THE PUZZLE OF PAIN, LOSS OF MOBILITY, EVASIVE MOVEMENTS AND THE SELF-MANAGEMENT

CLINICAL ASPECTS OF THE ASSESSMENT, TREATMENT AND DESIGN OF A HOME PROGRAMME FOR 2 PATIENTS. ONE WITH SEVERE SHOULDER PAIN OF NEURO-ORTHOPAEDIC ORIGIN, THE OTHER WITH PROBLEMS DUE TO A LESION OF THE CENTRAL NERVOUS SYSTEM.

By GISELA ROLF

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"The real purpose of scientific method is to make sure nature hasn't misled you into thinking you know something you don't actually know."
Robert M. Pirsig

INTRODUCTION

Around 1986 Maitland expanded, within his system of assessment and treatment in manual therapy, the investigation of the mobility and sensitivity of neural tissues as a possible cause of pain, loss of mobility and muscle spasm. In many patients he claimed to be able to demonstrate that antalgic postures and evasive movements of the trunk might result from neural tissues being at fault.

After Elvey described what he called (1979) the Brachial Plexus Tension Test, 1986 the Upper Limb Tension Test/ULTT (1986) and later the Upper Limb Neural Tissue Provocation Test / ULNP (Elvey 1995) physiotherapists could more clearly differentiate between joint tissues, muscle tissues and neural tissues as sources of pain and dysfunction. New clinical insights into the contribution of neural tissue dynamics to acute and chronic pain and its treatment were stimulated. The assessment and treatment of joint dysfunction, the loss of neural tissue mobility and protective muscle spasm were primarily thought of as "mechanical" (Elvey R L 1986) but an attempt to link it to scientific research on neural tissues` response to irritation was made.

Since *Mobilisation of the Nervous System (*Butler 1991) was first published, physiotherapists have increasingly considered and included neural structures in the assessment, reassessment and treatment of the symptoms and signs of their patients.

Shacklock (1995) suggested, "*The term neurodynamics may be deployed to include the link between mechanical and physiological types of mechanisms*". Physiotherapists have been encouraged to attempt to influence directly and indirectly the patho-biomechanical changes of neural tissues which so often include pathoneurophysiological reactions, such as vascular and biochemical processes which, in turn, appear to affect target tissues adversely. Target tissues are those structures such as muscles, connective tissues, joints, intervertebral discs, the vascular system, organs, and the connective tissues of the nervous system itself that are directly or indirectly innervated and controlled by the nervous system.

However, it is obvious that we lack much information in the scientific field about the nervous system and especially about *Neurodynamics*. Even recent studies on the nervi nervorum, suggested as the missing link between all the other factors contributing to neuropathic pain (Bove and Light, 1997; Ochoa 1997; Sorkin 1997) can offer only a hypothesis to support our clinical methods.

"The biomechanics of the peripheral and especially of the central nervous system sadly is neglected in research and although I asked many of my colleagues on international congresses who are especially involved in neurophysiology we do not know about any study in the field of neurodynamics and its relation to clinical states" (Prof. Dr. med J. Kesselring, neurologist, director of the Rehabilitation Centre, clinic Valens, Switzerland, correspondence February 2000).

All physiotherapeutic techniques, either by active treatment to improve selective muscle function and reduce pain, or passive techniques to similarly influence signs and symptoms are in essence mechanical since they employ movement. They aim to ease or eliminate pain, improve mobility and regain normal muscle tone and re-educate selective muscle function, all of this in order to restore normal function to the patient.

Shacklock (1999) writing on manual therapy reasons that the method of clinical management operates on all levels of the healing process: *"What is important is that treatment accommodates not just the brain and spinal cord, but all relevant stages in which input and output mechanisms are integrated with clinical reasoning. Each component as part of a reasoning model can be assessed and treated with purpose. A focus on learning (e.g. motor and cognitive) and conditioning processes should be key aspects, with the understanding that therapy must change the culpable mechanisms, whether they be generated in the brain or the body"*.

The holistic understanding of "a person in pain and with disability" demands from the physiotherapist many different levels of comprehension, skills and knowledge: e.g. an integrated scientific and clinical approach to biomechanics, to biochemist, to anatomy, to physiology and to pathology. Physiotherapists should also understand psychosocial processes, have knowledge about body awareness in order to modify habits and give adequate support to the patient in the development of confidence. They need to be informed about perception and significant learning strategies, about interaction and communication amongst their many skills.

"Whilst mobilisation of neural tissues remains a useful modality in the treatment of neural disorders, refinements are necessary. Consideration of relevant physiology in assessment and treatment of neurogenic pain is an important refinement if neural mobilisation is to become well founded, safe and effective. One of the dangers in treatment of neural tissues is the reliance on mechanics. In the not too distant past, this reliance may have led to inappropriate physical treatment to neural tissues" (Shacklock, 1995). Undoubtedly every day we learn from patients that we need more skills, experience, knowledge and further means of treatment. However the physiotherapist's ability to select appropriately and find the right balance with all the treatment tools that serve the individual needs of the patient is an essential goal.

WHEN SELECTIVE MUSCLE ACTIVITY FAILS PATHOLOGY IS FACILITATED AND PREDISPOSING PROCESSES DEVELOP IN TARGET AND NEURAL TISSUES PRODUCING SYMPTOMS AND SIGNS.

"*A disorder in the nerve at one site may predispose to another lesion either distally or proximally along the nervous system"* (Gunn and Milbrandt, 1978). Clinical experience confirms that one disorder can predispose an individual to another disorder in neural tissues as well as in target tissues and vice versa. For example the loss of selective trunk muscle activity, especially the selective oblique muscle activity of the abdominal wall (Davies 1990; Davies 2000), which normally pulls the ribs down diagonally and prevents the thorax and the shoulder girdle from lifting up presents the human body with many problems:

" *They regulate intra abdominal pressure, are involved in respiration, stabilise the body axis during equilibrium reactions against gravity, influence selective and pain free shoulder function and participate in equilibrium movements of the pelvis while walking* (S.Klein-Vogelbach, 1986).

Even if this selective trunk activity is only slightly weakened and therefore "imbalanced", other target and neural tissues will be overused proximally and distally due to evasive movements and postures. This occurs as the body tries to compensate for the loss of active stabilisation proximally, which normally forms a protection for all target and neural tissues.

The following diagram draws attention to the close the interrelationship and dependence of all tissues within the body, whether neural or target tissues, and therefore to the possibility that tissues can predispose each other to symptoms and signs.

Fig. 1

Neurodynamics, as defined by Shacklock (1995), summarises in this diagram the functional interrelationship between neurobiomechanical responses and neurophysiological responses within the nervous system. Patho-neurobiomechanics, as defined by Shacklock (1995), summarises the interrelationship between patho-neurobiomechanical and patho-physiological responses within the nervous system.

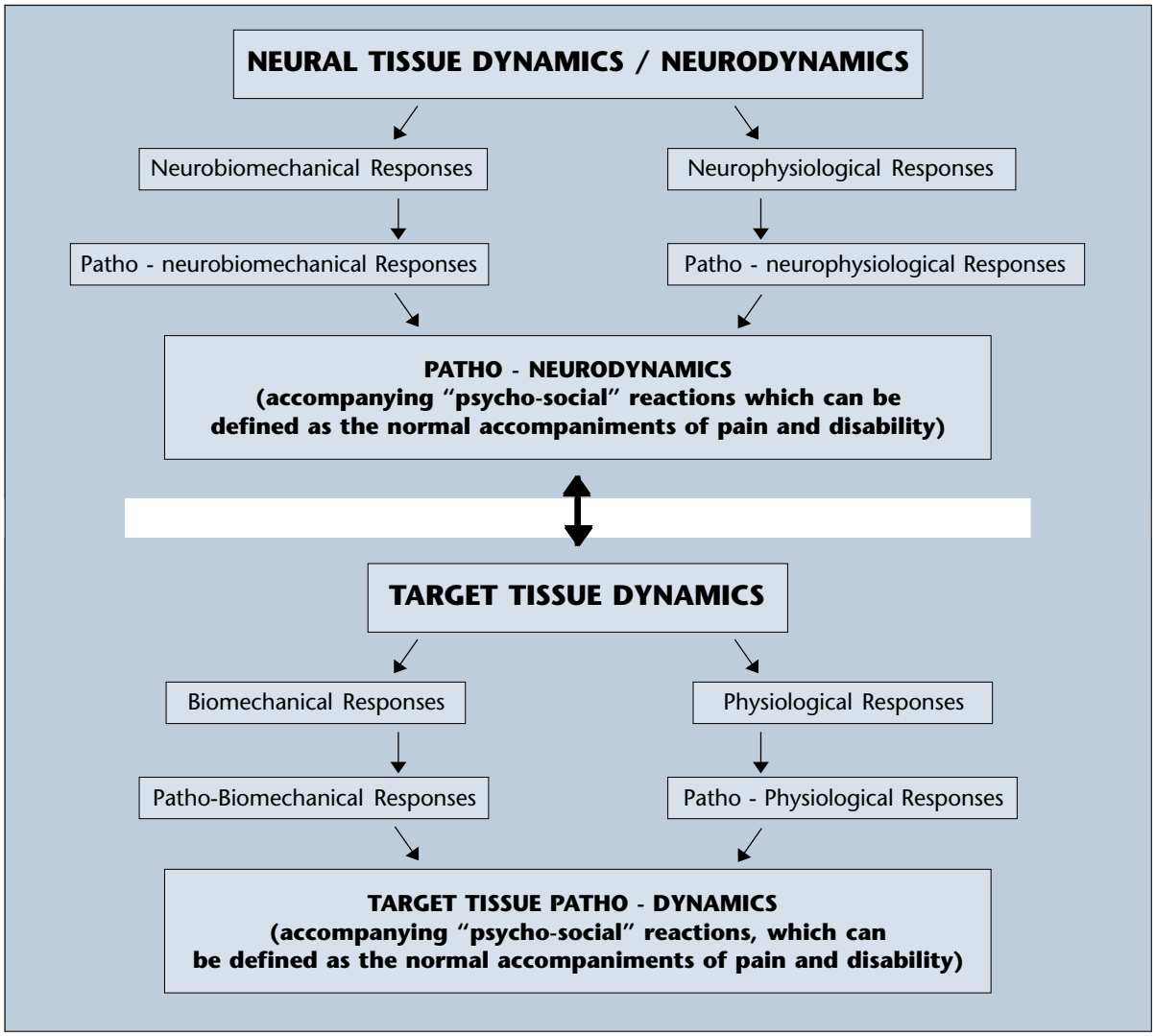


Fig. 1

Target Tissue Dynamics summarises in this diagram, the functional interrelationship between the biomechanical and the physiological responses of all tissues. Target Tissue Patho-Dynamics describes the interrelationship between patho-mechanical and patho-physiological mechanisms within all target tissue. The diagram attempts to relate the clinical observation of how neural tissue movement (neurodynamics) influences directly and indirectly non-neural tissue activity (target tissue dynamics) and, vice verse, how target tissue dynamics influence directly and indirectly the neurodynamics.

Because the nervous system is a continuous tissue tract the cervical neuraxis cannot be seen, in terms of dynamics, separately from the thoracic and the lumbar neuraxis. *NEURAXIS is a term used when the CNS is considered along its length irrespective of its bends and folds* (Bowsher,1988); *the neuraxis/spinal cord is a continuation of the medulla oblongata* (Butler, 1991). Certainly clinically these anatomical regions of the spinal cord influence each other with regard to pathological states such as restricted mobility (elongation and accessory movements such as antero-posterior, transverse and rotatory movements), pain responses to movement, evasive movements and antalgic postures, change in muscle tone, and autonomic reactions. In this regard special attention should be given to the thoracic spine as a main facilitator of lumbar and cervical vertebral pathology and muscle-skeletal complaints in the upper and lower limb. Diffuse autonomic or localised symptoms such as apparent visceral complaints in the upper and lower abdomen can also be provoked by the thoracic neuraxis.
The loss of selective trunk activities, depriving the trunk of the means of effective active stabilisation against gravity is likely to cause progressive mechanical problems by overusing target and neural tissues thereby predisposing to pathology, as the above diagram indicates. Therefore the re-education of selective muscle activity and at the same time the attempted reversal of all patho-biomechanical and patho-physiolocal tissue problems is of vital importance in order to prevent recurrent problems for the patient. The interdependence between a normal nervous system and normal target tissues cannot be emphasised enough. In order for the nervous system to mantain its functional integrity the target tissues must in turn, enable it to spread out and adapt to any position and body movement (Butler, 1991). If the nervous system is not mobilised and protected by muscle activities and free joint mobility - as is so often the case clinically in patients who have suffered a central lesion and are immobilised - it rapidly loses its normal biomechanical properties when tested and normal physiological responses like the appropriate autonomic activation are altered. The most important of all neuro-psychological responses is the impulse transport which is unique to the nervous system.

NEURO-DYNAMIC TESTING AND TREATMENT
The neuro-dynamic tests described by Maitland (1985), by Elvey (1986) and by Butler (1991) assist in diagnosis of neurodynamic faults. The authors explain how, by varying neural test components and their sensitising movements or by using palpation techniques for peripheral nerves it is possible in most cases to differentiate between neural and target tissues as components of a disorder. Investigation by movements of the neuraxis in the cervical, thoracic and lumbar regions, and the addition of components of the *Neural Tissue Provocation Tests* reveal how neural patho-dynamics frequently cause symptoms in spinal areas, the trunk, the limbs and the head such as nausea, and apparent organic pain such as attacks of angina pectoris, and gall bladder symptoms.

There are various ways in which the nervous system may be moved in which its forces may be modified and thus its patho-biomechanical properties and patho-neurophysiological responses altered. The examiner must be sure that the diagnostic tests for tissue structures/organs have been carried out thus indicating that neural tissue problems may be present.

INFLUENCING NEURAL TISSUE IN A DIRECT WAY
Using neurodynamic tests or their components (passive physiological movements for the nervous system), in different sequences and combinations, adding in sensitising movements and including the neuraxis in the neurodynamic tests offer a clinically reliable way to assess, treat and re-assess neural tissue movement and sensitivity. This is so especially, in conditions where inert resistance is the main limiting factor of movement, with pain appearing at the limit of neural mobility.

Palpation techniques such as local transverse movements of the nerve tissue (Butler, 1995) against the mechanical interface of the nervous system (this technique can be described as passive accessory movements to the nervous system) applied to peripheral nerves, nerve roots and the different parts of the plexi aid diagnosis and serve as therapeutic techniques (Butler, 1995). A second passive accessory movement of neural tissues is a locally applied "roll-over" of a peripheral nerve with some gentle pressure applied within its nerve bed.

*(**Fig. 2**) Accessory movement to neural tissue (Brachial plexus, Median nerve), transverse movement of the neural tissues against the mechanical interface*

INFLUENCING NEURAL TISSUES IN AN INDIRECT WAY
This occurs firstly with active muscle work such as selective stabilisation of the trunk followed by movement of the limbs or, vice versa, stabilizing the limbs and moving the trunk. An indicator of the quality of selective muscle function is the ability to rotate or counter any rotator activity actively and then to rotate against rotatory muscle function. Any method of motor learning and re-education of selective muscle

function may modify the biomechanics of neural tissues and influence therefore the physiology of the nervous system in an indirect way.

Secondly any mobilisation of interfacing tissues such as joint mobilisation constitutes an indirect method of influencing neural tissue dynamics e.g. passive unilateral postero-anterior mobilisation of T5/6 vertebral segments.

As a clinical observation, postural correction and regular re-positioning, as for example with a patient who suffered a hemiplegia, can alter directly and indirectly the biodynamic reaction of neural tissues, as may be seen in the progressive modification of allodynia and hyperaesthesia of neural structures.

ASPECTS OF TREATMENT

All the neurodynamic tests and test components can be applied as treatment, according to the clinical evaluation concept of Maitland, either in a very gentle way or more forcefully as necessary. However in the case of direct mobilisation of the neural tissue passive movement should be applied only as a rhythmical mobilisation and never as a prolonged stretching at the limit of range. In some cases it appears more favourable to encourage neural tissue mobility and ease symptoms when, for example, the trunk is moved rhythmically against the limb, which can be positioned in some or all components of a neurodynamic test sequence.

All such rhythmical neural mobilisations can be done either without pain, or by just provoking the pain, or going just beyond, reproducing "the pain", or encountering resistance with little pain, and preferably without pain, towards the limit of the movement. There is no justification for hurting the patient unnecessarily. The gentleness or force of the mobilisation will depend upon the assessment, re-assessment and the nature of the problem (Maitland, 1986).

The accessory movements to neural tissues such as the locally applied transverse mobilisation and "roll-over" mobilisation (palpation techniques) can be applied to influence severe pain syndromes most favourably. Although there is as yet no scientific proof to support these clinical observations, experience suggests that they have some validity in the treatment of severe pain, protective muscle spasm and loss of accessory and physiological neural tissue mobility in conditions with diffuse and strange symptoms e.g.: sympathetically maintained pain syndromes (McMahon, 1991: Melzack, R, 1991) which may develop after a lesion of the central nervous system.

It is of vital importance to re-assess, after each neural tissue mobilisation, possible changes in the quality of muscle activity because so often immediately after neurodynamic treatment this can change for the better. To retrain and regain normal muscle function immediately after a passive mobilisation of neural tissue is most important. Clinical experience has shown that neural mobility is quickly lost again and the symptoms reoccur if muscle activity and range of motion in both accessory and physiological joint movements cannot maintain the passively regained mobility of the nervous system. For the same reason any evasive movement or antalgic posture of the trunk and/or limbs has to be actively corrected to their active optimum after passive neural tissue mobilisation.

DESIGN OF A HOMEPROGRAMME AND ENCOURAGEMENT OF SELF-MANAGEMENT

From the first treatment the patient should be helped to understand the response of his symptoms to his daily activities, to his postural habits, to his active muscle control and to his psychological state. The patient must learn what provokes his symptoms and signs through a detailed and patiently maintained teaching and learning process (on both his and the therapists side) in order to perform a home programme and monitor self-management with a modified life style. To relearn physical awareness can take some time especially if the patient, has been taking analgesics, anti-inflammatory medication and mood altering drugs over a long period of time.
In order to protect his body tissues and to prevent further overuse and damage to them as part of his learning process, the patient often needs enhanced sensory input and guidance from the hand of the physiotherapist. The guidance will facilitate awareness of how much movement to perform, how not overdo movements and how to correct postures. The frequency and extent of tissue self-mobilisation, the exactness of active exercises, the recognition and correction of evasive or substitute movements and postures have all to be learned. This learning process guided by the evaluation of each sequence by the physiotherapist, is necessary in the beginning.
As soon as possible the patient should also be encouraged to make his own discoveries such as reducing his pain and improving mobility by auto-mobilisation of neural and target tissues. A self-management programme can begin to be established in his daily life.

> " I keep six honest serving-men
> (They taught me all I knew);
> Their names are *WHAT* and *WHY* and *WHEN*
> And *HOW* and *WHERE* and *WHO*.
> I send them over land and sea,
> I send them east and west;
> But after they have worked for me
> I give them all a rest "
>
> R. Kipling

The patient feels safer, as they describe it, if they can use these guidelines of knowing exactly *what* to do *how* and *when* it is necessary, *why* they must do it, and *who*, if anyone, should be involved to help.

The resultant reduction of his anxiety in response to unpredictable pain attacks, discomfort and stiffness

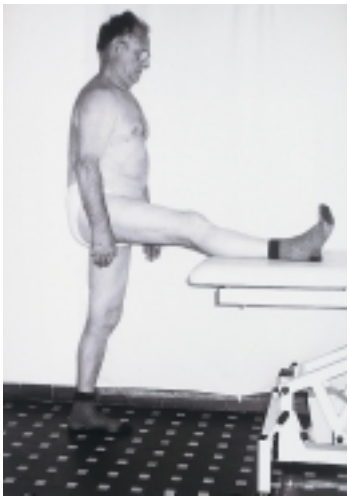
and his growing confidence in his ability to modify his symptoms will lead to a re-organisation of his life along self protection and prophylactic lines. *"There is increasing evidence that people want to understand more about their bodies, both in the prevention of ill health, as well as in the promotion of health and fitness"* (Watson, 1996).

SOME ASPECTS CONCERNING THERAPEUTIC EXERCISES AS A PRE-REQUISIT FOR SELECTIVE MUSCLE FUNCTION

Progress will be more rapid if the patient is in the position of contributing to the treatment with active exercises at home as well as tissue self mobilisation.

" A very specific type of therapeutic exercise has been devised which provides effective pain relief for chronic and recurrent back pain sufferers probably through enhanced segmental stabilisation".
(C. A. Richardson; Jull G A, 1995). These therapeutic exercises have been of great value for patients with acute and chronic back pain, and can, if segmental stabilisation is performed in positions which enhance neural and target tissue mobility, improve both the muscle stabilisation effect and the neurodynamic and target tissue symptoms and signs.

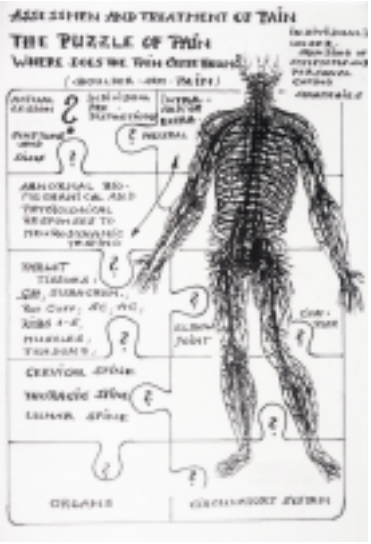
Active segmental stabilisation is much more difficult to maintain against neuro-dynamically generated symptoms and variations in muscle tone. However, it should be progressed step by step until the neural tissues are mobile enough to allow the target tissue to perform more easily, for example muscle tone being reduced and able to work more smoothly "through range" (Fig. 3, 4, 5).



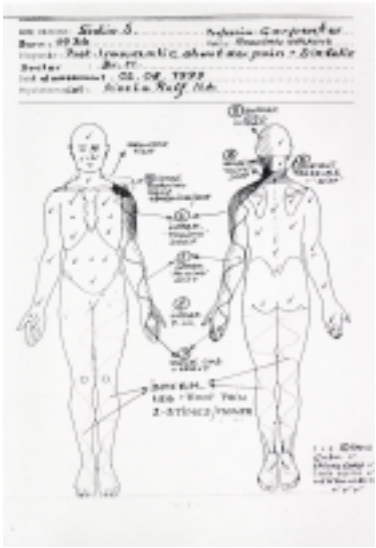
(Fig. 3) *SLR (lower limb neural tissue provocation test components) mobilisation "at limit" adding in dorsi flexion of the foot and performing active trunk stabilisation against the restriction of the SLR neural tissues.*

(Fig. 4) *ULNP (upper limb neural tissue provocation test) position at limit against the wall maintaining segmental stabilisation of the trunk at the same time and mobilising the neck away from the ULNP position: one foot is placed on a stool to avoid excessive lumbar extension/evasive movement.*

(Fig. 5) *Active antero-posterior auto-mobilisation of the cervical spine correcting upper cervical extension and lower cervical flexion; the ULNP position against the wall is maintained for the left arm, hand and fingers. One leg is positioned on a stool in order to keep the lumbar spine in a neutral position.*



(Fig. 6)
Puzzle of Pain



(Fig. 7)
Body Chart of Mr. S.

ASSESSMENT

History. Three weeks before calling for an appointment, Mr. S. slipped on the stairs and banged his head and his left shoulder against the wall. The patient felt a stabbing pain in his lower neck and left shoulder at the time of the accident, but this cleared and he did not feel any pain.

After a week he was wakened about 4 a.m. by severe shoulder pain left, when lying on his left side. He had to get up, because he could not find any comfortable position and about 8 a.m. the shoulder pain increased to such an extent that he could not move his arm at all and had to consult a doctor.

In the following 3 weeks he received 9 intra-articular injections, anti-inflammatory medicine and was supplied with painkillers and psycho-modifying drugs neither of which he took. After 3 weeks the shoulder pain was reduced by about 30%, and the neck felt very stiff. He suffered headaches each morning until about 11a.m. He was wakened by pins and needles in his left fingers each night about 4 a.m. He could not lie on his left side (his normally comfortable sleeping position) and he could not perform any shoulder nor arm movements without pain. His left arm felt heavy, cold and very weak which concerned him a lot. Being a carpenter, he had to lift heavy objects and deliver furniture to customers.

Mr. S. has been diabetic for 21 years. He did not take any other medication, felt well and healthy and did up to 10 hours of mountain walking each day at weekends. Once or twice a month he suffered severe diffuse leg and foot pains right and left for about 5-6 hours at night, which forced him to get up and walk about. He did not know why and no doctor could advise him what to do in order to change these severe pain attacks.

He had lived alone since his wife died of cancer 9 years ago and looked after his home.
He suffered 2 accidents in his life: when he was 25 he fell from a tree. After falling some 8 meters he landed on his bottom fracturing his coccyx. The severe local pain at the base of the spine gradually disappeared over a period of 3 months. The second accident happened when he was aged 46. He slipped on an ice patch and landed with great force in extension on his back and head. He suffered headaches for some 6 months and intermittent lumbar pain for one year, but after that he felt perfectly well, but markedly less mobile in his spine and especially his neck.

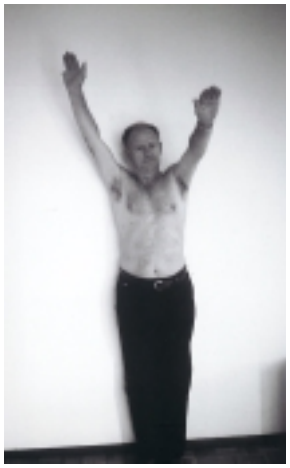
Subjective assessment/behaviour of the main areas of symptoms:
Symptoms in area 1 (see body chart) woke him frequently at night. The patient could not sleep well because every position was uncomfortable after a few minutes. Area 2, 4, 6 also troubled him at night, but with shifting around these areas of pain eased for a short period.

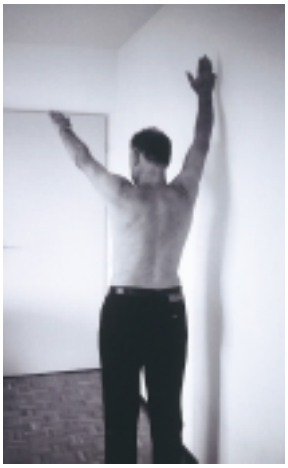
On waking the patient felt tired and stiff all over, but after a hot shower and moving around eased this stiffness. Area 1 and 2 were less troublesome and areas 4, 5, 7, 8 became less intense and were more bearable with walking. The patient could not even lift 2 kg, because of pain in area 1, 2, 4. Getting dressed and undressed gave more pain in area 1, 2, 3, 4, but this settled quickly.

Selected data from ongoing records are detailed below.
Physical assessment. Day 1, 9 June 1999: The first assessment procedures were carried out gently because of longstanding diabetes and severe left shoulder pain, which increased with every active movement.

Active movements
Left gleno-humeral joint: all movements reproduced pain in the areas marked on the body chart 1, 2, 4, 5, 6 and were severely restricted (Fig. 8-11)

(Fig 8) 1st assessment: antalgic posture of his left shoulder girdle; the swelling over the left shoulder is marked.





(Fig. 9 & 10) 1st assessment: G/H flexion with evasive movements anteriorly and with cervical lateral flexion.

(Fig. 11a) 1st assessment: active test movement left arm "hand on back".

(Fig. 11b) 1st assessment: G/H joint abduction with evasive movements of the left shoulder girdle into elevation and medial rotation of the G/H joint.

Right gleno-humeral joint: with regard to mobility there was a little stiffness in all directions, but no pain, except in area 6 with 100 ° abduction.

Left rotator cuff: all resisted movements in abduction, lateral rotation, medial rotation and elbow flexion showed markedly reduced muscle power and severe pain in area 1, 2, 3, 5,

Right rotator cuff: no pain and full strength

Passive physiological movements
Left gleno-humeral joint:
Flexion: 60°, reproduced neck and upper arm symptoms (2, 4)
Abduction: 60°, reproduced (4) sharply.
Extension: 0°, and gave diffuse pain (2, 4, 5)
Lateral and medial rotation were not possible to perform because of the severity of pain in area 1 and 4
Left gleno-humeral joint, accessory movements:
Postero-anterior (grade II- -) local pain and area 4 severe; antero-posterior (grade II) local pain and area 4 severe; antero-posterior (grade II): local pain plus area 4, severe; medial rotation (grade I): reproduced 1 and 4; lateral rotation (grade I): reproduced 4 and 5.

Re-assessment: no change in the gleno-humeral active and passive movements

Active Tests: Cervical Spine
All active movements were restricted. There was no movement in the upper and lower cervical spine for lateral flexion to the right and to the left. In the mid cervical lateral flexion was 30°, reproducing areas 2, 3, 4. Rotation to the left and to the right was 15°, reproducing areas 2, 3, 4. There was no extension in the lower cervical spine, but the spine was held from C5–T4 in 30° of flexion with a stiff upper cervical spine in extension. Neither cervical positions could be reduced because of severe pain in area 2.

Cervical Palpation (Patient Prone Lying)
The left arm had to be positioned alongside the trunk because of the severe shoulder pain.
All vertebral levels right and left felt very, very stiff with palpable scar tissue in the lamina and in the interspinous spaces. C3 and C5 were very deep set. The posterior neck muscles and soft tissues were very tight, hard and shortened, and thickened especially sub-occipitally. Sharp local pain was elicited with left unilateral postero-anterior mobilisation (grade II-) on C5/6, C3/4, C2/3 and 0/C1. All other levels on the left were very stiff, but painless (grade IV). From 0/C1 to T4/5 on the right (grade IV), were very stiff but no pain.

With antero-posterior mobilisation techniques applied unilaterally at C3/4 and C4/5 left (grade II- -) sharp local pain was elicited radiating to areas 2, 4, 5, 8. Other levels left and right anterio-posterior stiff but gave no pain (grade IV).

The left brachial plexus felt very tight and immobile at the mechanical interface in its upper and lower parts. The left shoulder girdle was held in elevation; no correction was possible in prone lying because of pain in areas 1, 2, 4. Accessory movements (transverse grade II- -) on the upper and on the lower part of the brachial plexus showed no movement in the axilla, but caused diffuse pressure and heaviness into the left axilla with pins and needles into the left hand and all fingers.

The re-assessment immediately after the testing showed no change in the pain nor in joint mobility of either the cervical spine or the gleno-humeral joint complex.

LOWER LIMB NEURAL
TISSUE PROVOCATION TESTS
Right Passive Straight Leg Raise (SLR) without dorsi-flexion: 35°, reproduced area 2 (diffuse and deep) and area 4, and caused evasive movement of the trunk with a transverse shift to the left. Correction of the trunk to middle position gave pain in area 2, 4 (sharp and severe). An evasive movement of the left shoulder girdle into elevation increased with SLR 30°; correction was not possible because of pain in the left arm areas 1, 2, 4, 5, 8.

Treatment: Ten neural tissue mobilisations SLR without dorsi-flexion 20° slowly and without pain (grade II-).

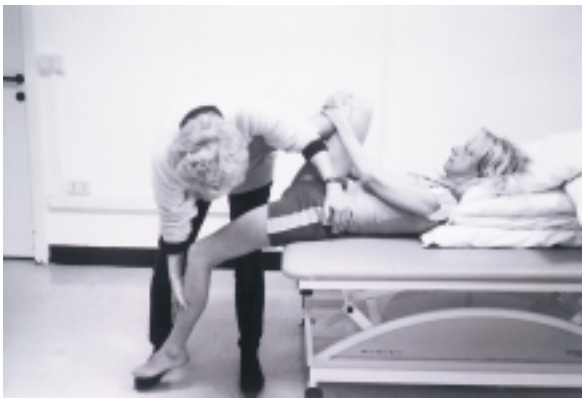
Re-assessment: a marked difference with cervical active movements in all directions. The gleno-humeral passive joint flexion improved from 60° to 100°, reproducing a little pain in area 1, 4;
Abduction from 60° to 90°, reproducing pain in area 1, 4. *Rotator cuff:* external rotation, abduction, medial rotation and elbow flexion could all be done actively a little, with some pain reproduced in area 1 and 4.

Left SLR without dorsi-flexion: 70°, gave a stretching feeling behind the knee, adding full dorsi- flexion gave more of a stretching feeling. Adding adduction and medial rotation produced no more discomfort.

Right Femoral Nerve Provocation Test in left side lying: was not possible because patient could not lie on his left side due to severe shoulder pain.

But by positioning the patient supine with flexion of the cervical and thoracic spines using pillows and with the left leg in flexion to keep the lumbar spine in neutral, hip extension showed restriction of 30° with the knee kept in 90° flexion. The patient felt a strong stretching discomfort on the anterior thigh. The attempt to add knee flexion increased the stretch of the anterior thigh markedly. Subtracting upper cervical and mid thoracic flexion abolished the stretching discomfort anteriorly on the thigh.

Left Femoral Nerve Provocation Test (first cervical and upper thoracic spine positioned in flexion without pain): hip extension was -20° and gave a stretching feeling over the anterior thigh, adding in 70° flexion of the knee (limit) provoked hyperextension of the upper lumbar spine, but no pain. Moving the cervical and thoracic spine back into neutral gave the hip more mobility into extension and 90° knee flexion without pain was possible.
Re-assessment: no difference in pain or mobility behaviour in the cervical spine and left shoulder movements.



(Fig. 12) *Femoral provocation test in supine, for a patient who suffered a left hemiplegia: The limit of the mobility with full hip extension, was 80° of knee flexion with pain on the anterior part of the thigh. The patient was unable to activate muscles to obtain this position. This position could be used for any patient who finds it difficult to lie on his/her side, or when stiffness of the hips and of the lumbar spine prevent accurate neural testing in side lying, or if muscle activity makes the side lying position too instable and therefore inadequate for accurate neurodynamic testing.*

UPPER LIMB NEURAL
TISSUE PROVOCATION TESTS
Right ULNP 1:
Gleno-humeral abduction 80° reproduced area 1, 4:
On reducing gleno-humeral abduction to 70° without pain adding full supination, abolished pain; adding wrist extension: 20° gave no pain; adding finger extension: all joints free range without pain; adding thumb abduction 45° and extension/interphalangeal joint 100° limit, no pain; adding gleno-humeral joint lateral rotation abolished pain; adding elbow extension to 100° reproduced severe pain 1, 2, 4, 5 and 8.
Re-assessment of cervical active movements:
More range was possible with lateral flexion to the left and right but the same pain responses in area 1, 2, 4, 6. The gleno-humeral active joint movements and pain responses were unchanged.

Left ULNP 1: not done at the 1st assessment

Right ULNP 2a with median nerve bias; right ULNP 2b with radial nerve bias; right ULNP 3a with ulnar nerve bias; right ULNP 3b with ulnar nerve bias: were all restricted in almost all components, however no pain was elicited, only a strong stretching feeling over the whole arm and hand anteriorly and posteriorly while adding the components.

Left ULNP 2a - 3b: not done on the day of the 1st assessment.

Right 1st rib: passive accessory movements: felt stiff at grade IV, no pain.

Left 1st rib: passive accessory movements: could not be touched because of severity of pain.

Right acromio-clavicular joint: passive accessory movements: felt restricted in all directions with grade IV, no pain.

Left acromio-clavicular joint: passive accessory movements: marked swelling on the joint and very warm. It could not be moved because of severity of pain.

Shoulder horizontal adduction was very restricted and gave severe pain in areas 1, 4

(Fig. 13) *1st assessment: horizontal adduction of the left shoulder, stressing the acromio-clavicular and sterno-clavicular joints.*

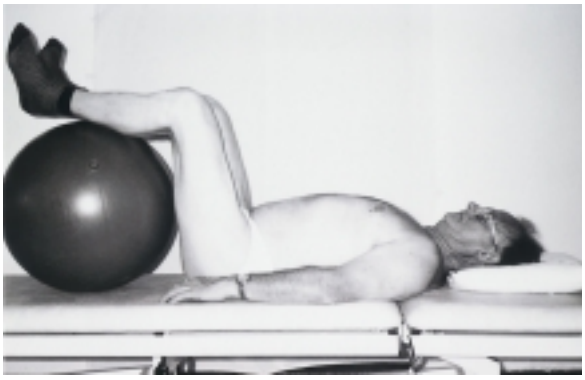
Left sterno-clavicular joint movements: in all directions local pain with grade II

Right sterno-clavicular joint movements: in all directions locally sensitive with grade IV

On observation no *selective abdominal muscle work could be seen during the examination.* The patient lay supine with excessive lumbar extension and the rib cage was extremely elevated (fig. 14).

Treatment and Teaching session for the home programme/supine: selective activity of the abdominal muscles short of pain or discomfort, legs positioned in hip and knee flexion 90° (fig. 15).

(Fig. 14) *1st assessment Mr. S. no abdominal muscle activity*



(Fig. 15) *Selective muscle activity/supine: legs supported, heels compressed against each other to encourage lower abdominal muscle activity; 3rd re-assessment and treatment*

Re-assessment of right SLR without dorsi-flexion: had increased to 60° with only slight reproduction of pain in area 1, 2, 4, 5. Gleno-humeral active joint movements unchanged but far less evasive movement of the left shoulder girdle into elevation occurred.

Treatment and Teaching session for the home programme: the patient was shown how to mobilise the right SLR without dorsi-flexion and without evasive movements of the left hip into flexion and using active abdominal bracing and thus avoiding lumbar and low thoracic hyperextension and elevation of the rib cage.

Re-assessment: the neck and the shoulder pain felt easier; no physical testing was done
Warning: the patient was asked for a detailed report of the pain behaviour over the next 2 days; the patient was given the telephone number because he should return the next day if any pain areas worsened.

Home programme for the following two days:

1. Abdominal muscle activities in lying with the legs supported in 90° flexion of the hips over a chair and with 90° knee flexion: 20 times in the morning before getting up and 20 times during the afternoon or evening, making sure that the rib cage moved diagonally down towards the navel. These active contractions of the abdominal muscles were to be carried out without provoking any pain. The patient had to re-assess his active shoulder movements for pain and mobility after each session.
2. Right SLR mobilisation in standing: the patient stood in the doorway and swung the right leg into hip flexion with firm extension of the right knee and some dorsi-flexion of the foot. Evasive movements of the left hip and pelvis were to be actively countered and the trunk actively stabilised with abdominal muscles. The patient was asked to perform a minimum of 20 movements with active stabilisation of the trunk, every day.
3. An ice pack was applied for a few minutes to the left acromio-clavicular joint for a minimum of 10 times a day by the patient.

2nd re-assessment and treatment: day 3
The patient reported that the pain in areas 1, 2, 4, 5, 6, 8 had lessened about 40%. He had followed his home programme as taught over the two days.
Body chart: pain in symptom area 2 was now intermittent, 1 less burning, 4 only when lying on the left shoulder. The patient was still wakened by pain in area 4 during the night when turning in his sleep on his left side. Pain in area 8 did not occur and area 7 felt cold and heavy only at times. The condition in area 6 was unchanged and the whole neck felt stiff.

The physical assessment
Active movements left gleno-humeral joint:
Flexion 130°, reproduced pain in area 1, 4*; abduction* 100°, reproduced pain in area 1, 2, 4, but less sharp*; lateral rotation* 20° reproduced pain in area 1 and 4; *medial rotation* 20° without pain; *hand on back* 30% less than right only reproducing a little pain in area 1, 2, 4
Left rotator cuff: the patient could counter medium resistance, but pain in area 1 and 4 was reproduced
Treatment: left gleno-humeral accessory movements left: 20 times postero-anterior mobilisation grade III- - without pain, 20 oscillations.

Re-assessment: gleno-humeral flexion 130°, but less pain in area 1, 4; gleno-humeral abduction 110°(improved) and less pain in area 1, 4. *Repeat treatment (same):* no change in gleno-humeral active movements.
Cervical active mobility has become less in all directions and the lateral flexion to the right was more painful in area 1, 2, 4. The cervical rotation to the left and to the right was unchanged.

Treatment of the cervical spine:
Right and left postero-anterior unilateral and central mobilisations grade III- -, at each level from 0/C1 to TH 6/7 with re-assessment of the cervical mobility after each mobilisation set at each level. The local pain responses reduced quickly during passive accessory central and unilateral mobilisations. The antero-posterior unilateral mobilisation grade III - - was performed at each level from C2/3 to C6/7 and cervical mobility was re-assessed after each mobilisation session on each level. The local pain response to movement lessened and reproduction of pain areas 2, 4, 5, 8 was only diffuse.

Re-assessment: the patient felt less stiff in the neck; the quality of the movements in all directions had improved and the lateral flexion to the left was the same as to the right, but reproduced slight pain in area 2 and 4. Left gleno- humeral range and pain response was the same.

Treatment and teaching session for the home programme: sitting on a firm chair. Active mobilisation of the lower cervical spine in an antero-posterior direction with chin tucking and with an active stabilisation of the trunk, keeping the left shoulder girdle down (correcting actively the evasive movement). All components of this sequence are done without pain aggravation, but into the feeling of resistance.

Re-assessment: "my neck feels free". *Active cervical movements:* rotation to the left 35°, no pain; rotation to the right 35°, gave no pain. Left lateral flexion (mid cervical) was 30° (upper and lower cervical lateral flexion was still very stiff) and no pain; overpressure was not applied. Lower cervical extension: virtually no movement but fixed in 30° flexion; upper cervical extension: full range, no pain. Right SLR without dorsi-flexion: 80° reproducing very slight pain in area 1, 2, 4.

Treatment: 20 times right SLR 70° without dorsi-flexion; mobilisation with the hip in some degree of medial rotation grade III without pain, then 20 times into adduction III without pain; then adding in dorsal flexion at limit 20 times touching the beginning of pain (P1) of areas 1, 2, 4.

Re-assessment: The shoulder *"feels good and free";* cervical mobility in all directions improved and lateral flexion to the left, rotation to the left and to the right did not reproduce any pain.

Repeat treatment: right SLR 70° right with some dorsi-flexion without pain: 30 times grade III- - -, then to 70° with dorsi-flexion and with very slight pain in area 2 and 4, 20 times SLR grade III. The evasive thoracic movements to the left were less marked and could be corrected and overcorrected without any pain. The antalgic posture into elevation of the left shoulder girdle could be corrected to the mid line without pain.

Re-assessment: the cervical active movements were without pain responses, however the through range quality of each movement did not look smooth. Oscillatory overpressure grade IV - - very slowly at the end of the available range of lateral flexion to the right and to the left and of rotation to the right and to the left reproduced, only with lateral flexion to the right, slight pain in area 2, 4, 5.

Slight oscillation (grade IV- -) into lower cervical extension at limit of the active range reproduced a sharp pain in area 6 and "pins and needles" in area 8.
Left acromio-clavicular joint: the swelling and the heat over the joint were less severe. The antero-posterior mobilisation on the joint line, on the clavicle and on the acromion with grade II - - produced a local pain, which the patient could accept.

Treatment: antero-posterior mobilisation (grade I), 20 oscillations with angulations to the right and to the left with a bearable local pain response, but after 18 mobilisations the joint started to develop more heat.
Re-assessment: with horizontal adduction of the shoulder and the shoulder girdle the elbow passed the middle line of the body, but at the limit of the

available active range the patient felt a sharp twinge of pain over the A/C joint.
Advice: continued use of ice treatment at home.

Treatment: *Left 1st rib: 20 longitudinal caudal mobilisations grade II without pain; then moving along the 1st rib with anterior posterior and posterior anterior mobilisations (grade III-) without pain for 5 minutes.*
Re-assessment: the antalgic posture of the left shoulder into elevation was reduced; the cervical active movements were the same; the range of gleno-humeral active joint movements were the same but now without pain.
Right ULNP 1: range of each component remained the same, but the pain response was "40%" less.

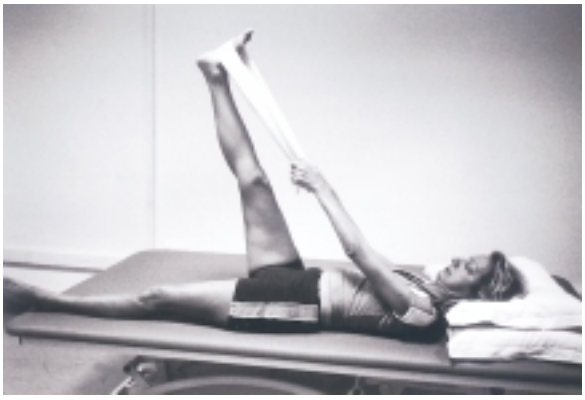
Treatment: mobilisation for 10 minutes of all components separately of the right ULNP 1 with grade III- reproducing a slight stretching discomfort.
Re-assessment: all active movements of the cervical spine were the same, but there was no pain now. All gleno-humeral movements to the limit of the active range were without pain: flexion 160°, abduction 130°, lateral rotation in adduction 30°, medial rotation 25°.
Left ULNP 1: gleno-humeral abduction 90° without pain, 100° gave pain in area 1, 2, 4. Going back to 90° of abduction and adding supination: full range, no pain. Adding wrist extension, 30°, no pain. Adding finger extension, very restricted, no pain. Adding lateral rotation in the gleno-humeral joint pain in areas 1, 2, 4, at 10° lateral rotation. Eliminating gleno-humeral lateral rotation, no pain. Adding elbow extension to 50° gave strong pain in area 1, 2, 4 and a strong stretching feeling in the anterior part of the elbow.

Treatment: mobilising the left ULNP 1 in individual components with grade II- without pain, 10 minutes
Re-assessment: active gleno-humeral joint movements now were without pain. Flexion 170° and abduction was full (similar to the right side), but with evasive movements into abduction with flexion and medial rotation with abduction.

Treatment: left ULNP 1 was repeated for 10 minutes, then the individual components of ULNP 1 were reversed in their sequence and an elbow extension of 110° without pain, was achieved.
Re-assessment: cervical active movements and gleno-humeral active movements were the same, but subjectively more comfortable for the patient.

Treatment: the reversed components of the left ULNP 1 were accumulated to 110° elbow extension with 50° of gleno-humeral joint abduction and full lateral rotation, without pain. This position was held and the patient performed lumbar and thoracic rotation movements to the left with right SLR "swinging" 20 times.
Re-assessment: the cervical active movements improved in all directions without any pain. Very gentle oscillatory overpressure movements did not hurt and the resistance at limit felt less rigid. The active left gleno-humeral joint movements seemed to be less laborious and there was no pain response at limit of the active range. The antalgic posture of elevation of the left shoulder girdle had disappeared. The acromio-clavicular joint was less warm and the swelling reduced by 70%.
Right ULNP 1 had lost the strong stretching sensation, but the range of all components was the same.
Left ULNP1 had increased mobility to a 100° abduction without pain and a 150° elbow extension without pain.

Teaching the home programme:
1. Ensuring the exactness of the SLR, swing grade III in the doorway without evasive movements of the pelvis and with active stabilisation of the trunk whilst keeping the chin "tucked" in.
2. Practising the right and left SLR in lying with the help of a towel and stabilising with the trunk muscles.



(Fig. 16) *Right SLR mobilisation with the help of a bath towel. Active trunk stabilisation whilst countering any evasive movement into flexion of the lumbar spine and of the contra -lateral hip and knee (patient with a left hemiplegia)*

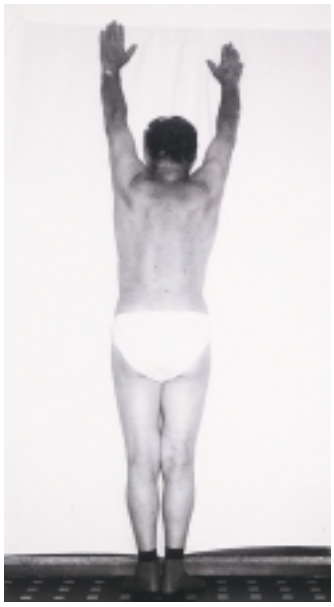
3. Practising the right SLR supine with lumbar rotation to the left, adding dorsi-flexion of the right foot.
4. Left ULNP 1 position against the wall and stabilising the trunk actively. To prevent lumbar hyperextension one leg was put unto a stool with hip and knee flexion 90° (see Fig. 5); rhythmical cervical lateral flexion away from the left ULNP position (see Fig. 4.).
5. Practising the selective activity of the abdominal muscles and adding in bilateral SLR (BSLR) whilst pressing the heels firmly together to enhance abdominal activity.

Warning: the patient was asked to give a detailed report about any pain response to these manoeuvres.

Follow- up treatments:
Every second day, a total of 7 sessions. Treatment was as described above with varying emphasis on target tissue (e.g. left 1st rib: left shoulder girdle complex; cervical zygapophyseal joints) and neural tis-

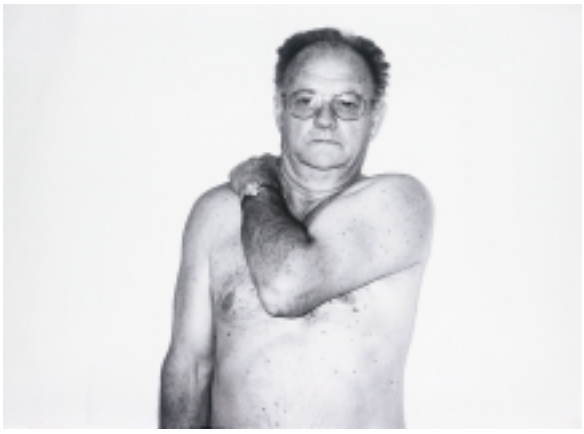
sues. Neural tissue mobilisation was progressed; the home programme was increased with an emphasis on postural correction whilst sitting in the car or watching television and working in the workshop. Correction of evasive movements by activating selective trunk muscles when carrying loads was emphasised.

After four weeks without treatment:
1 re-assessment and a treatment session: The patient's only complaint was that he was sometimes woken at night when he was lying on his left shoulder with his neck in flexion.
Positioning of his neck on a pillow at night was demonstrated and a folded towel put, under his upper thorax when lying on his left side in order to minimise shoulder compression.
The treatment of all neural and target tissues could be done at limit grade IV without reproducing pain.
The active movements of the left gleno-humeral joint were now equal to the other side.

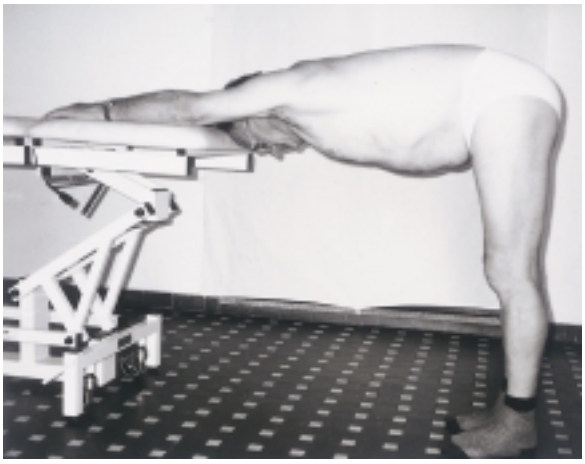


(Fig. 17) *Active test: left G/H flexion after 9 treatment sessions and daily auto mobilisation of Neural tissues and active exercises of the abdominal muscles by the patient.*

(Fig. 18) *Active test "hand on back" after 9 treatment sessions.*



(Fig. 19) *Horizontal adduction of the left shoulder girdle after 9 treatment sessions.*



(Fig. 20) *Home programme with both legs in 90° hip flexion, knee extension and 90° dorsi flexion of both the hint foot. The trunk is mobilised into thoracic extension while the arms rest with elbow extension on the table. Antero-posterior mobilisation of the cervical spine, correcting upper cervical extension and lower cervical flexion is then initiated as an auto-mobilisation at the limit of thoracic extension.*

The SLR plus DF at the limit of range reached 80° hip flexion without pain. The patient worked full time and suffered no pain while working, felt much more mobile than a few years ago and did not suffer under any night pain in both legs. After 6 months the patient phoned and said he feels o.k. and did not have any night pain. He thought this is due to the SLR mobilisation right with lumber rotation to the left and building up trunk muscle activity.

Part 2
CLINICAL OBSERVATIONS REGARDING NEURAL PATHO-DYNAMIC FEATURES IN THE ASSESMENT AND TREATMENT OF PATIENTS WHO HAVE SUFFERED A LESION OF THEIR CENTRAL NERVOUS SYSTEM
It is the purpose of the following remarks to make physiotherapists aware that testing and treating the neurodynamic problems of patients with a lesion of their central nervous system requires different handling to those with other neuro-musculo-skeletal problems, such as the example described above.

Physiotherapists can rely on patients with neuro-muscular-skeletal problems to report their symptoms in detail. The physiotherapist can detect resistance in passive movements and its behaviour through the test movements; evaluate changes in protective muscle tone, analyse antalgic postures and evasive movements and can define the quality of muscle activity as well as the autonomic response.

Re-assessing meticulously gives the therapist guidance on how accurately the problem-solving process has been as well as the effectiveness of treatment. Non-verbal and verbal communication is available, although it needs to be interpreted carefully. And the psychological reactions in individuals appear, in most cases, in proportion to their suffering and disability and to their efforts to maintain a normal life.

With patients, who have been affected by a central nervous system lesion the physiotherapist is faced with completely different input, output and feed back process on all levels of their physical, psychological and social state. It is obvious that the physiotherapists have therefore to modify their physical, psychological, social and communicative approach. Also the interpretation of symptoms and signs, the goals set for treatment in relation to the extent of a central lesion and the approach to the partners and family of the patient may also be difficult.
Over the last 50 years a large amount of scientific articles and clinical studies have been published in this field for those involved in the assessment and treatment of patients who have suffered a lesion of their central nervous system. More recent literature has become available that stresses the importance of assessing the neurodynamic state of each patient and integrating these findings into the assessment and treatment at all stages of the rehabilitation process (Davies, 1994, 2000; McKibbin, 1995; Consensus Report, 1998).

The sensory system, which initiates "feed forward" and "feed back" for body functions, and especially postural and selective muscle activity, have completely changed and are somehow "distorted" for the patient. Muscle function therefore no longer protects the nervous system or guarantees neural mobility. The nervous system loses its control over the target tissues and these in turn lose their ability to co-ordinate with other target tissues and with the nervous system. The vicious circle of biomechanical and physiological pathology in neural and target tissues is powerfully facilitated. In addition memory and learning as well as verbal and non-verbal communication abilities change and tactile- kinaesthetic modalities have been to some extend disengaged. The patient with a central lesion has, so to speak, lost his physical world.
Whether muscle tone is high, provoking muscle hyper-activity or muscle spasm or whether the muscle tone is low, resulting in muscle hypo-activity the nervous system as a plastic, continuous tissue tract appears to loose a great deal of mobility (i.e. elongation and transverse gliding at the interface) to varying extents, in different body areas, pulling the whole body out of alignment.

In the case of hemiplegia a SLR test (without dorsiflexion) of the so- called "better side" may be far less mobile than of the more affected side. When determining the point of onset of neural tissue resistance during this test the trunk may rotate backward on the more effected side. At the same time elevation of the shoulder girdle and excessive gleno-humeral subluxation (on the more affected side) can be seen with the cervical spine becoming laterally flexed to that side. The whole arm and hand with the fingers can be observed to move into "associated reactions" (evasive movements).
Likewise a ULNP test movement of the "better side" can provoke evasive movement of the trunk, of the leg and foot, of the arm and hand of the more affected side. Often extreme upper cervical extension occurs at the same time.
When investigating the upper cervical spine it is felt to be almost fixed in hyperextension, combined with sub-occipital tissue "thickening". All sub-occipital and neck muscles show marked hyper- tonicity and loss of elasticity. This situation has to be reversed urgently to create a more mobile spine with free movement of nerve roots, plexi and peripheral nerves in addition to re-educating selective muscle activity using target and neural tissue mobilisation. If this is not undertaken, poor balance reactions will be perpetuated and contractures will develop. In addition the face and the oral tract will suffer permanent damage and the shoulder girdle and all joints of the upper limb will become more and more contracted. This in itself distorts sensory feedback and makes the recovery of active selective muscle function much more difficult. Without such corrections normal alignment of the head and neck, and of the trunk, and pelvis and lower limbs likewise will not be possible, especially when the vertebral column and neuraxis are subjected to less and less normal movement.

" It seems that the measured mobility of the M.Trapezius descendens stands in direct relationship with the mobility of neural structures" (Dale E, Jull G, Sutton S,1997). This study was done with asymptomatic subjects and supports the clinical observation that target tissues are only as good as their nerve supply. Therefore continuous assessment and re-assessment, mobilising all neural and target tissues guarantees the best prophylaxis against adaptive shortening of target tissues when neural tissues guarantees their normal biomechanical characteristics following a lesion of the central nervous system. To maintain the mobility of the nervous system and all its target tissues is prophylaxis against soft tissue and joint contractures – that once allowed to develop – cause great discomfort and pain for the patient and prolong markedly the rehabilitation process.

Devising a home programme with tissue self-mobilisation demands regular re-assessment of all target

and neural tissues for the rest of the patients' life. This will assure optimal physical mobility and facilitate the recovery of muscle function. The progressive recovery of muscle function is the only way to further improve altered neurodynamics. Therefore the reactivation and re-education of selective muscle function against patho-mechanical resistance of any target and neural tissues should be undertaken as early as possible. "In order for the nervous system to maintain its functional integrity the target tissues must in turn, enable it to spread out and adapt to any position and body movement" (Butler 1991), (see Fig. 5).

Neurodynamic testing and treatment as well as joint mobilisation should be performed very slowly and deliberately, searching for the beginning of muscle spasm or inert tissue resistance, avoiding any pain or discomfort and stopping when the onset of spasm is reached and when any evasive movement-reactions and muscle tone changes begin (Rolf, 1997). Attempting to override resistance associated with neural tissue sensitivity (which is often accompanied by protective muscle spasm in the absence of pain) with accumulated neural test components only provokes discomfort afterwards and consequently makes muscle tone rise. Forceful techniques to neural tissues of the limbs and especially to the neuraxis are contraindicated. Furthermore, the evasive movement reactions are likely to increase when passive movement ignores and moves into spasm and inert tissue resistances, aggravating neural and target tissue reactions.

In this situation the nervous system is only "shifted" (pushed from one evasive movement into another) rather than be mobilised/elongated. Any successful rhythmical neurodynamic mobilisation with an adequate grade should result in a better ability of the neural tissues to elongate and the peripheral nerves should move more easily transversely at their mechanical interface. However, the treatment of neural tissues in patients who have suffered a central lesion of their nervous system needs to be performed towards the limit of range of movement of neural tissues in order to enhance full normal elongation.

Because of the distorted sensory modalities, which occur in any central lesion, pain variations in response to movement, as well as activities and time are very difficult if not impossible for the patient to assess. Therefore the physiotherapist cannot, in most cases, rely on the patient as a witness of the detailed behaviour of their pain.

It is therefore also essential for the physiotherapist to give, with the flat and comfortable hand sensory input (2–3 seconds) by way of intermittent gentle pressure on the contra- lateral trunk and limb. This facilitates sensory awareness before and during neurodynamic or joint testing and treatment. This intermittent sensory input is repeated after 30 seconds when the sensory stimulus begins to fade away. In this way tactile-kinaesthetic communication can be stimulated throughout the less affected side as well as to the other side of the body. The importance of sensory stimulation while treating a patient with a lesion of the central nervous system should not be underestimated. *"The motor disturbances are aggravated by sensory impairment. Patients with sensory deficit lack the urge to move and do not know how to move limbs or segments of limbs which they do not feel properly"* (B. Bobath, 1990).

There have been various ways discussed and developed how to stimulate and facilitate active muscle function in central lesions. However the means of sensory input in order to improve tactile- kinaesthetic communication which is essential for selective muscle work, has received little attention and discussion. One system, the Affolter concept, offers a way to improve *"perceptional modalities"* (Affolter 1980) by actively guiding the patient physically with any particular activity, emphasising sensory input. Readers may be interested to explore this method, which is the approach used by the author.

In most patients with a lesion of the central nervous system each component of neurodynamic test procedure has to be freed in some way, especially after a long period of immobility. The vertebral column looses mobility at almost all its levels and in all directions and therefore target tissues (e.g. the vertebral joint structure, the costo-transverse joints, proximal and distal limb joint complexes) lack their normal ranges of passive physiological and passive accessory motion. All muscular and connective tissues feel hard and inelastic when palpated. Adding the neuraxis as a sensitising movement (spinal flexion) to neurodynamic testing reveals marked limitation of the elongation of neural tissues and restrictions in adapting to normal movement reactions. Therefore, in many cases, the patient when he wants to move actively or change position has continuously to struggle against resistance.

The normal transverse gliding movement of peripheral nerves when adapting to tensile forces during postural changes or movements appear also to become very restricted. The peripheral nerves frequently are felt like immobile plastic tubes within their interfacing tissues. It is possible to imagine that the nervous system suffers an abnormally high proportion of compensatory intra- neural movement because the extra-neural movements are extremely restricted. This can provoke extreme autonomic reactions when for example positioning the patient, such as waves of sweating, difficulties in breathing as well as diffuse physical discomfort. Sadly in many patients a complete loss of extra- and intra-neural mobility can be found when contractures have been allowed to develop.

If a single neurodynamic test component has to be mobilised, localised techniques of mobilising the peripheral nerve, the plexus and the nerve roots with accessory movements are clinically beneficial:

e.g. transverse mobilisation against the mechanical interface of the lower part of the brachial plexus in order to achieve more gleno-humeral flexion, or rolling the peripheral nerve with gentle compression in its nerve bed, e.g. the median nerve at the anterior aspect of the elbow in order to achieve more elbow extension.

(Fig. 21a) *Hemiplegia (left): "roll-over" technique for a peripheral nerve in its nerve bed, accessory movement to neural tissue, the peroneal nerve in this case.*

(Fig. 21b) *Localised passive mobilisation of the peroneal nerve with accessory mobilisation technique of "roll-over" releases the protective spasm of the toe flexors (resistance related to neural tissue sensitivity) on the more affected side and improves sensory awareness at the same time.*

Another example is moving at the mechanical interface with a transverse or/and rotatory tissue technique against the nerve: e.g. moving the adductor muscles against the obturator nerve (interface mobilisation against the nerve) in order to reduce muscle tone and to achieve more hip abduction.

The restricted movement should then be slowly added in without encountering pain or resistance. The accessory mobilisation techniques for neural tissue are especially indicated in patients with severe pain syndromes such as SMP/*sympathetically maintained pain* (McMahon, 1991; Melzack 1991; Davies 1994; CRPS Consensus Report, 1998). Pain and the autonomic signs like swelling, sweating as well as feelings of hot-cold, heaviness, electrical shocks and other strange sensations are also reduced. At the same time the sensory awareness of that part of the limb or even of the whole limb can improve rapidly.

If movement of one of the neurodynamic test components has been restored another can be introduced. Again only to the point of onset of resistance which often is identical with spasm onset and always without pain. It is clinically impressive when the whole of a neurodynamic test sequence in a limb is gradually restored. With patients who have a great deal of pain it is advisable to introduce passive physiological movements of neurodynamic test components only when approximately 50% of the normal range of each test component has been achieved. But active stabilisation without pain is encouraged at a much earlier stage ensuring rhythmical active muscle work without evasive movements.

After local accessory neural mobilisation an immediate active muscle contraction, for instant of the peroneal muscles (see Fig.21B) or of the wrist and finger extensors, is possible. This activation should then be rhythmically repeated interspersed with the same neural mobilisation techniques of accessory movements along the peripheral nerve that supplies these muscles. Should the regained muscle activity become more accentuated with this treatment and show a better functional quality then other neurodynamic test components can be very slowly introduced continuing accessory mobilisation to the respective nerve.

Any neural, articular, connective tissue and muscle resistance (e.g. increased muscle tone) will counteract the regained selective muscle function in central lesions. Therefore the physiotherapist has to restore pain- free range of movement in all target tissues also. The mobilisation techniques used for joints need to be adapted to the sensory disturbance of a central lesion and be slow, never staccato-like at the limit of the available range, avoiding painful contact pressure from the therapist's thumb tips and avoiding pain and discomfort. It is important to add intermittent sensory stimulation.

Normalising muscle hyper- activity on the so-called "better side" is another important aspect in the rehabilitation of patients with a central lesion. It is possible that gentle neural mobilisation with SLR, PKB (prone knee bend), ULNP (grades of II and III-) with all the sensitising movements progressively added with intermittent sensory stimulation can reduce hyper-activity spontaneously.

Because patients with a central lesion of their nervous system in most of the cases tend to sit behind the transverse axis of their hips and have great difficulties in bringing their trunk and weight forwards in sitting as well as in standing and walking, the vertebral column suffers progressive stiffening. Therefore the lumbar, thoracic and cervical neuraxis should always be included in the mobilisation of neural tissues. For example positioning the vertebral column in rotation and lateral flexion to either side, mobilising into flexion and extension as well as in combined physiological movement are useful measures.

However intermittent sensory input, rhythmical active movements and the correction of evasive movements are essential also to reduce the hyperactivity of the less affected side, which so often provokes hypertonicity of the more affected side.

Frequently the shoulder complex (gleno-humeral and acromio-clavicular joints as well as the scapulo-thoracic structures and the ribs 1-5 included) of the more affected side shows extensive loss of mobility. A marked reduction of neural tissue mobility and a lack of any muscle activity in the arm and hand are, sadly, often seen in patients with a central lesion of their nervous system. The complete loss of gleno-humeral lateral rotation and of passive accessory movements predisposes the shoulder to rotator cuff and nerve lesions if the affected arm is not positioned and handled with great care. Many authors have commented in case studies on the important clinical aspects of treatment and some scientific studies have been published on the subject (Basmajian, 1979; Calliet, 1980; Smith et al.,1982; Roper, 1982; Najenson et al. 1971; Davies 1985, 1990, 2000). Pain- free mobility has to be restored and maintained in all neural and target tissues of the shoulder complex, of the trunk and rib cage, of the arm and hand in these cases. It is laborious, but essential work to slowly alter their patho-dynamics in order to facilitate the recovery of muscular activity.

It is particularly difficult to restore passive and active lateral rotation of the gleno-humeral joint. This movement encourages thoracic extension, stimulates trunk activity, scapula thoracic movements and seems to contribute to neural tissue mobility of the brachial plexus. Firstly the gleno-humeral joint needs to be mobilised with accessory movements at the limit of the available range of lateral rotation. This should be done without pain, in a fully adducted position and with mobilising techniques that use the whole palm of the hand of the physiotherapist rather than the thumbs alone, thus avoiding painful local pressure. In most cases the A/C joint, the ribs 1-5, the clavicle and the S/C joint as well as the scapula/ thoracic tissues need slow rhythmical mobilisation near the limit of their range and intermittent sensory stimulation.

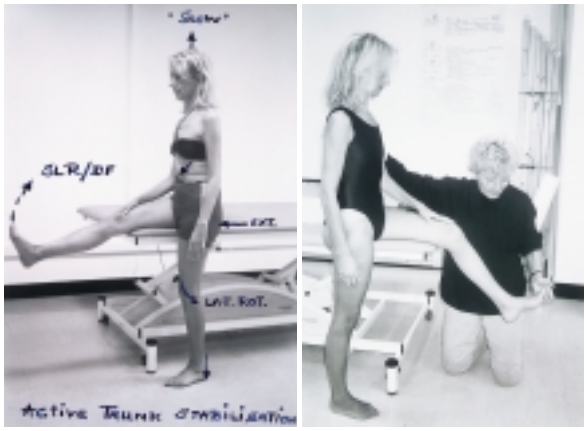
At the same time intermittent sensory input on the contra-lateral side should be carried out. To achieve full lateral rotation of the gleno-humeral joint the cervical nerve roots, the brachial plexus and the peripheral nerves should be mobilised with accessory movements, adding in shoulder depression, elevation, protraction, retraction, as well as all cervical physiological movements and components of ULNP movements.

Selective trunk activity with the emphasis of pulling the rib cage down to the umbilicus is then initiated with the shoulder held by the physiotherapist in depression and adduction at the limit of the obtained lateral rotation of the gleno-humeral joint. SLR and PKB positioning can be added in at any stage of the treatment. Should any muscle activity occur in the arm while this treatment procedure is being carried out (e.g. activity of the muscles deltoid, biceps, triceps, rotator cuff muscles) it is encouraged by accessory mobilisation to the respective nerve.
In patients with central lesions, the mobilisation of the neuraxis is an enormously important aspect of treatment. It is possible that, in long sitting with the trunk actively stabilised then mobilising into more hip flexion, and adding in abduction of the legs can provoke spontaneously muscle activity in the hand of the more effected side. Long sitting with and without abduction, mobilising the trunk into maximum flexion with large and rhythmical amplitudes grade III without pain and into the slump position and adding thoracic rotation away from the more effected side can also provide spontaneous selective arm, hand and finger activity.
At some stage the ULNP on the more effected side will become passively free without pain. This will aid the recovery of active shoulder, arm and hand function.

With respect to the prognosis for the patient with a central lesion all tissue mobility has to be restored to an optimal level to encourage muscle activity and in order to maintain mobility of all target and neural tissues. Therefore a home programme should be established at a very early stage of the treatment working on specific areas where the patient is most affected. The physiotherapist can then re-evaluate specific aspects of the home programme over time, teach and encourage rhythmical self-mobilisation, correct evasive movements and postures, and teach how to avoid hyper- activity. It is important to make sure that the patient can do *the home programme* himself without the help of another person.

(Fig. 22) *Patient with left hemiplegia. Home programme: the patient cannot yet avoid pelvic and lumbar evasive movements and some knee flexion with mobilising the left SLR (hands clasped under her left knee)*

(Fig. 23) *Patient with left hemiplegia. Home programme: the patient can actively stabilise the left trunk retraction by stabilising the trunk muscles and avoiding lumbar and pelvic flexion.*



(Fig. 24a & 24b) *Left hemiplegia: home programme: the patient with a left hemiplegia has learned to brace the muscles of her trunk; antero-posterior mobilisation of the cervical spine/neuraxis with the trunk stabilised, is carried out without evasive movements. At the same time the SLR position at limit of range on the less affected side is maintained; the left hip of the more affected side is corrected actively into more extension, avoiding the evasive movements of flexion and medial rotation of the left hip and lumbar extension. The photo is marked for the patient's home programme.*

Correct postures need also to be taught to every patient with a central lesion. It seems to be of help for the patient if such postures, self-mobilisation techniques and selective muscle activities are documented with photographs which are clearly marked (e.g.Fig.24 a). He can use these to be sure that the home programme he performs is exact.

The speed and rhythm of neural and target tissue auto-mobilisation and active muscle work sequences require a long time for the patient to learn and get right. However, this is a very necessary aspect of the treatment because these individuals have lost their rhythm of movement and are always forced to try to move against the resistance of their neural and target tissues. Therefore activity in groups or developing like golf and skiing/"Langlauf" should also be encouraged (Consensus Report, 1998; Gerber, 1995; Rasmussen, 1995; Malström, Johansson, Sallnäs, 1995; Davies, 2000). The more his sensory awareness will improve the more he will initiate movement. The practise of a home programme is often the beginning of the adaptation of the patient to his future life with a central nervous system lesion.

Great care should be taken right from the beginning to teach the *activities of daily life without evasive movements* since a patient with a lesion of his central nervous system will spontaneously perform movements with evasive movements (associated reactions). Moving from lying to sitting and from sitting to lying, getting up from sitting to standing, sitting down, dressing and undressing – as well as other daily and often repeated activities - should be carefully analysed. Abnormal resistance against movements and positional changes in target and neural tissues, hyper- activity and reduced sensory feedback provoke evasive movements. These in themselves give the wrong information to the patient and can facilitate further patho-mechanical and patho-physiological changes in neural and target tissues.

The spontaneous activities of daily life offer an excellent chance for analysis by the physiotherapist as to why, where, how and when movements go wrong and which kind of therapeutic intervention is therefore needed. A carefully devised home programme has little result as regard to neural and target tissue mobility and selective muscle function if the activities of daily life are performed with massive evasive movements and hyper-activity of the so-called better side.

There are many different ways to dress for instance, but in whichever way it is taught and performed it must not provoke any evasive movement or hyperactivity. With the relearning of daily movement sequences the patient needs intensive guiding in the beginning, as practised in the Affolter concept (Affolter, Stricker (eds) 1980). Also sensory input to the contra-lateral side should always be applied to ensure tactile-kinaesthetic awareness. The patient should be enabled to perform an activity without any help and recognize immediately if a movement goes wrong whilst he moves. He should then be able to eliminate all evasive movements. Once patients understand why and how they have to perform activities of their daily living in certain ways to achieve improvement and avoid adaptive shortening of tissues (e.g. Achilles tendon), they usually try conscientiously to perform the postural and movement sequences with the best possible quality.

Fig. 25a. *Dressing with evasive movements and hyperactivity.*

Fig. 25b. *Home programme: dressing without evasive movements and no hyperactivity.*

The home programme should be re-evaluated from time to time. So too should the detailed neurodynamics and condition of the target tissues and the activities of daily living in order to avoid any secondary changes such as adaptive shortening of tissues (e.g. loss of shoulder movement mobility, loss of Achilles tendon elasticity) that so easily can develop in these patients.

Patients who are mentally or physically not capable of performing a daily home programme or of initiating selective muscle activities of their daily life, will require someone to be taught how to help them maintain their mobility, prevent contractures and improve muscle activity.

CONCLUSION

This article has stressed the importance of the assessment and treatment of disordered neurodynamics and the associated changes in the muscular system, both in neuro-orthopaedic patients and those with central lesion disorders has been emphasised. The home programme to maintain and progress the improvements gained is an essential aspect of physiotherapy in these cases. Intermittent sensory stimulation needs to become an important aspect in the treatment of patients with a central lesion disorder.

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