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EDITORIAL

Guest Editor Prof EM Rouiller, PhD

Neuroplasticity

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 blind human 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 associative visual 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, 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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Prof Eric ROUILLER, guest editor

http://www.unifr.ch/neuro/rouiller



« Somatosensory Testing and Rehabilitation » ALee Dellon Baltimore : The Institute for Peripheral Nerve

surgery, 2000. ISBN 1-56900-049-2

The first section, « basic principles », deals with the anatomy and the physiology of peripheral nerves and cutaneous sensory receptors, as well as with their response to injury. The responses of « free » nerve endings and of specific corpuscles (Meissner, Pacini, Merkel and others) are presented in more detail, with some hypotheses in regard to mechanisms of recovery after injury. An entire chapter in this section is dedicated to the pathways of normal and altered proprioception. Reflex sympathetic dystrophy is discussed with hypothesis regarding physiopathology and treatment guidelines. Techniques of nerve repair, including grafting and entubulation, are discussed. However, end-to-side repair is not mentioned, as this very new concept was probably not yet accessible at time of printing. The second section is about « quantitative sensory testing ». Tests of sensibility are discussed with special emphasis on their validity, standardization, and reliability. These results contribute to quantify impairment and disability. Methods to detect malingering are presented. Concepts of threshold and innervation density are discussed, including a very interesting historical perspective, leading then to the detailed exposition of a large variety of instruments. Electrodiagnostic tests though are briefly presented from a rather critical point of view.

In the third section, grading systems of function are presented, including modifications of the Highet scales and the author's own numerical grading system. The chapter about cortical plasticity is fascinating, as it is the basis for the understanding of sensory reeducation. This is the topic of the following chapter, including techniques of desensitization. Usefulness of sensory reeducation is demonstrated via a meta-analysis of results after repairs. Reeducation protocols from five different institutions are presented.

The fourth section focuses on the particularities of the upper and lower extremities, the head and neck, the breast, the penis, with normative data very useful for comparison in clinical situations. A chapter is dedicated to neuropathy including observations about double crush syndrome-, in various diseases like diabetes, leprosy and others. A further fascinating chapter deals with cumulative trauma disorders. Management of specific situations at work or with musicians is presented. Even more fascinating is the chapter about plegia, where traditional concepts of sensory losses in plegia are challenged by recent observations.

Finally, useful information is given to the reader willing to proceed with a research project. At the end, the reader may test his knowledge via a self-assessment questionnaire.

« Somatosensory testing and rehabilitation » brings together in a single volume basic neurosciences, pathology and physiopathology, precise techniques of testing and reeducation, and insight in further developments. Therefore this book is certainly a must for any health professional involved in the treatment of peripheral nerves and related disorders, be it a surgeon, a neurologist, a therapist or any interested person. The reunion of these different chapters in a single volume is a perfect example of the necessity for these professionals to investigate and understand each other's field, in order to eventually improve the care we can give to our patients.

Georges Kohut, MD,

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Des dates pour s'arrêter – A few dates to take a break – Ein paar Daten um eine Pause zu machen

7 - 8 octobre 2004	Expériences en ergothérapie, XVIIème série
Lieu	La Grande-Motte, Hérault, France.
Info	ergotherapiemontpellier@wanadoo.fr; ++33 4 67 10 79 99
28 - 29 octobre 2004	Congrès de la Société Suisse pour l'Etude de la Douleur
Lieu	Bienne, Suisse.
Info	<u>Programme (pdf)</u>
12-13 Novembre 2004	2 ^{ème} Congrès des Trois Pays pour la Rééducation de la Main.
Lieu	Bâle, Suisse.
Info	<u>n.gruenert-pluess@sghr.ch</u>
18 - 20 novembre 2004 Lieu Info	4 ^{ème} Congrès de la Société d'Etude et de Traitement de la Douleur Montpellier, France <u>http://setd-douleur.org</u>
Mardi 23 novembre 2004	Les mardis du Centre : <i>Forum en rééducation sensitive du corps humain.</i>
Lieu	Fribourg, clinique Ste-Anne, centre de rééducation sensitive.
Info	<u>reeducation.sensitive@ste-anne.ch</u> ; ++41 26 3500 312
5. – 6. April 2005	Somatosensorische Rehabilitation: Kurs I Claude SPICHER, OTR, CHT; Irène INAUEN, OTR, Past- Presidentin SGHR
Ort	Fribourg, (Switzerland)
Info	<u>www.fribourgtourism.ch</u> ; +41 26 3500 11 11
15-18 June 2005	VIII Congress of the European Federation of Societies for Hand Therapy. X Congress of the Federation of the European Societies for Surgery of the Hand
Lieu Info	Gothenburg, Sweden <u>handcongress2005@gbg.congrex.se</u> ; ++46 31 708 60 00 <u>www.congrex.com/handcongress2005</u>
6 - 10 avril 2005	9th Congress of the European Association for Palliative Care
Lieu	Aachen, Deutschland
Info	http://www.eapcnet.org/Aachen2005

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17-18 Juin 2005	1 ^{er} congrès de l'Association suisse des Ergothérapeutes
Lieu	Zürich, Suisse
	http://iiufpc01.unifr.ch:81/upload/annonce%20congres%20ASE
	<u>%202005.doc</u>
21 - 26 août 2005	11th World Congress of the European Association for Palliative
	Care 11 th World Congress on Pain
Lieu	Sydney, Australia
Info	IASP@locke.hs.washington.edu
11-15 March 2007	10 th Triennial Congress of the International Federation of
	Societies for Surgery of the Hand (IFSSH).
	7 th Congress of the International Federation of Societies for
	Hand Therapy (IFSHT).
Lieu	Sydney, Australia
Info	www.hands2007.com
	www.indido2007.0011

e-News for Somatosensory Rehabilitation Des collègues qui souhaiteraient le recevoir – Colleagues who would like to get it– Kollegen die es bekommen möchten

N'hésitez pas à communiquer à la rédaction les adresses e-mail des personnes susceptibles d'être intéressées par e-News for Somatosensory Rehabilitation.

Bibliographie - Reference --- Referenz

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Full text in line: http://iiufpc01.unifr.ch:81/upload/Sadato,NeurosciLett,2004.pdf

Their MRIf Figure: http://iiufpc01.unifr.ch:81/upload/La%20Fig%201.gif

Somatosensory Rehabilitation Centre's Statistics

The results of the 61 patients that have been assessed from the 1^{st} of July till the 25^{th} of July 2004 (see <u>http://iiufpc01.unifr.ch:81/upload/e-News1(1).pdf</u>):

61 patients

Stage I	Stage II, II, IV & CRPS II	N.D	Treatment interrupted
6	45	3	7

Assessment : from the 1 st of July till the 25 th of July 2004						
McGill pain que	McGill pain questionnaire (St. II, III, IV & CRPS II; N = 48 patients ; ND = 3)					
Min. – Max.	Mean	Median	SD			
6 – 75	40.8	40.5	17,5			
Distribution of the scores						
6-20	21	- 60	61 - 75			
10.4 %	75	5 %	14.6 %			

After 2 months of Somatosensory Rehabilitation					
McGill pain questionnaire (St. II, III, IV & CRPS II; N = 45 patients ; ND = 3)					
Min. – Max.	Mean	Mean Median SD			
5 – 77	31.7	25	19,5		
Distribution of the scores					
5-20	21	- 60	61 - 75		
30.3 %	60	.6 %	9.1 %		

Esthesiography N= 64				
1. Pressure Perception Threshold	Ø if PPT > 15 grams	N = 45		
2. Ø	Vibration Perception Threshold	N = 19		

Assessment : from the 1 st of July till the 25 th of July 2004					
Pressure Perception Threshold N= 45					
Min. – Max. Mean Median SD					
0.2 – 79.0 gf 13.4 gf 8.7 gf 17.5 gf					

After 2 months of Somatosensory Rehabilitation					
Pressure Perception Threshold N= 45					
Min. – Max. Mean Median SD					
0.1 – 13.9 gf 3.2 gf 1.7 gf 3.0 gf					

Assessment : from the 1 st of July till the 25 th of July 2004				
Vibration Perception Threshold N= 19				
Min. – Max.	Mean	Median	SD	
0.11 – 1.00 mm	0.46 mm	0.38 mm	0.29 mm	

After 2 months of Somatosensory Rehabilitation			
Vibration Perception Threshold N= 19			
Min. – Max.	Mean	Median	SD
0.10 – 0.92 mm	0.29 mm	0.17 mm	0.25 mm

Allodynography N= 25



An example of Rainbow Pain Scale blue on a Nervus cutaneus surae fibularis lesion

Stimulus: esthesiometer 3.6 grams

Reaction: Visual Analogue Scale 5 + 1 = 6 / 10 centimeter (the pain level before testing was at 5 / 10).



IMPRESSUM

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