INTRODUCCIÓN A LA NEURODINÁMICA CLÍNICA 1/2

GENERALIDADES

by @paco.bautista

Bautista

PRESENTACIÓN

- Importancia clínica mayor a la disfunción patomecánica del SN como fuente de síntomas musculoesqueléticos.

- Distintos estudios establecen la implicación del sistema neuromeníngeo en distinta patología de carácter crónico (cervicalgias, lumbalgias, epicondilitis, STC, etc).

- Puesta de manifiesto mediante los test neurodinámicos.

INTRODUCCIÓN

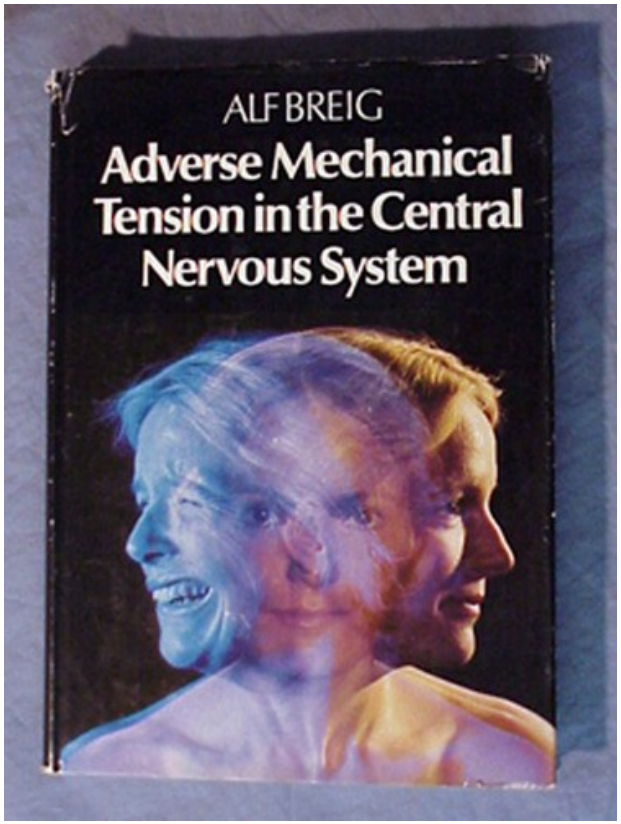
- Importancia de la irritación de los tejidos neurales en la producción de síntomas. (Grieve, 1970).
- Gran evolución en los últimos 50 años, tratados de neurodinamia y progresión clínica. (Butler 2002, Shacklock 2005).
- Integración de técnicas neurodinámicas en la neurociencia (Mosley y Butler, 2001).

INTRODUCCIÓN

- Primer termino acuñado para la disfunción neural “tensión neural adversa”.

- “Conjunto de respuestas fisiológicas y mecánicas anormales del tejido neural cuando se valora su amplitud de movimiento y capacidad de estiramiento normal”

(Alf Breig 1978)



INTRODUCCIÓN

- Los test de tensión valoraban sólo las capacidades mecánicas, no tenían en cuenta la fisiología.
- Sólo hacen referencia a una propiedades físicas, y aunque sirven para valorar el comportamiento mecánico, que va unido al fisiológico.
- Actualmente se denominan test neurodinámicos.

(Shacklock, 1995)

- Una tensión o compresión anómala del SN en cualquier parte de su trayecto puede causar disfunción neural y alterar su aporte vascular y ser fuente de los síntomas del paciente.

- IRRITACIÓN NEURAL, LESIÓN NEURAL MENOR, DISFUNCIÓN NEURAL

Manual Therapy (1998) 3(4), 187–194
© 1998 Harcourt Brace & Co. Ltd

Review article

Minor peripheral nerve injuries: an underestimated source of pain?

J. Greening, B. Lynn

Department of Physiology, University College London, London, UK

SUMMARY. **Chronic pain following partial nerve injury that is associated with objective signs of conduction loss presents few diagnostic difficulties. However, diagnosis of patients who present with diffuse symptoms in their limbs of unknown etiology is often a problem. Recent animal and human experiments suggest that diffuse painful symptoms may be a consequence of relatively minor nerve injuries in which signs of changed nerve function are not initially apparent. This paper reviews the experimental evidence that describes the rather surprising morphological and physiological consequences of such injuries and relates them to human studies on neuropathic pain. These studies demonstrate that pain and changed somatosensory thresholds may occur following relatively minor axonal damage and even nerve sheath inflammation when no axonal damage is present.**

It is proposed that in many patients with diffuse limb pain there may be a contribution from nerve injury, i.e. a neuropathic component. This has implications for treatment of these conditions. Manual techniques and exercises to relieve compressive effects and restore normal neural dynamics should be effective in the management of these conditions.

Physiotherapists, as part of their clinical case load, see patients in whom painful symptoms appear related to obvious partial peripheral nerve injury. In fact chronic neuropathic pain may be a more frequent sequela of partial nerve injury than complete nerve transection. A spectacular example is causalgia, a syndrome where pain and extensive hyperalgesia follows a partial nerve injury.

Recent experimental evidence in animal models suggests that prolonged painful symptoms can occur following relatively minor neural injury. This paper will review the experimental evidence and, where possible, relate it to human studies and clinical situations where a diagnosis of neuropathic pain may not have initially appeared obvious. It is hoped that this will give the clinician a physiological understanding of these conditions and the encouragement to search in a more subtle fashion for the cause and site of neuropathic symptoms so that early appropriate treatment can be instituted.

A short overview of normal peripheral nerve anatomy of a mixed peripheral nerve is presented in Figure 1 and a classification of sensory nerve fibres in Table 1. Nerve fibres are packed within endoneurial connective tissue and bundled into fascicles. Fascicles are surrounded by the perineurium, a connective tissue and cellular barrier with considerable tensile strength that acts to limit diffusion thus protecting the interior of the fascicles by maintaining their chemical environment. The epineurium is the outer connective tissue that supports and protects the fascicles and carries the vascular vessels that send branches through the perineurium and form the endoneurial vessel system (Lundborg 1988).

Animal models of painful peripheral neuropathy include tight partial nerve ligation (Seltzer et al 1990), ligation with partial nerve transection (Chung et al 1993), peripheral neuritis (Maves et al 1993; Zochodne & Lam 1993; Eliave et al 1996) and chronic constriction injury (CCI) (Munger et al 1992). In animals, body posture, measurements of paw withdrawal latency in response to heat, mechanical stimulation and sympathetic manipulation are used as indicators of spontaneous pain, allodynia and hyperalgesia (see Table 2). The animals affected limb posture (guarding posture) is assessed as an indicator of spontaneous pain behaviour.

Tight partial nerve ligation and ligation with partial nerve transection will not be considered further, since in both of these models neural damage is such that severe sensory loss and motor weakness are clearly apparent. However, the chronic constrictive and neuritis model

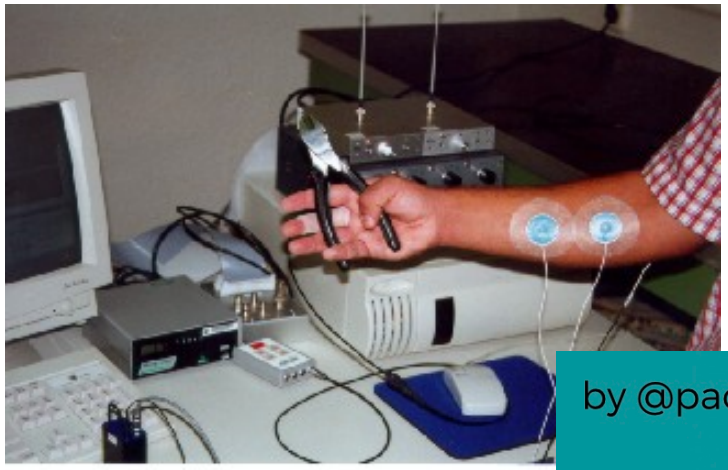
Jane Greening, MSc, MCSP, MACP, Research Fellow, **Bruce Lynn, PhD,** Reader in Physiology, University College London, Gower Street, London, WC1E 6BT, UK.
Correspondence to JG.

(Quintner JL 1989, Greening J 1998,2005, Butler D 2002, Shacklock 2007)

- Dicha irritación mantenida en el tiempo es fuente de impulsos ectópicos dolorosos y puede generar debilidad muscular.

- Impulsos ectópicos transmitidos a través del tejido conectivo del SNP.

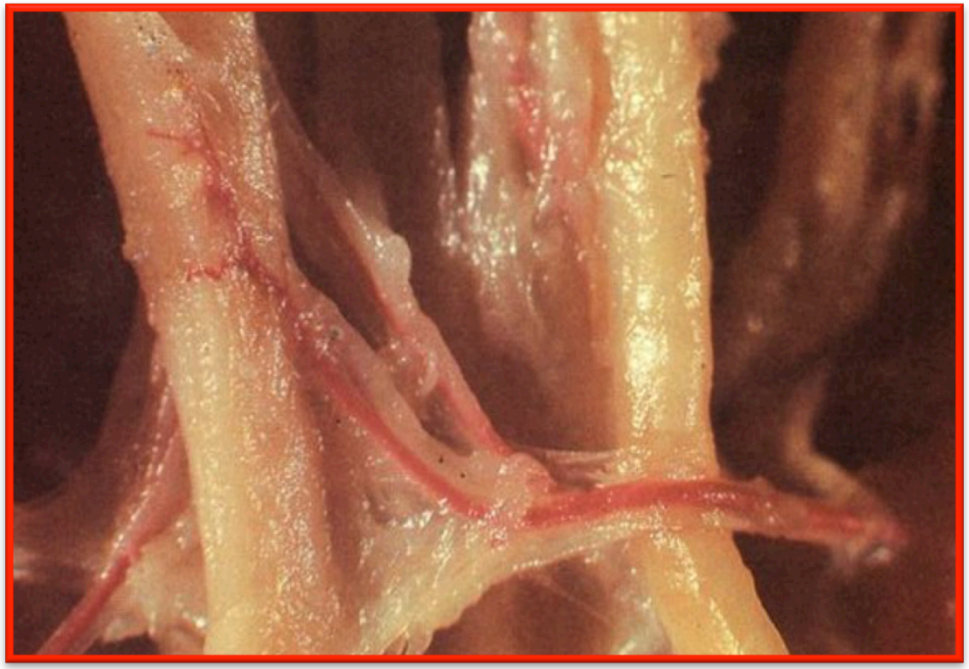
- Alteración de la conducción nerviosa por irritación del tejido de conducción, transporte axonal. (EMG +)



(Butler D 2002, Bove G 2003, Dilley A 2005, Elliott MB 2009, Bautista F 2010, 2011)

DISFUNCIÓN NEURAL

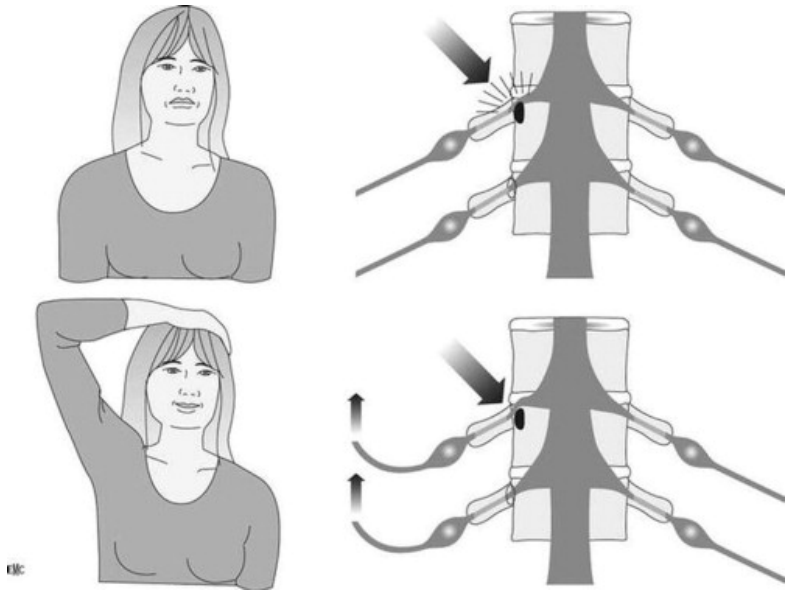
- Déficit vascular de los VASA-NERVORUM.
- Afectación de los NERVI-NERVORUM.
- Cambios de la mecanosensibilidad neural.



(De http://www.facebook.com/?ref=home#!/pages/Neurodinamica-en-la-practica-clinica/212580164860?ref=nf del album wall fotos 9/02/10 19:00)

Signos físicos de la disfunción neural

- Postura antálgica
- Alteración al movimiento activo (compensaciones)
- Alteración al movimiento pasivo
- Respuesta anómala al test neurodinámico
- Alodinia mecánica a la palpación neural
- Evidencia de causa local que la justifique



(Elvey 1997)

EVALUACIÓN FÍSICA A TRAVÉS DE LA NEURODINÁMICA

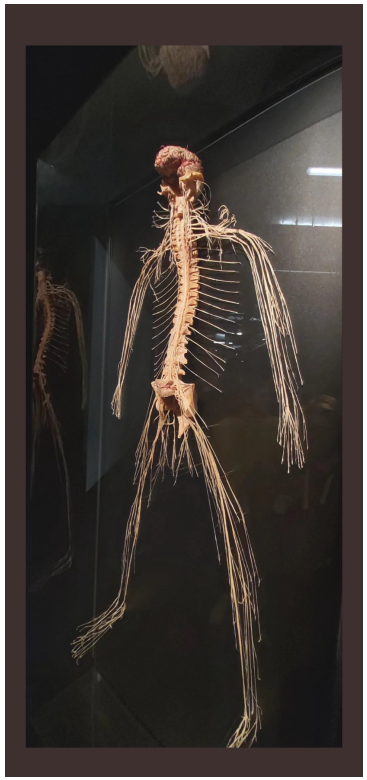
- Usamos movimientos poliarticulares para desafiar (la mecanosensibilidad) del sistema nervioso periférico o central.

- Pruebas reproducen la clínica: Secuenciación ND/ Test neurodinámicos

(Butler, 1991)

TRATAMIENTO NEURODINÁMICO

- La neurodinámica persigue la homeostasis del ambiente neural tanto intraneural, como en relación con al interface a través del movimiento.



(Basson A, 2017)

EVIDENCIA TRATAMIENTO NEURODINÁMICO

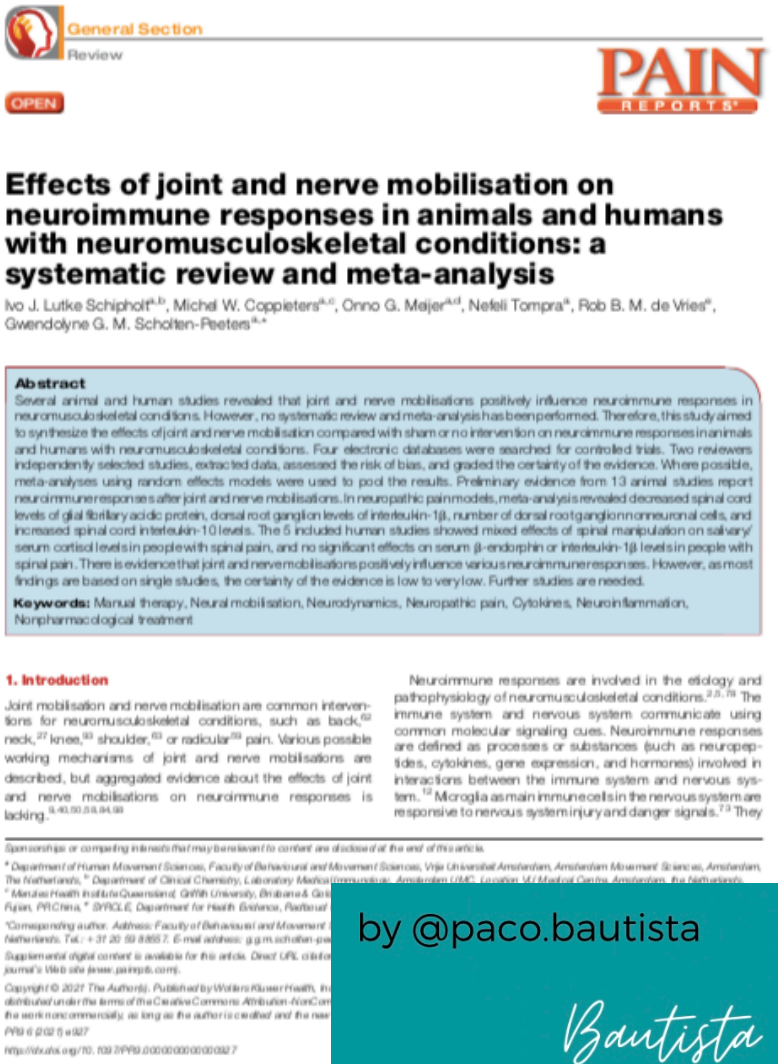
- Ha resultado efectivo en:
  - Cervicalgia
  - Lumbalgia inespecífica
  - STC (disminuye el edema intraneural)
  - Síndrome de canal de Guyon
  - Epicondilalgia
  - Talalgia (SLR)
  - Radiculopatía cervical

(Basson A, 2017)

MOVILIZACIÓN NEURAL Y RESPUESTAS NEUROINMUNES

- La movilización neural influye positivamente en las respuestas neuroinmunes en patología MSK.
- Con niveles disminuidos en el asta posterior y GRD de proteína ácida fibrilar glial, niveles de interleucina-1b, número de astrocitos. Sensibilizantes.
- Y niveles aumentados de interleucina-10. Inhibitorio
- Mejorando la hiperalgesia y procesos de sensibilización

(Schipholt IJ, et al. 2021)



General Section
Review
OPEN
PAIN REPORTS

Effects of joint and nerve mobilisation on neuroimmune responses in animals and humans with neuromusculoskeletal conditions: a systematic review and meta-analysis

Ivo J. Lutke Schipholt[a,b], Michel W. Coppieters[a,c], Onno G. Meijer[a,d], Nefeli Tompra[a], Rob B. M. de Vries[e], Gwendolyne G. M. Scholten-Peeters[a,*]

Abstract

Several animal and human studies revealed that joint and nerve mobilisations positively influence neuroimmune responses in neuromusculoskeletal conditions. However, no systematic review and meta-analysis has been performed. Therefore, this study aimed to synthesize the effects of joint and nerve mobilisation compared with sham or no intervention on neuroimmune responses in animals and humans with neuromusculoskeletal conditions. Four electronic databases were searched for controlled trials. Two reviewers independently selected studies, extracted data, assessed the risk of bias, and graded the certainty of the evidence. Where possible, meta-analyses using random effects models were used to pool the results. Preliminary evidence from 13 animal studies report neuroimmune responses after joint and nerve mobilisations. In neuropathic pain models, meta-analysis revealed decreased spinal cord levels of glial fibrillary acidic protein, dorsal root ganglion levels of interleukin-1β, number of dorsal root ganglion nonneuronal cells, and increased spinal cord interleukin-10 levels. The 5 included human studies showed mixed effects of spinal manipulation on salivary/serum cortisol levels in people with spinal pain, and no significant effects on serum β-endorphin or interleukin-1β levels in people with spinal pain. There is evidence that joint and nerve mobilisations positively influence various neuroimmune responses. However, as most findings are based on single studies, the certainty of the evidence is low to very low. Further studies are needed.

Keywords: Manual therapy, Neural mobilisation, Neurodynamics, Neuropathic pain, Cytokines, Neuroinflammation, Nonpharmacological treatment

1. Introduction

Joint mobilisation and nerve mobilisation are common interventions for neuromusculoskeletal conditions, such as back,[62] neck,[27] knee,[93] shoulder,[63] or radicular[59] pain. Various possible working mechanisms of joint and nerve mobilisations are described, but aggregated evidence about the effects of joint and nerve mobilisations on neuroimmune responses is lacking.[9,40,50,59,84,98]

Neuroimmune responses are involved in the etiology and pathophysiology of neuromusculoskeletal conditions.[2,5,78] The immune system and nervous system communicate using common molecular signaling cues. Neuroimmune responses are defined as processes or substances (such as neuropeptides, cytokines, gene expression, and hormones) involved in interactions between the immune system and nervous system.[12] Microglia as main immune cells in the nervous system are responsive to nervous system injury and danger signals.[73] They

*Corresponding author. Address: Faculty of Behavioural and Movement Sciences, Van der Boechorststraat 9, 1081 BT Amsterdam, The Netherlands. Tel.: +31 20 59 88557. E-mail address: g.g.m.scholten-peeters@vu.nl (G. G. M. Scholten-Peeters).

Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Web site (www.painrpts.com).

PR9 6 (2021) e927

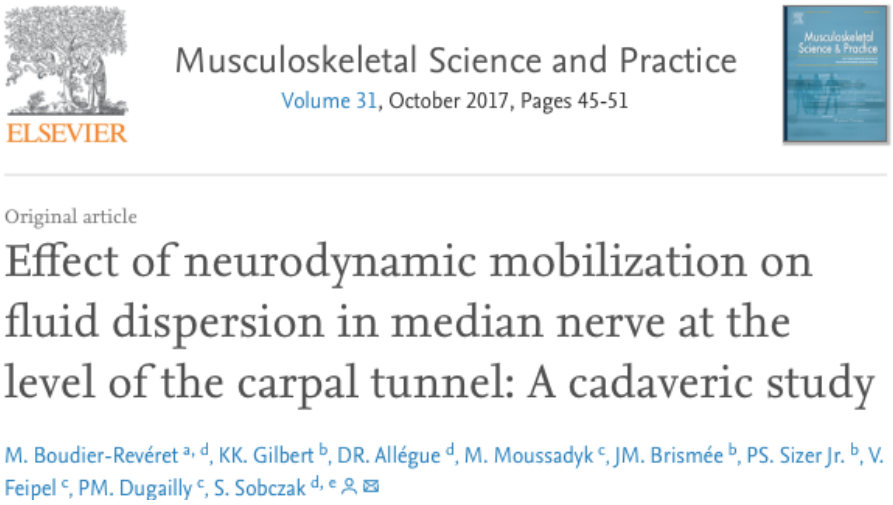
http://dx.doi.org/10.1097/PR9.0000000000000927

by @paco.bautista

Bautista

EFECTO DEL TRATAMIENTO NEURODINÁMICO

- Tanto el deslizamiento como las técnicas de tensión aplicadas durante 5 min, favorecen la dispersión del edema intraneural en cadáver.
- Ha resultado efectivo:
  - Técnicas de deslizamiento (p=0,016)
  - Técnicas de tensión (p=0,018)


ELSEVIER

Musculoskeletal Science and Practice
Volume 31, October 2017, Pages 45-51

Original article

Effect of neurodynamic mobilization on fluid dispersion in median nerve at the level of the carpal tunnel: A cadaveric study

M. Boudier-Revéret [a, d], KK. Gilbert [b], DR. Allégue [d], M. Moussadyk [c], JM. Brismée [b], PS. Sizer Jr. [b], V. Feipel [c], PM. Dugailly [c], S. Sobczak [d, e]

(Boudier M, 2017)

NEURODINÁMICA SOBRE EL SNC

J Bodyw Mov Ther. 2012 Jul;16(3):364-368. doi: 10.1016/j.jbmt.2011.12.003. Epub 2012 Jan 20.

Analysis of electromyographic activity in spastic biceps brachii muscle following neural mobilization.

Castilho J[1], Ferreira LAB[1], Pereira WM[1], Neto HP[2], Morelli JGDS[2], Brandalize D[1], Kerppers II[1], Oliveira CS[3].

- Parece que la movilización neurodinámica del lado sano disminuye la espasticidad del lado patológico en pc tras ACV.
- Y mejora la mecanosensibilidad del lado afecto, al movilizar el mediano del lado sano con el ULNT1.

Brain Inj. 2019;33(8):1039-1044. doi: 10.1080/02699052.2019.1606441. Epub 2019 Apr 26.

Upper Limb Neurodynamic Test 1 in patients with Acquired Brain Injury: a cross-sectional study.

Díez Valdés S[1], Vega JA[1,2], Martínez-Pubil JA[3].

(Castilho J, 2012; Diez-Valdés S, 2019)