

The invited clinical commentary is intended to provide experts in physiotherapy practice with an opportunity to expound personal viewpoints and where possible to offer scholarly critique of relevant evidence.

A Proposed Clinical Reasoning Model For Western Acupuncture

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ABSTRACT

The western approach to acupuncture as practised by many physiotherapists has been criticised because of the lack of clinical reasoning underpinning treatment choices. In accordance with recent advances in the understanding of neurophysiology and pain mechanisms western acupuncture can now be implemented with more reference to the basic sciences. A clinical reasoning model based on physiological mechanisms is proposed to aid physiotherapists in making treatment decisions and to provide a framework in which to guide alteration to interventions during the course of recovery. The model has yet to be validated by scientific research. **Brandham L. (2003) A Proposed Clinical Reasoning Model for Western Acupuncture. New Zealand Journal of Physiotherapy 31(1): 40-45.**

BACKGROUND

Two broad paradigms of acupuncture practice have been described, traditional Chinese acupuncture (TCA) which takes its origin from traditional Chinese medicine, and the more recently described scientific or western acupuncture (Filshie & Cummings, 1999). Western acupuncture is based on the basic sciences of anatomy, physiology and pathology. The western approach has been criticised for being too simplistic and prescriptive. In particular, practitioners of TCA have questioned whether the limited western model reduces the efficacy of the intervention as it ignores the subtleties and complexities of TCA. Recent advances in the understanding of pain neurophysiology and acupuncture mechanisms suggest a scientific basis for western acupuncture and would appear to support its use (Carlson, 2002; Filshie & Cummings, 1999).

A clinical reasoning model has been developed which uses current scientific knowledge to underpin clinical decision-making and attempts to provide a systematic approach to determine treatment choices. Following orthodox physiotherapy diagnosis, an acupuncture treatment plan is formulated based on the acupuncturist's impression of the predominant pain mechanisms involved in the individual patient's condition. Hence, a particular central nervous system (CNS) mechanism is targeted which is believed to provide an optimum treatment for the patient. As the condition becomes chronic, or pain mechanisms change with time, the acupuncture intervention may be altered to reflect the changing physiology of the individual. As the aim is to influence different levels or physiological mechanisms within the CNS this has been termed the "layering method" (Bradnam, 2001). Implementation of the model relies on an existing knowledge of anatomy; segmental and peripheral

nerve innervation of tissues and the neuroanatomy of the sympathetic nervous system. The proposed model has not yet been validated by clinical research, however, clinicians are encouraged to consider the possible impact of such a model on treatment outcomes for their patients.

CLINICAL REASONING

Clinical reasoning is the thinking underlying clinical practice, without which practice becomes a technical operation (Jones, 1995). Commonly, physiotherapists implement western acupuncture using neuro-anatomical principles to select acupuncture points. Points are chosen that are situated in close proximity to the injured body part, with the intention of inducing a strong segmental pain inhibitory effect. In many cases, particularly for acute nociceptive pain, this approach is extremely successful. However, when it fails to produce positive results alternative methods for change or progression of treatment are limited. Adding more needles, leaving the needles in situ for longer or applying greater intensity of stimulation are some methods used. Historically, these progressions were applied with limited knowledge of the underlying mechanisms or a clear clinical reasoning process. Anecdotal evidence suggests that many practitioners when faced with a recalcitrant problem switch to the TCA paradigm in the belief that the alternatives for the progression of acupuncture under the western model have been exhausted. Recent research into pain mechanisms and those mechanisms underlying acupuncture analgesia has identified possible progressions for western acupuncturists using different neural pathways within the CNS (Bradnam, 2001). Therefore the proposed model suggests treatment progressions for different patients so that clinicians do not need to change to the TCA paradigm.

MECHANISMS UNDERPINNING THE LAYERING METHOD

In addition to knowledge of anatomy, acupuncture points, peripheral neuroanatomy and segmental innervation, the clinician could find an understanding of the following mechanisms useful.

TISSUE MECHANISMS

Tissue mechanisms concern the stage of injury or healing of the patient's condition (Gifford & Butler, 1997). These range from acute or chronic inflammation to collagen remodeling. Knowledge of the injury and healing process, and the time frame of these processes will allow the acupuncturist to implement the optimum treatment for that particular stage of healing. Treatment can be tailored to the state of the injury by increasing or decreasing blood flow via encouragement of peripheral effects and/or manipulation of the sympathetic nervous system (Carlson, 2002; Lundeberg, 1998).

PAIN MECHANISMS

There are several methods of classifying pain and the one used here is that of Gifford and Butler (1997). Acupuncture may be more or less effective for different pain types so diagnosis of the predominant pain mechanism should underpin treatment decisions and determination of prognosis (Lundeberg & Ekholm, 2001).

Nociceptive Pain

Nociceptive pain relies on an intact nervous system as it results from nociceptor stimulation in the peripheral tissues. This can be via mechanical, inflammatory or ischaemic mechanisms and is usually associated with acute pain and/or tissue injury. Peripheral nerve endings and dorsal horn neurons in the spinal cord become sensitised, but these processes are self-limiting and resolve once the tissue has healed (Woolf & Costigan, 1999). A predictable stimulus-response relationship exists between pain provocation and pain experience (Gifford & Butler, 1997). Nociceptive pain has been demonstrated to respond positively in response to acupuncture treatment (Lundeberg, Hurtig, Lundeberg, & Thomas, 1988a).

Neurogenic pain

Neurogenic pain arises from a segment of nerve or the dorsal root ganglion due to axon damage (Myers, 1995). An atypical reaction to sensory inputs whereby non-noxious input will evoke or sustain pain results, hence neurogenic pain is characterised by allodynia and hyperalgesia (Wiesenfeld-Hallin & Zu, 1996). In addition, synthesis of the neuropeptide cholecystinin (CCK) increases following nerve injury. CCK is known to function as an endogenous opioid antagonist, therefore, the effect of the usual opioid pain inhibitory systems may be less effective (Wiesenfeld-Hallin & Zu, 1996). This suggests conventional acupuncture may not be useful for neurogenic pain. Preliminary information suggests that electroacupuncture applied with a high

frequency/low intensity paradigm may activate the noradrenergic (non-opioid) pathways in the spinal cord (White, 1999). Scientific investigation however is required to support high frequency/low intensity electroacupuncture as an option for relief of neurogenic pain.

Centrally-evoked Pain

This pain mechanism is related to altered CNS circuitry and processing. For unknown and probably multi-faceted reasons there is prolonged sensitisation of the spinal cord and regions of the sensory cortex in the brain after the initial injury has healed. It becomes, therefore, a component of chronic pain syndrome (Coderre, Arroyo, & Champion, 1993). The features of this pain type include ongoing pain after the injury has healed that present as unfamiliar anatomic pain patterns with atypical and unpredictable pain behaviors. Patients may exhibit variable responses to treatment and a poor response to medication (Gifford & Butler, 1997). Sympathetic nervous system (SNS) contribution to inflammation via activation of the lateral sympathetic horn in the spinal cord or by a centrally mediated autonomic response may be a feature of this pain type (Coderre, Arroyo, & Champion, 1993). The patient may exhibit sympathetic signs in related segments, including swelling and redness, but often there may be no signs. Slow healing musculoskeletal conditions might be related to inhibition of the SNS leading to trophic changes in target tissues (Bekkering & van Bussel, 1998). Lundeberg et al (2001) suggests acupuncture may be used in this pain mechanism, in particular in the early stages of chronicity when the level of dysfunction in the CNS is not known and some inhibitory pathways may be patent. These authors recommend decreasing sympathetic activity in the chronic pain patient by using a combination of warmth, gentle acupuncture or transcutaneous nerve stimulation with light stimulation only. According to Lundeberg & Ekholm (2001) ear acupuncture may also be used to increase parasympathetic activity. Lundeberg (1998) has also suggested that acupuncture may aid the chronic pain patient in terms of elevating mood and encouraging sleep. One investigation has been demonstrated that acupuncture releases neuro-chemicals in the brain a response that has the potential to affect the regulation of bodily processes, including both mood and sleep (Sjolund, Terenius & Eriksson, 1977).

Somatic Nervous System Mechanisms

Many acupuncture points are on or close to major peripheral nerve trunks. By needling these points, acupuncture may influence all tissues supplied by the nerve. As around 70% of acupuncture points lie in muscle tissue (Melzack, Stillwell, & Fox, 1977), the myotomal innervation of muscles should be learned and applied in practice. Alternatively, dermatomes and scleratomes can be chosen if the anatomical location of the acupuncture point is not in muscle tissue. Carlson (2002) proposes that the

acupuncturist may influence acute injuries without the need to place needles into injured tissue.

Sympathetic Nervous System Mechanisms

Acupuncture will induce immediate effects on the sympathetic nervous system (Liao, Urata, & Nishikawa, 1998). Segmental spinal afferents synapse with spinal preganglionic autonomic neurons to produce localised somato-autonomic reflexes mediated within the spinal cord. These reflexes exhibit strong segmental organisation and the effects on target organs are specific (Sato, Sato, & Schmidt, 1997). This may be utilised therapeutically to influence sympathetic outflow to regions of the body. The sympathetic efferent supply to the head and neck originates from the lateral horn of the spinal cord in the segments T1 – 4. The efferent outflow to the upper limbs is T5 – 9 and the lower limbs T10 - L2 (Bekkering & van Bussel, 1998). Spinal reflexes are under descending influences from the brain (Sato et al., 1997).

ACUPUNCTURE MECHANISMS

Three major categories have been described (Lundeberg, 1998).

Peripheral Mechanisms

This results from an axon reflex, whereby sensory neuropeptides such as Substance P and Calcitonin Gene Related Peptide (CGRP) are released from primary afferent nerve endings. Sensory neuropeptides produce local vasodilatation and modulate local immune responses (Lundeberg, Kjartansson, & Samuelsson, 1988b). In order to utilise the local effects of acupuncture, Lundeberg (1998) recommends needling close to the injured tissue and that it be performed with low intensity stimulation to encourage peripheral, rather than central neuropeptide release.

Spinal Mechanisms

These include attenuation of nociceptive input in the dorsal horn of the spinal cord (Lundeberg et al., 1988a), alterations in sympathetic outflow that are intensity dependant (Sato et al., 1997), and changes in motor output (Yu, Wang, & Wang, 1995). According to Sato et al (1997) high intensity needling may increase sympathetic outflow to tissues supplied by the segment in the short term, followed by a longer term decrease in outflow. Low intensity or non-painful input could reduce sympathetic outflow in the segment (Sato et al., 1997). It is proposed that spinal effects may occur immediately and have the potential to elicit strong analgesic effects. These effects may be obtained by needling the site of injury or any tissue innervated by the same spinal segment as the injured tissue according to Lundeberg (1998).

Supraspinal Mechanisms

There are many descending inhibitory systems from the brain that acupuncture is thought to activate. These include diffuse noxious inhibitory controls (DNIC), descending pain inhibitory pathways

from the arcuate nucleus in the hypothalamus, neurohormonal responses and central control of autonomic nervous system from the arcuate nucleus (Carlson, 2002; White, 1999). Lundeberg (1998) recommends longer periods of needling of up to 40 minutes to initiate supraspinal effects, as the brain systems are generalised and non-specific and may require high intensity stimulation over time to activate them. According to Lundeberg (1998) central responses may not be dependent on the site of needle placement, and may be more likely when strong points such as those in the hands and feet are needled. If a spinal segment is highly sensitised such as in acute nociceptive or centrally evoked pain, supraspinal effects should perhaps be elicited with needles placed extrasegmentally to the injured part to avoid exacerbating pain. Treatments should be repeated regularly as this is thought to stimulate the synthesis of neurohormones (Lundeberg, 1998).

In summary, Carlson (2002) suggests that acupuncture influences the nervous system in the periphery via release of neuropeptides from sensory nerve endings, in the spinal cord, by blocking pain signals in the dorsal horn, manipulation of sympathetic outflow and influencing motor output. In addition if the parameters of stimulation are sufficient, acupuncture may also activate nuclei in the brain for descending inhibition of pain and those controlling body processes such as autonomic and endocrine responses.

CLINICAL REASONING MODEL – THE LAYERING PROCESS

The clinical reasoning model suggested here is that the clinician asks a series of questions as to which acupuncture effects are desired for the patient, taking into account their individual pain and tissue mechanisms. Appropriate points and stimulation parameters can be chosen in an effort to provide an optimum intervention. Table 1 describes the layering technique for acupuncture treatment and contains questions that may be used for clinical reasoning. If healing of injured tissues is the main concern of the therapist then eliciting local effects of acupuncture could be one aim of therapy. Local effects may be maximised by using local acupuncture points, or simply by putting the needle directly into the damaged tissue. In addition, any points chosen for local effect could induce segmental effects. In acute pain, segmental effects may be desirable, as they could produce strong analgesia, but in the early stages of an injury associated local effects are potentially detrimental. The suggestion here is that in such a case, points anywhere that share an innervation via that spinal segment, can be chosen, be it other muscles, skin or bony periosteum, as long as the injured tissue is avoided (Lundeberg 1998).

It has been suggested that in cases of intense acute nociceptive pain fewer needles should be used, as the segment will already be sensitised by the painful afferent input from the injury. Too many needles in the segment, in this situation, may increase, rather than decrease the pain. A further suggestion is that

Table 1. The Layering Technique for Acupuncture Treatment of Musculoskeletal Conditions

Quick check list: Peripheral effects yes / no

If No: Needle away from injured tissue **If Yes:** What are the points or needle directly into tissue you want to influence. Use few needles and stimulate gently to maximise local effects. If treating superficial injuries, high frequency, low intensity electroacupuncture will aid blood flow to skin by reducing sympathetic tone.

2. Segmental / spinal effects yes / no

If No: Needle tissues with different segmental nerve (extrasegmental) supply to that of damaged tissue

If Yes: Do you want to needle into the damaged tissue?

If Yes: Choose local points situated anatomically near or in the damaged tissue

If No: Choose points in other tissues that are supplied by the same myotome, scleratome or dermatome as the damaged tissue. If choosing a myotome choose a muscle that is hypertonic, and/or in which the points are tender to palpation.

Acute Nociceptive pain use fewer needles in segment. (high frequency, low intensity electroacupuncture to maximise spinal cord inhibition – then progress to manual acupuncture or low frequency high intensity electroacupuncture)

Chronic Nociceptive pain – use more needles in segment **PLUS** Choose a distant point in the disturbed segment, in either dermatome, myotome or scleratome. If treating for pain a point in a bordering segment could be chosen as a distant point as nociceptive stimuli will affect bordering segments

OR Choose a distant point that stimulates the peripheral nerve that supplies the damaged tissue

Add a layer: Choose a spinal point that influences the segment sharing the nerve supply as the spinal level - e.g. Huatuo Jiaji point, Bladder point, or facet joint in cervical spine. Needle for 10-20 minutes

3. Supraspinal effects Yes/No

If No: needle for 10-15 minutes with moderate stimulation. Choose segmental points to damaged tissues and do not use "big" points.

If Yes: Choose extrasegmental points and "big points" of hands and feet (commonly used TCA points?). Needle for 20-40 minutes with strong stimulation. Activates descending inhibitory systems from hypothalamus and possibly diffuse noxious inhibitory controls (DNIC)

4. Sympathetic Outflow (When condition not improving with somatic treatment) Yes/No

If No: Think somatic nerve supply and treat according to above principles

If Yes: Choose segmental level of tissue you want to influence and needle Huatuo Jiaji or Bladder Point at that spinal level. Sympathetic outflow to head and neck is C8 to T4, Upper limb is T5-T9 and Lower Limbs are T10-L2. **PLUS:** Choose a distant point in tissues innervated with the same sympathetic segmental nerve supply as the tissue you wish to influence. Needle strongly for at least 10 minutes to increase sympathetic outflow, or gently to decrease outflow.

5. Central Sympathetic Effects Yes/No

Autonomic nervous system control by hypothalamus. Stimulated in same manner as analgesic supraspinal effects

Choose large points, hands and feet and stimulate strongly for 20-40 minutes

6. Immune Effects yes/no

Use Points at segmental level of spleen, lung, thymus Use general strong points that influence hypothalamus and regulate autonomic outflow (hands and feet)

Repeat Treatments, strong stimulation, 30 minutes

Auricular Points affecting vagal efferent activity

as the condition progresses from acute to chronic, more needles can be added into the segment (Lundeberg, 1998). Choosing distant points in the segment may be a method for doing this. Distant points are those in other muscles or tissues sharing an innervation with the injured tissue, but are further away from the injury site (Bradnam 2001). Another option is to use "spinal points" i.e. points on the Bladder channel or Huatuo Jiaji points in muscles close to the spinal level that share innervation with the injured part. With this application the segment is influenced via the dorsal rami. Supraspinal effects may be elicited by either segmental or extrasegmental needling and as these effects are time and intensity related any acupuncture point has the potential to induce this effect. Activating the descending pain inhibitory systems is thought to produce analgesia that is less strong than segmental inhibition, but has an effect of greater duration (Lundeberg et al., 1988a). When using these pathways to treat acute nociceptive pain, or centrally evoked pain, it may be prudent to activate them via extrasegmental needles to avoid overloading the sensitised segment. The hands in particular, and to a lesser extent the feet, have large representation on the somatosensory cortex in the brain, and so may be the points of choice to elicit supraspinal effects.

PROGRESSION OF TREATMENT – THE SYMPATHETIC NERVOUS SYSTEM

If a condition has a sympathetic component, such as Complex Regional Pain Syndrome, or if an injury is slow to heal, the proposed approach is that a specific manipulation of the sympathetic nervous system could be used to alter sympathetic outflow. A personal consideration is that this may be the mechanism clinicians unwittingly use when they treat using TCA as many strong TCA points are located in the extremities. According to Bekkering & van Bussel (1998) these points have a significant sympathetic innervation and so may be more useful in manipulating sympathetic responses. Needling at the spinal level supplying the target tissue or region or needling a point in the periphery sharing the segment could also stimulate the sympathetic nervous system to influence the target tissue. For example LI4 is located in the adductor pollicis muscle and has T1 innervation (Table 2). Needling LI4 may activate the sympathetic lateral horn at T1 level and alter the sympathetic outflow to the head and neck. Autonomic outflow is under central control by the hypothalamus regulating the sympathetic and parasympathetic nervous systems (Kandell, Schwartz & Jessel, 2000). It is believed that stimulation of this system is non-specific in terms of point location, being more

dependent on intensity and length of stimulation. In order to effectively activate central autonomic responses the use of "strong" points has been recommended, similar to those used to evoke central responses. Research has shown that acupuncture stimulation may increase or decrease sympathetic activity depending on the state of the target organ or tissue (Sato et al., 1997). For optimum treatment of body organs, Stener-Victorin, (2000) has recommended the use of high intensity, low frequency electroacupuncture to provide a strong stimulus to the CNS.

NEUROHORMONAL MECHANISM

According to Lundeberg, (1999) a further supraspinal mechanism that may be activated by acupuncture, given certain time and intensity parameters, is a neurohormonal mechanism. The suggestion is that Beta-endorphin and Adrenocorticotrophic hormone (ACTH) are released in equimolar amounts from the pituitary gland into the blood stream (Lundeberg, 1999). ACTH in turn, may influence the adrenal gland increasing the production of anti-inflammatory corticosteroids (Sato et al 1997). A further observation is that Beta-endorphin levels may fluctuate with changes in the number and activity of T-lymphocytes and natural killer (NK) cells (Lundeberg, 1999). These effects can be incorporated into a treatment plan and may optimise healing effects in slow healing conditions, such as those that affect immune deficient people or those with high-intensity demands on their bodies such as athletes. Lundeberg, (1999) proposes that to influence the organs producing T-lymphocytes, NK cells, the thymus, spleen and lung, the segments that supply sympathetic innervation to these organs should be needled. In addition, Lundberg (1999) suggests that sympathetic points and ear acupuncture points may also be considered because of their potential to influence vagal parasympathetic activity. Research is needed to investigate these opinions.

CLINICAL REASONING MODEL OF WESTERN ACUPUNCTURE: EXAMPLES OF CLINICAL APPLICATION

Example One: Proposed application for Lateral Epicondylitis (for all point locations refer to Table 2)

Local effects may be achieved by introducing a needle into LI11 or into the common extensor origin tendon. The same points may be used to achieve segmental effects so that both local and segmental effects may be achieved simultaneously. In an acute state where local effects may be contraindicated, but segmental effects are desired, point choices could include LI9,10 and/or LU5. These proposed points are segmental and close to the injury, but are not directly into the damaged region. Treatment may be progressed by adding more distant points for segmental effect. For example LU3,4 or LI8 are recommended by the author because they are in different muscles but they share the C5/6 myotome innervation. An additional or alternative point may

be chosen to influence the posterior interosseous nerve as this nerve supplies the affected tissue. The author recommends TE5 to access this nerve. Spinal points recommended are at the spinal levels of C5/6 and needling into Huatuo Jiaji or Bladder Channel points at these levels may influence the posterior rami for the segment. To activate supraspinal analgesic effects, LI4 or LR3 are recommended extrasegmental points. Needling points on the bladder channel between levels T5-9 may achieve stimulation of segmental sympathetic outflow. The intensity of needling will be determined by the treatment effect desired. It is proposed that central sympathetic points selected may be the same as those chosen for supraspinal analgesia.

Example Two: Proposed application for Knee Joint (for all point locations refer to Table 2)

Local effects may be achieved by using the knee 'eye' points, Hedong, BL40 and various forms of periosteal tapping to capsule and ligament tissues. As in example one it is proposed that these treatments may have the potential to produce segmental as well as local effects. Where local effects are not desired, tissues may be needled that share the innervation of the knee joint. The author recommends for example that in a case of posterior knee pain, innervated mostly by the tibial nerve, points in any muscles supplied by the tibial nerve may be used. Points in such muscles include BL36, 38, GB33, LR8, KI10 and SP9. To progress treatment it is recommended that anatomically distant points may be added. For example, the author recommends KI3 or GB30 for their associations with the tibial and sciatic nerves respectively. Spinal points recommended are at the spinal level of L3/4 on the bladder channel. To activate the supraspinal analgesic effects and possibly influence central autonomic outflow LR3, ST36 or LI4 and 11 are recommended. Needling the spine at T12 to L2 or selecting points SP12, LR10, or 11 that have L2 innervation may generate segmental sympathetic effects.

The author's viewpoint is that stimulation parameters of intensity and duration need to be considered in addition to point location when progressing treatment in order to access the different 'layers' of the CNS.

CONCLUSION

This clinical reasoning model was evolved by collaboration between academics and clinicians in New Zealand and proposes a theoretical framework for the application of western acupuncture. The model attempts to use current physiological theories to underpin and inform clinical decision-making and suggests a basis for treatment progression. It is recommended that clinicians measure outcomes and use reflective practice when implementing the model as it is not based on scientific evidence and requires primary research in a clinical setting to support its use.

Key Messages:

- The western approach to acupuncture as practised by many physiotherapists has been criticised because of the lack of clinical reasoning underpinning treatment choices. In accordance with recent advances in the understanding neurophysiology and pain mechanisms western acupuncture can now be implemented with more reference to the basic sciences.
- A clinical reasoning model-the 'layering process', based on physiological mechanisms, is proposed to assist acupuncturists in making treatment choices. The recommended progressions use different mechanisms in the central nervous system.
- The model relies on orthodox physiotherapy diagnosis and prior knowledge of anatomy, physiology, pathology and acupuncture point locations. Detailed knowledge of somatic and sympathetic neuroanatomy along with physiological mechanisms is also required to implement the model.
- While the model has not been validated by scientific investigation, physiotherapists practicing western acupuncture are encouraged to consider the layering process and its possible contribution to the practice of western acupuncture.

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References

- Bekkering R, van Bussel R (1998). Segmental Acupuncture. In J. Filshie & A. White (Eds.): *Medical Acupuncture: A Western Scientific Approach*. Churchill Livingstone.
- Bradnam L (2001). Western Acupuncture Point Selection: A Scientific Clinical Reasoning Model. *Meridian Worldwide – Newsletter of the International Acupuncture Association of Physical Therapists* 10(2), October, 9-18.
- Carlsson C (2002). Acupuncture Mechanisms for Clinically Relevant Long-term Effects - reconsideration and a hypothesis. *Acupuncture in Medicine* 20(2-3) 82-99.
- Coderre T, Arroyo J, Champion G (1993). Contribution of Central Neuroplasticity to Pathological Pain. A Review of clinical and experimental research. *Pain*, 52, 259-285.
- Filshie J, Cummings M (1999). Western Medical Acupuncture. In E. Ernst & A. White (Eds.), *Acupuncture. A Scientific Appraisal* (pp. 31-59). Oxford: Butterworth-Heinemann.
- Gifford LS, Butler DS (1997). The Integration of Pain Sciences into Clinical Practice. *Journal of Hand Therapy*, April-June, 87-95.
- Jones M (1995). Clinical Reasoning and Pain. *Manual Therapy*, 1, 17-24.
- Kandell E, Schwartz J, Jessell T (Eds) (2000). *Principles of Neural Science*. 4th Edition. McGraw-Hill Health Professions Division, USA.
- Liao T-J, Urata S, Nishikawa H (1998). Transient Decrease in Skin Resistance Response and Level at the Deh-Chi stage caused by Manual Acupuncture. *Tohoku Journal of Experimental Medicine*, 186, 19-25.
- Lundeberg T (1998). *The Physiological Basis of Acupuncture*. Paper presented at the MANZ/PAANZ Annual Conference, Christchurch, New Zealand.
- Lundeberg T (1996). Electrical Stimulation Techniques [comments]. *Lancet*, 348, 1672-1673.

- Lundeberg T (1999). Effects of Sensory Stimulation (Acupuncture) on Circulatory and Immune Systems. In E. Ernst & A. White (Eds.), *Acupuncture: A Scientific Appraisal* (pp. 93-106): Butterworth Heinemann.
- Lundeberg T, Ekholm J (2001). Pain - From Periphery to Brain. *Journal of the Acupuncture Association of Chartered Physiotherapists*, February 2001, 13-19.
- Lundeberg T, Hurtig T, Lundeberg S, Thomas M (1988a). Long Term Results of Acupuncture in Chronic Head and Neck Pain. *Pain Clinic*, 2, 15-31.
- Lundeberg T, Kjartansson J, Samuelsson U (1988b). Effect of electrical Nerve Stimulation on Healing of Ischaemic Skin Flaps. *Lancet*, ii, 712-714.
- Melzack R, Stillwell D, Fox E (1977). Trigger Points and Acupuncture Points for Pain, Correlations and Implications. *Pain*, 3, 3-23.
- Myers RR (1995). The Pathogenesis of Neuropathic Pain. *Regional Anesthesia*, 20(3), 173-184.
- Sjölund B, Terenius L, Eriksson M (1977). Increased cerebrospinal Fluid Levels of Endorphins after Electroacupuncture. *Acta Physiologica Scandinavica*, 100(3) 382-384.
- Sato A, Sato Y, Schmidt R (1997). *The Impact of Somatosensory Input on Autonomic Functions*. Heidelberg: Springer-Verlag.
- Stener-Victorin E (2000). *Acupuncture in Reproductive Medicine*. Goteborg University Sweden, Goteborg.
- White A. (1999). Neurophysiology of Acupuncture Analgesia. In E. Ernst & A. White (Eds.), *Acupuncture: A Scientific Appraisal* (pp. 60-92): Butterworth-Heinemann.
- Wiesenfeld-Hallin Z, Zu X (1996). Plasticity of Messenger Function in Primary Afferents following Nerve Injury - implications for Neuropathic pain. In G Carli & M Zimmerman (Eds.), *Progress in Brain Research* (Vol. 110): Elsevier Science.
- Woolf C, Costigan M (1999). Transcriptional and Posttranscriptional Plasticity and the Generation of Inflammatory Pain. *Proceedings of the National Academy of Science*, 96 July, 7723-7730.
- Yu Y-H, Wang H-C, Wang Z-J (1995). The Effect of Acupuncture on Spinal Motor Neuron Excitability in Stroke Patients. *Chinese Medical Journal*, 56, 258-63.

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