

**The treatment of Chronic Low Back Pain
following the principles of the Fascial
Distortion Modell (FDM)**

A quasi experimental study

Master Thesis to obtain the degree

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DECLARATION

Hereby I declare that I have written the present master thesis on my own.

I have clearly marked as quotes all parts of the text that I have copied literally or rephrased from published or unpublished works of other authors.

All sources and references I have used in writing this thesis are listed in the bibliography. No thesis with the same content was submitted to any other examination board before.

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Abstract

BACKGROUND: Chronic Low Back Pain (CLBP) is a burden for a high percentage of people and therefore a major field of activity for osteopaths. The present study introduces the Fascial Distortion Modell (FDM) and observes the efficacy of treatments following its principles.

METHODS: In a quasi experimental study 22 probands suffering from CLBP are twice treated following the principles of the FDM and are repeatedly tested for their functional status (ODI), pain (VAS), finger-floor distance and intake of analgesics. Three pre-treatment measurements act as base line values and are compared with two post-treatment measurements.

RESULTS: All four parameters statistically significant change for the better. Mean functional status measured by means of the ODI improves from 20, 64 to 12, 75. Mean VAS results drop from 3, 57 to 1, 43. Mean finger-floor distance is reduced from 8, 2 cm to 4, 7 cm in the first treatment respectively from 6, 6 cm to 4, 1 cm in the second treatment. Also the number of probands taking analgesics is reduced during study period

CONCLUSION: A treatment following the principles of the FDM is efficacious in treating patients suffering with CLBP.

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1. Introduction

Epidemiological studies show that the life-time prevalence of low back pain (LBP) is up to 84% (European Guidelines for the management of chronic low back pain [EGMCLBP] 2005) and is therefore a common burden for nearly everyone. LBP is not only a personal but also an economic problem: in the United States LBP leads to an estimated loss of 149 million workdays annually (*Guo, 1999*). Medical physicians, physiotherapists, chiropractors, acupuncturists, massage therapists as well as osteopaths are typical care providers in cases of low back pain (*Cote, 2005*). Despite the different treatments those care providers can offer the majority of patients suffering from LBP chooses **not** to seek care (*Walker, 2004 / Mortimer, 2003 / Balague, 2007*). Out of those people who seek care the majority chooses the general practitioner (30%), or a combination of GP and physiotherapist (55%; *Cote, 2005*). Although the quoted studies describe the situation in Australia I dare say that the situation is pretty the same in Austria: the medical physician is surely the first port of call.

Nevertheless in the osteopathic practise the majority of patients seek our help because of chronic low back pain (CLBP). They are being referred from general practitioners or orthopaedists for several diagnoses such as disc herniation (protrusion, prolaps, sequestrum...), vertebrostenosis, spondylarthrosis or chronic lumbago.

Most of them have already undergone x-ray and MRI to find the exact cause of their pain. Given the findings of the radiologist it should be easy to meliorate the patient's symptoms: stretch where tissues are too short, release and relax tissues that have too much tension, improve blood circulation for better nutrition of the tissue and so on. So far so good - unfortunately when I am doing my examination on those patients, I often realize that my clinical osteopathical findings do not really match the existing diagnosis. Especially in elderly patients MRI often show multiple lesions of each and every disc. Which of these lesions is causing the pain? Is it only one or is it the combination of all existing (visible) lesions harming the patient?

The MRI offers a perfect snap shot and reveals the current state of the patients' spine. But it does not reveal the source of the patients' pain without any doubt: As the pathological changes in the spine do not form all of a sudden but took time to develop, the degeneration of the vertebrae and discs might have existed long before the patient complains about pain. (I will refer to respective radiological studies in chapter 2.3) What was the last straw? What was the additional stress, which led to the patient feeling pain?

As far as answering these questions is concerned the common morphology-based explanation of the pathogenesis of LBP (blaming degenerative processes of bone, discs and ligaments for the pain) falls short.

With the Fascial Distortion Model (FDM) Stephen Typaldos D.O. suggests a new approach to musculo-skeletal injuries and pain, using a totally different perception of pathogenesis of pain. In his model Typaldos (2002) ignores degeneration, lesions or injuries of the tissues depicted by imaging techniques, and states that the fascial system is the major contributor of pain. Following Typaldos (2002) therapists shall only treat distortions of fasciae and by doing so help restore pain-free conditions for the patient.

The research question of this master thesis is:

Is a treatment following the principles of the FDM efficacious in treating patients suffering from chronic lumbar pain?

The underlying hypothesis of this study is that there is a considerable, measurable improvement with mere two treatments.

In order to examine this research question and to verify the hypothesis, 22 patients, suffering from chronic low back pain were observed in a study, using a quasi experimental design with repeated measurements.

Due to the fact that chronic lumbar pain leads to impairments in numerous activities of daily life, the most important parameter of this thesis is the functional status of the patients. This is examined by using the Oswestry Disability Index (ODI)

The second parameter in question is the intensity of pain measured on a visual analogue scale (VAS).

Besides these two main parameters this study also observes mobility and intake of analgesics as a third and fourth parameter.

2. Chronic Low Back Pain (CLBP)

Chronic low back pain is a topic that is often and profoundly investigated in countless scientific studies. Due to this fact this chapter will concentrate on high quality papers and provides a brief summary of the current state of knowledge concerning CLBP.

2.1 Definition of Chronic Low Back Pain

“Low back pain is defined as pain and discomfort, localised below the costal margin and above the inferior gluteal folds, with or without referred leg pain.” (European Guidelines for the management of chronic non-specific low back pain; [EGMCLBP] 2005, page 30)

Following the authors of the above quoted guidelines, chronic pain is defined as:

“pain persisting for at least 12 weeks”. (EGMCLBP, page 30)

This duration of 12 weeks includes subacute back pain, back pain that has lasted for a very long period of time and also cases of recurrent pain, with the current episode of pain lasting approximately 12 weeks. (EGMCLBP, page 30)

LBP can be divided into three categories:

- Specific spinal pathology (see 2.3)
- Nerve root pain/radicular pain (see 2.3)
- Non-specific low back pain (see 2.3)

2.2 Epidemiology of CLBP

Numerous studies (for example Anderson (1993); Bressler (1999); Ebbeloi (2002) and reviews (Hestbaeck (2003); Pengel (2003); Van Tulder (2002)) researched the epidemiological aspects of low back pain, including population-based surveys on the occurrence, socio-cultural determinants and psychosocial correlations.

Referring to five systematic reviews on this topic I would like to give a short overview about the prevalence of LBP.

Two of these reviews focus on Germany and one study assesses the Nordic population (in Denmark, Sweden and Iceland), assuming that the prevalence of LBP in those countries will be very similar to that of the Austrian population. (Unfortunately there are no studies that concentrate solely on Austria)

The study of *Schmidt (2007)* – conducted from 2003 to 2006- investigates the prevalence of LBP in Germany. The target sample consists of 15.750 women and men, drawn at random from municipal population registers of five German cities. Those 15.750 subjects (aged between 18 and 75) were contacted by a 3 stage mailing procedure. In a 10-page questionnaire, they are directly asked for point-, 1-year and lifetime prevalence of LBP. Furthermore, subjects are asked to fill in the Graded Chronic Pain Scale (GCPS) by *Von Korff (1992)* and to mark the location of their pain in a simple anatomical drawing.

9.387 subjects of the target sample returned completed questionnaires, out of which 9.263 were feasible for further analyzes. In Schmidt's study, point-prevalence of LBP is 34,2%, 1-year prevalence 75,5% and a lifetime prevalence 85,2%.

Another survey, assessing LBP prevalence in Germany is done by *Neuhauser (2005)*. In a nation-wide computer assisted telephone interview 8.318 persons (aged 18 years or older) are interrogated.

Neuhauser finds a point-prevalence of LBP of 22,3% and 1-year prevalence of 61,8%. The prevalence of CLBP is asked separately, with an outcome of 18,7% for the 1-year prevalence and 27,3% for lifetime prevalence respectively.

In the age groups 18 – 29, 30 – 39, 40 – 49 women show a significantly higher prevalence of LBP; in the age groups older than 60 years women also have higher,

but not significantly higher prevalence. Compared to the results of *Schmidt (2007)* Neuhauser finds lower prevalence in all categories. This might be traced back to the fact that the subjects are more involved (when filling in questionnaires) in *Schmidt's (2007)*, than when they are only interrogated.

The study of Leboeuf- Yde (1996) investigates the LBP prevalence of the Nordic population using data from a (in those days) recent study as well as data from two older Swedish, one Danish and an Icelandic survey (Svensson (1982); Svensson (1988); Raffnson (1982); Biering-Sørensen (1982)).

The 1-year prevalence in this study is reported to range between 44-65%, the lifetime prevalence between 60-65%.

The review of *Louw (2007)* concentrates on the prevalence of LBP in Africa. A total of 27 epidemiological studies are included (covering 32.059 individuals), the majority of those, conducted in South Africa and Nigeria in urban populations are assessing adolescents and adults.

The general assumption, that there is a comparatively lower LBP prevalence in Africa than in the developed countries, is contradicted for the findings of this review are similar to those quoted below (*Walker, 2000*).

Point prevalence in adults ranges between 16% and 59% with a mean of 32%, 1-year prevalence between 14% and 72% (mean 50%), and a lifetime prevalence of 28% to 74% (mean 62%)

In a systematic review *Walker (2000)* analyses 56 population studies of low back pain. Out of those 56 studies he identifies thirty of acceptable quality.

Walker concludes, that point prevalence of low back pain ranges from 12 -33%, 1 -year prevalence from 22-65% and lifetime prevalence from 11-84%.

Walker's (2000) study can act as a summary of the data collected in the last centuries. Through its comprehensive extent (reviewing 56 studies) the range of LBP

prevalence seems to be wide spread in *Walker's (2000)* results. This is surely caused by different study designs and different definitions of LBP.

The results of the studies quoted above (concentrating on Germany and the Nordic countries (*Schmidt (2007); Neuhauser (2005); Leboeuf –Yde (1996)*)) fit very well into the range described by Walker (2000). So I would assume that the prevalence of LBP and CLBP in Austria will be quite at the same level.

2.3 Causes of CLBP

Causes of CLBP are manifold (listed in table 1), *Deyo (2007)* summarizes:

“Experimental studies suggest that LBP may originate from many spinal structures, including ligaments, facet joints, the vertebral periosteum, the paravertebral musculature and fascia, blood vessels, the annulus fibrosus, and spinal nerve roots. Perhaps most common are musculoligamentous injuries and age-related degenerative processes in the intervertebral discs and facet joints. Other common problems are spinal stenosis and disc herniation.” (*Deyo, 2001, page 363*)

Even though imaging techniques like MRI are very accurate in finding alterations in the patient's spine, *Deyo (2001)* assumes that approximately 80% of patients with LBP cannot be given a precise diagnosis, for LBP symptoms, pathology and radiological findings are poorly correlated.

As well as *Deyo (2001)* *Nachemson (1992)* criticises the low specificity of the tests applied to patients with LBP.

Table 1 Differential diagnosis of LBP

Mechanical LBP or leg pain (97%)	Nonmechanical spinal conditions (1%)	Visceral disease (2%)
Lumbar strain, sprain(70%) *	Neoplasia (0.7%)	Disease of pelvic organs
Degenerative process of disks and facets (10%)	Multiple myeloma	Prostatitis
Herniated disc (4%)	Metastatic carcinoma	Endometriosis
Spinal stenosis (3%)	Lymphoma and leukaemia	Chronic pelvic inflammatory disease
Osteoporotic compression fracture (4%)	Spinal cord tumors	Renal disease
Spondylolisthesis (2%)	Retroperitoneal tumors	Nephrolithiasis
Traumatic fracture (<1%)	Primary vertebral tumors	Pyelonephritis
Congenital diseases (<1%)	Infections (0.01%)	Perinephric abscess
Severe Kyphosis	Osteomyelitis	Aortic aneurysma
Severe Scoliosis	Septic diskitis	Gastrointestinal disease
Transitional vertebrae	Paraspinous abscess	Pancreatitis
Spondylosis	Epidural abscess	Cholecystitis
Internal disk disruption or diskogenic LBP	Inflammatory arthritis	Penetrating ulcer
Presumed instability	Ankylosing spondylitis	
	Psoriatic spondylitis	
	Reiter's syndrome	
	Inflammatory bowel disease	
	Scheuermann's disease	
	Paget's disease of bone	

* "strain" and "sprain" are nonspecific terms with no pathoanatomical confirmation. "Idiopathic LBP" may be a preferable term.

Table 1 adapted from Deyo (2001)

Table 1 charts *Deyo's (2001)* assumption that the vast majority of LBP (97%) is caused by mechanical problems. Out of these 97% *Deyo (2001)* states that around 70% are idiopathic, but he also lists 15 conditions that might also lead to LBP. The second and third column of the table lists non-mechanical spinal conditions, possibly leading to LBP.

It is discouraging for patients, who suffer from LBP, to have their pain classified as "unspecific", as they are longing for a precise explanation for their situation. (*McPhillips-Tangum, 1998*) This leads to elusive terms such as "lumbago" or "sciatica" - avoiding medical diagnoses- but still giving the patient the feeling that the examiner understood his or her problem. (*Deyo, 2009*)

Patients with LBP who underwent x-ray or MR Imaging could easily be diagnosed with a bulging disc, osteoarthritis of the facet joints or spinal stenosis, blaming those radiological findings to be the reason for their pain.

But this conclusion falls short, given the results of the studies quoted below:

The results of *Jensen's MC (1994)* study require questioning the current morphological-based understanding of the pathogenesis of LBP.

On 98 asymptomatic people MRI examination is performed and is read by two neuroradiologists, who do not know the clinical status of the subjects, classifying their findings into normal/bulge/protrusion or extrusion of the five lumbar discs.

Additionally 27 abnormal MRI scans from people with LBP are mixed randomly with the scans from the asymptomatic people. Out of the 98-person sample group only 36% have normal disks at all levels, while 52% have a bulging at least at one level and 38% have an abnormality of more than one intervertebral disk.

27% have a protrusion and 1% even an extrusion. The prevalence of bulges increases with the subject's age. Anular defects are detected in 14% and facet arthropathy in 8%. Given the high percentage of bulgings found in asymptomatic people, the discovery of bulgings or protrusions in LBP patients may often be coincidental.

Weishaupt D (1998) does a very similar study on 60 asymptomatic volunteers, including only individuals who had never experienced "relevant LBP". From the 30 male and 30 female subjects -aged between 20 and 50- MRI scans are performed and analyzed independently by two musculoskeletal radiologists. The findings concerning the disks are classified as: normal/bulging/protrusion/extrusion and sequestration. In regard to osteoarthritis the facet joints are evaluated as normal/mild degenerative disease/moderate degenerative disease and severe degenerative disease. In 43 of the 60 subjects (72%) the examiners find advanced-stage intervertebral disk degeneration. In 13 (22%) of the subjects nerve root contact is detected, in two cases even with nerve root deviation. As far as osteoarthritis is concerned, 11 – 18 % (interrater differences) have mild degenerative diseases and 3 – 8 % have moderate degenerative diseases.

An article recently published in *Lancet* by *Chou R. (2009)* reports new results of a meta-analyzes about imaging strategies in LBP. The team of Chou compares immediate, routine lumbar imaging (or routine provision of imaging findings) of patients suffering from LBP versus usual clinical care without immediate lumbar imaging (or not routinely providing results of imaging). The results reveal that there is no significant difference in the outcome parameters (pain, function, quality of life)

between those patients being routinely imaged and those not being imaged. This is reported of both short and long term effects.

Chou (2009) states:

"In addition to lack of clinical benefit, lumbar imaging is associated with radiation exposure (radiography and CT), may not affect diagnostic or treatment plans, increases direct costs and may lead to increased use of expensive but potentially unnecessary invasive procedures." (Chou, 2009, page 472)

Given the findings of the above quoted studies, it is coherent, that the authors of the European Guidelines conclude "not to recommend radiographic imaging (plain radiography, CT or MRI, bone scanning, discography or facet nerve blocks for the diagnosis of non-specific Chronic LBP unless a specific cause is strongly suspected" (EGCLBP, 2004, page 5)

Nevertheless the former quoted Deyo R.A. (2009) summarizes the situation in the United States:

"Despite guidelines recommending parsimonious imaging, use of lumbar magnetic resonance imaging (MRI) increased in the Medicare population by 307% during a recent 12-year interval...Spine imaging rates vary dramatically across geographic regions, and surgery rates are highest where imaging rates are highest. When judged against guidelines, one-third to two-thirds of spinal computed tomography imaging and MRI may be inappropriate." (Deyo, 2009, page 62)

Deyo (2009) describes the situation in the US, but my daily routine suggests that the situation might be the same in Austria.

All the studies quoted above should lead to the question, if the morphology-based understanding of the pathogenesis of LBP is really incontrovertible. The poor correlation between radiological findings and the patients' symptoms suggest that there might be some other reason for LBP symptoms (Jensen (1994), Weishaupt (1998), Beattie (2009), Luoma (2000), Kjaer (2005), Waris (2007)) something that cannot be visualized by MRI.

The Fascial Distortion Model (FDM) offers a different approach to this problem. The FDM states that it is the fascias being distorted, causing the pain, and not some kind of degeneration of discs or bones (see chapter 3).

2.4 Treatment of CLBP

Given the manifold reasons for CLBP (listed in chapter 2.3) there are many different approaches for treatment.

Besides physical treatments (as for example interferential therapy, laser therapy, shortwave diathermy, therapeutic ultrasound) pharmacological interventions (such as NSAIDs, Muscle relaxants, Opioids, Antidepressants, Antiepileptic drugs, Capsicum pain-plasters) and invasive procedures (Injections and nerve blocks, Acupuncture) there are also manual approaches such as Traction, Mobilisation, spinal Manipulation and Massage. (*EGMCLBP, 2004*)

In Austria the appliance of both pharmacological interventions and invasive procedures is only accessible for medical doctors but not for osteopaths (most Austrian osteopaths are physiotherapists but not medical doctors). This is why I am concentrating on the manual approaches to CLBP such as traction, mobilisation and spinal manipulation. I will provide a short summary of the efficacy of those techniques. Furthermore this paper presents three studies exploring osteopathic treatments.

2.4.1 Traction

Traction is a manual approach to hypomobile joints, separating the two bones of the joint. (*Kaltenborn, 1992*)

“The exact mechanism through which traction might be effective is unclear. It has been suggested that spinal elongation, through decreasing lordosis and increasing intervertebral space, inhibits nociceptive impulses, improves mobility, decreases mechanical stress, reduces muscle spasm or spinal nerve compression (caused by

osteophytes) releases luxation of a disc or capsule from the zygo-apophysial joint, and releases adhesions around the zygo-apophysial joint and anulus fibrosus.”

(Clark J. 2006, p 1592)

Clarke J. (2006) does an updated systematic review on the efficacy of traction for LBP. 24 randomized controlled trials with a total of 2.177 patients are included. The study includes both patients with and without sciatica, traction (intermittent or continuous) being applied mechanically or by motorized force, manual traction and autotraction. As far as short or long-term outcomes in the mixed group of patients (with or without sciatica) are concerned, Clark does not find statistically significant difference between traction and sham, placebo or no treatment.

The authors of the European Guidelines came to the same conclusion: they cannot find any differences between traction and sham-traction procedure, which is why traction as a treatment for CLBP is not recommended.

(EGCLBP, 2004, page 15)

2.4.2 Spinal Manipulative Therapy and Mobilisation for CLBP

Spinal mobilisation is defined as a high velocity thrust to a joint beyond its restricted range of motion while spinal mobilisation is characterized by repetitive low velocity passive movements within or at the limit of joint range (or carried out constantly as a stretching).

The arguments for the application of spinal manipulation are manifold (Koes B; 1996)

- Reduction of a bulging disc
- Correction of the internal displacement of disc fragments
- Freeing of adhesions around a prolapsed disc or facet joints
- Inhibition of transmission of nociceptive impulses
- Relaxation of entrapped synovial folds or plica
- Relaxation of hypertonic muscles by sudden stretching

Because of the enormous quantity of studies evaluating the efficacy of spinal manipulative therapy I am going to present three review articles on spinal

manipulation (not looking on special osteopathic methods) and mobilisation in chronological order:

Koes (1996) includes 36 different randomized controlled trials in his systematic review. Eight out of those 36 studies concentrate on chronic low back pain only, comparing spinal manipulation with other conservative treatments such as general practitioner's care, physiotherapeutic interventions, back school and analgetics. five out of eight studies report positive results, two report negative results and the remaining study present no result.

In comparison to the 28 studies, which concentrated on acute or subacute low back pain, these results suggest that there is more evidence in favour of manipulation for more chronic conditions.

Koes arrives at the conclusion that "unfortunately, to date, we have not been able to identify the patients in whom manipulation might be most beneficial." (*Koes, 1996, p 2867*)

Seven years later *Bronfort (2003)* does a systematic review on the same topic, identifying 46 randomized controlled trials, 11 out of which investigated chronic low back pain. Bronfort's results suggest that there is moderate evidence that spinal manipulation and mobilisation is superior to general medical care and to placebo in short term, and superior to physical therapy in the long term for patient improvement. He also finds moderate evidence that spinal manipulation in combination with strengthening exercises is similar in effect to prescription NSAIDs with exercises for pain relief in both the short and long term.

As far as reducing disability in the long term is concerned there is moderate evidence that spinal manipulation is superior to physical therapy and to home exercise.

On the other hand it is found that there is only limited evidence that spinal manipulation is superior to sham manipulation in the short term.

Bronfort concludes that the use of spinal manipulation and/or mobilisation is a viable option for the treatment of LBP.

Another four years later *Assendelft (2007)* publishes a systematic review for the Cochrane Library including 39 studies, 14 out of those on patients with "unsure duration" of pain. Assendelft reports the following outcome measures: Level of pain in

the individual patients (expressed on VAS), functional status (expressed on back pain- specific scales, such as Oswestry Disability Index or Roland Morris Disability Questionnaire), as well as short and long term duration of effects. This is of special interest to me, because I also chose VAS and ODI for my study, which improves the comparability of my data.

Assendelft's (2007) data give only sparse support for spinal manipulation, both for acute and chronic low back pain. For chronic low back pain it is statistically significant that spinal manipulation is superior to sham manipulation, showing improvement of 10mm in short-term pain and improvement of long-term pain of 19mm and improvement of short-term function by 3.3 points on the RMDQ. Furthermore spinal manipulation is superior compared with groups of therapies judged to be ineffective or perhaps harmful (such as traction, corset, bed rest, home care, no treatment, diathermy).

Assendelft sees no evidence that spinal manipulation is superior to other standard treatments for patients with CLBP.

Summarizing these results the authors of the European Guidelines see:

- Strong evidence that manipulation and general practitioner's care are of similar effectiveness in the treatment CLBP
- Moderate evidence that spinal manipulation in addition to general practitioner's care is more effective than GP's care alone
- Moderate evidence that spinal manipulation is superior to sham manipulation (EGMCLB, 2004)

2.4.3 Osteopathic treatment for CLBP

Given the sparse scientific support for sole application of spinal manipulation the question raises how to combine HVLA techniques with other manual techniques to improve the treatment's outcome.

Following Dr. Arthur *Still's* (1910) second principle (of the human body being a unit in which no part can work independently) Osteopathy has a more holistic approach to CLBP than allopathy. Therefore an osteopathic treatment for CLBP problems will not only imply manual techniques on a structural level, but also on a visceral and cranio-

sacral level. As osteopathic treatment is always based on the examiners/osteopaths findings and therefore varies individually for each and every patient, there are no general guidelines for the treatment of low back pain.

In his masterthesis *Seifner (2006)* summarizes the osteopathic techniques that he used for the treatment of herniated lumbar discs:

- Thrust techniques for the thoracic spine and the thoraco-lumbar junction (foremost C7, Th4, Th6, Th9, Th10, Th12 – L1)
- Correction of the ilia and/or sacrum
- HVLA Correction of hip and ankle
- AORT Techniques for triggerpoints in the lumbar spine and pelvic region
- FDM Techniques
- Visceral techniques:
 - Correction of too low kidneys
 - Correction of diaphragmatic tensions
- Cranio-sacral techniques:
 - Membranous Balances Tension (MBT) of the sacrum
 - CV4
 - Synchronisation between occiput and sacrum

(Seifner, 2006 p 36)

Using these techniques *Seifner (2006)* compares osteopathic treatment of lumbar herniated discs with physiotherapeutic treatment. 13 patients are treated osteopathically, 9 patients are in the control group, receiving physiotherapeutic treatment. Seifner investigates the duration of sick leave, the number of treatments necessary, duration of treatment, change in pain, change in finger-floor distance (flexion, and sidebending left and right), change of proprioceptive skills and change of SLR Test in both the test and the control group.

In order to compare *Seifner's* results with the results of my study, I would like to present his outcomes concerning change in pain and change in finger-floor distance (flexion) above all. Concerning the change in pain the test group performs much better than the control group: in 85% of the test group pain can be reduced by 4 to 5 points on a VAS (with 6 degrees), while only 20% of the control group reach such advancement, with 10% of the control group reporting no advancement at all.

Concerning the finger-floor distance (flexion) *Seifner's* results are not in favour for osteopathic treatment: while the control group reduces the finger-floor distance by 29,44 cm (mean) the test group shows only advancement of 3,75 cm (mean).

Seifner's explanation for this is, that the test group already had better ratings in this test prior to the treatment, and so they could not reduce the distance to the extent that was reached in the control group.

While *Seifner's* study concentrates on patients with disc herniation only, the study of *Adorjan et al. (1999)* examines chronic LBP patients with various diagnoses. Their inclusion criteria is "a medical proofed diagnosis of low back pain, VAS score more than 30mm, and a Roland Morris Test score of more than 5".

A total of 57 patients fit in the inclusion/exclusion criteria and are divided in a test group consisting of 29 patients and control group (28 patients). Their modus operandi is: a pre-treatment VAS and Roland Morris questionnaire serve as a starting basis which is followed by 5 treatments at intervals of 15 days. Another 15 days later a final VAS and Roland Morris questionnaire has to be answered.

The test group is treated with 8 predetermined osteopathic techniques for the following five regions: thoraco-lumbar junction, Sacro-iliac joints, os coccygis, psoas and duodenum/colon, while the control group receives sham treatment by being tested only.

Concerning quality of life (Roland Morris questionnaire) the test group shows a consistent bettering, which is statistically significant higher than in the control group. From an almost equal mean of 8.7 (test group) respectively 9.1 (control group) of marked answers (out of 24 questions), the test group improves to only 3, while the control group came only to a mean of 6.7.

Concerning pain the test group also performs better: they reduce their arithmetic mean of 63mm VAS to 18 mm VAS, while the control group improves only slightly from 55mm to 52mm VAS. This result is also statistically significant.

Licciardone (2003) does a randomized controlled trial on patients with chronic non-specific LBP. 91 subjects, having had constant or intermittent CLBP in the last three months, are randomized into a test group (osteopathic manipulative therapy –OMT), a control group treated with sham manipulation and a group receiving no treatment.

The baseline assessment consisted of a Short Form-36 Survey (SF36), a 10 cm VAS and a Roland Morris Questionnaire.

Subjects of OMT and sham manipulation group are treated seven times within five months: first treatment one week after the baseline assessment, second another week later, third treatment one month after baseline assessment and the following treatments monthly thereafter.

Subjects of the OMT group are treated with one or a combination of the following techniques: HVLA thrusts, myofascial release, strain-counterstrain, muscle energy and soft tissue techniques and cranio-sacral therapy.

All subjects, regardless of which randomized group, are allowed to receive other low back care (but no OMT or chiropractic treatment for sham- respectively no-treatment group) to complement the trial interventions.

New data of SF-36; VAS and RM-Questionnaire are collected at one/three and six months.

The results of the SF-36 are statistically different over the time: at one month the OMT group reports more improvement than the no-treatment group. At three and six months it is the other way round with the sham-treatment group reporting more improvement.

Concerning the results of the VAS, both the OMT and the sham-treatment group show greater improvement than the no-treatment group.

In the Roland Morris Disability scores no significant differences can be found over time among the treatment groups.

So Licciardone concludes that both OMT and sham manipulation have some benefits when used in addition to usual care for CLBP.

3. The Fascial Distortion Model (FDM)

Due to the fact that the FDM is a rather new concept of explaining musculoskeletal dysfunctions, the publications of Stephen Typaldos the founder of the FDM, are still of fundamental importance.

Therefore this chapter is giving a very brief compendium of Typaldos' work, using and quoting his book "Clinical and Theoretical Application of the Fascial Distortion Model within the Practice of Medicine and Surgery".

Stephen Typaldos described his model as follows:

"The Fascial Distortion Model (FDM) is an anatomical perspective in which the underlying etiology of virtually every musculoskeletal injury (.....) is considered to be comprised of one or more of six specific pathological alterations of the body's connecting tissues (fascial bands, ligaments, tendons, retinacula, etc.)"

(Typaldos, 2002, p 3)

In the modern allopathic medicine destruction of structures (as in e.g. osteoarthritis, or a sprained ankle) and the accompanying inflammation are said to be the major generator of pain. In the FDM on the other side the distortions of the fascia are proposed to be the determining factor (and sensor) of pain.

(Typaldos, 2002, p 14)

Daily routine shows that patients' verbal expressions in the medical history describing "their" pain are very often accompanied by a certain body language. In my experience this body language is given only little attention both in allopathic and in osteopathic medicine. At least in three years of physiotherapy school and six years of osteopathic training I was neither advised to monitor the patient's body language nor taught how to interpret it correctly. In the FDM on the other hand the body language of the patient is vital, because the automatic and involuntary signs of body language help the therapist to detect which fascial distortions (or combinations of different fascial distortions) are causal for the pain. Each and every fascial distortion presents with a distinct kind of body language. (Typaldos, 2002)

3.1 Definition, function and dysfunction of fascia

Fascia as the primary connective tissue- is a three dimensional sheet of firm tissue, that is spreading across our body from head to toe. It presents in many different forms such as tendons, ligaments, retinacula, aponeuroses, fascial bands perimysium and epimysium of muscle fibers in our musculoskeletal system and as pericardial sac, pleura, meninges and many other structures in our visceral and cranial system. (Typaldos, 2002)

Typaldos (2002) states that fascia not only connects but surrounds, engulfs, encases, separates, compartmentalizes, divides, protects, insulates and buffers bones, nerves and muscles. It invests and sheaths every muscle fibre, muscle bundle, entire muscles as well as every group of muscles. Furthermore it invests bone, nerve, blood vessel and organ of our body.

Due to the fact that fascial tissue has to fulfil so many different functions (as listed above) different anatomical specifications of fascia are present in the body.

(Typaldos, 2002, p 9)

Typaldos (2002) differentiates four primary kinds of structural fascia: banded fascia, coiled fascia, folding fascia and smooth fascia.

Banded fascia is found in ligaments, tendons and aponeuroses, like the iliotibial tract. The main function of banded fascia is to protect joints and linear regions of trunk and limbs, blood vessels and tissues from perpendicular forces. Banded fascia can be affected with triggerbands and continuum distortions. (see Chapter 3.2.1 resp. 3.2.2)

Coiled fascia encircles entire portions of limbs, trunk, back, vessels and organs. The main function of coiled fascia is to protect non-jointed tissue from traction or compression forces. Coiled fascia can be affected by cylinder distortions. (see Chapter 3.2.5)

Folding fascia, which is present in capsules, intermuscular septa as well as interosseous membranes, protects joints from traction and compression forces.

Folding fascia can be affected by folding distortions (see chapter 3.2.3)

The fourth structural kind of fascia described by Typaldos is the **smooth** fascia. It lines joints, abdomen, viscera and makes up planes of non-folding fascial tissue. The

function of smooth fascia is to keep joints and tissues lubricated, in order to allow good gliding of one fascial structure on another.

(Typaldos,2002, p 9)

Following Typaldos (2002) every human being presents an individual percentage of those different types of fascia in the body, enabling and supporting the individual's daily routine. (In this connection Typaldos differentiates e.g. the bodies of weight lifters [higher percentage of banded fascia] and the bodies of ballet dancers [higher percentage of folded fascia])

(Typaldos,2002, p 9/10)

Typaldos (2002) argues that fascia - as being a living structure- is in the need of oxygen and nutrients to sustain itself, as well as a system of removal of waste products. In addition to the different functions of fascia (as listed above) in the FDM, fascia is also seen as fluid transport network, feeding its adjacencies with oxygen, hormones, minerals and nutrients respectively removing waste products. Hence, injuries to the fascia will lead to disrupted fluid flow to the downstream network of fascia, as well as a discontinuance of the upstream flow.

(Typaldos, 2002, p 10)

Besides the lesions caused by injuries to the fascial system itself, fascia can also be affected by viral or bacterial infections clogging its fluid transportation. In the case of malnutrition fascia becomes more easily affected by even minor external forces.

Typaldos (2002) states e.g. that the myalgia accompanying viral influenza, is caused by cylinder distortions (see 3.2.5), due to disrupted fascial fluid flow.

Summarizing Typaldos' (2002) model it can be said that malfunction of the fascial system due to injury, inflammation or posture may lead to an abnormal high pressure on all the covered, invested respectively fed structures. This results in pain, dysfunction as well as strange side effects and symptoms which often cannot be matched with an accurate allopathic diagnosis.

3.2 Principal types of Fascial Distortions

Typaldos (2002) found six different types of fascial distortions, which will be described in the following chapters. In addition to Typaldos' explanation of the particular distortion (mostly quoted from the FDM Textbook) I will provide a summary about that very distortion in the lumbar area, as far as possible locations, the patient's body language and verbal description in the medical history are concerned.

3.2.1 Triggerbands (TB)

Quoting Typaldos (2002) triggerbands are

“anatomical injuries to banded fascial tissues in which the fibers have become distorted (i.e., twisted, separated, torn, or wrinkled).”

The verbal description associated with triggerbands is a “burning” or “pulling” pain along a linear course. The accompanying body language is a sweeping motion with one or more fingers along triggerband pathway.

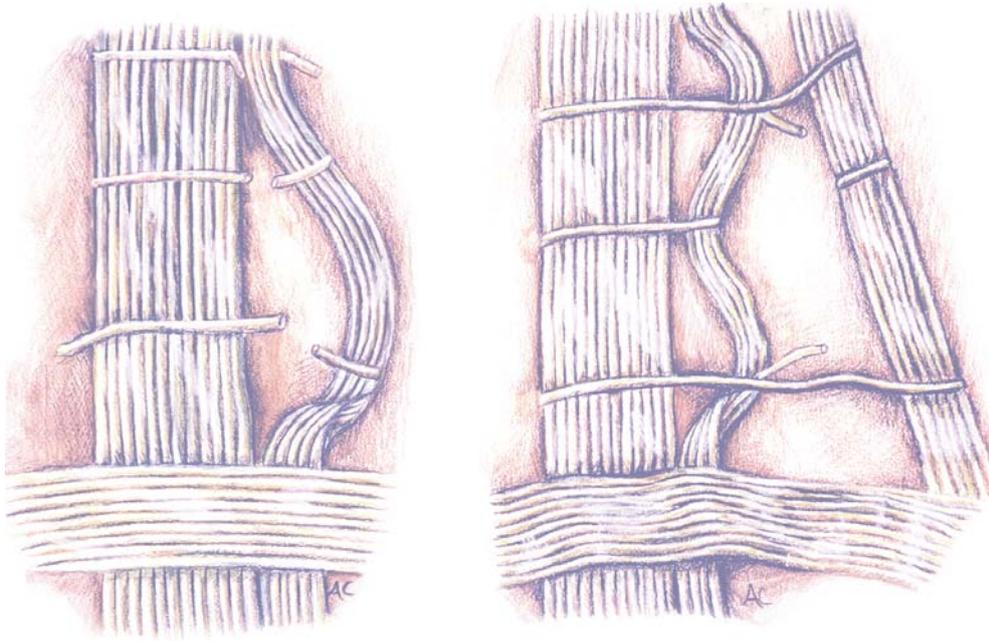
(Typaldos, 2002)

The body language directs the corrective treatment (the so called triggerband technique) specifically to the distorted fibers of the afflicted ligament, fascial band or tendon.

(Typaldos, 2002)

“The goal of the treatment is to physically break fascial adhesions (if the injury is chronic), untwist the distorted band or sub-bands (individual fibers of the band), and reapproximate the torn fibers. In essence, triggerband technique is accomplished by ironing out the wrinkled fascia with the physician's thumb. And although there are several subtypes all triggerbands are treated the same way, and that is with triggerband technique. “

(Typaldos, 2002)



In an acute triggerband (left) crosslinks have been fractured and some sub-bands (individual fibers) have twisted apart. Note that crossbands stop the fibers from tearing indefinitely and are the starting point for triggerband technique. If the torn crosslinks heal by attaching to structures other than their appropriate counterparts (right) they are called fascial adhesions and the injury is considered to be chronic.

Picture 1, Triggerbands

As quoted above the adequate FDM technique for the treatment is the Triggerband technique. The therapist determines the very triggerband, palpates its starting point and “irons out” the fascial band with firm force of his thumb, ending up at the anatomical structure that fixes the fascia (e.g. coccyx, Mastoid process...)

In the lumbar region there are three major Triggerbands to be found: the Posterior thigh triggerband, the Lateral thigh triggerband and the Paravertebral triggerband.

The **Posterior thigh triggerband** starts (respectively ends) at the sacrococcygeal junction, goes upward past the sacral base to the lumbar transverse processes, where it turns laterally in a small bow, past the iliac crest and then downwards posterior on the thigh until it reaches the groove between medial and lateral part of M. soleus.

The **Lateral thigh triggerband**, starts (ends) also at the sacrococcygeal junction, goes up to the sacral base, to the lumbar transverse processes and then veering laterally in a bigger bow downwards the lateral thigh ending at the lateral tibia, close to the tuberosity of Gerdy.

The third triggerband, the **Paravertebral triggerband**, starts (ends) at the sacrococcygeal junction, going cranially, with a slight veering laterally at the sacral base, then goes upwards paravertebral approximately to the dorso-lumbar junction (or even the mastoid process in hypermobile individuals) (Typaldos, 2002)

3.2.2. Herniated Triggerpoints (HTP)

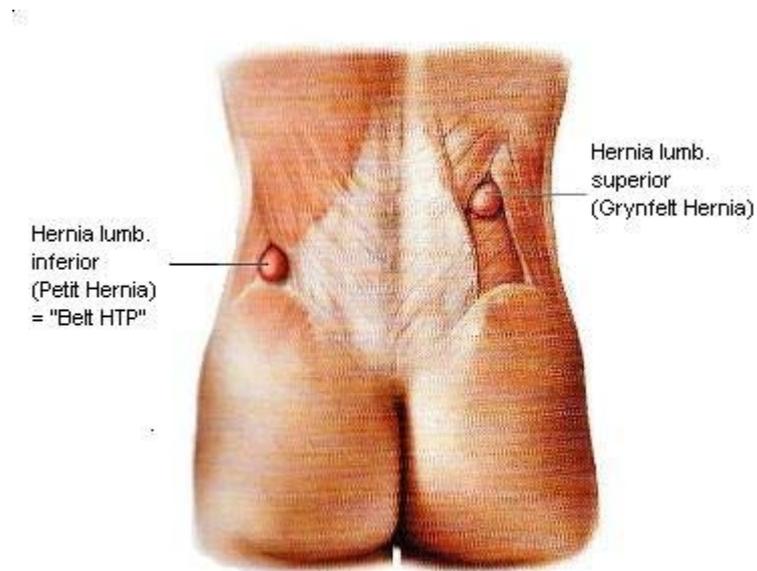
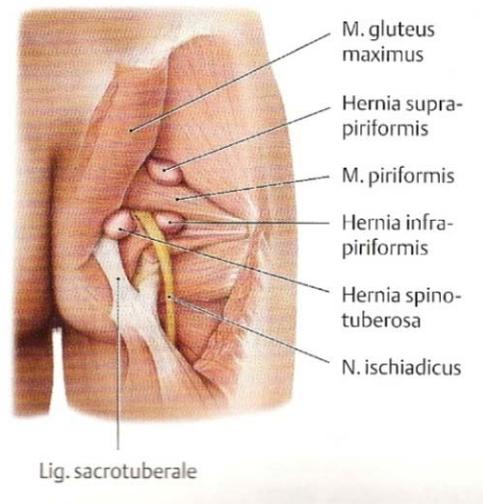
Typaldos (2002) defines Herniated triggerpoints as fascial distortions in which ***“underlying tissue has protruded through an adjacent fascial plane and has become entrapped.”***

Following Typaldos (2002) injuries of that kind are responsible for a wide range of painful complaints such as sore shoulders, neck aches, abdominal pain, renal colic pain and lumbar strains.

(Typaldos, 2002)

The corresponding body language with a HTP is that the patient pushes his or her fingers directly deep into the painful area. The FDM intervention (triggerpoint technique) is doing the very same: it forces the protruded and entrapped tissue back through the herniated fascial plane.

The most common herniated triggerpoints are the “Bull’s eye HTP”, located deep in the gluteal muscles and the “Belt HTP” above the Crista iliaca. A third common HTP is in the Grynfelt - triangle between the 12th rib and the spine.



Picture 2 and 3, „Bull’s eye HTPs” respectively lumbar HTPs, adapted from Prometheus 2005

3.2.3 Continuum Distortion (CD)

With the term Continuum Distortion Typaldos (2002) describes a lesion at the so called transition zone (TZ) between ligament (or tendon, capsule) and bone. Interesting what Typaldos (2002) states about osseous and ligamentous structures in our body:

“In the fascial distortion model, ligament and bone are envisioned as two opposite ends of one anatomical spectrum both structures are seen as merely compositional forms of each other. Bone is therefore a fascial tissue with a large percentage of osseous material, while ligament is a fascial tissue with minimal bony products.”

(Typaldos, 2002, p 31)

Following Typaldos (2002) the tissue of the transition zone has osseous as well as ligamentous physical properties.

Even more he states that the transition zone has the physiological capability to alter the percentage of its osseous components (by shifting bony components in or out), creating a “harder” respectively “softer” tissue. This shifting gives the ligament/bone unit the capacity to respond to external forces and by doing so protects the tissue from potential injuries, such as fractures or ligamentous tears.

(Typaldos, 2002)

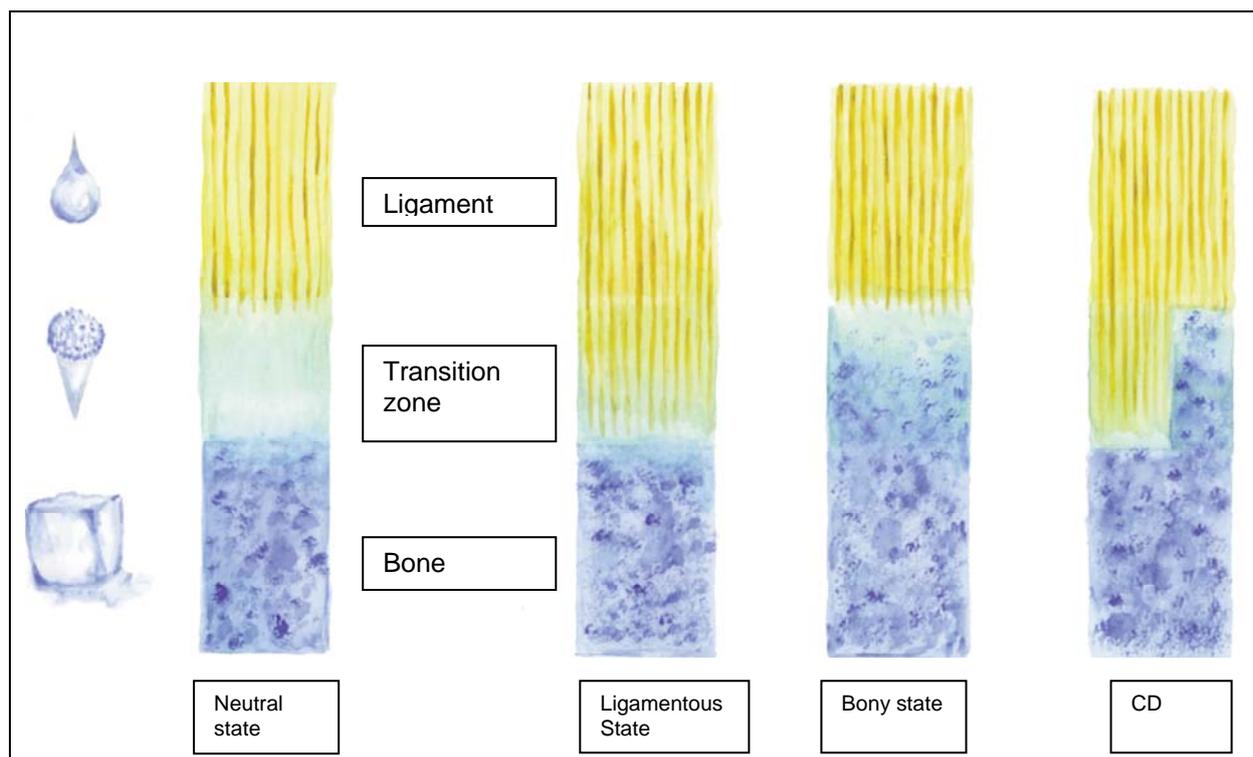


Diagram 1, Continuum distortion

In the case of a Continuum distortion the transition zone has lost its capability to shift between bony and ligamentous properties. This can occur *“when a portion of the transition zone is subjected to a unidirectional force at the same time as another portion of the same zone encounters a multidirectional force. The result is that the*

transition zone splits its identity — one part becomes osseous and the other ligamental.”

(Typaldos, 2002, p 32)

This imbalance in the transition zone disrupts the mechanical function of the ligament (for some fibres are stiff while others are flexible) and also harms its proprioception, transmitting uneven mechanical information to the cerebellum. This uneven information will then be interpreted as pain, located in the very spot of the transition zone.

Typaldos (2002) differentiates between two subtypes of CDs: the “**Everted Continuums Distortion**” (ECD, in which a portion of the transition zone is stuck in osseous configuration) and the “**Inverted Continuum Distortion**” (ICD, with a portion of the transition zone stuck in ligamentous configuration), both of which are treated with the continuum distortion technique: Pressure through the therapist’s thumb firmly and continuously applied directly in the continuum distortion forces the transition zone to shift.

As an alternative to the continuum technique in the case of an inverted continuum distortion Typaldos (2002) suggests to do a thrust manipulation. By the thrust tugging on the bony matrix, osseous components are being pulled back in the transition zone. Especially with CDs in the sacroiliac joint, the “scissors technique” (thrust manipulation on the SI-joint) is very effective.

(Typaldos, 2002)

The body language with CDs is distinct: one finger points to the spot(s) of pain. In the lumbar region there CDs are common in the region of the sacroiliac joint, on the spinal processes and on the coccyx.

3.2.4. Folding Distortions

The fourth described principal fascial distortion are folding distortions. Typaldos (2002) describes folding distortions as follows:

“When fascia in or around a joint becomes distorted from either traction or compression forces, this is called a folding distortion. These three-dimensional

injuries of the fascial plane hurt deep within the joint and diminish the ability of the fascia to protect against pulling or pushing injuries. Within the FDM there are two subtypes of folding distortions —unfolding and refolding. Unfolding distortions occur when a pulling and twisting force is introduced into a joint and the fascia unfolds, torques, and refolds contorted. The main structural ramification of this injury is that the fascia can't refold completely. Refolding injuries, in contrast, occur when the fascia becomes jammed or compressed onto itself and then can't unfold completely. “ (Typaldos, 2002, p 37)

Typaldos (2002) states that patients with folding distortions in the lumbar region will complain about feeling a pain deep inside, having the desire to be stretched or compressed (unfolding res. refolding). The body language often seen is that the patient pushes his/her fist into the lumbar region while they are extending or bending their spine.

Distinguishing between Unfolding and Refolding distortion in the lumbar spine is not always easily done. Typaldos (2002) suggests the following procedure: Foremost it is essential to know the mechanism of injury from the case history: have there been stretching or compressing forces, causing the problems? If this question cannot be answered satisfactorily it is helpful to find out which translation reduces or exacerbates pain: traction will be comfortable for the patient with Unfolding distortion but will aggravate symptoms for refolding distortion. Vice versa it is with compression. If the mere application of smooth traction or compression cannot help distinguishing between the two types (none of both considerably reduces or aggravates symptoms) the next step would be to try a thrust manipulation either with traction or compression (= unfolding technique res. refolding technique). Pain caused by an unfolding distortion will ease when unfolding techniques being applied, and just the other way around with refolding techniques. (Typaldos, 2002)

Typaldos (2002) finds that in the lumbar region, it is often the case that both unfolding and refolding distortions occur in the very same individual, caused by concurrent traction and compression forces. In that case Typaldos (2002) suggests to

apply both refolding and unfolding techniques, refolding first followed by unfolding. (Typaldos, 2002)

Folding techniques of any kind should not be painful! If there is pain both with unfolding and refolding it is often caused by another fascial distortion (e.g. triggerbands). In those cases the other fascial distortions have to be corrected first.

With the lumbar region the unfolding res. refolding technique of choice will be the “chair technique”.

Typaldos (2002) describes the chair technique for unfolding:

“In this procedure, patient sits backwards on a chair (i.e., straddles) so that he/she is facing towards the wall. The feet are tucked inside the legs of the chair that are closest to the wall and forearms are crossed so that each hand holds onto the opposite shoulder. To make the correction, physician stands behind patient and reaches around with nonthrusting hand and grips one or both elbows. Palm of thrusting hand is placed over the transverse process and paravertebral fascia of area to be manipulated. Simultaneously, both hands of the physician are used to traction and extend the spine. Once traction is maximized, the spine is rotated until the physiological barrier is reached. (When treating the right lumbar spine physician’s right hand is thrusting hand.) Once the physiological barrier is engaged, a quick lateral and superiorly directed thrust is made by the palm of the treating hand.”

(Typaldos, 2002, p 44)

For refolding the technique is similar, only the vectors of the thrust differ.

In the very same sitting position as described above, the therapist applies a thrust combining rotational and compressive forces. The compression is delivered through shoulder, arms and hands of the therapist.

3.2.5 Cylinder Distortions

The so called Cylinder distortions as the fifth principal fascial distortion affects the coiled fascia, which cylindrically encircles the extremities, trunk and back.

The affected coils of circular fascia are tangled and so act a tourniquet around muscles or other tissues. This entangling of coils inhibits the ability to uncoil and recoil and so diminishes the resilience to absorb pushing and pulling forces.

Cylinder distortions, which are located quite superficial paradoxically, can create deep pain in a non-jointed area.

(Typaldos, 2002)

Following Typaldos (2002) Cylinder distortions have the propensity to exhibit seemingly bizarre symptoms, resembling neurological conditions such as tingling, numbness or muscle cramps. Furthermore patients complain about a pain wandering or jumping from one area to another. This is because the coils of fascia are tangled in varying arrangement whilst movements, depending on the sequence of muscle contractions.

(Typaldos, 2002)

The body language of patients complaining about that sort of pain will be a repetitive squeezing of the affected soft tissue.

The whole lumbar spine area as well as the dorsal thigh can be affected by cylinder distortions.

In order to treat cylinder distortions some cylinder treatments can be applied: **double thumb technique** (stretching or compressing the affected tissue with both thumbs), **Indian burn** (stretching plus rotating the affected area), "**Squeegee-technique**" (capable foremost for lesion on the limbs: the therapist wraps his hands around the affected area and slowly slides his hands along the limb, always maintaining a squeezing tension. Another possibility is to use **cupping**. Especially when cupping is used while the affected area is doing movements the tangled circular coils of fascia are very well being separated from one another.

(Typaldos, 2002)

3.2.6 Tectonic Fixations

The sixth described principal fascial distortion type is the tectonic fixation.

In a tectonic fixation the fascial surface has lost its ability to properly glide, due to the loss of synovial fluid transport between two structures. The two layers of fascia, or two partners of articulation stick together like two magnets.

This very distortion is common and widespread for fixation of fascial surfaces can occur in any joint of the body.

(Typaldos, 2002)

In the lumbar spine tectonic fixations are found of course in the facet joints and are treated by thrust manipulation such as lumbar roll or the chair technique in neutral position (neither traction nor compression being applied as apposed to the application in case of folding distortions). Furthermore all techniques increasing synovial fluid circulation are very helpful. However, other concurrent fascial distortions should be corrected first, because they may be causal for genesis of tectonic fixations (foremost triggerbands with adhesions as well as folding distortions) (Typaldos, 2002)

The patient affected by tectonic fixation will express that his “back needs to be cracked.”

3.3 Studies about the efficacy of the FDM

In the FDM Textbook Typaldos describes 22 case histories of patients, who were treated using FDM for different problems in their musculo-skeletal system (e.g five patients complained about painful shoulders, four about ankle problems, three suffering from LBP symptoms....).

Unsurprisingly Typaldos only presents case histories with an enormous and fast bettering of symptoms as well as improvement of movements. Due to the fact that the quoted publication is a textbook, which is written for students of FDM, it cannot be expected to find highest impartiality. This is not specific to the very textbook but also

common for textbooks, teaching other techniques such as visceral- or cranio-sacral therapy.

Besides Typaldos' case histories only little scientific research has been carried out to verify the thesis of the FDM.

As the FDM is still a very new concept - known only to a little number of therapists, the pool of people, who do research on that topic is quite small. In my literature research I found three studies concentrating on the efficacy of treatments following the principles of the FDM:

Rossmys (2005) master thesis for the "College für angewandte Osteopathie" investigates the efficacy of treatment according to the principles of the FDM in the treatment of painful restricted abduction of the shoulder. His study is a randomized clinical study examining and treating 36 patients with different medical diagnoses such as impingement, osteoarthritis, posttraumatic pain and tendonitis. 19 of the participating individuals are treated in the FDM group, 17 in the control group. Rossmys chooses the following procedure for his study: after taking the medical history, the patient's shoulder abduction is measured (on the one hand with a goniometer, on the other hand with standardized digital photo which is evaluated afterwards with "Corel Draw 12"). The measurement is followed by the first treatment, after which the abduction is measured again. The same procedure is repeated before and after the second treatment (three or four days after the first) At last there is a terminal measurement 14 to 16 days after the second treatment. In total the shoulder abduction is measured 5 times.

While the FDM group is treated following the FDM, the control group receives articulation techniques and traction/distraction treatment for the glenohumeral joint as well as passive mobilisation of the scapula.

Rossmys' results are promising: while in the control group the abduction gains seven degrees, the FDM group manages an improvement of 42 degrees. Between the first and second treatment both groups lose their newly gained movement slightly, but then improve again: control group gains another six degrees, the FDM group another 16. The terminal measurement shows a persistent improvement of the FDM group (another four degrees) while the control group slightly worsens by two degrees.

After this terminal measurement the control group is given the opportunity to get two more FDM treatments. Also the control group is now showing a considerable gain of 36 degrees after the first measurement- respectively additional six degrees after the second FDM treatment.

Table 2: Rossmys's results

	FDM group mean (sd)	control group mean (sd)	p-value
first measurement	111	118	
terminal measurement	168 (+/- 12)	123 (+/- 34)	<0.0001
absolute difference before - after	55 (+/- 26)	5 (+/-15)	<0.0001
percental difference before - after	57 (+/- 38,7)	5 (+/- 12)	<0.0001

Table 2, Rossmys's results

The table above shows the FDM group performed significantly better than the control group.

Vis. (2006) wrote her master thesis for the “Vienna School of Osteopathy” titled “The treatment of lateral epicondylitis with the Fascial Distortion Model by Stephen Typaldos”. Her study compares FDM treatment for lateral epicondylitis of the elbow with treatment with NSAID therapy following the usual dutch standard (500 mg Naproxen twice daily). The 23 participating individuals are randomly divided in two groups (FDM group 11, control group 12). For measurement Vis chooses grip strength measurement (using a “Jamar dynamic hand dynamometer”,) on the one hand and a visual analogue scale (0-100) on the other hand. As far as pain is concerned there is also an additional verbal rating (no pain – severe pain) as well as numeric rating (0-10). Vis’ approach is as follows: The FDM group is treated 5 times in an interval of seven days (including three measurements: initial, after the third treatment and a final measurement one week after the last treatment). The control group starts with Naproxen medication at day one and continues for 15 days. The results are in favour for the FDM group: as far as pain is concerned (VAS) the NSAID medication does not reveal significant changes while the test group establishes significant improvement: mean difference between pretest and midtest

measurement is 28,64 points, delta between midtest and posttest measurement is 16,81. Also the frequency of pain is reduced in the FDM group: while beforehand 72,7% of the test group reported continuous pain, 54,5% are reporting only sporadic pain or are free of pain at the midtest, and so do 81,8 % at the terminal testing. In contrast, the control group does not show any improvement in pain frequency: the number of patients with regular pain (60%) or continuous pain (30%-40%) remains at a high level.

As far as grip strength is concerned, Vis differentiates between four parameters: pain free grip strength, maximum grip strength, pain threshold on grip and strength loss. There is a significant improvement in mean pain free grip and mean threshold on grip between pretest and terminal test. Concerning the other two parameters no changes could be verified. The results of the control group show no significant improvement in any of the parameters.

While the two studies mentioned above evaluated range of movement respectively pain and strength *Geiger (2007)* concentrates on stability, sensomotoric function (proprioceptive capabilities of a person) as well as symmetry in the upright stance on both legs. 75 asymptomatic probands take part in the study and are divided by random in experimental group, comparison group and control group. By means of a computer controlled measurement system every proband's ability to keep balance on an MFT board is examined. Afterwards the experimental group is treated using triggerband technique for the lateral thigh, the control group is treated with friction massage of the lateral thigh while the control group receives no treatment. Following those treatments the probands are examined once more.

Geiger's hypotheses were that by treating both the lateral thighs of probands their stability (hypothesis 1), sensomotoric function (hypothesis 2) and symmetry (hypothesis 3) can be improved. The hypotheses on sensomotoric function could be verified. The comparison group and control group improved as well, no significant differences have been found. The assumptions that stability and symmetry could be improved as well could not be verified.

Summarizing it can be said that two out of the three studies that I found report statistically significant improvement through the application of the FDM as far as the parameters pain, mobility and strength are concerned. The third study also shows

improvement through the application of FDM on the parameter sensomotoric function, but not statistically significant.

4. Methods

4.1 General Information

This study was carried out in quasi-experimental design with repeated measures. All participating patients were in one single group and underwent the same standardised procedure described below. Control was given by the repeated measures in the pre-, inter- and post-treatment period (three times pre-treatment, twice inter-treatment and three times post-treatment).

4.1.1 Reasons for choosing the repeated measures design

The research question of this thesis was if a treatment following the rules of the FDM is efficacious in treating patients with CLBP. In order to find an answer to this question I decided to use the repeated measures design for the following reason:

As the study deals with patients suffering from chronic pain, it was estimated that the probands would suffer from (measurable) consistent pain and consistent impairment in their functional status, showing only little up- and downturns. The three measurements during pre-treatment period tried to verify this hypothesis of more or less consistent pain. The analysis of the results of the pre-treatment questionnaires (see chapter 5) shows that the parameters were indeed rather constant. This confirmed the assumption that both pain and functional status of the probands would stay rather constant not only in the weeks of pre-treatment observation but also in the following weeks (in the case of receiving no treatment).

This consistency of the baseline parameters allowed using the supposable continuation of pain and functional status as a quasi substitute for a control group.

Nevertheless it is clear that without having a control group, the outcome of a clinical trial cannot be of maximum significance. The supposed consistency of parameters as a fictive control group can only be a weak substitute, but in this study I decided to do so for the following reasons:

- Given that I am working alone in my private practice it was obvious that it would be difficult to find enough patients for the study. If the 22 participating patients would have been split up in two groups, both the test group and the control group would have been very small in size, which makes it difficult to find significant differences.
- Uncertainties about how to treat a control group:
 - A waiting list design) would have taxed the participants' patience a big deal, for it needs a very good compliance to repeatedly fill in the questionnaires without receiving any treatment.
 - Manual sham treatment for the control group is hardly manageable and ethically not correct.
 - Treating the control group osteopathically without using the FDM would not be ethically correct for me personally, as it is part of my daily routine when treating patients with LBP.
 - Let the control group be treated by some other osteopath would also be questionable for me: the test group would be treated only by using FDM techniques, while the control group would consume the whole wide range of osteopathic treatments, including also visceral and cranial work.
 - Using the FDM concept exclusively would probably be a disadvantage, when being compared to an osteopathic black box treatment.
 - Furthermore this would test FDM's superiority to osteopathy rather than the efficacy of FDM.

4.2 Operating procedure

All in all 22 patients took part in my study. These patients - fitting in the inclusion and exclusion criteria (see chapter 3.2) – were referred by two Viennese physicians:

Dr. med. Univ. Johann Weiß (general practitioner, cooperating with me for several years), referred ten participants and Dr. med. Univ. Ilse Stracker-Jandl (general practitioner), five.. Both physicians referred long time LBP patients from their pool of patients. Seven participants came by word of mouth recommendation.

4.2.1 Inclusion and exclusion criteria

Inclusion criteria for my study were:

- Male or female Patients, aged between 18 and 80
- Chronic low back pain (LBP lasting for more than the last 6 weeks) with or without Sciatica
- Patients not receiving any other kind of treatment (except pain relief medication) during the 7 week study period

Exclusion criteria were:

- Lumbar or pelvic fractures
- Acute herniated vertebral disc (Achilles tendon reflex absence, PSR absence)
- Patients with a disc surgery in the last 6 months
- Spondylolisthesis
- Pregnancy
- Chronic intestinal diseases (Mb. Crohn, Colitis ulcerosa, chronic congestion)
- Nephrolithiasis
- Gallstones
- Diseases of the uretic system
- Malignancies and metastases
- Manic depression

Following *Deyo's (2001)* compendium of different diagnosis of LBP the above indicated visceral diseases and nonmechanical spinal conditions (such as neoplasia...) can be the important factor in LBP, but are not in Osteopath's

competence to treat with the FDM. Patients with manic depression were excluded because it was unclear how such a patient would react psychologically on a treatment, which could be considered as rude.

The patients, referred from the general practitioners named above were pre-checked by them, to confirm they matched the inclusion and exclusion criteria.

The remaining patients underwent special anamneses when they came for the first time, in order to see if they really fitted.

4.2.2 Procedure / Modus operandi

The whole study was divided in a pre-treatment period, two treatments with the corresponding inter-treatment time, as well as a post-treatment period.

4.2.2.1 Pre-treatment period

Patients referred to participate in my study first had to answer the German version of the Oswestry Disability Index questionnaire (ODI, version 2.1, see chapter 4) developed by *Mannion et al (2005)*, with an additional question concerning the current pain medication (no medication / occasional medication / constant medication) and an additional 10 cm Visual Analogue Scale (VAS).

The VAS was chosen for two reasons: on the one hand it offers the possibility to measure an ad hoc change of pain from before to after treatment, and on the other hand it delivers more precise data, as it detects even very little changes (VAS was measured in mm).

This questionnaire was handed out by the referring practitioners, respectively was sent via email to those patients, coming from word of mouth recommendation.

Exactly one week later the patients filled in the very same questionnaire, and so they did a third time prior to the first treatment in my practice.

4.2.2.2 Initial medical examination

After completion of the questionnaire, the anamneses started, which paid enormous attention to the body language used by the patients when they were describing their pain.

In order to reconfirm if the patients (still) matched the inclusion and exclusion criteria, (mainly to exclude new and acute problems) the initial medical examination, following the anamneses, included important neurological test (like Straight Leg Raise [SLR], Achilles and Patella- tendon reflex, Valsalva Test, Muscle strength and Sensibility).

4.2.2.3 Active mobility tests

As active mobility tests the standing flexion test, standing extension, sidebending and rotation were performed. While the standing flexion test was measured (see 3.3.3.2), the other tests were performed in the first instance to see if these movements caused pain.

If neither of these tests could provoke pain, patients were asked to do a special movement or perform a special activity, of which they were sure that it would provoke “their” pain.

4.2.2.4 Measurement of finger-floor-distance

When patients complain about - respectively their body language demonstrates - triggerband related pain in the lumbar spine it is a common finding to have a limited Flexion.

As I expected to see a lot of triggerbands (see Chapter 3.2.1) I decided to measure the finger-floor distance, in order to verify if there was an ad hoc improvement of Flexion after using the triggerband technique.

To improve the reproducibility of the standing flexion test it was performed on a special marked carpet, ensuring that there was always equal distance between right and left foot (17 cm).

Patients were asked to bend over, trying to touch the floor with their fingers.

This test was performed four times in a row, measured every single time. Out of these four different results I calculated the arithmetic mean. Unit of measurement was cm.

4.2.2.5 First treatment

According to the findings in the anamneses and the active mobility tests the first treatment was performed.

The treatment always followed the rules of the Fascial Distortion Model:

- Painful treatment first
- No thrust after a Continuums

After the treatment the standing flexion test was repeated and measured and the patients were asked to do a new mark on the VAS.

4.2.2.6 Inter-treatment period

Three days after the first treatment patients were asked to fill in the ODI questionnaire and the added VAS scale once more.

4.2.2.7 Second treatment

Prior to the second treatment patients filled in the ODI questionnaire and VAS. After that the same procedure as in the first treatment session was carried out.

4.2.2.8 Post-treatment period

Post-treatment period started right after the second treatment by marking the VAS. Furthermore patients were given two copies of the ODI and VAS, which they had to fill in one respectively two weeks after the second treatment.

5. The Oswestry Disability Index (ODI)

The ODI is a self-administered questionnaire, measuring the extent of the patient's back pain. Probands complete the ODI to give an account of their functional status on the very day. The ODI assesses the patient's difficulties in carrying out nine activities of daily life: personal care, lifting, walking, sitting, standing, sleeping, sex life, social life and travelling. A tenth sub item assesses pain.

Each item is scored from 0 – 5, with 0 meaning that there is no impairment and 5 representing greater disability. The “ODI score” is the result of the doubled sum of all 10 sub items.

The reasons for choosing the ODI are manifold:

Due to *Mannion's (2007)* work the ODI is available in German.

Fairbank JC (2000) wrote: “The ODI has stood the test of time and many reviews. It's usable in a wide variety of applications as a condition-specific outcome measure of spine-related disability (Spine 2000, Volume 25 (22)).

When choosing the ODI over the Roland Morris Questionnaire (which is also often used) it was decisive that the ODI is recommended for patients with persistent and severe disability, while the RM suites better for patients having mild to moderate pain. (*Roland, 2007*)

As far as interpreting the changes in the ODI is concerned, *Ostelo RWJ (2008)* published an “international consensus regarding Minimal Important Change (MIC) on frequently used measures of functional status and pain”. By means of literature review as well as an expert panel Ostelo and co-workers arrived at the following agreement: MIC for the ODI is proposed by a change of 10 points, respectively by a 30% change relatively to the baseline score.

For the VAS, which is used in this study as well, the authors agreed on a MIC of 15 mm, or also a 30% change to the baseline score.

6. Descriptive analysis of the sample

6.1 Gender distribution

All in all 22 subjects (n = 22) took part in the study, 18 of which are females and 4 are males.

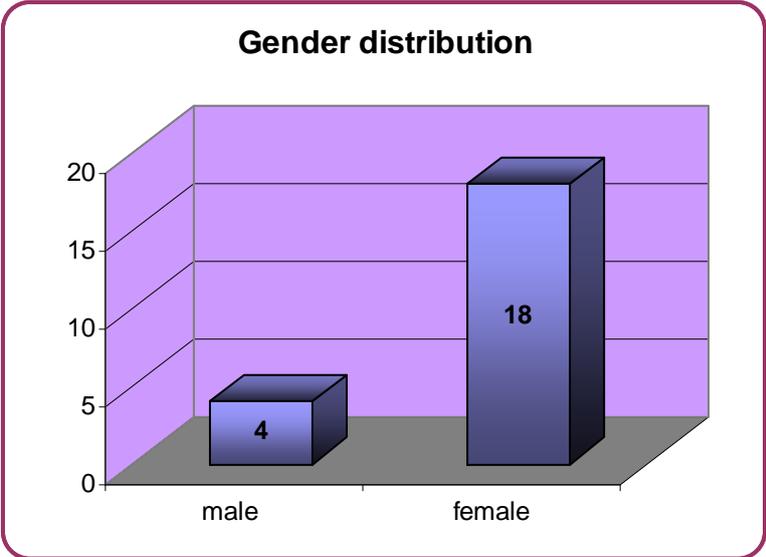


Diagram 2, gender distribution

6.2 Age distribution

Subjects in the study ranged from the age of 20 to 70.

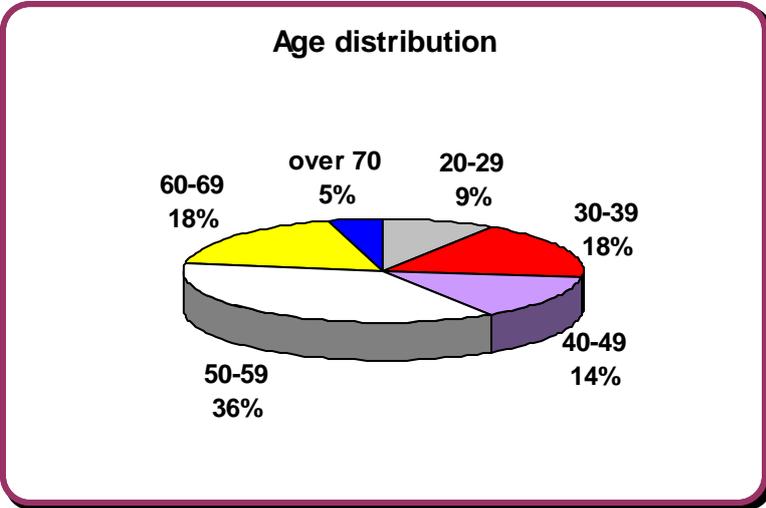


Diagram 3, age distribution

6.3 Medical diagnoses

The one group of probands referred by the supporting general practitioners have mostly already undergone MR examination and therefore could present precise diagnoses (such as disc herniation, disc protrusion, vertebrostenosis....).

For each of these subjects a combination of different degenerations was found. In the diagram below these probands with multiple diagnoses were grouped to the diagnosis that I considered as most severe.

Subjects of the other group, not being referred by their general practitioner, presented diagnoses that were less precise: chronic lumbalgia, hyperlordosis...

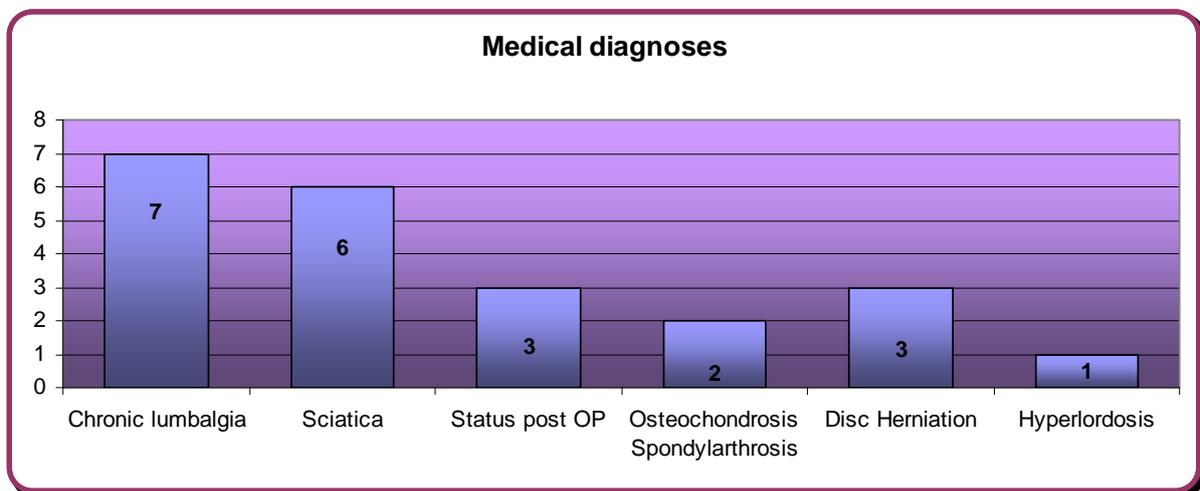


Diagram 4, medical diagnoses

7. Results

Using the repeated – measurement design, it led to a collection of data in each and every parameter (medication, functional status, VAS results, finger – floor distance). Those data were analysed by means of SPSS 10.

7.1 Changes of intake of analgesics during study period

At the beginning of this study the parameter “intake of analgesics” was not given the highest priority, as it was not expected to be impacted very much. This assumption turned out to be wrong as numerous participants were able to reduce their intake of analgesics.

The results of the first questionnaire show that 13 of the participating 22 persons do not take any painkillers, six take painkillers occasionally and three are on constant medication.

During study period, the number of patients, not using painkillers rises from 13 to 18 in the last questionnaire while the number of patients using them occasionally falls from six to four. The number of patients on constant medication could be reduced to zero.

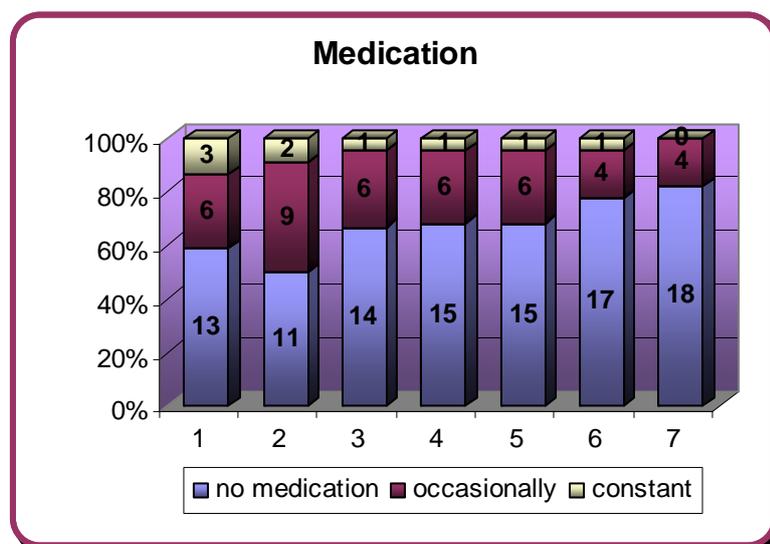


Diagramm 5, Intake of medication

In the third questionnaire one single person was taken out of consideration, as she was on constant medication because of pain in her knee. So the total number of persons in the third questionnaire is 21 instead of 22.

Those subjects who could reduce their medication during the treatment period were included in the statistical evaluation. Two individuals showed no continuity in their medication (jumping from no medication to constant medication and back) and were therefore excluded for the relevant analyses. Nevertheless they were yet included in those analyses which reveal ad hoc changes within one treatment.- these are pre – and post-test measurement of VAS and finger – floor distance

7.2 Changes of the functional status during the study period

As described before, 20 subjects could be evaluated in this regard by means of the Oswestry Disability Index (ODI). During pre-treatment observation time the mean ODI scores were rather stable ranging from 19,10 (in ODI 3) to 21,60 (in ODI 1). Starting with the treatment period the ODI scores begin to fall, resulting in a mean score of 13,20 in the first follow up ODI and – still improving - a score of 12,31 in the last follow up.

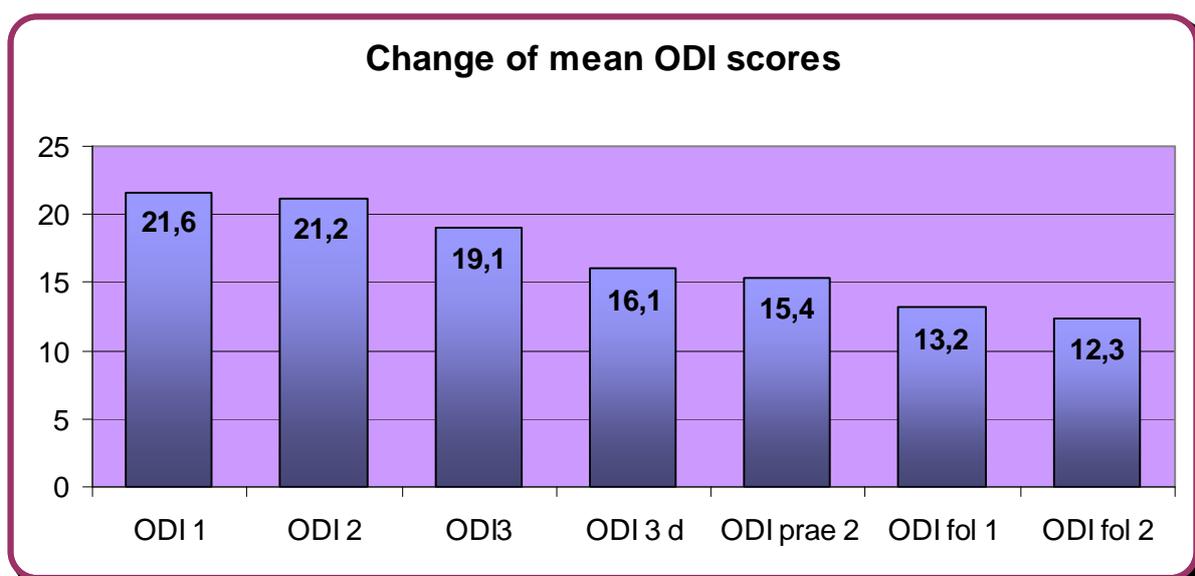


Diagram 6, Changes of mean ODI scores

Using the mean of all pre-treatment ODI scores (20,6) as well as the mean of all follow up ODI scores (12,8) this is an improvement of 7,8 or 38,12%. Referring to the Minimal Important Change (MIC) consensus of *Ostelo RWJ and co-workers (2008)* this can be referred to as clinical important change.

The T-test of paired samples comparing those 2 variables shows a highly significant improvement with a moderate correlation of +0,715.

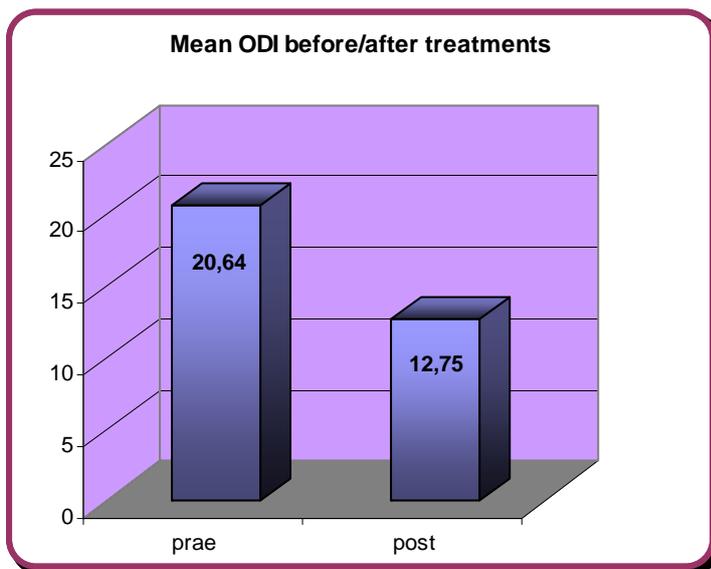


Diagram 7, Mean ODI before/after treatments

Statistics of paired samples					
		<i>m</i>	<i>N</i>	<i>sd</i>	<i>se of mean</i>
pair 1	ODI prae	20,635	20	11,919	2,665
	ODI post	12,750	20	14,249	3,186

Correlation of paired samples				
		<i>N</i>	<i>correlation</i>	<i>significance</i>
pair 1	ODI prae & ODI post	20	0,715	0,000

Test of paired samples									
		<i>m</i>	<i>sd</i>	paired diff. <i>se of m</i>	95% confidence interval		<i>T</i>	<i>df</i>	<i>sig.</i>
					lower	upper			
pair 1	ODI prae & ODI post	7,885	10,119	2,263	3,149	12,621	3,485	19	0,002

7.2.1 Changes of the ODI scores for the “worse half” of patients

It turned out that there are some difficulties for the ODI to detect changes in patients with rather low scores. So some subjects, who started with quite low ODI scores, soon reached a score of zero (or close to zero). This fraction then presented almost equal scores in the last measurements.

This is why I decided to do some extra analyzes of those 50% of patients, who reached lower ODI scores.

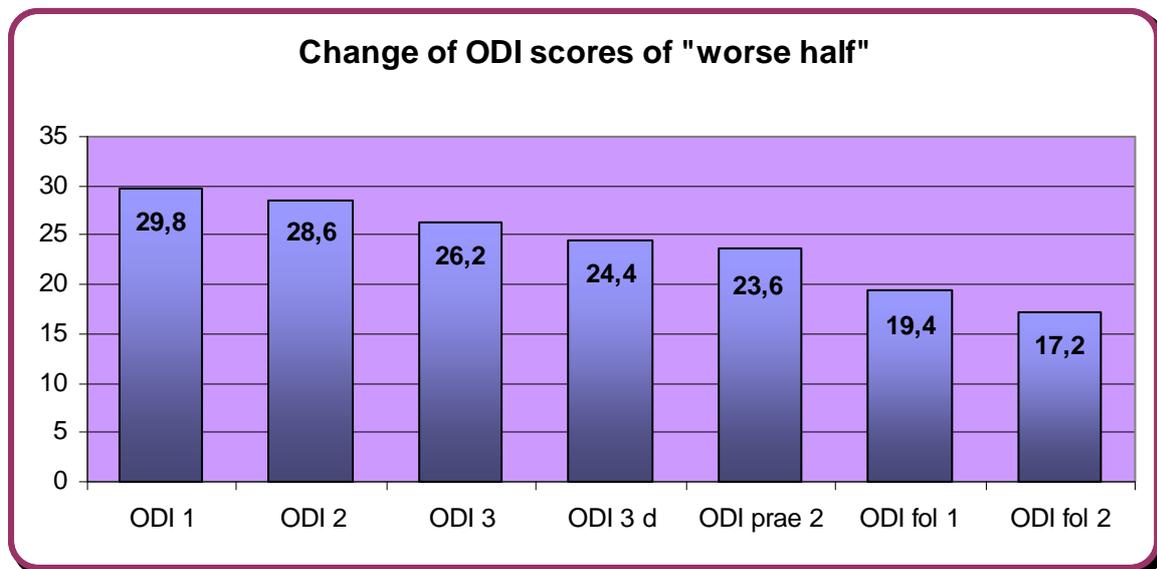


Diagram 8, Change of ODI scores of the “worse half”

Again, the mean of all ODI scores of this fraction during pre-treatment period is 28,4 and the mean ODI scores in the post-treatment period is 18,3. So for these patients an improvement of 10,1 points (35,6 %) could be reached.

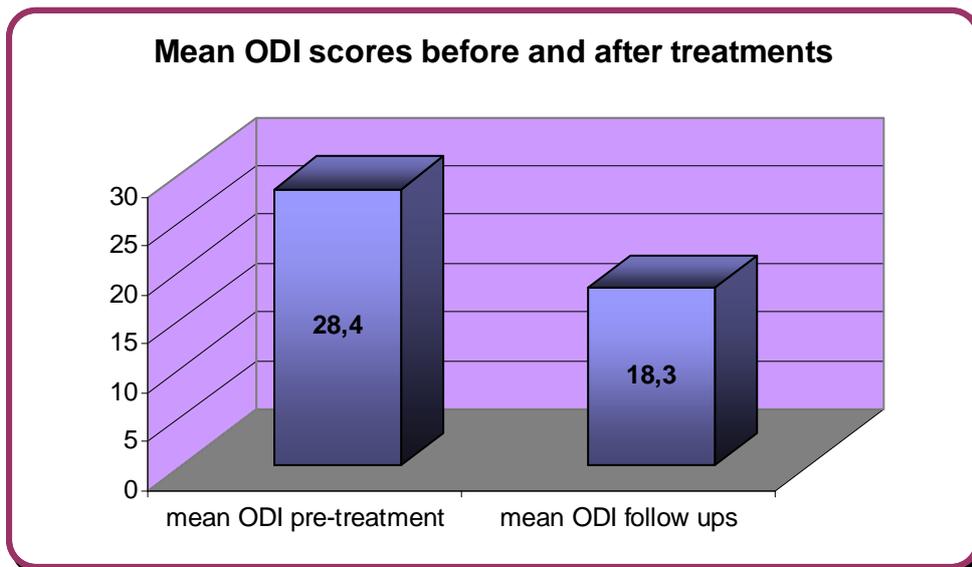


Diagram 9, Mean ODI scores before and after treatments of the worse half of patients

Statistics of paired samples					
		<i>m</i>	<i>N</i>	<i>sd</i>	<i>se of mean</i>
pair 1	ODI prae	28,200	10	12,127	3,835
	ODI post	18,300	10	18,415	5,823

Correlation of paired samples				
		<i>N</i>	<i>correlation</i>	<i>significance</i>
pair 1	ODI prae & ODI post	10	0,738	0,015

Test of paired samples									
		<i>m</i>	paired diff.		95% confidence interval	<i>T</i>	<i>df</i>	<i>sig.</i>	
			<i>sd</i>	<i>se of m</i>					
pair 1	ODI prae - ODI post	9,900	12,511	3,956	0,950	18,850	2,502	9	0,034

The T-Test shows also in this fraction of patients a highly significant change with a moderate correlation of 0,738.

7.3 Changes of VAS results during study period

The questionnaire, that was handed out to the patients included also a 10 cm Visual Analog Scale. Firstly the VAS, as being more precise in its ability to measure

intensity of pain (compared to the ODI), offered a second method to collect data. Secondly the VAS can be used directly after treatment, which helps to detect ad hoc changes. In addition to the seven questionnaires during the study phase, patients had to fill in the VAS another two times, directly after the first respectively the second treatment.

The mean VAS results in the pre-treatment observation period were rather stable ranging from 3,35 (being the result of the first questionnaires) to 3,57 in the last pre-treatment questionnaire. Having received the first treatment the mean VAS score was more than halved in value to 1,43. The mean VAS results on day three after the first treatment increased again to 2,46, but was still 31,1 % lower than the last pre-treatment VAS. The rising of the VAS results on that very date came not as a surprise, due to expected side effects of the FDM treatment, such as muscle sourness – caused by the triggerband technique.

The mean VAS prior to the second treatment was 2,17, and fell to 0,98 post treatment. The two follow ups showed a mean VAS of 1,52 respectively 1,3.

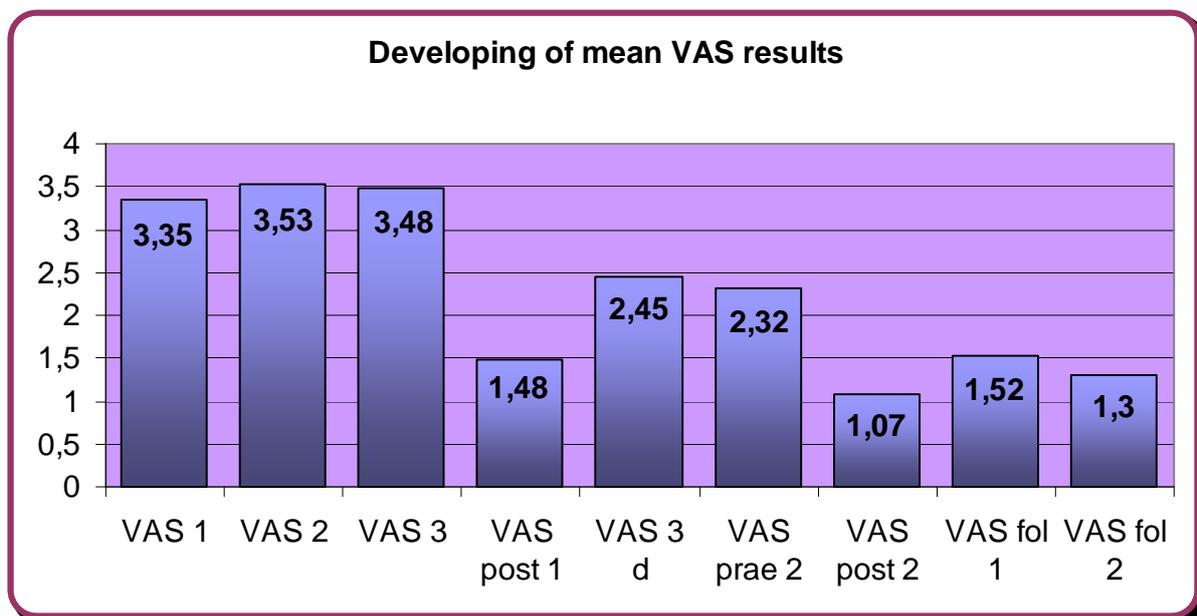


Diagram 10, Developing of mean VAS results

Using the mean of all pre-treatment VAS and comparing it with the mean of all post treatment VAS, show a pleasing result: mean VAS could be reduced from 3,48 to 1,41 which equals an improvement of 59,9%.

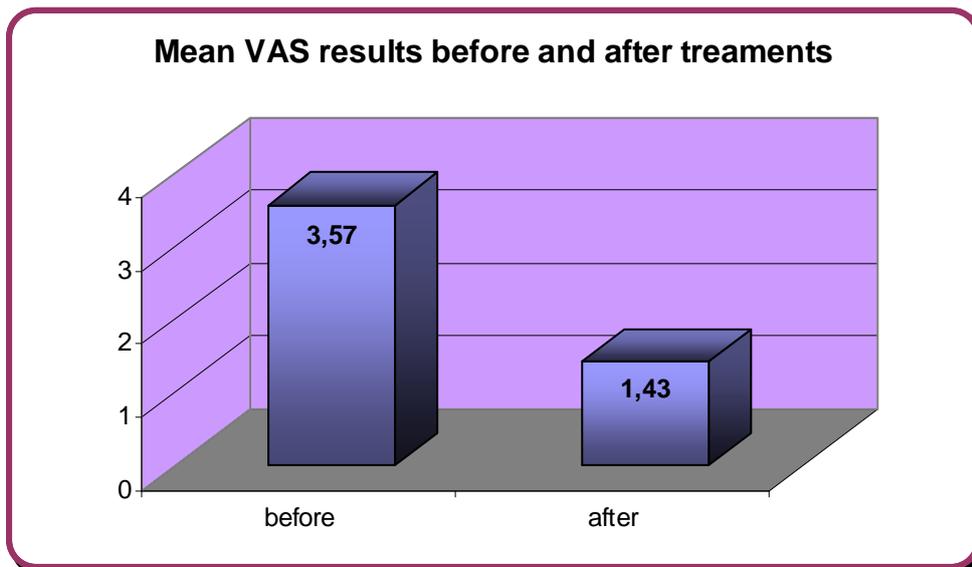


Diagram 11, Mean VAS results before and after treatments

The T-test results are not as distinct. The significance is also high in this fraction, but the correlation is only mild.

This is due to two participants, who marked extremely high VAS scores (doubling their pre-treatment values) in the post treatment period.

These participants showed also higher ODI scores in the post treatment period, but not as strong as in the VAS.

Statistics of paired samples					
		<i>m</i>	<i>N</i>	<i>sd</i>	<i>se of mean</i>
pair 1	VAS prae	3,452	20	2,119	0,474
	VAS post	1,405	20	1,886	0,422

Correlation of paired samples				
		<i>N</i>	<i>correlation</i>	<i>significance</i>
pair 1	VAS prae & VAS post	20	0,227	0,335

Test of paired samples									
		<i>m</i>	<i>sd</i>	paired diff. se of <i>m</i>	95% confidence interval		<i>T</i>	<i>df</i>	<i>sig.</i>
					lower	upper			
pair 1	VAS prae & VAS post	2,047	2,496	0,558	0,878	3,215	3,667	19	0,002

7.3.1 Ad hoc changes of VAS results before and after the treatments

By means of the VAS it was not only possible to detect long-term changes in the patient's perception of pain, but also to identify short-term effects of the treatment. In this analysis all participants (n=22) are included, for the effect of the analgesics is considered to be constant, with no changes between before and after treatment. Comparing the VAS results before and after the first treatment shows a change from 3,57 to 1,43 (an improvement of 59,9%). The analysis shows similar results before and after the second treatment: mean VAS results beforehand were 2,17, mean VAS results post treatment were 0,98 (an improvement of 54,8%) Both values exceed the suggested threshold of 30% (see quoted consensus above) by far.

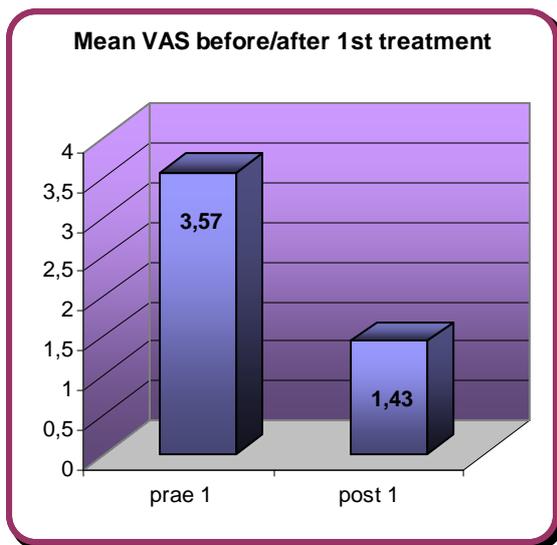


Diagram 12, Mean VAS before/after 1st treatment

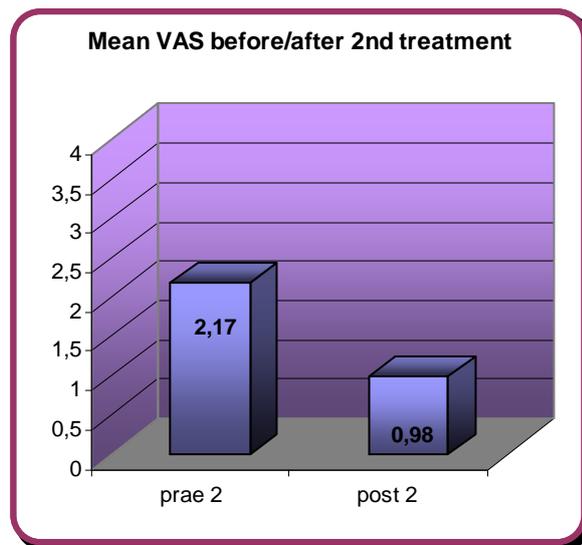


Diagram 13, Mean VAS before/after 2nd treatment

The T-Test of the variables VAS prae 1st and VAS post 1st treatment shows a high significant change with a moderate correlation of 0,627.

Statistics of paired samples		<i>m</i>	<i>N</i>	<i>sd</i>	<i>se of mean</i>
pair 1	VAS prae 1st	3,568	22	2,268	0,484
	VAS post 1st	1,427	22	1,427	0,304

Correlation of paired samples				
		N	correlation	significance
pair 1	VAS prae 1st & VAS post 1st	22	0,627	0,002

Test of paired samples									
		m	sd	paired diff. se of m	95% confidence interval		T	df	sig.
						lower	upper		
pair 1	VAS prae & post 1st	2,141	1,767	0,377	1,357	2,925	5,682	21	0,000

The results are even more explicit in the T-Test for the variables VAS prae 2nd and VAS post 2nd. The improvement is highly significant and there is a strong correlation of 0,789.

Statistics of paired samples					
		m	N	sd	se of mean
pair 1	VAS prae 2nd	2,173	22	2,184	0,466
	VAS post 2nd	0,977	22	1,614	0,344

Correlation of paired samples				
		N	correlation	significance
pair 1	VAS prae 2nd & VAS post 2nd	22	0,789	0,000

Test of paired samples									
		m	sd	paired diff. se of m	95% confidence interval		T	df	sig.
						lower	upper		
pair 1	VAS prae & post 2nd	1,195	1,345	0,287	0,599	1,792	4,168	21	0,000

7.4 Changes of the finger-floor distance

As described in chapter 3.3.2.2 the finger-floor distance was measured four times in a row before and after every single treatment. Out of these four results the arithmetic mean was calculated and used for further analyses.

In the FDM it is assumed, that the presence of triggerbands worsens the ability of bending forward, a diagnostic finding, which is very often seen in patients with CLBP. Again all participants are included in the observation (n=22)

Both in the first and in the second treatment the mean finger-floor distance could be reduced: from 8,2 to 4,7 in the first treatment respectively from 6,6 to 4,1 in the second treatment. Note that there is a absolute difference of 1,6 cm between the two pre-treatment measurings (an improvement of 19,5%), indicating that the improvement in the finger-floor distance was not only a short term effect.

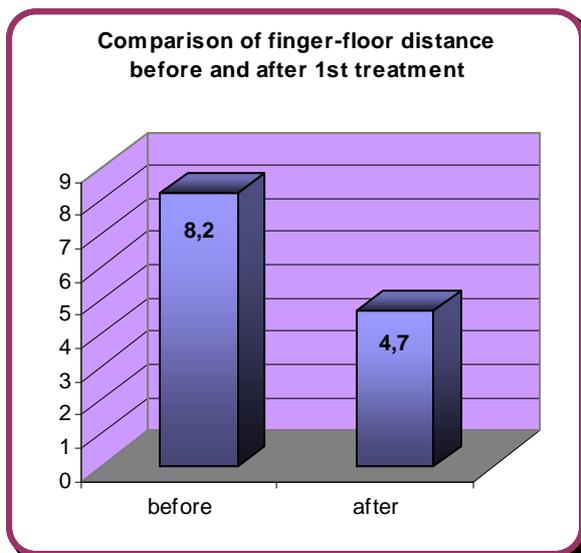


Diagram 14, Comparison of finger-floor distance before and after 1st treatment

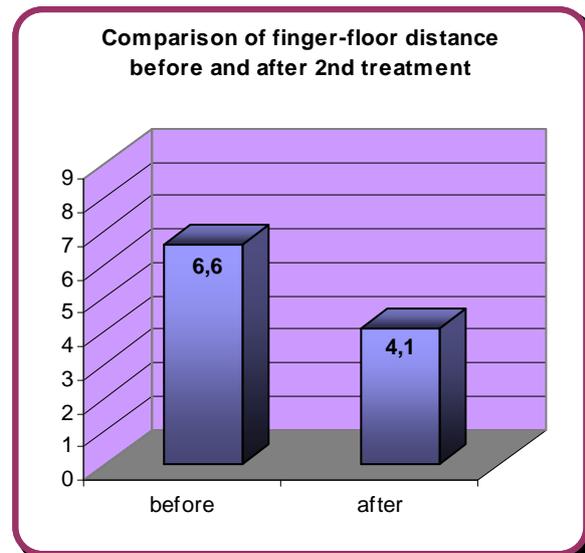


Diagram 15, Comparison of finger-floor distance before and after 2nd treatment

The T-Test of those two variables shows a highly significant change with a strong correlation of 0,940.

Statistics of paired samples						
		<i>m</i>	<i>N</i>	<i>sd</i>	<i>se of mean</i>	
pair 1	ffd prae 1st	8,200	22	10,119	2,157	
	ffd post 1st	4,682	22	6,271	1,337	

Correlation of paired samples				
		<i>N</i>	<i>correlation</i>	<i>significance</i>
pair 1	ffd prae & post 1st	22	0,940	0,000

Test of paired samples									
		<i>m</i>	<i>sd</i>	paired diff. se of <i>m</i>	95% confidence interval		<i>T</i>	<i>df</i>	<i>sig.</i>
						lower	upper		
pair 1	ffd prae & post 1st	3,518	4,735	1,009	1,419	5,617	3,485	21	0,002

Also the comparison between finger-floor distances in the 2nd treatment shows highly significant improvement with a strong correlation of 0,962.

Statistics of paired samples						
		<i>m</i>	<i>N</i>	<i>sd</i>	<i>se of mean</i>	
pair 1	ffd prae 2nd	6,566	22	8,396	1,790	
	ffd post 2nd	4,102	22	5,398	1,151	

Correlation of paired samples				
		<i>N</i>	<i>correlation</i>	<i>significance</i>
pair 1	ffd prae & post 2nd	22	0,962	0,000

Test of paired samples									
		<i>m</i>	<i>sd</i>	paired diff. se of <i>m</i>	95% confidence interval		<i>T</i>	<i>df</i>	<i>sig.</i>
						lower	upper		
pair 1	ffd prae & post 2nd	5,589	8,288	1,767	1,914	9,263	3,163	21	0,005

8. Discussion

The main objective of this study was to examine whether a treatment following the principles of the FDM can be efficacious in treating patients with CLBP. The efficacy was measured on the basis of two parameters: functional status and pain.

The hypothesis was that after two treatments following the FDM, there would be a measurable improvement of these two parameters.

The hypothesis on functional status can be verified: after two treatments patients show an improvement of 38,12%. These results are statistically significant and exceed the 30% improvement, indicating a MIC.

The hypothesis concerning pain can also be verified: comparing the mean of the pre-treatment results with the mean post-treatment results there is an improvement of 59,9%. Additionally to these positive long term effects, the study also shows delectable ad hoc improvements: after both treatments the mean VAS scores could be more than halved. Both short and long term improvements are statistically significant.

In addition to the above quoted main parameters also the third parameter –mobility - changes for the better. As ad hoc achievement the patients show an statistically significant improved flexion of approximately 40%.

Another achievement is the reduction in intake of analgesics during the observation period.

Summarizing the results of this study, I am able to conclude that the research question can be answered in favour of FDM: a treatment following the principles of the FDM is efficacious in treating patients with CLBP.

The choice of using this very study design might lead to less valid results than randomized double blind controlled trials. It is recommended to improve the validity of the results by conducting a follow up study, which makes use of a control group.

My private practice's code of conduct and ethical standards did not allow for conducting a waiting list design or a study design with the control group being sham-treated. This would have led to patients' dissatisfaction, something which is not compatible to the customer satisfaction strategy the practice is following.

By having the long pre-treatment phase, interrogating the basis values of pain and functional status, the study makes up for the absence of a control group. Assuming that these values would remain quite stable, they can be taken as a reference for the changing values of the test group.

Future studies about the FDM would be well advised to use another study design, such as a randomized controlled trial.

The number of participants (22) in this study was relatively small. Despite this fact the results showed statistically significant differences between before and after treatment. For this study it was statistically not a disadvantage, as the results of the single individuals showed a good correlation and significance. Nevertheless future studies would be well advised to increase the number of participants.

Another learning, concerning the study-process, is to have the treating process and the observation process done separately by another person. In the process of treating it is unavoidable for the therapist as well as the patient to socially interact with each other. This interaction is always based on an asymmetric relation: the (mighty and clever) investigator on the one hand and the help seeking patient on the other hand. In a kind of self-fulfilling prophecy the patient might find himself provoked to mark better values in the questionnaire in order to satisfy the investigator's (and so the known therapist's) desire.

On the flipside, during the observation period it happened only twice that the patients had to fill out the questionnaire with the investigator being present. At all other times the questionnaires were filled out in privacy and the results were brought in or sent via mail.

Furthermore it would improve the validity of the results if there would have been more (and more experienced) therapists conducting the treatments. By having only one

therapist doing the treatment it can easily lead to a bias, a systematic error in treatment. In the FDM this could be for example a constant misinterpretation of a certain sign of body language, a misinterpretation that will lead to a treatment that is not indicated and thus not helpful.

During the study it turned out that the Oswestry Disability Index (ODI) has difficulties to detect differences and changes in patients who have only little impairment in their functional status. In retrospective, it would have been an advantage to have the parameter "functional status" measured and compared with two different questionnaires, for example the Oswestry Disability Index and the Roland Morris questionnaire (RM), as the RM seems to be more suitable for patients with little impairment. (*Roland, 2007*)

For future studies I would recommend the use of two or three different questionnaires for the assessment of functional status. It would make it easier to detect all sorts of differences (little, moderate or large), but it could tax the participant's patience.

For future studies I would furthermore recommend to add another parameter of investigation, namely the patient's satisfaction. The instant effect of a treatment is not always measurable (as far as functional status but also pain is concerned).

Nevertheless the patient will feel satisfied or dissatisfied with the treatment and could mark this grade of satisfaction on a scale. This could help express, if the patient thinks that the treatment he/she has had was effective.

Despite the methodological weakness of this present study it has to be summed up that it led to a high significant improvement in all the observed parameters. So the hypothesis that a treatment following the principles of the FDM is efficient in treating patients with CLBP could be verified. The author would appreciate to have this study reproduced and controlled by future investigators.

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10. Appendix

10.1 Table of Abbreviations

AORT	Autonome Osteopathic Reposition Technique
CD	Continuums distortion
CLBP	Chronic Low Back Pain
corr.	Correlation
CT	Computed Tomography
CV 4	Compression of the fourth Ventrikel
D.O	Doctor of Osteopathy
Dr. med. univ.	Doctor medicinae universalis = Medical Doctor
ECD	Everted Continuums Distortion
EGMCLBP	European Guidelines for the Management of Chronic Low Back Pain
FDM	Fascial Distortion Modell
Ffd	Finger – Floor Distance
GCPS	Graded Chronic Pain Scale
GP	General Practitioner
HTP	Herniated Triggerpoint
HVLA	High Velocity Low Amplitude
ICD	Inverted Continuums Distortion
LBP	Low Back Pain
m	Mean
MBT	Membranous Balanced Tension
MRI	Magnetic Resonance Imaging
N	Number of Participants / Number of analyzed data
NSAIDs	Non Steroidal Anti Inflammatory Drugs
ODI	Oswestry Disability Index
ODI 1	Results of the first ODI
ODI 2	Results of the second ODI
ODI 3	Results of the third ODI
ODI 3d	Results of the ODI three days after the first treatment

ODI prae 2	Results of ODI prior to the second treatment
ODI fol 1	Results of the first follow up ODI
ODI fol 2	Results of the second follow up ODI
OMT	Osteopathic Manipulative Therapy
RMDQ	Roland Morris Disability Questionnaire
sd	Standard deviation
se	Standard error
se of mean	Standard error of mean
SF-36	Short Form – 36
SI-Joint	Sacro – Iliac Joint
sig.	Significance
SLR	Straight Leg Raise
TB	Triggerband
TZ	Transition Zone
VAS	Visual Analogue Scale
VAS 1	Results of the first VAS
VAS 2	Results of the second VAS
VAS 3	Results of the third VAS / VAS prior to the first treatment
VAS post 1	Results of the VAS after the first treatment
VAS 3d	Results of the VAS three days after the first treatment
VAS prae 2	Results of the VAS prior to the second treatment
VAS post 2	Results of the VAS after the second treatment
VAS fol 1	Results of the first follow up VAS, one week after the second treatment
VAS fol 2	Results of the second follow up VAS, two weeks after the second treatment

10.2 Table of Diagrams and tables

Diagram 1	Continuum distortion, adapted from FDM Textbook, chapt. 5, p 33
Diagram 2	Gender distribution
Diagram 3	Age distribution
Diagram 4	Medical diagnoses
Diagram 5	Intake of medication
Diagram 6	Change of mean ODI scores
Diagram 7	Mean ODI before and after treatments
Diagram 8	Change of mean ODI scores of the worse half of patients
Diagram 9	Mean ODI scores before and after treatments of the worse half of patients
Diagram 10	Developing of mean VAS results
Diagram 11	Mean VAS results before and after treatments
Diagram 12	Mean VAS results before/after the first treatment
Diagram 13	Mean VAS results before/after the second treatment
Diagram 14	Comparison of finger-floor distance before and after the first treatment
Diagram 15	Comparison of finger-floor distance before and after the second treatment
Table 1	Differential diagnoses of LBP, adapted from Deyo (2001)
Table 2	Results of Rossmly (2005)

10.3 Table of pictures

Picture 1	Triggerbands, adapted from the FDM Textbook, chapter 3, p 23
Picture 2	Location of Bull's eye HTP, adapted from Prometheus, page 189
Picture 3	Location of lumbar hernias, adapted from Prometheus, page 189

10.4 Letter to patients / Questionnaire



Werte Patientin, werter Patient!

Danke, dass Sie sich entschieden haben, bei meiner Studie über das Fasciendistorsionsmodell mitzumachen!

Bei meiner wissenschaftlichen Untersuchung geht es darum, herauszufinden ob man mit dieser speziellen Behandlung Rückenschmerzen (so wie die Ihren) effizient behandeln kann.

Um das Gelingen der Studie zu gewährleisten, ist es notwendig, gewisse Schritte ganz exakt auszuführen. Darum möchte ich Sie bitten, diese Zeilen aufmerksam zu lesen und daraufhin das Procedere genau zu befolgen.

- 1.) Rufen Sie mich bitte unter der Nummer **0699/11601914** an, um sich für die Teilnahme an der Studie anzumelden, und um den Termin für die erste Behandlung auszumachen.
- 2.) Füllen Sie dann bitte gleich den Fragebogen aus (blaue Schrift).
- 3.) Eine Woche später füllen Sie bitte den Fragebogen noch einmal aus (rote Schrift).
- 4.) Kommen Sie zum ausgemachten Termin zu mir in die Praxis (und nehmen bitte die ausgefüllten Fragebögen mit!) Die Behandlung ist für Sie kostenlos (Normalpreis 65€)
- 5.) Drei Tage nach dem ersten Termin füllen Sie bitte abermals den Fragebogen aus.
- 6.) Eine Woche später kommen Sie neuerlich zu einem Termin zu mir in die Praxis (und nehmen bitte den ausgefüllten Fragebogen mit!)
- 7.) Eine Woche später bitte noch einmal den Fragebogen (grüne Schrift) ausfüllen
- 8.) Noch eine Woche später bitte den letzten Fragebogen (violette Schrift) ausfüllen und auf dem Postweg zu mir in die Praxis schicken. (Postgebühr bezahlt Empfänger)

Sollten Sie Fragen haben, Unklarheiten oder Probleme auftreten, rufen Sie mich bitte ohne zu zögern an. Ich danke noch einmal für Ihre Mitarbeit und dafür, dass Sie mich mit Ihrer Teilnahme unterstützen!

Mit freundlichen Grüßen

Bitte füllen Sie diesen Fragebogen aus. Er soll uns darüber informieren, wie Ihre Rücken
(oder Bein-) Probleme Ihre Fähigkeiten beeinflussen, den Alltag zu bewältigen.
Wir bitten Sie, jeden Abschnitt zu beantworten.
Kreuzen Sie in jedem Abschnitt nur die Aussage an, die Sie heute am besten beschreibt.

Name _____ Datum _____

Geb. Datum _____

Abschnitt 1: Schmerzstärke

- 0 Ich habe momentan keine Schmerzen
- 1 Die Schmerzen sind momentan sehr schwach
- 2 Die Schmerzen sind momentan mässig
- 3 Die Schmerzen sind momentan ziemlich stark
- 4 Die Schmerzen sind momentan sehr stark
- 5 Die Schmerzen sind momentan so schlimm wie nur vorstellbar

Abschnitt 2: Körperpflege (Waschen, Anziehen etc.)

- 0 Ich kann meine Körperpflege normal durchführen, ohne dass die Schmerzen dadurch stärker werden
- 1 Ich kann meine Körperpflege normal durchführen aber es ist schmerzhaft
- 2 Meine Körperpflege durchzuführen ist schmerzhaft, und ich bin langsam und vorsichtig
- 3 Ich brauche bei der Körperpflege etwas Hilfe, bewältige das meiste aber selber
- 4 Ich brauche täglich Hilfe bei den meisten Aspekten der Körperpflege
- 5 Ich kann mich nicht selbst anziehen, wasche mich mit Mühe und bleibe im Bett

Abschnitt 3: Heben

- 0 Ich kann schwere Gegenstände heben, ohne dass die Schmerzen dadurch stärker werden
- 1 Ich kann schwere Gegenstände heben aber die Schmerzen werden dadurch stärker
- 2 Schmerzen hindern mich daran, schwere Gegenstände vom Boden zu heben, aber es geht, wenn sie geeignet stehen (z.B. auf einem Tisch)
- 3 Schmerzen hindern mich daran, schwere Gegenstände zu heben, aber ich kann leichte bis mittelschwere Gegenstände heben, wenn sie geeignet stehen
- 4 Ich kann nur sehr leichte Gegenstände heben
- 5 Ich kann überhaupt nichts heben oder tragen

Abschnitt 4: Gehen

- 0 Schmerzen hindern mich nicht daran, so weit zu gehen, wie ich möchte
- 1 Schmerzen hindern mich daran, mehr als 1-2 km zu gehen
- 2 Schmerzen hindern mich daran, mehr als 0,5 km zu gehen
- 3 Schmerzen hindern mich daran, mehr als 100 m zu gehen
- 4 Ich kann nur mit einem Stock oder Krücken gehen
- 5 Ich bin die meiste Zeit im Bett und muss mich zur Toilette schleppen

Abschnitt 5: Sitzen

- 0 Ich kann auf jedem Stuhl so lange sitzen wie ich möchte
- 1 Ich kann auf meinem Lieblingsstuhl so lange sitzen wie ich möchte
- 2 Schmerzen hindern mich daran, länger als 1 Stunde zu sitzen
- 3 Schmerzen hindern mich daran, länger als eine halbe Stunde zu sitzen
- 4 Schmerzen hindern mich daran, länger als 10 Minuten zu sitzen
- 5 Schmerzen hindern mich daran, überhaupt zu sitzen

Abschnitt 6: Stehen

- 0 Ich kann so lange stehen wie ich möchte, ohne dass die Schmerzen dadurch stärker werden
- 1 Ich kann so lange stehen wie ich möchte, aber die Schmerzen werden dadurch stärker
- 2 Schmerzen hindern mich daran, länger als 1 Stunde zu stehen
- 3 Schmerzen hindern mich daran, länger als eine halbe Stunde zu stehen
- 4 Schmerzen hindern mich daran, länger als 10 Minuten zu stehen
- 5 Schmerzen hindern mich daran, überhaupt zu stehen

Abschnitt 7: Schlafen

- 0 Mein Schlaf ist nie durch Schmerzen gestört
- 1 Mein Schlaf ist gelegentlich durch Schmerzen gestört
- 2 Ich schlafe auf Grund von Schmerzen weniger als 6 Stunden
- 3 Ich schlafe auf Grund von Schmerzen weniger als 4 Stunden
- 4 Ich schlafe auf Grund von Schmerzen weniger als 2 Stunden
- 5 Schmerzen hindern mich daran, überhaupt zu schlafen

Abschnitt 8: Sexualleben (falls zutreffend)

- 0 Mein Sexualleben ist normal, und die Schmerzen werden dadurch nicht stärker
- 1 Mein Sexualleben ist normal, aber die Schmerzen werden dadurch stärker
- 2 Mein Sexualleben ist nahezu normal, aber sehr schmerzhaft
- 3 Mein Sexualleben ist durch Schmerzen stark eingeschränkt
- 4 Ich habe auf Grund von Schmerzen fast kein Sexualleben
- 5 Schmerzen verhindern jegliches Sexualleben

Abschnitt 9: Sozialleben

- 0 Mein Sozialleben ist normal, und die Schmerzen werden dadurch nicht stärker
- 1 Mein Sozialleben ist normal, aber die Schmerzen werden dadurch stärker
- 2 Schmerzen haben keinen wesentlichen Einfluss auf mein Sozialleben, ausser dass sie meine aktiven Interessen, z. B Sport einschränken
- 3 Schmerzen schränken mein Sozialleben ein, und ich gehen nicht mehr so oft aus
- 4 Schmerzen schränken mein Sozialleben auf mein Zuhause ein
- 5 Ich habe auf Grund von Schmerzen kein Sozialleben

Abschnitt 10: Reisen

- 0 Ich kann überallhin reisen, und die Schmerzen werden dadurch nicht stärker
- 1 Ich kann überallhin reisen, aber die Schmerzen werden dadurch stärker
- 2 Trotz starker Schmerzen kann ich länger als 2 Stunden unterwegs sein
- 3 Ich kann auf Grund von Schmerzen höchstens 1 Stunde unterwegs sein
- 4 Ich kann auf Grund von Schmerzen nur kurze notwendige Fahrten unter 30 Minuten machen
- 5 Schmerzen hindern mich daran, Fahrten zu machen, ausser zur medizinischen Behandlung

Nehmen Sie zur Zeit Schmerzmittel? Wenn ja: welche und in welcher Dosierung?

- Nein
- Gelegentlich, bei Bedarf
- Ja, ständig.

Unten gezeichnete Linie ist eine Schmerzskala.
Zeichnen Sie bitte auf dieser Linie mit einem Strich jene Stelle an, die Ihren momentanen Schmerz am ehesten widerspiegelt.
Die Linie beginnt links ("habe momentan keine Schmerzen") und endet rechts ("die Schmerzen sind momentan so schlimm wie nur vorstellbar")

