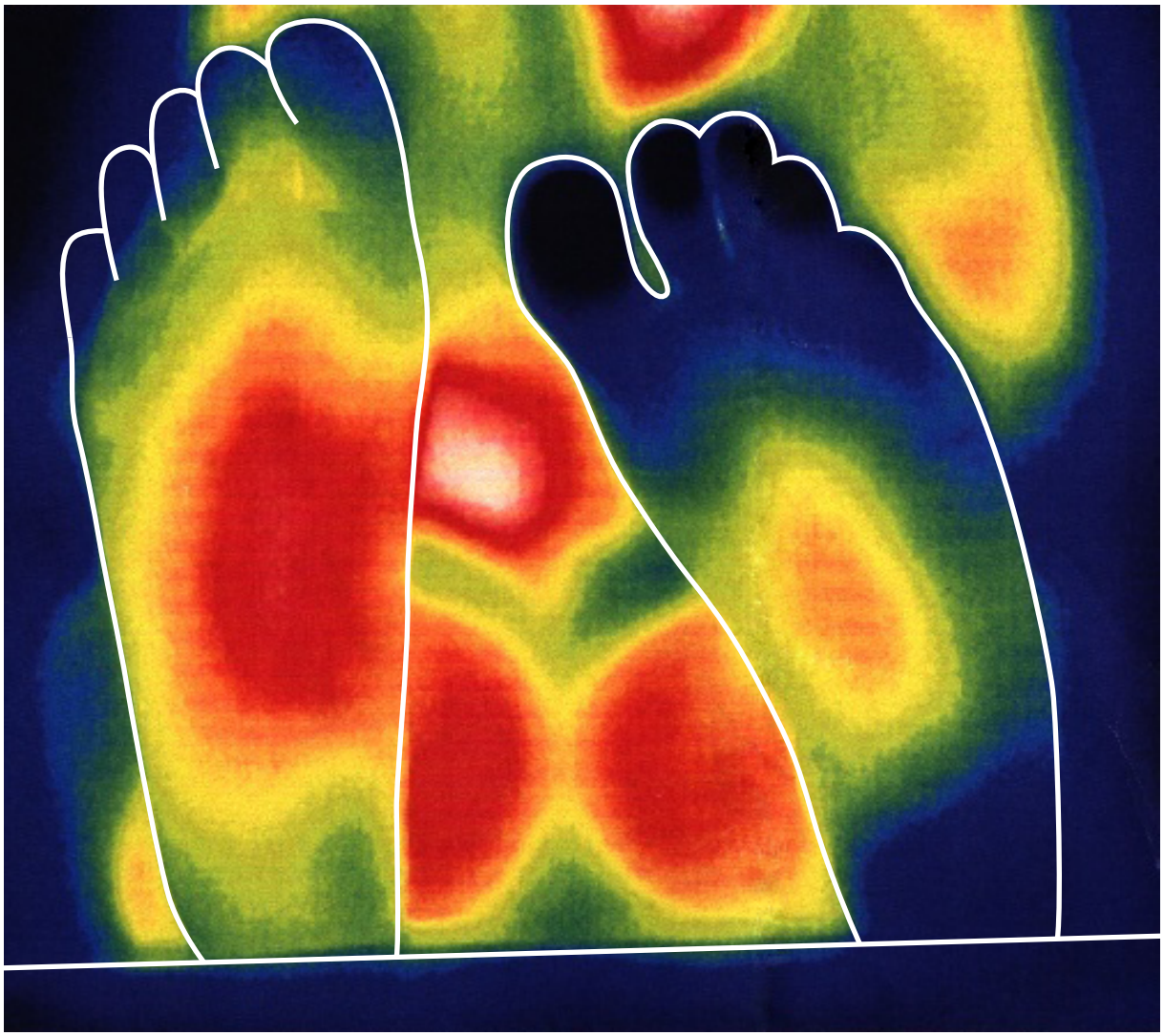
Is the Pain in CRPS-1 patients a result of continuing cramp and what is the importance of IMT in this?



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Master Thesis
by
Anton F.M. van Berkel

Is pain in CRPS-1 patients a result of continuing cramp and what is the importance of IMT in this?

By Anton F.M. van Berkel

Abstract

In this Thesis a conceivable explanation will be given about a part of the CRPS-1 problem. The comprehensive explanation in this study is limited to the lower extremity. CRPS-1 means: “Complex Regional Pain Syndrome”. The cause is unknown. An adequate therapy is unknown. Medicine already knows CRPS-1 for over 100 years. Complex means that several systems in the body are involved in the development and maintenance of this problem. The regionally of this syndrome will be denied.

There will be given a detailed specification of the consequences of the dysfunction of the Fibular Caput after inversion trauma. The reactions of the different tissues of the locomotors apparatus will be given in normal and subsequent abnormal gait.

The used therapy is: “Integrative Manual Therapy”™, hereinafter called: “IMT”. This treatment method is given at the Connecticut School of Integrative Manual Therapy (CSIMT) in Bloomfield. CT, USA.

From kinesiology, an extra symptom will be introduced to extend the diagnoses. The possible development of symptoms will be portrayed. A hypothesis will be introduced for the chronic character of CRPS-1. The proposed therapy for this group of patients is to correct the disfunctions of joints in the whole body. The Compression Syndrome Technique is used for improvement of blood circulation. Primary the mobilisation of the Fibular Caput to normalize his function is important for weight bearing transport during gait. The addition of an extra technique for muscle cramp proved to be essential for success.

If necessary, explanations will be given for the forearm.

Keywords:

Nerve pain, ischemic pain, fascia, tissue mobility, weight bearing, subluxation, fibular caput.

Is the pain in CRPS-1 patients a result of continuing cramp and what is the importance of IMT in this?

by

Anton F.M. van Berkel.

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science, the Department of Health Science of the Westbrook University.

July 29, 2010

Thesis Supervisor: David A. Frederick, Ph.D.

Department of Health Science

CERTIFICATE OF APPROVAL

MASTER OF SCIENCE THESIS

This is to certify that the Master of Science Thesis of

Anton F.M. van Berkel

has been approved by the Examining Committee and has fulfilled the thesis requirement for the Master of Science Degree in the department of Health Science.

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Gregory A. Baldt, DC., ND.

President

July 29, 2010.

Dedication

At the moment of finishing this thesis, I experience the feeling of achieving my goal. This was possible thanks to the help of many people. I like to thank them on this page.

Especially my wife, she gave me space for over forty years, to cultivate my profession on my way. During this long period I was often absent to collect new knowledge. She always has had a great understanding for my position. For this I am very grateful to her!

Anaesthesiologist Floor Groot, MD gave me the possibility to cooperate with him in the Pain Department in Breda, for twenty one years. We often could have a comprehensive discussion because he is an acupuncturist too. I thank him and I realise that my interest in CRPS-1 has started because of this cooperation.

Also, to my instructors of the Connecticut School for Integrative Manual Therapy in Bloomfield, I feel gratitude. The always interested and kind help of my professors at Westbrook University, the Open University in New Mexico, was very stimulating for me.

My patients should not be forgotten. I will thank them for their help to express their complains and problems. Including the help for photos in this thesis.

For the help with my writing I will thank Sharon Giammatteo, Ph.D., IMT.C. director of the IMT –School in Bloomfield, and Dr. Vincent Hoffmann MD, member of the Pain Department of the Amphia Hospital Breda. My colleague, Christine Dombroski. PT., IMT. for helping me with the translation into English.

Because of their help and spending time for brainstorming, I like to thank anaesthesiologist Henk van Driel MD, family doctor Roel Pierik GP, and anatomist Andry Fleeming, Ph.D.

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Preface

In 2005 I gave myself the assignment to write down my discoveries. In these times we had not any good therapy for CRPS-1. The results of my approach with IMT were very promising. In my opinion, the so-called Macedonian therapy, developed in 2006, is not good. This therapy gives unnecessary pain during the treatment.

My choice was to write a Master Thesis. By this I forced myself to give my statements a scientific foundation. Therefore this written therapeutic refinement will not be lost, and hopefully, a great deal of unnecessary sorrow can be prevented.

I hope my effort will be successful.

Almkerk (The Netherlands), July 2010

Anton F.M. van Berkel

1. Statement of the problem

1.1. The state of the art in CRPS-1

The International Association for the Study of Pain (I.A.S.P.) has given a classification for this syndrome in 1994 (Mersky and Bogduk).

a. The presence of an initiating noxious event, or a cause of immobilization.
b. Continuing pain, allodynia or hyperalgesea in which the pain is disproportionate to the event.
c. Evidence at some time of edema, changes in skin blood flow, or abnormal sudomotor activity in the region of pain.
d. This diagnosis is excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction.

The diagnosis is made by investigation of the signs and symptoms. How it looks, feels, moves and develops in time. There are four factors:

1. Pain; hyperalgesea, hyperesthesea and allodynia.
2. Temperature, asymmetry and color change.
3. Edema.
4. Motion; decreased range of motion, motor dysfunction and trophic symptoms.

The therapy consists of three parts: physical therapy, pain management for neuropathic pain and psychotherapy. If there is no progress in functional restoration and reanimation, the medication needs to change. Stronger analgesics are typically used. If after 10 to 16 weeks of no progress, a continuous epidural analgesia or neurostimulation (Spinal Cord Stimulation) is used.

The problem of CRPS-1 is complex, because of many causes without tissue damage to explain the syndrome. Trauma of the extremity is the cause in 65% of the cases. The use of cast after fractures, post surgical conditions, contusions, strains and sprains. Less common causes are spinal cord injury, CVA and cardiac ischemia (Wasner. 2003).

The syndrome has different problems in the acute and chronic states. In the acute state, joint and/ or skin pain, coordination and circulation failure are prominent. In the chronic state, problems with contractures, osteoporosis, muscular atrophy and psychological problems are prominent.

In general, the peripheral somatosensory abnormalities are difficult to quantify because of the subjective symptoms. And the complaints are not static. During investigations, the EMG is normal. Patient reactions to the therapies vary.

1.2. The cause and correct therapy is unknown

As already mentioned, the cause and the golden therapy are unclear. CRPS-1 usually begins with trauma, and the symptoms mimic neuropathic pain. Therefore, the therapeutic approach is as in neuropathic suffering. However nobody can explain this type of pain because of missing objectives. Scientists are looking for a motive to formulate a new classification and a new definition. On one hand it has symptoms of the central nervous system and on the other hand it looks like a rupture inside the peripheral nervous system. Why the persistent inflammation, pain and trophic disturbances at the distal aspects of the extremities? So far, there is no explanation for that question (Baron, Ralf. 2005).

Only the invasive Spinal Cord Stimulation (SCS), deep brain stimulation and motor cortex stimulation are successful in all patients.

1.3. Current therapy is not a "Golden Standard"

Because of the unknown cause of CRPS-1 we have no idea, which therapy to use to break the vicious circle (Why does not start definite repair?). Only invasive medical interference is successful in all patients for lowering the pain, like; sub cutaneous stimulation, deep brain stimulation and motor cortex stimulation. Quality of life can be influenced by the combination of pain management and physical therapy. Physical therapy alone has no good results (Kemler. 2000).

In present times Pregabeline is prescribed successfully. Pregabeline influences the pain conduction to the Central Nervous System. Distally Capsaicin ointment is used. It is known when early therapy has started; the syndrome will not grow into a chronic illness. Often rehab programs are successful after epidural blocks or after infusions.

During the acute phase all patients report of increasing pain, after sessions of physical therapy. Also here the reason is unknown. Pain management is multi- disciplinary. The rehabilitation specialist, the physical therapist, the occupational therapist and psychologist work close together. The therapy consists of lowering the pain (medication), physical function rehab, Pain Exposure Physical Therapy (time dependent) and if necessary psychological functional rehab (cognitive training) (Congress Nijmegen. 2009).

The therapy still consists of "Trial and Error" (Meijler, W.J. 2006).

1.4. The impact of CRPS-1 on patient's life

The impact of uncomprehended pain is enormous; physical as well psychological. The violation of this syndrome on patients' lives is particularly notable.

Children are often absent from school, they cannot play outside with their friends and time is lost from visiting rehab centres or hospitals. Delay risk of physical and psychological

development is present for them during a prolonged pain period. In children with CRPS-1 there is a preference for girls.

In adolescents and adult, there is usually we a big psychological impact. Anxiety for a forced change in their choice of profession is huge. Self-image, character, cognitive and behaviour patterns and ambitions will change (psychosocial disability factor). Pain-specific avoidance behaviour are developed and so functional disabilities arise. Because of the anxiety to move, the patient bumps the affected extremity more often as usual. Bumps always cause increased pain. There is more pain, after moving or weight bearing during gait. In an acute episode, patients can lie in bed for days. Outside the house they move by wheelchair and indoors they use crutches. The fact that the pain returns during all kinds of functioning implies the danger of anxiety for movement. They do not leave the house anymore and negativism and even deprivation can easy develop. The risk to become a chronic pain patient increases in such periods.

In the medical literature, it is emphasized that these patients use help aids during long periods. This is much longer than in the case of “normal” inversion traumas with less to none tissue damage. The use of crutches and wheelchairs can take months to years, and sometimes for the rest of their lives.

Abnormal functioning also provokes body pain outside the afflicted extremity, such as back pain and shoulder pain after walking with crutches. After treatment sessions or after “normal” functioning there is, in the acute phase, edema and stiffness. Patients often report tingling sensations as well. Because of the stiffness we hear complains of “ My legs are like lead”, increase in pain and loss of coordination. In extreme cases, after wrong therapy or bad luck, amputation can be necessary at the end of a long lasting suffering.

CRPS-1 is a general social problem. In severe cases, one has to look for another job. Sometimes resumption of work is impossible, because of the severe handicap. Public health

costs are increased by the monthly pay out, and the expensive adaptations for the patient, and/or the patient's home (Wheelchair, stairlift, bathroom etc.).

CRPS-1 is not only a great burden for the patient, but also for the whole family. We often see a divorce because of inability of the partner to handle the situation. The patient is left behind with another negative experience.

The experience of loss of work has the biggest impact on the quality of life. Research conclusion is that people do not always rate pain as affecting their quality of life; rather pain interferes with how life is lived and not its quality.

1.5. Can IMT improve blood circulation in CRPS-1?

Chronic neuropathic pain is characterized by spontaneous pain. One of the main problems in CRPS-1 is the diminished blood distribution into the extremities. Because of that repair can not be started in a sufficient way. The pain will be increased in time by central sensitisation. It is unknown if it is the sensitisation that holds the inflammatory situation. A cascade of immune cell types, such as mast cells, T-lymfocytes, TNFα, monocytes, substance P, etc. are involved for the sensation of pain (Thacker, M. A. 2007). Because of these mediators, vascular changes play a role, such as vasoconstriction and vascular leakage. Subsequently, there is an insufficient blood supply for normal tissue repair. This is called an ischemic situation.

IMT therapists have techniques to promote vasodilatation. These hands-on techniques are called "Compression Syndrome Techniques". By this technique the process of repair can be initiated (Several workbooks of IMT School). The skin color normalises and the edema disappears.

1.6. Conclusion of section one

CRPS-1 consists of several symptoms. It seems that one symptom leads to another symptom. Asking the patient, pain is the most important symptom. In a therapeutic way, pain is a complex problem. My opinion is that the pain and the insufficient blood circulation are the most elementary problems in CRPS-1. Both are immediately related. Both are present if the extremity is moved. As soon as the patient moves, a cycle of pain, impotence and a discoloration of the skin appear. So to speak: “The patient is caught in a complex circle of pain” To burst through this circle, we have to investigate the factors such are important during this movements.

These factors are:

a. The lack of understanding. Why the discrepancy between trauma and pain?
b. The Ischemia. Especially, pain and ischemia are related.
c. Wound repair. This is possible when there is sufficient blood supply. And, correct healing means correct moving during the healing process.
d. Correct moving. Correct, physiological movements depend of different levels. The condition of cells, joints, whole extremity and whole body.

2. Review of literature

Before orientating the medical literature, I have tried to base my therapeutic approach by attentive listening and observing. CRPS-1 is traditionally seen as a chronic neurological disorder. At the start of my research 95% of my patients had experienced a trauma. Because of this, the information collected for this thesis, not only explores neurology and anaesthesiology, but also kinesiology and sports medicine. In addition to the research of literature, I visited several congresses and consulted with scientists.

2.1. Publications of the International Association for Study of Pain (IASP)

1. Abstracts. 11th World Congress on Pain. Sydney, Australia. 2005.
2. Pain 2005. An updated review; 293-389.
3. The Path of Pain. 1975/ 2005.
4. CRPS: Current diagnosis and therapy. Progress in pain research and management volume 32.

2.2. Workbooks of the International Academy for Sport science

2.3. State of the Art on CRPS in the Netherlands. Several congresses, course informations and publications

1. Hoogeveen. Congress. Rehab Centre “Bethesda”. Januari 2005.
2. CRPS Course. Nederlands Paramedisch Instituut. 2006.
3. Pain Exposure Physical Therapy. Congress UMC St. Radboud. Universiteit Nijmegen. NL. March. 2009.
4. Publications, Nederlandse Vereniging voor Dystrofie Patiënten [Dutch Association for CRPS-1 patients]

5. Nederlands tijdschrift voor fysiotherapie [Dutch Journal for Physical Therapy] Febr. 2003; 113: (1): 2-10.
6. Nationale Pijndagen [National Pain days]. Amersfoort 2006.
7. Nationaal Pijnfonds [National Association for Pain]. Leiden.

2.4. The Internet Libraries

1. The Cochrane Library.
2. Pub Med. US. Nat. Library of Medicine.

2.5. Publications of the Interdisciplinairy World Congresses on Low Back & Pelvic Pain

1. San Diego, USA. 1995.
2. Vienna, Austria. 1998.
3. Barcelona, Spain. 2007.

2.6. Fascia Research. Congress at Boston. October 2007

2.7. Dissertation en publications concerning the psychological facets of pain

1. Over de Pijn. Prof. Dr. F.J.J. Buytendijk. 1943.
2. Chronische Pijn, het Kruis van de Geneeskunde. A.M.E. Vrancken. MSc.
3. The role of fear of movement/ (re)injury in pain disability. Vlaeyen, J.W.S.et al.

2.8. Energy Medicine

1. Pain congress. Utrecht. NL. 2008. Coherence in Health.
2. Energy Medicine, the scientific basis. James L. Oschman. 2000.
3. Energy Medicine in Therapeutics and Human Performance. J.L. Oschman. 2005.

2.9. Anatomical Atlasess

1. Gray's Anatomy. 15th. Edition. 1901.
2. Handatlas der Anatomie des Menschen. Spalteholz-Spanner. Edition 16th. 1960.
3. Gray's Anatomy. Edition 38. 1995.

3. The method

3.1. A new rehabilitation program for CRPS-1 patients in 2006

After the CRPS-1 congress in Hoogeveen, Holland, change was introduced to the therapeutic approach in dystrophy patients. Until the year of 2005, physiotherapy sessions were within the pain threshold. Doctors and therapists of the Rehab Center “Bethesda” in Hoogeveen heard about great success of patients who visited a female therapist, Mrs. Shinka in Macedonia. The therapy, used by Mrs. Shinka is as followed. With one hand she massages the injured tissue while the other moves the adjacent joint at the same time. She does not pay attention to the pain. Immediately after this mobilisation the patient has to move as much as possible. This is repeated usually for three days. The patients relate painful treatments, but the success was great. Approximately 55% were completely healed after one week. A click was often sensed during the treatment, sometimes heard too. After that the healing was more progressive (Ek, .J.W. 2004).

The intention is to promote this kind of therapy in the Netherlands. Directly after massage of the trigger points, the joints are moved in a passive way. This will be done as much it takes until the patient can move by himself. The subtitle of the Macedonia therapy in Holland is: “Function first pain later”.

During this therapeutic approach, the case history is not taken; during 45 minutes two physiotherapists are working with one patient. The total sessions are five, once a week. The partner of the patient is present during the sessions to prevent a mild attitude to the patient. Talking about pain is forbidden. Hoogeveen claims a good result of 42%.

What I noticed:

1. During the treatment of above written method, the volume of pain is huge (Observation by myself).
2. The great step forwards after the “click”. In the Australian literature we can read this statement too (Congress book Sydney 2005).

At the moment of trauma two factors are important and both are present:

A. Pain.

B. Instability.

Fig. 1. Pain and instability.

“The most significant feature of the bodily reactions in pain…..is that they are of the nature of reflexes”.

(Cannon MD, W.B. 1920).

3.1.1. About pain in CRPS-1

The definition for pain, given by the I.A.S.P. is: “Pain is an unpleasant sensory and emotional experience with actual or potential tissue damage, or described in terms of such damage”.

In my opinion, this is too gentle of an expression. The CRSP-1 patient does not talk about unpleasant, but about continuing excruciating pain, especially in the acute phase, if the foot is placed on the ground or touched. International we speak about derailment of the innervations of the sympathetic nervous system. Therefore the patients find themselves in a vicious circle of negative factors which influence each other.

The pain system

This consists of nociceptors in the periphery, a peripheral nerve, the dorsal root of the spine, a central nerve and the cerebral cortex where the pain awareness is. The brainstem plays a crucial role in the transmission of the nociceptive stimulus of the cortex. The PAG is an important intermediate stage. Out of the brainstem a descendent network consists of pro- and anti-nociceptive components. In this, the Insula is important as well.

Nociceptors are receptors for pain, such as the organelles of Pacini, Meissner, Golgi, Krause etc. Once activated, they transport impulses to the brain as mentioned above. The nociceptors are situated the skin, locomotor apparatus (muscles, tendons, capsules, fascia), parietal membrane, visceral membrane and parenchyma of the organs. The density of the nociceptor innervations is decreasing from exterior to interior. In the skin we find the highest and in the organs the lowest number of nociceptors. The fact that all these nociceptors are spread throughout the whole body is important to understand. We always have to realise about the attendance of pain in a number of locations. Often the patient points to the spot where they feel the most intensive pain (Kellgren. 1938).

The sensory nerves are divided into:

a. C-fibre nerves not myelinated, and have slow impulse transport. Create nagging and emotional pain. Peripheral sensitization, which is the enhanced responsiveness of primary afferent nociceptors to natural stimuli plays a significant role in hyperalgesia. Under inflammatory conditions sensory neurons are commonly sensitized to mechanical stimuli, especially from deep tissue.
b. The Aδ- fibres myelinated. They are important for the first pain awareness, and have fast impulse transport. It is to pinpoint the pain and the withdraw reflex.

General arrangement of neurological pain:

1. Nociceptive pain, this is normal pain, such as a pin-prick.
2. Inflammation pain, if a sterile inflammation arises in the area of a nerve.
3. Referred pain, the cause of the pain is not locally situated. For example, pain in the skin stemming from organ disorders and myofascial pain caused by inter vertebral joints (Travell, J.G. & Simons, D.G. 1983).
4. Neuropathic pain, in the case of damaged tissue.
5. Sensitization pain, chronic pain which is experienced worse because of lowering the threshold of the pain. This occurs because of neuroplasticity of the dorsal root ganglion, for example after chronic inflammation.

In CRPS-1 patients we find the first four kinds of pain. In the case of chronic conditions all five are present.

Important remark about pain:

Pain is individual!

One of the known pain researchers, Dr. P.D. Wall published in 1985 the next three findings:

1. The active threshold for C-fibres is not the same as for pain. Sub-groups of C-fibres transport another impulse frequency.
2. The threshold for pain is individual and is different for every kind of pain. Pain by electric shock, pain after frost, etc. (Everybody has his own, individual network out of the brainstem. The "body-self neuromatrix" Tracey, I. 2007).
3. Different frequencies of noxious stimuli can produce pain awareness. Some frequencies do not produce pain, such as 0.4 Hertz.

Pain has an alarm function

The patient thinks pain has the function of a warning system. Because of the intensive experience, every patient is convinced of much tissue damage. Most of the time it is not. Also, the EMG does not give abnormalities in CRPS-1. Because of this, anxiety for moving arises (LeDoux, J. 2000., LeDoux, J. 2002., Delgado, M.R. 2008).

Specific for CRPS-1 pain is:

- Sensitization- peripheral because of mechanical stimuli are converted into electrical signals in the case of local inflammation. Central sensitization increases because of excitation of the dorsal root ganglion of the central nervous system. The nociceptive C-fibres, our chronic alarm fibres are not adaptable. As long as the cause of the pain does not disappear, the alarm signal continues to grow (Graven-Nielsen and Arendt-Nielsen. 2003).

- Hypersensitivity for thermal stimulus - the Aδ-HTM-nociceptors, which are sensitive for mechanical influences, are sensitive to thermal stimuli as well (Kajander. K C. 1987). Often, after returning from a warm country, we hear patients complain about more pain as in their holiday surroundings.
- In the case of tissue damage around a joint, the joint afferents will sensitize (Schaibe. 1993). Patients complain about increasing pain after repetitive bumping of the foot. The sensitization is created by the neuroplasticity of the dorsal root.
- Painful touch can be present. This is a serious handicap for the patient. During sleep the contact with blankets, during a shower bath with the jet of water, etc. The pain originates because of central sensitization in the brain. This is demonstrated with the help of fMRI, during research of pain transport in the brain by the team of Dr. Irene Tracey of the university in Oxford, England. In patients with this problem the team found changes at the posterior parietal cortex, in the somatosensory region (Leknes, S. 2008).
- Hormones play an important role as pain mediators in female dystrophy patients. During wound healing pain is created by Pain Producing Substances as histamine, substance P, serotonin, bradykinin, etc. The pain increases during menstruation (Veldhuijzen, J. 2005). The female hormone progesterone is a steroid hormone important for peripheral nerve repair. It is produced by the ovaries. Young girls have a return of their pain during the first periods of menstruation. Perhaps important relations can be noticed? (Koenig, H. L. 2000).
- Ischemia during isometric muscle contraction is not understood. CRPS-1 patients are walking with these contractions because of inflexibility in the foot. It appears the leg is in continued muscle cramp. Moving means increase of pain.

The therapeutic approach of pain in CRPS-1 after 2008:

We distinguish three subdivisions; the first is least understood:

1. Structural model - somewhere there is tissue damage in the locomotor apparatus.
2. Psychological model - if no tissue damage is found, cause must be psychological.
3. Neurophysiologic model- long-lasting pain creates sensitization.

The problem with chronic pain is there is little differentiation between the divisions. In 2009, the third model has the focus. CRPS-1 pain is treated like neuropathic pain despite no tissue damage. The conclusion is, chronic muscle-skeletal pain is explained by a neurophysiologic model. This is based on the physiology of the nervous system, instead of on structural damage of the peripheral tissues like bones, ligaments, muscles, etc.

The conclusion is: CRPS-1 patients are never in the same class. Traumas cannot be compared with one another. Everybody interprets pain in his personal situation of experience and hereditary character. Furthermore, the duration is important. Pain can still be experienced centrally, while the distal cause already has stopped. Pain in dystrophy patients often does not disappear notwithstanding movement stops. This can continue for days after minor movements. Pain behaviour and the experience of pain can give an inside view of this obstinate syndrome. In my opinion, pain during movement is the most important symptom. I start with the “hands-on expert”.

3.1.2. Pain perception in CRSP-1 patients, some interpretations

a. My pain can be a burning feeling, like a nurse had pulled tight a bandage around my foot. I feel this the whole day. Sometimes it feels like stabs, and the bones feel cold and warm however the foot is always cold.
b. My pain is burning. It never goes away. I cannot imagine being without my pain.

c. My pain does not feel the same in my whole body. In different places it has different feelings. One spot can feel very sharp, another spot feels too short as if the muscles are too short. And there is a tingling pain. Sometimes I feel threatened by the pain. It gives me the feeling that any moment my bones can break. During these moments I can be very anxious and I lose control of the situation.
d. My pain is nagging. Localized in my back. Beside that I have a continuous headache. It feels like they are fighting with each other. If I'm tired, let's say after a walk, the pain feels like fire but I can't remove it. I have no control and lie down waiting for the moment it will fade away.
e. I feel it as pain in my nerves, same feeling as a nerve is touched by the dentist. My right leg hurts that way, like my toes get electricity out of my back. It feels like my leg is always accompanied with electricity - sometimes more, sometimes less. It feels like a numb leg and someone sticks needles or knives in it.
f. During good periods, when I have less pain, I set off for doing more. Then, a little bump is sufficient, to get back the old, burning pain.

3.1.3. Observations of the CRPS-1 patient by the therapist

In general, weight bearing on the affected foot is impossible. Most of the time, the foot does not rest on the ground. If the foot is placed on the ground, we see a kind of clubfoot. The knee is in hyperextension. Weight bearing is on the lateral side of the foot (see Fig. 2).

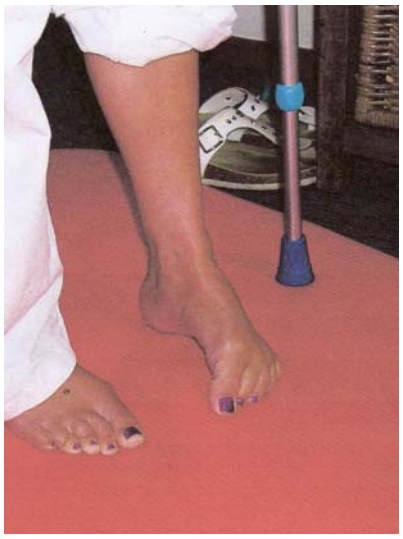

Fig. 2. Weight bearing.

The leg is positioned in internal rotation. The longitudinal arch of the foot is kept high, the meta-tarsus kept to medial and during walking, no propulsion of the body-weight is seen with the traumatized foot (see Fig. 3).

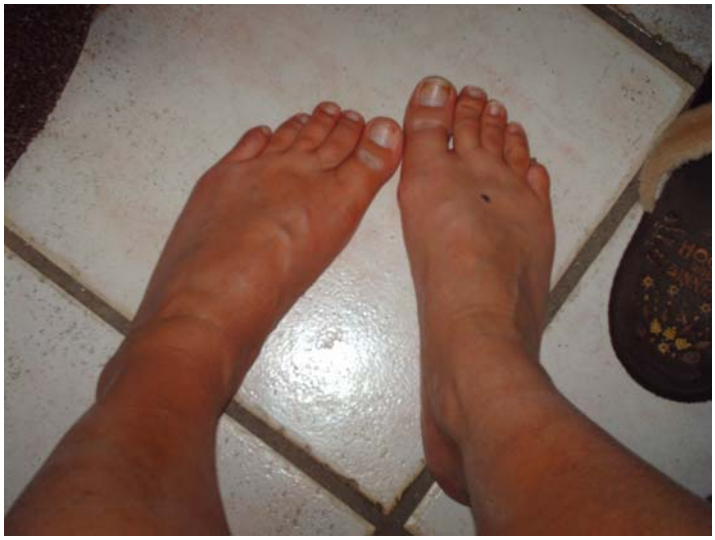

Fig. 3. Long arch of foot.

No heel strike exists. If the foot does not rest on the ground, the foot is drooping in inversion. We see a discoloration because of blood circulation problems (see Fig. 4).

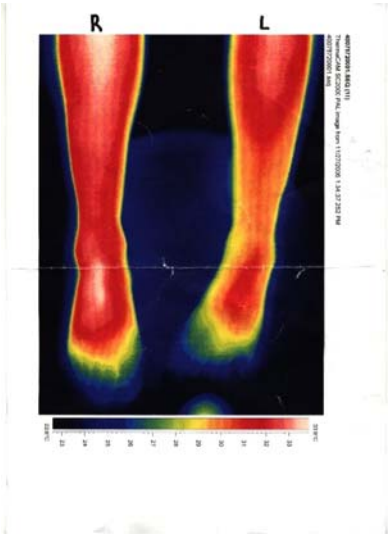

Fig. 4. Discoloration of foot.

Physical inspection shows a limit of eversion and dorsal flexion. Often, active motion of the ankle is disturbed. The toes are shortened/ shrunk, and joint-play has disappeared (see Fig. 5).

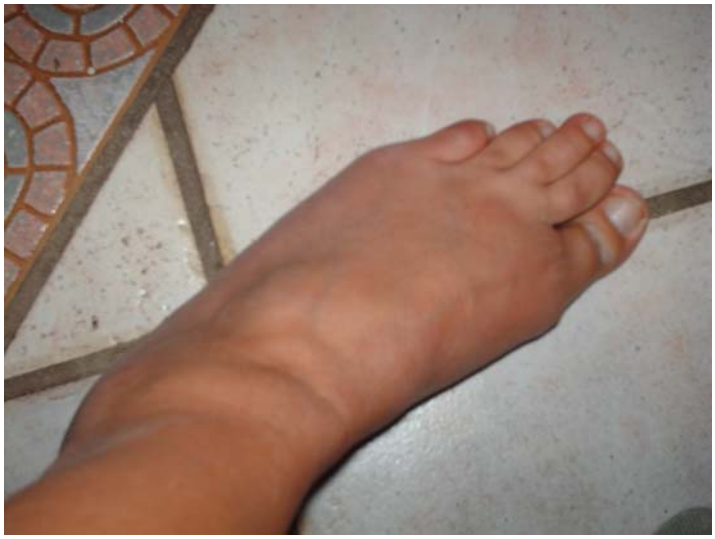

Fig. 5. Shortened big toe.

Active eversion and dorsal flexion of the foot is often impossible, forced by a temporary pareses (see Fig. 6).

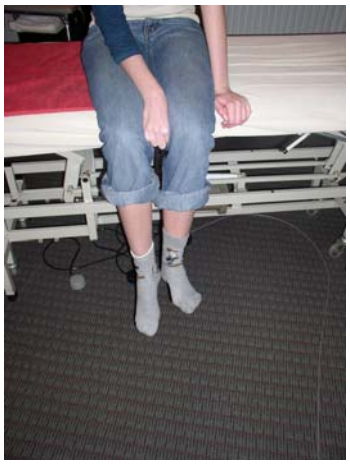

Fig. 6. Disturbed eversion.

The patient is trapped by his/her lower leg! Besides pain in deeper tissue during the acute phase, hyperalgesia may be present in the whole leg or the lower leg. However, if the patient has improvement, some places show preference for localisations where the pain stays longer.

These pain regions are (see Fig. 7):

1. Metatarsal one, two, four and five.
2. Tuberosity of fifth metatarsal.
3. Navicular-Cuneiform joint.
4. Calcaneum lateral side.
5. Lower leg lateral side.
6. Anterior intercondylar area of Tibia.
7. Lateral side of Anterior Superior Illiac Spine and Iliac Crest/ greater Trochanter region.
8. Sacro-Iliac joint.
9. Thoracic between the Scapulae and Cervical.

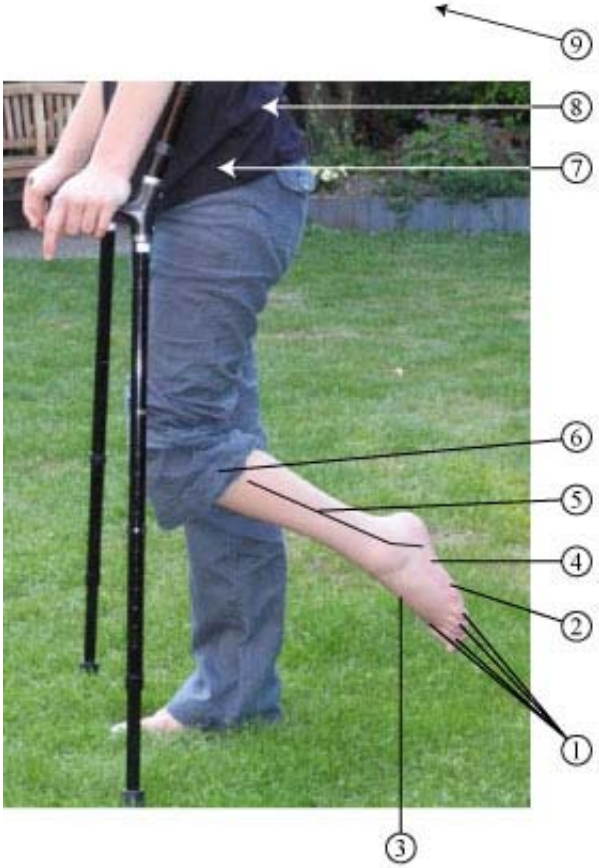

Fig. 7. Locations of pain.

If patients have suffered from CRPS-1 for many years, we see a diminishing of dimensions in the foot or the hand (size of shoe/ glove).

3.1.4. Pain, compression syndromes and IMT

A compression syndrome is a sustained neuro-reflexogenic protective mode. The body has the ability to protect itself. This self-protection is reflexogenic, autonomic and automatic. It can go from the little contraction, as in wound healing from a little cut in the skin, to the biggest contraction when we shrink into our self, in the case of a big danger. In between these two, we can find protective contractions at all levels in the body. Protective modes are useful in the case of tissue damage. The damaged tissue needs this protection until it heals. But, if the protection mode lasts after tissue healing, it will affect health. The compression becomes pathologic, and is called a compression syndrome.

Often, compression syndromes are situated in diaphragms, ligaments, tendons, joints and especial in connective tissue. The result of a compression syndrome is a long lasting contraction, which can create pain. This means decreased movements where the syndrome is localized in the body. Decrease of normal blood circulation and lymph will start chronic inflammations.

Health means to have the ability of free movement with free information throughout the body (Oschman, J.L. 1993. / Oschman, J.L. & Oschman, N.H. 1994). If movements are restricted locally, free flow of information to other parts of the body is restricted. On the electric level, we must think of information by the piezoelectric effects are disturbed inside a compression syndrome (Barnus, A.J. 1995. / Szent-Gyõrgyi, A. 1941). In this case, healing can not begin. If we do not neutralize the syndrome we become ill. So, if complete health does not return after physical or emotional injury or trauma, we have to look for the sites where continuity of mobility and motility is restricted. Therefore we look for problems of joint dysfunction, muscle spasm, circulation insufficiency, pain, limitations of motion, and more. With correct functioning connective tissue, existing everywhere in the body, movements are performed well. This connective tissue, fascia, is the tissue which envelops the locomotor apparatus. I will give an extensive writing to this subject in section 3.2.7.

During practical training for Integrative Manual Therapist many techniques are instructed to eliminate a compression syndrome (Workbooks School for IMT).

3.1.5. About pain and muscle cramp.

The definition of muscle cramp is: “An involuntary and forceful contraction that does not relax”.

A cramp can be present in all kinds of muscles. It exists in both skeletal muscles and smooth muscles. In skeletal muscles, there is a preference of the tonic muscles like the calf

muscles, quadriceps and hamstrings. This phenomenon occurs often, the cause is sought in a deficit of oxygen in muscles, however more research is needed. A cramp is always accompanied with pain and can vary from mild to unbearable (Rüger, L.J. 2008).

Possible causes for cramps are incorrect stretching and fatigue. At the moment of fatigue, the coordination is not correct anymore. Movements are not good. Dehydration and depletion of electrolytes result. This happens especially in endurance sports, the cramp occurs in the above mentioned muscles.

The treatment is: “Stop the activities” Perform a quiet stretch and wait until the muscle relaxes. According to the Cochrane Register, Dry-needling of Trigger points is the only effective conservative therapy in muscle cramp. Trigger points are little local contracted regions which can restrict movement, because of that the locomotion is bad too (Arendt-Nielsen, L. 2009).

In conclusion, muscle cramp can give unbearable pain. It consists of components of neuropathic pain if it is prolonged (CLI or Critical Limb Ischemia). (Bennet. M L. 2005). Partiality localised in the lower limb and front arm. One of the causes is insufficient blood circulation. In particular, incorrect moving and postural strain are often mentioned (Lundberg, U. 1999). Important in CRPS-1 is the deficit of oxygen during chronic infections, it can produce a cramp with accompanying pain. Muscle pain with movement anxiety will give a bad locomotion too (Vlaeyen. J.W.S. 2000).

3.1.6. Is the pain created by a neuritis?

CRPS-1 is treated like a neuropathic illness, notwithstanding the lack of tissue damage. The EMG is not anomalous. Often, the functioning has decreased, but not always! The question is: “Has the experienced pain a neuritis cause?”

A neuritis factor was demonstrated by a research team of the Erasmus University in Rotterdam in 2006. Pro-inflammatory mediators were shown in CRPS-1 by F.J. Huijgen. MD. The fluid of artificial made blisters was studied. The healthy extremity was compared with the afflicted extremity. The levels of IL-6, TN-alpha and ET-1 were increased in the afflicted extremity versus the healthy extremity. The conclusion was that NOx/ET-1 ratio is disturbed and therefore there is a vasoconstriction with diminished blood circulation in the afflicted extremity (Groeneweg, J.G. 2006). The sympathetic efferent nerves in the peripheral tissues have synaptic contacts with sensible afferents. Therefore it is possible that CRPS-1 patients are caught in a neurologic circle, because of the above mentioned neuritis of the sympathetic neurological system.

3.2. About stability of the CRPS-1 patient

3.2.1. Normal gait analyses

To understand the pain and stability difficulties in deviated gait, normal walking will be analysed (Perry, J. 1992), particularly since we do not find abnormalities in CRPS-1 patients.

Walking is divided in two phases, stance phase and swing phase. Stance phase will be divided into two parts because the patients cannot make heel strike, and don't make weight propulsion. The two parts of the stance phase are:

a. The heel strike.

b. The body-weight propulsion.

The muscular activities:

Subsection a. At the moment of heel strike the hip is flexed, the knee near full extension. This is stabilised by the gluteus maximus, the quadriceps muscle and the

hamstrings in the upper leg and calf muscles of the lower leg. Directly after heel strike the hamstrings relax. During normal walking, floor contact of the calcaneum is abrupt after a free fall of one cm. As soon as the heel contact is made the anterior tibial and peroneal longus muscles tighten for absorption of the free fall. The origin of the anterior tibial muscle is at the proximal tibia and the interosseous membrane, medial side, and crural fascia. The origin of the peroneal longus muscle is fibular head and also the crural fascia but the lateral side. Both have their insertion at the head of the metatarsal one and medial cuneiform bone. Both muscles act as a stirrup to catch the impact of the heel strike. Peroneus longus tendon has a special route around the lateral aspect of the cuboid bone and is used as a hypomochlion.

Patellar tendon at the knee also acts for shock absorption at the moment of heel strike.

Subsection b. First contact of body-weight with the ground is heel strike. After that, the peroneus longus and anterior tibial muscles activity change from impact braking to guiding the lower leg. They stabilize medial and lateral movements of the ankle joint, together with the peroneus brevis muscle. Then body-weight propulsion proceeds to midfoot, to the metatarsals and to the phalanges. During this motion the knee is stabilized by the adductor magnus at the medial side and the long head of the biceps femorus at the lateral side of the upper leg. Meanwhile the gluteus maximus stays tense. The quadriceps muscle and plantar flexors work together at midstance. In the terminal stance, just before toe-off, the plantar flexors are the most active.

The muscular movements during swing phase:

Three moments, during swing phase, are important for the CRPS-1 patient.

1. Pre-swing phase - This is, as soon as the foot departs the ground, the extension activity of the quadriceps stops. Hip flexion and prolongation of knee flexion starts

with tightening of the psoas muscle. The short head of the biceps femoris muscle has a small moment of tightening. The biceps femoris (short head) has its origin at lateral intermuscular septum. Insertion is at fibular head. The fibular head moves into dorsal position by the short pull of biceps femoris brevis.

2. Knee movement. The lower leg swings forward and at 60 degrees flexion of the knee, the tibia makes a rotation of 8 degrees in transverse plane and also 8 degrees in coronal plane.
3. Swing through until heel strike. The ankle is at the moment of toe-off in plantar flexion. Along the swing phase, from back to front, the position of the foot will change from plantar to dorsal flexion by the foot extensors. Swing phase ends at the moment of heel strike. Preparation to control heel strike is done by tightening the gluteus maximus. Besides stabilizing the hip, the ilio-tibital tract is tightened by the gluteus maximus to prepare the control of the extension of the knee. The ilio-tibial tract of fascia lata pulls the fibular heaad back in anterior position. Extension of the knee proceeds by tension of the quadriceps muscle and stabilized by tension of the hamstrings. Heel strike ends the swing phase.

The distal part of the fibula (lateral malleolus) has made a forward/ backward movement. During the swing phase it is posterior, during stance phase it is anterior. The fibular bone can made a superior/ inferior movement too. We need this possibility if the surface of the ground on which we stand is not level (see Fig. 8).

During this movement of the fibula we see a kind of

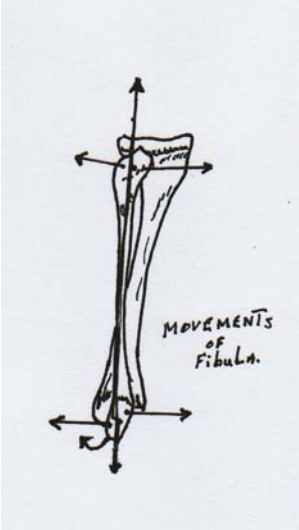

Fig. 8.

Movements of filula.

locking and unlocking. Because of the forward/ backward movement of the proximal fibula the cuboid bone is rotated in the foot at the distal side (Bojsen-Møller, F. 1979).

In this chapter I have showed the importance of the cooperation of the anterior tibial muscle and the longus peronal muscle for correct heel strike. CRPS-1 patients have a great problem with that (see also “closed packed position” under section 3.2.2.).

3.2.2. Why do CRPS-1 patients need crutches for stability?

A closer look at standing and gait, a possible cause for CRPS-1:

The inability to bear bodyweight is a frequent symptom in CRPS-1 patients. After trauma, crutches are needed for an abnormally long period.

The cause can be:

1. Neurologic.
2. Orthopaedic.

Often, it is both.

Subsection 1. Often, the neurological cause is a strain of the peronal communal nerve during an inversion trauma. The motor and sympathetic functions of the nerve, which create the possibility of the movements and maintaining the sensibility of the lower leg, are ceased. The duration of a paralysed peroneal nerve in CRPS-1 is very variable.

Subsection 2. An orthopaedic cause can be found in the functioning of the knee and the tarsal bones of the foot by deviated bodyweight load bearing or sudden muscular hypertension at the lateral side of the ankle.

To understand the functioning of the foot I will describe the movements of the bones. The muscular movements are written in section 3.2.1.

The movements of the bones during stance phase:

Osseous movements, during stance phase of gait, are traditionally written as segmental movements of the longitudinal arch (Bojsen-Møller, F. 1979). The arch is important for weight bearing during walking and standing. We distinguish three segments:

1. The rear foot or calcaneum.
2. The midfoot.
3. The first ray or MT-I.

The tibia and fibula are situated on top of the arch. The talus is situated in between the calcaneum and the tibia.

During stance phase, weight bearing can occur in two ways. The arch can be loaded in two different ways:

1. Toe off is performed by the first ray. Like ordinary walking is.
2. Toe off is performed by the four meta-tarsal bones of digits two through five. Like walking on tiptoe is.

Most part of the researches makes theoretical statements about motion around the longitudinal axis of the midfoot, which is opposite to the rear foot. This longitudinal axis is from the calcaneus to the MT-1 head. See Fig. 9, axes A. The navicular bone performs inversion movement at the moment of heel strike. When the bodyweight is transported forward, the navicular bone makes eversion (Shephard, E. 1951).

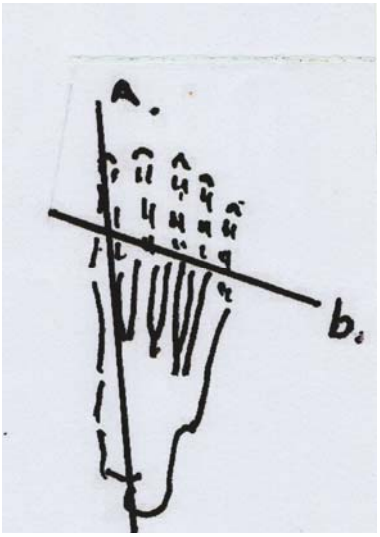

Fig. 9. Axes of foot.

The magnitude of this navicular movement relative to the calcaneum was found to be 1.9°. The movement of the midfoot means the motion of the midtarsal joint. Consequently the midtarsal joint moves during the stance phase, when the bodyweight comes forward. This joint consists of four bones. At the proximal side of the foot, the talus and the calcaneum and at the distal side the navicular and cuboid bones. Anatomically we have two joints. Apparently, there is an assumption that the navicular and the cuboid bones move the same way. It is supposed one physiological moving unit. But don't exist movements between navicular and cuboid bones? (CC joint). Most of the research, done about weight bearing during walking, is transport by the first ray.

Let us look to axes A

Hicks has a "windlass mechanism" explained in 1954. This is the mechanism for tension of the arch of the foot during walking (Hicks, J.H. 1954). The stance phase used for this mechanism is from heel strike to (Big) toe-off. The effectiveness of this windlass depends on the length of the plantar aponeurosis in relation to the longitudinal arch, and the radius of the aponeurosis. This mechanism explains the approximation of the bones in the longitudinal arch. The plantar aponeurosis inserts anteriorly on the proximal phalanges of the toes. When the toes are dorsiflexed during propulsion, the aponeurosis is wound around the heads of the

metatarsal bones. Mostly, on the MT-1 side because of the two sesamoid bones. The two extremities of the longitudinal arch are thereby approximated and the arch is tied together. At the same time the cuboid bone is rotated into inversion (pronation). by tightening the peroneous longus (see Fig. 10 and Fig. 11). The cuboid bone comes in a close packed position. This is a closed packed position for the CC-joint. Then the cuboid bone is in a locked position. In this case the stability of the midfoot is excellent if the push off is made by the big toe.

The second axes B

Another oblique axis can be used during push off. This is the line through the heads of metatarsal 2/ metatarsal 5 (see Fig. 9). The CC-joint is loose packed in that situation, and therefore the stability is decreased. As mentioned, we can compare this with walking on tip toe.

The anatomical shape of the CC joint

For the diagnostician and therapist in CRPS-1 patients it is extremely important to understand the CC joint (see Fig. 10). The knowledge of the closed packed and loose packed position of the cuboid bone during the stance phase will give clearance.

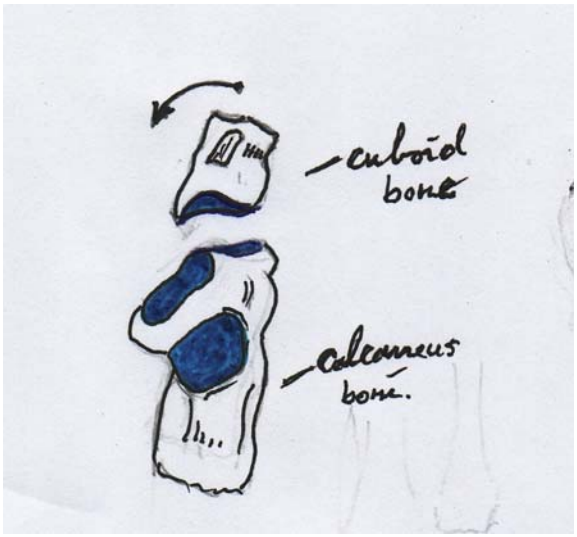

Fig. 10. The CC-joint.

The CC-joint is a concave-convex joint. Not like the saddle joint in the hand but partly like the shape of a glass of spectacles. The axis of movement is longitudinal through the foot, and goes off to the calcanean process of the cuboid. This shape makes possible a rotation of the cuboid on the calcaneum with the calcanean process as a pivot, and with the flat peripheral

part of the joint surface as a guiding flange. In neutral position the dew-drop like inferolateral extension of the cuboid joint surface is not opposed by the calcaneum, but at pronation it slides upward until the joint reaches its close packed position. It is held in that position by the dorsal border of the calcaneum, the tension of the long tendon of peroneus muscle and the tightening of the plantar, dorsal and lateral ligaments of the CC-joint.

If using the oblique axis B for push off, the long plantar ligament is loose, the sesamoid bones are not in use. The cuboid bone slides with the calcaneum process medial on the convex curvature of the calcaneum. The navicular bone follows the cuboid bone in a medial movement on the talus bone. The fit between the two surfaces of the CC-joints is lost. The movement is limited by the tension in the tarsal joint ligaments. Like the bifurcate ligament.

At full supination of the CC-joint, the ligaments of this joint limit further movements. The joint is locked, but not in a close packed position because of the articulating surfaces only oppose each other partly (The normal lock position is in pronation). It does not fit. Actually, it is a kind of sub-luxation (Elftman, H. 1960).

I have drawn the movement of the cuboid bone. See Fig. 11 for the plantar side of the right foot.

I think that carrying the bodyweight by a not fitting joint is very painful. The situation written above is like standing on a spike. Many CRPS-1 patients mention this experience. This is the reason they push the cuboid bone outward (Claw position of the foot). And of course, by using the crutches they lower the

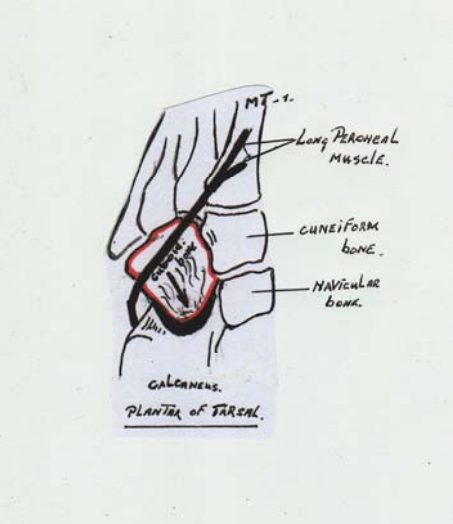

Fig. 11. Cuboid bone rotation.

pressure of the sub-luxated joint. As long as the patients are not liberated they will use crutches.

During a trauma, a lot of problems can exist in the many joints of an extremity. I myself, I am very amazed about the extreme harmony of our locomotor apparatus. In appendix III, it is possible to read more in detail, about the movements of every joint of the foot during gait.

3.2.3. The importance of blood circulation during movement.

Every cell in the body needs oxygen for functioning and staying alive. Blood is the only way of transport of oxygen, so blood circulation has a primary importance in functioning. CRPS patients can not perform correct movements after trauma. Everybody has a local oxygen failure after trauma. But not everybody becomes a CRPS-1 patient after trauma.

The most existing way in which CRPS-1 develops is a distortion of a joint in the forearm or in the lower leg. For a person with CRPS-1, “simple” distortion of the ankle joint can give complains for many years. While a “serious” distortion, for someone without CRPS-1, inclusive skin, ligament, muscle, bone and nerve lesions can be healed after a short period.

The “normal” situation:

The body has intrinsic, adaptive mechanisms to fight infections and for the damaged tissue repair. The body will react “normally” with activating some systems for maintenance homeostasis.

Analyses of the phenomenon trauma with “normal” result:

During a “normal” inversion trauma muscles are contracted for equilibrium and pain is experienced at the same time. Because of this action a vasoconstriction will be started.

Vasodilatation will start if the danger has gone. This is “the Retro-Stress-Relaxation” mechanism of the heart, the muscles and baro-receptor reflex. A pre-capillary vasoconstriction starts, followed by increase of heartbeat but decrease of tension by contraction of the myocardium. This is the result of a sympathetic trauma reaction impulse. This limits blood loss out of a wound, as in the case of an open wound with fracture. At the same time during the trauma, our brain influences behavioural functions also. We are alarmed because of danger, anxiety to fall; anxiety is a sympathetic action too. We have to decide immediately:” Fight or flight”. Or because of tissue damage this is not possible. In the case of fight/ flight, the central vasoconstriction of the trunk stays and the extremities receive more blood. To activate this central reflex, loss of blood is not necessary. Great fear is enough to activate this (LeDoux, J.L. 2000. / Tracy, I. 2007). In the case of tissue disruption vasoconstriction will be continued until the bleeding stops.

The kidneys also give an endocrine response by producing hormones, to regulate homeostasis. The Renine-Angiotensine and Aldosterone system (RAAS).

The above-mentioned situation is a “normal” preparing for the consequences of a trauma. In the case of nerve damage, pain can be experienced. This is a temporary pathophysiological electric hyper excitation of the nerve rupture.

After an accident, wound healing in “normal cases” goes in phases: inflammation phase, proliferation phase and remodelling phase. After three to six weeks, most of the patients are healed and functioning is normal. All this is possible because of sufficient blood circulation.

We speak about normal blood circulation if there is equilibrium between arterial and venous blood circulation. From central, the arterial vessels are influenced by the sympathetic nervous system. Intensive signalling gives constriction of the vessel and less signalling gives

dilatation. There are not a great number of sympathetic nerves near peripheral arteries. The tone of the vessel wall will be regulated by the central nervous system, at night when in rest.

Most important is the local situation. During active movements, local situation is dominant. Local influences are:

1. Myogenic reactions- A pull on the vessel wall gives vasoconstriction.
2. Hypoxia and metabolites- CO2, Adenosine and Potassium.
3. Vaso-active substances from endothelial - Histamine, NO, EDF, Serotonin.
4. Hormones – such as Aldosterone.
5. Pace-maker activity- Contractile rhythm of the vessel wall via gap junctions out of the heart.
6. Heat.

The return of venous blood to the heart is determined by the suck up of the heart itself, the vein valves and the pump action of working muscles (Moving along the fascias and through the fascias, perforating veins). Correct movement without pain means correct blood circulation. The healing will be without problems.

Analysis of a trauma with "abnormal result":

An abnormal situation after trauma can result into CRPS-1. Why the existence of an abnormal posttraumatic situation? Why are the movements and the blood circulation disturbed? What is abnormal? What are the characteristics of a trauma to become CRPS-1? My experience is, that most of the time, it is a torsion or a blunt trauma of the distal joints of an extremity or the vertebral column. Examples of torsion injuries are; inversion trauma of the ankle (60 % of the cases, get off a bicycle, come down the sidewalk, distortion on the

playground)., distortion of the fingers at volleyball, distortion of the wrist at the moment of handling baggage from a moving carousel.

Examples of blunt traumas are; a blow against the vertebral column during a car crash (thoracic gives dystrophy in the arm and a lumbar blow gives dystrophy in the leg), a hefty blow against the calcaneum at the moment of fall from the stairs, bumping against a heavy object, receiving a blow in the wrist when tumble headfirst. Etc.

And yet, extended tissue damage is hardly found in CRPS-1. We always find blood circulation problems and at the same time functioning is painful.

In my opinion, the fundamental difference between “normal” posttraumatic situations and CRPS-1 situations is: “In normal we see a relaxed extremity; in abnormal we see a tense extremity” In “normal” situations we see damaged tissue, blood flow and fractures are visible. The extremity is relatively relaxed. In the case of CRPS-1 we see no damaged tissue, no blood flow, and the extremity is hypertonic or atonic!

In the case of an inversion trauma, the leg is held stiff and is in endo-rotation. The knee is in hyper-extension and the foot is held like a claw. It looks like this painful, posttraumatic position is the result of torsion forces on the periosteum, the muscles, the nerves and ligaments, created by the patient. Some hemorrhaging may exist in the above mentioned situation, yet the extremity is shown as in extreme danger! Safety reflexes are continued when the patient comes in. The extremity is held small, like living tissue reacts when in extreme danger. Blood supply is easier when the patient shortens her extremity. This is very useful in the case of blood circulation problems. See Fig. 12. This patient has held her arm for fourteen years in this position.

Fig. 12. Arm in shortened position.

Various causes of blood circulation disorders:

1. Circulation disorder after displacement of the nerve ending.

After inversion trauma, it is possible to have a temporary loss of the peroneal nerve because of a sudden pull at the motor neuron of this mixed nerve. Its sensory nerve terminals lie more than1µm away from the muscle cells of the blood vessel (Luff, S.E. 1987). They are situated in the adventitia of the arterioles (no endplates). The terminal portions of the axons is varicose, bulbous portions run amid collagen fibres, fibroblasts, lymphatics and vasa vasorum, close to the vessel. Because of that, it is not necessary to have tissue damage after displacement of the nerve. When we have a sore leg after sitting in the same position, we do not have tissue damage! The functional reflex is to rub the sore spot; the nerve will return to its normal position. Displacement means a disturbance of the electromagnetic field. The communication between the nerve and the arteriole is bad or not possible. This communication takes place with bio-photons (Wijk van, R. 2005). This electronic communication is much faster than the electric communication in nerves (more information in 3.2.4.)!

2. Circulation disorder after nerve lesion:

Because of a nerve injury, a nociceptive pain starts. Normal mechanic stimulation can create abnormal discharges in traumatic displaced nerves. In the case of neuropathy the response on these stimuli is abnormal. The aberrant impulse transport of the nerve, the lesion can be partial, and the sensory ganglia will pass on these discharges into the CNS.

Due to this, central sensitization arises in the dorsal root ganglion. This is a sensory reaction to normal stimuli. Nearby “healthy” nerves can join this pathologic behaviour (extension). Neuropathic pain is felt in the skin because it has more nociceptors than muscles have. Patients complain about from being touched while using the shower and wearing clothes. A nerve inflammation creates a tactile allodynia and a vasodilatation, consequently erythema. According to Wasner et al., redness during the acute phase in CRPS-1 is because of a complete functional loss of cutaneous sympathetic vasoconstrictor activity (Wasner, G. 2001 / 1999). According to Birklein et al., subjects with cold skins represent a central sensitisation in the cortex (Birklein, F. 1998). We see during the acute phase a swollen, red/ purple foot as proof of poor blood circulation. Because of the disconnection we notice in the acute phase a retarded propriocepsis too (Kleinrensink, G. J. 1997). So, the chance in bumping and strain of the ankle is increased. We see this very often in CRPS-1!

3. Blood circulation disorder in nerve entrapment:

In this case there is a burning pain. The nerve may have an internal hemorrhage or a tear inside because of a longitudinal pull. The continuity of the nerve is lost. In this, we have to think of the anatomic situation of a myelin sheath outside and nerve fibrils inside. The rupture will have a frayed character. Electric signalling is disturbed in this nerve. Repair is slow because of the difficult discharge of necrotic tissue by phagocytosis (Every dead cell

must be replaced). Blood circulation of the nerve itself is insufficient during entrapment. So the wound healing is. During wound healing known sensitization mediators are created. Like the Nerve Growth Factor and Protein Kinase C (Lewin, G.R. 2004). These can modulate mechanically activated channels in sensory neurons. These mechanosensitive channels are present in the dorsal root ganglion and create central sensitization. This happens because of prolonged wound repair. The necrotic tissue creates a neuropathic vasculitis, especially in nerves with myelin sheaths. Nerves when damaged always have poor blood circulation. Because of ischemia there is a disturbed axon repair with much pain. In the case of entrapment, the ischemia is part of a vicious circle. Because of the pain, muscles will tighten, creating stiffness and ischemia. Increased ischemia means more pain. The CRPS-1 patients call this “muscle cramp pain”.

4. Blood circulation disorders in nerve elongation:

Pathological elongation of a myelin nerve can give a temporary loss of motor function. Frequently we see functional loss of the peroneal musculature in CRPS-1 patients. Stretching of the Schwann sheath expands the distance between the Ranvier nodes. Because of that the function of the nerve is poor. Research in rabbits shows, tibial nerve can be stretched by 40% (Wey van der, L.P. 1995). Vasculitis neuropathy is frequently seen in CRPS-1 patients in the superficial peroneal nerve. This could be interpreted as poor blood circulation as well (Lacroix, C. 1991).

5. Blood circulation disorder in the case of vessels gets jammed:

Not only the skin but also the fascias are well provided with nociceptors. The peronal nerve is anatomically related to the lateral compartment of the lower leg. Walking on the lateral aspect of the foot, without bodyweight bearing, creates a pathological traction to the

peronal nerve and to the compartment. A pathologic function of this connective tissue can produce pain by an isotonic muscle contraction during and after walking. The pain is located at the outside of the leg and the underside of the foot. The periosteum of the tibia can create "shinbone pain" too.

The blood vessels will be congested by these traction and pressure. If tissue damage follows after pathologic walking we see an increase of mastcell activity starting the next inflammation. Not only after pathological movements but also in ischemic situations, cells will die. Because of this, pathological movements the character of the pain will change from nociceptive into neuropathic pain (Güger, L.J. 2008).

In the extremities, many muscle compartments exist. These are constituted by fascia. In pathological walking the pressure inside the compartment is not physiologic any more. During walking with a leg held in endo-rotation, an entrapment of the deep veins and perforating veins can be created. Hundred and fifty perforating veins are located in the lower leg. The connection with the superficial veins is blocked when trapped.

The saphena magna vein and the saphena minor vein are situated superficial and connected with each other by three perforating veins. These three perforating veins are situated in the space between the medial malleolus of the ankle and the mid calf. If blocked, an arterial congestion comes into existence. In sports medicine it is well known that compartment syndrome of the lower leg is very painful (Platz, F. 1976). See Fig. 13.

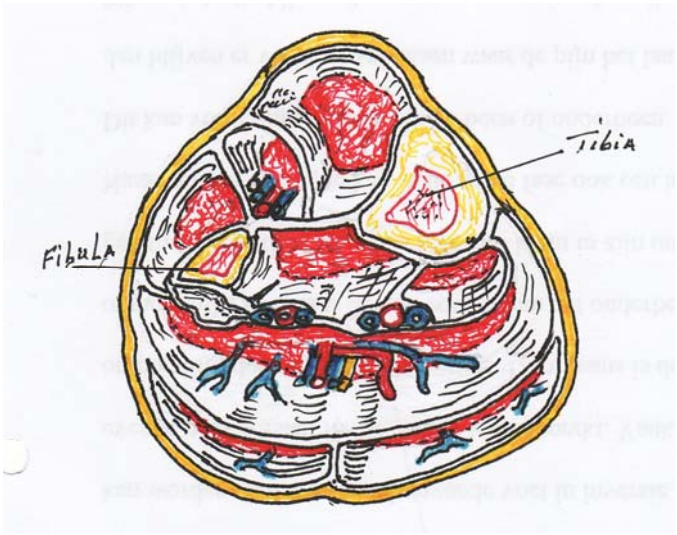

Fig. 13. Lower leg compartments.

3.2.4. Physiological change of the cardiovascular system during instability

There are two information systems, or communication systems, that inform us about the situation of our body (Rothwell, N. et al. 1994). All body parts communicate with each other by information waves. These are the brain waves from the Thalamus, with the Alpha rhythm (Brain pacemaker). Other waves are Beta, Delta and Theta waves. They circulate in the brain. The heart also creates waves, such as electromagnetic, sound, pressure and temperature waves. Every cell of the body receives information from both systems. These systems are not only physiological but are anatomically connected with each other. Blood vessels are connected with nerves by connective tissue (vessel-nerve bundle). In these bundles the blood vessels run perineural. The connective tissue of this perineural system acts as an information network. Nerves use electrical energy. The perineural system uses electronic energy. This functions by mechanical impulses. Living liquid crystals can generate piezoelectric effects for producing bio-electronic signals (Pienta, K.J. 1991). No pull means no information! The cardiovascular system functions like a semiconductor (Becker, R.O. 1995 / 1991). It is a high-speed communication network, much faster than nerves. It has a key role in wound repair.

The vessel-nerve bundles are part of a continuous integrated system extending throughout the body. In this way, the brain is informed about the whole body by this "living Matrix". Signals from this living Matrix have effects on the whole body. It influences our general health or the need of local repair. Moving correctly is essential for staying healthy and correct wound repair (Oschman, J.L. 1993).

In CRPS-1 there is an incorrect pull or no pull upon the vessel/ bundle. The effect is incorrect information or no information. Local pathological movements produce pathological force in this super molecular network. Hence, alignment influences two kinds of communication: nerve impulses and semi conduction through the tensegrity network. Therefore we must look to the whole body. Emotional distress can also contribute to incorrect movements too.

Endothelial cell dysfunction occurs very early in trauma-hemorrhage. Because of the hormonal changes vasoconstriction starts. This occurs in the local blood vessels, as well as central in the aorta. This can persist despite the disappearance of the danger (Wang, P. 1993/ 1995).

Maybe, prolonged elongation of the perineural system has a role in this vasoconstriction?

3.2.5. Instability by blood circulation problems in and near bones

Osteoporosis is a well known complication in chronic CRPS-1 patients. When the patient refuses to touch the ground and blood circulation is disturbed for a long time, osteoporosis can occur. Osteoporosis is more frequent in adults than in children. The periosteum of children has more blood vessels. There is a superior blood communication with the vessels of the shaft as in adults. The osseous blood circulation consists of one nutrient artery; it runs through the cortical bone without branching. In the medullar cavity it then divides into ascending and descending branches. Centripetal branches end in the middle of a

long bone, draining in a central venous sinus. In this thesis the blood capillaries of the outside of the bone shaft is important. Peritoneal pain can be present in CRPS-1 patients. The blood capillaries of the bone surface are connected with the periosteal plexuses, which are formed by arteries from muscle attachments. If the muscle function is not correct, the blood circulation is not correct. The blood support can diminish and in this way the quality of the periosteum will decrease.

Myelinated and non-myelinated nerves occur widely in peritoneal tissue. The nerves accompany the nutrient vessels into the marrow of the bone and exist also in Haversian canals (Gray's Anatomy).

Overloading effects of bone tissue was studied in rats. Immunohistochemical changes were found after repetitive motion. At periosteal surfaces, an increase of mononuclear cells, ED 1 and osteoclasts was found. Most were found at places of attachments of muscles and interosseous membranes.

Malfunction of bones also create nerve function disorder or inflammation, increase of macrophages etc. Bone inflammation produces osteolysis of periosteal bone tissue (Barr, A. E. 2003).

The fine motor control will also be declined. Essential movement feedback is incorrect (Elliott, M.B. 2008).

3.2.6. Energy consumption in the lower extremity in CRPS-1

Energy needed for bipedal locomotion occurs from muscle activity and rebound force against gravity. CRPS-1 patients do not have the rebound movements from the surface they are moving upon. Normal healthy walking consists of the movement of one limb while balanced on the other. That means muscular performance of flexion- extension in the joints of the lower limbs, mostly at the knee and foot level. This is written in section 3.2.1, yet there is more.

Not only an erect moving forward exists but also a pendulum like movement mixed with spring like movements. These movements are produced by:

1. Increasing and decreasing of the vertebral curvatures, which can be observed in a lateral view of the spinal column. This is the increase and decrease of intervertebral space. This gives consequently the up/ down movement of the body height during walking.
2. The circular rotations and counter-rotations of the vertebrae around the axis of the spinal column. The criss-crossed ligaments of the spinal column when stretched will accumulate elastic energy. During counter-rotations the stretched ligaments release and energy is decompressed (Granovetsky, S. 1988).
3. Compression and decompression of the intervertebral discs. The discs are highly elastic and highly compressible, and their rebound is easy.
4. At the moment, just before heal strike, we have seen in section 3.2.1. a free fall of the foot of about 1 cm. The rebound force of the ground is stored into the Achilles tendon and into the ligaments of the arch of the foot. This enables the body to bounce along like a rubber ball (Ker, R.F. 1987).

All these elastic ligaments, menisci and discs around the joints of the leg and spinal column, inclusive the cartilage of the joint itself, are capable of compression and decompression. These qualities of the joints give the body the possibility to store and release elastic energy. This will recuperate some of the muscular energy wasted during locomotion (Alexander, R. 1987).

In CRPS-1 patients, after ankle trauma, these pendulum and spring movements during gait are absent. Because of holding the affected leg stiff, there is no bounce movement. When

heel strike is performed, the rebounded force of the ground will not spread out in the body as it should.

We know that with aging there is the loss of elasticity. The elderly often know that going up the stairs is not as painful as going down. In descending stairs, the motion is abrupt and involves sudden compression of the joints at each step. In CRPS-1 the compression of the joints is made by the patient herself (see Fig. 14).

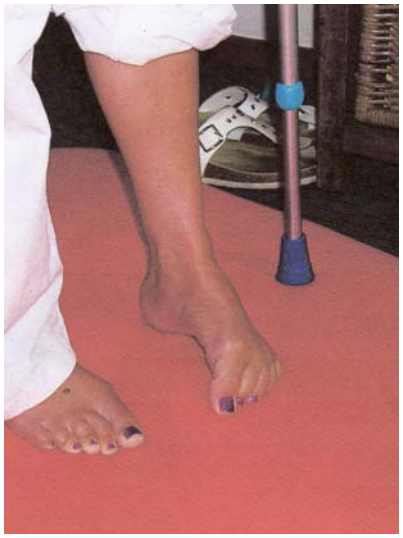

Fig. 14. Compression in the foot.

Most of the time, the bones are not held in alignment. Stressed joints can't move freely, the recoil actions of the joints are reduced. This means, ground force is going into the affected lower limb and will be increased with every step. The energy stays in the bones of the lower limb like collision energy. This raises the internal warmth and will expand the pain during inflammation (Danenberg, H.J. 1992. / Aguayo, A. 1971).

Long-term use of crutches reduces free movement as well.

Tissue repair is compromised when a lower extremity is not acting against gravity. Normal movements of compression/ decompression of the joint cartilage, ligaments, tendons, skin, long plantar ligament etc. produce tiny electric pulsations (Oschman, J.L. 2000). These oscillations are representative information about the precise nature of these movements. The

role of this electronic information, made by the piezoelectric effect, is how to repair and control form (Wolff's law). The bones of the leg are less filled with blood, and are held in torsion. Due to this, the leg loses its flexibility. It suffers more from bumping and putting down on solid ground than a flexible leg.

The patient expresses an increase of pain with every step. Knocking with the leg against something also increases the pain. Theoretically, there is a danger for bone fracture, but stress fractures are unknown in CRPS-1. We see the patient stop walking before bone fractures will exist. They may have "micro cracks" or bone bruises. Nevertheless, distance walking is limited in CRPS-1. We can this name "CRPS-1 patients have limited Seismic Bone Force ". Research by Ozan Akkus, Purdue's Weldon School of Biomedical Engineering (Akkus, O. 2006). The decline of Seismic Bone Force can be explained by the loss of flexibility.

3.2.7. Modern ideas about the function of fascia tissue

It was formerly thought that fascia tissue was a passive tissue; Serving to envelope parts of the body, such as bones, muscles, joints and organs.

1. The bio-mechanic (passive) function.

Besides the function of enveloping, fascia can resist and transfer traction forces because of the presence of collagen filaments. For example, fascia envelops muscle and assists in the transferring muscle strength in joints. Fascias are also places of origin for muscles. It has an average thickness of 1 mm. Formed of numerous layers of parallel collagen fiber bundles. Each layer is separated by a thin layer of adipose tissue. This permits sliding one on the other (Stecco, C. 2007).

Walking and standing is possible thanks to a correct tension of these fascia membranes. Standing upright is determined by the crural fascia, fascia lata and the fascia

lumbo-dorsalis. This fascial chain starts at the calcaneum bone and goes up to the nuchal lines of the cranium. In reality, it goes much farther, internal the cranium as falx cerebri. To walk upright and to make it possible to perform functions with the hands, we have to enlarge the facia chain with the fascia of the latissimus dorsi muscle. So, the functional fascia chain is from calcaneum to the upper arm and cranium.

The anatomical unit

The crural fascia starts at the medial edge of the calcaneum as two sheaths enveloping the flexor muscles in the calf. If this structure has a disconnection, the coordination and the mobility of the leg is disturbed. The rotation of the tibia will be improved during swing phase and the EMG of the peroneal muscle shows a decreased activity (Stahl, V.A. 2007).

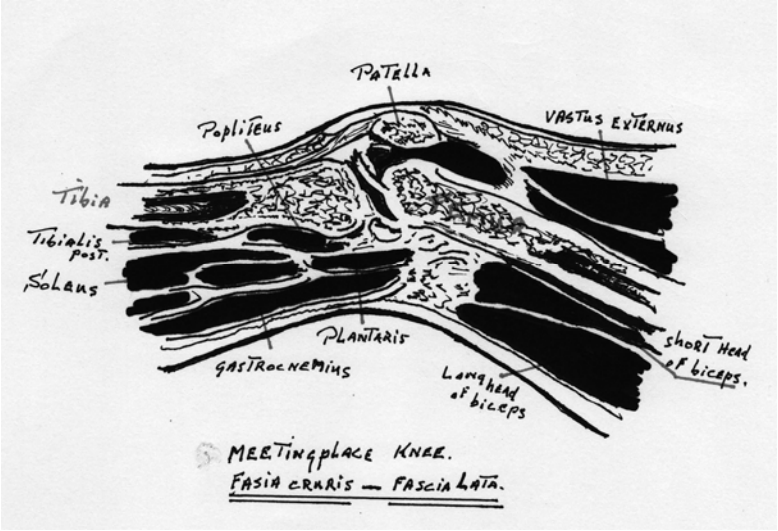

Fig. 15. Knee fascias.

The crural fascia widens and creates more muscle compartments in the lower leg. The long and short peroneal muscles are separated from the front and back compartments. They generate the lateral compartment. The front and back compartment are separated by the interosseous membrane. At the knee, the crural fascia ends frontal at the patella and bilateral condyles of the tibia. At the posterior side of the knee it creates the poplitial fossa. Here are the insertions of the biceps femoris muscle and other hamstrings (see Fig. 15). In the upper leg, the fascia continues as fascia lata. From the tibia-plateau, the femur condyles and the fibular head, a deep fascia is formed, it is fastened at the linea aspera. This split off the vastus

lateralis of the quadriceps muscle from the short head of biceps femoris muscle. From the lateral condyle of the tibia a superficial part thickens to form the ilio-tibial tract. This inserts lateral at the iliac crest as tensor fasciae latae. This creates at the posterior side a place for insertion of the gluteus maximus muscle. The fibers of the fascia continue dorsal to the sacrum and the coccyx. Lateral up to the iliac crest and anterior up to the inguinal ligament. Medial to the ischial tuberosity and through the sacro-tuberal ligament it broadens out cephalad at the dorsal side of the sacrum as part of the deep layer of the lumbo-dorsal fascia (see Fig. 16). At nutation of the sacrum (forward bending of the sacrum to the iliac bones) tension of both sacrotuberous ligaments will increase. Compression arises in both SI- joints, this means greater stability of the pelvic. The place of insertion of the long head of the biceps femoris muscle is the ischial tuberosity. In 50% of the patients, the sacrotuberal ligament is in line with the long head of biceps. The long dorsal sacroiliac ligament acts as antagonist for counternutation with the sacrotuberous ligament. The erector spinae is the pivotal muscle that loads and extends spine and pelvis. The tension of the erector spinae and the latissimus dorsi muscles inside the lumbo-dorsal fascia influences the functioning of

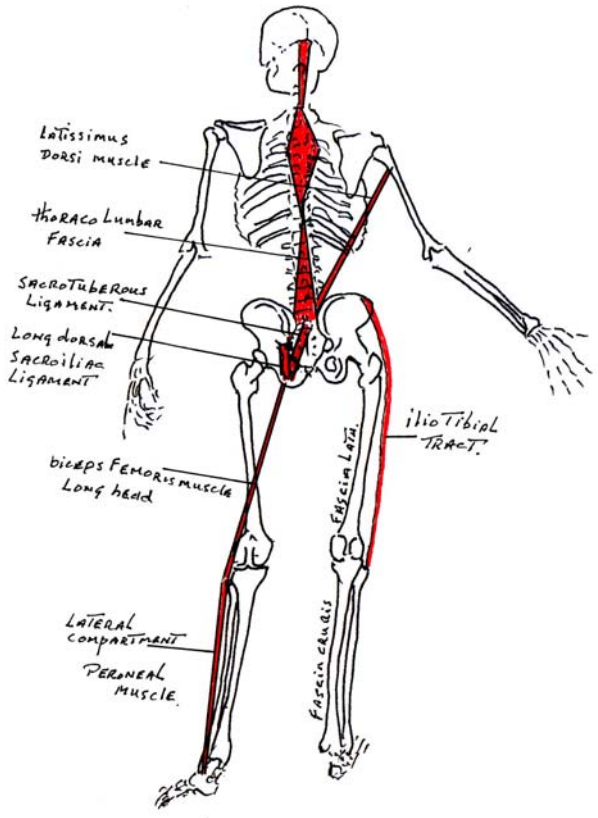

Fig. 16. Muscle trains.

the ligaments. This close collaboration is called “the muscle-tendon-fascia sling”. The force of these slings gives rise to a selflocking mechanism. This is a combination of form closure and force closure and establishes stability in the SI-joints (Snijders, C.J. 1993).

The lumbo-dorsal fascia spreads out from sacrum and iliac crests to the cranium. At every level of the vertebral column this fascia is also connected with the subflavia ligaments. The lumbo-dorsal fascia creates a place of origin for different dorsal and abdominal muscles, and is connected with muscles of upper and lower extremity. Because of their anatomical location, the piriformis and gluteus maximus muscles can optimise the force closure. The importance of the tendon of the biceps femoris muscle depends of the flexion in the hip (Vleeming. A.1990).

Correct functioning of the fascias, written above, is very important for stability in standing and walking in CRPS-1 patients. This knowledge is essential for rehabilitation. If the patient persists in a pathological attitude, it is important to realize that to change one aspect of the chain will influence the whole chain. So, one muscle action can influence the whole body within the fascia relationship. These “myofascial slings” are called by Thomas W. Meyers: “The anatomy trains” (Meyers, T. W./ Jarmey, Ch.).

2. The function of sensitivity in fascia.

The function of sensitivity is shown in the importance of fascia for the proprioception. Professor J. Staubesand of the Albert-Ludwigs-University of Freiburg, Germany, demonstrated myelin and not-myelin nerves present in the crural fascia. Because of this, the relationship was shown between fascias and the autonomic nervous system. This means; every intervention in the fascia system has consequences for the autonomous system (Staubesand, J. 1996). This means; fascias play a role in the neuromuscular reflex control. Also, fascias contain pain receptors by which they influence the autonomic relation in the

case of damaged or ill positioned fascia. This autonomous connections will influence the central processing at spinal and cortical level where the consciousness of pain exists (Emotional memory - LeDoux, J. E. 2008).

3. The active function of the fascias.

Besides passive and sensory functions, fascias have active functions too. It is shown that fascias, beside pain receptors and nerve endings, also have smooth muscle cells. This is published by Robert Schleip. He detected the presence of large concentrations of myofibroblasts in the perimysium of fascias. Investigations with mepyramine showed a tonic contraction of smooth muscle tissue during minutes until two hours (Schleib, R. 2005 / 2006).

The conclusion is that fascias can make and hold a pre-stress to support other muscular activities. Bad tension of fascias means bad neurological control, and this means stiff muscles. Every movement has a bad control because of wrong pre-stress. This is extremely important at the moment of heel strike during gait. But, taking up a cup of tea with the hand can only be realized because of pre-stress in the back, shoulder and elbow muscles.

Living creatures use a system that stabilises itself. In architecture this is used too. It is called “tensegrity”, a construction in which pressure forces and pull forces are in balance. Also, it is maintained during movements (Buckminster Fuller, Architect) (See Fig. 17).

Because of the above mentioned discoveries, fascia tissue is elevated into a higher functional plan.

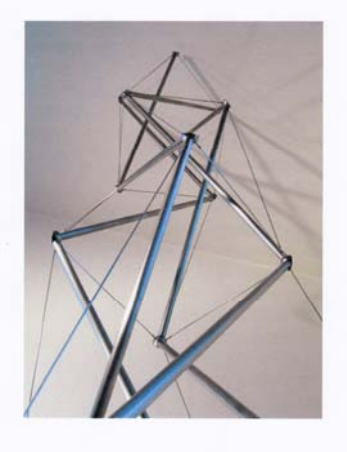

Fig. 17. Tensegrity.

3.2.8. Why is CRPS-1 situated in the lower limb and forearm?

During the acute phase of CRPS-1, the symptoms are observed at the distal parts of the extremities. Pre-eminently, the syndrome arises after trauma to the forearm and lower leg. These body parts are delicate because they have to execute complex movements. The combined actions of the elbow, wrist and fingers are very delicate.

Most of the musculature is multi-articular in this region. According to right functioning of one joint, we mostly need a physiological unit of joints. One mistake in the chain of locomotion can be the origin of a serious functional disturbance.

The coordination is complex. Supination and pronation is unique for the lower limb and forearm.

During trauma, more mobility is needed for peripheral nerves, in contrast with the more central nerves. Nerves are vulnerable because of pulley force during traumas. Especially during distortions and sprains, more joints are involved. These are the most occurring causes of CRPS-1 (Inversion trauma of the ankle is for 60% the cause of CRPS-1).

Functional examination is difficult in small disorders. Often, in the case of CRPS-1, the functional examination is not possible by the pain behaviour of the patient. Because of that, small function disorders can not be noticed at all.

3.2.9. About subluxation and nerves in the lower leg in CRPS-1

If the head of a joint is dislocated a little, we call this a subluxation. This can be permanent or temporary. Consequence of this is that it is out of alignment and not moving as it should. It also creates a transformation of the adjoining tissues. Usually, nerves and blood vessels traverse their well-defined paths in the extremities without difficulties. But the vulnerability of all nerves is not the same. Places of common occurrence, which may produce a pathological situation, is given by anatomical structures next to the nerve. This can be where the nerve passes through a fibro-osseous canal. It can be relatively more fixed in that position and can be impinged by a connective tissue band, a muscle crossing over or by bone. Any mechanical trauma can produce an entrapment of a nerve (Harumitsu, I. 2005).

Peripheral entrapment neuropathy is not easy to identify or diagnosed. Especially since the cause can be at more than one location at the same time. In the acute phase, the patient shows us their physical complaints and findings most of the time in a distal part of the nerve distribution (Nordin, M. 1984). After an inversion trauma, a physiologic block or neuropraxia can occur during a temporary subluxation somewhere in the chain of movement. Recovery can take hours to weeks. Symptoms of pain, muscle weakness and percussion give a Tinel sign.

If the myelin sheath is partially damaged, healing can take three months. But if the axon itself is damaged, it can take three years before final healing is completed. And the conduction velocity down the nerve would initially be very slow. This is never more as 75-80% of normal and the myelin sheath stays thin.

In CRPS patients after inversion trauma, we see a leg in a forced posture. I gave the specifications already before. The cause of this forced posture can be one or more blocked joints but this is never proved. In the literature, there is what is called a “therapeutic click”.

This is published in the Dutch “Medisch Contact” 12 maart 2004; 59:11. And also mentioned by Australian physical therapists during the Pain Congress in Melbourne in 2005. After the “click”, the patient immediately has more freedom in movement with less pain.

Joint movements during gait have already been mentioned, but now I will proceed to the unique movements of pronation and supination. In the lower limb, these movements are made by the proximal and distal tibiofibular joints with the interosseous membrane.

1. The proximal tibiofibular joint consists of the lateral condyle of the tibia and the fibular head. The surface is covered with hyaline cartilage. They are connected by the anterior and posterior ligaments, the fibular collateral ligament and the arcuate popliteum ligament. The latter runs cephalad, obliquely with the popliteal tendon (see Fig. 18).

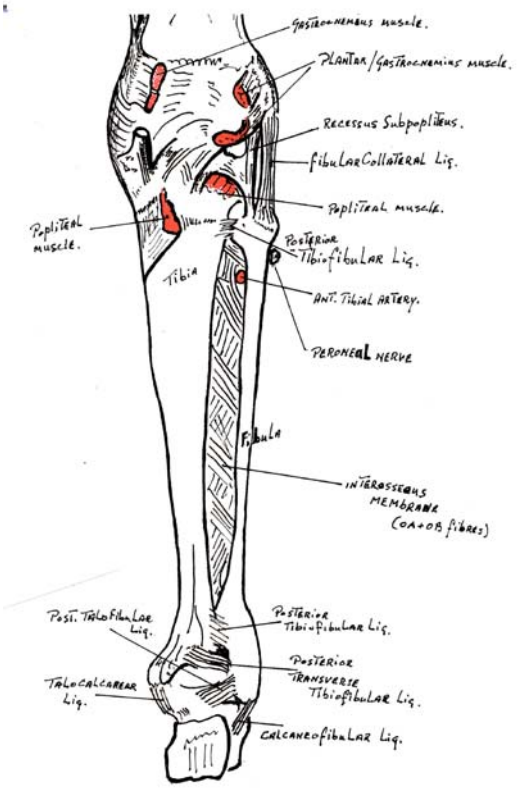

Fig. 18. Posterior ligaments of the right knee.

2. The interosseous membrane connects the shafts of the tibia and the fibula. There is an opening in the cranial portion to give way to the anterior tibial artery. The membrane consists of oblique A and B fibers, perpendicular to each other (Kapandji, orthopaedic surgeon). Because of the perpendicular orientation of the fibers, little movements of the tibia

and fibula are possible like superior/ inferior and translation movements, and they limit these movements as well.

3. The distal tibiofibular joint is a syndesmosis. The osseous membrane clasps both bones and is assisted by some ligaments, the posterior tibiofibular ligament, posterior transversal tibiofibular ligament and the posterior talofibular ligament (see Fig. 19).

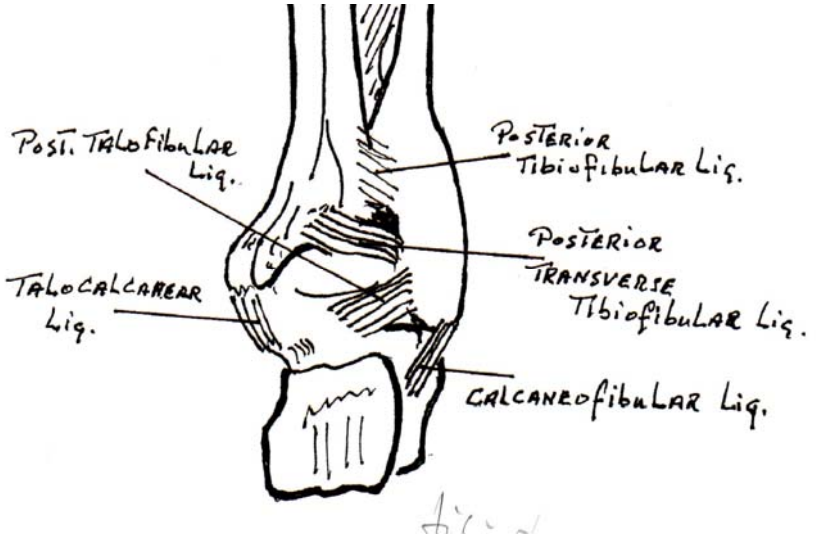

Fig. 19. Posterior ligaments of the right ankle.

The anatomical atlas: "Handatlas der Anatomie des Menschen" of Spalteholz-Spanner, 16th edition, shows a disc between tibia and fibula and mentions this at page 165, plate 320 a "villus synovialus et art. Tibiofibularis".

Of course, the talocalcaneal and calcaneofibular ligaments are helpful with closing the articulation. During walking on a sloping surface, the talus will create an increased malleolar gap by slight lateral rotation of the fibula, by "give" at the syndesmosis and the head of the fibula will make a glide. The calcaneofibular ligament resist these movements. The normal gap in women is ≤ 5.2 mm. In men ≤ 6.5 mm. If the talus moves laterally in the frontal plane, the fibula will make a caudal movement. If the talus moves medial in the frontal plane, the fibula will move cephalad.

It is important to realize that all these movements take place at the same time during walking on a sloping surface! (see Fig. 8).

As already said, a peripheral entrapment neuropathy is difficult to diagnose because many joints can be involved in a traumatic event. This is extreme difficult in CRPS-1 after inversion trauma.

To diagnose a drop foot in a patient after a nine hours flight is much easier!

An entrapment neuropathy is a nerve lesion caused by pressure or mechanical irritation from anatomical structures next to the nerve. Or in the case of our flight passenger, the pressure of her body-weight against a plane chair, at the height of her fibular head.

My conclusion is: a subluxation is possible after inversion trauma. Pull at the lateral ankle means; pull at the proximal fibular head too!

The most vulnerable nerve in the lower leg is the peroneal nerve (Hollis, M. H. 2005). There are three places of preference:

1. The fibular neck. The common peroneal nerve arise from the region of the heads of both biceps femoris muscles, courses around the fibular neck and passes through a tough fibro-osseous opening, makes a sharp bend and travels inferior along the peroneal long muscle (see Fig. 20).

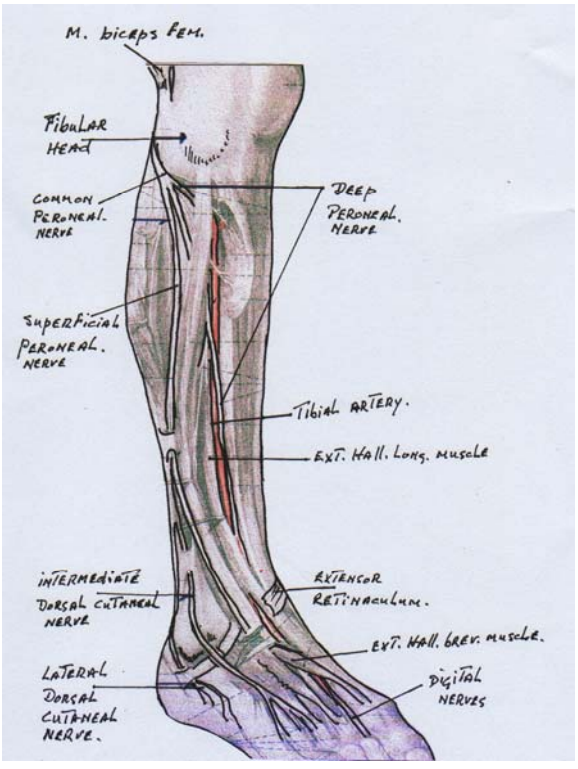

Fig. 20. Peroneal nerve.

2. Both sides of the lateral ankle at the extensor retinaculum. The common peroneal nerve divides into two branches, a

superficial and deep branch, after passing the fibular neck. The superficial peroneal nerve travels distally, up to one third of the lower leg. Then goes anteriorly. Here it divides in sensible branches. One to the front of the ankle (intermedial). and one to the hind foot.(lateral). The intermedial dorsal cutaneus nerve passes the crural fasia from the distal fibula, 4-6 cm. proximally of mortise (see Fig. 20). The nerve endings can be in a fixed position by the fascial attachments. Aberrant situations are mentioned by Styf and Morberg in 1997.

3. The anterior tarsal canal. The deep peroneal nerve starts also at fibular neck and travels close to the interosseous membrane in the anterior compartment to distal. The nerve is accompanied by the anterior tibial artery and vein. Approximately at 4 cm. distance of mortise, the extensor hallucis muscle crosses the nerve. The nerve passes beneath the extensor retinaculum. At the height of meta-tarsal 1 the extensor hallucis brevis muscle crosses the nerve ending to digit one. Below the retinaculum the nerve traverses through the tough anterior tarsal canal. In this canal are 4 tendons (EHL, TP, EDC, EHB), one artery (Dorsalis pedis), one vein (perforance lateral vein) and the deep peroneal nerve. The bones give after inversion trauma impingement symptoms (see Fig. 21).

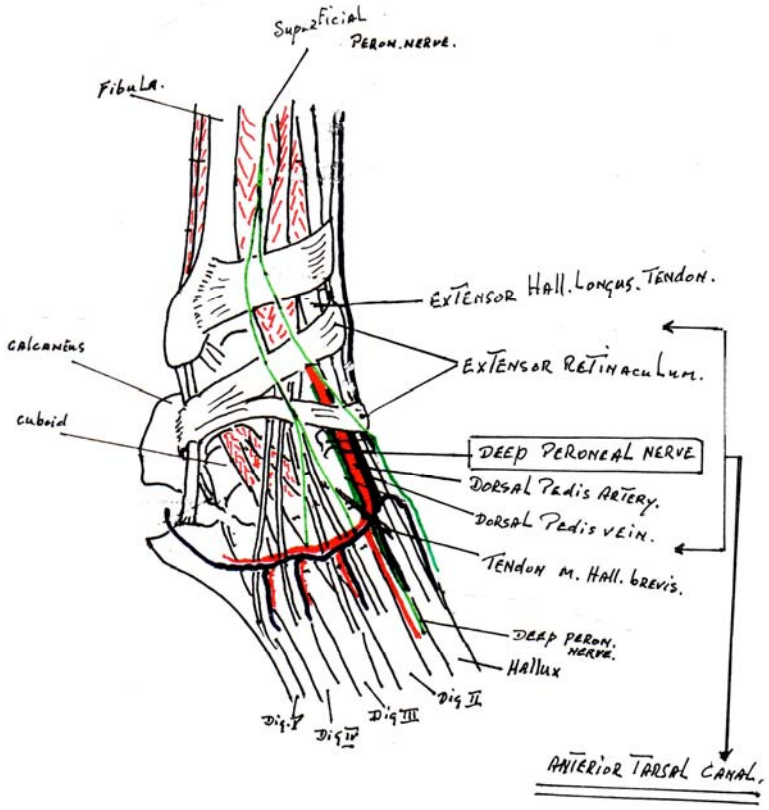

Fig. 21. Anterior Tarsal Canal.

Symptoms, we find in CRPS-1 can be realised at any level of the course of the nerve. These are; burning pain, cramps over dorsum of foot inclusive the first web space. Some shoes increase complaints by minimizing movement of the nerve. A positive Tinel's sign from the neck of fibula until the ankle is well known in CRPS-1.

To endorse the importance of the possibility of entrapment in CRPS-1, I refer to the research results of Ducic and Adkison.

Ducic writes about the motor and sensible innervation of the superficial peroneal nerve. He noticed a subdivision in four anatomical variations (Ducic, I. 2006).

1. In 69,4% the nerve is in the lateral compartment. 2. In 16,2% the nerve divides and stays in lateral and goes also to anterior compartment. 3. In 6,3% the nerve is in the innermuscular septum. 4. In 8.1% the whole course is in the anterior compartment.

Adkison et al. report about the cutaneus dorsalis course of the nerve (Adkinson, D.P. 1991). It is in 73% of the cases in the lateral compartment. In 12% it consists of two parts in both, anterior and lateral compartment. In 14% the nerve meanders from anterior to lateral. In 1% the nerve travels close to the fascia. A pass through of the fascia exists between three to eighteen cm. proximal of the malleolus.

When we do not find a solution to release the tightness, to release the peroneal nerve after inversion trauma, the symptoms will increase after every movement. That is exactly what strikes me most in CRPS-1! Any movement is experienced as the next trauma! The extreme posture of the knee in recurvation and holding outside is often seen in dystrophy patients, but this is a dangerous situation for the fibular head. Together with endorotation of the tarsal bones, the patient brings on danger in the anterior tarsal canal too. If weight bearing is possible but normal movement of the foot is not, the disturbed leg shows us a blocked proximal tibiofibular joint. The foot points more laterally (see Fig. 22).

The same situation exists in the forearm. The ulna and the radius also pronate and supinate. The radial nerve is most vulnerable in the forearm if the radial head is in subluxation.

Fig. 22. Foot to lateral.

3.2.10. Is a signature left in the cardiovascular system after CRPS-1 trauma?

It has struck me that CRPS-1 patients have limited radius of action. If, during one day, many movements are made, the next day looks like payback. The patient must rest more as normal. The question is: “Why?”

If every movement means a new trauma, could Aldosterone be involved? This could mean besides traumatic cause of CRPS-1 a hormonal role can be present too. Aldosterone is released from adrenal cortex by the action of ACTH and angiotensin II. Today, the number of individuals with a high aldosterone level is increasing. Increased concentrations are found in patients with hypertension and metabolic syndrome. One of its roles in aldosterone production goes quicker after repeated, also insignificant, bleedings (Marney, A. M. 2007. / Demaria, E. J. 1989). Increase of aldosterone arises if a patient experiences two events after each other. For instance, this may happen during a minor trauma after surgery in the past. Aldosterone plays a role in the control of fluid and electrolyte balance in injured patients. When inappropriately high in concentrations, it can influence cardiovascular remodelling and increase endothelial dysfunction and inflammation. Every time when patients hurt themselves

it means vasoconstriction. This vasoconstriction is present in the whole system, also when danger is gone (Wang, P. 1995). Maybe in this way, central vasoconstriction can influence the refill of the blood vessels in the extremities?

Vasoconstriction with pain after bumping can also be part of the Emotional Memory, written by Joseph LeDoux and Irene Tracey. This is a "cerebral signature" we can find after trauma (Tracey, I. 2007. / LeDoux, J. E. 2002). With fMRI is shown that a neurogenic respond can be followed into the CNS, up to the brain of the Amygdala region, by asking questions about anxiety. The process of sensitization through the dorsal horn increases easy after repeated bumping.

During menstruation, when the blood circulation changes in the pelvis, female patients experience more pain (Velthuijzen, J. 2005).

Because of these entire processes takes place at the same time, it looks like pain is increasing in CRPS-1 patients. We could conclude: "Indeed, a persisting change is present in the blood vessels because of the presence of hormones".

3.3. My experiments during treatment of CRPS-1 patients

In section one, I have written that the cause or causes of CRPS-1 are not clear. In this section, I enumerated several possible causes. This gives possibilities for therapeutic interventions. The issues for successful interventions are:

1. Muscle cramp.
2. Compression syndromes of the extremities.
3. Mobilising the radial and fibular head.
4. Relapse during menstruation.
5. "Muscle Trains".

6. Reduced “Seismic Bone Force”.
7. Allodynia.

Subsection 1.

In patients with muscle cramp and/ or fascial pain, Trigger points are localised. They were treated with dry-needling (Essential is the local twitch response!) After that passive elongation was used.

Subsection 2.

The use of compression syndrome techniques was successful. The pain diminished and often, after one to four treatments, most patients were able to stand on both feet.

Subsection 3.

Mobilising the radial head increases pronation and supination. Mobilising the fibular head means a great improvement of the mobility of the foot.

Subsection 4.

Female patients, who had already good improvements, can sense more pain during menstruation. To do only compression syndrome techniques in the pelvis has proven successful to let disappear pain in the foot.

Subsection 5.

During the rehab period, it has proven important, to hold the patient in their body alignment. Besides correcting the movements of the injured extremity, we have to pay attention to the rest of the body.

Subsection 6.

When “Seismic Bone Force” was reduced, the use of a pad was advised. To buy a soft sole for the shoes or to fix a spongy buffer at a glove or a brace (Tremble of a steering wheel).

Subsection 7.

In the case of allodynia an artificial skin was advised.

In general, during the first year, I saw the patients twelve times. During the second year five times. And during the third year two times. In the course of the first two years exercises of daily living were performed. In this period, much attention is given to avoid bumping. During the third year jogging is started and specific sports are coming in.

4. The findings

4.1. Analyses of my experiments

1. Muscular cramp was primary treated by performing compression syndrome techniques. The cramp will disappear fairly quickly. If this did not happen, the head of the radius or the fibula did not move properly. Then the forearm patients, complain about pain at the ulnar styloid process. The lower leg patients will point to the insertion place of the peronal tertius at tuberosity of fifth metatarsal (MC-5). In both cases we must perform compression techniques to relax the areas of pisiform bone of the wrist or the lateral malleolus of the ankle. After that, an elongation will be performed at MC-5, or at tarsus plus MC-5. By doing this, the cuboid bone is liberated out of his “loose packed position”. Applying dry-needling techniques for Trigger points is exceptional. It is used if the passive elongation harms too much and if it is obvious that the trigger point is the cause of the pain.
2. When I started my experiments with the CRPS-1 patients, I used all known compression syndrome techniques for extremities. The techniques near the joints, in which the tension of the fascias and the ligaments were changed, were most effective (Eleven out of eighty techniques are now used in the lower extremity). The results after performance of these techniques indicate that influencing fascias have influence at the autonomic nervous system. The patient has less pain and we see an improved blood circulation.
3. I mentioned already the improper movements of the fibula and the radius. Improving the mobility of the fibula and the radius has great results for the function of wrist and ankle. If the fibula is unrestricted, the surrounding of the cuboid bone is not painful any more and the forced lining of the anterior tarsal

canal will be finished too. The eversion of the foot is normalised and weight bearing is increased.

4. In many women more pain is produced during the last phase of the menstruation by changes in the progesterone/estrogen balance (J. Velthuijzen). The anatomical course of the iliac artery and vein is partly situated at the abdominal side of the SI-joints. Maybe, the mobility of these joints play a role in the tone of blood vessels? Pain decrease in the foot after only performing compression technique of the pelvis, can point to this.
5. I have mentioned successive fascias. I have emphasized the fact of one system – the fascial chain. Patients, who have experienced an injury in one extremity, often have dysfunctions in the vertebral column and/ or the pelvis. This is because they try to intercept their stumbling with a straight arm. The impact of the fall will be transferred from arm to vertebral column. We must inspect all the joints and treat all the dysfunctions. It is not exceptional that CRPS-1 pain in the foot disappeared after mobilising the first cervical vertebra. In whiplash patients we have seen the opposite. Trauma of the head can give an increase of pain in the posterior tibial muscle at the ankle joint (Koelbaek, J. 1999).
6. The “Seismic Bone Force” is always decreased in CRPS-1! During minimal touch, patients experience an increase of stiffness. For example in the leg, when during walking the amount of steps increases. Or in the arm, during car riding when handling the wheel. Walking in the gym on a soft surface is very significant for a longer session period. I came to this, because patients reported me that they could walk easier on the wet shore line on the beach! Adaptation was advised to decrease incoming forces. A cushion in a hand brace and softer soles of shoes. With one of my patients a special insert was made to correct the habitual subluxation of the

fibula. When started with jogging, it was first performed in a wooded surrounding. Look for further information at the following pictures.

7. In the case of allodynia, it is highly important that an artificial skin will not move with the real skin. Direct contact with the skin must be impossible. A good fitting glove during the night is very useful for forearms. At daytime a good fit stocking is important for decreasing the touch of a pair of trousers, especially in the case of tough jeans.

Fig. 23. Shoe aid.

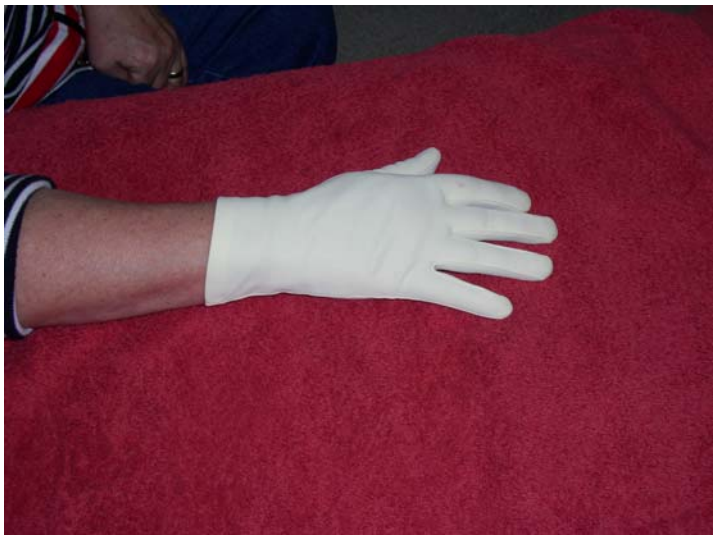

Fig. 24. Hand skin protection.

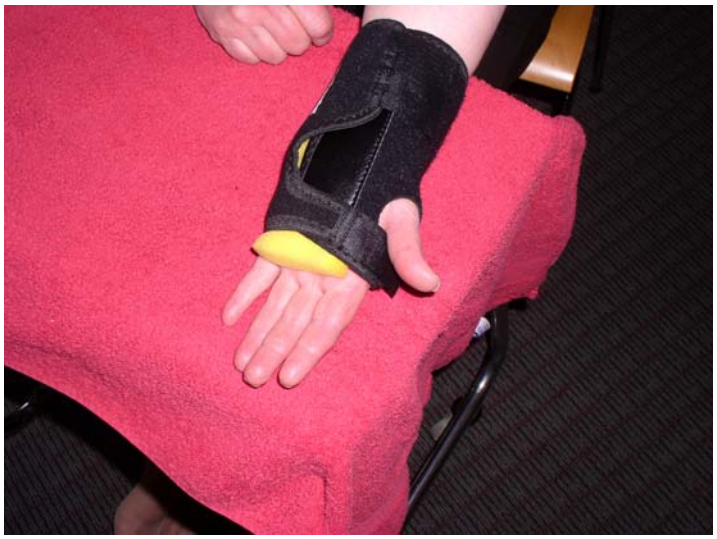

Fig. 25. Hand protection against fibration.

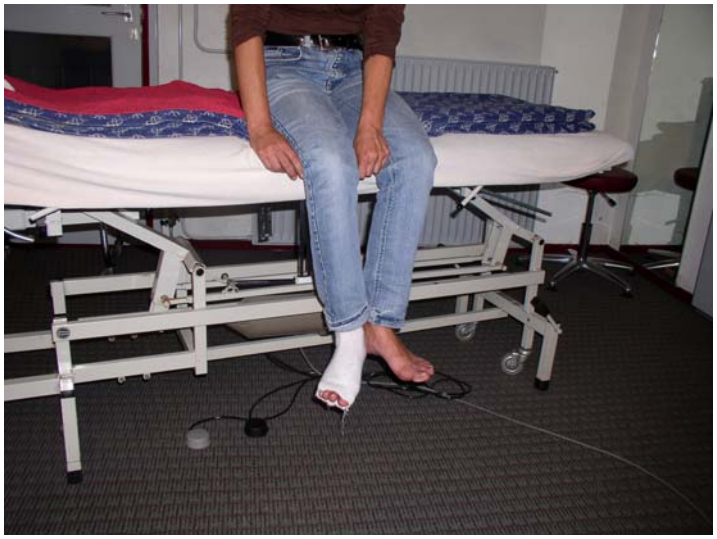

Fig. 26. Skin protection near ankle joint.

4.1.1. The IMT techniques for treatment of the CRPS-1 patients

The patients show their pain in the direct surrounding of the wrist or the ankle. However, the special IMT diagnostics give indications to start the therapy in the trunk. The treatment proceeds from central to distal. This has the advantage that the pain is decreased already if the pain area is reached. In the case of CRPS-1 in the lower leg, the treatment starts below the heart, directly at the abdominal diaphragm. At the height of vertebra Th 10. In the case of CRPS-1 in the forearm, the treatment is started above the heart (Vertebra Th 7). For

the techniques at the forearm I refer to the workbooks; “Diaphragm Compression Syndromes” and “Compression Syndromes of the Upper Extremity” (Workbook School IMT).

The ten used techniques in CRPS-1, in the case of injured lower legs are as follows. The force used during the performance is not more as five Newton.

For the region of the pelvis, technique 1, 2 and 3.

For the region of the knee, technique 4, 5 and 6.

For the region of the ankle, technique 7 and 8.

For the region of the foot, technique 9 and 10.

1. Influence of the thoraco-lumbar fascia region (Bi-lateral).

   Point of departure: Supine, with cushion below the knees.

   Push both anterior superior iliac spines to medial with help of your hand and elbow of one arm. At the same time, with the other hand, move spinal processes of Th 10 till L 3, one by one, to anterior/ inferior. Wait at each vertebra, until the inter-vertebral space releases.

2. Influence at the area of long dorsal ligament (Bi-lateral, see Fig. 27).

   Point of departure: Supine, with cushion under knees.

   One hand gives pressure from Sacral 2 into the direction of the knee. At the same time, with the other hand, pressure at pubic symphysis to posterior. Wait until release.

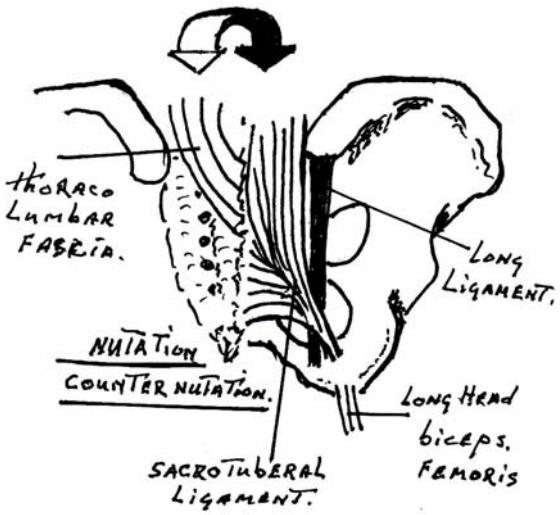

Fig. 27. Ligaments of sacrum.

3. Influence at region sacro-tuberal ligament (Bi-lateral).

Point of departure: Supine, with cushion under knees.

One hand gives pressure from Sacral 2 in the direction of the foot. At the same time, with the other hand pressure against the lateral side of the ichial bone into the direction of umbilicus. Wait until the release. After these three techniques the cushion under the knees is taken away.

4. Influence at region aperture of anterior tibial artery (interosseous membrane) and ligaments of the fibular head (see Fig. 18).

   Point of departure: Supine.

   The fibular head is pushed cranial (with thumb). At the same time with the other hand, two centimetres below the tubercle of tibia, press tibia medial (of the patient). Wait until the release.

5. Influence at region insertion of crural fascia in the knee.

   Point of departure: Supine.

   The tibia is moved cranial and dorsal. At the same time, the femur is moved to medial with the other hand. Wait until a release.

6. Influence at region of inter-muscular septum of the fascia lata.

   Point of departure: Supine.

   The tibia is moved cranial. At the same time, the femur is moved medial. Wait until the release.

7. The region of interosseous membrane.

   Point of departure: Supine.

   Press on the lateral side of the tubercle of the tibia medial, and the distal part of tibia to lateral and superior. Wait until the release.

8. The region of the origin of crural fascia.

   Point of departure: Supine.

Take the achillus tendon to lateral and move with the other hand the calcaneum bone to lateral at the same time. Wait until the release.

9. + 10. Influence at the ankle capsule and anterior tarsal canal (see Fig. 28).

Point of departure: Supine.

Two techniques are made as one. Technique 9 has more influence at origin of crural fascia and inferior extensor retinaculum. And technique 10 is more the release of deep fascias and deep ligaments in the foot.

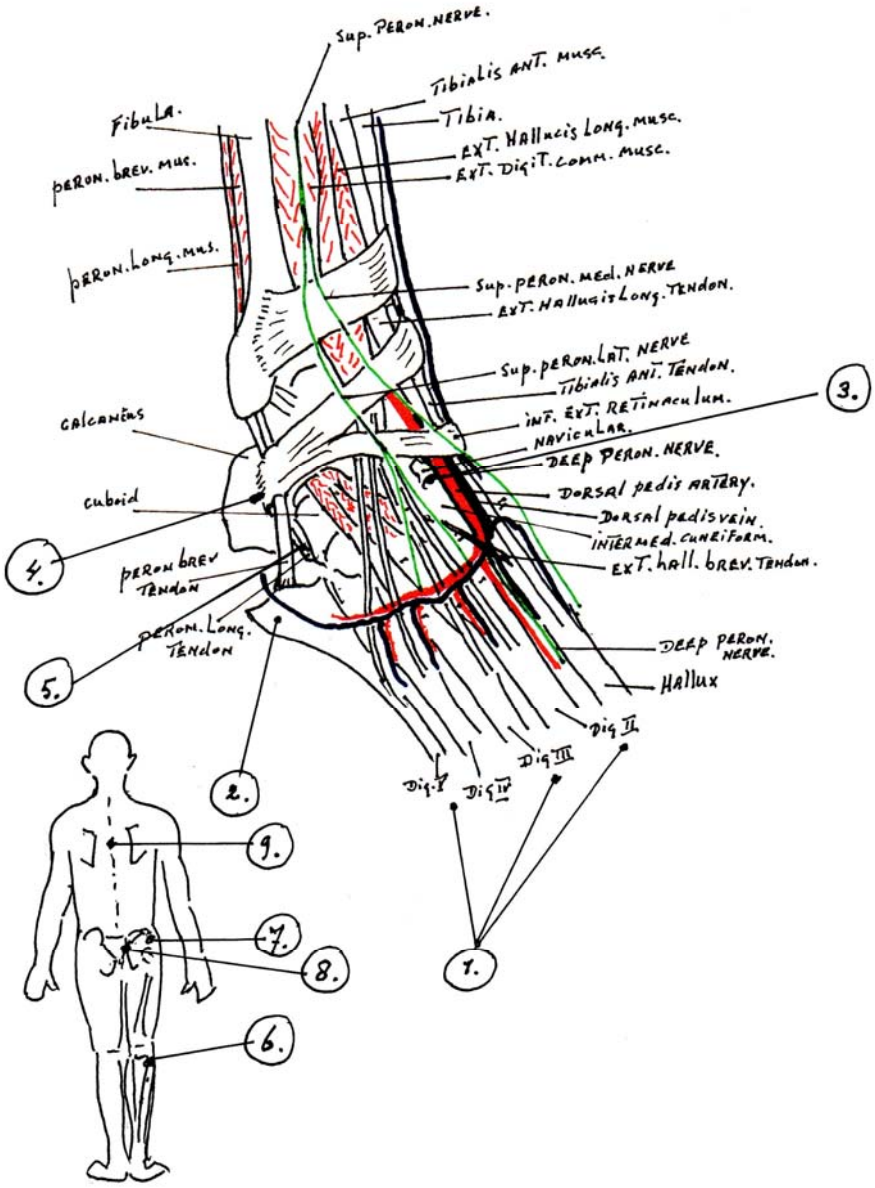

Fig. 28. Metatarsal pain.

9. The therapist stands with his face in the direction of the foot of the patient.
   Set the fork of patient's ankle with one hand. With the other hand, pull the tarsus inferior perpendicular out of the fork. Wait until release. Move the pulling hand slowly from calcanium bone to the first ray. Hold your pull. Move to meta-tarsal one. Pull longitudinal. Wait until release. Move slowly to the first phalanx. Hold your pull. Wait until release. Move slowly to the last phalanx. Hold your pull and wait until release.

10. Stay in the same position. Hold longitudinal pull at first ray with one hand and hold this during the whole technique.
    Bring, with the other hand, the second meta-tarsal bone closer to the first meta-tarsal bone. Wait for release. Pull the second meta-tarsal longitudinal as which the capsule and ligaments of cuneiform joint will release. Move this last hand from meta-tarsal to first phalanx. Hold your pull. Wait until relaxation. Move further to the next phalanx. Hold your longitudinal pull. Wait until release. Move to next phalanx. Hold your pull. Wait until release. Etc. When the first and second ray ligaments are released, take them together in your first hand. Hold the pull. Proceed to the next, third meta-tarsal bone. Bring this bone closer to the both rays you have in your hand. Wait until release. Pull the third meta-tarsal longitudinal. Wait until the release. Move your second hand to the first phalanx of the third ray. Pull longitudinal and wait. Proceed one after the other phalanx to the end of the toe. Take the three rays in relaxed position in your, still pulling, first hand. Proceed with your second hand to the fourth meta-tarsal bone. Bring this fourth close to the third meta-tarsal bone. Wait. Pull. Wait. Move. Pull. Wait. Move. Pull. Wait until the last phalanx joint ligaments are relaxed. Take the four rays in your first hand

and maintain the pull. With your second hand, grab the fifth meta-tarsal bone, bring it to the other four meta-tarsals and wait. After that you pull. Wait. Move. Pull. Wait. Move. Pull. Wait. At the end we have relaxed the foot piece by piece. At last the whole sole is relaxed. If not quite right, you have to repeat technique eight (relaxation of ray five!).

Do we have back the “joint play” in the extremity? If so, we must inspect the function of the proximal fibular joint:

The patient sits on the table. Stabilize the foot of the patient on your crossed lower legs. Immobilize the tibia with one hand and move for mobilization the fibular head with your other hand. Push posterior and pull anterior. In both rotation positions of the ankle (see Fig. 29).

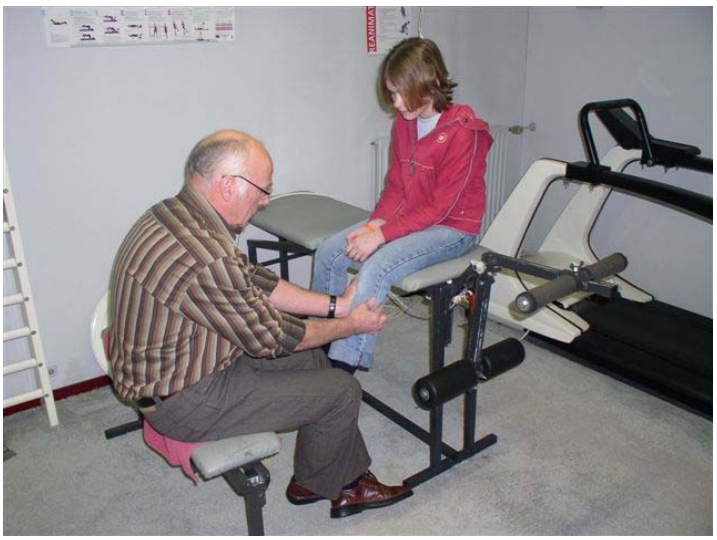

Fig. 29. Fibula mobilisation.

Explanation of the performance of the technique:

The essence of IMT compression techniques is changing the local pressure in the body during a sufficient time. Give time for healing and correction of self. Not compulsory. Sometimes a shear movement is used too (see appendix I).

4.1.2. Tender points of the lower leg in CRPS-1

Now, after writing a number of causes for pain and dysfunction in the preceding sections, we can have a better look at the locations of pain in the CRPS-1 patient (already mentioned in section 3.1.3., see Fig. 7 or Fig. 28).

1. Pain at meta-tarsal 1, 2, 4 and 5.

This can be explained anatomically, because of the location of the superficial peronal nerve. This nerve ends in the three cutaneous nerves, the medial, inter-medial and dorsal cutaneous nerve. It looks like the third ray has less skin nerve innervations; perhaps that is the reason involvement of ray three is rare. Meta-tarsal one can be a direct consequence of an entrapment of the nerve ending of the peronal profundus nerve in the anterior tarsal canal (see Fig. 21).

2. Pain at tuberosity of fifth metatarsal.

This is the place of insertion of the peronal brevis muscle. This muscle works together with long peronal muscle, and helps to increase the pressure in the calcanocuboid joint, this hurts when the cuboid bone is in “loose packed” position (CC-joint). Pointing for pain at the lateral side of the calcaneum bone can also mean that the lateral dorsal cutaneous nerve is injured (see Fig. 20).

3. Pain between cuneiform and navicular bone.

This can come into existence by the profundus peronal nerve (Anterior tarsal canal, see Fig. 21).

Using the second axis of the foot during toe off can create local osseous pain in that area (see Fig. 9).

4. Pain at lateral side of calcaneum.

A “loose packed” position of the calcaneocuboid joint. Created by the lateral dorsal cutaneous nerve.

Abnormal pull of the lateral ligaments of the ankle. Bad mobility of fibular head.

5. Pain at lateral side of lower leg.

Decrease of mobility of fibular head. Subluxation of the head creates elongation of the common peronal nerve, this means pain in the whole route of the nerve until the foot.

Abnormal pull at the fibula from the interosseous membrane.

6. Pain at anterior lateral tibial head.

Failed function of fibular head.

Blocked sacro-iliac joints.

Using axis two during gait.

7. Pain at lateral side of iliac crest.

The pain can be showed at the iliac crest, at origin of iliac tract. If the fibular head is blocked, the pain can expand to this point. The function of the long biceps muscle is often disturbed at the same time. We have to inspect the fibular head first, and then the sacro-iliac joint.

8. Pain at sacro-iliac joint.

This joint is part of the ”tendo-fascial-muscle train”.

9. Cervical and thoracic pain.

As already mentioned, disturbances of parts of the vertebral column will develop during an accident. We have to inspect this, because it is part of the “muscle train”. After time, walking with crutches will give stress for this part of the body because of overuse. Extra attention will be paid for this area, not only the osseous blockage but also for the muscular pain. We often can find trigger points in the trapezius muscle and rhomboideus muscle.

We can make a general remark about the above written locations. Decrease of the blood circulation can be the primary reason for pain by ischemia. This depends in which pain position the patient is (see 3.2.3. underneath various blood circulation disorders). We have to realize what is primary and what is secondary? Most of the time, we must treat both in CRPS-1!

4.1.3. Treatment reactions

1. Freedom of movement in the foot and equilibrium change. A great number of CRPS-1 patients have complains in the lower leg because of a bad function of the fibula. We can test this function by making eversion with both lower legs at the same time (see Fig. 30). Reduced eversion means dysfunction in that leg. Or we can tap the fibula and the tibia with little force. Is the tap against the fibula more painful, the fibula is suspected. Mobilizing the fibular head is often experienced as a release from prison! Not only the eversion normalizes but than weight bearing is possible too! Mobilizing the fibula is always performed, after accomplish all the compression syndrome techniques. Usually, earlier mobilization of the fibular head is impossible because of pain. Immediately after treatment we see patients walk unsure for a moment. We must pay attention to this short change in equilibrium. CRPS-1 patients after casting are always suspicious to dysfunction of the fibular head.

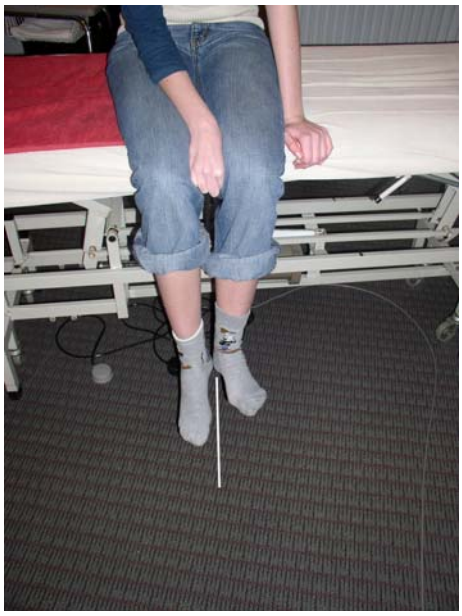

Fig. 30. Eversion test.

2. Increase of pain at styloid process of ulna, and at cuneiform bone I and /or II. The patient persists in dysfunctioning of the pronation and the supination of the forearm. Look to the radial head. Persists in using the second axis of the foot in walking. In this case we have to execute the “bone bruise” technique too.
3. Increase of pain after a (quick) manual therapy technique. Executing manual therapy (quick mobilisation/ chiropractics) is only useful after all the compression syndrome techniques are done. Maybe, this pain exists because of the close relation between the vertebral column and the SI-joints with the blood vessels at the abdominal side, combined with a tiny vasoconstriction of the aorta? The aorta has a close connection with each vertebra. Pull at the vertebra is pull at the aorta. “Quick” manual therapy can be useful at the end of a therapeutic session if somewhere pain still exists. It is my experience that pain at the end of a session, lasted at metatarsal-5, and disappeared after mobilizing Co/ C1. Or pain in the knee disappeared after manipulation of thoracic vertebra 6/ 7.
4. Increase of pain in the CRPS arm/ hand after jogging. This will draw our attention to dysfunctions in the vertebral column or the pelvis.
5. Pain is coming up in a central part while we execute elongation distally. We must proceed with elongation despite upcoming pain. Usually, the central pain will disappear slowly. If not, most of the time, trigger points are the cause of this stubborn pain.
6. Increase of headache and/ or shoulder pain during the treatment of CRPS-1 in the forearm. In the case of CRPS-1 in the forearm, we start with compression syndrome techniques above the heart. If the thoracic inlet is not completely free these complains can be developed.

7. Cramps at night. Ever returning pain during the night can be resolved mostly by a delicate pull at the fingers or toes during 30 seconds. Wait until the pain disappears.
8. Retarded factors for a quick result. In patients, mostly older than 40 years, incidental circumstances can influence the perception of pain in CRPS-1 patients. We can think about extra complains of cervical artrosis, achilles tendon shortening, frozen shoulder and especial osteoporosis is notorious in CRPS-1. Long lasting osteoporosis can be the reason for amputation of hand or lower leg!
9. If pain can not be explained. If a CRPS-1 patient insists in complaining about not logic pain or tells us: “The pain is everywhere!” And these pains does not diminish, we have to ask for psychological help.
10. A correct function of the fibular head. We can control the function of the fibula by performing an exercise. I call this the cross exercise (see Fig. 31). During side steps the leg is alternately placed in front or behind. CRPS-1 patients with dysfunction of fibula, can not perform this exercise.

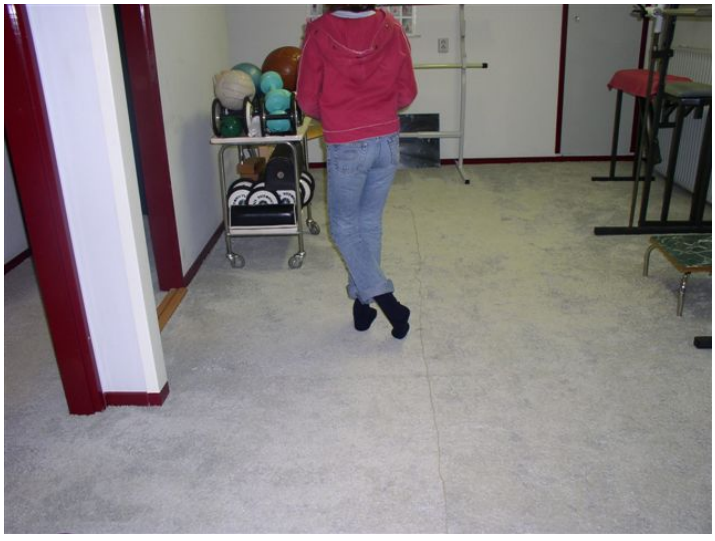

Fig. 31. Cross test.

4.1.4. Therapeutic results during developing the therapy

Before the year of 2004, I started the treatments at the distal part of the injured extremity. Where the patients pointed to be injured and the pain was experienced. The results were with varying success. The successes and failures could not be explained logically. In the case of a good result, good results lasted only for a short period.

The first big improvement after 2004 was the fact that I started the treatment in the trunk. In the case of CRPS-1 in the lower leg, I started below the heart. In the case of CRPS-1 in the forearm, I started above the heart, at thoracic inlet. In all patients I used the same techniques. The results, immediately after treatment, were much better, but also lasted short. The length of the history of the injury had no influence on the results. Of course, the factors mentioned in 4.1.3. point 8 had there importance (Osteoporosis etc.)

In general, the pain disappeared quickly and the blood circulation together with the mobility of the joints, improved during one session.

4.1.5. Changing the treatment

The results of starting the treatment in the trunk were good. But the results did not stay for a long period. I found an improvement of my therapy by implementing a longitudinal traction. This was executed after the compression technique. The traction is performed when the extremity is hold in line. After performing the compression techniques of the pelvis, traction of the hip joints is done in a row. After the compression techniques of the knee, traction of this joint will be carried out (Hands of therapist directly below the patella). After that, the technique for interosseous membrane is done. Continued by traction at the ankle. The hands of the therapist around the malleoli. We end with technique ten, traction is already written in this technique.

Because of these tractions, the therapeutic result was much better and the intervals between the sessions could be enlarged.

4.1.6. Discussion why this improvement?

I wrote in section 3.2.7. about new research of several functions in fascia tissues at the universities in Ulm (Germany) and Rotterdam (Holland). We now know more functions of the fascia tissue, like the function for local blood circulation.

Increase in tone of the fascia will create active vasoconstriction. But if the tone of the fascia will be decreased, and we wait for the reaction of the body, vasoconstriction will diminish. Before improving the distal blood circulation we must be sure about a correct proximal blood circulation. So, first proximal release must be realized. Otherwise the distal therapeutic attempts will be minimized in a reflexogenic way.

Also, I have written about the fact that fascia can maintain a contraction by smooth muscle cells. To “reset” the fascia we can perform a slow traction to neutralize this contraction. Of course, this can be done successfully, when the blood circulation is sufficient. Because of these two steps, normalizing blood circulation and fascia tone, pain will disappear.

Executing longitudinal traction can be painful, especial at the start in the foot. But this pain will fade while we hold the traction.

4.1.7. What is essential for complete success?

Executing all the techniques in one session is most important. The whole path has to be done. For the forearm, starting above the heart and ending at the tips of the fingers. For the lower leg, starting below the heart and ending at the tips of the toes. We must realize; the “fascia train” is one unit. If one part stays in contraction, this part will neutralize the therapeutic work shortly there after. A correct body scheme, meaning a correct tone of the collagen network is of great importance. If we can normalize the communication inside the body, the result of our treatment will stay longer.

Good communication means good self regulation. Our system of blood vessels is, together with the heart, the fastest communication system of our body. The signals run in an

electro magnetic way (Ingber, D.E. 1993. / Oschman, J.L. 2000) Noise in one level means noise in the whole (Information of the piezoelectric effect). This means; we must “reset” the whole!

The importance of a correct body scheme is endorsed by many scientists (Hoogeveen Symposium 2005 / Congress Nijmegen 2009 / Oschman, J.L. 2004).

4.2. Postulate hypotheses

A lot of compression syndrome techniques are written down in the workbooks of the Connecticut School of Integrative Manual Therapy. We know over eighty techniques for the leg, but only ten were helpful in CRPS-1. The now presented ten techniques proved to be useful in all my patients with injured lower legs. It concerns techniques at joints, and they were finished with a longitudinal traction.

Furthermore, the order of working is determined by the start in the trunk. The start is always at the vertebral column below the heart. The treatment always ends at the most distal phalanxes.

The head of the fibula has a great importance for the body scheme.
Now we can postulate three hypotheses, because of these three important factors.

4.2.1. Hypothesis I

“Fascias are important in the treatment of CRPS-1 patients to improve the blood circulation?”

Peripheral blood circulation of the capillaries in tissues of arms and legs is in particular regulated by changes of the lumen of the arterioles. A delicate local self-regulation is possible because of the alternating contractions and relaxations of several muscles, which are surrounded by fascias. This happens in an individual pattern.

This self-regulation consists of:

1. Myogenic factor.
2. Endothelial factor.

Pressure changes in the interior and exterior of arterioles is the biggest cause of capillary blood circulation. Muscle pump action, a muscle contains more blood during relaxation. The availability of oxygen or a neurological factor doesn't play a role in this (Berne, R. 1993). Anyway, research of the university in Nijmegen (Holland) has demonstrated that there is no difference in oxygen in CRPS-1 (Vaneker, M. 2006). Smooth muscles in the tunica media of the arterioles react on changes of transmural pressure. Reduced transmural pressure (intra-vascular pressure minus extra-vascular pressure) stimulates relaxation of the pre-capillary vessels. The endothelial tissue will produce an "Endothelial Dirived Relaxing Factor" (EDRF). Release of EDRF can also be stimulated by a shear stress of the endothelial tissue.

Fascias are the exterior boundary of a local extra-vascular space. It is conceivable that compression syndrome techniques decrease pressure of this extra-vascular space by changing the pressure during a shear at the local fascias. Performing compression techniques means; change the pressure and wait until the release. Only after that we can proceed.

The positive results of the longitudinal traction at joints afterwards can also be explained as an artificial shear effect to the endothelial tissue. Also, it is possible that longitudinal traction, executed in a slow manner at fascias, have influence in the tone of the smooth muscles of the fascia itself (Schleib, R. 2005). By doing so, the pressure inside the space of the fascia will be diminished more.

The behaviour of a CRPS-1 patient shows us the opposite. Because of their convulsive attitude the inter-fascia pressure or extra-vascular pressure will increase. Because

of this, the smooth muscles of the blood vessels are stimulated for contraction. The result is a bad blood supply for the tissues.

4.2.2. Hypothesis II

"Exist post traumatic signature in cardiovascular system in CRPS-1 patients?"

A large dose of aldosterone is a remembrance for the body; it means the body has experienced trauma. Incoming impulses like bumping create aldosterone quickly, and with that create vasoconstriction.

The cardiovascular system is a closed system. This can be divided in several parts. The best known segmentation is the small and the large blood circulation (lungs and heart, rest of the body). In this way, the rest of the body can be divided into smaller functional parts. The result of this is that every part has an influence on other parts. In this way, dysfunctions between the functional units of the blood circulation can be a reason for a quick return of vasoconstriction. This is why it is important to treat the whole distance from heart to outer phalanx.

We know the appearance of endothelial tissue in between the smooth muscles of vascular walls. These are called the myoendothelial junctions. A functional interaction exists between several blood vessels at a distance. The electrical and mechanical conduction is performed by the gap junctions. The extension of this interaction is unknown (closed system is in fact the whole system). The need to treat the healthy hip too is an empiric finding. A CRPS-1 vasoconstriction in the left lower leg has an influence on the right pelvis, but not into the right thigh. Maybe, the aortic bifurcation is a functional unit? Conclusion until now is that after treating the pelvis, we can confine the techniques to the involved leg. It is to imagine that a serious trauma can limit interactions between other functional units. Because we do not know the existence of a functional unit, we must start at the most proximal part. If we don't, the CRPS-1 complains will be increased by another part of a larger unit. This is called "a

signature". Such a "signature" can still be located inside the fascia, three years after trauma during the IMT therapy.

The good results after starting the therapy at the vertebral column can stand for an increased volume of blood into the extremity. Mechanical pull at the aorta influences the baroreceptors. These receptors determine the volume of blood.

4.2.3. Hypothesis III

"Is enlargement of the diagnostic criteria necessary in CRPS-1?"

Immobilizing the extremities with a cast often increases the pain, although the goal is to decrease the pain. Many CRPS-1 patients relate that the start of their illness came after "a too tight cast". In reality, when a cast is used, the ability of the patient to self-correct the sub-luxation by normal, physiologic movements is removed.

Looking back, it turns out that a correct judgement about the diagnostic function of these joints was not made. It is known, moving directly is important for good results (Finniss, D. 2005). After mobilizing the fibular head, with or without a "crack", spontaneous or by the hands of the therapist, there is a successful healing, often after one or two sessions.

During an inversion trauma, the chance to get a subluxation is enlarged. It is a common cause of CRPS-1. Normalizing the rotations of the lower leg there can be a quick advancement. In the acute stage, the problem is that the patient often does not tolerate diagnostic touch. But the diagnostic inspection of radius, fibula and cuboid bone is simple by asking the patient to move by themselves (Cross test. see Fig. 31). This gives essential diagnostic information, and in my opinion, this belongs to the standard inspection of CRPS-1.

5. Discussion

5.1. An efficient therapy is urgently needed

The number of CRPS-1 patients is growing and has huge financial consequences. The Netherlands has 16, 5 million residents, three million of them experience pain daily. Of this, forty percent experience pain longer then seven years. In Holland, CRPS-1 costs among 32, 5 and 47, 3 million Euros per year. In 2003 the Dutch Ministry of Economic Affairs has reserved 90 million Euros for research on CRPS-1. The research is accomplished by seven research centers, called the TREND consortium (Trauma RElated Neuronal Dysfunction). (Trendconsortium. 2007)

Worldwide twenty to thirty percent of children and fifty percent of the adults experience abnormal pain during their life. After three months, thirty five percent and after six months twenty percent still experience pain. In this, ten percent is suffering from serious pain. One percent of the European citizens suffer from neuropathic pain. To support the importance of these patients, the European division of the IASP has formulated a statement in which chronic and periodical pain has to be considered as an illness of its own (Niv, D. 2005).

Every year the number of CRPS-1 patients in Holland is growing by eight thousand new cases. If IMT can give a contribution to the solution, then this will be a very valuable one.

In my capacity of physical therapist, working with IMT techniques, I have written in section three as much as possible reasons why CRPS-1 can come into being. At the same time I wrote why it can develop into a chronic disorder. I have reported besides anatomical and neurological problems also important hormonal and psychological influences. The above mentioned factors increase each other, therefore the patient feels imprisoned in pain.

In section four, I wrote about the symptoms which can be treated with IMT.

When I have set up a hypothesis, I gave a possible explanation how the IMT techniques can be used to set free the patient out of her “prison of pain”. It looks like IMT uses the properties of the sympathetic nervous system. The implementation of the compression syndrome techniques is never imperative. But is more inviting for the injured tissue of the body to normalize. The therapist follows the process, the patient marks the pace. Therefore a session lasts an hour.

I will help to bring the patient into her original situation. Therefore the patient can continue to live without adaptations. It is advisable to follow the patient for three years with help in rehabilitation (Today, the maximum in Holland is two years). In my opinion, this is important because this syndrome is typically only treated as a neurological problem. The improving use of the Subcutaneous Neuro-stimulator is seen as a “Problem killer”.

Above, I have tried to show the great extension of the CRPS-1 problem. We must use every sufficient therapy to resolve this big problem. The good results of IMT are promising and therefore we must use it.

During this discussion about the effectiveness we also have to discuss the coaching of the patient, the cooperation with other disciplines and the financial meaning.

5.2. Good information is important; CRPS-1 has a great negative impact in patients

A good explanation is always important for the result of a treatment (In Holland regulated by law in 1995, the WGBO). With CRPS-1 this is especially important since there much anxiety created by unclearness can be taken away (Keefe, F. J. 2004). Many securities become insecure. What is likely for the near and distant future? Clarity is very important. Having a job or going to school is one of the most important factors for the quality of life (Jeffrey, J, E. 2005). What is the meaning of CRPS-1 for the patient, for the partner and for

the family? A good explanation about the current situation will improve optimism and acceptance. We must prevent depression. The information given by medical doctors and therapists must be controlled, including internet information! Wrong interpretation by the patient can create big commotion! But also, a wrong interpretation by the therapist, about the behaviour of the patient, can be a reason for misunderstandings. Long lasting use of walking aids can give impression to the therapist of avoidance behaviour. One wrong remark of the therapist can create depressive or aggressive feelings in the patient. Until now, I have not noticed in my patients avoidance behaviour because of anxiety to move. I did notice avoidance behaviour only in the case of clear illness profit. This behaviour is tested scientifically. With the assistance of the Dutch Tampa scale an increase of kinesiophobia could not be demonstrated for CRPS-1. Patients with chronic low back pain, chronic neck pain, chronic fatigue and fibromyalgia scored higher (Berg van de, M.E. 2008).

The fact that R. Carmen and M.D. Green found a generation gap in patients with chronic pain is not surprising! (Carmen, R. 2007). People younger than fifty years have more difficulties with chronic pain and tend more to depressiveness. In my opinion, it would be very strange if this was not the case! To become a CRPS-1 patient is a real, huge shock for a young person. The future changes immediately. It is a long way to go, to give chronic pain a place in your life.

Remarks from therapists like: “Pain is not important, first the function”, is a dangerous saying. As a doctor or therapist, we must take care about the psychological reaction of the patient. Therefore a good IMT treatment has to include a good guidance, inclusive of a comprehensive explanation.

The guidance has also to include homework exercises (see Fig. 33). Explanation to the family is important. If the patient has a temporary regression, family members can resolve

problems at the end of the treatment period (see Fig. 32). Instruction to the family makes them less dependant on the therapist. So, it gives them more self-reliance.

Fig. 32. Mother treats child.

Fig. 33. Homework.

5.3. Is it possible to diminish the financial consequential loss of chronic pain by IMT?

The financial consequences of chronic pain are enormous. When a rehab program has started, we expect no further financial expenses outside this rehab program. We expect that patients restart earlier with their work. But until now, this is not the case! In addition to the visits to a rehab centre, the visits to a Pain Clinic are also going on. In serious cases, a lot of

money is spent on adjustments to the patient and to her surroundings. In Holland, the costs of chronic patients are very high. Therefore the government has invested four hundred million Euros for research on CRPS-1.

I treated ten patients during the period 2004-2009. They had finished their rehab program because of no progression. They visited the program in an average of two years. Eight patients made very good progress in my program. One had fair progress and one had no progress. The total costs for IMT for these patients were an average of one week rehab centre.

5.4. Is an Integrative Manual Therapist member of a multi-disciplinary team?

Cooperation with an anaesthesiologist is essential, especially in cases with allodynia, medical assistance is vital. In all cases, we must avoid central and peripheral sensitization. If the patient makes a quick consult with the anaesthesiologist, the medical intake can be limited to one week or perhaps only several weeks.

In the case of depression or not logical developments, psychological help is necessary too.

The treatment of CRPS-1 is always multi-disciplinary. The anaesthesiologist has no results without a physical therapist and the therapist has no results without medical help. The answer for the above written question is a clear “yes!” (Kemler. 2000).

6. Summary and conclusions

6.1. The four types

To assess the therapeutic results at the start of the treatments, I have made a distinction between four types:

Type 1. Acute CRPS-1 after trauma. Often presents with the vasomotor dysfunction, the motor and trophic changes and allodynia. The IMT treatment has immediately good results.

Type 2. Chronic CRPS-1 after trauma. Here we see less vasomotor dysfunction, the motor function is painful. The IMT treatment has immediately good results.

Type 3. Sub-acute CRPS-1 without trauma. Primarily presents with vasomotor dysfunction, and sudomotor (edema). The IMT treatment is limited, temporary results.

Type 4. CRPS-1 after cast and/ or after operation. In this case, we see all variations of CRPS symptoms. Joint dysfunction is for sure. The IMT treatment has immediately good results after removal of cast. It takes more time to achieve good results after an operation (Over three months). A cicatricose after operation

Fig. 34. Acupuncture scar technique.

can be treated by acupuncture scar technique. This can increase a good result. The performance is very painful, but promising. This problem is well known in CRPS-1 patients after tunnel syndrome operations (see Fig. 34). Therefore, during the Pain Congress in Sydney, operation room nurses had pleaded for a correct anatomical position of numb extremities in the case of fixation during operations (Zago, M. A. B. 2005).

6.2. Making a diagnosis for CRPS-1

The criteria for the diagnosis for CRPS-1 is well defined by the IASP, yet it is useful to remember the last line.

It is written: “If another diagnosis can’t be made”.

In this thesis I have tried to increase the diagnostic research and because of that the therapeutic possibilities too. The question is: if we find a malfunction of the fibula, can we change the CRPS-1 diagnosis into “subluxation of the fibula?” I don’t think so! Recognizing this fibula phenomena will enlarge our inside view of CRPS-1. I admit it is surprising, that such a small abnormality in a seemingly insignificant joint, has such an important role.

The finding of these subluxations has had a crucial role for therapeutic success. In my opinion, we must incorporate these findings as a symptom often seen in this complex syndrome. It is my experience that normalizing the blood circulation, again and again, is the key role to health. We have to optimise the body scheme after we have it normalised. We must persist in going through with it until the body can do it by itself. In serious cases, it can take three years.

6.3. Conclusion

1. Is CRPS-1 a long lasting cramp?

On that question it now can be said: “It looks like it is!”

Characteristic is the diversity of causes that creates the connection of intermittent pain in a CRPS-1 patient. The muscle contractions that maintain this syndrome and create a forced position, can in my opinion be called “a muscle cramp”. And, due to the simultaneous ischemia, it has a neuropathic aspect. Type three is an exception since there is no trauma in the anamnesis. It appears that the hormonal factor may be the cause for type three.

2. What is the importance of IMT for the treatment in CRPS-1 patients?

IMT is not an invasive kind of treatment. The results are promising. It fits in a multi-disciplinary therapeutic approach. The underlying theory endorses scientific research. The financial costs are low.

3. Is the treatment of the forearm the same?

Yes, the treatment for the forearm follows the same rules as for the lower leg.

The one exception is for the extra function of the radial head. The radial head is also important for extension of the arm. The fibula has no function in extension of the leg.

In supplement V is extra information about the diagnostics of the forearm.

4. Is the name “Complex Regional Pain Syndrome” correct?

In this thesis I have demonstrated that the “R” is not correct in CRPS-1. I showed an amount of proof, that it is not regional, the whole body is involved! In practice too, we are confronted with patients telling us about enlargement of the pain to another extremity. Research of the last decade, about the role of sensitization in CRPS-1, points also to the disappearance of the R.

To understand this pain syndrome, we must let go to divide the patient in different parts! (Weiss, P.A. 1977).

5. Addition.

The mentioned techniques are also successful in post operative orthopaedic patients.

7. Future research

1. A comparative investigation of IMT method and the so called Macedonia method. To me it looks useful to compare the outcomes of both methods. Particularly with the difference between these methods is the used therapeutic force. The pain experience of the patient, during the treatment, is less in IMT method.
2. Enlarging the rehab period. I like to plead for a longer period of time, because serious cases of CRPS-1 need this extension. In my opinion the time of rehabilitation in a rehab centre, is often too short (in Holland two years maximum!) If the patient, because of circumstances, has prolonged complaints of CRPS-1, the central sensitization has made requisition. This means a long way back for these patients. The reprogramming of the central nervous system takes more time. This means that inside the rehab program, there is a need for a longer period of treatment. My experience is that these serious cases need three years before total health. Particularly for patients under the age of forty in which no osteoporosis is detected, an enlargement of rehab-time looks very useful! An investigation of the financial and therapeutically result of a third year is significant! I can relate this: the patient comes in during the third year, only over a great period, to "reset" the last complains. The financial cost is at a minimum and the grief of the patient is also at a minimum. This extra year minimizes the costs and gives the patients a maximum chance on total rehabilitation!
3. Research about the hormonal factor. The unknown factor, important in CRPS-1, is the role of the hormone system. Research was started because of this factor, in Rotterdam, The Netherlands. On the day of September the 9$^{th.}$ 2009 J.G. Groeneweg promoted the use of the medicine Tadalafil or ISDN (Isosorbide dinitrate). This affects the disturbed layer of endothelial tissue in the blood vessel

wall. The medication decreases pain in patients (Groeneweg, J.G. 2009). Further research is needed (this research is part of Trend 2007).

4. The future report of the research centres of TREND will give direction to future research.

8. Conclusion of this Thesis

It is my wish that this thesis will bring forth an improvement in the diagnostic and an understanding about the CRPS-1 syndrome.

My conclusion is that the observation of, and the talking with, the patients has had great value. I was able to describe more possible causes of CRPS-1 besides the already known neurological causes. It is out of the question that several factors must be investigated as a reason of the cause of this pain syndrome. I sincerely hope that this thesis is a reason to enlarge the diagnosis. I have seen too many patients, after having pain for years, released from pain after only one treatment!

Several scientific researches, I have mentioned, were assumptions for me five years ago. Time worked in my favor. Particularly the fascia research groups in the Netherlands, Germany and America were very important for realization of this thesis. The problems of the sympathetic neural system, maintained by pathological fascia pulling, can now be interpreted and therapeutically understood better. Including the circle of pain and the importance of an aberrant body scheme.

I sincerely hope, that in the nearby future the treatment will be less painful and the endocrine factor will be discovered soon.

Appendix I, what is IMT?

Integrative Manual Therapy (IMT) is a hands-on therapy assesses and intervenes with the anatomy, mechanics, physiology and psychology of the body and it's regulatory mechanisms. Most important in this therapeutically approach is the recognition of patterns, the recognition of patterns in and between the systems of the body in the case of illness or trauma.

The body is seen as one, but consists of different systems, working together in a healthy way. In the case of illness or traumatic injury, the body will start protective mechanisms. The goal of IMT diagnostics is to make an investigation of these patterns. We use Patterns of protection, Patterns of responses, myofascial Patterns, Patterns within a particular system of the body and more. This investigation is called: "Integrative Systems Approach™." This is a unique critical thinking, developed by Dr. Sharon Giamatteo-Weiselfish over the past 30 years. She developed hundreds of techniques for tissue repair.

Sometimes, protective modes do not appear to self correction because of the underlying initial trouble is still present. Therefore the IMT professional is focused from the inside. The lack of flow or communication with the injury will be restored by working with the body's protective wisdom. To access the initial trouble in this way is the key for non-invasive IMT recovery. The therapist works from inside to outside. The protective barriers are reduced without passing the barrier. An integrative healing process for free functioning and structural regeneration is the ultimate goal.

To day, hundreds of students are training in IMT programs like; Advanced program, Diploma program and Certification program. The student has the possibility to follow the Bachelor of Science, the Master of Science, and Doctor of Science program.

Appendix II, history of pain research and pain control on CRPS-1

In our times, pain control from the seventies until now, differs from the previous period. After the seventies, the pharmaceutical industry developed a huge scale of chemical pain killers. This was a logical consequence after the extension of the knowledge of the functioning of the neural system. The nervous system was the origin of the cause of pain. When during operations the nervous system was blocked, pain was not experienced. It was certain that the pain transport must be along the nervous system, or the nervous system was the reason for being aware of pain. The increased knowledge about diversity of nerves and the working of synapses, where the electric energy changed into chemical energy, was broadened systematically. Chemical pain medications such as Paracetamol, Morphine plasters, segmental blocks etc. are used today.

Besides the chemical painkillers also electric devices are used. Examples are the TENS- device on the skin (Transcutaneous Electric Nerve Stimulation). or the Neurostimulator, situated under the skin. These are used frequently in CRPS-1 treatments. Nowadays, pain control is used locally with great accuracy, and is increasingly refined.

However, when a patient complaints of local pain it can easily lead to a local treatment by a doctor. A possible origin of the pain outside the showed pain location can be easily misjudged.

Pain research, before the seventies, consisted of observations of the behaviour of the pain patient. The reactions on pain impulses were recorded. After that, with the knowledge of those days, various conclusions were established, such as: “Pain is the result of the reaction of the patient because of abnormal pulse structure” (Auersperg. 1949). Auersperg did experiments in which he showed that existence of pain was produced during ischemia.

Normal impulses give information about the world around us. Normal means we can cope with the information and interpret. Abnormal impulses mean we cannot cope with the information and we cannot interpret. Pain occurs after passing a threshold. This is called being “over-stimulated”. Pain gives imbalance. It could mean danger; it makes us insecure because we do not understand.

The possibility to feel pain is essential for survival. A pain signal is interpreted as useful if danger exists. However, if pain stays without danger, it cannot serve as a function of warning. Pain is then experienced as useless. This is the fundamental principle of the psychotherapy in CRPS-1.

Pain makes the patient angry or anxious. This reaction on pain is already written in 1920 by the American physiologist Cannon. He calls this: “the flight or fight reaction” or the Effort-syndrome. He draws up several changes in the body which are created by pain, such as an increase of adrenaline by the adrenal glands. This decreases the peristalsis of the bowels. With the contraction of the small arteries in the abdomen, hypertension exists in the case of fear. Through this, the skeletal muscles receive more blood at the moment of danger for better flight or fight. At the same time, the capacity of blood coagulation improves and blood sugar level increases. Maybe, because of this relationship, increasing pain during menstruation can be explained in female CRPS-1 patients? These pain reactions are classified as sympathetic reactions.

Pain behaviour of animals was also studied by professors Buytendijk, Plessner and Max Niehans (Buytendijk F.J.J. 1943) (Niehans M. 1938).

Five basic types were written down: 1) reaction for flight 2) motoric disorientation 3) aggressive behaviour 4) decrease of activities 5) care for injured body part, with or without sound. Important under two is the fact that the movements of the animals were disorientated,

but the coordination was not disturbed! In point three, there is a sign for battle, preparing for action. Fight or flight. Action against danger, to gain security.

Human pain behaviour is described by Bilz (Bilz R. 1940. p. 100). He described the oral and brachial activity when in pain. The muscles of the jaws often contract when shouting. A safe space is made with the arms during an attack, or holding the arms close to the body to make oneself smaller.

We see this looking for safety in animals too. A dog takes care and defends his broken leg as if it is his puppy. Humans are not permitted to approach. There may be growling. Because of the pain, the dog experiences his leg in a different way than normal. The leg will be protected, moved in another way or not moved at all. That is the proof in that moment, that the animal feels himself twofoldness. Hurt physically as well psychologically. The broken leg is no longer a logical part of the whole. After we experience pain in the arm we notice we have an arm. Often we hear the aggressive saying of CRPS-1 patients: “My arm does not belong to me any more, cut it off!” Also CRPS-1 patients move their injured extremity aberrant or not at all. Their behaviour is like a trapped animal. And like a trapped animal, the patient does not dare to move the extremity. As if every movement increases the pain and the danger too. In acute CRPS-1 it is not permitted to touch the injured extremity, not even the skin. Stimulating to move in spite of pain makes them aggressive. Exercises produce more pain at the start of the rehab period. Therefore patients often become more aggressive or depressive. The pain is incomprehensible and long lasting. If we think about the small trauma cause, pain is accompanied by sympathetic reactions in the body like: blood circulation disturbance, vomiting, sweating, anxiety, restlessness and often depressiveness. This brought the French surgeon R. Leriche to write in 1937 in his book; La chirurgie de la douleur: “Le symphatique est le grand nerf de la douleur”. He pointed on the fact that pain is an individual experience, not transferable and inclusive suggestive behaviour. This means beside the body

reaction, the mind also is important in the experience of pain. For example, attention of the partner for the pain of the patient increases pain experience in chronic patients (Hoogeveen Symposium. 2005).

Irene Tracey wrote about three mechanisms important for pain perception. These are: attention, expectation and reappraisal (Tracey I. at al. 2002).

If we are overwhelmed, like in a situation of war, torture, exhaustion and ecstasies often pain is not felt, in spite of an extended trauma. Even pain can be interpreted as positive during sex. Pain is individual. It confronts with the self. All of a sudden there is a dystrophy leg or arm! R. Leriche, 1879-1955, had made so much interest in CRPS-1 that his name is bound to the Südeck-Leriche syndrome. He divided the pain into two categories: Cerebral-spinal and Sympathetic. Nowadays, even in the case of a tiny tissue trauma, an outstanding vegetative reaction of the body can occur. Local trauma can produce reactions in the whole body. All systems are connected. Now, local and central sensitization has the attention of the researchers. In spite of the formulated guideline in Holland we do not have a golden therapy. We still have to look forward for more information. J.P. Robinson writes in IASP Press: “The boundary between the structural model and the neurophysiological model is likely to remain hazy for the foreseeable future” (Robinson J.P. 2005).

A short neurological vision of chronic pain was already given in section 3.

In my opinion, the conclusion of pain control history must be that we have to be aware of the fact that CRPS-1 is a total body disturbance. Because of new interventions the tendency of pain killing by Neurostimulators exists. But the therapy must stay multidissiplinary.

Appendix III, osseous movements during stance phase

Osseous movements during stance phase of gait, are traditionally written as segmental movements of the longitudinal arch. This arch is important for weight bearing during walking and standing. These segments consist of three parts.

The calcaneum or rear foot, the midfoot and the first ray. On top of the arch is the tibia. In between the calcaneum and the tibia the talus is situated. Much literature reports measurements of motion between; a. tibia and calcaneum, b. navicular bone relative to the calcaneum, and c. the first ray to the navicular bone. Professor Mark Cornwall did research in Arizona with a 6D-research electromagnetic motion analysis system (Cornwall. M. W. & McPoll. Th. G. 2002). With kind permission of professor Cornwall I can report as follows.

A. Motion of the Tibia relative to the calcaneum at the moment of heel strike is a quick internal rotation. This stands during 25% of the stance phase. Then it reverses in maximal external rotation and stays at the moment of toe-off.

Calcaneum plantar/ dorsal motion, relative to tibia.

Just before heel strike, the calcaneum is in zero position. After heel strike a rapidly plantar flexion occurs during 14% of the stance phase. Then it reverses to dorsal flexion by 73% of the stance phase. The last period, the calcaneus is again in plantar flexion until toe-off.

Inversion/ eversion motion. At heel strike, the calcaneus is inverted and gradually everts during 55% of the stance phase. Just prior to the toe-off there is a quick inversion.

B. Navicular bone of plantar/ dorsal flexion movement relative to calcaneum.

Before heel strike the navicular bone is plantar flexed. At heel strike it makes a rapidly dorsal flexion to its maximum at foot flat. Then gradually reverses to plantar flexion at toe-off. Inversion/ eversion movements. At heel strike it changes from everted rapidly into inversion

until zero degrees during 24% of the stance phase. At that moment the bone reverses to eversion until toe-off. The navicular bone is never actually inverted during stance phase.

Adduction of navicular bone is only 1,5 degree during 75% of stance phase.

C. First ray plantar/ dorsal motion relative to navicular bone.

The first metatarsal is plantar flexed at heel strike and gradually dorsiflexed to its maximum at 70% of stance phase. After that moment it reverses rapidly to plantar flexion.

Inversion/ eversion motion. At heel strike it is in its zero position. Everts by 26 % of the stance phase, reverses then in a position of inversion, to its maximum value at toe-off.

Adduction of the first metatarsal at heel strike is 0,7 degree but abducts during 52% of the stance phase of – 0,8 degree. Movements of the first ray are small. The excursions of the first ray relative to the navicular bone is 10,2 degrees in the sagital plane, 4,5 degrees in frontal and 2,5 degrees in transversal plane.

Eversion, dorsiflexion and abduction shows up at all five metatarsal bones after internal rotation of the tibia (pronation of the sub-talar joint) (Oldenbook L.L., Smith C.R. 1979).

Appendix IV, the tests

During five years I have treated one hundred patients. Eighty percent are completely cured. A number of patients, approximately ten percent, did not return to their old situation. The last ten percent of patients have complications like osteoporosis, serious contractures and psychological problems. It has struck me that most of these patients are over forty five years old.

It is noticeable that the eighty percent cured patients think about return of the old CRPS-1 if they notice pain. Important for these patients is to give a “normal” treatment if they have “normal problems” of their locomotion apparatus. Performing of CRPS-1 techniques must be avoided, for confirmation of the healing from CRPS-1.

The VAS score:

The VAS was used to measure the results of IMT treatments.

At the start of the treatment the pain was set at ten. Directly after the first session the average score was five.

After one year of guidance (about 12 sessions), at the start of the thirteenth session, the pain was set at an average of score four. Directly after the thirteenth session the pain score was two.

Most important reasons for increase of complains:

- Bumping.
- Continuing movements too long, despite pain.
- Therapist is not careful or malfunctioning device.
- Continuing to walk on solid soles of shoes.

- In children, but also in adults, the therapist or doctor did not take the patient seriously.

Appendix V, remarks on diagnostics of the forearm

Not only in tonic muscles of the lower leg, but also in the more phase muscles of the forearm pain is well-known, like we see in dystrophy patients. For example in Carpal Tunnel Syndrome, Chronic Compartment Syndrome of the forearm and the Pronator Syndrome, pain starts during wrong movements. The median nerve can be trapped locally at the carpal ligament in the wrist. In the elbow, the nerve may be trapped at the pronator muscle, the Struthers ligaments and the aponeurotic expansion of biceps tendon. The radial nerve can sense a pathological pressure at the location of the arcade of Frohse and the supinator muscle itself. These pains are known by the name; impingement or compression syndromes. Neuropathic pain is often present. Shaking hands, wrist extension or flexion and especial supination is very painful (Wilbourn A.J. 1981). These movements are also disturbed in CRPS-1 patients.

Bibliography

Adkinson, D. P., Bosse, M. J., Gaccione, D. R., Gabriel, K. R (1991) Anatomical variations in the course of the superficial peroneal nerve. *Bone Joint Surg. Am. 1991 Jan; 73 (1):* 112-4.

Aguayo, A., Mair, C. P. V., Midgley, R (1971) Experimental progressive compression neuropathy in the rabbit. *Archives of Neurol. Vol 24; 1971*: 359-364.

Akkus, O (2006) Perdue University. New system monitors tiny "Earthquakes" in bones to prevent fractures. *PHYSorg.com.* Sep. 13-2006.

Alexander, R., Bennet-Clark, H. C (1977) Storage of elastic strain energy in muscle and other tissues. *Nature 1977*; 265: 114-117.

Arendt-Nielsen, L. et al (2009) Evaluations of sympathetic vasoconstrictor response following nociseptive stimulation of latent myofascial trigger points in humans. *Acta Physiologica* 2009*; vol. 196, (4), Aug*: 411-417.

Auersperg et al (1949) Disturbances of sensation occasioned by experimental arrest of blood flow. *Arquivos de Neuro-Psiquiatria. Vol. 2. 1949*: 371-392.

Barnus, A. J., Tsuzaki, M., Yamato, J. et al (1995) Mechanoreception at the cellular level: the detection, interpretation, and diversity of responses to mechanical signals. *Biochem. Cell Biol. 73; 1995 (7-8*): 349-365.

Baron, R., Stanton-Hicks, M.,Huijgen, F., Maier,. Ch (2005) Mini-symposium. Pain Congress Sydney 2005. Lecture number 1122.

Barr, A. E. et al (2003) Repetitive, negligible force reaching in rats induces pathological overloading of upper extremity bones. *Bone Miner Res. 2003 No; 18* (11): 2023-32.

Becker, R. O.(1991) Evidence for a primitive DC electric analog system controlling brain function. *Subtle Energies, vol. 2; 1991*: 71-88.

Becker, R. O., Selden, G (1995) The body electric electromagnetism and the foundation of life. William Morrow. New York.

Bennett, M.L., Smith, B. H., Torrance, N., Potter, J (2005) S-Lanns score for identifying pain of predominantly neuropathic origin: validation for use in clinical and postal research. *.J. Pain. 2005.*

Berg van de, M. E., Bouma.M.E., Beerthuisen, A.,Verkerk, K.,'t Spijker van, A., (2008) Kinesiofobie en het ontstaan van het CRPS type 1. FysioPraxis. Juni 2008.

Berne, R. M., Levy, M. N (1993) Physiology. International Edition. 3th. Edition. Mosby Year Book, Inc. St-Louis. Missouri. 1993.

Bilz, R (1940) Ein Beitrag zur Pathologie menschlicher Affecte und Organfunktionen. *Leipzig. 1940.* seite 100.

Birklein, F., Roedl, B., Neundörfer, B., Handwerker, H. O (1998) Sympathetic vasoconstrictor reflex pattern in patients with CRPS. *Pain. 1998 Mar; 75*: 93-100.

Bojsen-Møller, F (1979) Calcaneocuboid joint and stability of the longitudinal arch of the foot at high and low gear push off. *J. Anat. 1979. 129; (1):* 165-176.

Breivik, Harald. et al (2005) Prevalence and impact of chronic pain: a system review of epidemiological studies on chronic pain. Pain Congress Sydney. 2005, Aug. 24. Lecture number 1106-P346.

Buytendijk, F. J. J (1943) Over de Pijn. Universiteit Utrecht. NL.

Cannon, W. B (1920) Bodily changes in pain, hunger and rage. New York 1920.

Congress UMC. St. Radboud Nijmegen. 09-03-2009. Pain Exposure Physical Therapy.

Cornwall, M. W., McPoll, Th. G (2002) Motion of the calcaneus, navicular andfirst metatarsal during stance phase of walking. *J. of the Am. Podiatric Med. Ass. Vol 92.* No. 2. Feb. 2002.

Danenberg, H. J (1992) Subtle gait malfunction and chronic musculo-skeletal pain. *J. Orth. Med. 1992; 14*: 18-25.

Delgado, M. R., Nearing, K. I., LeDoux, J. E., Phelps, E. A (2008) Neural circuitry underlying the regulation of conditioned fear and its relation to extinction. *Neuron. 2008; 59*: 829-838.

Demaria, E. J., Lilly, M. P., Gann, D. S (1989) Aldosterone secretion following non-hypotensive hemorrhage is potetiated by prior blood loss. *Journal of Trauma. 1989.*

Ducic, I., Dellon, A. L., Graw, K. S (2006) The clinical importance of variations in the surgical anatomy of the superficial peroneal nerve in the mid-third of the lateral leg. *Ann. Plast. Surg. 2006 Jun; 56 (6):* 635-8.

Ek, J. W., Gijn van, J (2004) Een wonderbaarlijke genezing. *Medisch Contact 2004; 59 (11)* : 401 – 403.

Elftman, H (1960) The transverse tarsal joint and its control. *Clinical Orthopaedics. 1960*; 41-46.

Elliott, M. B., Barr, A. E., Kietrys, D. M., Al-Shutti, T., Amin, M (2008) Peripheral neuritis and increased spinal cord neurochemicals are induced in a model of repetitive injury with low force and repetition exposure. *Brain res. 2008 Jul. 7*; 1218: 103-113.

Fairhurst, M. Wiech, K., Duckley, P., Tracey, I (2008) Anticipatory brainstem activity predicts neural processing of pain in humans. *Pain. 2008. Oct. 26*; 17070996 (P,S,G,E,B,D).

Finniss, D., Murphy, P., Brooker, Ch., Cousins, M., Overton, S., et al (2005) Early mobilisation, specific exercise and cognitive behavioural therapy are the key components for the treatment of CRPS-1 patients in children and adolescents. Pain Congress Sydney. 2005 Aug. 26. Lecture 1822-P325.

Granovetsky, S (1988) The spinal Engine. Springer-Verlag, NY. Inman VT. 1966. Human locomotion. *Can. Med. Assoc. J. 94*: 1047-1057.

Graven-Nielsen, T., Arendt-Nielsen, L (2003) Central sensitization in fibromyalgia and other musculoskeletal disorders. *Curr Pain Headache Rep. 2003; 7*: 355-61.

Gray's Anatomy. Edition 38: 467-470.

Green, C. R (2007) Generation gap found in chronic pain. *Pain resources 2007: News.*

Groeneweg, J. G., Huijgen, F. J (2006) Increased endothelin-1 and diminished nitric oxide levels in blister fluids of patients with CRPS-1.

Groeneweg, J. G (2009) Cold case: Vascular dysregulation in chronic CRPS-1. Dissertation. 09-09-2009. Erasmus Universiteit Rotterdam. NL.

Güger, L. J., Irnich, D., Abahjio, T. N. et al (2008) Characteristics of chronic ischemic pain in patiens with peripheral arterial disease. *Pain. 2008 Sep. 30; 139 (1*): 201-208.

Harumitsu, I. et al (2005) Distribution of sodium channels during nerve elongation in rat peripheral nerve. *Journal of Orthop. Sci. Springer Japan. 2005; vol. 10*. No 2/ March.

Hicks, J. H (1954) The mechanics of foot. II. The plantar aponeurosis and the arch. *Journal of Anatomy. 1954; 88*: 23-31.

Ho, M. W. Popp, F.A., Warnke, U (1994) Bioelectrodynamics and biocommunication. World Scientific. 1994. Singapore.

Hollis, M. H., Lemay, D. E., Jensen, R. P (2005) Nerve entrapment syndromes of the lower extremity. 2005, Jan. 27. from www.emedicine.com/othoped/topic422.htm.

Hoogeveen symposium (2005) Eerst de functie dan de pijn. Jan. 20. Verpleeghuis "Weidesteyn", Holland.

Ingber, D. E (1993) Cellular tensegrity: defining new rules of biological design that govern the cytoskeleton. *Journal of Cell Science. 1993; 104*: 613-627.

Jarmey, Ch. Anatomic Train Concept. The atlas of musculo-skeletal anatomy. North Atlantic Books.

Jeffrey, J. E (2005) What is the nature of the relationship between chronic pain and quality of life? Pain Congress Sydney. 2005, Aug. 24. Lecture 969-P211.

Kajander, K. C., Giesler jr, G. J (1987) Effects of repeated noxious thermal stimuli on the responses of neurons in the lateral cervical nucleus of cats: evidence fora n input from A-nociceptors to the spinocervicothalamic pathway. *Brain Research. 1987. Vol. 436;* (2) : 390-395.

Keefe, F. J., Rumble, M. E., Scipio, C. D., Giordano, L. A., Perri, L. M (2004) Psychological aspects of persistent pain: current state of the science. *The journal of pain. 2004; 5*: 195-211.

Kemler, A. M., (2000) A two year follow up. *N. Eng. J. Med. 2000; 343*: 618-624.

Ker, R.F., Bennett, M. B., Bibby, S. R (1987) The spring in the arch of the human foot. *Nature 1987; 325*: 147-149.

Kleinrensink, G. J (1997) Enkelverzwikking leidt tot geleidingsvertraging in zenuwen. Dissertation. Jan. 24 1997. Erasmus Universiteit Rotterdam. NL.

Koelback, J., Graven-Nielsen, T., Schou, O., Arendt- Nielsen, L (1999) Generalised muscular hyperalgesia in chronic whiplash syndrome. *Pain 1999; 83 (2*): 229-234.

Koenig, H. L., Gong, W. H., Pelissier, P (2000) Role of progesterone in peripheral nerve repair. *Journals of reproduction and fertility. 2000; 5*: 189-199.

Lacroix, C., Hautoshi, F., Gérard, S (1991) Vulnerability of nerve fibres to ischemia. *Brain. Vol. 114; No 4*: 1929-1942, 1991.

LeDoux, J. E (2002) Emotion, Memory and the brain. *Sci. Am. 2002; 12*: 62-71.

LeDoux, J. E (2000) Emotion circuits in the brain. *Annu. Rev. Neurosci. 2000; 23:* 155–184.

LeDoux, J. E., Lanuza, E., Moncho-Bogani, J (2008) Unconditioned stimulus pathways to the amygdale: Effects of lesions of the posterior intralaminar thalamus on foot-shock-induced c-Fos expression in the subdivisions of the lateral amygdale. *Neuroscience. 2008 Aug. 26.*

Leknes, S., Brooks, J. C. W., Wiech, K., Tracey, I (2008) Pain relief as an opponent process: a psychophysical investigation. *Eur. J. Neurosci. 2008 Jul 30*; 1736-1867.

Lewin, G. R., Uhtaek, Oh., Cesare, P (2004) Mechanosensation and Pain. *J. Neurobiol. 2004 Oct; 61 (1)* :30-44.

Luff, S. E., McLachlan, E. M., Hirst, G. D. S (1987) An ultra structural analysis of the sympathetic neuromuscular junctions an arterioles of the submucosa of the guinea pig ileum. *J. Comp. Neurol. 1987; 257*: 578-594.

Lundberg, U (1999) Stress responses in low status jobs and their relationships to health risk: musculoskeletal disorders. Am. New York Acad. Sci. 1999; 896: 162-172.

Marney, A. M., Brown, N (2007) Aldosterone and end-organ damage. *Clinical. Science. 2007, vol. 113.*

Meijler, W. J (2006) Pijnperiodiek. Aug.

Meyers, Th. W. Bodywork KMI, Kinesis Myofascial Integration.

Niehans, M (1938) Wege zum Verständnis der Tiere. Niehans Verlag. Zürich-Leipzig.

Niv, D., Devon, M (2005) If you suffer from pain, your doctor should consider it a disease. *EurekAlert. 2005. Jan. 14.*

Nordin, M., Nystrom, B., Wallin, V., Hagbarth, K. E (1984) Ectopic sensory discharges and paresthesiae in patients with disorders of peripheral nerves, dorsal roots and dorsal columns. *Pain 1984; 20*: 231-245.

Nuutti, V. V. et al. Central processing of tactile and nociceptive stimuli in CRPS-1.

Oldenbook, L. L., Smith, C. R (1979) Metatarsal head motion secondary to rearfoot pronation and supination: an anatomical investigation. *JAPA 69; 24, 1979.*

Oschman, J. L. & Oschman, N. H (1994) Physiological and emotional effects of acupuncture needle insertion.

Oschman, J. L (1993) A biophysical bases for acupuncture.

Oschman, J. L (1993) Sensing solitons in soft tissues. *Guild News. Vol. 2; 1993*: 11-14. p. 242.

Oschman, J. L (2000) Energy Medicine. ISBN 0-443-06261-7. Chapter 12: 165-174. Churchill Livingstone books.

Oschman, J.L (2004) Energy Medicine in Therapeutics and Human Performance. Butterworth- Heinemann. ISBN 0-443-06261-7.

Perry, J (1992) Gait analyses. Publ. Slack. ISBN. 1-55642-192-3.

Pienta, K. J., Coffey, D. S (1991) Cellular harmonic information transfer through a tissue tensegrity-matrix system. *Med.Hypotheses.1991 Jan; 34 (1):* 88-95.

Platz, F., Adelmann, G (1976) Zur anatomie der 'vena arcuata cruris posterior' und ihrer Tiefen anastomosen. *Verh. Anat. Ges. Jena. 1976; 70*: 709-714.

Robinson, J. P (2005) Path of Pain. p. 364. IASP-Press. Seattle.

Rothwell, N. J., Berkenbosch, F (1994) Brain control of responses to trauma. Cambridge University Press.

Rüger, L. J., Irnich, D., Abahji, T. N. et al (2008) Characteristics of chronic ischemic pain in patients with peripheral arterial disease. *Pain. 2008 Sep. 30; 139 (1):* 201-208.

Schleib, R. et al (2006) Passive muscle stiffness may be influenced by active contractility of intramuscular connective tissue. *Med. Hypotheses, 2006; 66:* 66-71.

Schleib, R., Klinger, W., Lehmann-Horn, F (2005) Active fascial contractility: fascia may be able to contract in a smooth muscle-like manner and thereby influence musculoskeletal dynamics. *Med. Hypotheses, 2005; 65*: 273-277.

Shephard, E (1951) Tarsal movements. *Journal of bone and joint surgery. 1951. 33B,* 258-263.

Snijders, C. J., Vleeming, A., Stoeckart, R (1993) Loading of the sacroiliac joints when lifting in stooped posture. *J. of Clin. Biomech. 1993; 8*: 295-301.

Stahl, V. A., Gottschall, J. S., Nichols, T. R (2007) A biomechanical analysis of the crural fascia in the feline hindlimb during locomotion and intra-muscular stimulation. Fascia Research Congress, Boston. 2007. Congress book page 214; Lecture 8.1.3.

Staubesand, J., Li, Y (1996) Zum feinbau der fascia cruris mit besonderer Berücksichtigung epi- und intrafaszialer Nerven. *Manuelle Medizin, 1996; 34*: 196-200.

Stecco, C., Porzionato, A., Stecco, A., Aldegheri, R (2007) Histological study of the deep fasciae of the limbs. Fascia Research Congress, Boston 2007; Congress book page 132; Lecture 5.3.1.

Styf, J., Morberg, P (1997) The superficial peroneal tunnel syndrome. *J. Bone Joint Surg (Br) 1997*; 79-B: 801-3.

Szent-Gyõrgyi, A (1941) The study of energy-levels in biochemistry. *Nature 1941;* 148 : 157-159.

Thacker, M. A. et al (2007) Pathophysiology of peripheral neuropathic pain: Immuun cells and molecules. *Anesth. Analg. 2007; 105*; 838-847.

Tracey, I (2007) The cerebral signature for pain perception and its modulation. *Neuron. 2007 Aug. 2; 55 (3):* 377-91.

Tracey, I., Ploghaus, A., Gati, J. S., Clare, S. Smith, S (2002) Imaging how attention modulates pain in humans using functional MRI. *Univ. of Oxford. 2002.*

Travell, J. G., Simons, D. G. Myofascial Pain and Dysfunction. The Trigger Manual. Williams & Wilkins Books.

Trend. 2007 < www.Trendconsortium.nl > ; The Dutch Ministry of Economic Affairs (BSIK03016)

Vaneker, M., Wilder-Smith, O. H., Schrimbges, P., Oerlemans, H. M (2006) Impairments as measured by ISS do not greatly change between one and eight years after CRPS-1 diagnosis. *Eur. J. Pain. 2006 Oct; 10 (7):* 639-44.

Velthuijzen, J (2005) Effects of pain. Dissertation Aug. 9. UMC Utrecht. NL.

Vlaeyen, J. W. S., Linton, S.J (2000) Fear avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain 2000; 85*: 317-332.

Vleeming, A (1990) Transfer of lumbosacral load to iliac bones and legs. *Spine 1990; 15 (2):* 133-136.

Wang, P., Ba, Z. F., Chaudry, I.H (1993) Endothelial cell dysfunction occurs very early following trauma-hemorrhage and persists despite fluid resuscitation. *Am. J. Physiol. 1993 Sept.*

Wang, P., Ba, Z. F., Stepp, K. J., Chaudry, I. H (1995) Pentoxifylline attenuates the depressed endothelial cell function and vascular muscle contractility following trauma and hemorrhagic shock. *Am. J. Physiol. 1995.*

Wasner, G., Heckmann, K., Maier, C., Baron, R (1999) Vascular abnomalities in acute CRPS: complete inhibition of sympathetic nerve activity with recovery. *Arch. Neurol. 1999 May; 56*: 613-20.

Wasner, G., Schattschneider, J., Heckmann, K., Maier, C., Baron, R (2001) Vascular abnormalities in CRPS-1: mechanisms and diagnostic value. *Brain. 2001 Mar.; 124*: 587-99.

Weiss, P. A (1977) A new image of man in medicine. Vol.1. Toward a man centered medical science. Futura Publishing Company, Mount Kisco. NY. 1977.

Wey van der, L. P (1995) Peripheral nerve elongation by laser Doppler flowmetry-monitored expansion. Dissertation. Nijmegen Universiteits Ziekenhuis.

Wijk van, R., Wijk van, E., (2005) An introduction to human biophoton emission. *Forsch. Komplementärmedizin Klass. Naturheilkd. 2005; 12*: 77-83.

Wilbourn, A. J (1981) A study of carpal pressure. *Journal of Bone joint Surgery. 1981.*

Zago, M. A. B., Posso, M. B. S., Correa, A. L., Posso, I. P (2005) Postoperative pain resulted from the surgical positions: A nursing review. Pain Congress Sydney. 2005, Aug. 24. Lecture 960-P202.

Biography

Anton van Berkel is a Dutch general practitioner in physical therapy since 1970. He has a private practice in the centre of Holland. He received his Bachelor degree in 1980 and a certification in Sports Physical Therapy and Rehabilitation Training in 1990 from the International Academy for Sport science in Nijmegen. His certification in IMT (Integrative Manual Therapy) from the IMT- School in Bloomfield, CT. USA, was in 2004. He started his research on CRPS-1 in 2005 when he started his Master Program at Westbrook University. New Mexico. USA.

From 1972 until 1998 he studied acupuncture and alternative medicine in Europe and Asia, he taught acupuncture and was speaker at national and international acupuncture conferences. A close cooperation with the Pain Department of the Amphia Hospital in Breda started in 1986. He has had in-depth training in many areas, including ear acupuncture, cranial fascial and cranial mandibular pain, soft-laser therapy and is Alumnus of the Upledger Institute in Miami, Florida (Cranial-Sacral Therapy). He studied Experimental Anatomy at the Free University of Brussels in Belgium and momentary, besides CRPS-1, post operative Hand Plastic Surgery patients are his focus now.

In 2005 he started an Office for Pain Control besides his Institute for Physical Therapy <www.antonvanberkel.nl>.