gehrts 2006)

The therapeutic horse must be selected with great care. Key factors here are the horse's physical build, movement, temperament and performance. The horse should not be too large and should have a well-muscled back, to permit riding without a saddle. It should move smoothly and with a steady rhythm. Important, too, is a regular and diligent pace, and a smooth trot which enables the rider to sit easily. (Strauß 2006)

Equally important as the horse's exterior (outward appearance), however, are its character and temperament. A therapeutic horse must have a balanced personality. It should not be too timid or nervous. It must react to all kinds of human handling in a friendly way, and in addition, must be receptive and eager to learn. (Strauß 2006)

Moreover, the training of these horses is an extremely relevant factor. It guarantees the highest safety factor when performing therapy, and makes full use of the entire range of the horse's movements. (Hemmelmayer 1997)

The therapeutic horse, as every other dressage horse, should be worked regularly under saddle and by hand, in order to maintain its forward movement, tempo and flow. In addition to training, forwards riding and the ability to surmount small obstacles in the terrain are

important. Above all, work with the lunge is essential; here the voice must take the place of riding aids. (Hemmelmayr 1997)
For use in hippotherapy, the horse's patience, above all, must be trained. As a result, the horse becomes accustomed to standing completely still, even when confronted with uncontrolled noise or pounding extremities, if a patient is lifted on to its back. It learns to accept several persons in its close vicinity and even to carry two riders at once. (Strauß 2006)

The training of a therapeutic horse runs parallel to the schooling of a young horse. A (Class L) fully-trained horse can be retrained as a therapeutic horse in a relatively short time. Older, veteran horses are seldom suitable for use in therapy because their backs lack adequate flexibility. (Daes 1983)
The time factor is also important, i.e., the extent of time in which the horse can be used for therapy. Since the horse has to concentrate on its work, it cannot be used for hours. Daily use is not advisable either, since horses also need other activities.

The horse trainer must be a suitable person who is familiar with the therapeutic horse and who can follow the therapist's directions. He/she should be able to work in harmony with patient, therapist and horse. (Strauß 2006)

2.3 Effective Factors in Hippotherapy

A patient being carried on horseback for 20 – 30 minutes at a walking pace is not a really spectacular sight. Nevertheless, even at this slow forward pace, a great deal is happening. The patient has to process and compensate approximately 100 complex stimuli of movement per minute. The effects of hippotherapy on many disorders significantly surpass the effects of conventional remedial gymnastics. (Hellwig 2000)

2.3.1. Physical Effects

For a clearer understanding of the effects of the horse's movement on the patient, the forces which are thereby transferred to the patient or which develop are defined below.
The following physical forces have an effect on the patient while sitting on the horse:
-three-dimensional impulses of vibration/oscillation
-velocity
-centrifugal force

The horse moves rhythmically and harmoniously. At a walking pace, we are speaking of a **three-dimensional impulse of vibration/oscillation** from the horse's back, in which 90 – 100 impulses per minute are transferred. This three-dimensional equine movement is, in effect, a method of torso training for the patient, which simulates walking. (Tauffkirchen 1997, Sterba 2007, Strauß 2006)

In this exercise, the pelvis moves around the sagittotransversal axis on the frontal plane. This causes a caudal dipping of the pelvis, alternately right and left, of approximately 5 cm. This shifts the pelvis 7 – 8 cm to the side, causing a lateral flexion of the spine (lumbar vertebral column) relative to the sacrum of 16°. This results in abduction/adduction of the hip joints (Tauffkirchen 1997, Sterba 2007, Strauß 2006)
The movement around the frontosagittal axis causes an 8° rotation of the pelvis around the torso's longitudinal axis. Rotation of the spine around the torso's longitudinal axis is 19°. This results in internal and external rotation of the hip joints.

Movement around the frontotransversal axis causes a dorsal tilt of the pelvis, which springs back again. This causes extension and flexion of the hip. (Tauffkirchen 1979, 1996, Riede 1983, Strauß 2006)

These movements are transferred from horse to rider. (Ill. Nr. 2)

In 1971, Baumann already recorded this in a most informative film study, using marking points on the iliac crest, spinal process and shoulder blades. He verified that these movements of the pelvis, spine and shoulder girdle regions correspond to the physiological gait of humans. (Gehrts 2006)

It is observed that when the pelvis is centred the spine is stretched and the effort of maintaining an upright position and balance causes continuous variations in the interplay of muscles in the torso - the stomach and back muscles - and also the muscles of the shoulder girdle. The rotation of the spine is effected by a movement of the shoulder girdle counter to the pelvis. In this way, reflex posture and balance reactions are produced and the perception of posture and movement is trained. Hence there is a twofold result: improvement of head - torso control and activation of the erector muscle groups.

At the same time it is evident that the easing of the patient's own body weight when straddling the horse has a positive effect on the freedom of movement in the torso and the extremities.

The rhythmic stimuli from the horse's back facilitate even movement and guarantee continuous alternation between states of tension and relaxation in the active gripping muscles. Specific exercises on the horse make higher demands on the patient's balance, coordination and spatial orientation. In this way, muscle training in the sense of stretching, relaxation and strengthening is performed. (Rieger 1978)

Equally effective are the forces of **velocity,** by virtue of the variations in speed, and **centrifugal force,** by virtue of the changes in direction, both created by forwards movement. In hippotherapy, the patient mostly sits on the horse's back with no saddle and has to adapt to the constantly changing movements of the horse. He/she is supported and corrected by one or two therapists. (Ölsböck 1997, Gehrts 2006, Hellwig 2000)

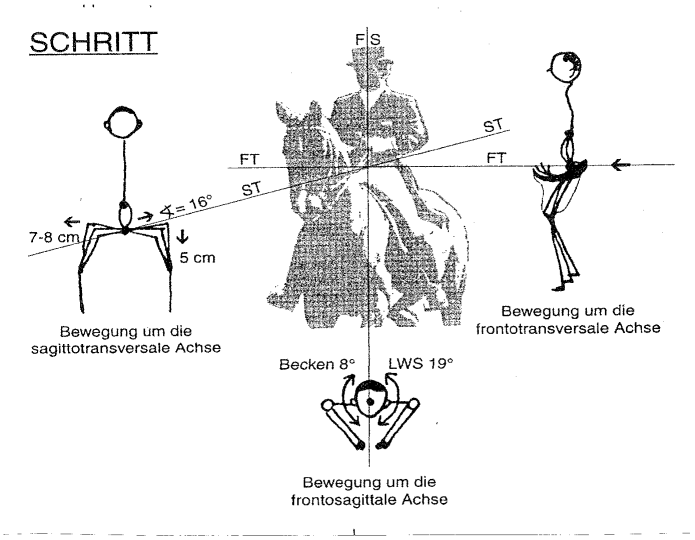


Fig. 2. Illustration of the three-dimensional movements of the pelvis on horseback

WALK/PACE/ movement around the sagittotransversal axis/ hip 8° spine 19°/ movement around the frontotransversal axis movement around the frontocapital axis

The gait simulation training of the torso, of the ventral as well as the dorsal muscles results in synergetic muscle strengthening.

A further effect is the **mobilisation of the joints**, especially in the region of the spine, hip joints and shoulder girdle. The regular movement leads to **dynamic stabilisation** of the thoracic spine and improved breathing. (Sterba 2007). At the same time, non-stop **balance training** is being performed, as the patient constantly adapts to the horse's movements (Benda, McGibbon, Grant 2003)

Traction is produced by the patient's dangling extremities and his/her body weight. If the horse's back is too broad, however, this pendular movement is not possible.

Shortened muscles, especially the adductors, inner rotators and ischio-crural muscles are stretched by the patient's sitting astride the horse.

A further result is **improvement of abnormal movement patterns** and **suppression of tonic reflex activities**, this being in direct connection with the facilitation and automation of movements brought about by continuous repetition and changes of direction. (Debuse, Chandler, Gibb 2005) Thus, different corrective reactions are continually demanded and promoted.

Coordination and, therefore, **sensomotor training** is called for.

Sensory integration training implies the use of all the senses. (See Physiological Effects, P. 17) The brain must select, be sensitive to and process stimuli. (Meregillano 2004)

Breathing can also be successfully incorporated into the therapy. Breathing out reduces the tonus and this can be combined with voice exercises. (Ölsböck 1991, Tauffkirchen 1993)

2.3.2 Physiological Effects

The physiological factors in hippotherapy play a vital role in the patient's wellbeing.
In particular, activation of the sensory system with its functions of sight, hearing, smell, balance, skin and deep sensibility is extremely important. A rider moving at a walking pace has a pulse rate of 120 beats per minute, 124 beats per minute at a trot and 180 beats per minute at a gallop. (Tauffkirchen 1993)
A study was designed with the aim of documenting the heart rate response to therapeutic riding of children with cerebral palsy. The result was that the average resting, median and peak heart rate values, as well as the peak percentage heart rate reserve values, were significantly higher in the wheelchair-dependent group. (Dirienzo et al. 2007) This study shows the influence of hippotherapy on the patients' coronary-circulatory system. However, only eight children took part in the study over a period of 10 weeks. The small number of test persons is a point of criticism in this study.
Furthermore, hippotherapy brings about improvements in breathing and has a positive influence on abdominal, intestinal and urinary tract peristalsis, relaxation of the muscular system because of the horse's 1° higher body temperature, and activation of the sensory system.(Ölsböck 1997, Debuse, Chandler, Gibb 2005)

2.3.3 Psychological Effects

As a result of the increasing interaction between patient and horse, arising from increased sensory perception through intensive body contact, and from a gradual mutual understanding of each other's movements and forms of expression, modification of the patient's social and personal behaviour is possible. (Riesser 1997)

Children with cerebral palsy who have often lost all trust in themselves and their abilities because of countless negative experiences and frustrations from the past must first of all build up trust again and feel completely confident that the horse will carry and accept them. (Rieger 1978)

The gradual process of being able to assess the horse's movements and behaviour varies in time, depending not only on the patient's disability, but also on his/her sensitivity and attitude – basic inclination, fondness for animals, inherent sense of trust and degree of anxiety. It may also happen that a patient simply cannot adjust, mostly in the case of children with multiple handicaps. In such a case the therapy is terminated to prevent any negative or aggressive reactions arising. (Ölsböck 1997)

It must also be stressed that hippotherapy is greatly appreciated by parents because they are the best judges of the positive effects it produces.

Brauer, in his medical dissertation, (1971) states that this psychosomatic reciprocal relationship creates a state of relaxation and that the patient's willpower is activated through this "charging his/her batteries with joy" – an aspect which has also been mentioned by many other authors after studying patients' behaviour during therapy with and on the horse. (Rieger 1978)

In scientific studies by Zinke and Kröge (1969, 1973) the horse acts as a training agent and as a type of "probation officer," helping the patient to change and correct his/her behaviour successfully. Baumann and Danielcik /1971) too, point out the psychological influence of hippotherapy. The emotional-mental retardation and behavioural disturbances which can be a consequence of early physical handicaps should not be ignored when drawing up medical indications. Without question, the motivation generated by hippotherapy rates this form of therapy superior to other forms of treatment. It is also an important factor in overcoming resignation and therapy-fatigue, when progress is at a standstill after years of physiotherapy. (Rieger 1978)

In our day and age, human beings are controlled by technology, and a sick person or a person with special needs is becoming more and more a mere "case" - simply an object to be treated.

Hippotherapy, with its physical and psychological interactive aspects, fulfils all the requirements of a holistic method of treatment, which is no longer merely practical medicine, but which can present a basis of scientific facts. These, however, still need to be further examined and developed if they are to withstand necessary criticism in medical circles. (Rieger 1978)

2.4. Indications / Contra-indications for Hippotherapy

Hippotherapy is indicated primarily for children from their fourth year on (depending on their size), but also for adults. My test person, Matthias, has been receiving this form of therapy regularly since the age of five.
The therapy has proven especially effective in treating diseases of the central and peripheral nervous system, such as, e.g. cerebral palsy* (CP), central hypotension*, multiple sclerosis, myelominigocele*, conditions after strokes and craniocerebral trauma, transverse lesion of the spinal cord with paraplegia, spina bifida*, diseases of the spinal cord and minimal cerebral dysfunction. (Tauffkirchen 1989, Rieger 1987, Strauß 2006, Gehrts 2006)
It is also exceedingly effective in treating musculoskeletal disorders, such as, for example, vertebral disk problems, hip disorders, scoliosis, postural weakness, muscular dysbalance or conditions following amputation.
Further disorders for which hippotherapy is indicated are muscular and metabolic disorders, syndromes, chromosomal anomalies, as well as gynaecological problems or pelvic floor insufficiency occurring more frequently in adults. (Tauffkirchen 1989, Gehrts 2006)
It is mainly indicated for cerebral palsy, as in the case of Matthias in this study, and in addition, for multiple sclerosis, postural weakness and defective posture, especially in children and adolescents. (Tauffkirchen 1989, Rieger 1978, Strauß 2006, Gehrts 2006)

CONTRA-INDICATIONS for hippotherapy would be insoluble spasticity, which would prevent the patient from sitting astride a horse, as well as luxation of the hip joint. Lack of stable head and/or torso control or atlantoaxial instability, post-traumatic operative correction of the spine (which can also affect children with cerebral palsy due to excessive curvature of the spine) or simply, excessive anxiety or panic caused by the horse.
Further contra-indications would be an allergy to horse hair, coronary-circulatory insufficiency or inflammatory processes in the spine. Another contra-indication would be an active phase in multiple sclerosis or osteoporosis, which occur not only in old age, but are also a possibility in cerebral palsy due to the lack of pressure on the bones. Another excluding factor would be incipient thrombosis or embolism as well as haemophilia.

* See chapter 8.2

Opinions differ in the case of slipped disks, and here the doctor's instructions must determine the form of treatment, as in the case of epileptic seizures, due to the higher risk of accidents. If the patient remains free of symptoms with medication for three years, however, participation in hippotherapy or riding as a sport for the disabled is a possibility. (Straußß2006, Gehrts 2006, Rieger 1978, Tauffkirchen 1998)

From a medical point of view, none of these contra-indications applied to Matthias, my test person!

2.5 Osteopathy – Hippotherapy and their Development

2.5.1 The Development of Osteopathy

Palpation or the manual examination to diagnose blockages of movement was developed a good hundred years ago in America and spread to Great Britain at the beginning of the 20th century. In China, palpation has been in use for thousands of years. (Sammut, Searle-Barnes 1998)

Osteopathy was developed by von A.T. Still in the second half of the 19th century, in the mid-west of the United States of America. He contended that because God had created flawless human beings, it must then follow from a mechanical point of view that total health must exist if the body structures are correctly ordered and function normally and if at the same time neither the blood flow nor the transmission of nerve impulses is restricted. Still, therefore, tried to understand the functions of the support and locomotor system and the intestines on the basis of the knowledge of anatomy and physiology of that day. Using digital manipulation, he improved the mobility of the joints and loosened the muscles. He regarded osteopathy not only as a form of treatment for musculoskeletal disorders, and also treated many other forms of illness with it. (Sammut, Searle-Barnes 1998)

In 1892 Still opened a School of Osteopathy in Kirksville and trained a rapidly increasing number of students. (Searle-Barnes 1998)

In the 70's, Audrey Smith developed this concept further.

From this the current concepts of osteopathy developed, which were formulated as principles on the basis of Still's original ideas and are still valid today. They are:

1. Movement is life.
2. Structure and function mutually influence each other.
3. The law of the artery. The unrestricted transport of body fluids is an absolute prerequisite for health.
4. The body is a unit.
5. The self-healing potential of the body (Ligner 2002)

2.5.2 The Development of Hippotherapy

Hippotherapy uses the “unconventional” medium of the horse. Xenophon (430-354 BC), who ranks as the founder of hippology or the science of horses, defined hippology as follows: “It includes the human being on the horse, the rider, too – as well as all other factors which develop from this relationship between man and horse.” (Hengst & Reichenbach 1972, Gehrts 2006)

The ancient Greeks knew long ago that horses are ideal for therapeutic purposes. The Greek doctor Hippocrates (430-370 BC), whose name, literally translated, means “horse dominator” wrote in his works about the “sacred rhythm” of riding. (Riesser 1997, Rieger 1981)

Galenos of Pergamon (121 – 180 AD), the Roman Emperor Marcus Aurelius’ personal physician, described riding as a form of gymnastic exercise. In the 16th century Hieronymus Cardanus (1501 – 1576) in his dietary study “De sanitate tuenda” recommended riding as a therapeutic measure. In the same century Mercurialis’ study in 1569 - “De arte gymnastica” - alludes to the health-promoting and preserving value of riding. He also understood the effect riding had on the senses.

In Diderot’s famous “Encyclopaedia” published in 1751 one can read that the unity of rider and horse necessitates continual flexion and relaxation of the muscles, which makes riding a universal exercise for all parts of the body. (Riesser 1997) Therefore, this eminent French philosopher recognized an important principle of modern hippotherapy almost two-and-a-half centuries ago. (Riesser1997)

After the Second World War, soldiers who had lost a leg were set on horseback to retrain their impaired statics. (Mellin 1995)

At the beginning of the 1950’s, particularly in Scandinavian countries, cerebrally motor handicapped children were treated on horses. The phenomenon of “being moved” was rediscovered as a method of physiotherapy. The new factor was the discovery that the horse’s movements were transmitted to the patient’s body. (Rieger 1978)

In Germany, it was probably the Lower Franconian Doctor Max Reichenbach who recognised this fact in 1953 and used it specifically on his patients. He only published his findings, however, in 1965. Four years previously, in 1961, Dr. Eberhard Durschky had issued what is probably the first publication in the German language on “Therapeutic Riding”. After this, one publication has followed the other up to the present day.

The introduction of the term "hippotherapy" in specialised medicinal terminology is attributed to the current Rector of the University of Basle, Prof. Dr.med. H .R. Kaeser. This “terminus technicus” has been adopted in all German-speaking countries and is becoming increasingly popular in the rest of Europe. (Riesser 1997)

The fact that the human body reacts “with every fibre in it” in the truest sense of the word to the movements of the horse leads to the conclusion that man and horse share something in common, namely, the urge to move forwards. (Ölsböck 1997)

2.6 Comparisons/Similarities between Osteopathy and Hippotherapy

Right at the very start of my osteopathic training, I discovered again and again parallel factors which reminded me of hippotherapy.
Above all, general osteopathic treatment (GOT) called AOB in German and TGO in France, in which the joints of the entire body are moved systematically and rhythmically, can be compared to movements on the horse. (Chapter 2.3)
In GOT the entire body is systematically moved and its functions tested. The individual joints are worked over with slow, rhythmic movements. The osteopath harmonizes with the movements in the same rhythmic way. The patient can be treated sitting, lying on his/her back, on his/her side or in an abdominal position. The diagnosis flows smoothly into the therapy. Palpation plays a significant role in this. Working with his /her hands, the therapist performs palpatory anatomy - manual examination of the anatomical structures of the body. (Ligner 2001)
The osteopath must find the correct rhythm to enable the patient to relax. In this, the therapist is actually working on a neurological and psychological level.
In this way the entire body is scanned, even if the patient has only indicated a problem in one part of the body. (Ligner 2001)
The therapist begins treatment at the pelvis, the central point in the middle of the body, which is vital for stability as well as mobility.
In GOT the pelvis is also a region of exceptional significance. It is from this region that the horse's movements are conveyed to the upper and lower extremities. (See Chapter 2.3, Effective Factors in Hippotherapy) One of Still's four basic Rules of Osteopathy (see Chapter 6, The Development of Osteopathy) is **"Motion is life"** or **"Life is motion"**. In osteopathy, mobility is assessed. "We know life only by the motions of material bodies." (Still 1902 p.255) - "Motion is the first evidence of life." (Still 1899 p.196) - "If it moves, it can take care of itself." (Schiowitz 2005)
Sutherland, too, recognised that the vital motions were represented by a rhythmic force he generically called Tides. He likened the processes of life to the periodic motions of the ocean. (Paulus 2001)

These four statements by different osteopaths show the importance of motion in the human body. Here motion plays a key role. Accordingly, the motion of each individual cell is vital for the optimal functioning of the common whole (the body). Motion is just as important in the structural system as in the cranial system and body fluids. Motion is the first sign of life and the last. (Ligner 2001)

I find the previous statements exceedingly meaningful in connection with hippotherapy, since here too, motion has been ordained a key role.

In hippotherapy we find the following similar views: “Life is motion, and the reverse is also true: motion is life.” (Strauss 2008 p19)

Strauss also claims: “If appropriate forms of movement generate development, harmony and preservation of health in healthy persons - how much more will a sick person, especially one who has problems with movement, be in need of help and healing through appropriate methods of being moved.”

(Strauss 2008 p19)

Every individual working with movement is actually working with the “essence of life” and every person who encourages and teaches movement, also encourages and teaches how to live! (Dietze 2003)

These claims about hippotherapy make it clear how important movement is for the human body. In this form of therapy, movement is transmitted via the horse to the patient. The concept of helping or encouraging the patient is present as it is in osteopathy.

Handicapped persons whose freedom of movement is often greatly restricted are systematically and thoroughly moved on the horse and – an important fact - moved forwards, since the patients are often incapable of doing so by themselves. In a manner similar to the GOT osteopathic method of rhythmic joint manipulation, the patient is systematically and rhythmically moved by means of the swinging movements of the horse. The individual joints of the body are thereby mobilised. (See Chapter 2.3)

A further point in common is the idea of the **unity of the body.**

In both forms of therapy the human being is regarded as a whole. One definition of osteopathy is: “a comprehensive diagnostic and therapeutic system based on the inter-relationship between anatomy and physiology for the examination, prevention and treatment of disorders. “ **It regards the human organism as a whole, as a mechanical unit,** which is closely connected to its inner fluid milieu as well to its outer surroundings.” (Magoun 1976 p19)

The body is a unit, and wants to be treated as such. (Ligner 1995)

I would like to quote Still on this point, too: “We look at the body in health as meaning perfection and harmony, not in one part, but as the whole”. (Still 1986 p44)
Still contended further that if the body structures are ordered correctly and function normally, and if at the same time neither the blood flow nor the conduction of nerve impulses is impaired, the wellbeing of the body is then assured. Here again, we see in the foreground the interplay of elements in the human body and the importance of its functioning as a unit. The objective of osteopathy is to treat the body as a whole and stimulate its self-healing powers.
In hippotherapy, too, the human being is treated holistically, i.e., emotionally, mentally and socially. (Molnar-Mignon 1997)
Thus hippotherapy, too, is regarded as a holistic therapy, in which the patient is treated in his/her living environment. He is perceived as a bio-psycho-social entity. (Strauss 2008)
The patient is motivated and inspired with the aid of the horse. (Lisinski, Stryla 2001, Tauffkirchen 1978))
A very recent study investigates the horse as a social factor. The conclusion is that a wide range of positive effects not yet fully appreciated by the public results from working with horses. I would merely like to mention here the development of personality and self-confidence. These social components definitely upgrade this form of therapy. (Preiskammer, Josef 2008)

Hippotherapy is an accepted form of therapy for children with cerebral paresis. (See Chapter 3.1)
It is a holistic (multifactorial) form of therapy, hence successful with children and adolescents with infantile cerebral paresis, who have multifactorial problems. (Ölsböck 1997)
The explanation for this can be seen in the positive influence of the therapy on the control processes of the central nervous system, resulting in improvement of functions, performance and wellbeing. (Rieger 1978)
In hippotherapy the patient is, then, treated physically, emotionally, mentally and socially through holistic stimulation. (Molnar-Mignon 1997)

These claims about osteopathy and hippotherapy clearly show that the patient is regarded and treated as a whole in both forms of therapy. Not only his/her symptoms, but the individual as a psycho-physical entity are of intrinsic importance to the therapist. By means of external mobilisation the therapist attempts to normalise muscle tone and facilitate physiological movements.

Basic muscle tone is the main problem for children with cerebral palsy, as in the case of Matthias, my test person, and this can be successfully reduced by hippotherapy. (Sterba et al. 2002) After hippotherapy, the osteopath can work in a gentler and more efficient way, using the wide range of osteopathic treatments on several levels (structural, visceral, cranial and fascial). Knox 2005)

Subsequent osteopathic treatment can also treat the joints directly, since the musculature is then more relaxed and the treatment is accordingly less painful or unpleasant and is better tolerated by children.

I would like here to quote some statements by well known osteopaths who have worked with children with cerebral palsy.

"Growing brains have enormous capacity to compensate. Osteopathic treatment can help to remove obstacles in the way of optimum compensation." (Carreiro 2003, Hayden 2000, Hearsey 1994)

"Cranial articular lesions and resulting membranous imbalances can be reduced successfully with osteopathic treatment." (Frymann 1993)

The authors believe that children can be helped by osteopathy.

To date, osteopathy is not classified as a standard method of treatment for children with cerebral palsy. Physiotherapeutic methods such as Bobath, Vojta und hippotherapy, as well as therapeutic concepts such as Castillo-Morales, sensory integration, Petö, Affolter and Frostig are seen as accepted methods. (Maurer 2002) These forms of therapy are described in Chapter 2.6. Some studies, however, have examined and confirmed the positive effects of osteopathy on these children's motor skills and their general state of health. (See Chapter 3.1)

2.7 Motor Development

In this chapter the normal development of the central nervous system is first described, for a clearer understanding of how this differs in a child with cerebral palsy, which will be described later.

Static-motor development from the neonate to the adult is dependent on the maturation of the central nervous system. The course of this development is determined by genetically set patterns of development and by environmental stimulation. The brain, in its capacity as an integrative and coordinatory organ, responds to these stimuli with automatic, complex reactions. (Flehming 1987)

Each sequence of movement is always carried out by optimal adaptation to outer stimuli. The patient's organism and environment function inter-dependently in this respect. According to Schilling (1979) motor development or the stage of motor development is always dependent on the environment and, as a result, dependent on the situation. (Flehming, 1987)

For Christian, (1952) motion is not the result of organs which have become functionally reliable, but rather, the utilisation of functioning organs.

Tonus regulation is one of the prime factors in motor skills. Suppression of deep-seated central regions of the brain and stimulation of more highly integrated central brain regions by means of finely-graded regulation and counter-regulation, are always mutually influential processes. Here, one step must necessarily follow the other. In this way, a genetically imprinted pattern can attain full development through interreaction with environmental stimuli, which partly inhibit and partly support this process. (Flehming 1987)

The fact that this complicated system can fail due to a great number of disturbances lies in the magnitude of the regulatory systems. Their coordinated interplay in the microcosm of a developing child with its psyche, its ability to react to environmental stimuli, with all the potential of its sensory and sensitive systems and its intellect to react to such stimuli is what we call development. (Flehming 1987)

If the defect occurs in a system which is not yet fully functional, the chances of adjustment are higher than in a fully mature system in which the channels are already set. This is called the plasticity or the dynamics of the brain. The more developed an organism is, the more complex are its reactions and the more susceptible the entire system is to faults.

Therapy should therefore begin as soon as possible. (Arbuckle 1955)

Established classic forms of therapy include neurophysiological and physical therapies such as hippotherapy and the therapeutical concepts described below. (Maurer 2002)

The Bobath Technique:

This method was the first consistent concept created after the Second World War for children with infantile cerebral paresis. Bobath places special emphasis on restriction of pathological movement and promotion of physiological movement in everyday situations. Compliance on the part of the child is necessary. Handling practices play an important part – the correct manipulation of the child (neonate) in everyday situations. (Maurer 2002)

The Voita Technique:

Newborn children have reflexes for crawling and turning over, two inborn coordinating mechanisms which can be activated by applying stimuli to specific regions. In therapy, pressure is applied to points in precisely predefined areas, with a view to activating normal spontaneous motor skills in the patient. (Maurer 2002)

Castillo-Morales Orofacial Therapy:

In this country we are especially familiar with orofacial regulatory therapy. While the motor nerves are at rest, the therapist works on the regulation of the tonus in the mouth region. Mandibular control, activation of the muscles of facial expression, of the lips, cheeks and tongue as well as sensitisation or desensitisation of the mouth area (heightened retching reflex) are central tasks. (Maurer 2002)

The Petö Technique:

This is a holistic form of therapy which is not classed as part of the recognized sphere of physiotherapy, but is, rather, a conductive multidisciplinary therapy which is actually an educational method. This therapy includes a lot of group work to train conditioning. (Maurer 2002)

The Ayres Method of Sensory Integration:

Stimulation of different sensory impressions is thought to result in improved integration and processing of perception. In particular, visual spatial perception is considered to be better

structured as a result and hence, more accurate. This form of therapy is mostly used by occupational therapists. (Maurer 2002)

The Affolter and Frostig Therapy:

This takes the form of perception training therapies for older children, in which the focal point is tactile- kinaesthetic perception. Many learning programmes are based on Affolter's work. Frostig concentrates on the visual motor senses and everyday visual situations with special emphasis on handling. This entails the correct manipulation of the child (infant) in everyday situations. Very many learning and coaching programmes to some extent in everyday use in schools originate from Marianne Frostig's work.

All these forms of therapy can and should help to provide cerebral palsied children for whom no curative therapy is feasible with good means of achieving the physical goals possible for them in their daily life. (Maurer 2002)

Osteopathy is not yet regarded as one of the classic forms of therapy open to children with cerebral palsy. Many parents are not aware of this option. This should change. Osteopathy, too, should be made available to these children, as well as the above-mentioned forms of therapy.

2.8 Infantile Cerebral Paresis –Historical Progression

Although long known as having a distinct clinical picture, it was not until the middle of the 19th century that infantile cerebral paresis distanced itself in particular from the then prevalent and widespread poliomyelitis. The English orthopaedist Little was the first doctor to research the nature and treatment of this disorder. The first studies on the treatment of foot misalignment following asphyxia* and premature birth are by him. (1861) The disorder was primarily named after him. After the Second World War, improvements in obstetrics and the option of intensive medical care for newborn babies made it necessary to re-define the clinical picture. The AACP (American Academy for Cerebral Palsy) played the leading role in this, publishing a first definition in 1957. This was redefined to some extent by the Spastic Society in 1966 and has remained valid since that time. The definition is as follows:

"Infantile cerebral paresis is a permanent, but not unalterable disorder of posture and movement as a result of a pre-, peri- or postnatal cerebral functional disorder occurring before the brain has completed its maturation and development." (Maurer 2002)

The new version of the definition created in 2000 by the international network "Surveillance of Cerebral Palsy in Europe" (SCPE) consists of four points. Infantile cerebral paresis is:

- a manifestation of different disorders
- it is permanent, but not unchangeable
- it is a disorder of posture and/or movement and motor skills
- it is caused by a non-progressive disorder/lesion/abnormity originating in the immature brain

This fairly similar definition gives a brief and distinct review of the individual points. (Maurer 2002)

Similar statements about cerebral paresis follow.

"Cerebral paresis," or "CP" for short, is a concept and not a disease. (J. Carreiro 2004)

The concept is used to describe a range of non- progressive, but changing motor disorders resulting from brain damage during development. (Menkes u. Sarant 2000)

The most common causes are perinatal asphyxia* and complications in connection with premature birth. Perinatal asphyxia is a condition, in which the brain is exposed to hypoxia,*

*See Chapter 8.2

ischaemia* and hypercapnia*, leading to cerebral oedema and metabolic changes. (Volpe 1995) Investigations suggest that the insult is intra-uterine in 40-50% of cases, in 30-40% peripartal and in approximately 10% postpartal. (Brown et al. 1974, Low et al. 1989, Volpe 1995)

Prenatal brain insults are the predominate factors associated with cerebral palsy. (Ceney, Palmer 1997)

The prevalence of cerebral palsy in Europe is 2 - 3 out of 1000 live births with wide variations in different weight classes. The statistics in a study made in Graz in 2002 which deals exclusively with premature births in intensive care which have a birth weight lower than 1500g, range between 10%-20%. (Maurer 2002)

The morphological cause of infantile cerebral palsy is always damage to the first motor neuron. Primarily, and in most cases (85%) the pyramidal tract (tractus corticospinales) is affected, but extrapyramidal tracts (injury of the basal ganglia) or the cerebellum (injury to the tractus cortocopontocerebellaris) can also be affected. Primary or secondary injury to cortical regions can also occur. Magnetic resonance imaging has shown that in affected children not only the brain tracts (white matter) but also the cerebral cortex (grey matter) is thinner than in healthy children. (Maurer 2002)

Which area of the brain is affected depends on the duration of the insult and the gestational age of the foetus, since the sensitivity of particular areas changes during pregnancy according to the development of the pattern of blood vessels in the brain.

Since brain damage mostly occurs during pregnancy or at birth, the child is quite capable of learning motor skills, which however, because of cortical damage, vary from the norm. (Maurer 2002)

In the course of their development children must learn new strategies, refining and integrating them in order to interact with their environment.

It is therefore a sensomotor disorder of posture and movement, caused by permanent but non-progressive damage to the developing brain. (Carreiro 2004)

*See Chapter 8.2

2.8.1 The Clinical Picture of Cerebral Paresis

The clinical picture of cerebral paresis always reveals a disorder of posture and motion. (Cheney, Palmer 1997, Arbuckle 1955, Maurer 1997)

MOTOR SKILLS in the cerebrally palsied child:

Primarily, the central control structure of the locomotor system is disturbed. The disorder in reciprocal innervation leads to faulty coordination between agonistic and antagonistic groups of muscles. In some the tonus is too low, in others too high. The accompanying symptom of strabismus in children with cerebral palsy is caused by a lack of coordination between the extraocular muscles. Furthermore, coordination of the skeletal musculature is affected. Voluntary movements are inhibited, pathological patterns of movement can occur and the joints are held in extreme positions. (Diel 1999)

The extent of spasticity varies from child to child. In extreme spacitic, the child can develop contractures, mostly affecting ankles and feet, but which can spread to other joints. 27% of children with spastic diplegia suffer from cerebral seizures, often in the form of grand-mal* seizures (tonic-clonic). (Menkes, Sarant 2000) If untreated, they can also contribute to raised tonus. (Carreiro 2004)

Impaired balance of physical strength and muscular imbalance are responsible for bio-mechanically triggered secondary changes such as functional shortening of the extremities, pes valgus, flat feet (fallen arches), talipes equines, as well as deformities of the thorax, kyphosis, and scoliosis, asymmetry of neck and rump, and contractures which develop over time. Over the years asymmetric muscular traction leads to hip joint subluxations.

If these symptoms persist for a number of years, they result in tertiary mutations such as mutation of the bones, an irreparable final condition found in children with severe forms of cerebral palsy, caused by the long-term effects of defective biomechanics and accompanying pain. (Diel 1999)

The SENSORIUM of the cerebrally palsied child:

Children with cerebral palsy have enormous deficits in perceived and integrated sensory stimulation compared with children who have good motor skills. This hinders the optimal development of the child's individual senses, of higher levels of coordination and a physical integrity identity system, as well as the ability to "grasp" (understand) the environment,

*See Chapter 8.2

the development of abstract imagination, communicative facilities in line with the child's age and a "healthy self confidence." Therapy can only be successful if all the sensory deficits of the child are taken into account. (Diel 1999)
The connection between spasticity and cognitive abilities is not yet clear. It is often wrongly assumed that a child with infantile cerebral palsy suffers from severe cognitive disorders. Studies dating from the 60's show a strong correlation between low IQ levels and spasticity of the upper extremities. (Ingram 1964) Some more recent studies present contradictory information. (Krägeloh-Mann 1995, Olsen et al. 1998)
However, when all is said or done, cognitive abilities can not even be accurately predicted by magnetic resonance screening.
(Carreiro 1999)

Further accompanying disorders may be DEFECTIVE VISION. This can take the form of strabismus convergens* (which affects spatial vision, perception of heights, slants, distances, and which leads, if untreated, to weak-sightedness in the misaligned eye), paralysis of the eye (e.g. abducens paralysis), reduction of acuity of vision caused by premature birth retinopathy,* homonymous hemianopsia, (e.g. complete loss of the visual field through damage to the right tractus opticus) or optical agnosia (in which what is seen cannot be recognized, interpreted or processed). (Mumenthaler 1986, Flehming 1987, Rossi 1989)

Another accompanying disorder can be DEFECTIVE HEARING. One must differentiate here between peripheral impaired hearing (disturbances in ventilation of the tubes in orofacial dysregulation often result in effusion and inflammation of the middle ear) and centrally impaired hearing (caused by damage to the auditory pathway or acoustic agnosia*) (Mumenthaler 1986, Flehming 1987, Rossi 1989)

Yet another disorder which can accompany cerebral palsy in children is EPILEPSY. Epilepsy can occur particularly in spastic tetraparesis, but less often in spastic hemiparesis. Furthermore, the lack of active movement, lack of verticalisation on the intestinal mass and a frequently faulty diet can result in OBSTIPATION. For the same reasons PERSISTENT INFECTIONS OF THE URINARY TRACT can also arise.

*See Chapter 8.2

Inconsequent parental training, an exceptionally close mother-child relationship, increased dependency etc. also lead to BEHAVIOURAL and EMOTIONAL PROBLEMS. (Diel 1999). PSYCHOLOGICAL PROBLEMS, the strongest form of which can be termed intentional paresis should also be included here. This implies a very early loss of pleasure in movement and the loss of trust in one's own capabilities and abilities. This can go as far as a symbiotic fusion with the mother, since the child performs a great many voluntary activities and actions through the mother, living a part of his/her life, as it were, through the mother. This brings about an even closer mother-child relationship, as described above, which for a long time does not allow separation, and thereby hinders social integration. The end result is that the child is even less willing to attempt to exercise his/her motor muscles than he/she would normally be on account of his/her handicap. These children are often extremely difficult to motivate in therapy. (Maurer 2002, Marcovich 2003, Savage 2008)

2.8.2 Outward Forms of Infantile Cerebral Paresis

Classic cerebral palsy takes the form of spasticity in 70-80% of all cases. (Dormans u. Palligrino 1998)

Classification of cerebral palsy (according to Freud's phenomenology, modified by Ingram-Hagberg-Michaelis et al. /Tübingen classification)

The **1st** form**, spastic cerebral paresis** is manifested in spastic hemiparesis, which can affect arms, legs, or arms and legs to an equal degree.

Spastic tetraparesis can affect the legs alone, or three extremities can be affected (one arm being less affected); it may be one-sided, crossed, (affecting the upper extremity on one side and the lower extremity on the other side) or complete.

In addition, there is the **2nd** form, **dyskinetic* cerebral paresis**. The most common form of this is athetosis.

In the **3rd** form, **ataxic* cerebral paresis**, the degree of spasticity can be more or less pronounced.

The **4th** form**, cerebellar paresis** is found less frequently than the other forms. It results from damage to the cerebellum. It is often only first noticed when the child begins to progress to upright posture (sitting, standing, walking). (Adams 1993) At the end of pregnancy, the cerebellum is at its most sensitive to injury. (Rossi 1989, Diel 1999)

"Pure forms" of cerebral palsy are seldom found, mixed forms being more common. In evaluation the components involved should be listed - firstly, the main symptoms, followed, if possible, by the pattern of distribution. One diagnosis might be: "spastic tetraparesis with athetoxic components in the upper extremities". (Rossi 1989, Diel 1999)

Ad 1: In spastic cerebral palsy the brain lesion is always central. (gyrus praecentralis* in the cortex cerebri- tractus corticospinalis)

The spastic muscular system shows flexible resistance to passive stretching. Dysfunctional co-contractions of the agonist and antagonist are produced, leading to restricted movement. The innate muscular reflexes are heightened and pyramidal tract signs are present. (Babinski, Gordon, Oppenheim) Tonic reflex patterns are present (asymmetrical tonic neck reflex (ATNR), tonic labyrinth reflex (TLR), symmetrical neck reflex (SNR) as well as oral

* See chapter 8.2

extension reaction). This results in pain and restriction of movement, or stiffness.
Spasticity is present mainly in the tonic muscles which are responsible for anti-gravitational postural function in the locomotor system. Every active movement of a physical region or psychological agitation leads to raised tonus in the pathological postural pattern, resulting in associated reactions*. (Maurer 2002)

Ad 2: In dyskinetic cerebral palsy, the point of lesion is in the basal ganglia, in particular in the striatum (nucleus caudatus + putamen) and in the pallidum. Characteristic for athetosis are abnormal, worm-like twisting movements carried out at a slow pace. These movements affect the distal segments of the extremities and are interrupted only by sleep.
The major problem lies in the erratic muscle tonus, which at rest is reduced and in activity is too high. The result is impaired and greatly reduced physical coordination. Maintenance of posture is very badly affected. Voluntary movements can not be gauged and balance reactions are poor. Pathological tonic reactions are seen (ATNR, SNR, oral extension reactions, eye movements affected by the tonic pattern). The patients experience fear and lack of confidence because of their poor levels of motor stability and self control.
Typical symptoms often found in athetosis are uncoordinated facial movements such as discoordination of the motor functions of the eye, mouth and tongue, as well as speech disorders and flat, uncontrolled breathing. The functions of the upper extremities are usually more affected, while the intelligence is mostly normal. (Rossi 1989, Diel 1999, Maurer 2002)

Ad 3: Ataxia in ICP (infantile cerebral paresis) is rare. The lesion is situated in the cerebellum. Ataxia means “without order”. The symptoms are hypotension* and poor coordination of all movements. This leads to asynergy (lack of exactness in the fine play of muscles) and the rebound phenomenon (an extremity pressed against resistance displays a rebound motion after the resistance is removed). The patient shows dysmetria* and hypermetria*, i.e. the restrictive function of the antagonist sets in too late. Movements are excessive. Further, we find intention tremor* and dys- or adiadochikinesis (lack of rhythmic, alternating interplay of the antagonistic muscles).

* See chapter 8.2

Speech is slow and halting, formation of syllables uneven and articulation poor. A further symptom is nystagmus*.

Balance reactions are poor, because the torso is unstable. Patients have a wide gait.
Newborn children are floppy infants*. Their progression to an upright posture against gravity only develops slowly. Balance reactions are extremely disturbed. The children start to walk very late and have an insecure (atactic) gait. Intelligence is often noticeably reduced. In rare cases forms of epilepsy are found. (Rossi 1989, Diel 1999, Maurer 2002)

*See 8.2

2.9 Botulinum Toxin Type A in Childhood Spasticity

Since Matthias, my test child, was also treated with botulinum toxin A, I would like to examine briefly this type of therapy which is relatively new (in Austria).
Botulinum toxin A is a relatively new, non-invasive treatment option for children with cerebral palsy, providing an effective short-term intervention to reduce spasticity. Mulligan, Wilmshurst 2006)
Over the past years several studies, including placebo-controlled studies, (see below) have shown that spasticity can be reduced by administering intramuscular injections of botulinum A (BTX A). Cerebral palsy with spasticity is one of the most common disorders of the developing locomotion system in children. (Strobl 2000)
Muscular imbalance produces compensatory mechanisms, retardation of sensomotor development and secondary deformities of the locomotor system (luxation, arthrosis…). Prevention of these consequences is the main aim in treating spastic children. (Strobl 2000)
BTX A is a valuable complement to other treatments such as therapy, orthotic devices, casts, etc., for reducing spasticity in particular muscle groups. BTX intensifies the tonus-reducing effect of other therapeutic methods often applied. In many cases it is only these injections which make it possible for the patient to wear therapeutic casts or functional orthotic devices. (Strobl 2000)

In Austria, a prerequisite for successful treatment is the exact selection of patients, based on statomotor and dynamic examination, as well as 3-D gait analysis and dynamic electromyography.
No complications were observed. Systemic side effects occurred in a few cases in the form of temporary muscular weakness. (Strobl 2000)
Treatment of cerebral palsied children with botox is still relatively new, which is obvious in the studies. All the studies deal with the same theme, namely, how spasticity can be reduced in the affected muscle groups. Their conclusions are also very similar, namely, that the treatment does actually reduce spasticity in the affected muscle. However, the long-term effects are not yet known. How does a muscle react when treated with botox over a period of years? This leads to modified statics, because the tonus has been altered. In Matthias' case, it has led to a deterioration in his hollow back and to flat feet.

The following studies on BTX A, Booth et al. 2003; Chambers 2002; Detrembleur et al. 2002; Flett et al. 2001; Galle et al. 2001; Graham et al. 2000; Mall et al. 2001; Ong, Chong, Yap 2001; Reddihough et al. 2002; Wong 2003, found a reduction in spasticity.

2.7 State of Research/Scientific Studies

In my research for this study I discovered a great deal of information on studies dealing with hippotherapy and cerebral palsy. However, relatively few studies exist on osteopathy and cerebral palsy. My information is taken from various sources, including MEDLINE, EMBASE, CINAHL, Pascal Biomed, PsycINFO, CDSR, ACP Journal Club, DARE, CCTR, CLCMR, CLHTA, CLEED, BIOSIS Previews, PSYNDEXplus Literature and Audiovisual Media, PSYNDEXplus Tests, CanChild Centre for Childhood Disabilities, Medical University Library of Vienna and the Osteopathic Library of Midstone.
I shall begin with the existing studies on osteopathy.
One of the most recent studies carried out by Davis et al. (2007) in Arizona (USA) deals with cerebral palsied children and osteopathy. The method of this study is a structured objective form designed to assist osteopathic physicians in the evaluation of fascial and spinal motion restriction. A system of muscle spasticity rating was developed for use during osteopathic musculoskeletal structural examinations. In this study, confirmatory factor analysis was used to examine the relationships between fascial and spinal motion restrictions in addition to spasticity. In fifty- seven children with spastic cerebral palsy, latent factors for fascial restrictions and spinal motion restriction fit the data well and both factors were correlated with a visual analogue scale rating of the child's muscle spasticity. These findings provide preliminary evidence for the factoral and concurrent validity of fascial and spinal motion restrictions, demonstrating the benefits of an instrument for assessing the results of osteopathic musculoskeletal structural examinations.
In this study, the connection between muscular and fascial structures is clear. The relatively high number of test persons and the statistical significance of the results validate the study.

Another study dealing with cerebral palsied children was written by Duncan et al. (2004). In this study fifty children were involved in a randomized, controlled trial to evaluate the effectiveness of either osteopathic manipulation or acupuncture as a 6-month therapeutic therapeutic adjunct for children with cerebral palsy. The osteopathic physician concentrated on craniosacral and myofascial release techniques. Ninety-six percent of parents reported some improvement while their child was receiving treatments but the gains varied from child to child.

The most frequent gains were seen in improvement in the use of the arms or legs, and more restful sleep in the osteopathic and acupuncture groups, respectively. Improvement in mood and improved bowel function were also very common benefits noted by the parents in both groups. The impact of the changes for these children and their families was significant.
This study has a high validity rating on the grounds of its length and the number of test persons involved. The improved sleeping rhythm reported by the parents can be interpreted as an improvement in the child's overall general condition and increased sense of wellbeing. This results in a better quality of life for both children and parents. This factor is extremely difficult to test, since children with cerebral palsy find it extremely difficult, if at all, to gauge their own general sense of wellbeing. In this study only the parents were asked to complete a questionnaire on their children's condition. The test persons themselves were not in a position to supply information on their sense of wellbeing, or on any improvements. This was either because of the severity of their handicap, or because they were too young. Their ages varied from eleven months to twelve years, whereby the best results were achieved with the younger children. (See also the study by Arbuckle.)
A critical factor in this study is that the children's condition was only assessed by means of a questionnaire and therefore the results are not objective. External assessment would be more desirable, or else measurements of movements or functions. Observation over a longer time period would also be very interesting.

In my study I attempted to record my test person Matthias' sense of wellbeing on a Smiley scale. However, as he always said he felt really fine, apart from one day after falling down some stairs, I could not use his statements in my study. This shows how difficult it is to measure these children's sense of wellbeing. Most data comes from parental observation of children's behaviour, as in the above-mentioned study.

Another study by Steingrube-Bradtke (2003) covers a period of two years with 50 children aged 0-16 years. The intention is to clarify whether the multimodal (manual therapeutic/osteopathic) therapy concept has an influence on the well-being of children with cerebral palsy. The Wilcoxon test and descriptive statistics show significantly improved values after single treatment, as well as over a longer time span.
Since this study was carried out over a relatively long period of time, its validity and the medical significance of its findings are of considerable consequence.

An American study by Arbuckle (1955) includes a description of the importance of osteopathy in cases of cerebral paresis. In cranial treatment, rhythmic, deeper breathing is one of the first reactions to be noticed. The authors also treat patients with “Chapman reflexes”. They regard the patient as an entity, an individual, and his/her difficulty or handicap is superimposed upon this unit. So the patient must be treated as an individual of a certain age and not merely as an object to be forced into a pattern of structural and temperamental uniformity. In the patient’s best interests, treatment should be initiated before undesirable motor patterns have become fixed. Prevention of such patterns is far easier and less time-consuming. When the infant is in the nascent stage, osteopathic manipulative therapy constitutes the entire program over and above that of the usual care given all newborn children. Older children require more treatment. Occupational therapy must be added. Occupational therapy consists of any mental or physical activity which is professionally prescribed and adapted to help a patient recover from his/her disability. Osteopathic manipulative treatment helps to overcome the cause of the symptoms and difficulties of cerebral palsy, whereas occupational therapy aids the patient’s rehabilitation.
The authors also write that fun must be substituted for fear and anxiety, because it is exactly these latter emotions which are factors that hinder progress.
To sum up, it can be said that this study describes methods of therapeutic treatment for children with cerebral palsy, as well as the importance of osteopathy in cerebral palsy, in particular, the osteopathic treatment of newborn children. An equally important factor is individual therapy for every child with cerebral palsy, because one universal “prescription” for treatment does not exist.

In addition, previous studies by Frymann and colleagues and Romansky record improvements in children with cerebral palsy, arising from the use of osteopathic manipulation treatment. Measurements of improvement have included speech, balance, improved concentration and attention span, decreased spasticity, and improved mobilization of respiratory secretions, pulmonary function and fluid balance in general. Remarkable improvements have been seen in the patient’s quality of life as reflected in the above parameters.

Also the single-case study by Knox (2005) shows that the osteopathic treatment given to the proband, James, an eight-year old boy, was successful. His breathing and eczema improved and the improvement in diaphragmatic excursion had a positive effect on his overall body functions.

However, since this is a single-case study, the results can not apply to all other children with cerebral palsy. However, it does provide yet another indication that osteopathy has a positive influence on the general condition of a child with cerebral palsy, thus improving his/her quality of life.

The Centre for Childhood Disability Research at McMaster University created the Gross Motor Function Classification System (GMFCS). This is a tool that can be used to classify children with cerebral palsy into five levels based on their functional abilities and their need for assistive technology and wheeled mobility. The focus is on the child's self-initiated movement functions in sitting and walking. The Ontario Motor Growth (OMG) Study (Avery et al 2002) has given more information to answer parents' questions. They have developed a system which defines more accurately the range of gross motor functions in children with cerebral palsy. The GMFCS level, therefore, tells us a lot (but not everything) about how much or how well a child's motor development may progress. Factors such as a child's interests, motivation, health and other aspects of development also make a difference to a child's motor development. The GMF Measure assessment, however, tells us nothing about the quality of motor control (smoothness, efficiency, ease of movement) used by children to do things.
In my opinion, the "Gross Motor Function Measure" is an excellent method of assessing the extent of impairment of motor functions in children with cerebral palsy and of giving parents a prognosis. In Austria, this form of testing is, however, not commonly used and is not found in medical reports on children with cerebral palsy. For this reason I have not included it in my study.

In short, it can be said that osteopathy has a positive effect on children with cerebral palsy. The best results have been attained on younger children. (Duncan et al. 2004, Arbuckle 1955)

Over twenty studies investigate the effects of botulinum toxin A on children with cerebral palsy. (See Chapter 3.2)

There are a great many scientific studies on children with cerebral palsy and the effects of hippotherapy. All reach a similar conclusion, namely, that hippotherapy has a positive effect on the locomotor system of children with cerebral palsy. I shall present various studies on hippotherapy in order to give a wider overall picture.

The effects of hippotherapy on muscular tonus, joint flexibility, breathing, gland secretion, heart and circulation, and, not least, on the psyche, are empirically certifiable and partly measurable, too. (Hemmelmayr 1997)

As early as 1987, electromyographic examinations were carried out to test the patellar tendon, which established that the patellar tendon reflex was significantly reduced following hippotherapy, which can be seen as an indication of the reduction in muscular tonus. (Bausenwein 1987)

Furthermore, coordination of the muscles of the torso while the patient was walking was examined before and after hippotherapy, to judge their influence on torso control. Highly significant improvements in the children tested were observed. (Ölsböck 1997)

A more recent electromyographic examination was made by Benda, McGibbon and Grant (2003). The study is entitled: "Improvements in muscle symmetry in children with cerebral palsy after equine-assisted therapy (hippotherapy)."

Fifteen children between four and eight years of age were examined. With the aid of electromyography, muscular activity was measured on the torso and lower extremities. The children's musculature was measured on the horse and on a barrel. The result was that eight minutes of hippotherapy, but not stationary sitting astride a barrel improved symmetry in muscle activity in children with spastic cerebral palsy. These findings suggest that it is the movement of the horse rather than passive stretching that accounts for the improvements measured.

A prospective, randomised therapeutic study by Ziegler (1997) investigates the effect of hippotherapy on post-operative spinal disc patients. The result was positive in 87% of the cases. 12% of the patients experienced no effects from hippotherapy and 0% of the patients experienced pain during or after treatment.

In the study, 16 patients were examined during stationary therapeutic treatment following nucleotomy of the lumbar column and compared with a controlled group. Accordingly, orthopaedic hippotherapy can be seriously considered a valid therapeutic concept for post-operative treatment of lumbar disc patients, which in combination with therapeutic programmes presently used leads to improved rehabilitation results. This study is interesting although it was not carried out on children with cerebral palsy, because the adult patients were able to give direct feedback on hippotherapy.

Another study on the Effectiveness of Hippotherapy on Patients with Multiple Sclerosis (Künzle 1993) was carried out over a period of six years in Switzerland. It is a neurological

disorder. The therapeutic results were evaluated from three sides.
From the patient's point of view: The positive assessments by the patients surprised even the most optimistic therapists.
From the point of view of the doctors who prescribed the therapy. In general, they were particularly critical of new methods. The high degree of positive opinions expressed from this quarter was, therefore, all the more remarkable.
From the point of view of the therapists involved: All the physiotherapists involved were completely convinced about hippotherapy.
The assessment of hippotherapy by patients, doctors and therapists resulted in 96.9% positive statements. Only in a few cases were the critics unable to make clear statements and there were even less than 1% negative opinions in the survey. The statements about the effects of hippotherapy were classified into the following groups.

- improvement of motor skills (balance, coordination, posture, gait)
- pain relief
- relaxation, slackening of muscles, reduction of tonus
- improvement of general condition and independence, better quality of life

The fact that hippotherapy gives pleasure upvalues this form of therapy.
I have included this study because it ran over six years and the clientele consisted of neurological patients. They were able to give details of their personal experience during therapy and assess the above-mentioned effects objectively. Another important aspect of this study is the assessment from three different points of view (patient, doctor, and therapist).

I have listed some of the latest studies on children with cerebral palsy.
One of the latest studies from 2007 is by Hamill, Washington und White. The purpose of this single subject study was to examine the effects of a once-weekly, 10-week hippotherapy programme designed for three children with cerebral palsy, aged 27-54 months. Parental perception of the hippotherapy intervention was assessed by questionnaires. None of the children made improvements by any of the standardized results measured. However, parental perception was extremely positive, with reported improvements in the range of motion and head control.
This study was carried out in the USA. In Austria, children of this age would not be given hippotherapy (the earliest age would be four years, depending of course on the size of the child (see Chapter 2.4).

Moreover, I find a 10-week therapy span too short to expect improvements in development in cerebral palsied children of this age. However, the positive findings of the parents lead us to assume that hippotherapy was effective, after all.

The study entitled „Heart rate response to therapeutic riding in children with cerebral palsy: an exploratory study," was written by Dirienzo and Baceski in 2007.
The study was designed to document the heart rate response to therapeutic riding of children with cerebral palsy. Eight young riders were divided into two groups: ambulatory and wheelchair dependent.
The young patients with multiple disabilities who were wheelchair dependent s higher heart rate values during therapeutic riding than their ambulatory counterparts. In view of these higher rate values, further studies are needed to assess the role of monitoring heart rate response during therapeutic riding.
This study is included because it shows the influence on the heart and circulatory system.

Another study dating from 2007 from Canada reads: "Horseback riding as therapy for children with cerebral palsy: is there evidence of its effectiveness?" (Snider et al 2007)
This study is a systematic review of the literature on horseback riding therapy as an intervention for children with cerebral palsy. The conclusions of this study are that hippotherapy is indeed effective for treating muscle symmetry in the trunk and hip and that therapeutic horseback riding is effective in improving gross motor functions when compared with regular therapy or the time the patient might spend on a waiting list.
This study is interesting because no probands were questioned or tested, but the results were extracted from literature. In this way, information from many previous studies was summarised. This shows how many different ways there are of approaching this subject.

Since many studies on hippotherapy and cerebral palsy exist which have come to similar conclusions, namely, that these therapies have a positive influence on the motor senses in childhood, a few are listed here:
Casady, Nichols-Larsen 2004; Cherng et al. 2004; Debuse, Chandler, Gibb 2005; Duncan et al. 2004; Gehrts 2006; Gerster 1976; Hael, Hellwig 2000; Gehrts 2006; Giuliani, Lewis 1999;

Hengst 1976; Horster et al. 1976; Kuczynski, Quint, Toomey 1998; Slonka 1999; Land et al. 2001; Tauffkirchen 1978; Tuttle 1987; Liptak 2005; McGibbon et al. 1998; Meregillano 2004; Maurer 2002; Rieger 1978; Sterba 2007; Sterba et al. 2002

3. Methodology

3.1 The Test Person

3.1.1 Matthias' Case History

Matthias was born in Vienna in April 1997. He has a twin brother who is also affected. The twins were born by Caesarean section in the 27$^{th.}$ week of pregnancy following premature rupture of the membranes. Matthias' birth weight was 1034 g, his head circumference 25.2 cm and the head/toe measurement 36 cm. APGAR was 7/8/9. Because of the posthemorrhagic hydrocephalus external ventricular drainage was performed. He was allowed home on 9.7.1997.

Matthias developed spastic tetraparesis with accentuation on his left side. From birth on he received physiotherapy, early coaching and cranio-sacral therapy. At approximately one year of age he began to make seal-like, floppy movements and later to crawl. The disparity between his left and right sides was then quite obvious. When he began to stand, he was given walkers and specially fitted shoes, as well as a tilt-table. The upper left extremity clearly showed weakness in muscular strength and automatic muscle reflexes. Both lower extremities showed medium to noticeably heightened muscle tonus and increased automatic reflexes as well as easily triggered pathological reflexes such as adductor reflex, heel reflex, Rossolimo reflex and crossed adductor reflexes.

His behaviour at play at the age of 2 was in line with his age, disregarding his motor handicap and his sense of exploration was good. He had a large vocabulary even at this early age. His psychological development was similarly unremarkable. At the age of two and a half Matthias was given night calf splints for overcorrection of both legs. He started to walk at three. From his fourth year on he had blocks of hippotherapy.

In February 2002, when he was 5 years of age, Matthias had his first intramuscular treatment with botulinum toxin A in the ischio-crural musculature on both sides and in the upper left extremity. Following this, he was given lower leg redression walking casts (lower leg/foot walking casts) on both legs. Since the function of the upper left extremity as well as the legs had improved after the botox injection, this therapy was repeated a year later and in 2004. From this time on the groin and popliteal ligament and the upper extremity were treated, and later on, the left thenar. Since then, this therapy has been repeated once yearly.

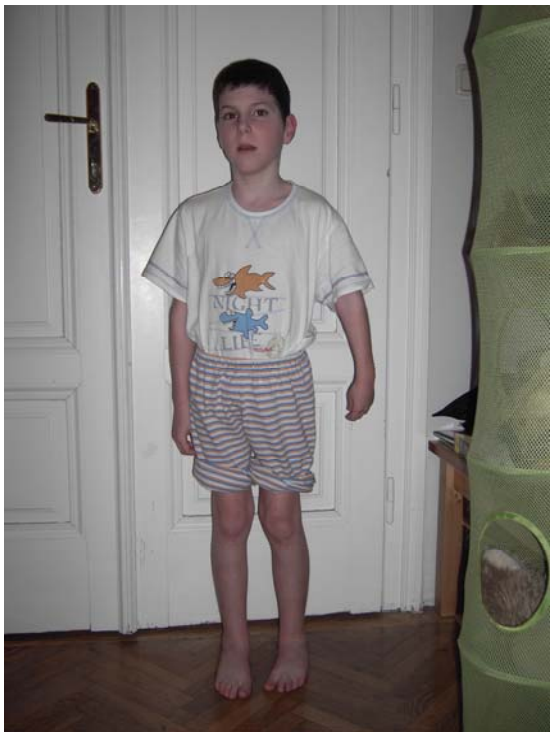


Fig. 3. Stance – ventral view
Schönau 2006

Fig. 4. Stance with abducted arms
Schönau 2006

Matthias' Present Status/ February 2007

Height: 131 cm
Weight: 30 kg
Head circumference: 52 cm

Matthias is a happy, bright 10-year old with a remarkably large vocabulary. He attends the second class of a primary school. He always tries his best and is very much supported by his parents but he still finds it difficult to keep up with healthy children in general, as well as with schoolwork. He is particularly weak at mathematics. However, he has no trouble making friends quickly and makes a very communicative impression.
He can do some things which are often problematic for other children of this age. For example, he could tell the time perfectly at six years of age. He knows every car make and is a great automobile fan. He can play with his toys for hours, an activity which often falls too short in his busy daily plan. He has hippotherapy on a regular basis, alternating with physiotherapy, and is sometimes given osteopathic and ergotherapeutic treatments and all kinds of supplementary coaching to improve his performance at school.
His health is very stable.
Since he was four, Matthias has been able to walk freely and climb steps. Two years ago he learned to swim without a swimming aid and he can dive well. He rides a bike with supports and is already very independent in his daily life.

Latest Medical Report:

Tetraparesis is seen in both legs and in the left arm. The latter is the more badly affected. Matthias hardly ever uses his left arm. Actively, it can only be moved a little. His hand is usually held closed in a fist and can not be opened actively. If asked, Matthias opens the fingers of the left hand with the help of his right hand. He also used to crawl with this wrist flexed to the maximum.

If the left wrist is extended passively, Matthias finds the pull of muscles in the hand extensors uncomfortable. Simultaneous extension of his wrist and finger joints is not possible. The left thumb is held in the fist (atrophy of the m.opponens). The elbow can be extended passively to the maximum, actively a few degrees less. Both arms show strong hair growth on the radial side of the lower arms.

His right arm functions normally.

Fig. 5: Stance with abducted arms/Schönau 2006

Fig. 6 On all fours /Schönau 2006

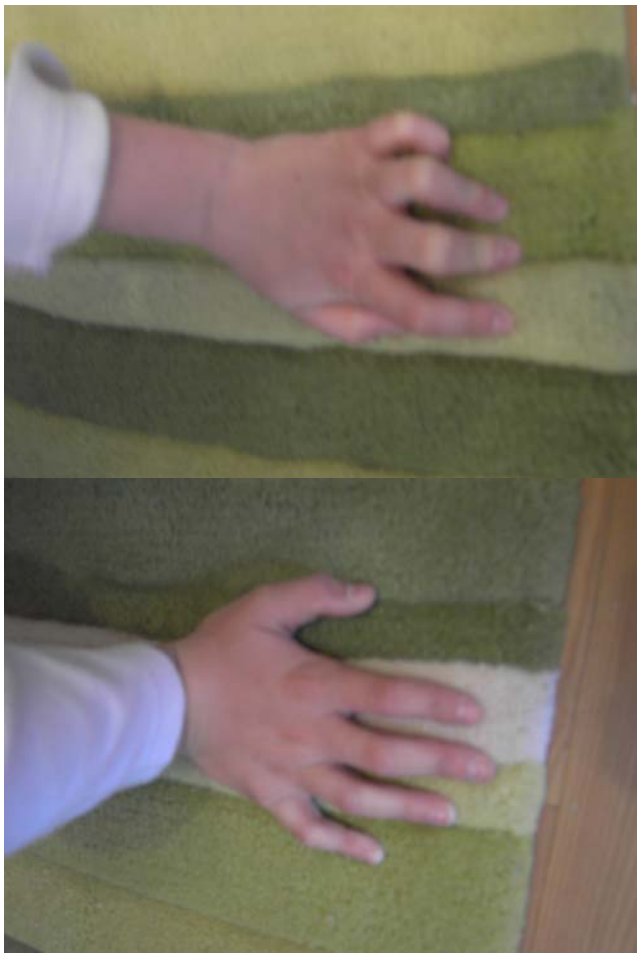


Fig. 7 Hands pressing down / Schönau 2006

In the lower extremities the difference is not as great as in the upper extremities. Both legs are almost equally affected, the left leg somewhat more so.
The hips are flexible on both sides, although in the left hip there is greater tension in abduction and outer rotation. Matthias admits to feeling pain in the knee joint of his left leg when it is completely extended, because the ischio-crural musculature is stretched.
Originally due to his spasticity Matthias' had talipes equines in both feet. Through the botox treatment and the subsequent casts his feet are now in a 90% position. Actively, he can only move them a little and passively, they cannot be moved much either. Hence he has developed talipes planus in both feet.

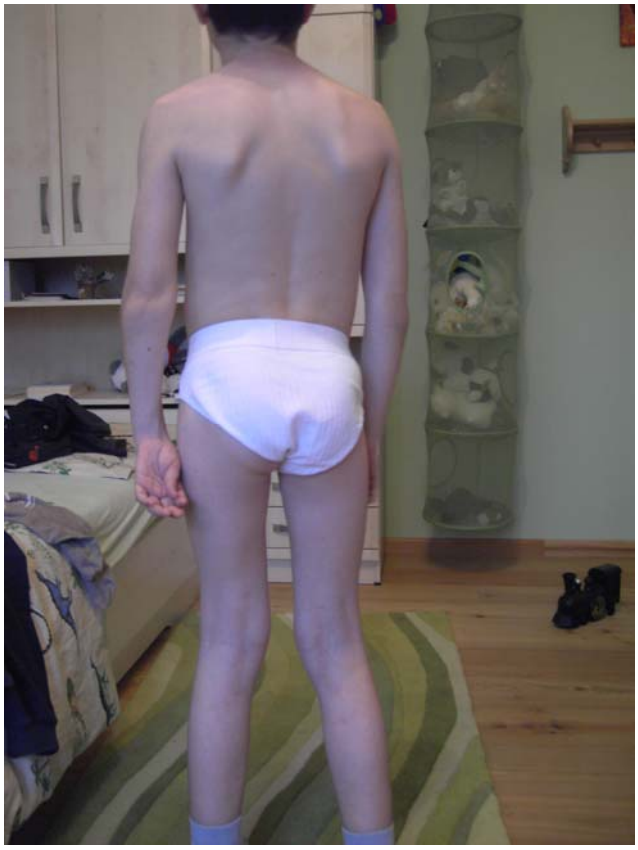


Fig. 8 Standing – dorsal view/Schönau 2006

Fig. 9 The feet - dorsal view / Schönau 2006

The entire right side is more ventral.

The spine shows a long-curved scoliosis with convexity to the right. Segmental flexibility is good. The respiratory movement of the ribs is better on the right than on the left side. The back and shoulder musculature is markedly weaker on the left side. The left arm appears shorter, being somewhat flexed due to the spasticity in that joint.
From the anterior, it is noticeable that the position of the left clavicula is more raised. The lumbar column shows hyperlordosis, which is more extreme when Matthias moves.

Matthias has a narrow, high head form. This “extended skull” is combined with a raised palate Therefore there is not much room for his teeth. His palate is being stretched with a brace. After this treatment he will get fixed brackets.
The SSB is in an extension/torsion pattern.

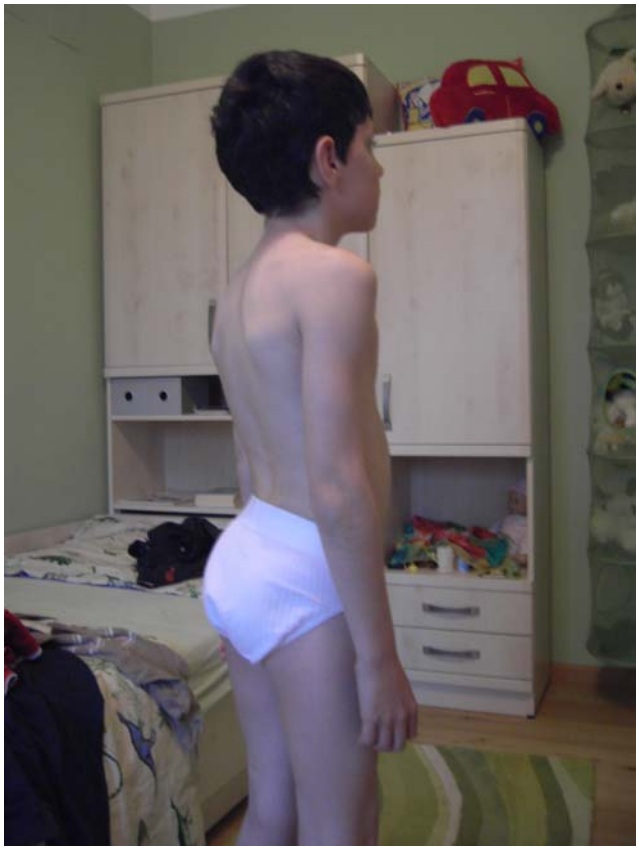


Fig.10 Standing – view from the right side / Schönau 2006

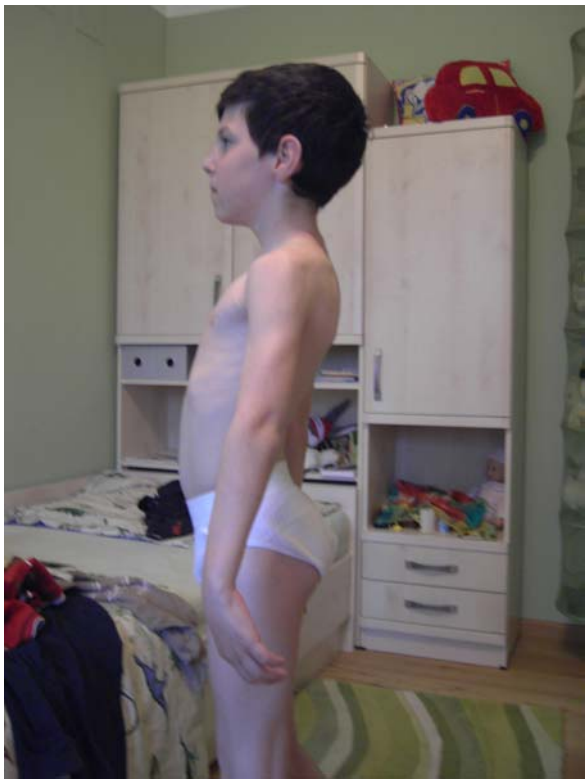


Fig.11 Standing – view from the left side / Schönau 2006

3.2 Osteopathic Treatment

Osteopathic treatment was carried out in five 45-minute sessions. Various osteopathic techniques were used, which were individually adapted to the patient's diagnosis and therapeutic situation.

"Osteopathic and cranial-sacral treatment is always individual, for no patient is like another, and even the same patient differs from one day to the next in the state of tension in his tissues and in his energy potential." (Liem 2001 p 334)

Treatment can, therefore, not be diagnosed and prescribed for all children with cerebral palsy alike.

The main focus of Matthias' treatment is on:

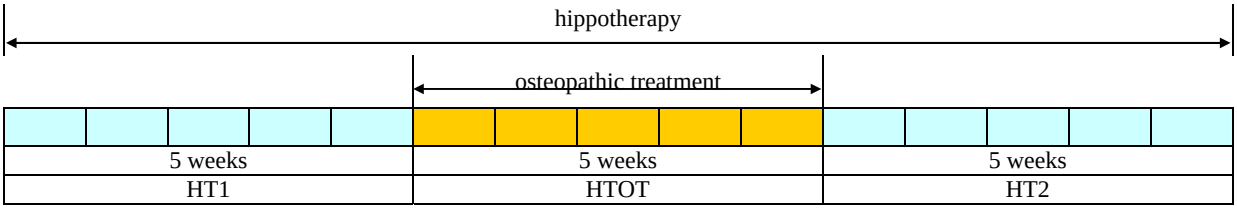
- alleviation of tension in the musculature of the left side
- techniques for balancing the right and left halves of the body.
- mobilisation of the joints, in particular the lower extremities and the left arm
- improvement of respiratory movement
- working on the extension pattern of the skull to alleviate tension

The first aim is to alleviate chronic muscle spasms which can hamper his normal everyday activities and disturb sleep. In older children capable of following instructions, such as Matthias, osteopathic techniques for passive or isometric muscular stretching are especially effective, such as, e.g. muscle-energy techniques and strain-counterstrain techniques, in particular in the region of the pectoral or abdominal girdle. Balanced ligamentous tension techniques and other "indirect" techniques are also well tolerated by these patients and have proven effective. (Carreiro 2004)

Another aim in osteopathic treatment of children with infantile cerebral palsy consists of changing the proprioceptive input of joints, connective tissues and muscles, which have an adverse affect on posture, balance and movement. For contractures of the joints, scoliosis and hip dysplasia are all complications which originate from chronic muscle spasms, abnormal postural strength and altered joint mechanics. (Carreiro 2004)

3.3 Procedure

For this study I used a withdrawal design:
Matthias received hippotherapy once a week for 15 weeks. Additionally, he was treated osteopathically five times in weekly intervals (cf. Chapter 3.2) on the same days as he had hippotherapy. His timetable is shown in Ill. 1.



Ill. 1: Timetable

Every evening during this period three variables were measured by his mother in order to assess the mobility of the musculoskeletal system:

- the right and left knee-floor distance while sitting crossed-legged, measured in centimetres (**rkf** [cm] and **lkf** [cm], respectively)
- the fist-floor distance in a forwards bent standing position (**st** [cm])

Fig. 12a fist-floor distance 12b fist-floor distance/ side view 12c knee-floor distance

In these tests, characteristic flexion and abduction in the coxofemoral joint, flexion of the lumbar spine, extension and flexion of the knees and the dorsal extension of the foot are observed.
Additionally, Matthias' general sense of well-being is assessed by means of a “Pain faces” scale (by Glaxo Smith Kline).

3.4 Evaluation of the Data

The statistics program used was SPSS 12.0.®

The dependent variables are:

- lkf
- rkf
- st

The “Pain faces” scale was not evaluated due to uniform data.

The independent variables are:

- Test sequence
  - nT no treatment
  - HT1 hippotherapy
  - HTOT hippotherapy and osteopathic treatment
  - HT2 hippotherapy
- Day after treatment (0-6)

Sample sizes:

Sequence	n	Days after treatment						
	total	0	1	2	3	4	5	6
nT	2	-	-	-	-	-	-	-
HT1	34	5	5	5	5	4	5	5
HTOT	35	5	5	5	5	5	5	5
HT2	33	5	5	5	5	5	4	4

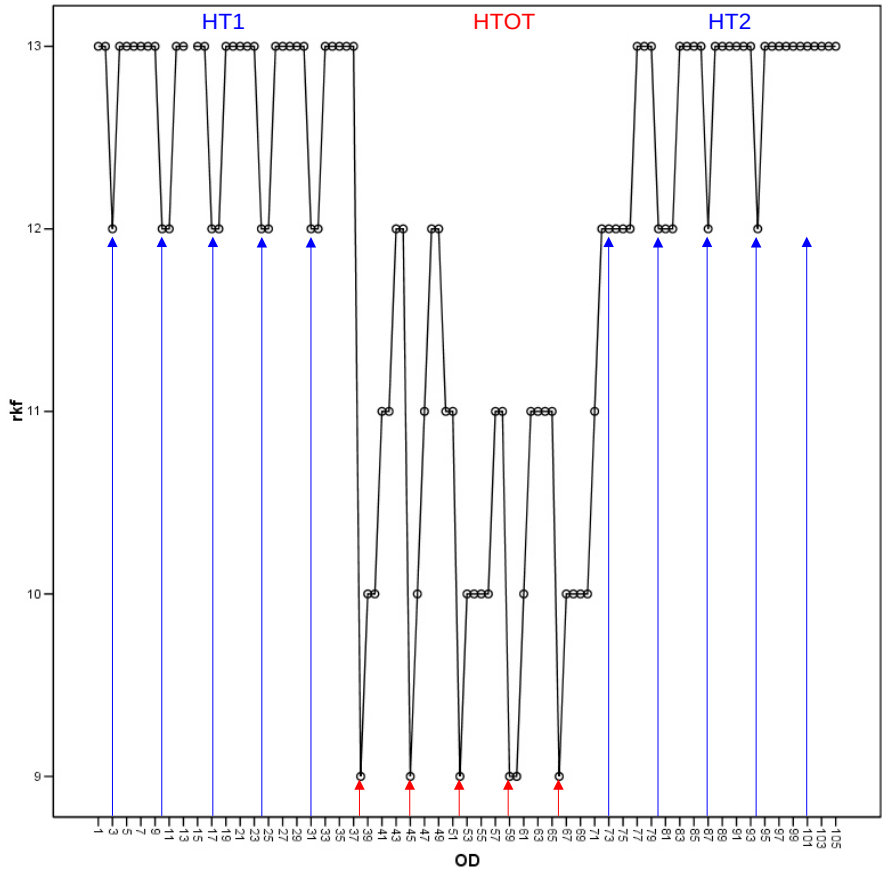
First of all, a descriptive analysis was carried out comprising mean values, standard deviation and 95%-confidence intervals of the dependent variables grouped by the independent ones. The data of the four samples were compared by means of 95% confidence intervals.
In addition, the dependent variables were tested with GLM (univariate analysis of variance) with regard to differences between therapy periods and differences between days after therapy (factors). Since sample sizes are unequal, in addition to the least significant difference test (Fisher's LSD) the Turkey-Kramer test was used for significance testing (Type III sum of squares, level of significance = 0.05).

For the graphical representation of the data error bar plots were used (mean values ± 95% confidence interval).

4. Results

4.1 The Right Knee-Floor Distance

In Ill. 2 an overview of the changes in the right knee-floor distance during the observation phase is given.



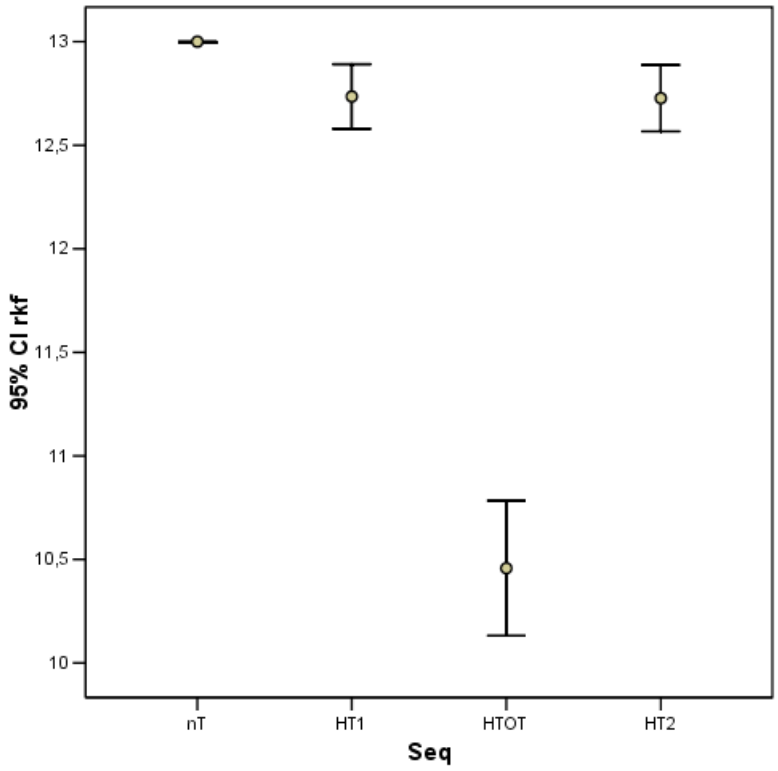
Ill. 2: Matthias' right knee-floor distances (rkf) during the observation phase. "OD" designates the observation day. Days when Matthias had hippotherapy are marked with blue arrows, days when Matthias had additional osteopathic treatment with red arrows.

On the days when Matthias had some form of therapy (hippotherapy: blue arrows, hippotherapy and osteopathic treatment: red arrows), the knee-floor distance is reduced, indicating increased mobility of the musculoskeletal structure. Hippotherapy with no osteopathic treatment results in a smaller improvement (maximum 1 cm) than both therapies combined (maximum 4 cm).

Mean values, 95%-confidence intervals (95%CI) and standard deviations for the different periods (nT, HT1, HTOT and HT2) are summarised in Table 1. An overview of mean values and 95%-confidence intervals of the right knee-floor distance (rkf) in the different phases of therapy is seen by comparison with Ill. 3.

	n	Mean	±95%CI	Std. dev.
nT	2	13	0.00	0
HT1	34	12.7	0.16	0.45
HTOT	35	10.5	0.33	0.95
HT2	33	12.7	0.16	0.45

Table 1: Mean values, 95%-confidence intervals (95% CI) and standard deviations of the right knee-floor distance during the different periods of therapy.



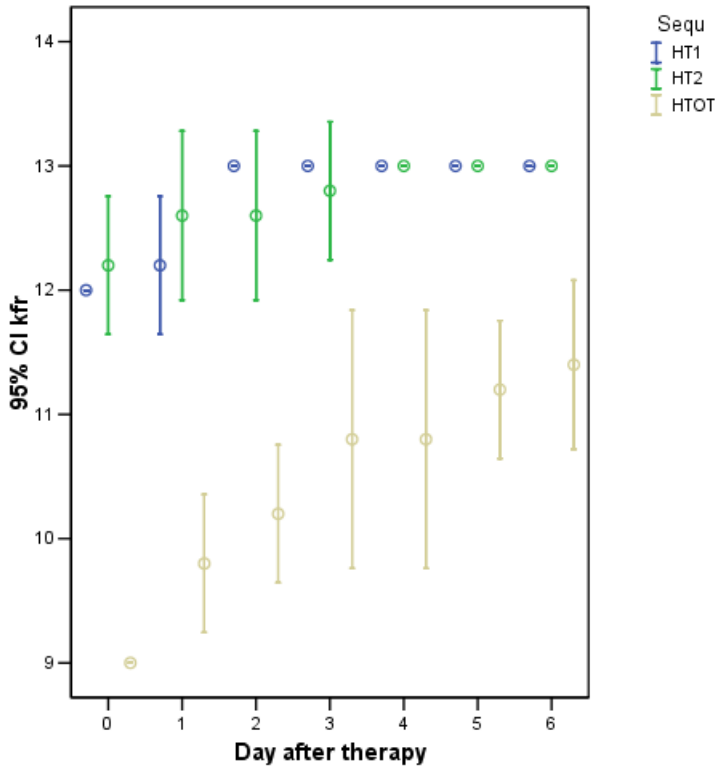
Ill. 3: Mean values and 95%-confidence intervals of Matthias' right knee-floor distance (rkf), for each therapy phase (nT: no therapy, HT1: first hippotherapy period, HTOT: hippotherapy and osteopathy, HT2: second hippotherapy period).

The baseline of Matthias' right knee-floor distance (nT) before receiving some form of therapy is 13 cm. By means of hippotherapy (both periods) an average improvement of 0.3 cm could be attained. During the period with additional osteopathic treatment the average right knee-floor distance was 10.5 cm - that means an average improvement of 2.5 cm. The 95%-confidence intervals as well as the results of analysis of variance (cf. Table 2) indicate a **significant reduction of the right knee-floor distance** during this period compared to the baseline before the therapies, as well as the knee-floor distances during the periods when Matthias had hippotherapy alone. The mean improvement is 2.28 cm compared to the preceding HT1 period (95% CI: -2.69 to -1.86 cm).

(I) Seq	(J) Seq	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
HTOT	HT1	-2.28(*)	0.159	<0.001	-2.69	-1.86
	HT2	-2.27(*)	0.161	<0.001	-2.69	-1.85
	nT	-2.54(*)	0.481	<0.001	-3.80	-1.29

Table 2: Mean differences in the right knee-floor distance during the HTOT period and the other phases, standard error, 95% confidence intervals of the mean differences and statistical significance of the differences.

As can be already seen in the overview chart (Ill. 2), Matthias' mobility decreases over time after therapy, which can be observed in an increase in the knee-floor distance.
In Ill. 4, a summary is given of the right knee-floor distance grouped by days after treatment.



Ill. 4: Mean values and 95% confidence intervals (95% CI) of the right knee-floor distance on different days after treatment.

In Ill. 4 it can be observed that in the first hippotherapy period (HT1) the original state of the right knee-floor distance in all cases (no 95%CI -whiskers) is reached on the second day after treatment, whereas in the second hippotherapy period (HT2) this state is reached on the fourth

day. During the HTOT period the extent of deterioration is slower and within one week after treatment the original state is not reached again.

An analysis of variance with the data of each individual day after therapy (0-6) shows that significant differences between the right knee-floor distances during the HTOT phase compared to the other two periods (HT1 and HT2) can be observed for each day (cf. Table 3 and Table 4). That means additional osteopathic treatment results in a more distinct and also longer-lasting reduction of the right knee-floor distance. In addition, the original restriction of mobility (nT and HT1) is not reattained during the HTOT phase.

Day after therapy	Sequ.	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
before therapy	nT	13.00			
0	HT1	12.00	0.115	11.75	12.25
	HT2	12.20	0.115	11.95	12.45
	HTOT	9.00	0.115	8.75	9.25
1	HT1	12.20	0.216	11.73	12.67
	HT2	12.60	0.216	12.13	13.07
	HTOT	9.80	0.216	9.33	10.27
2	HT1	13.00	0.183	12.60	13.40
	HT2	12.60	0.183	12.20	13.00
	HTOT	10.20	0.183	9.80	10.60
3	HT1	13.00	0.245	12.47	13.53
	HT2	12.80	0.245	12.27	13.33
	HTOT	10.80	0.245	10.27	11.33
4	HT1	13.00	0.252	12.44	13.56
	HT2	13.00	0.226	12.50	13.50
	HTOT	10.80	0.226	10.30	11.30
5	HT1	13.00	0.121	12.73	13.27
	HT2	13.00	0.135	12.70	13.30
	HTOT	11.20	0.121	10.93	11.47
6	HT1	13.00	0.148	12.67	13.33
	HT2	13.00	0.165	12.64	13.36
	HTOT	11.40	0.148	11.07	11.73

Table 3: Mean values, 95%-confidence intervals (95% CI) and standard deviations of the right knee-floor distance for the days after therapy grouped by the different therapy periods.

Even on the 6th day after therapy the upper bound of the 95% confidence interval (95% CI) during the HTOT period is lower than the lower bound of the 95% CI of the day of therapy during the hippotherapy periods, indicating a more sustainable effect of osteopathic therapy.

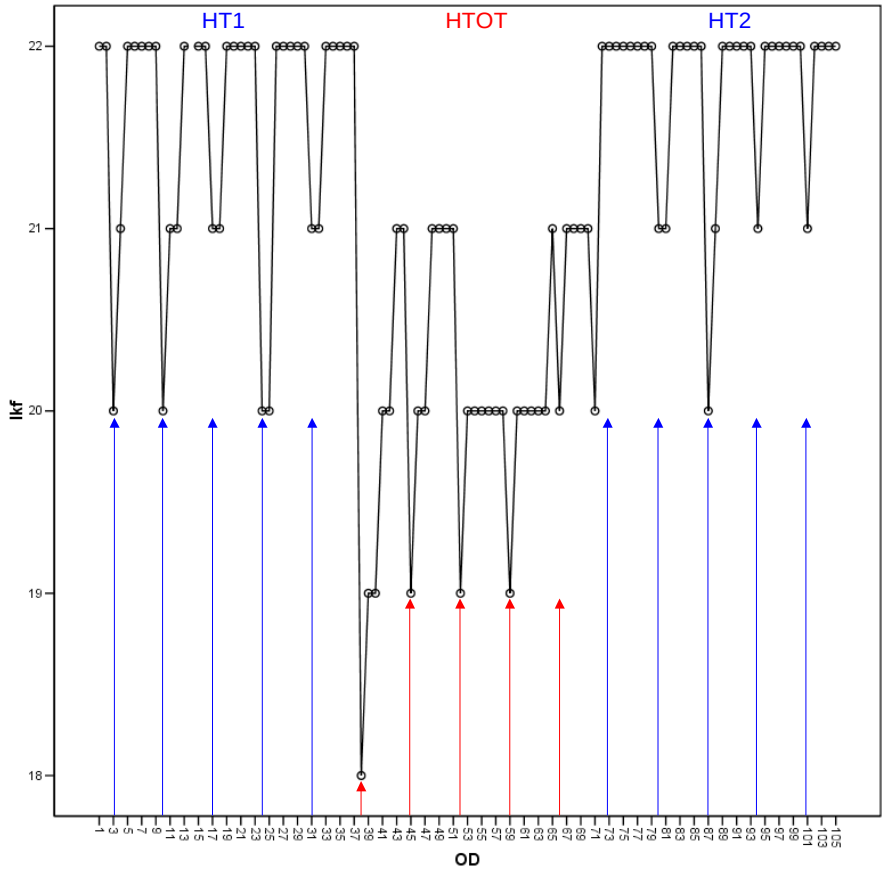
Day after therapy	(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval for Difference	
						Lower Bound	Upper Bound
0	HTOT	HT1	-3.00	0.163	<0.001	-3.36	-2.64
		HT2	-3.20	0.163	<0.001	-3.56	-2.84
1	HTOT	HT1	-2.40	0.306	<0.001	-3.07	-1.73
		HT2	-2.80	0.306	<0.001	-3.47	-2.13
2	HTOT	HT1	-2.80	0.258	<0.001	-3.36	-2.24
		HT2	-2.40	0.258	<0.001	-2.96	-1.84
3	HTOT	HT1	-2.20	0.346	<0.001	-2.95	-1.45
		HT2	-2.00	0.346	<0.001	-2.75	-1.25
4	HTOT	HT1	-2.20	0.338	<0.001	-2.94	-1.46
		HT2	-2.20	0.319	<0.001	-2.90	-1.50
5	HTOT	HT1	-1.80	0.171	<0.001	-2.18	-1.42
		HT2	-1.80	0.181	<0.001	-2.20	-1.40
6	HTOT	HT1	-1.60	0.209	<0.001	-2.06	-1.14
		HT2	-1.60	0.222	<0.001	-2.09	-1.11

Table 4: Mean differences in the right knee-floor distance between the HTOT period and the other phases, standard error, 95% confidence intervals of the mean differences and statistical significance of the differences after grouping by days after treatment.

The mean difference in the right knee-floor distance during the HTOT phase compared to the hippotherapy phases ranges from maximum 3.2 cm on the day of treatment to minimum 1.6 cm six days after the treatment. Differences are statistically significant on each day. Therefore it can be concluded that a higher efficacy is gained by a combination of the treatments. In addition, a more sustainable improvement in the patient's mobility can be achieved.

4.2 The Left Knee-Floor Distance

Matthias' left body side is more seriously affected than his right side. This can be seen in the greater initial knee-floor distance on the left than on the right. The changes in the left knee-floor distance during the observation phase can be observed in Ill. 5.



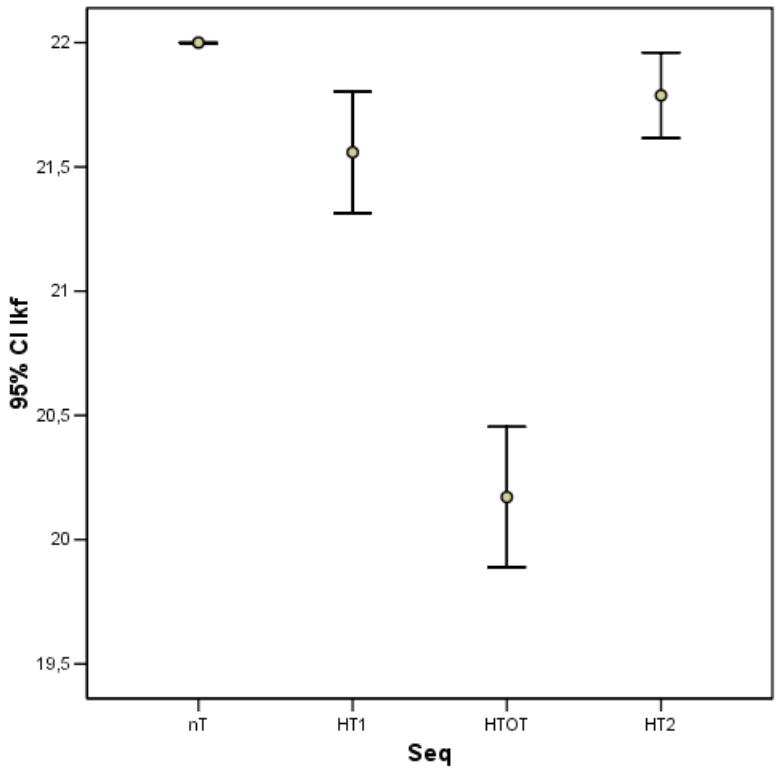
Ill. 5: Matthias' left knee-floor distances (lkf) during the observation phase. "OD" designates the observation day. Days when Matthias had hippotherapy are marked with blue arrows, days when Matthias had additional osteopathic treatment with red arrows.

Improvements in Matthias' mobility on the left side of his body can also be seen on the days when he had some form of therapy (hippotherapy: blue arrows, hippotherapy and osteopathic treatment: red arrows). Again, hippotherapy with no osteopathic treatment results in a smaller improvement (maximum 2 cm) than the combination of both therapies (maximum 4 cm). Mean values and standard deviations for the different periods and 95% confidence intervals (95% CI) are summarised in Table 5. For easier comparison, mean values and 95% CI of the

left knee-floor distance (lkf) during the different therapy phases (nT, HT1, HTOT and HT2) are additionally summarised in Ill. 6.

Sequ.	n	Mean	±95%CI	Std. dev.
nT	2	22	0.00	0
HT1	34	21.6	0.25	0.70
HTOT	35	20.2	0.28	0.82
HT2	33	21.8	0.17	0.48

Table 5: Mean values, 95%-confidence intervals (95% CI) and standard deviations in the left knee-floor distance during the different periods of therapy.



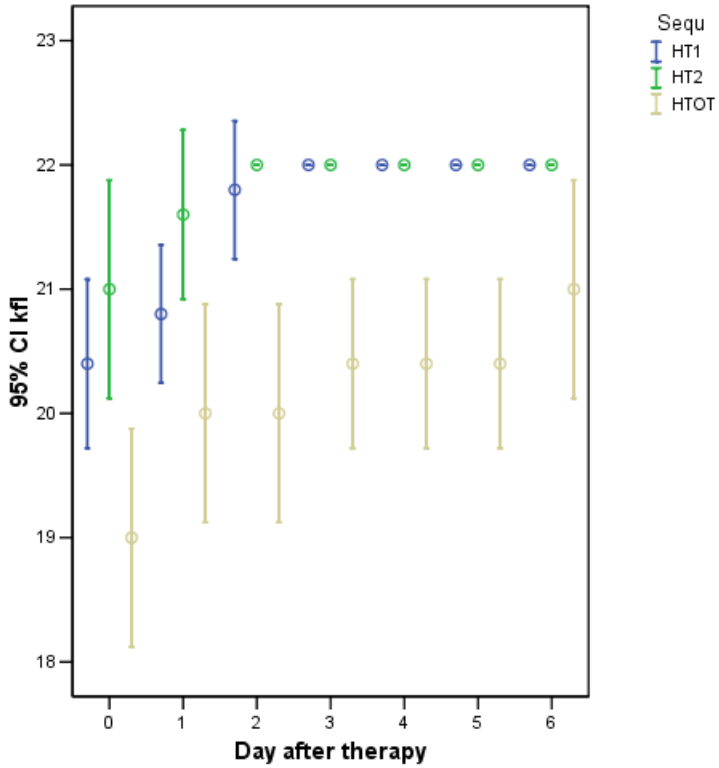
Ill. 6: Mean values and 95%-confidence intervals in Matthias' left knee-floor distance (lkf), for each therapy sequence (nT: no therapy, HT1: first hippotherapy sequence, HTOT: hippotherapy and osteopathy, HT2: second hippotherapy sequence).

The baseline of Matthias' left knee-floor distance before the different therapies (nT) is 22 cm. During the period with combined hippotherapy and osteopathic treatment (HTOT) the average left knee-floor distance is 20.2 cm. According to the 95%-confidence intervals and analysis of variance (results cf. Table 6). these differences are statistically significant.

(I) Seq	(J) Seq	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
HTOT	HT1	-1.39(*)	0.165	<0.001	-1.82	-0.96
	HT2	-1.62(*)	0.166	<0.001	-2.05	-1.18
	nT	-1.83(*)	0.498	0.002	-3.13	-0.53

Table 6: Mean differences in the left knee-floor distance during the HTOT period and the other periods, standard error, 95% confidence intervals of the mean differences and significance.

As can be observed in the overview chart (Ill. 5), Matthias' left-hand mobility decreases over time after therapy, too. In Ill. 7, a summary of the left knee-floor distance grouped by days after treatment is given.



Ill. 5: Mean values and 95% confidence intervals (95% CI) of the left knee-floor distance on different days after treatment.

In Ill. 7 it can be observed that in the first hippotherapy period (HT1) the original state of the left knee-floor distance **in all cases** (no 95%CI -whiskers) is reached on the third day after treatment, whereas in the second hippotherapy period this state is reached one day earlier. During the HTOT period a deterioration of mobility to the original state can only be observed once, at the 6th day after the fifth treatment (cf. Ill. 5). Nevertheless, the data indicate that the

combination of the treatments results in a more sustainable improvement in the patient's mobility.

An analysis of variance with the data of each individual day after therapy (0-6), shows that significant differences between the left knee-floor distances during the three periods can be observed for each individual day after therapy (cf. Table 7 and Table 8) In addition, the original restriction of mobility (nT and HT1) is not reattained during the HTOT phase. This means, additional osteopathic treatment results in a more distinct and also longer-lasting reduction of the left knee-floor distance.

Day after therapy	Sequ.	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
before therapy	nT	22.00			
0	HT1	20.40	0.294	19.76	21.04
	HT2	21.00	0.294	20.36	21.64
	HTOT	19.00	0.294	18.36	19.64
1	HT1	20.80	0.258	20.24	21.36
	HT2	21.60	0.258	21.04	22.16
	HTOT	20.00	0.258	19.44	20.56
2	HT1	21.80	0.216	21.33	22.27
	HT2	22.00	0.216	21.53	22.47
	HTOT	20.00	0.216	19.53	20.47
3	HT1	22.00	0.141	21.69	22.31
	HT2	22.00	0.141	21.69	22.31
	HTOT	20.40	0.141	20.09	20.71
4	HT1	22.00	0.165	21.64	22.36
	HT2	22.00	0.148	21.67	22.33
	HTOT	20.40	0.148	20.07	20.73
5	HT1	22.00	0.148	21.67	22.33
	HT2	22.00	0.165	21.64	22.36
	HTOT	20.40	0.148	20.07	20.73
6	HT1	22.00	0.191	21.58	22.42
	HT2	22.00	0.213	21.53	22.47
	HTOT	21.00	0.191	20.58	21.42

Table 7: Mean values, 95%-confidence intervals (95% CI) and standard deviations of the left knee-floor distance for the days after therapy grouped by the different therapy periods.

The effect of additional osteopathic treatment is lower on the left side than on t right, where even on the 6th day after therapy the upper bound of the 95% confidence interval during the HTOT period is lower than the lower bound of the 95% confidence interval of the day of therapy during the other periods. On the left side an overlapping of the confidence intervals of the data from the different periods can already be noted on the first day after therapy. Nevertheless, deterioration of Matthias' mobility is slower during the period from two to six days after treatment.

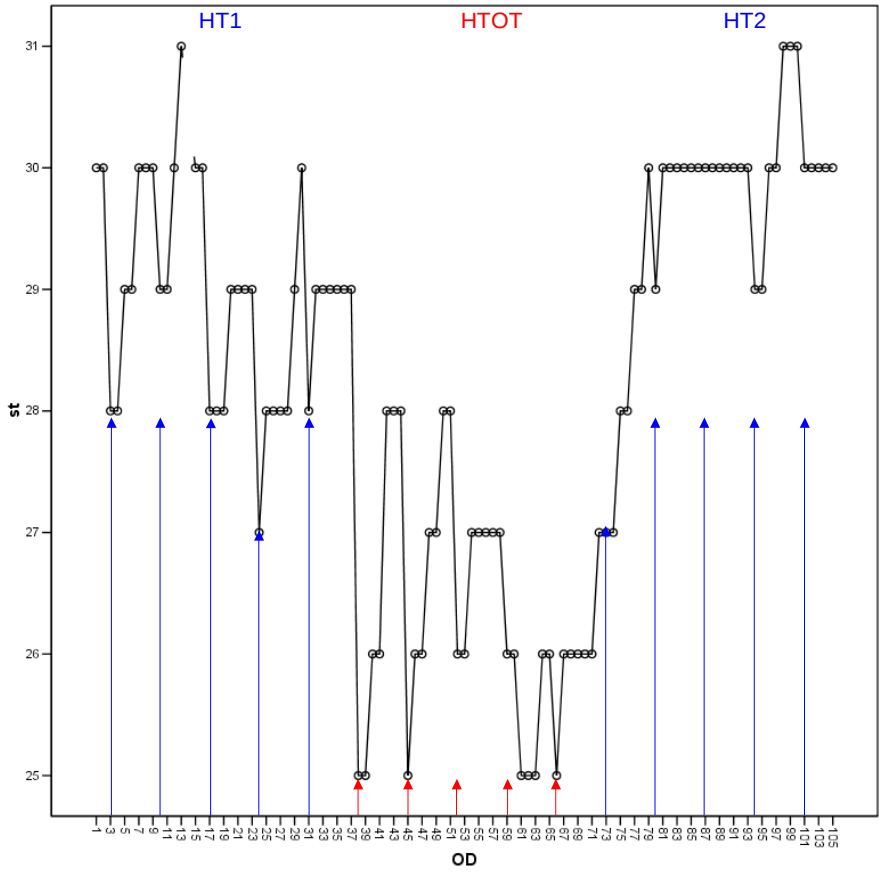
Day after therapy	(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval for Difference	
						Lower Bound	Upper Bound
0	HTOT	HT1	-1.40	0.416	0.006	-2.31	-0.49
		HT2	-2.00	0.416	<0.001	-2.91	-1.09
1	HT1	HT2	-0.80	0.365	0.049	-1.60	0.00
	HT2	HT1	0.80	0.365	0.049	0.00	1.60
	HTOT	HT1	-0.80	0.365	0.049	-1.60	0.00
		HT2	-1.60	0.365	0.001	-2.40	-0.80
2	HTOT	HT1	-1.80	0.306	<0.001	-2.47	-1.13
		HT2	-2.00	0.306	<0.001	-2.67	-1.33
3	HTOT	HT1	-1.60	0.200	<0.001	-2.04	-1.16
		HT2	-1.60	0.200	<0.001	-2.04	-1.16
4	HTOT	HT1	-1.60	0.222	<0.001	-2.09	-1.11
		HT2	-1.60	0.209	<0.001	-2.06	-1.14
5	HTOT	HT1	-1.60	0.209	<0.001	-2.06	-1.14
		HT2	-1.60	0.222	<0.001	-2.09	-1.11
6	HTOT	HT1	-1.00	0.270	0.003	-1.59	-0.41
		HT2	-1.00	0.286	0.005	-1.63	-0.37

Table 8: Mean differences in the left knee-floor distance during the HTOT period and the other phases, standard error, 95% confidence intervals of the mean differences and statistical significance of the differences after grouping by days after treatment.

The mean difference in the left knee-floor distance during the HTOT phase compared to the phases when Matthias has hippotherapy alone ranges from maximum 2.0 cm on the day of treatment to minimum 0.8 cm one day after the treatment. Due to the longer-lasting effect of osteopathic treatment, again higher differences can be observed on the days 2-6 after treatment. The differences are statistically significant for each day after treatment. Therefore it can be concluded that a higher efficacy is gained by a combination of the treatments. In addition, a more sustainable improvement in the patient's mobility can be achieved.

4.3 The Fist-Floor Distance

The changes in the fist-floor distance during the observation phase are summarised in Ill. 8.



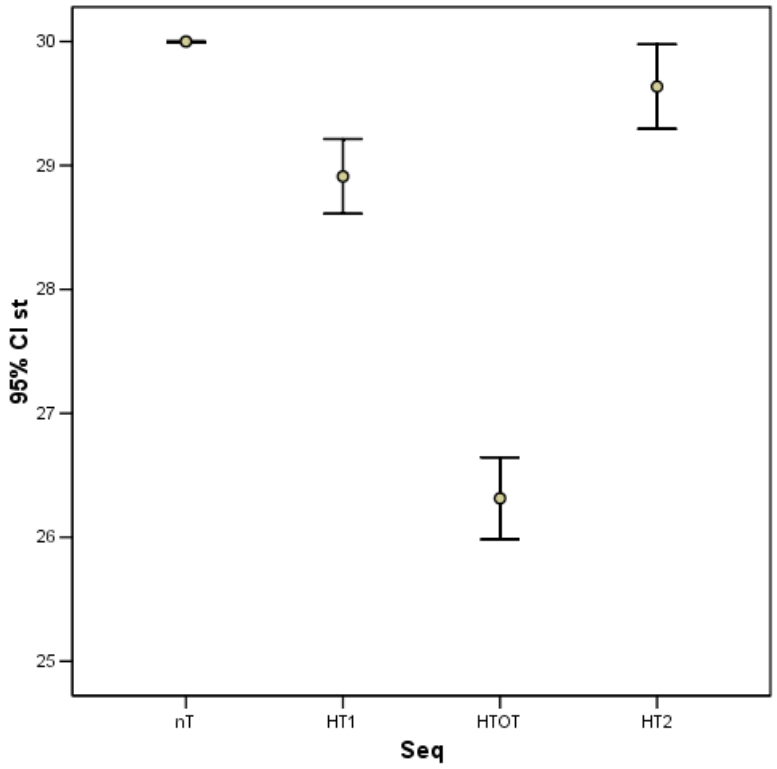
Ill. 8: Matthias' fist-floor distances (st) over the observation phase. "OD" designates the observation day. Days when Matthias had hippotherapy are marked with blue arrows, days when Matthias had additional osteopathic treatment with red arrows.

Improvements in Matthias' mobility can be noted on the days when he had some form of therapy (hippotherapy: blue arrows, hippotherapy and osteopathic treatment: red arrows). Again, hippotherapy with no osteopathic treatment results in a smaller improvement (maximum 2 cm) than the combination of both therapies (maximum 4 cm).

Mean values of and standard deviations in the fist-floor distance during the different periods and 95% confidence intervals are summarised in Table 9. For easier comparison, mean values and 95%-confidence intervals of the fist-floor distance (st) of the different therapy phases (nT, HT1, HTOT and HT2) are summarised in addition in Ill. 9.

Sequ.	n	Mean	±95%CI	Std. dev.
nT	2	30	0.00	0
HT1	34	28.9	0.30	0.87
HTOT	35	26.3	0.33	0.96
HT2	33	29.6	0.34	0.96

Table 9: Mean values, 95%-confidence intervals (95% CI) and standard deviations in the fist-floor distance during the different therapy periods.



Ill. 9: Mean values and 95%-confidence intervals of Matthias' fist-floor distance (st), for each therapy sequence (nT: no therapy, HT1: first hippotherapy sequence, HTOT: hippotherapy and osteopathy, HT2: second hippotherapy sequence).

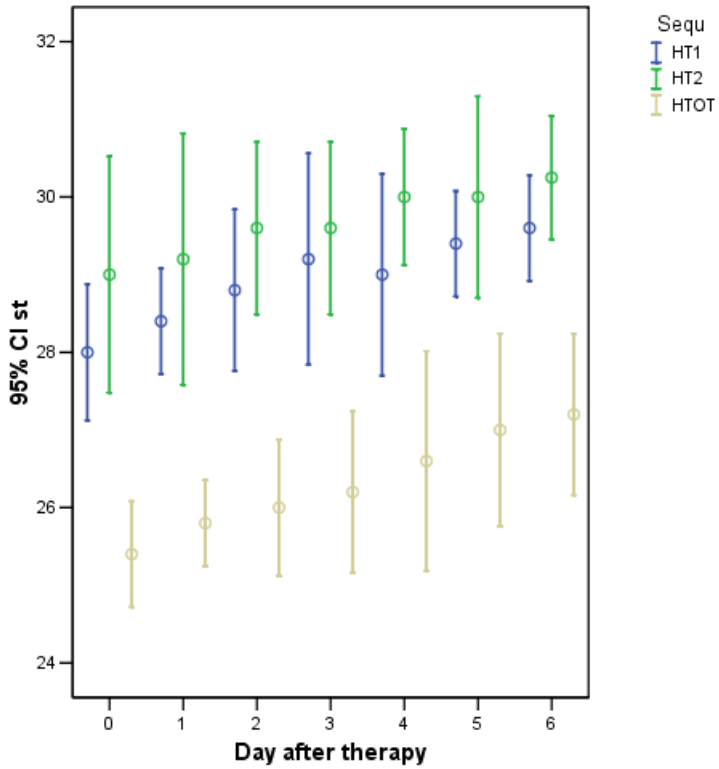
The baseline of Matthias' fist-floor distance before the different therapies (nT) is 30 cm. During the period with combined hippotherapy and osteopathic treatment, the average left knee-floor distance is 26.3 cm. According to the 95%-confidence intervals and analysis of variance these differences are statistically significant.

Moreover, significant differences in the fist-floor distance between the first and the second periods of hippotherapy can be observed. In the second period of hippotherapy mobility is restricted to a greater extent.

(I) Seq	(J) Seq	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
HT1	HT2	-0.72(*)	0.227	0.010	-1.32	-.13
	HTOT	2.60(*)	0.223	0.000	2.01	3.18
HTOT	HT1	-2.60(*)	0.223	0.000	-3.18	-2.01
	HT2	-3.32(*)	0.225	0.000	-3.91	-2.73
	nT	-3.69(*)	0.674	0.000	-5.45	-1.93

Table 10: Mean differences in the fist-floor distance during the HTOT period and the other periods, standard error, 95% confidence intervals of the mean differences and significance.

As could already be observed in the overview chart (Ill. 8) Matthias' mobility decreases after therapy over time. In Ill. , a summary of the fist-floor distance grouped by days after treatment is given.



Ill. 10: Mean values and 95% confidence intervals (95% CI) of the fist-floor distance on different days after treatment.

Compared to the knee-floor distances variability is higher. Again, original values of the fist-floor distance are not reached during the period when Matthias had both hippotherapy and

osteopathic treatment (HTOT). During the periods when he had hippotherapy alone (HT1 and HT2) even higher values can be observed than those before the therapies.
An analysis of variance with the data of each individual day after therapy (0-6) shows that significant differences between the fist-floor distance can be observed between the hippotherapy periods and the HTOT period for each individual day (cf. Table and Table). Moreover, the original restriction of mobility (nT and HT1) is not reattained during the HTOT phase. Therefore, additional osteopathic treatment results in a more distinct and thus longer-lasting reduction of the fist-floor distance.

Day after therapy	Sequ.	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
before therapy	nT	30.00			
0	HT1	28.00	0.392	27.15	28.85
	HT2	29.00	0.392	28.15	29.85
	HTOT	25.40	0.392	24.55	26.25
1	HT1	28.40	0.383	27.57	29.23
	HT2	29.20	0.383	28.37	30.03
	HTOT	25.80	0.383	24.97	26.63
2	HT1	28.80	0.365	28.00	29.60
	HT2	29.60	0.365	28.80	30.40
	HTOT	26.00	0.365	25.20	26.80
3	HT1	29.20	0.424	28.28	30.12
	HT2	29.60	0.424	28.68	30.52
	HTOT	26.20	0.424	25.28	27.12
4	HT1	29.00	0.457	27.99	30.01
	HT2	30.00	0.409	29.10	30.90
	HTOT	26.60	0.409	25.70	27.50
5	HT1	29.40	0.362	28.60	30.20
	HT2	30.00	0.405	29.11	30.89
	HTOT	27.00	0.362	26.20	27.80
6	HT1	29.60	0.294	28.95	30.25
	HT2	30.25	0.329	29.53	30.97
	HTOT	27.20	0.294	26.55	27.85

Table 11: Mean values. 95%-confidence intervals (95% CI) and standard errors in the fist-floor distance for the days after therapy grouped by the different periods of therapy.

On the 3rd day after treatment the upper bound of the 95% confidence interval during the HTOT period is lower than the lower bound of the 95% confidence interval of the day of therapy during the other periods. On the 6th day after treatment, average fist-floor distance is

still lower than on the second day after hippotherapy with no additional osteopathic treatment.

Day after therapy	(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval for Difference	
						Lower Bound	Upper Bound
0	HTOT	HT1	-2.60	0.554	0.001	-3.81	-1.39
		HT2	-3.60	0.554	<0.001	-4.81	-2.39
1	HTOT	HT1	-2.60	0.542	<0.001	-3.78	-1.42
		HT2	-3.40	0.542	<0.001	-4.58	-2.22
2	HTOT	HT1	-2.80	0.516	<0.001	-3.93	-1.67
		HT2	-3.60	0.516	<0.001	-4.73	-2.47
3	HTOT	HT1	-3.00	0.600	<0.001	-4.31	-1.69
		HT2	-3.40	0.600	<0.001	-4.71	-2.09
4	HTOT	HT1	-2.40	0.613	0.002	-3.75	-1.05
		HT2	-3.40	0.578	<0.001	-4.67	-2.13
5	HTOT	HT1	-2.40	0.512	0.001	-3.53	-1.27
		HT2	-3.00	0.543	<0.001	-4.19	-1.81
6	HTOT	HT1	-2.40	0.416	<0.001	-3.31	-1.49
		HT2	-3.05	0.441	<0.001	-4.02	-2.08

Table 12: Mean differences in the fist-floor distance during the HTOT period and the other phases, standard error, 95% confidence intervals of the mean differences and statistical significance of the differences after grouping by days after treatment.

The mean reduction of the fist-floor distance during the HTOT phase compared to the phases when Matthias has only hippotherapy ranges from 2.6 cm on the day of treatment to 3.05 cm on the sixth day after treatment. The differences are statistically significant for each individual day. Therefore it can be concluded that a higher efficacy is gained by a combination of the treatments. Moreover, a more sustainable improvement in the patient's mobility can be achieved.

5. Discussion

5.1 Discussion of the Method

This study examines the question whether osteopathy in addition to hippotherapy is a medically sensible therapeutic measure for the treatment of children with cerebral palsy. The hypothesis of this study is that osteopathic treatment combined with hippotherapy is indeed more effective than hippotherapy alone in increasing the extent of movement and the general wellbeing of a child with cerebral palsy,
I have chosen a withdrawal design since children with cerebral palsy usually receive hippotherapy or physiotherapy on a regular basis and they cannot be expected to forego this therapy for a longer period of time. My study covered a period of 15 weeks.

The difficulty began with the choice of a suitable proband. I have several children with cerebral palsy as patients and finally decided to choose Matthias since both he and his mother were extremely cooperative and I could be certain of their compliance. Another reason was that I had occasionally treated Matthias with physiotherapy; however, his hippotherapy took place at a centre for therapeutic riding, which seemed sensible for the purposes of this study. I contacted the hippotherapist and asked if she would agree to the proposed appointments. Naturally, in the case of Matthias' being unavailable (e.g. through illness) I had a reserve proband at my disposal.
After we had talked, Matthias' parents were immediately compliant and we started with the study as agreed which, however, was not very easy, because the intention was that the measurements be taken without a break over a period of fifteen weeks. No charge was made for the osteopathic treatments.
As independent variable I chose the fist-floor distance standing in a flexed back position, as well as the knee-floor distance right and left in a cross-legged position.
To increase reliability I suggested two measurements per day (in the morning and in the evening). Unfortunately, this proved impossible, because Matthias' mother had another handicapped child and a younger, healthy small child to look after. To expect her to cope with the morning stress involved in getting all the children out of the house on time would have been an unreasonable demand. I therefore decided to take three measurements in the evening and select the mean value.

As a valid measuring instrument one centimetre was used and the measurements were taken before bedtime and recorded in a notebook.
Once a week, I carried out the measurements myself in order to get a personal overview and control the regularity.
To test Matthias' sense of wellbeing, I asked him to record how he felt every day on a Smiley scale. Unfortunately, this measurement was much too inexact and could not be evaluated. Every day, Matthias recorded his status as being very good, apart from one time, when he had fallen downstairs and later still felt pain. For this reason it would be a good idea if further studies in this field designed a questionnaire with specific questions for the parents as well as the patients.
Matthias had therapy every Wednesday. Hence only two days could be used to calculate the baseline of mobility. This is also the reason that there are no data for the fifth and sixth days after treatment in the last week. Additionally, one day no measurements were taken. The missing data are the reason for different sample sizes, which would be a precondition for analysis of variance (ANOVA) calculations. Nevertheless, they are considered to be valid despite violating this assumption and the Turkey-Kramer method for multiple comparisons also used does not have this limitation. The Smiley scale was not evaluated due to uniform data.
ANOVA requires relative homogeneity of variances in order to be able to distinguish between differences of group means. This homogeneity is not fully given, but even despite this limitation, the differences in the means are high enough to show the effects of the additional osteopathic treatment. Therefore, no non-parametric tests have been performed. Generally, for ANOVA but other statistical methods, too, greater sample sizes give more reliable results. Therefore, longer observation periods would have been preferable.
Matthias has had hippotherapy and other therapies before. Nevertheless, in spite of a longer break in therapy, the original status (baseline of knee-floor distance and fist-floor distance) is unknown. Since I would only like to evaluate the additional influence of osteopathic treatment, this fact does not influence the essence of the result.
Because this is a single-subject study, the results must not be genera

5.2 Discussion of the Results

By means of additional osteopathic treatment a statistically significant higher degree of efficacy was attained compared to hippotherapy alone. Moreover, a more sustainable improvement of the patient's mobility could be achieved.

On the days when Matthias had some form of therapy (hippotherapy or hippotherapy plus additional osteopathic treatment), knee-floor distances as well as fist-floor distance were reduced, indicating improved mobility of the musculoskeletal structure. Hippotherapy without osteopathic treatment resulted in a smaller improvement than both therapies combined and during the phase when he had both therapies, original restrictions of mobility were not reached anymore.

Matthias' mobility deteriorated more slowly after having had additional osteopathic treatment before. Even on the 6th day after each treatment, knee-floor distances and fist-floor distance were normally lower than before the first osteopathic treatment (with only one exception). This means that during the whole period of additional osteopathic treatment his mobility was higher than in the hippotherapy periods before and afterwards.

The main results are summarised in Table 13.

Right knee-floor distance (rkf)		
Sequ.	Mean value rkf	Mean Difference HTOT- Sequ.
nT	13	-2.54
HT1	12.7	-2.28
HTOT	**10.5**	-
HT2	12.7	-2.27
Left knee-floor distance (lkf)		
Sequ.	Mean value lkf	Mean Difference HTOT- Sequ.
nT	22	-1.83
HT1	21.6	-1.39
HTOT	**20.2**	-
HT2	21.8	-1.62
Fist-floor distance (st)		
Sequ.	Mean value st	Mean Difference HTOT- Sequ.
nT	30	-3.69
HT1	28.9	-2.60
HTOT	**26.3**	-
HT2	29.6	-3.32

Table 13: Mean values and mean differences of the distances between the HTOT period and the other periods [cm].

In Table it can be noted that less improvement was reached by additional osteopathic treatment on the left body side compared to the right side. Nevertheless, the left knee-floor distance also decreased by an average of approx. 1.4 cm compared to the prior HT1 period. Generally, in spite of the improvements during the interjacent phase of combined hippotherapy and osteopathic treatment, no improvements compared to the first hippotherapy period can be observed during HT2. The fist-floor distance even increases from 28.9 cm (HT1) to 29.6 cm (HT2).

On the basis of the statistic results, osteopathy indeed proves a suitable form of therapy for Matthias. The osteopathic studies listed in Chapter 2.7 showed similar results.
The single-case study by Knox (2005) likewise showed the success of osteopathic treatment of the eight-year old boy.
Similarly, in the Steingrube-Bradtke study a significant improvement in the 50 children with cerebral palsy who were treated was recorded.
In the randomised, controlled study made by Duncan et al. in 2004, 96% of parents reported an improvement in their children, particularly in their arm and leg movements and sleep patterns.
Unfortunately not many studies in this field exist, perhaps because there are so many various forms of cerebral palsy, or because cerebral palsy is incurable and one can only alleviate its symptoms or prevent later damage.
From the results of these studies it can be said that osteopathy does indeed have a positive effect on children with cerebral palsy. Further studies are necessary in order to make more use of osteopathy in this field, too.
It would be interesting to carry out this study with several children with cerebral palsy. Similarly, as already mentioned, it would be interesting to determine the general condition of these children by the use of a questionnaire designed for both parents and children. Specific questions would be appropriate here, such as, for example, sleep-wake rhythm, harmony, appetite…
From a more critical point of view, a longer period of observation or a longer phase of osteopathic treatment would have been necessary in my study in order to give it greater significance.

6. Conclusion

In the introduction the common factors and similar scientific approaches of the two forms of therapy (osteopathy and hippotherapy), which motivated me to write this study are explained. In the general part, hippotherapy is described. The division of therapeutic riding (Thr) into three sub-areas, which are very often confused or lumped together, is very important for full understanding of the subject. Subsequently, the training of the hippotherapist and the training horse is explained and the manifold effective factors of this form of therapy are summarised. Since hippotherapy is used to treat not only children with cerebral palsy, all other indications/contra-indications have also been listed. The development of osteopathy, as well as hippotherapy, summarised in Chapter 2.5 is extremely interesting. The general section also includes a description of the clinical symptoms of infantile cerebral palsy.

Here I have summarised the historical course of the symptoms and the various causes of cerebral palsy. This is followed by the clinical picture and the manifestations of infantile cerebral palsy and a description of botulinum toxin therapy. This chapter ends with a review of the present state of research of scientific studies in the field of cerebral palsy – osteopathy – hippotherapy. Here I have listed individual studies on this subject and summarised studies which came to similar conclusions.

The chapter on methodology describes my test person, Matthias (11 years of age), from his birth to his present condition. He is a twin and was born by Caesarean section in the 27th week of pregnancy. He has a mentally and physically challenged brother. At present, Matthias attends an integrated class in the fourth form of a primary school.

Osteopathic treatment and applied techniques and a description of the latter lead into the methodological part of the study. A withdrawal design was chosen (A-B-A) with a duration of 15 weeks. Matthias was regularly given hippotherapy and, in phase B, additional osteopathy. As dependent variables I chose right and left knee-floor distances in a cross-legged seated position, and the fist-floor distance in a standing position. The independent variables result from the following sequences:

- no treatment
- hippotherapy 1
- hippotherapy and osteopathy
- hippotherapy 2

as well as the day after treatment.

The Glaxo Smith Kline Scale for assessing the general condition could not be used in evaluation.
Evaluation of the data follows.
Statistical results of the right and left knee-floor distances confirm the success of osteopathic treatment. All three results show a significant improvement when a combination of osteopathy and hippotherapy is used.

On the days when Matthias had some form of therapy (hippotherapy or hippotherapy and additional osteopathic treatment), knee-floor distances as well as fist-floor distance were reduced, indicating improved mobility of the musculoskeletal structure. Hippotherapy without osteopathic treatment resulted in a significantly smaller improvement than a combination of both therapies. Compared to the original state of mobility, the right knee-floor distance was reduced by an average of 0.3 cm by hippotherapy and 2.5 cm by the combination of hippotherapy and osteopathic treatment. The data for the right knee-floor distance are 0.4 cm and 1.8 cm and for the fist-floor distance 1.1 cm and 3.7 cm, respectively.
Moreover, deterioration of Matthias' mobility was slower when he had had additional osteopathic treatment before and his original restrictions of mobility before he had any therapy were not attained any more.

By means of additional osteopathic treatment a statistically significant greater degree of efficacy was attained compared to treatment with hippotherapy alone. Moreover, a more sustainable improvement in the patient's mobility could be achieved.

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8. Anhang

Die Prinzipien der Osteopathie

Still, der Begründer der Osteopathie, betont die Zusammenhänge zwischen der Mobilität der einzelnen Strukturen (“Leben ist Bewegung“), der Zirkulation sämtlicher Körperflüssigkeiten („Das Gesetz der Arterien“) und der wechselseitigen Beeinflussung von Körperstrukturen und – Funktionen („Die Struktur regiert die Funktion, und die Funktion formt die Struktur.“) Letztendlich sollte ein gutes Zusammenspiel dieser Komponenten dem Körper helfen („Der Körper funktioniert als Einheit“), über seine Autoregulationsmechanismen („Selbstheilungsmechanismen“) zur Gesundheit zu gelangen. (Ligner, Van Assche 1993)

Also people with CP need to use all the opportunities that there are. It is part of good osteopathic treatment to manage the patient well; which might include referral to other treatment modalities like physio-, occupational- or speech therapy. Educating the patient or the patient’s parents about the basic mechanisms in which he/she develops and functions and about different treatment modalities is an essential part of good management. (Knox 2004/05)

8.1 Einwilligungserklärung

Hiermit bin ich einverstanden, dass mein Sohn Matthias, als Proband in dieser Einzelfallstudie teilnimmt. Ich wurde über den Ablauf der Studie und die dafür anfälligen Messungen ausreichend informiert und habe sie ordnungsgemäß durchgeführt.
Ich bin einverstanden, dass Matthias auf Bildern zu sehen ist und seine Krankengeschichte dargelegt wird.

8.2 Osteopathische Techniken, die in dieser Studie verwendet wurden

Ich habe folgende Techniken aus dem großen Repertoire der Osteopathie ausgewählt und sie je nach Verfassung meines Patienten adaptiert.

Strukturelle Techniken:

- TGO an der WS, obere und untere Extremitäten
- Weichteiltechniken der WS, Schultergürtel
- Mobilisation der Gelenke an oberer und untere Extremität, sowie des Schulterblattes und der Rippen
- Mitchell Techniken (MET) an Becken, beiden UE und linker OE

Craniosacrale Techniken:

- Direkte Techniken an SSB
- Intraossärer Release für Occiput, Temporale, Parietale, Sacrum
- Ausgleich Occiput – Sacrum
- Lösen erhöhter Duraspannung
- Midline erarbeiten
- Fasciale Ausgleichstechnik
- Balanced ligamentous Techniken (BLT)

Viszerale Techniken an der Lunge und am Diaphragma

8.3 Erklärung der verwendeten Techniken

Die TGO, bei uns auch AOB (allgemeine osteopathische Behandlung) ist ein durchscannen des gesamten Körpers. Die einzelnen Gelenke werden der Reihe nach rhythmisch durchbewegt.
Der Therapeut passt sich dem Patienten an und geht mit der Bewegung mit. Sie dient der Diagnose und geht fließend in die Behandlung über. Wichtig ist es, dabei den richtigen Rhythmus für den Patienten zu finden. Man betreibt palpatorische Anatomie während man Hand anlegt. (Ligner 2004
Diese Behandlung kann in BL, RL, SL und im Sitz durchgeführt werden. Es können auch nur einzelne Teile davon verwendet werden.

Weichteiltechniken wurden in der HWS und im Schultergürtel und Thoraxbereich in RL und in der SL durchgeführt. Für die LWS wurde auch in der BL gearbeitet. (Ligner 2003)

Für die Mobilisation des Zwerchfells wählte ich die RL und mobilisierte L3, da hier der Hauptansatz des Diaphragmas liegt. Die Dreiphasenatmung (Bauch, Brust, Sternum) wurde erarbeitet. (Ligner 2003)

Die Mitchell Technik, MET (Muskelenergietechnik) ist eine nicht traumatische Technik, deshalb auch bei Kindern gut anzuwenden, da sie toleriert wird. Man verwendet bei der MET isometrische und konzentrisch isotonische Muskelkontraktionen. Die Effekte sind sehr vielfältig:

- das Gleichgewicht zwischen Tonus und Kraft wird wieder hergestellt
- es kommt zum Stretchen der Fascien
- Dehnen anderer Bindegewebsstrukturen
- Bewegung der interstitiellen Flüssigkeit
- Lymphatische und venöse Drainage
- Einfluss auf den arteriellen Kreislauf (Marcer 2004)

Die Balanced Ligamentous Techniques werden bei Kindern gern angewendet. Sie wurden ursprünglich von Sutherland entwickelt. Er wollte so wenig Kraft wie nur möglich verwenden, um den Körper zu unterstützen, sich selbst zu helfen. Es soll der biomechanische Neuralpunkt in jeder Ebene (knöchern, membranös, Flüssigkeitskomponente) gefunden werden. Bei der BLT werden die Informationen in den Bändern (Ligamenten) verändert. (Carreiro 2007)

Fascientechniken gehen auf Rollin Becker zurück. Schon die Geburt hat einen Einfluss auf die ganze fasciale und damit die posturale Entwicklung. Das Fasciensystem hat eine durchgehende Kontinuität, die sich immer weiter fortsetzt. Die meisten Fascien setzen am Occiput an, das heißt, dass dieses auch den meisten Einfluss auf die Fascien hat. Das ganze System macht eine rhythmische Bewegung, bei der die Strukturen an der Mittellinie eine Flexion und Extension und die paarigen Strukturen eine AR und IR machen. Bei der Bewegung der SSB legt die anteriore Fascie bei der Flexion mehr Weg zurück als die posteriore Fascie. Da Ziel dieser Technik ist es, einen Spannungsausgleich in allen Ebenen (Membranen, Ligamenten, Fascien) zu erreichen. (Woodhead 2006)

Für die restlichen cranialen und viszeralen Techniken werden hier keine Erklärungen angeführt, da sie individuell an den Patienten angepasst werden.

8.4 Abkürzungen

AACP = American Academy for Cerebral Palsy

AOB = allgemeine osteopathische Behandlung

AR = Außenrotation

ATNR = Asymmetrisch tonischer Nackenreflex

BL = Bauchlage

BLT = Balanced Ligament Technique

BWS = Brustwirbelsäule

CP = Cerebralparese

GMFCS = Gross Motor Function Classification System

GMFM = Gross Motor Function Measure

GOT = englische Abkürzung für AOB

HWS = Halswirbelsäule

ICP = infantile Cerebralparese

IR = Innenrotation

KThR = Kuratorium für therapeutisches Reiten

Lkf = left knee floor

LWS = Lendenwirbelsäule

MET = Muskelenergietechnik

Nt = no treatment

OE = obere Extremität

OMG = Ontario Motor Growth

RL = Rückenlage

rkf = right knee floor

SCPE = Surveillance of Cerebral Palsy in Europe

SSB = Synchondrosis sphenobasilaris

SNR = Symmetrischer Nackenreflex

SL = Seilage

St = fist floor

TGO = französische Abkürzung für AOB

TLR = Tonischer Labyrinthreflex

UE = untere Extremität

WS = Wirbelsäule

ZNS = Zentralnervensystem

8.5 Begriffsverzeichnis

Asphyxie: Atemstillstand infolge Atemlähmung oder Atemwegsverlegung, Erstickungszustand des Feten oder Neugeborenen als Folge des Absinkens des Sauerstoffgehalts bzw. der Kohlendioxid Anreicherung im Fetalblut.

assoziiert: verknüpft, verbunden, gemeinsam auftretend mit.

ataktisch: (gr. atakos - ungeordnet), unregelmäßig, zittrig

Cerebralparese/Cerebral palsy: Muskelhypotonie u. dadurch bedingte Muskelschwäche bei perinatalen Großhirn-, Kleinhirn- u. Hirnstamm Schäden

Dysmetrie: (gr. metron – Maß), unbeherrschte, überschießende oder zu kurz bemessene Bewegungen

Dyskinesie: (gr. Kinesis – Bewegung), motorische Fehlfunktion

Floppy infant : (eng. T**o be** floppy – schlaff**;** infant – Kind), Verminderung des kontraktilen, v.a. reflektorischen Muskeltonus, kongenital oder in frühester Kindheit

Grand-mal: primär generalisierte Anfälle bei Epilepsie

Gyrus praecentralis: Windung vor der Zentralfurche des Gehirns

Homonyme Hemianopsie: (gr. onoma – Name, Benennung), auf beiden Augen die linke oder die rechte Hälfte betreffende Sehstörung; Halbseitenblindheit

Hypotonie: (gr. tonos – Spannung), Tonuserniedrigung

Hyperkapnie: (gr. kapnos – Dunst, Gas) Erhöhung der CO Spannung im arteriellen Blut

Hypermetrie: Bewegungsübermaß bei Zielbewegungen

Hypoxie: Sauerstoffmangel, Sauerstoffnot

Intensionstremor: (lat. tremor – zittern) bei Bewegungen auftretende, weitgehend **rhythmisch** aufeinanderfolgende Kontraktionen antagonistisch wirkender Muskeln

Ischämie: Unterbrechung oder spürbare Verringerung der Durchblutung

isometrisch: Bez. Für Spannungsänderung des Muskels bei gleichbleibender Länge

isotonisch konzentrisch: Aktive Verkürzung des Muskels

Myelomeningocele: Bruchsack mit Vorfall der Meningen und des Rückenmarks

Nystagmus: Augenzittern, unwillkürlich, **rhythmisch**, schnell aufeinanderfolgende Zuckungen der Augäpfel

Obstipation: (lat. stipare – stopfen) Stuhlverstopfung, verzögerte Kotentleerung

Strabismus convergens: (gr. strabizein – schielen) Konvergenzschielen, Einwärtsschielen

Spina bifida: (lat. bifidus – zweigeteilt), angeborene Spaltbildung der Wirbelsäule, meist an der hinteren Seite des Wirbelbogen d. **Lumbal-** oder Sakralteils

zerebellar/zerebellare Symptome: Störungen der Koordination der Bewegungen/Sprache

8.6 List of Plates

8.7 List of Tables

8.8 List of Charts and Graphs

8.9 Appendix 1a

Results
ANOVA
Right Knee-Floor Distance

RKF - Univariate Analysis of Variance

Between-Subjects Factors

		N
Sequ	HT1	34
	HT2	33
	HTOT	35
	nT	2

Descriptive Statistics

Dependent Variable: rkf

Sequ	Mean	Std. Deviation	N
HT1	12,74	,448	34
HT2	12,73	,452	33
HTOT	10,46	,950	35
nT	13,00	,000	2
Total	11,97	1,265	104

Levene's Test of Equality of Error Variances(a)

Dependent Variable: rkf

F	df1	df2	Sig.
14,871	3	100	,000

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.
a Design: Intercept+Sequ

Tests of Between-Subjects Effects

Dependent Variable: rkf

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power(a)
Corrected Model	121,065(b)	3	40,355	92,032	,000	,734	276,096	1,000
Intercept	4067,982	1	4067,982	9277,291	,000	,989	9277,291	1,000
Sequ	121,065	3	40,355	92,032	,000	,734	276,096	1,000
Error	43,849	100	,438					
Total	15069,000	104						
Corrected Total	164,913	103						

a Computed using alpha = ,05

b R Squared = ,734 (Adjusted R Squared = ,726)

Parameter Estimates

Dependent Variable: rkf

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared	Noncent. Parameter	Observed Power(a)
					Lower Bound	Upper Bound			
Intercept	13,000	,468	27,764	,000	12,071	13,929	,885	27,764	1,000
[Sequ=HT1]	-,265	,482	-,549	,584	-1,221	,691	,003	,549	,085
[Sequ=HT2]	-,273	,482	-,566	,573	-1,229	,684	,003	,566	,087
[Sequ=HTOT]	-2,543	,481	-5,282	,000	-3,498	-1,588	,218	5,282	,999
[Sequ=nT]	0(b)	.	.	.	.	.	.	.	.

a Computed using alpha = ,05

b This parameter is set to zero because it is redundant.

Post Hoc Tests **(Sequ)**

Multiple Comparisons

Dependent Variable: rkf

	(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
Tukey HSD	HT1	HT2	,01	,162	1,000	-,41	,43
		HTOT	2,28(*)	,159	,000	1,86	2,69
		nT	-,26	,482	,947	-1,52	,99
	HT2	HT1	-,01	,162	1,000	-,43	,41
		HTOT	2,27(*)	,161	,000	1,85	2,69
		nT	-,27	,482	,942	-1,53	,99
	HTOT	HT1	-2,28(*)	,159	,000	-2,69	-1,86
		HT2	-2,27(*)	,161	,000	-2,69	-1,85
		nT	-2,54(*)	,481	,000	-3,80	-1,29
	nT	HT1	,26	,482	,947	-,99	1,52
		HT2	,27	,482	,942	-,99	1,53
		HTOT	2,54(*)	,481	,000	1,29	3,80
LSD	HT1	HT2	,01	,162	,961	-,31	,33
		HTOT	2,28(*)	,159	,000	1,96	2,59
		nT	-,26	,482	,584	-1,22	,69
	HT2	HT1	-,01	,162	,961	-,33	,31
		HTOT	2,27(*)	,161	,000	1,95	2,59
		nT	-,27	,482	,573	-1,23	,68
	HTOT	HT1	-2,28(*)	,159	,000	-2,59	-1,96
		HT2	-2,27(*)	,161	,000	-2,59	-1,95
		nT	-2,54(*)	,481	,000	-3,50	-1,59
	nT	HT1	,26	,482	,584	-,69	1,22
		HT2	,27	,482	,573	-,68	1,23
		HTOT	2,54(*)	,481	,000	1,59	3,50
Dunnett T3	HT1	HT2	,01	,110	1,000	-,29	,31
		HTOT	2,28(*)	,178	,000	1,79	2,77
		nT	-,26(*)	,077	,009	-,48	-,05
	HT2	HT1	-,01	,110	1,000	-,31	,29
		HTOT	2,27(*)	,179	,000	1,78	2,76
		nT	-,27(*)	,079	,009	-,49	-,05
	HTOT	HT1	-2,28(*)	,178	,000	-2,77	-1,79
		HT2	-2,27(*)	,179	,000	-2,76	-1,78
		nT	-2,54(*)	,161	,000	-2,99	-2,10
	nT	HT1	,26(*)	,077	,009	,05	,48
		HT2	,27(*)	,079	,009	,05	,49
		HTOT	2,54(*)	,161	,000	2,10	2,99

Based on observed means. * The mean difference is significant at the ,05 level.

Homogeneous Subsets

rkf

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b,c)	HTOT	35	10,46	
	HT2	33		12,73
	HT1	34		12,74
	nT	2		13,00
	Sig.		1,000	,872

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,438.
a Uses Harmonic Mean Sample Size = 6,799.
b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.
c Alpha = ,05.

RKF/Days after therapy - Univariate Analysis of Variance

Between-Subjects Factors

Day after therapy			N
.	Sequ	nT	2
0	Sequ	HT1	5
		HT2	5
		HTOT	5
1	Sequ	HT1	5
		HT2	5
		HTOT	5
2	Sequ	HT1	5
		HT2	5
		HTOT	5
3	Sequ	HT1	5
		HT2	5
		HTOT	5
4	Sequ	HT1	4
		HT2	5
		HTOT	5
5	Sequ	HT1	5
		HT2	4
		HTOT	5
6	Sequ	HT1	5
		HT2	4
		HTOT	5

Descriptive Statistics

Dependent Variable: rkf

Day after therapy	Sequ	Mean	Std. Deviation	N
.	nT	13,00	,000	2
	Total	13,00	,000	2
0	HT1	12,00	,000	5
	HT2	12,20	,447	5
	HTOT	9,00	,000	5
	Total	11,07	1,534	15
1	HT1	12,20	,447	5
	HT2	12,60	,548	5
	HTOT	9,80	,447	5
	Total	11,53	1,356	15
2	HT1	13,00	,000	5
	HT2	12,60	,548	5
	HTOT	10,20	,447	5
	Total	11,93	1,335	15
3	HT1	13,00	,000	5
	HT2	12,80	,447	5
	HTOT	10,80	,837	5
	Total	12,20	1,146	15
4	HT1	13,00	,000	4
	HT2	13,00	,000	5
	HTOT	10,80	,837	5
	Total	12,21	1,188	14
5	HT1	13,00	,000	5
	HT2	13,00	,000	4
	HTOT	11,20	,447	5
	Total	12,36	,929	14
6	HT1	13,00	,000	5
	HT2	13,00	,000	4
	HTOT	11,40	,548	5
	Total	12,43	,852	14

Levene's Test of Equality of Error Variances(a,b)

Dependent Variable: rkf

Day after therapy	F	df1	df2	Sig.
0	7,111	2	12	,009
1	,821	2	12	,463
2	10,667	2	12	,002
3	5,908	2	12	,016
4	9,629	2	11	,004
5	6,286	2	11	,015
6	84,857	2	11	,000

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a Test is not computed for one or more split files because there are less than two nonempty cells.

b Design: Intercept+Sequ

Tests of Between-Subjects Effects

Dependent Variable: rkf

Day after therapy	Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power(a)
.	Corrected Model	,000(b)	0	.	.	.	.	.	.
	Intercept	338,000	1	338,000	.	.	1,000	.	.
	Sequ	,000	0	.	.	.	.	.	.
	Error	,000	1	,000					
	Total	338,000	2						
	Corrected Total	,000	1						
0	Corrected Model	32,133(c)	2	16,067	241,000	,000	,976	482,000	1,000
	Intercept	1837,067	1	1837,067	27556,000	,000	1,000	27556,000	1,000
	Sequ	32,133	2	16,067	241,000	,000	,976	482,000	1,000
	Error	,800	12	,067					
	Total	1870,000	15						
	Corrected Total	32,933	14						
1	Corrected Model	22,933(d)	2	11,467	49,143	,000	,891	98,286	1,000
	Intercept	1995,267	1	1995,267	8551,143	,000	,999	8551,143	1,000
	Sequ	22,933	2	11,467	49,143	,000	,891	98,286	1,000
	Error	2,800	12	,233					
	Total	2021,000	15						
	Corrected Total	25,733	14						
2	Corrected Model	22,933(e)	2	11,467	68,800	,000	,920	137,600	1,000
	Intercept	2136,067	1	2136,067	12816,400	,000	,999	12816,400	1,000
	Sequ	22,933	2	11,467	68,800	,000	,920	137,600	1,000
	Error	2,000	12	,167					
	Total	2161,000	15						
	Corrected Total	24,933	14						

Day after therapy	Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power(a)
3	Corrected Model	14,800(f)	2	7,400	24,667	,000	,804	49,333	1,000
	Intercept	2232,600	1	2232,600	7442,000	,000	,998	7442,000	1,000
	Sequ	14,800	2	7,400	24,667	,000	,804	49,333	1,000
	Error	3,600	12	,300					
	Total	2251,000	15						
	Corrected Total	18,400	14						
4	Corrected Model	15,557(g)	2	7,779	30,559	,000	,847	61,117	1,000
	Intercept	2083,446	1	2083,446	8184,967	,000	,999	8184,967	1,000
	Sequ	15,557	2	7,779	30,559	,000	,847	61,117	1,000
	Error	2,800	11	,255					
	Total	2107,000	14						
	Corrected Total	18,357	13						
5	Corrected Model	10,414(h)	2	5,207	71,598	,000	,929	143,196	1,000
	Intercept	2128,985	1	2128,985	29273,538	,000	1,000	29273,538	1,000
	Sequ	10,414	2	5,207	71,598	,000	,929	143,196	1,000
	Error	,800	11	,073					
	Total	2149,000	14						
	Corrected Total	11,214	13						
6	Corrected Model	8,229(i)	2	4,114	37,714	,000	,873	75,429	1,000
	Intercept	2151,938	1	2151,938	19726,103	,000	,999	19726,103	1,000
	Sequ	8,229	2	4,114	37,714	,000	,873	75,429	1,000
	Error	1,200	11	,109					
	Total	2172,000	14						
	Corrected Total	9,429	13						

a Computed using alpha = ,05

b R Squared = . (Adjusted R Squared = .)

c R Squared = ,976 (Adjusted R Squared = ,972)

d R Squared = ,891 (Adjusted R Squared = ,873)

e R Squared = ,920 (Adjusted R Squared = ,906)

f R Squared = ,804 (Adjusted R Squared = ,772)

g R Squared = ,847 (Adjusted R Squared = ,820)

h R Squared = ,929 (Adjusted R Squared = ,916)

i R Squared = ,873 (Adjusted R Squared = ,850)

Parameter Estimates

Dependent Variable: rkf

Day after therapy	Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared	Noncent. Parameter	Observed Power(a)
						Lower Bound	Upper Bound			
.	Intercept	13,000	,000	.	.	13,000	13,000	1,000	.	.
	[Sequ=nT]	0(b)	.	.	.	.	.	.	.	.
0	Intercept	9,000	,115	77,942	,000	8,748	9,252	,998	77,942	1,000
	[Sequ=HT1]	3,000	,163	18,371	,000	2,644	3,356	,966	18,371	1,000
	[Sequ=HT2]	3,200	,163	19,596	,000	2,844	3,556	,970	19,596	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
1	Intercept	9,800	,216	45,365	,000	9,329	10,271	,994	45,365	1,000
	[Sequ=HT1]	2,400	,306	7,856	,000	1,734	3,066	,837	7,856	1,000
	[Sequ=HT2]	2,800	,306	9,165	,000	2,134	3,466	,875	9,165	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
2	Intercept	10,200	,183	55,868	,000	9,802	10,598	,996	55,868	1,000
	[Sequ=HT1]	2,800	,258	10,844	,000	2,237	3,363	,907	10,844	1,000
	[Sequ=HT2]	2,400	,258	9,295	,000	1,837	2,963	,878	9,295	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
3	Intercept	10,800	,245	44,091	,000	10,266	11,334	,994	44,091	1,000
	[Sequ=HT1]	2,200	,346	6,351	,000	1,445	2,955	,771	6,351	1,000
	[Sequ=HT2]	2,000	,346	5,774	,000	1,245	2,755	,735	5,774	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
4	Intercept	10,800	,226	47,866	,000	10,303	11,297	,995	47,866	1,000
	[Sequ=HT1]	2,200	,338	6,500	,000	1,455	2,945	,793	6,500	1,000
	[Sequ=HT2]	2,200	,319	6,895	,000	1,498	2,902	,812	6,895	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
5	Intercept	11,200	,121	92,865	,000	10,935	11,465	,999	92,865	1,000
	[Sequ=HT1]	1,800	,171	10,553	,000	1,425	2,175	,910	10,553	1,000
	[Sequ=HT2]	1,800	,181	9,950	,000	1,402	2,198	,900	9,950	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
6	Intercept	11,400	,148	77,178	,000	11,075	11,725	,998	77,178	1,000
	[Sequ=HT1]	1,600	,209	7,659	,000	1,140	2,060	,842	7,659	1,000
	[Sequ=HT2]	1,600	,222	7,221	,000	1,112	2,088	,826	7,221	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.

a Computed using alpha = ,05

b This parameter is set to zero because it is redundant.

Post Hoc Tests **(Sequ)**

Multiple Comparisons

Dependent Variable: rkf

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
							Lower Bound	Upper Bound
0	Tukey HSD	HT1	HT2	-,20	,163	,462	-,64	,24
			HTOT	3,00(*)	,163	,000	2,56	3,44
		HT2	HT1	,20	,163	,462	-,24	,64
			HTOT	3,20(*)	,163	,000	2,76	3,64
		HTOT	HT1	-3,00(*)	,163	,000	-3,44	-2,56
			HT2	-3,20(*)	,163	,000	-3,64	-2,76
	LSD	HT1	HT2	-,20	,163	,244	-,56	,16
			HTOT	3,00(*)	,163	,000	2,64	3,36
		HT2	HT1	,20	,163	,244	-,16	,56
			HTOT	3,20(*)	,163	,000	2,84	3,56
		HTOT	HT1	-3,00(*)	,163	,000	-3,36	-2,64
			HT2	-3,20(*)	,163	,000	-3,56	-2,84
	Dunnett T3	HT1	HT2	-,20	,200	,704	-,95	,55
			HTOT	3,00	,000	.	3,00	3,00
		HT2	HT1	,20	,200	,704	-,55	,95
			HTOT	3,20(*)	,200	,000	2,45	3,95
		HTOT	HT1	-3,00	,000	.	-3,00	-3,00
			HT2	-3,20(*)	,200	,000	-3,95	-2,45
1	Tukey HSD	HT1	HT2	-,40	,306	,417	-1,22	,42
			HTOT	2,40(*)	,306	,000	1,58	3,22
		HT2	HT1	,40	,306	,417	-,42	1,22
			HTOT	2,80(*)	,306	,000	1,98	3,62
		HTOT	HT1	-2,40(*)	,306	,000	-3,22	-1,58
			HT2	-2,80(*)	,306	,000	-3,62	-1,98
	LSD	HT1	HT2	-,40	,306	,215	-1,07	,27
			HTOT	2,40(*)	,306	,000	1,73	3,07
		HT2	HT1	,40	,306	,215	-,27	1,07
			HTOT	2,80(*)	,306	,000	2,13	3,47
		HTOT	HT1	-2,40(*)	,306	,000	-3,07	-1,73
			HT2	-2,80(*)	,306	,000	-3,47	-2,13

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
1	Dunnett T3	HT1	HT2	-,40	,316	,535	-1,34	,54
			HTOT	2,40(*)	,283	,000	1,56	3,24
		HT2	HT1	,40	,316	,535	-,54	1,34
			HTOT	2,80(*)	,316	,000	1,86	3,74
		HTOT	HT1	-2,40(*)	,283	,000	-3,24	-1,56
			HT2	-2,80(*)	,316	,000	-3,74	-1,86
2	Tukey HSD	HT1	HT2	,40	,258	,304	-,29	1,09
			HTOT	2,80(*)	,258	,000	2,11	3,49
		HT2	HT1	-,40	,258	,304	-1,09	,29
			HTOT	2,40(*)	,258	,000	1,71	3,09
		HTOT	HT1	-2,80(*)	,258	,000	-3,49	-2,11
			HT2	-2,40(*)	,258	,000	-3,09	-1,71
	LSD	HT1	HT2	,40	,258	,147	-,16	,96
			HTOT	2,80(*)	,258	,000	2,24	3,36
		HT2	HT1	-,40	,258	,147	-,96	,16
			HTOT	2,40(*)	,258	,000	1,84	2,96
		HTOT	HT1	-2,80(*)	,258	,000	-3,36	-2,24
			HT2	-2,40(*)	,258	,000	-2,96	-1,84
	Dunnett T3	HT1	HT2	,40	,245	,391	-,52	1,32
			HTOT	2,80(*)	,200	,000	2,05	3,55
		HT2	HT1	-,40	,245	,391	-1,32	,52
			HTOT	2,40(*)	,316	,000	1,46	3,34
		HTOT	HT1	-2,80(*)	,200	,000	-3,55	-2,05
			HT2	-2,40(*)	,316	,000	-3,34	-1,46
3	Tukey HSD	HT1	HT2	,20	,346	,835	-,72	1,12
			HTOT	2,20(*)	,346	,000	1,28	3,12
		HT2	HT1	-,20	,346	,835	-1,12	,72
			HTOT	2,00(*)	,346	,000	1,08	2,92
		HTOT	HT1	-2,20(*)	,346	,000	-3,12	-1,28
			HT2	-2,00(*)	,346	,000	-2,92	-1,08

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
3	LSD	HT1	HT2	,20	,346	,574	-,55	,95
			HTOT	2,20(*)	,346	,000	1,45	2,95
		HT2	HT1	-,20	,346	,574	-,95	,55
			HTOT	2,00(*)	,346	,000	1,25	2,75
		HTOT	HT1	-2,20(*)	,346	,000	-2,95	-1,45
			HT2	-2,00(*)	,346	,000	-2,75	-1,25
	Dunnett T3	HT1	HT2	,20	,200	,704	-,55	,95
			HTOT	2,20(*)	,374	,011	,80	3,60
		HT2	HT1	-,20	,200	,704	-,95	,55
			HTOT	2,00(*)	,424	,009	,65	3,35
		HTOT	HT1	-2,20(*)	,374	,011	-3,60	-,80
			HT2	-2,00(*)	,424	,009	-3,35	-,65
4	Tukey HSD	HT1	HT2	,00	,338	1,000	-,91	,91
			HTOT	2,20(*)	,338	,000	1,29	3,11
		HT2	HT1	,00	,338	1,000	-,91	,91
			HTOT	2,20(*)	,319	,000	1,34	3,06
		HTOT	HT1	-2,20(*)	,338	,000	-3,11	-1,29
			HT2	-2,20(*)	,319	,000	-3,06	-1,34
	LSD	HT1	HT2	,00	,338	1,000	-,74	,74
			HTOT	2,20(*)	,338	,000	1,46	2,94
		HT2	HT1	,00	,338	1,000	-,74	,74
			HTOT	2,20(*)	,319	,000	1,50	2,90
		HTOT	HT1	-2,20(*)	,338	,000	-2,94	-1,46
			HT2	-2,20(*)	,319	,000	-2,90	-1,50
	Dunnett T3	HT1	HT2	,00	,000	.	,00	,00
			HTOT	2,20(*)	,374	,011	,80	3,60
		HT2	HT1	,00	,000	.	,00	,00
			HTOT	2,20(*)	,374	,011	,80	3,60
		HTOT	HT1	-2,20(*)	,374	,011	-3,60	-,80
			HT2	-2,20(*)	,374	,011	-3,60	-,80

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
5	Tukey HSD	HT1	HT2	,00	,181	1,000	-,49	,49
			HTOT	1,80(*)	,171	,000	1,34	2,26
		HT2	HT1	,00	,181	1,000	-,49	,49
			HTOT	1,80(*)	,181	,000	1,31	2,29
		HTOT	HT1	-1,80(*)	,171	,000	-2,26	-1,34
			HT2	-1,80(*)	,181	,000	-2,29	-1,31
	LSD	HT1	HT2	,00	,181	1,000	-,40	,40
			HTOT	1,80(*)	,171	,000	1,42	2,18
		HT2	HT1	,00	,181	1,000	-,40	,40
			HTOT	1,80(*)	,181	,000	1,40	2,20
		HTOT	HT1	-1,80(*)	,171	,000	-2,18	-1,42
			HT2	-1,80(*)	,181	,000	-2,20	-1,40
	Dunnett T3	HT1	HT2	,00	,000	.	,00	,00
			HTOT	1,80(*)	,200	,002	1,05	2,55
		HT2	HT1	,00	,000	.	,00	,00
			HTOT	1,80(*)	,200	,002	1,05	2,55
		HTOT	HT1	-1,80(*)	,200	,002	-2,55	-1,05
			HT2	-1,80(*)	,200	,002	-2,55	-1,05
6	Tukey HSD	HT1	HT2	,00	,222	1,000	-,60	,60
			HTOT	1,60(*)	,209	,000	1,04	2,16
		HT2	HT1	,00	,222	1,000	-,60	,60
			HTOT	1,60(*)	,222	,000	1,00	2,20
		HTOT	HT1	-1,60(*)	,209	,000	-2,16	-1,04
			HT2	-1,60(*)	,222	,000	-2,20	-1,00
	LSD	HT1	HT2	,00	,222	1,000	-,49	,49
			HTOT	1,60(*)	,209	,000	1,14	2,06
		HT2	HT1	,00	,222	1,000	-,49	,49
			HTOT	1,60(*)	,222	,000	1,11	2,09
		HTOT	HT1	-1,60(*)	,209	,000	-2,06	-1,14
			HT2	-1,60(*)	,222	,000	-2,09	-1,11

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
	Dunnett T3	HT1	HT2	,00	,000	.	,00	,00
			HTOT	1,60(*)	,245	,007	,68	2,52
		HT2	HT1	,00	,000	.	,00	,00
			HTOT	1,60(*)	,245	,007	,68	2,52
		HTOT	HT1	-1,60(*)	,245	,007	-2,52	-,68
			HT2	-1,60(*)	,245	,007	-2,52	-,68

Based on observed means.

* The mean difference is significant at the ,05 level.

Homogeneous Subsets

rkf

Day after therapy=0

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b)	HTOT	5	9,00	
	HT1	5		12,00
	HT2	5		12,20
	Sig.		1,000	,462

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,067.

a Uses Harmonic Mean Sample Size = 5,000.

b Alpha = ,05.

Day after therapy=1

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b)	HTOT	5	9,80	
	HT1	5		12,20
	HT2	5		12,60
	Sig.		1,000	,417

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,233.
a Uses Harmonic Mean Sample Size = 5,000.
b Alpha = ,05.

Day after therapy=2

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b)	HTOT	5	10,20	
	HT2	5		12,60
	HT1	5		13,00
	Sig.		1,000	,304

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,167.
a Uses Harmonic Mean Sample Size = 5,000.
b Alpha = ,05.

Day after therapy=3

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b)	HTOT	5	10,80	
	HT2	5		12,80
	HT1	5		13,00
	Sig.		1,000	,835

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,300.
a Uses Harmonic Mean Sample Size = 5,000.
b Alpha = ,05.

Day after therapy=4

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b,c)	HTOT	5	10,80	
	HT1	4		13,00
	HT2	5		13,00
	Sig.		1,000	1,000

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,255. a Uses Harmonic Mean Sample Size = 4,615.
b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

c Alpha = ,05.

Day after therapy=5

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b,c)	HTOT	5	11,20	
	HT1	5		13,00
	HT2	4		13,00
	Sig.		1,000	1,000

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,073.
a Uses Harmonic Mean Sample Size = 4,615.
b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.
c Alpha = ,05.

Day after therapy=6

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b,c)	HTOT	5	11,40	
	HT1	5		13,00
	HT2	4		13,00
	Sig.		1,000	1,000

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,109.
a Uses Harmonic Mean Sample Size = 4,615.
b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.
c Alpha = ,05.

LKF - Univariate Analysis of Variance

Between-Subjects Factors

		N
Sequ	HT1	34
	HT2	33
	HTOT	35
	nT	2

Descriptive Statistics

Dependent Variable: lkf

Sequ	Mean	Std. Deviation	N
HT1	21,56	,705	34
HT2	21,79	,485	33
HTOT	20,17	,822	35
nT	22,00	,000	2
Total	21,17	,990	104

Levene's Test of Equality of Error Variances(a)

Dependent Variable: lkf

F	df1	df2	Sig.
4,123	3	100	,008

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.
a Design: Intercept+Sequ

Tests of Between-Subjects Effects

Dependent Variable: lkf

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power(a)
Corrected Model	54,016(b)	3	18,005	38,416	,000	,535	115,248	1,000
Intercept	12431,620	1	12431,620	26524,222	,000	,996	26524,222	1,000
Sequ	54,016	3	18,005	38,416	,000	,535	115,248	1,000
Error	46,869	100	,469					
Total	46724,000	104						
Corrected Total	100,885	103						

a Computed using alpha = ,05

b R Squared = ,535 (Adjusted R Squared = ,521)

Parameter Estimates

Dependent Variable: lkf

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared	Noncent. Parameter	Observed Power(a)
					Lower Bound	Upper Bound			
Intercept	22,000	,484	45,446	,000	21,040	22,960	,954	45,446	1,000
[Sequ=HT1]	-,441	,498	-,886	,378	-1,429	,547	,008	,886	,142
[Sequ=HT2]	-,212	,499	-,425	,671	-1,201	,777	,002	,425	,071
[Sequ=HTOT]	-1,829	,498	-3,674	,000	-2,816	-,841	,119	3,674	,953
[Sequ=nT]	0(b)	.	.	.	.	.	.	.	.

a Computed using alpha = ,05

b This parameter is set to zero because it is redundant.

Post Hoc Tests **(Sequ)**

Multiple Comparisons

Dependent Variable: lkf

	(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
Tukey HSD	HT1	HT2	-,23	,167	,522	-,67	,21
		HTOT	1,39(*)	,165	,000	,96	1,82
		nT	-,44	,498	,812	-1,74	,86
	HT2	HT1	,23	,167	,522	-,21	,67
		HTOT	1,62(*)	,166	,000	1,18	2,05
		nT	-,21	,499	,974	-1,51	1,09
	HTOT	HT1	-1,39(*)	,165	,000	-1,82	-,96
		HT2	-1,62(*)	,166	,000	-2,05	-1,18
		nT	-1,83(*)	,498	,002	-3,13	-,53
	nT	HT1	,44	,498	,812	-,86	1,74
		HT2	,21	,499	,974	-1,09	1,51
		HTOT	1,83(*)	,498	,002	,53	3,13
LSD	HT1	HT2	-,23	,167	,174	-,56	,10
		HTOT	1,39(*)	,165	,000	1,06	1,71
		nT	-,44	,498	,378	-1,43	,55
	HT2	HT1	,23	,167	,174	-,10	,56
		HTOT	1,62(*)	,166	,000	1,29	1,95
		nT	-,21	,499	,671	-1,20	,78
	HTOT	HT1	-1,39(*)	,165	,000	-1,71	-1,06
		HT2	-1,62(*)	,166	,000	-1,95	-1,29
		nT	-1,83(*)	,498	,000	-2,82	-,84
	nT	HT1	,44	,498	,378	-,55	1,43
		HT2	,21	,499	,671	-,78	1,20
		HTOT	1,83(*)	,498	,000	,84	2,82
Dunnett T3	HT1	HT2	-,23	,147	,542	-,63	,17
		HTOT	1,39(*)	,184	,000	,89	1,89
		nT	-,44(*)	,121	,005	-,78	-,10
	HT2	HT1	,23	,147	,542	-,17	,63
		HTOT	1,62(*)	,163	,000	1,17	2,06
		nT	-,21	,084	,096	-,45	,02
	HTOT	HT1	-1,39(*)	,184	,000	-1,89	-,89
		HT2	-1,62(*)	,163	,000	-2,06	-1,17
		nT	-1,83(*)	,139	,000	-2,22	-1,44
	nT	HT1	,44(*)	,121	,005	,10	,78
		HT2	,21	,084	,096	-,02	,45
		HTOT	1,83(*)	,139	,000	1,44	2,22

Based on observed means. * The mean difference is significant at the ,05 level.

Homogeneous Subsets

lkf

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b,c)	HTOT	35	20,17	
	HT1	34		21,56
	HT2	33		21,79
	nT	2		22,00
	Sig.		1,000	,636

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,469.

a Uses Harmonic Mean Sample Size = 6,799.

b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

c Alpha = ,05.

Appendix 1b

Results
ANOVA
Left Knee-Floor Distance

LKF/Days after therapy - Univariate Analysis of Variance

Between-Subjects Factors

Day after therapy			N
.	Sequ	nT	2
0	Sequ	HT1	5
		HT2	5
		HTOT	5
1	Sequ	HT1	5
		HT2	5
		HTOT	5
2	Sequ	HT1	5
		HT2	5
		HTOT	5
3	Sequ	HT1	5
		HT2	5
		HTOT	5
4	Sequ	HT1	4
		HT2	5
		HTOT	5
5	Sequ	HT1	5
		HT2	4
		HTOT	5
6	Sequ	HT1	5
		HT2	4
		HTOT	5

Descriptive Statistics

Dependent Variable: lkf

Day after therapy	Sequ	Mean	Std. Deviation	N
.	nT	22,00	,000	2
	Total	22,00	,000	2
0	HT1	20,40	,548	5
	HT2	21,00	,707	5
	HTOT	19,00	,707	5
	Total	20,13	1,060	15
1	HT1	20,80	,447	5
	HT2	21,60	,548	5
	HTOT	20,00	,707	5
	Total	20,80	,862	15
2	HT1	21,80	,447	5
	HT2	22,00	,000	5
	HTOT	20,00	,707	5
	Total	21,27	1,033	15
3	HT1	22,00	,000	5
	HT2	22,00	,000	5
	HTOT	20,40	,548	5
	Total	21,47	,834	15
4	HT1	22,00	,000	4
	HT2	22,00	,000	5
	HTOT	20,40	,548	5
	Total	21,43	,852	14
5	HT1	22,00	,000	5
	HT2	22,00	,000	4
	HTOT	20,40	,548	5
	Total	21,43	,852	14
6	HT1	22,00	,000	5
	HT2	22,00	,000	4
	HTOT	21,00	,707	5
	Total	21,64	,633	14

Levene's Test of Equality of Error Variances(a,b)

Dependent Variable: lkf

Day after therapy	F	df1	df2	Sig.
0	,052	2	12	,949
1	,250	2	12	,783
2	1,806	2	12	,206
3	96,000	2	12	,000
4	84,857	2	11	,000
5	84,857	2	11	,000
6	2,357	2	11	,141

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a Test is not computed for one or more split files because there are less than two nonempty cells.

b Design: Intercept+Sequ

Tests of Between-Subjects Effects

Dependent Variable: lkf

Day after therapy	Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power(a)
.	Corrected Model	,000(b)	0	.	.	.	.	.	.
	Intercept	968,000	1	968,000	.	.	1,000	.	.
	Sequ	,000	0	.	.	.	.	.	.
	Error	,000	1	,000					
	Total	968,000	2						
	Corrected Total	,000	1						
0	Corrected Model	10,533(c)	2	5,267	12,154	,001	,669	24,308	,978
	Intercept	6080,267	1	6080,267	14031,385	,000	,999	14031,385	1,000
	Sequ	10,533	2	5,267	12,154	,001	,669	24,308	,978
	Error	5,200	12	,433					
	Total	6096,000	15						
	Corrected Total	15,733	14						
1	Corrected Model	6,400(d)	2	3,200	9,600	,003	,615	19,200	,940
	Intercept	6489,600	1	6489,600	19468,800	,000	,999	19468,800	1,000
	Sequ	6,400	2	3,200	9,600	,003	,615	19,200	,940
	Error	4,000	12	,333					
	Total	6500,000	15						
	Corrected Total	10,400	14						
2	Corrected Model	12,133(e)	2	6,067	26,000	,000	,813	52,000	1,000
	Intercept	6784,067	1	6784,067	29074,571	,000	1,000	29074,571	1,000
	Sequ	12,133	2	6,067	26,000	,000	,813	52,000	1,000
	Error	2,800	12	,233					
	Total	6799,000	15						
	Corrected Total	14,933	14						
3	Corrected Model	8,533(f)	2	4,267	42,667	,000	,877	85,333	1,000
	Intercept	6912,267	1	6912,267	69122,667	,000	1,000	69122,667	1,000
	Sequ	8,533	2	4,267	42,667	,000	,877	85,333	1,000
	Error	1,200	12	,100					
	Total	6922,000	15						
	Corrected Total	9,733	14						

Day after therapy	Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power(a)
4	Corrected Model	8,229(g)	2	4,114	37,714	,000	,873	75,429	1,000
	Intercept	6380,554	1	6380,554	58488,410	,000	1,000	58488,410	1,000
	Sequ	8,229	2	4,114	37,714	,000	,873	75,429	1,000
	Error	1,200	11	,109					
	Total	6438,000	14						
	Corrected Total	9,429	13						
5	Corrected Model	8,229(g)	2	4,114	37,714	,000	,873	75,429	1,000
	Intercept	6380,554	1	6380,554	58488,410	,000	1,000	58488,410	1,000
	Sequ	8,229	2	4,114	37,714	,000	,873	75,429	1,000
	Error	1,200	11	,109					
	Total	6438,000	14						
	Corrected Total	9,429	13						
6	Corrected Model	3,214(h)	2	1,607	8,839	,005	,616	17,679	,913
	Intercept	6500,000	1	6500,000	35750,000	,000	1,000	35750,000	1,000
	Sequ	3,214	2	1,607	8,839	,005	,616	17,679	,913
	Error	2,000	11	,182					
	Total	6563,000	14						
	Corrected Total	5,214	13						

a Computed using alpha = ,05
b R Squared = . (Adjusted R Squared = .)
c R Squared = ,669 (Adjusted R Squared = ,614)
d R Squared = ,615 (Adjusted R Squared = ,551)
e R Squared = ,813 (Adjusted R Squared = ,781)
f R Squared = ,877 (Adjusted R Squared = ,856)
g R Squared = ,873 (Adjusted R Squared = ,850)
h R Squared = ,616 (Adjusted R Squared = ,547)

Parameter Estimates

Dependent Variable: lkf

Day after therapy	Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared	Noncent. Parameter	Observed Power(a)
						Lower Bound	Upper Bound			
.	Intercept	22,000	,000	.	.	22,000	22,000	1,000	.	.
	[Sequ=nT]	0(b)	.	.	.	.	.	.	.	.
0	Intercept	19,000	,294	64,540	,000	18,359	19,641	,997	64,540	1,000
	[Sequ=HT1]	1,400	,416	3,363	,006	,493	2,307	,485	3,363	,870
	[Sequ=HT2]	2,000	,416	4,804	,000	1,093	2,907	,658	4,804	,993
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
1	Intercept	20,000	,258	77,460	,000	19,437	20,563	,998	77,460	1,000
	[Sequ=HT1]	,800	,365	2,191	,049	,004	1,596	,286	2,191	,522
	[Sequ=HT2]	1,600	,365	4,382	,001	,804	2,396	,615	4,382	,980
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
2	Intercept	20,000	,216	92,582	,000	19,529	20,471	,999	92,582	1,000
	[Sequ=HT1]	1,800	,306	5,892	,000	1,134	2,466	,743	5,892	1,000
	[Sequ=HT2]	2,000	,306	6,547	,000	1,334	2,666	,781	6,547	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
3	Intercept	20,400	,141	144,250	,000	20,092	20,708	,999	144,250	1,000
	[Sequ=HT1]	1,600	,200	8,000	,000	1,164	2,036	,842	8,000	1,000
	[Sequ=HT2]	1,600	,200	8,000	,000	1,164	2,036	,842	8,000	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
4	Intercept	20,400	,148	138,109	,000	20,075	20,725	,999	138,109	1,000
	[Sequ=HT1]	1,600	,222	7,221	,000	1,112	2,088	,826	7,221	1,000
	[Sequ=HT2]	1,600	,209	7,659	,000	1,140	2,060	,842	7,659	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
5	Intercept	20,400	,148	138,109	,000	20,075	20,725	,999	138,109	1,000
	[Sequ=HT1]	1,600	,209	7,659	,000	1,140	2,060	,842	7,659	1,000
	[Sequ=HT2]	1,600	,222	7,221	,000	1,112	2,088	,826	7,221	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
6	Intercept	21,000	,191	110,125	,000	20,580	21,420	,999	110,125	1,000
	[Sequ=HT1]	1,000	,270	3,708	,003	,406	1,594	,556	3,708	,921
	[Sequ=HT2]	1,000	,286	3,496	,005	,370	1,630	,526	3,496	,888
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.

a Computed using alpha = ,05

b This parameter is set to zero because it is redundant.

Post Hoc Tests **(Sequ)**

Multiple Comparisons

Dependent Variable: lkf

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
							Lower Bound	Upper Bound
0	Tukey HSD	HT1	HT2	-,60	,416	,352	-1,71	,51
			HTOT	1,40(*)	,416	,014	,29	2,51
		HT2	HT1	,60	,416	,352	-,51	1,71
			HTOT	2,00(*)	,416	,001	,89	3,11
		HTOT	HT1	-1,40(*)	,416	,014	-2,51	-,29
			HT2	-2,00(*)	,416	,001	-3,11	-,89
	LSD	HT1	HT2	-,60	,416	,175	-1,51	,31
			HTOT	1,40(*)	,416	,006	,49	2,31
		HT2	HT1	,60	,416	,175	-,31	1,51
			HTOT	2,00(*)	,416	,000	1,09	2,91
		HTOT	HT1	-1,40(*)	,416	,006	-2,31	-,49
			HT2	-2,00(*)	,416	,000	-2,91	-1,09
	Dunnett T3	HT1	HT2	-,60	,400	,408	-1,80	,60
			HTOT	1,40(*)	,400	,025	,20	2,60
		HT2	HT1	,60	,400	,408	-,60	1,80
			HTOT	2,00(*)	,447	,006	,68	3,32
		HTOT	HT1	-1,40(*)	,400	,025	-2,60	-,20
			HT2	-2,00(*)	,447	,006	-3,32	-,68
1	Tukey HSD	HT1	HT2	-,80	,365	,113	-1,77	,17
			HTOT	,80	,365	,113	-,17	1,77
		HT2	HT1	,80	,365	,113	-,17	1,77
			HTOT	1,60(*)	,365	,002	,63	2,57
		HTOT	HT1	-,80	,365	,113	-1,77	,17
			HT2	-1,60(*)	,365	,002	-2,57	-,63
	LSD	HT1	HT2	-,80(*)	,365	,049	-1,60	,00
			HTOT	,80(*)	,365	,049	,00	1,60
		HT2	HT1	,80(*)	,365	,049	,00	1,60
			HTOT	1,60(*)	,365	,001	,80	2,40
		HTOT	HT1	-,80(*)	,365	,049	-1,60	,00
			HT2	-1,60(*)	,365	,001	-2,40	-,80

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
1	Dunnett T3	HT1	HT2	-,80	,316	,097	-1,74	,14
			HTOT	,80	,374	,181	-,35	1,95
		HT2	HT1	,80	,316	,097	-,14	1,74
			HTOT	1,60(*)	,400	,013	,40	2,80
		HTOT	HT1	-,80	,374	,181	-1,95	,35
			HT2	-1,60(*)	,400	,013	-2,80	-,40
2	Tukey HSD	HT1	HT2	-,20	,306	,793	-1,02	,62
			HTOT	1,80(*)	,306	,000	,98	2,62
		HT2	HT1	,20	,306	,793	-,62	1,02
			HTOT	2,00(*)	,306	,000	1,18	2,82
		HTOT	HT1	-1,80(*)	,306	,000	-2,62	-,98
			HT2	-2,00(*)	,306	,000	-2,82	-1,18
	LSD	HT1	HT2	-,20	,306	,525	-,87	,47
			HTOT	1,80(*)	,306	,000	1,13	2,47
		HT2	HT1	,20	,306	,525	-,47	,87
			HTOT	2,00(*)	,306	,000	1,33	2,67
		HTOT	HT1	-1,80(*)	,306	,000	-2,47	-1,13
			HT2	-2,00(*)	,306	,000	-2,67	-1,33
	Dunnett T3	HT1	HT2	-,20	,200	,704	-,95	,55
			HTOT	1,80(*)	,374	,006	,65	2,95
		HT2	HT1	,20	,200	,704	-,55	,95
			HTOT	2,00(*)	,316	,008	,82	3,18
		HTOT	HT1	-1,80(*)	,374	,006	-2,95	-,65
			HT2	-2,00(*)	,316	,008	-3,18	-,82
3	Tukey HSD	HT1	HT2	,00	,200	1,000	-,53	,53
			HTOT	1,60(*)	,200	,000	1,07	2,13
		HT2	HT1	,00	,200	1,000	-,53	,53
			HTOT	1,60(*)	,200	,000	1,07	2,13
		HTOT	HT1	-1,60(*)	,200	,000	-2,13	-1,07
			HT2	-1,60(*)	,200	,000	-2,13	-1,07

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
3	LSD	HT1	HT2	,00	,200	1,000	-,44	,44
			HTOT	1,60(*)	,200	,000	1,16	2,04
		HT2	HT1	,00	,200	1,000	-,44	,44
			HTOT	1,60(*)	,200	,000	1,16	2,04
		HTOT	HT1	-1,60(*)	,200	,000	-2,04	-1,16
			HT2	-1,60(*)	,200	,000	-2,04	-1,16
	Dunnett T3	HT1	HT2	,00	,000	.	,00	,00
			HTOT	1,60(*)	,245	,007	,68	2,52
		HT2	HT1	,00	,000	.	,00	,00
			HTOT	1,60(*)	,245	,007	,68	2,52
		HTOT	HT1	-1,60(*)	,245	,007	-2,52	-,68
			HT2	-1,60(*)	,245	,007	-2,52	-,68
4	Tukey HSD	HT1	HT2	,00	,222	1,000	-,60	,60
			HTOT	1,60(*)	,222	,000	1,00	2,20
		HT2	HT1	,00	,222	1,000	-,60	,60
			HTOT	1,60(*)	,209	,000	1,04	2,16
		HTOT	HT1	-1,60(*)	,222	,000	-2,20	-1,00
			HT2	-1,60(*)	,209	,000	-2,16	-1,04
	LSD	HT1	HT2	,00	,222	1,000	-,49	,49
			HTOT	1,60(*)	,222	,000	1,11	2,09
		HT2	HT1	,00	,222	1,000	-,49	,49
			HTOT	1,60(*)	,209	,000	1,14	2,06
		HTOT	HT1	-1,60(*)	,222	,000	-2,09	-1,11
			HT2	-1,60(*)	,209	,000	-2,06	-1,14
	Dunnett T3	HT1	HT2	,00	,000	.	,00	,00
			HTOT	1,60(*)	,245	,007	,68	2,52
		HT2	HT1	,00	,000	.	,00	,00
			HTOT	1,60(*)	,245	,007	,68	2,52
		HTOT	HT1	-1,60(*)	,245	,007	-2,52	-,68
			HT2	-1,60(*)	,245	,007	-2,52	-,68

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
5	Tukey HSD	HT1	HT2	,00	,222	1,000	-,60	,60
			HTOT	1,60(*)	,209	,000	1,04	2,16
		HT2	HT1	,00	,222	1,000	-,60	,60
			HTOT	1,60(*)	,222	,000	1,00	2,20
		HTOT	HT1	-1,60(*)	,209	,000	-2,16	-1,04
			HT2	-1,60(*)	,222	,000	-2,20	-1,00
	LSD	HT1	HT2	,00	,222	1,000	-,49	,49
			HTOT	1,60(*)	,209	,000	1,14	2,06
		HT2	HT1	,00	,222	1,000	-,49	,49
			HTOT	1,60(*)	,222	,000	1,11	2,09
		HTOT	HT1	-1,60(*)	,209	,000	-2,06	-1,14
			HT2	-1,60(*)	,222	,000	-2,09	-1,11
	Dunnett T3	HT1	HT2	,00	,000	.	,00	,00
			HTOT	1,60(*)	,245	,007	,68	2,52
		HT2	HT1	,00	,000	.	,00	,00
			HTOT	1,60(*)	,245	,007	,68	2,52
		HTOT	HT1	-1,60(*)	,245	,007	-2,52	-,68
			HT2	-1,60(*)	,245	,007	-2,52	-,68
6	Tukey HSD	HT1	HT2	,00	,286	1,000	-,77	,77
			HTOT	1,00(*)	,270	,009	,27	1,73
		HT2	HT1	,00	,286	1,000	-,77	,77
			HTOT	1,00(*)	,286	,013	,23	1,77
		HTOT	HT1	-1,00(*)	,270	,009	-1,73	-,27
			HT2	-1,00(*)	,286	,013	-1,77	-,23
	LSD	HT1	HT2	,00	,286	1,000	-,63	,63
			HTOT	1,00(*)	,270	,003	,41	1,59
		HT2	HT1	,00	,286	1,000	-,63	,63
			HTOT	1,00(*)	,286	,005	,37	1,63
		HTOT	HT1	-1,00(*)	,270	,003	-1,59	-,41
			HT2	-1,00(*)	,286	,005	-1,63	-,37

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
6	Dunnett T3	HT1	HT2	,00	,000	.	,00	,00
			HTOT	1,00	,316	,084	-,18	2,18
		HT2	HT1	,00	,000	.	,00	,00
			HTOT	1,00	,316	,084	-,18	2,18
		HTOT	HT1	-1,00	,316	,084	-2,18	,18
			HT2	-1,00	,316	,084	-2,18	,18

Based on observed means.

* The mean difference is significant at the ,05 level.

Homogeneous Subsets

lkf

Day after therapy=0

	Sequ	N	Subset 1	Subset 2
Tukey HSD(a,b)	HTOT	5	19,00	
	HT1	5		20,40
	HT2	5		21,00
	Sig.		1,000	,352

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,433.

a Uses Harmonic Mean Sample Size = 5,000.

b Alpha = ,05.

Day after therapy=1

	Sequ	N	Subset 1	Subset 2
Tukey HSD(a,b)	HTOT	5	20,00	
	HT1	5	20,80	20,80
	HT2	5		21,60
	Sig.		,113	,113

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,333.
a Uses Harmonic Mean Sample Size = 5,000.
b Alpha = ,05.

Day after therapy=2

	Sequ	N	Subset 1	Subset 2
Tukey HSD(a,b)	HTOT	5	20,00	
	HT1	5		21,80
	HT2	5		22,00
	Sig.		1,000	,793

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,233.
a Uses Harmonic Mean Sample Size = 5,000.
b Alpha = ,05.

Day after therapy=3

	Sequ	N	Subset 1	Subset 2
Tukey HSD(a,b)	HTOT	5	20,40	
	HT1	5		22,00
	HT2	5		22,00
	Sig.		1,000	1,000

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,100.
a Uses Harmonic Mean Sample Size = 5,000.
b Alpha = ,05.

Day after therapy=4

	Sequ	N	Subset 1	Subset 2
Tukey HSD(a,b,c)	HTOT	5	20,40	
	HT1	4		22,00
	HT2	5		22,00
	Sig.		1,000	1,000

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,109.
a Uses Harmonic Mean Sample Size = 4,615.
b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.
c Alpha = ,05.

Day after therapy=5

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b,c)	HTOT	5	20,40	
	HT1	5		22,00
	HT2	4		22,00
	Sig.		1,000	1,000

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,109.
a Uses Harmonic Mean Sample Size = 4,615.
b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.
c Alpha = ,05.

Day after therapy=6

			Subset	
	Sequ	N	1	2
Tukey HSD(a,b,c)	HTOT	5	21,00	
	HT1	5		22,00
	HT2	4		22,00
	Sig.		1,000	1,000

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,182.
a Uses Harmonic Mean Sample Size = 4,615.
b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.
c Alpha = ,05.

Appendix 1c

Results
ANOVA
Fist-Floor Distance

ST - Univariate Analysis of Variance

Between-Subjects Factors

		N
Sequ	HT1	34
	HT2	33
	HTOT	35
	nT	2

Descriptive Statistics

Dependent Variable: st

Sequ	Mean	Std. Deviation	N
HT1	28,91	,866	34
HT2	29,64	,962	33
HTOT	26,31	,963	35
nT	30,00	,000	2
Total	28,29	1,710	104

Levene's Test of Equality of Error Variances(a)

Dependent Variable: st

F	df1	df2	Sig.
1,409	3	100	,245

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.
a Design: Intercept+Sequ

Tests of Between-Subjects Effects

Dependent Variable: st

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power(a)
Corrected Model	215,432(b)	3	71,811	83,584	,000	,715	250,751	1,000
Intercept	22426,794	1	22426,794	26103,615	,000	,996	26103,615	1,000
Sequ	215,432	3	71,811	83,584	,000	,715	250,751	1,000
Error	85,915	100	,859					
Total	83526,000	104						
Corrected Total	301,346	103						

a Computed using alpha = ,05

b R Squared = ,715 (Adjusted R Squared = ,706)

Parameter Estimates

Dependent Variable: st

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared	Noncent. Parameter	Observed Power(a)
					Lower Bound	Upper Bound			
Intercept	30,000	,655	45,772	,000	28,700	31,300	,954	45,772	1,000
[Sequ=HT1]	-1,088	,674	-1,614	,110	-2,426	,250	,025	1,614	,359
[Sequ=HT2]	-,364	,675	-,539	,591	-1,703	,976	,003	,539	,083
[Sequ=HTOT]	-3,686	,674	-5,469	,000	-5,023	-2,349	,230	5,469	1,000
[Sequ=nT]	0(b)	.	.	.	.	.	.	.	.

a Computed using alpha = ,05

b This parameter is set to zero because it is redundant.

Sequ

Multiple Comparisons

Dependent Variable: st

	(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
Tukey HSD	HT1	HT2	-,72(*)	,227	,010	-1,32	-,13
		HTOT	2,60(*)	,223	,000	2,01	3,18
		nT	-1,09	,674	,376	-2,85	,67
	HT2	HT1	,72(*)	,227	,010	,13	1,32
		HTOT	3,32(*)	,225	,000	2,73	3,91
		nT	-,36	,675	,949	-2,13	1,40
	HTOT	HT1	-2,60(*)	,223	,000	-3,18	-2,01
		HT2	-3,32(*)	,225	,000	-3,91	-2,73
		nT	-3,69(*)	,674	,000	-5,45	-1,93
	nT	HT1	1,09	,674	,376	-,67	2,85
		HT2	,36	,675	,949	-1,40	2,13
		HTOT	3,69(*)	,674	,000	1,93	5,45
LSD	HT1	HT2	-,72(*)	,227	,002	-1,17	-,28
		HTOT	2,60(*)	,223	,000	2,15	3,04
		nT	-1,09	,674	,110	-2,43	,25
	HT2	HT1	,72(*)	,227	,002	,28	1,17
		HTOT	3,32(*)	,225	,000	2,88	3,77
		nT	-,36	,675	,591	-1,70	,98
	HTOT	HT1	-2,60(*)	,223	,000	-3,04	-2,15
		HT2	-3,32(*)	,225	,000	-3,77	-2,88
		nT	-3,69(*)	,674	,000	-5,02	-2,35
	nT	HT1	1,09	,674	,110	-,25	2,43
		HT2	,36	,675	,591	-,98	1,70
		HTOT	3,69(*)	,674	,000	2,35	5,02
Dunnett T3	HT1	HT2	-,72(*)	,224	,011	-1,33	-,12
		HTOT	2,60(*)	,220	,000	2,00	3,19
		nT	-1,09(*)	,148	,000	-1,50	-,67
	HT2	HT1	,72(*)	,224	,011	,12	1,33
		HTOT	3,32(*)	,234	,000	2,69	3,95
		nT	-,36	,168	,197	-,83	,10
	HTOT	HT1	-2,60(*)	,220	,000	-3,19	-2,00
		HT2	-3,32(*)	,234	,000	-3,95	-2,69
		nT	-3,69(*)	,163	,000	-4,14	-3,23
	nT	HT1	1,09(*)	,148	,000	,67	1,50
		HT2	,36	,168	,197	-,10	,83
		HTOT	3,69(*)	,163	,000	3,23	4,14

Based on observed means.

* The mean difference is significant at the ,05 level.

Homogeneous Subsets

st

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b,c)	HTOT	35	26,31	
	HT1	34		28,91
	HT2	33		29,64
	nT	2		30,00
	Sig.		1,000	,140

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,859.
a Uses Harmonic Mean Sample Size = 6,799.
b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.
c Alpha = ,05.

ST/Days after therapy - Univariate Analysis of Variance

Between-Subjects Factors

Day after therapy			N
.	Sequ	nT	2
0	Sequ	HT1	5
		HT2	5
		HTOT	5
1	Sequ	HT1	5
		HT2	5
		HTOT	5
2	Sequ	HT1	5
		HT2	5
		HTOT	5
3	Sequ	HT1	5
		HT2	5
		HTOT	5
4	Sequ	HT1	4
		HT2	5
		HTOT	5
5	Sequ	HT1	5
		HT2	4
		HTOT	5
6	Sequ	HT1	5
		HT2	4
		HTOT	5

Descriptive Statistics

Dependent Variable: st

Day after therapy	Sequ	Mean	Std. Deviation	N
.	nT	30,00	,000	2
	Total	30,00	,000	2
0	HT1	28,00	,707	5
	HT2	29,00	1,225	5
	HTOT	25,40	,548	5
	Total	27,47	1,767	15
1	HT1	28,40	,548	5
	HT2	29,20	1,304	5
	HTOT	25,80	,447	5
	Total	27,80	1,699	15
2	HT1	28,80	,837	5
	HT2	29,60	,894	5
	HTOT	26,00	,707	5
	Total	28,13	1,767	15
3	HT1	29,20	1,095	5
	HT2	29,60	,894	5
	HTOT	26,20	,837	5
	Total	28,33	1,799	15
4	HT1	29,00	,816	4
	HT2	30,00	,707	5
	HTOT	26,60	1,140	5
	Total	28,50	1,743	14
5	HT1	29,40	,548	5
	HT2	30,00	,816	4
	HTOT	27,00	1,000	5
	Total	28,71	1,541	14
6	HT1	29,60	,548	5
	HT2	30,25	,500	4
	HTOT	27,20	,837	5
	Total	28,93	1,492	14

Levene's Test of Equality of Error Variances(a,b)

Dependent Variable: st

Day after therapy	F	df1	df2	Sig.
0	,664	2	12	,533
1	2,633	2	12	,113
2	,371	2	12	,698
3	,031	2	12	,969
4	,985	2	11	,404
5	,929	2	11	,424
6	,901	2	11	,434

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a Test is not computed for one or more split files because there are less than two nonempty cells.

b Design: Intercept+Sequ

Tests of Between-Subjects Effects

Dependent Variable: st

Day after therapy	Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power(a)
.	Corrected Model	,000(b)	0	.	.	.	.	.	.
	Intercept	1800,000	1	1800,000	.	.	1,000	.	.
	Sequ	,000	0	.	.	.	.	.	.
	Error	,000	1	,000					
	Total	1800,000	2						
	Corrected Total	,000	1						
0	Corrected Model	34,533(c)	2	17,267	22,522	,000	,790	45,043	1,000
	Intercept	11316,267	1	11316,267	14760,348	,000	,999	14760,348	1,000
	Sequ	34,533	2	17,267	22,522	,000	,790	45,043	1,000
	Error	9,200	12	,767					
	Total	11360,000	15						
	Corrected Total	43,733	14						
1	Corrected Model	31,600(d)	2	15,800	21,545	,000	,782	43,091	1,000
	Intercept	11592,600	1	11592,600	15808,091	,000	,999	15808,091	1,000
	Sequ	31,600	2	15,800	21,545	,000	,782	43,091	1,000
	Error	8,800	12	,733					
	Total	11633,000	15						
	Corrected Total	40,400	14						
2	Corrected Model	35,733(e)	2	17,867	26,800	,000	,817	53,600	1,000
	Intercept	11872,267	1	11872,267	17808,400	,000	,999	17808,400	1,000
	Sequ	35,733	2	17,867	26,800	,000	,817	53,600	1,000
	Error	8,000	12	,667					
	Total	11916,000	15						
	Corrected Total	43,733	14						
3	Corrected Model	34,533(f)	2	17,267	19,185	,000	,762	38,370	,999
	Intercept	12041,667	1	12041,667	13379,630	,000	,999	13379,630	1,000
	Sequ	34,533	2	17,267	19,185	,000	,762	38,370	,999
	Error	10,800	12	,900					
	Total	12087,000	15						
	Corrected Total	45,333	14						

Day after therapy	Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power(a)
4	Corrected Model	30,300(g)	2	15,150	18,114	,000	,767	36,228	,998
	Intercept	11272,862	1	11272,862	13478,421	,000	,999	13478,421	1,000
	Sequ	30,300	2	15,150	18,114	,000	,767	36,228	,998
	Error	9,200	11	,836					
	Total	11411,000	14						
	Corrected Total	39,500	13						
5	Corrected Model	23,657(h)	2	11,829	18,071	,000	,767	36,143	,998
	Intercept	11484,554	1	11484,554	17545,846	,000	,999	17545,846	1,000
	Sequ	23,657	2	11,829	18,071	,000	,767	36,143	,998
	Error	7,200	11	,655					
	Total	11574,000	14						
	Corrected Total	30,857	13						
6	Corrected Model	24,179(i)	2	12,089	27,996	,000	,836	55,992	1,000
	Intercept	11658,004	1	11658,004	26997,483	,000	1,000	26997,483	1,000
	Sequ	24,179	2	12,089	27,996	,000	,836	55,992	1,000
	Error	4,750	11	,432					
	Total	11745,000	14						
	Corrected Total	28,929	13						

a Computed using alpha = ,05
b R Squared = . (Adjusted R Squared = .)
c R Squared = ,790 (Adjusted R Squared = ,755)
d R Squared = ,782 (Adjusted R Squared = ,746)
e R Squared = ,817 (Adjusted R Squared = ,787)
f R Squared = ,762 (Adjusted R Squared = ,722)
g R Squared = ,767 (Adjusted R Squared = ,725)
h R Squared = ,767 (Adjusted R Squared = ,724)
i R Squared = ,836 (Adjusted R Squared = ,806)

Parameter Estimates

Dependent Variable: st

Day after therapy	Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared	Noncent. Parameter	Observed Power(a)
						Lower Bound	Upper Bound			
.	Intercept	30,000	,000	.	.	30,000	30,000	1,000	.	.
	[Sequ=nT]	0(b)	.	.	.	.	.	.	.	.
0	Intercept	25,400	,392	64,866	,000	24,547	26,253	,997	64,866	1,000
	[Sequ=HT1]	2,600	,554	4,695	,001	1,393	3,807	,648	4,695	,990
	[Sequ=HT2]	3,600	,554	6,501	,000	2,393	4,807	,779	6,501	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
1	Intercept	25,800	,383	67,368	,000	24,966	26,634	,997	67,368	1,000
	[Sequ=HT1]	2,600	,542	4,801	,000	1,420	3,780	,658	4,801	,992
	[Sequ=HT2]	3,400	,542	6,278	,000	2,220	4,580	,767	6,278	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
2	Intercept	26,000	,365	71,204	,000	25,204	26,796	,998	71,204	1,000
	[Sequ=HT1]	2,800	,516	5,422	,000	1,675	3,925	,710	5,422	,999
	[Sequ=HT2]	3,600	,516	6,971	,000	2,475	4,725	,802	6,971	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
3	Intercept	26,200	,424	61,754	,000	25,276	27,124	,997	61,754	1,000
	[Sequ=HT1]	3,000	,600	5,000	,000	1,693	4,307	,676	5,000	,995
	[Sequ=HT2]	3,400	,600	5,667	,000	2,093	4,707	,728	5,667	,999
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
4	Intercept	26,600	,409	65,038	,000	25,700	27,500	,997	65,038	1,000
	[Sequ=HT1]	2,400	,613	3,912	,002	1,050	3,750	,582	3,912	,944
	[Sequ=HT2]	3,400	,578	5,878	,000	2,127	4,673	,759	5,878	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
5	Intercept	27,000	,362	74,624	,000	26,204	27,796	,998	74,624	1,000
	[Sequ=HT1]	2,400	,512	4,690	,001	1,274	3,526	,667	4,690	,989
	[Sequ=HT2]	3,000	,543	5,528	,000	1,805	4,195	,735	5,528	,999
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.
6	Intercept	27,200	,294	92,556	,000	26,553	27,847	,999	92,556	1,000
	[Sequ=HT1]	2,400	,416	5,775	,000	1,485	3,315	,752	5,775	,999
	[Sequ=HT2]	3,050	,441	6,919	,000	2,080	4,020	,813	6,919	1,000
	[Sequ=HTOT]	0(b)	.	.	.	.	.	.	.	.

a Computed using alpha = ,05

b This parameter is set to zero because it is redundant.

Post Hoc Tests **(Sequ)**

Multiple Comparisons

Dependent Variable: st

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
							Lower Bound	Upper Bound
0	Tukey HSD	HT1	HT2	-1,00	,554	,209	-2,48	,48
			HTOT	2,60(*)	,554	,001	1,12	4,08
		HT2	HT1	1,00	,554	,209	-,48	2,48
			HTOT	3,60(*)	,554	,000	2,12	5,08
		HTOT	HT1	-2,60(*)	,554	,001	-4,08	-1,12
			HT2	-3,60(*)	,554	,000	-5,08	-2,12
	LSD	HT1	HT2	-1,00	,554	,096	-2,21	,21
			HTOT	2,60(*)	,554	,001	1,39	3,81
		HT2	HT1	1,00	,554	,096	-,21	2,21
			HTOT	3,60(*)	,554	,000	2,39	4,81
		HTOT	HT1	-2,60(*)	,554	,001	-3,81	-1,39
			HT2	-3,60(*)	,554	,000	-4,81	-2,39
	Dunnett T3	HT1	HT2	-1,00	,632	,378	-2,98	,98
			HTOT	2,60(*)	,400	,001	1,40	3,80
		HT2	HT1	1,00	,632	,378	-,98	2,98
			HTOT	3,60(*)	,600	,004	1,63	5,57
		HTOT	HT1	-2,60(*)	,400	,001	-3,80	-1,40
			HT2	-3,60(*)	,600	,004	-5,57	-1,63
1	Tukey HSD	HT1	HT2	-,80	,542	,336	-2,24	,64
			HTOT	2,60(*)	,542	,001	1,16	4,04
		HT2	HT1	,80	,542	,336	-,64	2,24
			HTOT	3,40(*)	,542	,000	1,96	4,84
		HTOT	HT1	-2,60(*)	,542	,001	-4,04	-1,16
			HT2	-3,40(*)	,542	,000	-4,84	-1,96
	LSD	HT1	HT2	-,80	,542	,165	-1,98	,38
			HTOT	2,60(*)	,542	,000	1,42	3,78
		HT2	HT1	,80	,542	,165	-,38	1,98
			HTOT	3,40(*)	,542	,000	2,22	4,58
		HTOT	HT1	-2,60(*)	,542	,000	-3,78	-1,42
			HT2	-3,40(*)	,542	,000	-4,58	-2,22

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
1	Dunnett T3	HT1	HT2	-,80	,632	,547	-2,89	1,29
			HTOT	2,60(*)	,316	,000	1,66	3,54
		HT2	HT1	,80	,632	,547	-1,29	2,89
			HTOT	3,40(*)	,616	,007	1,29	5,51
		HTOT	HT1	-2,60(*)	,316	,000	-3,54	-1,66
			HT2	-3,40(*)	,616	,007	-5,51	-1,29
2	Tukey HSD	HT1	HT2	-,80	,516	,304	-2,18	,58
			HTOT	2,80(*)	,516	,000	1,42	4,18
		HT2	HT1	,80	,516	,304	-,58	2,18
			HTOT	3,60(*)	,516	,000	2,22	4,98
		HTOT	HT1	-2,80(*)	,516	,000	-4,18	-1,42
			HT2	-3,60(*)	,516	,000	-4,98	-2,22
	LSD	HT1	HT2	-,80	,516	,147	-1,93	,33
			HTOT	2,80(*)	,516	,000	1,67	3,93
		HT2	HT1	,80	,516	,147	-,33	1,93
			HTOT	3,60(*)	,516	,000	2,47	4,73
		HTOT	HT1	-2,80(*)	,516	,000	-3,93	-1,67
			HT2	-3,60(*)	,516	,000	-4,73	-2,47
	Dunnett T3	HT1	HT2	-,80	,548	,425	-2,42	,82
			HTOT	2,80(*)	,490	,001	1,34	4,26
		HT2	HT1	,80	,548	,425	-,82	2,42
			HTOT	3,60(*)	,510	,000	2,07	5,13
		HTOT	HT1	-2,80(*)	,490	,001	-4,26	-1,34
			HT2	-3,60(*)	,510	,000	-5,13	-2,07
3	Tukey HSD	HT1	HT2	-,40	,600	,787	-2,00	1,20
			HTOT	3,00(*)	,600	,001	1,40	4,60
		HT2	HT1	,40	,600	,787	-1,20	2,00
			HTOT	3,40(*)	,600	,000	1,80	5,00
		HTOT	HT1	-3,00(*)	,600	,001	-4,60	-1,40
			HT2	-3,40(*)	,600	,000	-5,00	-1,80

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
3	LSD	HT1	HT2	-,40	,600	,518	-1,71	,91
			HTOT	3,00(*)	,600	,000	1,69	4,31
		HT2	HT1	,40	,600	,518	-,91	1,71
			HTOT	3,40(*)	,600	,000	2,09	4,71
		HTOT	HT1	-3,00(*)	,600	,000	-4,31	-1,69
			HT2	-3,40(*)	,600	,000	-4,71	-2,09
	Dunnett T3	HT1	HT2	-,40	,632	,892	-2,29	1,49
			HTOT	3,00(*)	,616	,004	1,15	4,85
		HT2	HT1	,40	,632	,892	-1,49	2,29
			HTOT	3,40(*)	,548	,001	1,78	5,02
		HTOT	HT1	-3,00(*)	,616	,004	-4,85	-1,15
			HT2	-3,40(*)	,548	,001	-5,02	-1,78
4	Tukey HSD	HT1	HT2	-1,00	,613	,274	-2,66	,66
			HTOT	2,40(*)	,613	,006	,74	4,06
		HT2	HT1	1,00	,613	,274	-,66	2,66
			HTOT	3,40(*)	,578	,000	1,84	4,96
		HTOT	HT1	-2,40(*)	,613	,006	-4,06	-,74
			HT2	-3,40(*)	,578	,000	-4,96	-1,84
	LSD	HT1	HT2	-1,00	,613	,131	-2,35	,35
			HTOT	2,40(*)	,613	,002	1,05	3,75
		HT2	HT1	1,00	,613	,131	-,35	2,35
			HTOT	3,40(*)	,578	,000	2,13	4,67
		HTOT	HT1	-2,40(*)	,613	,002	-3,75	-1,05
			HT2	-3,40(*)	,578	,000	-4,67	-2,13
	Dunnett T3	HT1	HT2	-1,00	,516	,246	-2,64	,64
			HTOT	2,40(*)	,653	,022	,40	4,40
		HT2	HT1	1,00	,516	,246	-,64	2,64
			HTOT	3,40(*)	,600	,003	1,54	5,26
		HTOT	HT1	-2,40(*)	,653	,022	-4,40	-,40
			HT2	-3,40(*)	,600	,003	-5,26	-1,54

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
5	Tukey HSD	HT1	HT2	-,60	,543	,530	-2,07	,87
			HTOT	2,40(*)	,512	,002	1,02	3,78
		HT2	HT1	,60	,543	,530	-,87	2,07
			HTOT	3,00(*)	,543	,000	1,53	4,47
		HTOT	HT1	-2,40(*)	,512	,002	-3,78	-1,02
			HT2	-3,00(*)	,543	,000	-4,47	-1,53
	LSD	HT1	HT2	-,60	,543	,293	-1,79	,59
			HTOT	2,40(*)	,512	,001	1,27	3,53
		HT2	HT1	,60	,543	,293	-,59	1,79
			HTOT	3,00(*)	,543	,000	1,81	4,19
		HTOT	HT1	-2,40(*)	,512	,001	-3,53	-1,27
			HT2	-3,00(*)	,543	,000	-4,19	-1,81
	Dunnett T3	HT1	HT2	-,60	,476	,552	-2,21	1,01
			HTOT	2,40(*)	,510	,008	,79	4,01
		HT2	HT1	,60	,476	,552	-1,01	2,21
			HTOT	3,00(*)	,606	,005	1,15	4,85
		HTOT	HT1	-2,40(*)	,510	,008	-4,01	-,79
			HT2	-3,00(*)	,606	,005	-4,85	-1,15
6	Tukey HSD	HT1	HT2	-,65	,441	,340	-1,84	,54
			HTOT	2,40(*)	,416	,000	1,28	3,52
		HT2	HT1	,65	,441	,340	-,54	1,84
			HTOT	3,05(*)	,441	,000	1,86	4,24
		HTOT	HT1	-2,40(*)	,416	,000	-3,52	-1,28
			HT2	-3,05(*)	,441	,000	-4,24	-1,86
	LSD	HT1	HT2	-,65	,441	,168	-1,62	,32
			HTOT	2,40(*)	,416	,000	1,49	3,31
		HT2	HT1	,65	,441	,168	-,32	1,62
			HTOT	3,05(*)	,441	,000	2,08	4,02
		HTOT	HT1	-2,40(*)	,416	,000	-3,31	-1,49
			HT2	-3,05(*)	,441	,000	-4,02	-2,08

Day after therapy		(I) Sequ	(J) Sequ	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
	Dunnett T3	HT1	HT2	-,65	,350	,263	-1,73	,43
			HTOT	2,40(*)	,447	,003	1,03	3,77
		HT2	HT1	,65	,350	,263	-,43	1,73
			HTOT	3,05(*)	,450	,001	1,65	4,45
		HTOT	HT1	-2,40(*)	,447	,003	-3,77	-1,03
			HT2	-3,05(*)	,450	,001	-4,45	-1,65

Based on observed means.
* The mean difference is significant at the ,05 level.

Homogeneous Subsets

st

Day after therapy=0

	Sequ	N	Subset 1	Subset 2
Tukey HSD(a,b)	HTOT	5	25,40	
	HT1	5		28,00
	HT2	5		29,00
	Sig.		1,000	,209

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,767.
a Uses Harmonic Mean Sample Size = 5,000.
b Alpha = ,05.

Day after therapy=1

	Sequ	N	Subset 1	Subset 2
Tukey HSD(a,b)	HTOT	5	25,80	
	HT1	5		28,40
	HT2	5		29,20
	Sig.		1,000	,336

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,733.
a Uses Harmonic Mean Sample Size = 5,000.
b Alpha = ,05.

Day after therapy=2

	Sequ	N	Subset 1	Subset 2
Tukey HSD(a,b)	HTOT	5	26,00	
	HT1	5		28,80
	HT2	5		29,60
	Sig.		1,000	,304

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,667.
a Uses Harmonic Mean Sample Size = 5,000.
b Alpha = ,05.

Day after therapy=3

	Sequ	N	Subset 1	Subset 2
Tukey HSD(a,b)	HTOT	5	26,20	
	HT1	5		29,20
	HT2	5		29,60
	Sig.		1,000	,787

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,900.
a Uses Harmonic Mean Sample Size = 5,000.
b Alpha = ,05.

Day after therapy=4

	Sequ	N	Subset 1	Subset 2
Tukey HSD(a,b,c)	HTOT	5	26,60	
	HT1	4		29,00
	HT2	5		30,00
	Sig.		1,000	,263

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,836.
a Uses Harmonic Mean Sample Size = 4,615.
b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.
c Alpha = ,05.

Day after therapy=5

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b,c)	HTOT	5	27,00	
	HT1	5		29,40
	HT2	4		30,00
	Sig.		1,000	,518

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,655.
a Uses Harmonic Mean Sample Size = 4,615.
b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.
c Alpha = ,05.

Day after therapy=6

	Sequ	N	Subset	
			1	2
Tukey HSD(a,b,c)	HTOT	5	27,20	
	HT1	5		29,60
	HT2	4		30,25
	Sig.		1,000	,327

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = ,432.
a Uses Harmonic Mean Sample Size = 4,615.
b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.
c Alpha = ,05.