

Persistent Pain State, Compromised Neural Plasticity and Reflextherapy

Gunnel Alice Berry*

Hunters Moon Cottage, Preston Candover, Basingstoke, RG252EP, UK

*Corresponding author: Gunnel Alice Berry, Lecturer/Clinician, Private Practice, Hunters Moon Cottage, Preston Candover, Basingstoke, RG252EP, UK, Tel: 01256 – 389722, E-mail: gunnel.berry1@gmail.com

Rec date: May 23, 2016; Acc date: July 04, 2016; Pub date: July 08, 2016

Copyright: © 2016 Berry GA. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Citation: Berry GA (2016) Persistent Pain State, Compromised Neural Plasticity and Reflextherapy. J Nov Physiother 6: 301. doi:10.4172/2165-7025.1000301

Optimisation of the use of reflexology

'Persistent Pain' [1] describes the notion of 'chronic pain' where unpleasant sensations continuously rise and fall to unimaginable heights and lows on a daily basis. Persistent pain may begin after an accident or other grievance but may seemingly have no underlying cause.

'Neural Plasticity' [2] is normal, ongoing interchanges of messages within the nerve tissue suggesting adaptability in the system. 'Compromised Neural Plasticity' [3] is an abnormal adaptability, resulting from disturbance in the nerve tissue. An interruption of interchanges of peptides in the axonal flow has occurred. The synaptic deficits in the periphery and the central brain areas are the most obvious areas of excitatory and inhibitory peptide exchanges relaying driving influencing and passing on information for muscle contraction, hormone release, kidney distillation and heart rate function etc.

The effect of injury and grievances occur where a deceleration force such as a whiplash incidence, fall from a height, tripping downstairs or emotional upset expose the body to direct physical and/or mental trauma. The sequel is an interrupted neural plasticity coherence which has an immediate effect on the nervous system as a whole. We do know that neural interfaces can be disturbed with minor injuries, [4] the blood flow is interrupted and prohibited to reach the tissue matrix. Continuous compromise in blood flow changes nerve tissue content and reduces effective conduction to intended targets. As well as 'pain', changes in vasoconstriction/dilatation indicate compromise in autonomic nerve tissue supply. Pain is driven by overload of excitatory peptides. Minor disturbances of intrinsic supply systems may be insignificant in small portions but collectively may become a major lack of plasticity. How do we turn the tap off? What mechanism inhibits production of sufficient innate descending peptides, enkephalins, endorphins and opioids to overcome excitatory overload? Presently intake of pharmaceutical opioids may provide pain-relief but are there other options?