Sophie DESROCHERS
Guesteditor

e-News for Somatosensory Rehabilitation

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ORIGINAL

GUESTEDITORIAL
CRPS: un résumé de l'approche de rééducation sensitive et des résultats qui peuvent être obtenus

To MD To neuroscientist To patient To therapist

Sophie DESROCHERS, OT[1]

Au cours des dernières années plusieurs changements sont survenus au niveau de l'appellation du syndrome douloureux régional complexe (CRPS) et au niveau de son traitement. De nos jours, les diagnostics sont plus précoces et les patients ont accès à un choix plus diversifié de thérapies. Toutefois, malgré l'évolution au niveau des traitements possibles, certains référents demeurent hésitants en ce qui à trait à la reconnaissance de ce syndrome, car il demeure pour plusieurs, difficile à traiter.

Jusqu'à ce jour, malgré les recherches, les publications et le développement de nouvelles approches thérapeutiques, il demeure difficile pour le thérapeute de faire face au défi de traiter une personne souffrant d'un CRPS. Au cours des 15 dernières années au Québec, plusieurs cliniques de la douleur ont vu le jour et sont devenue « spécialistes de la prise en charge» des personnes atteintes de CRPS. Toutefois, malgré leurs interventions, plusieurs de ces personnes demeurent avec des douleurs persistantes et des difficultés fonctionnelles suite aux traitements et c'est souvent dans ce contexte qu'ils sont reçus dans notre clinique d'ergothérapie.

Auparavant, mon approche avec cette clientèle était orientée vers la reprise de la fonction en favorisant un contrôle de la douleur avec différentes techniques antalgiques locales, l'utilisation d'aides techniques, de principes d'hygiène posturale et de protection articulaire ainsi que l'adoption d'un programme de conditionnement physique adapté à la personne (fait conjointement avec un kinésiologue dans ce cas).

Il y a deux ans, suite à la formation de rééducation sensitive reçue en sol canadien, j'ai mis en application, dès le lundi suivant, ce nouvel outil d'évaluation et de traitement dans ma pratique. Les succès ont été au rendez-vous et je peux désormais aider certaines personnes (avec lesquelles je n'aurais pas eu autant de succès) atteintes de ce syndrome à diminuer de façon significative leur douleur et améliorer leur fonction. Toutefois, certaines difficultés persistent, nos référents connaissent encore peu l'approche et il demeure difficile pour le moment de diffuser nos résultats, étant donné le nombre limité de gens formés au Québec (*n*=56 thérapeutes[2]).

J'ai donc profité de l'occasion qui m'a été offerte, soit par la revue des publications des 28 volumes du *e-News for Somatosensory Rehabilitation* sur le CRPS, afin de choisir celles qui selon moi offriront un vue d'ensemble concernant le phénomène du CRPS, l'approche de

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[2] Note de la rédaction

rééducation sensitive et les résultats obtenus lors de l'application de celle-ci. Je souhaite que ce volume constitue une référence pour tous ceux qui désirent améliorer l'impact de leur thérapie et offrir à leurs collègues en réadaptation un résumé de l'approche de rééducation sensitive et des résultats pouvant être obtenus.

Les articles choisis touchent, entre autres, différents thèmes tels que la recherche, le phénomène de la douleur, la neuroplasticité, les résultats obtenus avec l'utilisation de la méthode de rééducation sensitive et les témoignages des gens atteints de CPRS suite à l'utilisation de cette méthode. Bref, un ensemble d'articles qui m'ont permis d'apprendre davantage sur cette atteinte particulière et d'intervenir plus efficacement.

J'espère que les lecteurs, autant novices qu'avancés, en interventions auprès des CRPS et au niveau de l'approche de rééducation sensitive seront interpellés par ce volume spécial et qu'ils seront motivés tout comme nous à poursuivre leur implication auprès de cette clientèle et favoriser la diffusion d'information auprès de nos collègues.

C R P S

COMPLEX REGIONAL PAIN SYNDROME

Volume Spécial e-News 2004 - 2011

Special Issue

Choosen by Sophie DESROCHERS from MONTREAL

e-News 2004; 1(2)

GUESTEDITORIAL
Neuroplasticity

To MD To neuroscientist To patient To therapist

Eric M. ROUILLER, PhD[3]

Although neuroplasticity was for a long time thought to be reserved to immature central nervous system, it is now widely accepted that it occurs also at adult stage. Two main events are known to induce neuroplasticity in the central nervous system. First, learning was shown to induce changes of cortical maps, such as an increase of the representation of a given finger in the primary somatosensory cortex (S1) after sustained tactile stimulation applied to this finger (e.g. Recanzone et al., 1992). Along the same line, repetitive discrimination of a tone of a given frequency results in an increase of the representation of this frequency in the tonotopic map in the primary auditory cortex (A1; Recanzone et al., 1993). The increase of representation for a finger in S1 or a tone frequency in A1 occurs at the expense of neighbouring body territories or frequency domains, either unstimulated or behaviourally less relevant. Second, a peripheral lesion will modify the central representation. This has been demonstrated for instance in S1 as a result of finger or hand amputation (Merzenich et al., 1984; Florence and Kaas, 1995) as well as of peripheral nerve injury (Merzenich et al., 1983; Garraghty and Kaas, 1991; Pons et al., 1991; Florence et al., 1994) or spinal cord injury interrupting the dorsal column (Jain et al., 1997). Similarly, a lesion restricted to a small portion of the cochlea will modify the tonotopic representation of the contralateral A1 (Robertson and Irvine, 1989; Irvine and Rajan, 1997). In general, the representation of the corresponding lesioned body territory or frequency domain in the cochlea becomes absent or under represented, a cortical area invaded by adjacent body territories or frequency domains. Transmodal neuroplasticity has also been observed, such as the well known case of blindhuman subjects exhibiting an activation of the visual cortex elicited by Braille reading (e.g. Cohen et al., 1997, 1999; Sadato et al., 1996, 1998, 2002). In such a case, one may wonder whether the re-organization of the visual cortical areas in order to be responsive to tactile stimulation (Braille reading) derives from de-afferentation of the visual areas due to blindness and/or to learning of the Braille reading, thus emphasizing the cutaneous inputs, as compared to a normal (non-Braille reader) subject. To address this crucial question, Sadato and collaborators (2004) studied with fMRI the foci of cortical activity elicited by the discrimination of two distinct Braille characters. The originality of this study was to compare the two following groups of subjects: i) two recently blind subjects naive to Braille; ii) nineteen sighted control human subjects, also naive to Braille. In relation to the tactile discrimination, the authors observed in the two blind subjects an activation in associativevisual areas (Brodmann areas 37 and 19) that was absent in the control (sighted) subjects, although they performed the same task with a comparable behavioural score. In conclusion, taking advantage of the rare chance to investigate blind subjects naive to Braille,

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these data strongly support the notion that this neuroplasticity is not learning dependent, but is more likely to rely on de-afferentation. In other words, as claimed by the authors (Sadato et al., 2004), visual and tactile inputs are competitively balanced in the occipital cortex. As a result, in relation to the execution a demanding tactile task, de-afferented associative visual areas are recruited in blind subjects, but not in sighted ones. Sensory influences would thus play a more prominent role than learning influences (Sadato et al., 2004).

In the context of neuroplasticity of the cerebral cortex, two further issues deserve further comments. First, does the cortical neuroplasticity take its origin purely in the cerebral cortex? There is now evidence that similar changes also take place subcortically, for instance in the thalamus (Garraghty and Kaas, 1991) or even in the brainstem, suggesting that the changes observed cortically may derive, at least in part, from a plasticity already present more peripherally (see for review Jain et al., 1998). Second, what is the time course of such plastic changes? There is evidence that neuroplasticity spreads on a long period of time, starting by very early (immediate) changes occurring a few minutes to hours post-lesion (Sanes et al., 1988; Jain et al., 1998), followed by modifications taking place in the short term (days to weeks) as well as in the long term (months to years). It is thus clear that the observations of neuroplastic events following a lesion may be highly variable depending on the precise time point at which the measurements were performed. Along this line, performing a dynamic observation of cortical representation of the hand in the primary motor cortex, Schmidlin et al. (2004) demonstrated a disappearance of the hand representation in M1 as a result of cervical hemisection, followed by a progressive, though incomplete, re-appearance of the lost territory during a few (4-5) weeks post-lesion, following a time course paralleling the behavioural recovery. Finally, what are the mechanisms underlying neuroplasticity? Although they are not yet fully elucidated, several possible mechanisms have been proposed (see for review Jain et al., 1998). First, a peripheral lesion may cause a dis-inhibition of suppressed inputs. In the normal situation, there are latent inputs suppressed by inhibitory interneurons, themselves activated by the excitatory inputs. The release of the inhibition thus results from a lack of excitatory inputs due to the lesion. This interpretation is consistent with a decrease of GABA immunostaining in the cerebral cortex after peripheral nerve injury (Garraghty et al. 1991). Second, one may also consider a synaptic mechanism of potentiation of inputs ineffective in the normal situation. Finally, the above functional adaptations are accompanied by morphological changes, such as regeneration and/or collateral sprouting of axons, although such events are limited unless they are enhanced by neurotrophic factors or by manipulating the environment in order to stimulate the growth of axons for instance (see e.g. Schwab, 2002, 2004).

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FORUM DE REEDUCATION SENSITIVE

http://nte.unifr.ch/moodle/mod/forum/view.php?f=1053

To MD To neuroscientist To patient To therapist

Voici un exemple de question avec sa réponse que vous pouvez trouver sur le forum de rééducation sensitive du Centre de Nouvelle Technologie et Enseignement de l'université de Fribourg (Webmaster : Isabelle QUINTAL, BSc erg.).

Question :

Bonjour,

J'ai une jeune patiente de 17 ans en traitement pour un problème au niveau du genou. Lésions ligamentaires du genou G à 8 ans puis à 12 ans. Elle ressent des gènes fonctionelles dès ce moment, arrête la danse, tennis et snowboard. En juin 2004, arthroscopie : RAS; mais elle ressent des douleurs très fortes en post-op. Quelques séances physio avec notamment drainage sur un genou très oedémateux et inflammatoire.

Depuis, elle ne parvient plus à l'étendre ni le fléchir complètement, décharge le MIG et ne peux marcher que peu longtemps.

Elle consulte un autre médecin fin août 05 qui prescrit l'ergo. Scintigraphie et IRM sp.

A l'évaluation, toute la musculature postérieures G (cuisse, fesse, lombaire, dos, nuque) est hypertendue et douloureuse (déficit fonctionnel mm).

Esthésiographie pas très nette (toucher différent). Site d'irradiations provoquées au Vibralgic sur le nerf saphène (interne) à hauteur du haut de la rotule (irradiations suivant le trajet du rameau infra-patellaire et n. saphène). Le toucher, d'abord qualifié de désagréable, montre finalement une allodynie sur une zone allant de 2 cm au-dessus de la rotule jusqu'à mi-jambe, plutôt interne.

QDSA : chaleur (0), fourmillements (0), en éclairs (4), rayonnante (2) et étirement (2) derrière le genou.

Les douleurs apparaissent à l'activité, la mobilisation et sont plutôt vesperales et nocturnes (avec réveils).

L'aspect est cliniquement sans particularités, mais elle évoque des fluctuations au niveau vasomoteur (température) et oedème (ce qui nous donnerait un diag CRPS positif selon Bruehl).

L'hypothèse est donc un CRPS II, depuis plus d'un an, peu floride, qui n'aurait pas été diagnostiqué en tant que tel jusqu'ici; avec troubles musculo-fonctionnels secondaires. A noter qu'en plus des gènes et douleurs, elle est passablement déprimée par ce problème.

L'annonce des examens paracliniques normaux a été un coup dur, comme lui ôtant la justification médicale de ses problèmes/douleurs.

J'ai commencé un traitement de l'allodynie et demandé du Neurodol à son médecin.

Questions :

- Selon votre expérience, quelle médication serait utile à ce stade (après plus d'un an) ?

Quid des AD tricycliques (douleurs de fond)?

Elle a pris Voltarène et Tramal, mais l'aide est peu efficace.

- Connaissez-vous une pratique utile-pratique-efficace à ce niveau ? Je pense au Stress Loading Program pour la main, le bain tourbillonnant, ...

Merci d'avance !

Nicolas Chabloz
Ergothérapeute
Madeleine 28
1800 Vevey

Réponse :

Concerne: question d'expert

Cher Nicolas,

Liminaire:
La complexité du CRPS II, et de son traitement, augmente le nombre de paramètres dont il faut tenir compte EN MÊME TEMPS. Cette complexité me pousse petit à petit à parler de ""stade "V" de lésions axonales "".

1. Le CRPS II du genou est la 4ème localisation en fréquence du corps humain après la main, le pied et l'épaule. Nous en avons évalué 13 ces 14 derniers mois.

2. Le CRPS II du genou n'est pas floride. Généralement l'oedème a de la place et s'observe difficilement. Ce tableau de causalgie majeure est proche de son tableau cousin: la névralgie crurale.

3. La névralgie crurale est un tableau de 6 éléments somesthésiques à évaluer:

a) la région antérieure de la cuisse:
Branches cutanées antérieures du nerf fémoral

b) la région inféro-latérale de la rotule
Branche infrapatellaire du nerf saphène

c) la région médiale du genou
Branche fémorale médiale du nerf saphène

d) la région antéro-médiale de la jambe
Branches crurales médiales du nerf saphène

e) la région du creux plantaire
Branche terminale du nerf saphène

Le point de l'aine : inguinalgie

4. Dans la situation de ta patiente ces régions sont-elles altérées et alors tu as un CRPS II de la branche crurale médiale du nerf saphène et tu évites les autres territoires du tableau.

Ou tu as des lésions axonales du nerf obturateur (tiers moyen de la face médiale de la cuisse) Mais là sincèrement je n'ai jamais observé un CRPS II de ce nerf.

5. Pour la médication, classiquement le traitement du CRPS est le Miacalcic, le traitement du CRPS II est le Neurontin en petites doses ou le Rivotril. Les tricycliques sont aussi donnés mais dans un 2ème temps.

Bonne chance

Claude SPICHER

En français
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GUESTEDITORIAL
Complex Regional Pain Syndrome: Myth, madness or miscommunication?

To MD To neuroscientist To patient To therapist

Candy McCabe, PhD, RGN[4]

Myth and madness?

A patient enters your clinic room holding their right arm awkwardly, as if it has been paralysed by a stroke. They move cautiously so that their limb cannot be accidently knocked and as they position themselves in the clinic chair they angle their body so that they can ignore the limb as they speak to you. They tell you of the intense burning pain that affects this oedematous, shiny skinned arm, how their sleep is disturbed at night and about the cradle they have made to protect their arm from the weight of the bedclothes. They speak of a loathing of that painful limb, disgust and a strong desire to amputate it. Furthermore, they can draw a line across their arm where this hypothetical amputation should take place so that 'it', their arm, no longer interferes and torments every aspect of their waking lives. The right arm has not moved throughout this conversation though the left has been used in normal gestures. The patient is extremely distressed and exhausted by the pain. You see from their medical records that these symptoms have been present for more than a year after minor trauma and imaging evidence confirms that no peripheral damage remains to explain these ongoing problems. The patient appears to be describing symptoms that are illogical when considered against traditional pain mechanism theories. It is perhaps reasonable to conclude they are either fabricating this story for some ulterior motive or simply mad.

The above scenario describes a typical presentation of Complex Regional Pain Syndrome (CRPS) which is defined by the presence of sensory, motor, and autonomic disturbances, usually in a single limb, that may arise after nerve injury (type II), minor trauma, or spontaneously (type I)[1].The cause is unknown and there is currently no cure. Despite being first described by Weir Mitchell nearly one hundred and fifty years ago, then termed causalgia

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from the Greek ‘causos’ for heat and ‘algos’ for pain[2], the symptoms can still be dismissed as ‘psychosomatic’ by some physicians. The problems in diagnosis lie in the multiple names by which this condition has been known (e.g. Reflex Sympathetic Dystrophy, causalgia, Sudeck’s atrophy, algodystrophy to name but a few), the variability in signs and symptoms across time, the apparently bizarre nature of these symptoms in the absence of objective diagnostic criteria and a variability between individuals in response to therapies. The 1995 International Association for the Study of Pain revised taxonomy[1] aimed to address some of these issues but despite a number of subsequent suggested refinements to improve sensitivity and specificity[3,4] there remains a lack of uniformity across these criteria. Without a diagnostic gold standard for CRPS, confusion for both patients and physicians will continue[5]. It has recently been suggested that these problems could be resolved by applying the proposed revised definition of neuropathic pain, ‘‘pain arising as a direct consequence of a lesion or disease affecting the somatosensory system”[6] and classifying patients into ‘‘definite, probable, or possible” CRPS depending on objective data from their clinical presentation[7]. This seems a sensible route to take and may help to validate CRPS as a genuine condition for those who continue to remain sceptical.

Pain is the cardinal symptom of CRPS and this pain is larger in area and intensity than expected for the original injury. In a study by Birklein and colleagues[8] of 145 patients with CRPS Types 1 or 2, pain was described by over three-quarters of the subjects when their affected limb was at rest. Almost all subjects reported pain when their limb was moved or touched. Pain to light touch or other normally non-noxious stimulation is termed allodynia[9] and animal studies have demonstrated that nerve injuries can produce intense hyperalgesia in skin areas which are adjacent to that served by the damaged nerve and which share nerve trunks, roots or central terminations with the injured nerves[10,11]. This may explain how a relatively minor nerve injury can evoke pain over such a wide area and why partial denervation of the skin, rather than lowering sensation, appears to in fact increase sensitivity. However, Spicher and colleagues[12] have elegantly demonstrated that when minor nerve damage occurs, hypersensitivity, as measured by a standardised assessment for mechanical allodynia, and hypoaesthesia co-exist with the former masking the presence of the latter. This can be confirmed by regular use of distant vibrotactile counter stimulation (DVCS), a tactile and vibratory device, to reduce the area of hypersensitivity which then reveals the region of hypoaesthesia beneath; if the area of hypoaesthesia is not treated quickly then the mechanical allodynia returns after a few weeks[12]. The authors conclude that although the nerve damage is

peripheral the mechanisms for pain sensitisation are probably centrally driven with referral back to the peripheries where it is perceived as painful hypoaesthesia.

Miscommunication?

Allodynic pain states are defined by the presence of 'central sensitisation', driven by increased nociceptor activity occurring in the absence of an appropriate stimulus. Recent work has used non-invasive neuroimaging techniques to study cortical reorganisation in those with allodynia, finding that the representation of the painful body part becomes enlarged or shrunken within the primary motor and sensory maps in the brain. Associated changes may also occur within the thalamus, visual cortex and brain stem[13-15]. Studies have shown that there is a direct relationship between perceived pain and the extent of cortical remapping, with mechanical allodynia reducing as the changes on the somatopic map start to reverse[16,17]. These structures that are vulnerable to cortical remapping are also integral to the motor planning system which ensures the conduct of smooth, co-ordinated movements and prepares the body for the consequences of that movement[18,19]. This suggests that a relationship exists between abnormalities in the motor control system and the generation and perpetuation of pain.

Fink et al[20] demonstrated that by performing congruent and incongruent movements, whilst viewing only one limb in a mirror, cortical activity changed. Critically, when the limbs moved out-of-phase and yet were seen to move in-phase, cortical activity was increased in the right dorsolateral prefrontal cortex, signalling sensorimotor conflict. When this study design was replicated with healthy volunteers and subjects were asked to describe the sensations they experienced as they performed in- and out-of phase movements, over 60% of subjects described changes in temperature, weight, loss of ownership of a limb, discomfort or even pain in the limb hidden behind the mirror during out-of-phase movements[21]. These sensations can be turned 'off' and 'on' by corrective or abnormal visual feedback; thereby demonstrating that a range of sensations, including pain can be generated in the absence of neural damage. If the same protocol is repeated in those who already have chronic pain, their symptoms are exacerbated but importantly only transiently while visual input is pertrubed[22].

We know that these peripheral changes, arsing from a conflict between motor and sensory systems, are associated with objective central changes as described above[20]. The right dorsolateral prefrontal and parietal cortices are known to be active during complex motor

tasks[23] and those that require increased motor effort[24]. Interestingly, in some patients with CRPS and Fibromyalgia, (a chronic, widespread pain state), when they try to perform bilateral, synchronised upper limb movements, with one limb obscured from vision by a mirror, they are convinced that they are performing these movements but the hidden limb remains static. Our group has termed this phenomena motor extinction and we have observed that it occurs in those with the highest level of pain and greatest body perception disturbances (publication in process).

Relieving the pain of CRPS

As cortical remapping appears to be so closely related to the level of pain perceived then therapies that target the reversal of such changes are highly relevant in CRPS. Recent work[25,26] has demonstrated that therapeutic protocols designed to improve sensorimotor perception in CRPS improve tactile discrimination, give a modest reduction in pain, and correlate with a reversal of cortical reorganisation. Furthermore, intensive Electrical Sensory Discrimination Therapies, when applied to the painful limb, have been demonstrated to significantly reduce cortical re-mapping and allodynic pain (60% reduction) in those with phantom limb pain and have been suggested as being effective in other chronic pain states[27]. Somatosensory rehabilitation techniques[28], such as rehabilitation of hyposensitivity, or more precisely of the hypoaesthetic territory and DVCS as described previously[12] for mechanical allodynia, fit well within this new approach to allodynic pain. A hierarchy of textures for the patient to apply to their painful limb is also routinely used in clinical practice and appears to have good analgesic benefit as long as the patient truly attends to the target area. However, as this technique is rarely used as a stand alone therapy it has been difficult to objectively assess the added value it gives within a multi-disciplinary programme and to determine the optimum frequency and duration of treatments. It is important to note that all of these approaches require careful mapping of the affected limb using quantitative sensory testing techniques to establish the extent, intensity and nature of the sensory abnormality prior to commencement of therapy. Whether these therapies work by normalising peripheral sensation which then influences cortical re-mapping or vice versa is yet to be established but in a condition where so few therapies offer analgesic benefit these are exciting advances.

Mirror visual feedback (MVF) is another therapeutic option for the correction of sensorimotor incongruence in CRPS[29]. It is thought to work by providing false but congruent visual feedback of the unaffected limb thereby restoring the normal pain-free relationship

between sensory feedback and motor intention[29-31]. It appears to be more effective at relieving deep tissue pain (e.g. pressing, taut) than more superficial pain (e.g. knife-like, burning) or pain associated with temperature (e.g. burning, freezing) [32]. For those where motor extinction is present on baseline assessment, (i.e. the patient cannot perform bilateral, congruent movements), then MVF should not be persisted with as it may increase pain as described in the healthy volunteer and chronic pain studies above[,21,22]. What maybe more appropriate for these patients is to progress through the graded motor imagery programme, as described by Moseley[33,34]. However, even with this pre-motor training programme it has been shown that some patients with CRPS develop adverse side effects such as an increase in pain on imagined movements or the limb laterality task, and development of pain in an unaffected limb (ipsilateral (lower or upper limb to that affected) or contralateral limb) [35]

Conclusion

Complex Regional Pain Syndrome is not a myth but a very real and highly distressing condition. It is only now, with highly sophisticated assessment techniques that we are starting to gain an insight into the mechanisms that initiate and perpetuate the bizarre symptoms that typify CRPS. In the past, our ignorance has led us to doubt the existence of such symptoms, in much the same way that amputee phantom limb pain was once disbelieved but by working across specialities and sharing knowledge and expertise from a range of disciplines we are beginning to understand CRPS and how to care for those who suffer with it.

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CASE REPORT
4-Year Follow-Up

To MD To neuroscientist To patient To therapist

Rapid Relief of a Long-standing Posttraumatic Complex Regional Pain Syndrome type II Treated by Somatosensory Rehabilitation And its 4-Year Follow-Up

Spicher, CJ[5], OT, Swiss certified HT, University scientific collaborator
Degrange, B[6], OT

CASE REPORT AND TEST CONDITIONS

Medical History

Mrs. E, a right-handed 43–year-old, Caucasian, was referred to our Somatosensory Rehabilitation Centre for a chronic pain treatment in the right hand. In May 2001, she had a trauma on the dorsal side of the right hand. She benefited between 2001 and 2002 from numerous treatment of hand therapy in another centre.

Clinical Examination

First assessment 7th of July 2004
Upon assessment Mrs. E presented IASP diagnostic criteria **for a Complex Regional Pain Syndrome** (Bruehl *et al*., 1999):
(1): Continuing pain which is disproportionate to any inciting event, with a McGill Pain Questionnaire score (Melzack, 1975; Boureau, 1984) from 52% (the last 24 hours) to 58 % (sometimes during the last week before the assessment). In particular, she presents the following symptoms and signs:
(2) Symptoms:
Sensory: positive diagnostic testing of axonal lesions (see at the bottom).
Vasomotor: reports of temperature asymmetry, skin color changes and skin color asymmetry
Sudomotor: reports of oedema
Motor: evidence of decreased range of motion, weakness.
(3) Signs:
Sensory: hyperesthesia.

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http://www.unifr.ch/neuro/rouiller/collaborators/spicher.php

[6] Somatosensory Rehabilitation Centre (2004 – 2007); General Clinic; 6, Hans-Geiler, St., 1700 Fribourg, Switzerland, Europe

Sudomotor: oedema at the palmar side of the wrist.

Positive Diagnostic testing of axonal lesions 7th July 2004 (Spicher, 2003a, 2006)**:**

- Positive aesthesiography at 0,7 gram (Létiévant, 1869, 1873, 1876; Head, 1905; Trotter & Davies, 1907; Tinel, 1916, 1917; Sunderland, 1978; Inbal *et al*., 1987; Spicher & Kohut, 2001; Spicher, 2003a, 2006, 2003b) (Fig. 1 and appendix).

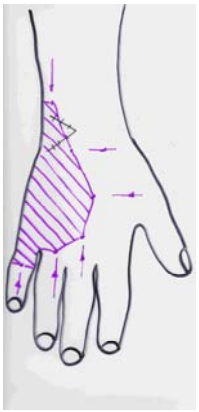

Fig. 1: *Aesthesiography at 0.7 gram (aesthesiometer 3.84). The points are marked, on each line, the first point non-felt.*

- The static two-point discrimination test is failed at 66 mm: one point is not identified from two points which are distanced from 66 mm (Weber, 1835; 1852; McDougall, 1903; Önne, 1962; Comtet, 1987; Dellon, 2000; Spicher, 2003a, 2006).
- Tingling signs:
  a) one static tingling sign (Dellon, 1984; Spicher *et al*., 1999) (Fig. 2)
  b) five advancing tingling signs: T^{++} on each digital proper dorsal nerve of the dorsal branch of the ulnar nerve (Fig. 2) (Dellon, 1984; Spicher *et al*., 1999).

Fig. 2: *The site of axonal lesions and five advancing tingling signs (T^{++}) on each digital proper dorsal nerve of the dorsal branch of the ulnar nerve.*

- Qualifiers: Peripheral neurological symptomatology in the McGill Pain Questionnaire (Melzack, 1975; Boureau, 1984): “Pricking”, “Tingling”; “Numb”. **Mrs. E. reports hot sensations IN the wrist** (it was not anymore burning as a couple of months ago).

Conclusion: Long-standing posttraumatic **Complex Regional Pain Syndrome type II**

All assessments were done by two therapists. Both are teaching the Somatosensory Rehabilitation Method. All assessments were done in the same place. The evaluations occurred at each session of treatment.

Administration Test

A. Pressure Perception Threshold

(von Frey, 1896; Semmes *et al*., 1960; Malenfant *et al.,* 1998; Levin *et al*., 1978; Spicher, 2003a, 2006)

Position

The hand to be examined is stable, if necessary aided by the examiner.

Type of stimulation

The pressure applied to the aesthesiometer by the therapist is the minimum required to bend the nylon filament of the aesthesiometer.

Stimulation of the skin only last 2 seconds and the interval between successive stimulations is 8 seconds. The time between each aesthesiometer application (ISI) is thus 10 seconds, to be counted mentally.

Explanation to the patient

The aesthesiometers are shown to the patient, who is told that he is going to be touched by some of them in order to determine the smallest pressure that he can perceive. He is asked to look away by turning his head slightly to the side. The patient replies by "touch" as soon as he perceives a stimulus.

Localization

In order to improve accuracy, the tested zone is marked with an ink spot and confirmed in comparison to an external reference point in order to be able to repeat the test at a later date.

Reference

Roughly, and every 3 aesthesiometers (5.88, 5.07, 4.56, 4.08, 3.22) in descending order, in order we are looking for the last aesthesiometer perceived, which becomes the reference. For this reference, **the mark on the aesthesiometer is noted. This will ensure that it is not included by error in the average of the six aesthesiometers retained at a later date.**

Note: Annie Malenfant's idea of the prior establishment of a reference value is based on two observations. On one hand, it is difficult for the patient to start with an ascending order where he does not perceive the first 6 to 10 aesthesiometers, and on the other, to reduce the number of aesthesiometers to 7 (3 below, the reference, 3 above) shortens the administration test administration.

Ascending order

In ascending order (A), starting 3 aesthesiometers below the reference, we look for the first aesthesiometer discerned.

Descending order

In descending order (D), starting 3 aesthesiometers above the reference, we look for the last aesthesiometer discerned.

Carry out 6 sequences: ADADAD. The 6 marks are noted on the retained aesthesiometers, in grams, according to the following table 1:

2.83 D	0.1 g.*	3.22 E	0.2 g.	3.61 F	0.4 g.	3.84 G	0.7 g.	4.08 H	1.2 g.
4.17 I	1.5 g.	4.31 J	2.1 g.	4.56 K	3.6 g.	4.74 L	5.5 g.	4.93 M	8.7 g.
5.07 N	11.7 g.	5.18 O	15.0 g.	5.46 P	29.0 g.	5.88 Q	75.0 g	6.10 R	ø

Table I: *Semmes-Weinstein Utilization Table*

*The mass applied is rounded up or down to the first decimal point; the precise value is 0.08 gram.

Comments: The aesthesiometer marks correspond to $\log_{10}$ of the application force expressed in 10^{-4} grams. Examples: ref.: 4.56 A: 1.5 D: 2.1 A: 2.1 D: 1.2 A: 1.5 D: 1.2

Result

We calculate the arithmetic average of the 6 forces applied.
This is the pressure perception threshold in grams.

B. Tingling sign in the periphery: the distal regeneration sign (advancing tingling sign)

(Trotter & Davies, 1909; Hoffmann, 1915a, 1915b; Tinel, 1915, 1916; Spicher *et al.,* 1999; Spicher, 2003a, 2006)

Position

The hand to be examined is stable, if necessary aided by the examiner's hand.

Type of stimulation

Amplitude vibration of 0.4 mm (Vibradol™)[7] or amplitude vibration of 3.0 V 160 Hz (Vibralgic™). The pressure to be applied by the therapist in the first instance is exerted by the weight of the generator probe. Subsequently, once the site has been localized, the pressure exerted is negligible (20 grams), just enough so that the 3 mm^2 tip does not slip, remaining on the point. In order to do this, the therapist's hand carry the weight of the probe.

Explanation to the patient

The probe is shown to the patient. It is then placed somewhere on his controlateral side in order to demonstrate a **localized** vibration. He is asked to look away by turning his head slightly to the side. The patient replies by "stop" when he perceives a **radiation towards the periphery**.

Localisation

The distal regeneration sign is found on the nerve path between the extremity and **the site of axonal lesions.**

Reference procedure

Proceed slowly from DISTAL to PROXIMAL in a large zigzag, but closely spaced over the presumed damaged nerve. When the patient signals for progression to be stopped, the probe is raised by the therapist, who marks the centre of the circle left by the tip of the pen on the skin with a point.

Controlled pressure

Site identification is verified using minimum probe pressure on the previously marked point. This is feasible if done promptly, but difficult to do when effecting a zigzag mobile displacement.

Result

The point is transferred onto the graph paper and, further, the patient is asked in which finger and on which side the radiation travels. This is also duly noted.

[7] www.vibradol.ch. The authors have no benefit in the sales of this apparatus.

TREATMENT

The treatment began the 7th July 2004 and finished the 28th October 2004. Mrs. E. benefited from 10 sessions of treatment. The duration of each session was between 30 minutes and 75 minutes (mean: 45 minutes).

Theory

The rehabilitation of hyposensitivity is based on the neuroplasticity of the somatosensory system (Rouiller, 2004).

Each session was divided into 3 parts: 1. Testing. 2. Adaptation of the exercises at home. For example: the hands-on therapy. 3. Stimulation by mechanical vibrations.

Technique

A. Hands-on Therapy

Definition

This is a manner in which the patient **explores everything that comes into his hand** during the day and then **verifies its sensations** with his other, non-damaged, hand (Fig. 3). In short, the patient compares the strange sensations with known ones.

Hands-on Therapy

Part of the dorsal side of your hand lacks sensitivity, making it difficult to use.

Your nervous system can still learn to identify things better. In order to do this, we ask you to:

- 8 X per day,
  - **Perceive a texture with your right hand, then**
  - **Compare the sensation with the other hand**

  For example: touch the numb area with rabbit fur, your jeans, your face, etc.

In short, anything that comes to hand**.**

Your hand therapist will help you assess your progress:

Your sensations will become less and less strange.

Good luck!

Fig. 3: *The hand-on therapy's document given and adapted to each patient for his exercises at home.*

This aspect of rehabilitation of hyposensitivity is very important, since it allows a comparison with the touch beforehand, with life before the accident. However, for the therapist, it necessitates a meticulous weekly follow up, without which, the patient often becomes discouraged by attempting differentiations that are too difficult.

B. Stimulation by mechanical vibrations

The somatosensory rehabilitation comprises three treatment phases: 1. Distant vibrotactile counter stimulation (DVCS), in the presence of a possible allodynic territory. 2. Rehabilitation of hyposensitivity. 3. Desensitization by mechanical vibrations at the site of axonal lesions.

The stimulation by mechanical vibrations is one of the techniques to practise the rehabilitation of hyposensitivity. The authors had here intentionally not used the concept of transcutaneous vibratory stimulation (TVS), which remains ambiguous. The mechanical vibrations can be used during the different phases of somatosensory rehabilitation (i.e. Distant Vibratory counter-stimulation, Rehabilitation of hyposensitivity, Desensitization at the site of axonal lesions).

During the rehabilitation of hyposensitivity stimulation by mechanical vibrations is administrated during ten minutes on the hypoaesthetic territory: the aesthesiography. The frequency is at 100 Hz. The value of the amplitude vibration equals: the Vibration Perception Threshold + 0.1 mm with the Vibradol™ [for different generators (Favre, 2002)].

C. Stress Loading Program

(Watson & Carlson, 1987; Carlson & Watson, 1988; Carlson, 1998)

The Stress Loading Program began the 30th September at 2 kg, 3 times **1 minute,** per day with the Dystrophile™. It was interrupted the 28th of October at 2 kg, 3 times **3 minutes,** per day because Mrs. E. had no more pain.

RESULTS

The tables II& III show how the hypoaesthesia decreased.

Dates	PPT
07.07.2004 (t_0)	ND: aesthesiography
14.07.2004 (t_7)	2.1 grams
26.07.2004 (t_{19})	1.2 gram
16.08.2004 (t_{40})	1.0 gram
30.08.2004 (t_{54})	0.6 gram
30.09.2004 (t_{84})	0.5 gram
28.10.2004 (t_{109})	0.4 gram
4-year Follow-Up	
19.10.2008	0.1 gram

Table II: *The evolution of the Pressure Perception Threshold (PPT)*

Dates	2-point discrimination test
07.7.2004 (t_0)	66 mm failed
23.8.2004 (t_{47})	12 mm
4-year Follow-Up	
19.10.2008	12 mm

Table III: *The evolution of the 2-point discrimination test*

The table 4 shows how the chronic neuropathic pains decreased during the same period.

Dates	McGill Pain Questionnaire
07.07.2004(t_0)	52 points
14.07.2004 (t_7)	47 points
26.07.2004 (t_{19})	37 points
23.08.2004 (t_{47})	14 points
28.10.2004 (t_{109})	0 – 5 points
4-year Follow-Up	
19.10.2008	0 – 5 points

Table IV: *The evolution of the chronic neuropathic pain by the McGill Pain Questionnaire; 4-year Follow-Up: 27 days per month: no pain; 3 – 4 days per month 5 points / 100.*

Figure 4 shows the disappearance of the tingling signs during the rehabilitation of the hyposensitivity.

Fig. 4: *The evolution of the five advancing tingling signs (T^{++}) on each digital proper dorsal nerve of the dorsal branch of the ulnar nerve during rehabilitation of hyposensitivity at: t_0, t_{40}, t_{47}, t_{54}, t_{64} & t_{85}.*

Mrs. E. finished the 3rd period of CRPS II rehabilitation (Stanton-Hicks *et al*., 1998; Spicher, 2003a, 2006) on the 28.10.2004. She tolerated a 3 x 3 min. stress loading program with the Dystrophile™ without pain neither in the shoulder nor the hand. She withstood a pressure on the hand with a dorsal flexion of 90 degrees without pain. Sometimes, she would still perceived weak tingling and weak squeezing on the wrist. However, the hot sensation completely disappeared, as well as the numbness sensation. The Pressure Perception Threshold was indeed normal: it passed from 2.1 grams to 0.4 gram.

4-year Follow-Up 19th of September 2008

Four years after the interruption of her treatment Mrs. E. was assessed for a Follow-up.

- The aesthesiography at 0.7 gram was still negative
- The value of the static two-point discrimination test was: 12 mm
- The five advancing tingling signs: T^{++} were still absent
- The only qualifiers which still annoyed (1/5) Mrs. E. 3 days per month were: painful cool sensation in the fingers 1/5; tugging 1/5; numb sensation in the wrist when she was driving a long trip
- At least, the value of the Pressure Perception Threshold was: 0.1 gram (see also Tables 2, 3 & 4)

DISCUSSION

The first publications in English (Ottoson, 1981; Lundeberg, 1984a, 1984b) that showed interest in using vibrations for their antalgic effect spoke of vibratory stimulation, whilst subsequent French authors (Romain *et al*., 1989), spoke of transcutaneous vibratory stimulation (Spicher & Kohut, 1996). This term is, however, ambiguous, since it does not clearly describe the effect of the vibratory stimulation. In fact, the first effect of the vibrations is antalgic and the second – and only the second – is to stimulate sensory recovery.

The author (Spicher & Kohut, 1997) published a patient's clinical result showing that mechanical vibrations had clearly modified the vibrotactile sense status to a considerable extent. Yves Allieu's (MD) comments about this article are highly interesting. "The improvement in sense obtained by the author using TVS on a 19-year-old ulnar lesion can, legitimately, surprise a reader not informed about this technique. However, we can confirm that we have, on several occasions, personally observed an improvement in sense discrimination of old lesions using vibratory stimulation, perhaps not always in such a significant way."

The observations of Silas Weir Mitchell (1874) and Cheryl Chrisman (1998): *"the causalgia* (CRPS type II) *comes from the cohabitation of altered axons and non-altered axons"* on the same area show us the way to reeducate this disease. This 4-year follow-up without any change of its successful result gives us a robust confirmation to go on this physical therapy way.

CONCLUSION

The rehabilitation of hyposensitivity based on the somatosensory system neuroplasticity has numerous indications. This case report shows one of its possibilities: Somatosensory rehabilitation of CRPS type II (causalgia) without mechanical allodynia.

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APPENDIX Experimental protocol for the aesthesiography.

Objective:

To map the boundaries of the hypoaesthetic territory.

Material:

- A4 millimetric graph paper
- Set of 20 Semmes-Weinstein pressure aesthesiometers.

Test procedure:

The hand to be examined is stable, if necessary stabilized by the examiner's hand.

Type of stimulation:

The pressure applied to the aesthesiometer by the therapist is the minimum force required to bend the nylon filament. The stimulation on the skin last for 2 seconds and the interstimulus interval (ISI) is 8 seconds. The time between each aesthesiometer application is thus 10 seconds, to be counted mentally.

Choice of aesthesiometer by the therapist:

In a descending series, the last aesthesiometer detected on the contralateral side is determined. i.e. it is 0.2 gram (mark: 3.22) on the dorsal face of the hand. Subsequently, select two aesthesiometers next to the first aesthesiometer detected both in the ascending and descending directions i.e. 0.7 gram for this case report. This series of five aesthesiometer is then used for

delineating the hypoaesthetic territory. If the aesthesiometer is too small, the contour will be imprecise. If on the contrary, it is too large, there will be no hypoaesthetic territory.
Explanation on the determination of the aesthesiography are given to the patient: the aesthesiometers are shown to her, who is told that she is going to be touched by some of them in order to determine the territory where she feels less than normal. She is asked to look away by turning her head slightly to the side. The patient replies by touched as soon as she detects the stimulus.

Localization:
In order to help the therapist trace the final polygon, it is easier to place the graph paper besides the hand and parallel to it, so that he only has to mentally do a transfer between the hand and the recording paper (and not also a rotation).
Longitudinal axis:
The first point not-detected by the patient is identified. On the longitudinal axis, from the proximal to the distal, the first stimulating site not-perceived by the patient is determined, advancing centimeter by centimeter. Move then back from distal to proximal in order to find the first detected point. Finally, the first point not-detected along this axis is found by moving forward again from proximal to distal, but now advancing millimeter by millimeter.
Transverse axis:
Search the first point not-detected by the patient along the axis perpendicular to the presumed damaged nerve. On the axis from right to left (e.g. for a palm face of a right hand, in case of lesion of the ulnar nerve), search the first point not detected by the patient, advancing centimeter by centimeter. Then return towards the right to find the first point detected. The next step is to return towards the left, but advancing millimeter by millimeter, in order to find the first point not-detected on the transverse axis. Finally, mark the point found on the paper and trace with an arrow the axis that was considered. If necessary, continue the search for other points on the lines: transverse axis of the metacarpal heads, transverse axis of the PIP, longitudinal axis from distal to proximal, etc.

Result:
We trace a polygon joining up the points determined, reflecting the extent and position of the hypoaesthetic territory.

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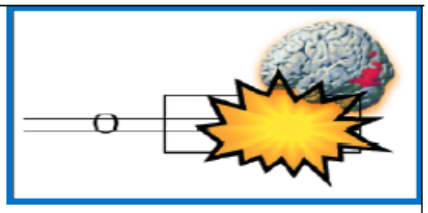

CONTINUOUS EDUCATION

4th Week for Somatosensory Rehabilitation
5th to 8th March 2012

4th WEEK for SOMATOSENSORY REHABILITATION 2012

Problem

- When the patients that are placed in our care have been suffering too much for too long, when their facial expression remains frozen, how can the hope of a better tomorrow be rejuvenated: a future with less shooting pain, with less burning sensations - simply put - with a decrease of **neuropathic pain**.
- Most patients suffering from chronic pain have cutaneous sense disorders. A decrease in the hypoaesthesia (for example the pressure perception threshold) will, at the same time, cause a decrease of their chronic neuropathic pain (for example the McGill Pain Questionnaire).

Overall Aim

- To rehabilitate the disorders of the cutaneous sense on the basis of the neuroplasticity of the somaesthetic system so as to lessen chronic neuropathic pain.
- To avert the outbreak of painful complications by rehabilitating the cutaneous sense.
- To build bridges between rehabilitation, medicine and the neurosciences.

Specific Objective

- To evaluate disorders of the cutaneous sense: aesthesiography, static 2-point discrimination test, tingling signs and somaesthetic symptoms, pressure perception threshold, etc.
- To evaluate painful complications with the McGill Pain Questionnaire: mechanical allodynia, reflex sympathetic dystrophies, neuralgia, etc.
- To implement planned rehabilitation procedures within the context of chronic pain complications.
- To adapt the knowledge of mainstream neurology for use in rehabilitating neuropathic pain and vice versa.

Teachers

- Claude Spicher, BSc OT, Swiss certified HT, Manager & therapist in the Somatosensory Rehabilitation Centre, University scientific collaborator www.unifr.ch/neuro/rouiller/collaborators/spicher.php
- Rebekah Della Casa, OT, therapist in the Somatosensory Rehabilitation Ctr.
- Isabelle Quintal, BSc OT, therapist in the Somatosensory Rehabilitation Ctr.

Guestspeakers (on Monday, at 6 p.m.)

- Dr Josef Strehle, MD, Fusszentrum Bern www.myfeet.ch, BERN, Switzerland
- Prof Jean-Marie Annoni, MD, Neurology Unit, Department of Medicine, University of Fribourg, FRIBOURG, Switzerland
- Dr Sebastian Dieguez, PhD, Psychologist, Neurology Unit, Department of Medicine, University of Fribourg, FRIBOURG, Switzerland

www.unifr.ch/neuro/rouiller/teaching/continedu.php

Date	5th to 8th of March 2012
Time Table	9am – 12am & 1pm – 5 pm
Duration	28 hours
Place	Clinique Générale; 6, Hans-Geiler Street ; Friburg
Price	CHF 990 / 1050 CAD Dollars / 1070 US Dollars / € 780 / £ 660 (Work Documents in English + Handbook + Atlas)
Reference	Spicher, C.J. (2006). *Handbook for Somatosensory Rehabilitation.* Montpellier, Paris: Sauramps Médical. Spicher, C.J., Desfoux, N. & Sprumont, P. (2010). *Atlas des territoires cutanés du corps humain.* Montpellier, Paris: Sauramps Médical.

4th Week for Somatosensory Rehabilitation

5th to 8th of March 2012

REGISTRATION FORM

Deadline: Monday, 6th February 2012

Name:

First (given) name:

Professional occupation:

Address:

e-mail address:

Please fill and return to:

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Rue du Musée 5
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or

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e-News 2006; 3(2)

Patienten Übersicht Nr. 4

«Krankheitverlauf»

To MD. To neuroscientist To patient To therapist

Meine Krankheitsgeschichte begann im Januar 2001.
Damals hatte ich nach längerer Pause wieder mit dem Joggen begonnen und deshalb neue Laufschuhe gekauft.
Sehr bald bekam ich Schmerzen am rechten Fuß, genauer an der tibialis anterior- Sehne in Knöchelnähe.
Ich ging sofort zu einem Facharzt, der den Fuß zunächst mit Verbänden, dann per Gipsschiene mehrmals für 10-14 Tage ruhig stellte und mir Schmerzmittel verschrieb.
War der Fuß eingegipst, hatte ich vor den Schmerzen weitgehend Ruhe, war der Gips entfernt, waren die Schmerzen nach wenigen Tagen umso stärker zurück.

Im Sommer 2001 war der gesamte Fuß dick und rot angeschwollen, mittels Kernspin wurde eine akute Reizung des oberen und unteren Sprunggelenks diagnostiziert.
Mit wachsender Verzweiflung versuchte ich ärztlich Hilfe zu finden, konnte ich doch nur noch an Krücken gehen.
Leider musste ich dabei keine guten Erfahrungen machen.
Die einen Ärzte meinten, ich hätte halt eine chronifizierte Sehnenscheidenentzündung, das sei sicher unangenehm, aber kein Grund sich anzustellen, andere Ärzte sagten, bei mir sei offensichtlich eine initiale Polyarthritis im Gange, meine Blutwerte seien zwar vollkommen in Ordnung, aber das läge am sog. diagnostischen Fenster. Ich solle hoch dosiert Voltaren einnehmen und regelmäßig mein Blut untersuchen lassen.

Was ich alles versuchte, nur um meine Fußprobleme zu beheben, würde den Rahmen eines normalen Berichts bei weitem sprengen.
Meine Wege führten mich von Heidelberg bis München, sämtlichen physiotherapeutischen Maßnahmen ließ ich über mich ergehen.
Letztlich endeten aber sämtliche Therapieversuche mit dem Spritzen von Cortison, das tangential an die Sehne injiziert wurde.
Durch die ständigen Gipsschienen rechts, bekam ich durch die Überlastung nun auch noch Schmerzen am linken Fuß.

Schließlich ließ ich, als ich einfach nicht mehr weiter wusste, im November 2001 in Heidelberg eine „Exploration“ am rechten Fuß durchführen, um eine vermeintlich mechanische Ursache der Schmerzen beheben zu lassen.
Im Verlauf der Operation wurde die tibialis anterior-Sehnenscheide gespalten und gespült.

Waren die Schmerzen vor der Operation schon schlimm, so waren sie jetzt katastrophal.
Rechts konnte ich so gut wie gar nicht mehr auftreten, geschweige denn abrollen oder gehen.

Der gesamte Fuß war jetzt nicht nur dick und rot geschwollen, in der Nähe und an bzw. auf der Sehne bildeten sich knubbelartige Verhärtungen, die die Schmerzen noch steigerten.
Zusätzlich traten nun auch Probleme mit dem Nagel der Großzehe auf, der plötzlich anfing einzuwachsen und zu eitern.

Mein berufliches und soziales Leben kam fast völlig zum erliegen, ich konnte mich nicht mehr selbst versorgen und zog wieder bei meinen Eltern ein.

Zwischen März 2002 und Januar 2003 unterzog ich mich in München 3 Nachoperationen, im gleichen Zeitraum wurden 6 Eingriffe am rechten Zehennagel vorgenommen (Emmert-Plastik), was den Nagel nicht davon abhielt, auch zum 7.mal zu eitern.
Zwischen den Operationen wurde der geschwollene Fuß 3-4 mal in der Woche mit Querfriktionen behandelt, um die Sehnenverhärtungen zu mobilisieren.
Die Schmerzen waren einfach unsäglich, aber man sagte mir, Querfriktionen müssten weh-tun, wenn sie helfen sollten.

Nach dem Eingriff im Januar 2003, kam es zu einer weiteren dramatischen Verschlechterung.
Die Farbe meines Fußes wandelte sich von Dunkelrot zu Purpur, die Haut fing an zu glänzen, als ob sie mit einer dicken Wachsschicht überzogen gewesen wäre und fühlte sich ständig heiß an.
Als Therapie schlug man mir eine weitere Operation vor, bei der ein Nerv in einem Loch, das in meinen Schienbeinknochen gebohrt werden sollte, versenkt werden sollte.

Ich weiß nicht, was aus mir geworden wäre, wenn ich nicht zufällig Anfang März 2003 im Internet das Zentrum für Fußchirurgie, Bern gefunden hätte.
Bei Dr. Strehle hörte ich erstmalig den Begriff „Morbus Sudeck“, der sich bei mir unmittelbar im Übergang von Phase 2 zu Phase 3 befand.
Meine Therapie wurde vollkommen umgestellt, u.a. wurde der Fuß geschont, in Salzwasser gebadet, ich nahm Calcitonin als Nasenspray ein, der Zehennagel erhielt eine Spange.

Zudem sollte in Absprache mit Dr. Strehle in Deutschland eine ambulante Schmerztherapie vorgenommen werden.
Dies war zwingend notwendig geworden, da ich inzwischen vor lauter Schmerzen nicht einmal mehr richtig liegen konnte.
An ein normales Durchschlafen war schon lange nicht mehr zu denken, da ich mich wegen der extremen Schmerzempfindlichkeit, die brennend bis zu meinem Knie ausstrahlte, nicht mehr auf die Seite drehen konnte und der Fuß hoch gelagert werden musste.
Veränderte ich im Schlaf meine Lage, wachte ich wegen der Schmerzen sofort auf, das kam pro Nacht ca. 5-10 mal vor, so dass ich schließlich dazu überging, meinen Unterschenkel nachts mit Paketklebeband zu fixieren.

Als besonders problematisch erwies sich das bei der Schmerztherapie u.a. verwendete Medikament Neurontin.
Hiervon sollte ich zunächst 3600 mg pro Tag einnehmen.
Die auftretenden Nebenwirkungen waren schon nach wenigen Wochen sehr unangenehm.
Innerhalb kurzer Zeit nahm ich stark an Gewicht zu. Ich war ständig müde und erschöpft, es viel mir immer schwerer mich auf eine Sache zu konzentrieren.

Dies ging nach ca. 2 Monaten so weit, dass ich beim Lesen nach 3 Buchseiten nicht mehr genau wusste, was zuvor in der Geschichte passiert war.

Nach 3-4 Monaten fing ich an zu stottern, kurz danach fielen mir beim Sprechen mitten im Satz bestimmte Worte nicht mehr ein.
So weiß ich z.B. noch genau, dass ich einmal zu meiner Mutter sagen wollte, dass das Telefon geklingelt habe, ich sah das Telefon auch geistig vor mir, konnte mich aber partout nicht daran erinnern, wie das seltsame Ding mit den Nummern auf den Tasten denn eigentlich heißt.

Gegen die Schmerzen half das Medikament allenfalls bedingt, deshalb sollte die Dosis auf 7200 mg täglich verdoppelt werden.
Daraufhin brach ich Therapie ab.

Bis zum September 2004 hatte sich der Morbus Sudeck zumindest soweit zurückgebildet, die Schwellung und Farbe des Fußes hatten sich einigermaßen normalisiert, sodass ich mich bei Dr. Strehle in Bern einer weiteren Operation unterziehen konnte.
Dies war durch die extremen Verhärtungen im Gewebe und die Nervenschmerzen, die durch eine in Deutschland durchgeführte Neuraltherapie weiter verschlimmert worden waren, leider unumgänglich.

Es stellte sich heraus, das ein Nerv sogar schon abgestorben war und deshalb durchtrennt und verlegt werden musste.
Außerdem entfernte Dr. Strehle das extrem ausgebildete Narbengewebe, das sich nach den vorhergehenden Operationen immer wieder neu gebildet hatte.
Nach der Operation von Dr. Strehle blieben die Verhärtungen dauerhaft verschwunden.

Allerdings hatte ich jetzt große Probleme im Bereich der Großzehe und in einem Gebiet über dem Außenknöchel, so dass mir normales Gehen ohne Krücken und sogar das Tragen eines Schuhs unmöglich war, außerdem waren der brennende Schmerzen an der Hautoberfläche wieder stärker geworden.

Wieder war es Dr. Strehle, der Rat wusste und mir die Behandlung bei Herrn Spicher, in der St. Anne-Klinik in Fribourg ermöglichte.

Nach einem ersten Gespräch im Januar 2005 war meinen Eltern und mir innerhalb kürzester Zeit klar, dass ich der von Herrn Spicher vorgeschlagenen Therapie eine Chance geben sollte, auch wenn dies bedeutete, dass mein Vater mich jeden 2. Freitag von Mannheim nach Fribourg fahren musste.
Ich brauchte Herrn Spicher eigentlich gar nicht erklären, welch brennende Schmerzen ich hatte und wie quälend diese waren, er untersuchte den Fuß und erzählte mir stattdessen vollkommend zutreffend, welche Beschwerden ich hatte.

Zunächst kam es mir sehr seltsam vor, als Herr Spicher mit einem Mäppchen zu mir kam, in dem sich lauter schmale Plastikstäbchen befanden und er mit diesen leicht in meinen Fuß piekste.
Ich lernte aber schnell den Zusammenhang zu der Regenbogenskala des Schmerzes und deren unterschiedliche Schmerniveaus kennen.

Auch die Intensität mit der Herr Spicher mich über die Eigenart und Qualität meiner Schmerzen regelmäßig befragte, war für mich absolutes Neuland.

Ist der Schmerz brennend oder schneidend? Stechend oder pieksend? Kratzend oder doch eher juckend?

Was ist eine mechanische Allodynie?

Am glücklichsten war ich allerdings über die Tatsache, dass bei der Behandlung mein Fuß in Ruhe gelassen wurde, und die Gegenstimulation der Nerven knapp unterhalb des Knies begann und sich dann erst Schritt für Schritt langsam nach unten arbeitete.
Auch nicht gewaltsames Laufen ohne Krücken wurde mir verordnet, sondern nach wie vor vorsichtige Schonung.
Die Verwunderung darüber, dass ich plötzlich zu Hause damit begann, an meinem Unterschenkel zunächst mit einem Kaninchenfell, dann mit einem Pinsel und schließlich mit

unterschiedlichen Stoffen zu reiben und regelmäßig auf eine bestimmte Stelle meines Schienbeins zu klopfen, war verständlicherweise groß.

Der Erfolg der Gegenstimulation ließ die anfängliche Skepsis schnell verschwinden.

Die größte Überwindung kostete mich, wieder das Neurontin einzunehmen.
Herr Spicher versicherte mir jedoch, dass ich nur eine Tagesdosis von 300 mg einnehmen sollte, und deshalb die schlimmen Nebenwirkungen nicht zu erwarten wären und eine derartig niedrige Dosierung außerdem eine deutlich bessere Wirkung gegen die Schmerzsymptome hätte.
Wieder behielt er Recht.

Nun, nach über einem Jahr Therapie in Fribourg, ist der Zustand meines Fußes so weit verbessert, dass ich wieder damit beginnen kann einen Schuh zu tragen.
Sämtliche Verfärbungen haben sich normalisiert, das gesamte quälende Brennen ist verschwunden, lediglich die Phantomschmerzen, die durch den abgestorbenen Nerv verursacht werden, müssen noch weiter behandelt werden.

So bleibt mir mich nur mich zu bedanken, bei Herrn Dr. Strehle, dem Arzt bzw. Operateur, der mir nach Jahren falscher Diagnosen und Therapien endlich helfen konnte und der mich nicht nur durch seine fachliche Kompetenz, sondern auch durch seine Menschlichkeit tief beeindruckt hat.
Außerdem bei Herrn Spicher und Frau Degrange, die mich kurzfristig in Ihr Therapieprogramm aufnahmen, mir immer Mut machen, geduldig mein Französisch ertragen und mir terminlich immer entgegenkommen.
Bei meiner Reha-ärztin, Frau Monika Herbert in Bammental bei Heidelberg, die mich treu und verständnisvoll seit etwa 3 Jahren begleitet und meine einzig verbliebene Anlaufstelle in der Nähe meines Wohnortes geblieben ist.
Und natürlich bei meinen Eltern. Was sie die letzten Jahre mit mir durchmachten, lässt sich nach diesem Bericht allenfalls erahnen.

Danke schön!

Björn Dill, im März 2006

e-News 2008; 5(4)

POSTER

To MD. To neuroscientist To patient To therapist

INTÉRÊT DE LA CORTISONE DANS LE SDRC I

Docteurs : B. Leroy, C. Lemaire, E. Heuse et I. Pevée (CHR la Citadelle de Liège)

La kinésithérapie, les bains écossais, les antalgiques et un antioxydant ont effet de preuve dans la prise en charge des SDRC I.

Médrol
32mg durant 4 j
16mg durant 8 j
8mg durant 4 j
4mg durant 4 j

La prise d'un traitement de courte durée de Médrol améliore-t-il la prise en charge des patients atteints de SDRC I ?

Dans la physiopathologie du SDRCI, la composante inflammatoire n'est que peu traitée. L'étude porte sur l'intérêt de la prise d'un traitement de courte durée de Médrol versus placebo pour des patients présentant un SDRC I aux membres supérieurs ou inférieurs. Une scintigraphie devait le confirmer. Il pouvait survenir après une fracture, de la chirurgie, une entorse ou une contusion.

Etude

Les patients présentant un SDRC I adressés au service sont répartis en deux groupes de manière aléatoire. Des 35 patients enrôlés, 28 ont été validés. Tous les patients recevaient le traitement validé. Un groupe reçoit du Médrol 32mg durant 4 j, 16mg durant 8 j, 8mg durant 4 j et 4mg durant 4 j ; l'autre reçoit un placebo.
Nous prenons les paramètres ci-dessous à la première consultation et à la dixième semaine. Les patient sont vus à deux reprises durant l'intervalle. Les deux groupes sont identiques pour les âges, le genre, la taille, la BMI et le temps écoulé depuis le début de la maladie.

Résultats

Différence de douleur à la pression

	Mi-coude (ns)	Mi-avant bras (ns)	Poignet (ns)	Mi-rotule (ns)	Mi-tibia (ns)	Cheville
Groupe P	3	2	5	1	1	3
Groupe M	3	5	6	1	2	10

Différence de périmètre

	Mi-coude	Mi-poignet	Mi-rotule	Mi-torse
Groupe P	0,3	6.6	0,05	1.22
Groupe M	1,04	1,3	0,7	1.28

Goniomètre

	Flexion (ns)	Extension
Groupe P	13.2	7.1
Groupe M	21.8	18.8

	EVA	HAD (ns) Anxiété	HAD (ns) Dépression	SF36
Groupe P	16	1.9	1.4	6.3
Groupe M	64	2.4	0.9	10.3

ns = non significatif

Conclusion

Dans cette étude nous observons que la prise de cortisone à dose décroissante apparaît comme efficace dans le traitement de la SDRC I. L'effet est bénéfique, avec un recul de 10 semaines, sur l'œdème, l'extension, la qualité de vie et la douleur. Cela semble plus efficace pour la cheville.

Commentaires

- Douleurs à la pression n'ont pu montrer de différences significatives sauf à la cheville avec 95% de certitude.
- Périmètre (oedème) : différence significative partout. Le périmètre diminue avec 95% de certitude plus nettement pour les patients sous Médrol. (Et même 99% dans le mi-tarse).
- Goniomètre : l'extension augmente avec 99% de certitude. La flexion n'affiche pas de différence significative.
- EVA : diminue avec 99% de certitude.
- L'HAD ne varie pas de manière significative.
- La SF 36 s'améliore avec 95 % de certitude.

SDRC: syndrome loco-régional douloureux complexe
[CRPS : Complex Regional Pain Syndrome]
Groupe P: Placébo **Groupe M**: Médrol® : Méthylprednisolone
EVA: Echelle Visuelle Analogique de 10 cm

e-News 2010; 7(4)

GUESTEDITORIAL
Cortical Plasticity in Complex Regional Pain Syndromes

To MD. To neuroscientist To patient To therapist

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Keywords: CRPS, neuropathic pain, hyperalgesia, plasticity, reorganization

The Complex Regional Pain Syndromes (CRPS; previously called "reflex sympathetic dystrophy" or "causalgia") belong to the remarkably large group of neuropathic pain syndromes. CRPS may develop in about 5 % of all traumas or nerve lesions in the extremities. Based on either the absence or presence of nerve lesions, CRPS is classified as CRPS 1 or 2. Clinically, these syndromes are characterized by a typical constellation of symptoms[1-3]: autonomic and inflammatory changes, motor symptoms and sensory disturbances (see Fig. 1).

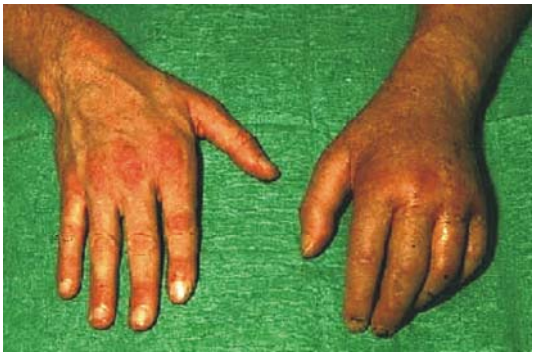

Fig. 1: *Acute CRPS: pain, swelling, reddish skin and decreased active range of motion in the left hand.*

Autonomic and inflammatory symptoms include presence of distal edema, changes of the skin temperature and color, and excessive sweating. Motor symptoms in CRPS are weakness, tremor, dystonia and myoclonic jerks.[4] Among the sensory symptoms spontaneous pain and stimulus-evoked pains are hallmarks. Basically, two types of stimulus- induced pains can be differentiated in CRPS. Pin- prick hyperalgesia means that a normally painful pin- prick stimulus induces an increased pain perception on the affected limb.[5] Dynamic-mechanical allodynia means that non-painful gentle stroking the affected skin is perceived as painful.[6] As mandatory for diagnosis, pain and hyperalgesia must not be limited to a single nerve or nerve root territory.[7] Moreover, sensory abnormalities are usually distributed in a stocking or glove like manner. The reasons for these puzzling phenomena are largely unknown. However, they may be explained by changes within the CNS. In the last couple of years our group has employed functional imaging techniques and explored possible CNS pathology in CRPS.[5,6,8-10] Using functional magnetic resonance imaging (fMRI) and magnetoencephalography

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(MEG), we provided evidence that the pathophysiology of this syndrome is not only limited to the peripheral nervous system. Most of the complex sensory features of CRPS were found to be crucially linked to characteristic CNS changes. The aim of our first MEG study[8] was to assess a possible cortical reorganization within the primary somatosensory cortex (S1) in 12 CRPS patients and correlate these changes to sensory, motor or autonomic complaints. Pneumatic tactile stimulations were used to explore the cortical representation of digits 1 (D1), 5 (D5) and the lower lip, both on the unaffected and CRPS- affected side. Projections of the respective MEG activities onto axial and coronal MRI slices demonstrated that the location of the SEF sources in the contralateral S1 cortex were arranged in a somatotopic manner, showing the cortical representation of the lower lip, D1, and D5 (Fig. 2).

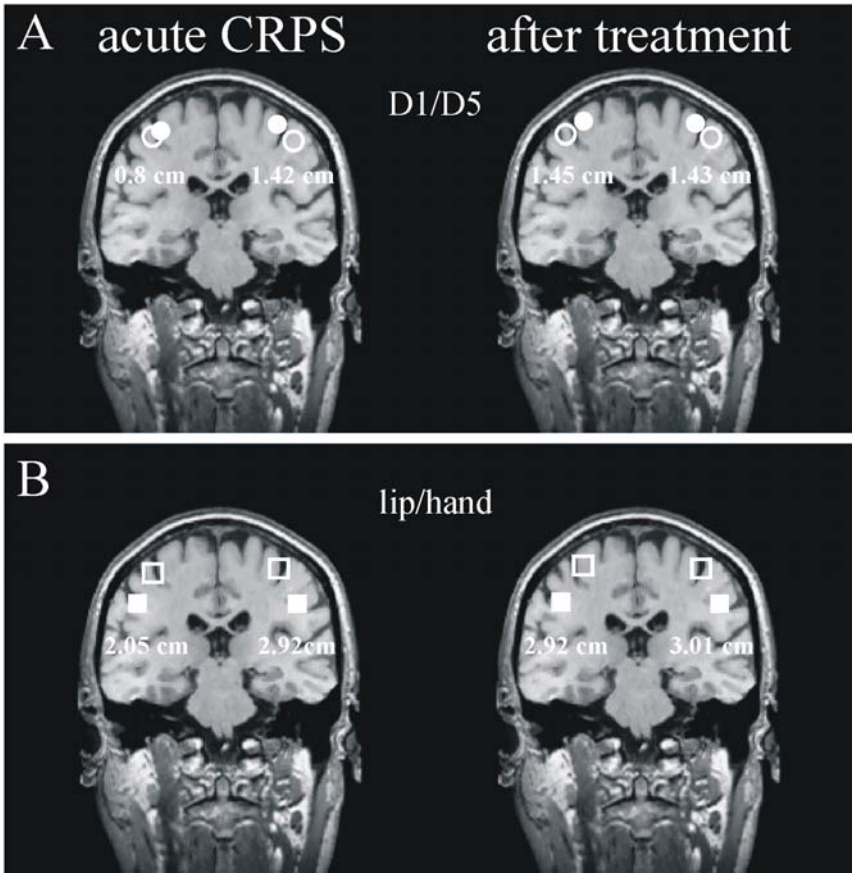

Fig. 2: *(A) Projection of the equivalent current dipole (ECD) localizations for D1 (filed circle) and D5 (open circle) onto individual MRI slices for one representative patient. There was a reduction of the hand extension from 1.42 cm (unaffected side) to 0.8 cm (CRPS-affected side) during acute CRPS. After the follow up time of 62 months, the distance between D1 and D5 increased to 1.45 cm on the affected side, whereas the corresponding distance on the unaffected side remained unchanged (1.43 cm).*
(B) Projection of the ECDs for the center of the hand (open squares) and the lower lip (filled squares) onto individual MRI slices. The distance between the center of the hand and the lower lip increased from 2.05 cm to 2.92 cm following therapy on the CRPS-affected side, whereas the respective ECD localizations for the unaffected side remained unchanged (distance hand-lip 2.92 versus 3.01 cm, before and after treatment).

To assess changes in the somatotopic map, the center of the hand representation itself was deemed to be the mid- point between the cortical representations of D1 and D5. Surprisingly, the distance between the cortical representation of the hand and the lip was markedly decreased on the affected CRPS side compared to the unaffected side (Fig. 2). The mean distance on the unaffected side was 2.76 cm compared with 1.95 cm on the affected side ($p < 0.05$). Additionally, the distance between D1 and D5 was reduced on the painful affected side (1.37 versus 0.80 cm for the unaffected and affected sides; $p < 0.05$). However, the degree of cortical reorganization was significantly correlated to the magnitude of CRPS pain, assessed with the McGill Pain questionnaire (MPQ; $r = 0.792$; $p < 0.05$) and the area of mechanical hyperalgesia ($r = 0.810$; $p < 0.05$). The latter was found to be the best predictor, using a multiple regression model. There was no correlation with other clinical symptoms or epidemiological data. In particular, there was no correlation between the duration of CRPS, pain during movement, the impairment of hand function (active range of motion) or autonomic scores. In order to investigate whether these S1 changes are reversible and how they change after treatment, we performed a follow up study[9] and traced the somatotopy within the S1 cortex of our patients employing MEG at least 1 year after therapy. Figure 2 shows a representative specimen, in which the distance between D1 and D5 increased from 0.80 cm during acute CRPS to 1.45 cm 1 year later and the distance between hand and lower lip accordingly increased from 2.05 to 2.92 cm. In contrast, the dipole locations on the unaffected side remained unchanged. For the whole group, at time of second investigation the distance between cortical representation of D1 and D5 on the CRPS side increased from 0.86 ± 0.1 cm (acute CRPS) to 1.16 ± 0.1 cm ($p < 0.005$). In contrast, there was no difference on the unaffected side (1.1 ± 0.1 vs 1.2 ± 0.1cm). According to hand representation, the distance between hand and lower lip increased on the CRPS side following therapy (1.6 ± 0.2 vs 2.2 ± 0.1 cm; $p = 0.009$). In order to determine which clinical improvement at best predicts recovery from cortical reorganization, we performed multiple regression analysis including the change of cortical reorganization (difference) between measurement one and two as dependent variable, and as independent variables the results obtained from quantitative sensory testing. The only factor that predicted the reduction of cortical reorganization was the reduction of pain, as measured by the MPQ (beta weight = 0.8; $p = 0.006$). Thus, reduction of neuropathic pain was directly correlated with recovery from cortical reorganization. Furthermore, symptoms associated with central nociceptive sensitization processes, in particular mechanical hyperalgesia seem to be most important for the induction of plastic CNS changes in CRPS. Therefore, in order to get more insights into the neuronal matrix involved in the central processing of stimulus- evoked pains (hyperalgesia), we performed another study[5] and used fMRI to explore brain activations during pin- prick hyperalgesia in CRPS patients. Twelve patients, in whom previous quantitative sensory testing revealed the presence of hyperalgesia to punctuate mechanical stimuli (i.e. pin-prick hyperalgesia), were included in the study. Pin-prick-hyperalgesia was elicited by von-Frey filaments at the affected limb. For control, the identical stimulation was performed on the unaffected limb. The latter stimulation predominantly led to activations of contralateral S1 cortex (Fig. 3), parietal association cortex and bilateral S2 cortices. Furthermore increases of the BOLD signal were seen in the contralateral insular cortex. The identical stimulation on the affected

side was markedly painful (i.e. pin-prick hyperalgesia) and we found the recruitment of a complex cortical network involved in the encoding for this CPRS feature (Fig. 3).

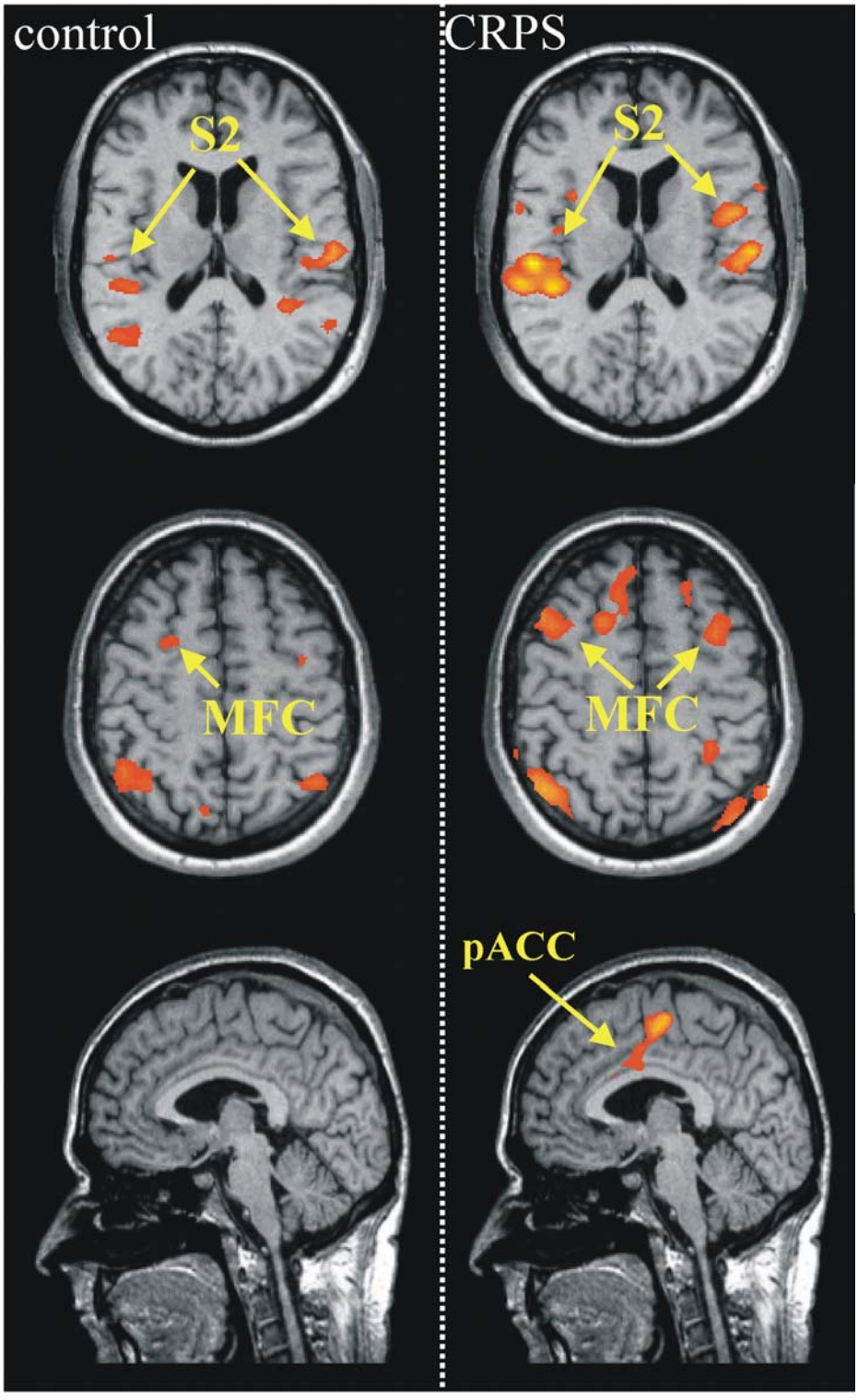

Fig. 3: *Brain activations during non- painful pin- prick stimulation at the unaffected control hand and during pin- prick hyperalgesia at the CRPS- affected hand. Note the increased activations in S2, frontal cortices (in particular middle frontal cortices; MFC) and posterior part of the anterior cingulate cortex (pACC).*

Most important areas within this network were the S1 cortex (contralateral), S2 (bilateral), insula (bilateral), associative- somatosensory cortices (contralateral), frontal cortices and parts of the anterior cingulate cortex. Recently, cortical activations underlying motor dysfunction of CRPS were also investigated.[11] During finger tapping of the affected extremity, CRPS patients showed a significant reorganization of central motor circuits, with an increased activation of primary motor and supplementary motor cortices (SMA). Furthermore, the ipsilateral motor cortex showed a markedly increased activation. When the individual amount of motor impairment was introduced as regressor in the fMRI analysis, it could be demonstrated that activations of the posterior parietal cortices, SMA and primary motor cortex were correlated with the extent of motor dysfunction.[11]

Finally, in a recent study[12] we tested changes in endogenous pain modulation in CRPS patients compared to age-matched healthy controls. We applied repetitive noxious electrical stimuli (stimulation frequency 1Hz) at the dorsal aspect of affected and unaffected hands in patients and to corresponding hands in controls. As known from previous studies this protocol simultaneously activates inhibitory and facilitatory pain modulating systems. This results in (i) adaptation to the repetitive noxious stimulus, but also, simultaneously and at the same site, in (ii) development of an area of pin- prick hyperalgesia. We measured (i) pain adaptation during the course of stimulation and (ii) the provoked area of pin-prick hyperalgesia. The parameters “pain adaptation” and “area of pin-prick hyperalgesia” were used as activity measures of pain inhibitory and pain facilitatory systems. As both measures result from gross inhibitory and gross facilitatory activity in pain modulatory systems, pain adaptation reflects net pain inhibition and area of pin-prick hyperalgesia net pain facilitation. We found (i) decreased adaptation to painful electrical stimuli on both affected and unaffected hands of CRPS patients compared to healthy controls and (ii) increased areas of hyperalgesia on affected hands of CRPS patients compared to unaffected hands of CRPS patients and healthy controls. These findings imply a shift from inhibition towards facilitation of nociceptive input in CRPS patients, based on differential activation of subcomponents of the endogenous pain modulatory system. The differences were not correlated with duration of the disease, pain intensity, autonomic or motor function scores, presence or degree of evoked pain. However, significant correlation was found with the extent of adaptation and hyperalgesia on the unaffected hand. Thus, we hypothesized that differential activity in endogenous pain modulating systems may be not only a result of CRPS, but a potential risk factor for its development.[12]

In summary, we provide several lines of evidence that patients with CRPS have characteristic CNS changes. How does cortical reorganization and re-reorganization in response to CRPS pain modulation occur? Basically, functional or structural reorganization may underlie plastic changes within the CNS. Functional reorganization includes rapid changes in the impulse processing of synaptic circuits. Afferent input to the S1 cortex following peripheral stimulation of mechanosensitive afferent fibers has been shown to modulate intracortical inhibition in area 3b within the S1 cortex of animals.[13] In fact, enhanced intracortical inhibition of motor cortex, which was accentuated in CRPS patients with hyperalgesia, has been recently demonstrated.[14] The plastic changes demonstrated herein may therefore be

explained by unmasking of preexisting, but previously latent afferent connections to S1 neurons. Alternatively, given a time frame of at least one year in our follow- up study, structural reorganization like axonal sprouting cannot be excluded. Cortical changes and altered sensorimotor processing[8,15] may explain several sensory features common in CRPS. Firstly, pain and hyperalgesia spread and are not limited to peripheral innervation territories. Instead, sensory symptoms are usually distributed in a glove- or stocking like manner.[2,3] Secondly, some patients with CRPS have even hemisensory deficits.[16] These include decreased temperature and pin-prick sensations and hereby indicate the involvement of other cortical areas than those corresponding to the primarily affected limb. Thirdly, some patients with CRPS appear to have referred sensations following somatosensory stimulation on the affected limb.[17] These referred sensations are modality specific (touch and pinprick) and perceived in the body part immediately adjacent to the S1 hand area on Penfield's cortical homunculus. Such findings could be explained by plastic S1 changes. We and others have shown that pain induces cortical reorganization, both in experimental and clinical settings.[6,18] For CRPS there is evidence for facilitated neurogenic inflammation.[19] A particular subtype of afferent C-fibers, the mechanoinsentitive ones, are important for neurogenic inflammation, which may explain peripheral features of CRPS.[20] These mechanoinsentitive C-fibers are further essential for development and maintenance of mechanical hyperalgesia.[21] Due to the fact that cortical S1 changes are correlated with mechanical hyperalgesia, at least in acute CRPS, we suggest that cortical reorganization may be induced by activation or sensitization of these particular C-fibers. The mechano-insensitive C-fibers usually are silent.[20] They were "woken-up" (meaning spontaneously active or sensitized to forthcoming stimuli) by peripheral changes after trauma. Thus, these fibers appear to be the most peripheral substrates of neuronal plasticity in the nociceptive system, which finally leads to CRPS if plastic changes propagate to the CNS. If it is possible to desensitize these C-fibers by CRPS therapy, peripheral symptoms ameliorate and central changes were reversed. In chronic CRPS, however, when axonal damage and deafferentiation become more important, common peripheral therapy often is ineffective and thereby CNS changes may become irreversible and chronic. Our studies suggest that cortical reorganization in acute CRPS may be reversible following successful therapy. Therefore, strategies interfering with maladaptive plasticity may be promising new treatment approaches for this disease.[22-24]

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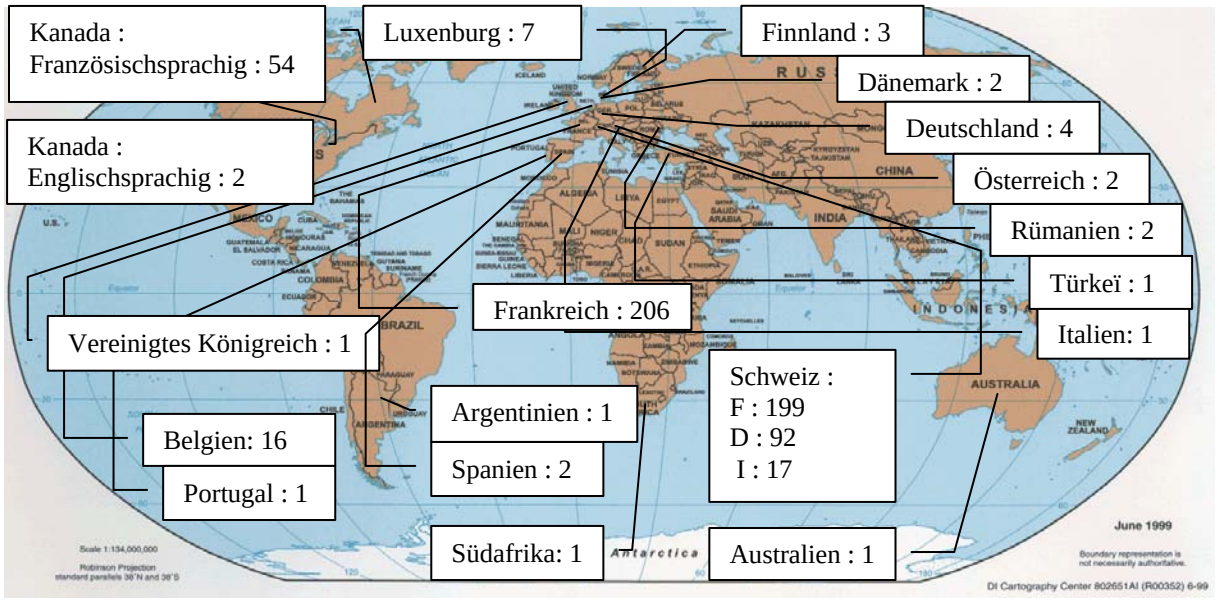
Somatosensorische Schmerzrehabilitation TherapeutInnen in der Welt

To MD. To neuroscientist To patient To therapist

1992 gab es die erste Mitteilung über somatosensorische Schmerzrehabilitation anlässlich des ersten Kongresses SGHR (Schweizerische Gesellschaft für Handrehabilitation). Das erste Mal wurde diese Methode 2001 unterrichtet. Bis zum 23. November 2010 erreichten wir mit unserer Weiterbildung zur somatosensorischen Rehabilitation 500 TherapeutInnen/Ärzte. Bei dieser Gelegenheit wollen wir die neue Rubrik eröffnen, um darzustellen, wo all die ausgebildeten Therapeuten mit ihrer Praxis domiziert sind.



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4	Kanada : Französischsprachig	54
5	Schweiz : Italienischsprachig	17
6	Belgien : Französischsprachig	16
7	Luxenburg	7
8	Deutschland	4
9	Finnland	3
10	Kanada : Englischsprachig	2

11	Dänemark	2
12	Österreich	2
13	Rumänien	2
14	Spanien	2
15	Italien	1
16	Vereinigtes Königreich	1
17	Türkei	1
18	Südafrika	1
19	Australien	1
20	Argentinien	1
21	Portugal	1

TOTAL 611

Participant Point of View No 1

«3rd WEEK FOR SOMATOSENSORY REHABILITATION
7-10.3.2011, SWITZERLAND»

To MD. To neuroscientist To patient To therapist

Nikolajew Sari M., PT[9]

Last autumn, our handsurgeon gave me a tip, which could be a useful education for my work with hand- and painpatients. I went to the address of the website and the content was interesting. Then I forgot about it. In January 2011 I remembered that education and decided to go to Switzerland. I contacted the course organizers; they had the e-mail addresses of two occupational therapists in Finland, who had assisted to the course the previous year. So I could take some advice for both, the journey and the lodging in the city. After a little bit of time on Internet, the flights and hotel were reserved.

The course was organized in the city of Friburg. This is where the Somatosensory Rehabilitation Center is located. It is easy to go to Friburg, from Finland by plane to Geneva, Bern, or Zürich, and is then easily accessible by train. I took a flight to Geneva, and took the

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train to Friburg. It is a 1.5 hour ride. At the course, there were 13 participants, 8 (7 OT, 1 PT) from Switzerland, 1 OT from Argentina, 1 PT from Australia, 1 MD from Turkey, 1 engineer from Germany and 1 PT from Finland. The teachers were the method developer, Claude J. Spicher, and an OT from the Somatosensory REhabilitatin Centre: Rebekah Della Casa. The course language was English, but some parts of the teaching were carried out in two groups, one in German and one in English.

The teaching was done between 9 a.m. - 5 p.m., plus a lecture on the first evening, that only part of the group was able to attend – Now a few words about the content of the course:

Somatosensory Rehabilitation is a method, which aim is to increase the quality of touch or even normalize the sensation of touch. Because, when hypoesthesia decreases, neuropathic pain decreases. Regular and rigorous assessment of the quality of hypoesthesia in terms of pressure perception threshold is an important part of this rehabilitation.

Sometimes, the hypoaesthetic territory – aesthesiography- is masked by a patch of skin which is painful to touch and is therefore not accessible. Since 1979, this stimulus induced pain is to be called allodynia in medicine. The original definition comes from Merksey and Bogduk (1994) ”Pain due to a stimulus witch does not normally provoke pain”. In such situations, while doing the diagnostic testing of axonal lesions at the first occupational or physical therapy session, the two point discrimination test is impossible.

The presence of mechanical allodynia hinders other physical treatments. For the reason that, any contact on the hypersensitive territory, although it can be bearable on the moment, can induce several hours of a very painful post-effect or even several sleepless nights. This hypersensitivity to touch is induced by the peripheral nerve lesion of the large myelinated A-beta fibers.

In other words, after a peripheral nerve leasion, aberrant sprouting can occure in the dorsal horn which can explain that a non-noxious stimulus is perceived as being noxious. This is one of the explanatory models of the different mechanisms of central sensitization.

We practiced the method testings and drew the maps on millimeterpaper. We learned to use the McGill Pain Questionnaire. We got information about hyposensitivity, mechanical allodynia, CRPS-patients in rehabilitation, desensitization by mechanical vibrations and followed, in small groups, patient treatments. In the evening we had free conversation, with local delicacies. The program was known, the teaching room had sufficient space for each participant, the experiences of therapist from different countries, was valuable to creat a network. Fortunately, the Handbook was included in the course as well as the just published: Atlas of cutaneous territories.

The price was EUR 763, which included teaching, breaks and snacks (which were really rich), as well as the handbook and the atlas. I recommend the course for those who already work with cutaneous sense or/and pain patient and otherwise interested on the topic and the

treatments. The method can only be currently certified in French. The 4th Week for Somatosensory Rehabilitation will be take place from the 5th of March 2012 until the 8th of March 2012. You can see more information on the continuous education link of the university of Fribourg.

Finally I would like to thank our long-suffering and inspiring teachers Rebekah Della Casa and Claude Spicher. Special thanks to the patients, who shared their rehabilitation times with us.

Participant Point of View No 1

«4. Kurs i somatosensorisk rehabilitation d. 7- 10 marts 2011»

To MD To neuroscientist To patient To therapist

Viime syksynä käsikirurgimme antoi minulle vinkin kurssista, josta saattaisi olla hyötyä työssäni käsi- ja kipupotilaiden kanssa. Kävin saamani osoitteen sivuilla ja sisältö vaikutti mielenkiintoiselta, asia jäi muhimaan. Vuodenvaihteen jälkeen kurssi alkoi askarruttaa minua uudelleen ja päätin lähteä katsomaan mitä maailmalla on tarjolla. Otin yhteyttä kurssin järjestäjään, hänellä oli yhteystiedot edellisenä vuonna, Suomesta olleille toimintaterapeuteille. Sain sähköpostiosoitteet, jotta voisin kysyä kurssista lisää sekä matkaa ja hotelleihin liittyviä vinkkejä. Kävi ilmi että kyseessä olivat kurssikaverini, muutaman vuoden takaiselta yläraajan kuntoutumisen – erikoistumisopinnoilta, Oulusta. Pientä selailua internetin ihmeellisessä maailmassa ja lennot sekä hotelli oli varattu.

Kurssi järjestettiin Friburin kaupungissa, jossa sijaitsee Somatosensory Rehabilitation center. Friburiin on helppo mennä Suomesta. Lentäen Berniin, Geneveen tai Zyrichiin, kaikista pääsee helposti junalla kaupungin ytimeen. Itse lensin Geneveen, josta junalla siirtymä oli 1,5 tuntia. Saavuin kurssia edeltävänä iltana pimeässä Friburiin. Aamulla paljastui kaupungin kauneus, sillä hotellini sijaitsi keskiaikaisessa kaupunginosassa.

Kurssillamme oli 13 osallistujaa, 8 Sveitsistä (7 tt, 1ft), 1 tt Argentiinasta, 1 ft Australiasta, 1 lääkäri Turkista sekä 1 ft Suomesta. Kurssin vetäjinä toimivat aivan loistavat toimintaterapeutit, metodin kehittäjä Claude Spicher sekä Rebekah Della Casa. Kurssikielenä oli englanti, mutta osa ryhmäopetuksesta toteutettiin Saksan ja Englannin kielellä.

Päivät olivat pitkiä, 9-17, jonka jälkeen oli vielä ulkopuolisia luentoja. Paikan päällä kykeni omaksumaan vain pienen osan. Päivien sisältö oli luotu erinomaisen vaihtelevaksi.

Somatosensory Rehabilitation on metodi, jossa on viisi menetelmää. Sen tarkoituksena on testata, ennalta ehkäistä sekä kuntouttaa ihotunnon häiriöitä. Kuuntelimme luentoja, jossa kävimme läpi käsitteitä liittyen kipuun, tuntoon sekä metodiin. Harjoittelimme metodiin kuuluvia testejä ja piirsimme millimetripaperille karttoja(aesthesiography, allodynogrphy, Rainbow Pain Scale). Perehdyimme McGill kipu kyselyyn. Saimme tietoa hyposensitiivisen, mekaanisen allodynia, crps-potilaiden kuntoutuksesta, tunnon uudelleen kouluttamisesta mekaanisen vibraation avulla sekä pääsimme seuraamaan pienissä ryhmissä potilastilanteita.

Lisäksi iltaisin oli vielä vapaata keskustelua paikallisia herkkuja maistellen. Tuli tunne että kurssilla oli riittävästi tilaa ihmisten omille kokemuksille ja niitä arvostettiin, eri maissa toteutuvien käytäntöjen vaihdolle sekä verkostoitumiselle. Onneksi kurssiin sisältyi käsikirja sekä juuri julkaistu atlas, ihon territorioista.

Kurssin hinta oli 763 euroa, joka piti sisällään opetuksen, aamiaiset ja välipalat(jotka olivat todella runsaat), sekä edellä mainitsemani käsikirjan sekä Atlaksen. Suosittelen kurssia niille, jotka työssään ovat tekemisissä aiemmin mainitsemieni ihotunnon häiriöiden kanssa tai muuten kiinnostuneet kivusta ja sen hoidoista. Metodiin voi tällä hetkellä sertifioitua vain Ranskassa, mutta seuraava eli 4th Week Somatosensory Rehabilitation järjestetään 5-8.3.2012, lisätietoja saat esimerkiksi yhdistyksen sivuilla olevan linkin kautta.

Lisäksi haluan kiittää kärsivällisiä sekä innostavia opettajiamme Rebekkah Della Casa:a sekä Claude Spicher: ä sekä erityiskiitos potilaille, jotka olivat valmiit jakamaan kuntoutustilanteitaan kanssamme.

Certificat en rééducation sensitive de la douleur

To MD. To neuroscientist To patient To therapist

En été 2010,

Après 16 ans de communication (n = 215),

Après 10 ans de cours de 2, 4, voire 8 jours (n = 34),

La 1ère volée du **Certificat CREA - Haute Ecole Libre de Bruxelles (HELB) en rééducation sensitive de la douleur** a vu le jour, sous l'égide de M. Pierre Castelein. Merci.

Cette formation compte 56 heures, ou équivalents.

Lauréates et lauréats de la 1ère volée 2010 du Certificat CREA - Haute Ecole Libre de Bruxelles (HELB) en *Rééducation sensitive de la douleur* (par ordre alphabétique) :

- Cynthia Lathion, Montana,
- Irene Inauen, Rheinfelden,
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- Nadège Desfoux, Fribourg,
- Nathalie Drezet-Munch, Genève,
- Paolo Signorino, Bruxelles,
- Pascal Latière, Genève,
- Rebekah Della Casa, Fribourg,
- Sandrine Clément-Favre, Fribourg,
- Séverine Landreau, Fouquière-les-Lens.

Expertisé par **Dr Mélanie Kaeser (PhD), Research Associate, Unit of Physiology and Program in Neurosciences, Department of Medicine, University of Fribourg, CH**

N'hésitez pas à vous inscrire pour devenir Lauréate de la 2ème volée !!!

Vous découvrez depuis l'été 2010, *e-News 7*(3), leur travail final de Certificat.

Fait clinique

To MD. To neuroscientist To patient To therapist

Névralgie dorso-intercostale incessante avec allodynie mécanique: Fait clinique d'une diminution des douleurs neuropathiques chroniques par la normalisation de la sensibilité cutanée.

ABSTRACT

Des lésions axonales d'un nerf cutané peuvent être à l'origine de douleurs spontanées survenant plusieurs années après un épisode traumatique ou infectieux. Le territoire de distribution cutanée, innervé par le nerf lésé, peut se modifier peu à peu, le toucher peut devenir « bizarre », désagréable, « émoussé », voire douloureux. Lors d'une prise en charge en rééducation sensitive, il est essentiel, dans un premier temps, de déterminer la branche cutanée du nerf lésé à l'aide du questionnaire de la douleur de St Antoine (QDSA), de cartographier le territoire hypoesthésique ou allodynique et de mettre en place le traitement en proposant la technique de traitement adéquate, visant à atténuer, voire supprimer les symptômes neurogènes. Lors d'une allodynie mécanique, il faut cependant attendre d'avoir accès au territoire hypoesthésique sous-jacent pour le traiter et voir ainsi diminuer les douleurs neuropathiques spontanées.

Mots clés :
Allodynie mécanique, lésions axonales, contre-stimulation vibrotactile à distance, hypoesthésie, douleurs neuropathiques.

Clément-Favre[10], S. lauréate de la 1ère volée du Certificat CREA - Haute Ecole Libre de Bruxelles (HELB) en *Rééducation sensitive de la douleur*

Expertisé par **Dr Mélanie Kaeser (PhD), Research Associate, Unit of Physiology and Program in Neurosciences, Department of Medicine, University of Fribourg, CH**

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e-mail : reeducation.sensitive@cliniquegenerale.ch

INTRODUCTION

Un territoire cutané initialement hypoesthésique peut devenir allodynique, névralgique ou les deux à la fois (1).

Dans 87% des cas, l'herpès du zona génère des allodynies mécaniques (2) sur le territoire de distribution cutanée de la branche sensitive lésée, voire même en dehors de celui-ci.

Le but de ce fait clinique est de montrer que sous l'effet de stimulations cutanées ciblées, la récupération de la sensibilité cutanée et la diminution de la symptomatologie neurogène sont possibles, plusieurs années après la lésion.

PATIENT & METHODES

Anamnèses générale et clinique

M. G., 75 ans, chef monteur-électricien retraité, nous est adressé le 17.09.2009 pour des douleurs thoraciques chroniques (42 mois). M. G. a eu un épisode de zona en 2003. Le patient est fortement gêné pour manger, se doucher, s'habiller, marcher et ne peut plus aller à la pêche, aux champignons. Les plaintes douloureuses sont localisées en zone dorsale au niveau du ganglion rachidien [arrivée du 10ème nerf thoracique (TH10)] et sur la face antérieure du tronc. Le QDSA oscille entre **34 et 41 points**, avec des sensations d'« engourdissement », d'« irradiation » et de « brûlures ». M. G. est très sensible au moindre effleurement de sa peau.

L'hypothèse de lésions axonales est :

Névralgie dorso-intercostale incessante de la branche perforante antérieure du 10 ^{ème} nerf thoracique avec allodynie mécanique (stade IV de lésions axonales).

Evaluation somesthésique

Le 17.09.2009, un territoire allodynique est mis en évidence par une allodynographie (Fig. 1) avec un arc-en-ciel des douleurs INDIGO (l'application de **8,7 grammes** augmente l'échelle visuelle analogique (EVA) de 1 cm).

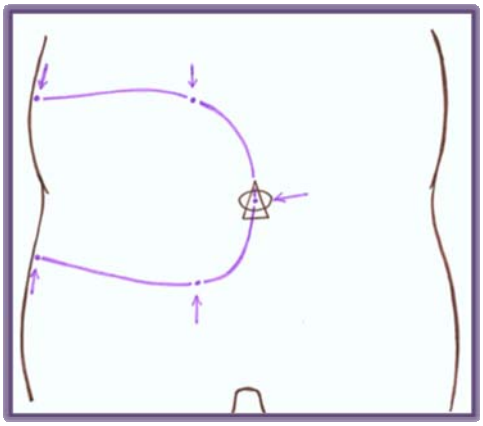

Fig. 1 : *Allodynographie à 15,0 grammes (esthésiomètre de Semmes-Weinstein 5.18) de la branche perforante antérieure du 10ème nerf thoracique, sur la face antérieure du tronc, le 17.09.2009, avec une EVA à 5+1=6 / 10 cm.*

Méthodes de traitement

1. Temps allodynique : contre-stimulation vibrotactile à distance, 8 fois par jour pendant 1 minute, sur la zone confortable (zone antérieure de travail : en dessus de TH5)
2. Rééducation de l'hyposensibilité sous-jacente au moyen de la thérapie du touche-à-tout de manière progressive sur le territoire hypoesthésique sous-jacent, dès le 29.09.2009.

RESULTATS

Le 29.09.2009, après 12 jours de contre-stimulation vibrotactile, l'allodynie mécanique a disparu, laissant place à une hypoesthésie sous-jacente qui s'est normalisée de la manière suivante (Tableau I) :

Dates	**Somesthésie : hypoesthésie sous-jacente**		**Douleurs neuropathiques**
	Seuil de perception à la pression (SPP)	**Test de discrimination deux points statiques (2pts)**	**Questionnaire de la douleur St-Antoine (QDSA)**
29.09.2009	2,9 g	Non déterminé	34 à 41 points (le 17.09.2009)
05.10.2009	Non déterminé	48 mm	Non déterminé
12.10.2009	0,9 g	41 mm	Non déterminé
26.10.2009	0,4 g	35 mm	30 à 36 points
02.11.2009	**0,3 g**	25 mm	Non déterminé
09.11.2009	Normalisé	Non déterminé	6 à 22 points
16.11.2009	Normalisé	**21 mm**	Non déterminé
22.11.2009	**Normalisé**	**Normalisé**	**0 à 14 points**

Tableau I : *Diminution de l'hypoesthésie sous-jacente corrélée avec la diminution des douleurs neuropathiques.*

DISCUSSION

Les patients présentant une allodynie mécanique utilisent souvent le terme d'hypersensibilité au toucher. Dans une étude (3), il a été montré chez des patients ayant des douleurs neuropathiques chroniques et traités par rééducation sensitive, que chacune des allodynies masquait une hypoesthésie sous-jacente, provoquées par des lésions de fibres aβ. L'hypersensibilité au toucher est donc une hypoesthésie douloureuse (1). Chez M. G., la rééducation de l'hypoesthésie sous-jacente – basée sur la neuroplasticité du système somesthésique – a permis de voir diminuer les symptômes neurogènes. Parallèlement à l'amélioration régulière de la somesthésie, le score au QDSA a diminué progressivement de **41 à 14 points**, passant ainsi la barre des 20 points, signe d'une situation stabilisée (4). M. G. a pu reprendre petit à petit ses activités quotidiennes sans ou avec des douleurs « modérées ».

CONCLUSION

Lors de douleurs neuropathiques, le thérapeute se doit, dès le début de la prise en charge, de présumer le nom de la ou des branches nerveuses lésées et évaluer la sensibilité cutanée de manière rigoureuse, afin de pouvoir proposer les techniques de traitement appropriées à l'état somesthésique du moment. Pour voir diminuer les douleurs neuropathiques spontanées, il est essentiel de pouvoir accéder à la peau, donc de faire disparaître l'allodynie, puis de rééduquer la sensibilité cutanée, car « réveiller la peau, c'est endormir les douleurs neuropathiques» (5).

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Ombre et pénombre

To MD To neuroscientist To patient To therapist

Le silence, pour moi, est une condition indispensable pour le travail

"Le silence pour moi est une condition indispensable pour le travail, alors évidemment c'est un peu spécial de parler de silence quand on a un bruit de ponceuse, de meuleuse, tous ces bruits qui ponctuent la vie de l'atelier, mais à l'intérieur de soi-même, il faut avoir ce silence. Je veux dire, au moins deux fronts, si ce n'est plus, mais en tout cas il y : le front qui résonne, qui travaille avec le problème technique, c'est le mental, qui gère les problèmes qui arrivent au fur et à mesure, les dimensions, enfin des choses tout à fait pratiques, et puis tout le reste n'est qu'une écoute, une écoute où la forme va naître, autour de la naissance de cette forme, il faut pour moi du silence, c'est-à-dire, que cette vie, qui apparaît petit à petit, sorte de l'inconnu, et cet inconnu doit avoir pour moi, ni son, ni forme, rien : c'est un grand vide qui doit être habité de silence."

Extrait du DVD :

de Riedmatten, E. (2008) – Christine AYMON, un portrait. Verossaz:
christine.aymon@bluewin.ch

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Somatosensory Rehabilitation Centre's Statistics

To MD To neuroscientist To patient To therapist

From the 1st of July 2004 until the 24th of May 2011, **1441 patients** have been assessed.

	136 patients with somatosensory disorders without pain (Stage I)			
		277 patients who were only assessed		
			301 patients who have interrupted their treatment	
				90 patients still on treatment
1441 patients assessed	1305 chronic painful patients assessed	1028 chronic painful patients treated	728 chronic painful patients who have not interrupted their treatment	**637 patients who have finished their treatment**

Table I: ***637 chronic painful patients*** *who have finished their somatosensory rehabilitation have been included. 21 patients have been excluded from the statistics (17 not determined and 4 desactivations). Consequently,* ***616 patients****, with* ***1172 axonal lesions****, took part in the prospective study.*

Evolution of the Pressure Perception Threshold (PPT)

***n* = 1172 axonal lesions**
616 patients

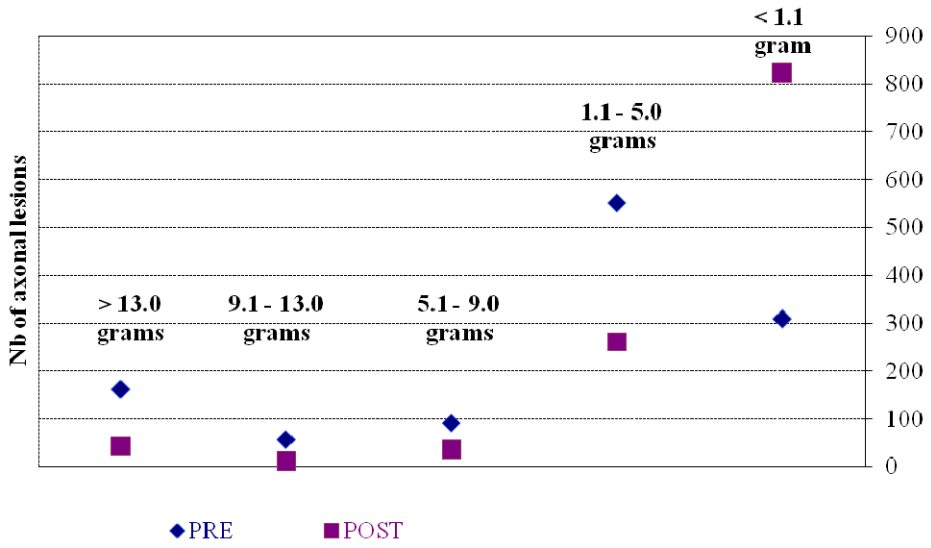

Fig. 1: *Distribution of the PPT values* ***before*** *&* ***after*** *somatosensory rehabilitation. Note: with the improvement of the perception ability, the undergroup "*POST: < 1.1 gram*" (n=823) includes axonal lesions from the 5* PRE *undergroups.*

Témoignage No 34 d'une patiente

«Bannir les pantalons et de ne porter que des robes ou des jupes»

To MD  To neuroscientist  To patient  To therapist 

En avril 2010, j'ai subi une laparoscopie et au réveil de l'opération j'ai immédiatement senti que je n'avais plus de sensibilité au niveau de la jambe droite.

Mon médecin était très étonné car selon lui ce problème ne lui était jamais arrivé avant. Il me disait que ça allait revenir et qu'il ne fallait pas s'inquiéter, d'une part parce que j'avais de la force dans la jambe et d'autre part parce qu'à ce moment là je n'avais pas de douleurs. De plus, il ne comprenait pas pourquoi j'aurais pu perdre de la sensibilité au niveau d'un membre inférieur alors qu'il m'avait opéré au ventre.

Au deuxième jour d'hospitalisation, j'étais contente car je ressentais des picotements et je pensais que finalement tout allait rentrer dans l'ordre. Mal m'en a pris d'avoir eu l'audace de me réjouir si vite. Les picotements étaient le commencement de douleurs indescriptibles. Elles sont arrivées aussi brutalement qu'inattendues. Piqûres, brûlures, déchirures, coups de poignard et j'en passe….. Et toujours pas de sensibilité sur toute la cuisse.

C'était vraiment paradoxal comme situation, j'avais mal et n'avais pas de sensibilité au touché. J'expliquais ça aux infirmières, à mon médecin ou à mon entourage et je voyais bien qu'on me regardait bizarrement. Dans l'esprit des gens, on ne peut pas avoir mal et ne rien sentir en même temps. C'était une situation qui n'était pas « logique ».

Trois semaines plus tard, mon médecin m'a prescrit des vitamines B9 « Elevit ». J'ai suivi son traitement et en parallèle j'ai essayé l'acupuncture. Rien n'a marché. Les douleurs étaient toujours aussi intenses et je commençais à fatiguer, à avoir le moral en baisse.

D'habitude on dit de moi que je suis un «petit rayon de soleil », mais petit à petit je suis devenue malgré moi plus irritable et à fleur de peau. Je repoussais même ma fille dès qu'elle voulait venir me faire un câlin trop près, de peur qu'elle me touche ou plutôt qu'elle frôle ma jambe. Je ne pouvais plus la prendre sur mes genoux et ça c'était très dur pour moi. Même la nuit je ne supportais plus mon duvet et je ne pouvais pas dormir sur le côté droit.

Nous sommes partis en vacances en juillet en Turquie et je n'ai pas pu nager. La pression de l'eau me faisait trop mal. A mon retour, à force de répéter à mon médecin que j'avais mal et toujours pas de sensibilité, il m'a envoyé chez un neurologue début août. Avant d'y aller j'ai quand-même voulu savoir ce qu'il s'était passé durant l'opération pour que tout ça m'arrive ou du moins pour essayer de comprendre. Rien, pas de réponse, j'attends toujours… Pour lui il ne s'est rien passé, il ne comprend pas il a cherché des explications où il n'y en avait pas (une év. hernie discale), donc je ne saurais jamais le fin mot de l'histoire et pourtant à ce moment là j'aurais juste eu besoin qu'il me dise c'est arrivé, « on » s'excuse.

Bref, j'étais contente d'aller chez le neurologue, il pourrait sans doute faire quelque chose pour moi. Il faut rester positif dans la vie. Il m'a fait quelques tests et m'a dit que j'avais une méralgie paresthésique du nerf fémoral et qu'il n'y avait rien à faire, sauf à aller mettre un

cierge à Bourguillon et prier bien fort. Voilà, le moral redescend au plus bas, vous repartez de là en prenant votre voiture, en mettant votre ceinture de sécurité qui fait très mal car elle appuis sur l'aine une partie de votre corps également douloureux et vous vous dites que vous devrez vivre avec et essayez d'apprivoiser cette douleur mais que ce n'est pas gagné.

Mes seuls soutiens pendant ces moments pénibles ont été mon compagnon et ma sœur. Les autres ne comprenaient toujours pas. Ils me disaient, tu sais, tel avait la même chose et ça a passé après 1 mois, mais moi, je m'en fichais des autres, c'est moi qui souffrais, c'est mon corps qui était blessé et chacun est différent. Et qui me disait que l'autre avait la même chose ?

Je sais au fond de moi que je n'ai pas trop le droit de me plaindre, il y a bien pire que moi. Des gens qui souffrent beaucoup plus. J'en ai côtoyé pas mal quand je faisais ma radiothérapie au CHUV il y a environ 8 ans et ça m'a appris à être plus forte et à ne pas trop me plaindre. Alors j'essayais de faire bonne figure devant les autres, à ne rien laisser paraître quand la douleur était plus forte ou quand on me touchait la cuisse.

Mon ami était en traitement chez le chiropraticien Aymon et je lui ai demandé de lui posé la question s'il pensait pouvoir faire quelque chose pour moi. Un rdv a été pris et dès le 1er contact il m'a de suite parlé de Monsieur Spicher et prit un rdv au Centre de rééducation sensitive de la Clinique Générale.

Enfin une lueur d'espoir à mon entretien lorsque Monsieur Spicher m'a dit qu'il y avait quelque chose à faire pour ma jambe! Mais ce qui m'a le plus surprise c'est qu'il connaissait tout de mes douleurs avant même que je lui en parle. Il pouvait me les décrire comme si c'était lui qui les subissait, c'était dingue ! Trop fort….. Par contre quand il m'a expliqué que je devais utiliser une peau de lapin pour commencer la thérapie, là j'ai hésité entre piquer un fou rire ou partir en courant.

Ensuite en lisant les newsletters, j'ai mieux compris les techniques utilisées par les thérapeutes pour dans un premier temps diminuer les douleurs pour pouvoir ensuite traiter la sensibilité de la peau.

Monsieur Spicher m'a conseillé de bannir les pantalons et de ne porter que des robes ou des jupes ! Encore une « torture » de plus pour moi. Je n'en portais jamais ou très rarement ! Il m'a fallu 10 jours pour me décider à aller faire les boutiques avec ma sœur. C'était la galère ! On ne s'imagine pas à quel point un problème aussi bénin que vestimentaire peut psychologiquement être aussi dur. Bref, je pouvais bien mettre une jupe le week-end, mais au travail c'était quelque chose d'insurmontable pour moi. Je l'ai quand-même fait et les résultats ne se sont pas faits attendre, sans trop de pression sur ma jambe j'avais moins mal et surtout moins de décharges électriques. En parallèle j'utilisais ma peau de lapin sur une zone « saine » proche de ma jambe.

Madame Della Casa m'a expliqué que je ne devais plus toucher ma jambe et que lorsqu'elle me grattait, ce qui m'arrivait souvent, il fallait gratter la peau de lapin ou autre chose (souvent le dos de mon fils). Même sous la douche il était conseillé de ne pas toucher la zone sensible. En résumé, il était important de faire comme si cette partie du corps n'était pas là. Ca n'a pas toujours été facile d'ignorer cette cuisse, mais j'ai appliqué au mieux ces règles.

Dès que les douleurs ont bien diminué, nous avons commencé à toucher la cuisse. C'était de nouveau très surprenant comme technique. J'ai pu choisir les tissus que j'emploierais pour la

toucher. Avec l'aide de M. Spicher j'ai choisi un foulard, une patte pour essuyer les lunettes et un mouchoir en papier. Le traitement consistait au début à passer le tissu sur les zones définies. Peu de temps et assez souvent et plus le traitement avançait, le temps se prolongeait mais je devais le faire moins souvent. A chaque rendez-vous on contrôlait si la sensibilité revenait et en même temps je sentais bien que les douleurs qui n'avaient pas complètement disparues avec la peau de lapin diminuaient de fois en fois.

Et voilà, la fin du traitement est arrivée en mars 2011. Grâce à Monsieur Spicher et à Madame Della Casa, j'ai enfin retrouvé MA jambe, c'est vraiment le jour et la nuit entre ma première visite à la Clinique Générale et aujourd'hui. Je les remercie vivement pour tout ce qu'ils ont fait pour moi et leur dédie un petit rayon de soleil pour ce commencement du printemps.

Coralie Gobet

[Vous pouvez lire ci-dessous, le No Comment N^{o} 23 sur cette patiente qui souffrait d'une névralgie crurale avec allodynie mécanique depuis 6 mois, lors de sa première séance de rééducation sensitive.]

No Comment N° 23

To MD. To neuroscientist To patient To therapist

Della Casa, R. (OT, ST certified HELB), Aymon M. (DC) & Spicher, C.J. (BSc OT)

Lors de l'évaluation initiale effectuée au Centre de rééducation sensitive de Fribourg le 6 octobre 2010, Madame G., 36 ans, présentait des douleurs neuropathiques **depuis 6 mois**.

Status post compression du N. fémoral avec hyperalgésie dermatome L2-L3 ; traitement manuel lombaire local L3-L4.

Diagnostic somesthésique:

Névralgie crurale permanente des branches cutanées antéieures du nerf fémoral avec allodynie mécanique (Stade IV de lésions axonales)

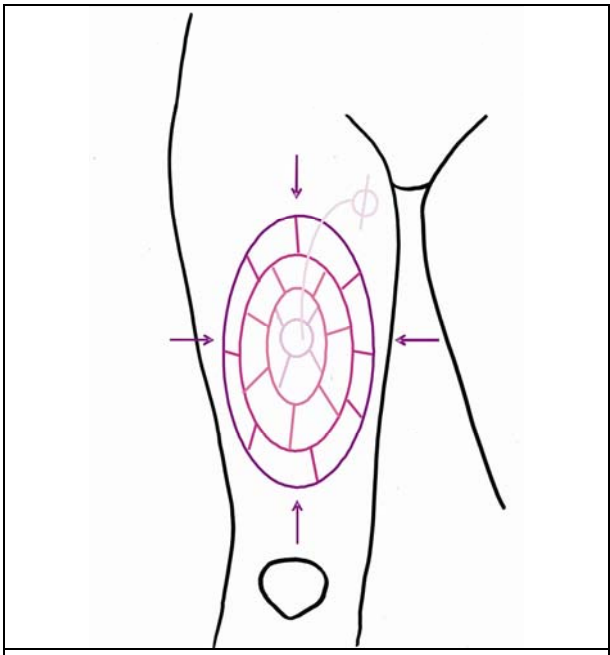

Fig. 1 : *Allodynographies successives à **15,0 grammes** des branches cutanées antérieures du nerf fémoral, effectuées du 6.10.2010 (t_0) au 6.1.2011 (t_{92}), sur la face antérieure de la cuisse.*

L'allodynographie circonscrit le territoire allodynique: la portion de peau où l'application de 15 grammes est perçue comme douloureuse (EVA : 3/10 cm). Les flèches indiquent les axes le long desquels la perception douloureuse de 15,0 grammes a été testée. Les points indiquent la localisation où l'application de l'esthésiomètre de 15,0 grammes (marque 5.18) est perçue de manière nociceptive.

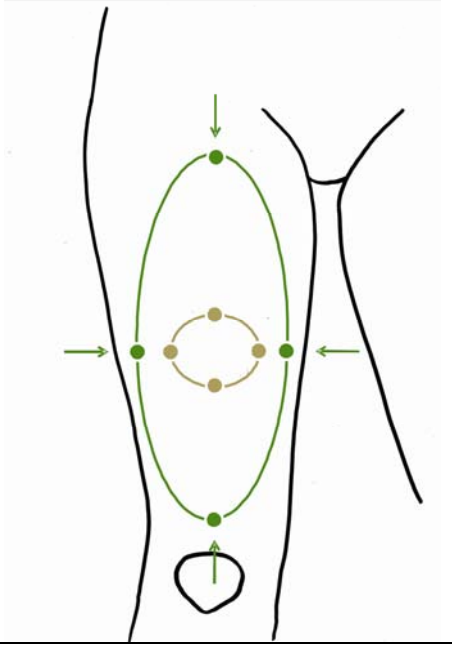

Fig. 2 : *Esthésiographies secondaires successives à **0,7 gramme** des branches cutanées antérieures du nerf fémoral effectuées du **6.1.2011** (t_{92})* au *18.2.2011 (t_{136}), sur la face antérieure de la cuisse. L'esthésiographie secondaire circonscrit le territoire hypoesthésique donc la portion de peau où cet esthésiomètre n'est pas détecté. Les flèches indiquent les axes le long desquels il est appliqué.*

Les points indiquent l'endroit où l'application de l'esthésiomètre de 0,7 gramme (marque 3.84) n'est pas détectée.

Date	Douleurs neuropathiques	Somesthésie				Stade
	Questionnaire de la douleur St-Antoine	Territoire de distribution cutanée	Arc-en-ciel des douleurs	SPP	2 pts	
06.10.2010	40 à 73 points	Allodynie	VERT	Intestable	Intestable	IV
14.12.2010	17 à 59 points	Allodynie	BLEU	Intestable	Intestable	III
28.12.2010	6 à 53 points	Allodynie	INDIGO	Intestable	Intestable	III
06.01.2011	Non déterminé	Hypoesthésie sous-jacente (Fig. 2)	Ø	2,1 g	Non déterminé	III
11.01.2011	Non déterminé	Hypoesthésie sous-jacente	Ø	Non déterminé	77 mm	III
21.01.2011	3 à 41 points	Hypoesthésie sous-jacente	Ø	1,5 g	Non déterminé	III
28.01.2011	Non déterminé	Hypoesthésie sous-jacente	Ø	Non déterminé	56 mm	III
11.02.2011	2 à 19 points	Hypoesthésie sous-jacente	Ø	Non déterminé	41 mm	I
18.02.2011	Non déterminé	Hypoesthésie sous-jacente	Ø	1,4 g	Non déterminé	I
25.02.2011	Non déterminé	Hypoesthésie sous-jacente	Ø	0,5 g	Non déterminé	I
18.03.2011	0 à 3 points	Hypoesthésie sous-jacente	Ø	Normalisé	30 mm	I

Tableau I : *La diminution des douleurs neuropathiques est corrélée avec la disparition de l'allodynie mécanique, puis avec la diminution de l'hypoesthésie sous-jacente.*
SPP : seuil de perception à la pression ; 2 pts : test de discrimination de 2 points statiques

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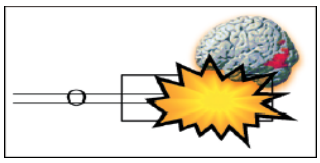

Date: 5-8 March 2012

4th Week for Somatosensory Rehabilitation

Claude Spicher, BSc OT, swiss certified Hand Therapist
Rebekah Della Casa, OT
Isabelle Quintal, BSc OT

Place : Somatosensory Rehabilitation Centre, Fribourg**,** Switzerland, Europe

Info : This *issue 8*(3) **page 133**

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Certificat en rééducation sensitive de la douleur

VIII^{ème} COURS :

Le traitement des syndromes douloureux neuropathiques par la rééducation sensitive de la douleur

Troubles de base I & II, Complications douloureuses I & II

Lieu : Institut de Formation en Ergothérapie, Montpellier, France

Info : http://www.ergotherapiemontpellier.com/formation.html

Ces formations peuvent être comptabilisées pour :
le Certificat en rééducation sensitive de la douleur

19–20 September 2011	**Satellite Symposium** **Structural Plasticity and Reorganization in Chronic Pain**
Place	Heidelberger, Germany
Info	www.painandstructuralplasticity.de conference@pharma.uni-heidelberg.de

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20–23 September 2011	**22nd Annual Clinical Meeting of** **American Academy of Pain Management**
Place	Las Vegas, USA
Info	jillian@aapainmanage.org

21–24 September 2011	**7th Congress of EFIC** European Federation of IASP® Chapters
Place	Hamburg, Germany
Info	http://www.efic.org/index.asp?sub=QFIY5RWIb074C4

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17 - 18 octobre 2011	**Certificat en rééducation sensitive de la douleur :** **Module 1: Troubles de base I & II**
Lieu	Québec, Province de Québec, Canada
Info	info@mouvementsante.com;

20 - 22 octobre 2011	**Module 2 : Complications douloureuses I, Analyse de pratique & Anatomie clinique I**
Lieu	Hôtel Holyday Inn Express, St-Hyacinthe, Québec, Canada
Info	info@mouvementsante.com;

16–18 novembre 2011	**Certificat de rééducation sensitive : module 3** **Gestion du lien thérapeutique, Anatomie clinique II & Complications douloureuses II**
Lieu	CREA-HELB, Campus ERASME, Bruxelles
Info	www.crea-helb.be / crea@helb-prigogine.be www.anfe.fr / sfc.secretariat@anfe.fr

17–18 novembre 2011	**45ème Congrès annuel SSCM** **13ème Congrès suisse SSRM**
Lieu	Palais des Congrès, Bienne
Info	http://www.congress-info.ch/sgh-sghr2011/?l=2

25 Novembre 2011	The NSF for Long-Term Conditions: Five Years On British Society of Rehabilitation Medicine
Place	Royal College of Physicians, London
Info	http://events.rcplondon.ac.uk/details.aspx?e=1758 conferences@rcplondon.ac.uk

4 - 6 February 2012	**6th Congress World Institute of Pain**
Place	Miami Beach, FL, USA
Info	wip@kenes.com http://www2.kenes.com/wip/Pages/Home.aspx

27 - 30 June 2012	**15th World Congress of Pain Clinicians**
Place	Granada, Spain
Info	http://www2.kenes.com/wspc/Pages/home.aspx?ref2=db1

9-11 mai 2012	**Certificat en rééducation sensitive de la douleur: module 2 Complications douloureuses I, Analyse de pratique & Anatomie clinique I**
Lieu	CREA-HELB, Campus ERASME, Bruxelles
Info	www.crea-helb.be / crea@helb-prigogine.be www.anfe.fr / sfc.secretariat@anfe.fr

2–6 October 2012	**14th World Congress on Pain** International Association for the Study of Pain
Place	Yokohama, Japan
Info	http://www.iasp-pain.org/Yokohama

14–16 novembre 2012	**Certificat en rééducation sensitive de la douleur : module 3 Gestion du lien thérapeutique, Anatomie clinique II & Complications douloureuses II**
Lieu	CREA-HELB, Campus ERASME, Bruxelles, Europe
Info	www.crea-helb.be / crea@helb-prigogine.be www.anfe.fr / sfc.secretariat@anfe.fr

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