## Review

# CHRONIC PAIN AND PHYSICAL ANALGESIA: THE IMPACT OF PHYSICAL MODALITIES TO REDUCE PAIN

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## **Summary**

Current paper proposes personal opinions on some contemporaneous theories of pain and therapeutic concepts of analgesia, including physical analgesia. Millions of patients suffer from chronic pain. All modern scheduled drugs used for treatment of the persistent pain (opioids, NSAIDs, COX-2 inhibitors) are associated with limitations and side effects. Our purpose was to remind the wide public of the impact of physical modalities in pain management in adults. We explain different natural and preformed physical modalities, with effectiveness in clinical practice. The author formulates a conception of pathogenetical mechanisms of physical analgesia. Declared opinions and conclusions of the author are based on the traditions of Bulgarian rehabilitation school, on analysis of scientific literature (including electronic media), on our modest 20 years experience - clinical observations, scientific and applied investigations; and on the results from systematic interviews with in-patients (treated in the National Physical therapy and rehabilitation hospital (Sofia) and in the Rehabilitation Clinic of Pleven University Hospital) and with outpatients (of some Medical centers of Sofia and Pleven).

**Key words:** physical modalities, rehabilitation, pain, analgesia

The formulation of the gate-control theory [1] for explanation of pain deposited the base of a new epoch in the development of orthodox medicine. This was the introduction of the principle of "contrastimulation" final effect reticence by stimulation of inhibiting systems or else, final effect stimulation by embarrassment of inhibiting systems. The science proved the existence of unsuspected reflectory relations and dependences between processes, apparently independent. There appear conditions for infringement of traditional therapeutic thinking and for formulation of a fundamentally new approach for creation of modern, or for explanation of existing anti-pain methods from the domain of electrology, thermo and cryotherapy, manual techniques, reflexology.

In our opinion, the anti-pain effect of physical modalities is very important, with a high level of efficacy. Physical analgesia has not side effects, and may be applied in combination with other therapeutic factors.

In the era of evidence-based medicine is important to precise details of correspondent therapeutic complex [2, 3, 4, 5], concerning applied physical modalities, methods and respective dosage.

**Pain** is one of the most frequent sensations, formed in the nervous system, with different individual characteristics. Pain is a subjective experience, defined by nociceptive activation; by changes in sensory nerves and paths, in cerebral centers regulators of stress, affections and motivation [6]. Different factors (physical, chemical, psychic) may influence pain sensation.

The biological significance of pain sensation is to defend the organism from noxious external influences (signal of attention). The pain informs the organism and causes a *reflex defensive reaction* of the individual.

Contrary to the medico-philosophical systems explaining pain by a metaphysical conflict between life and death, the French philosopher René Descartes formulates a new methodology oriented to the correct thinking and the system of knowledge. In his famous works "Discourse on the Method" (1637, in French) and "Principles of philosophy" (1644, in Latin) Descartes formulates the famous statement "Cogito ergo sum" (I think, therefore I am; or better, I am thinking, therefore I exist). In "Meditations on first philosophy" (1641, 1647) he explained the idea of the rational animal, the mind connection and of the defensive character of pain (baby fire, boy fire), including its capacity to unchain a reflectory reaction "pulling on a thread".

In nine volumes of the first didactic tractate on physiology "Elementa physiologiae corporis humani" (Physiological elements of the human body), the Swiss poet, scientist and Doctor Albrecht von Haller explain the difference between irritability and sensibility (1757-1766). Antoine-Laurent de Lavoisier, the father of contemporaneous chemistry, after experiments concluded that "la respiration est donc une combustion", that is, respiratory gas exchange is a combustion, like that of a candle burning and "life is a burning process". Slowly but definitively, in 19th century physiological sciences leave metaphysical speculations and target to the principles of physics, natural sciences and clinical medicine.

The Holland surgeon Willem Noordenbos [2] proposed the hypothesis for the multi-synaptic transmission of pain signal (1959): "One-one

synaptic transmission must be the exception rather than the rule in the nervous system. Any nerve cell located in the anterior corn . . . could hardly be expected to synapse at higher level with one such similar cell only. It will probably send ramifications to many other locations, and in turn be acted upon by the ramifications of many other cells. . . Far from being a continuous chain of short neurons, these fibers must constitute links in an extremely complicated nerve net in which, within limits, everything synapses more or less with everything else."

In 1965 the collaboration between two individual investigators the British physiologist Patrick Wall and the Canadian psychologist Ronald Melzack, generated the theory of gate control. Their common article "Pain Mechanisms: A New Theory" [1] was qualified like "the most influential ever written in the field of pain". Melzack and Wall suppose the existence of a controlling mechanism in the spinal medulla, which is closed in response to the normal stimulation of fast fibers of tactile sense, but is open if the slow fibers of pain perception transport numerous and intensive sensory signals. The gate is closed (and the pain transmission is interrupted) in case of a new stimulation of the fast fibers.

The scientific literature mentioned various contemporaneous theories for pain perception: *specific* (specific pain receptors nociceptors); *non-specific* (patterns theory pain perception depends on decoding, probably at spinal level, of temporo-spatial organization of patterns signals perceived by intensive stimulation of non-specific receptors). Most of authors support *combined theories*.

The following levels of pain perception are mentioned: *receptors* (nociceptors and free nerve terminals); *sensory roots; posterior columns of the spinal medulla; thalamus opticus; reticular formation; cerebral cortex*. In some cases (stress-analgesia) the psycho-emotional state of the individual is considered as very important.

There exist different types of pain: *acute* and *chronic (persistent) pain; nociceptive* and *neuropathic pain; others (central pain)* [7-13]. It is important to remember that the pain in clinical practice is generally combined with nociceptive and neuropathic components, and the latter usually prevails.

Some authors [13] consider that the combination of nociceptive (inflammatory hyperalgesia) and neuropathic mechanisms in

every individual case is the main cause for our therapeutic helplessness, as regard pain.

In the neuropathic type of pain, the direct irritation of the nociceptor (receptor) is not necessarily present. Neuropathic pain includes mechanisms of long-time potentiation (LTP) augmented expression of Na<sup>+</sup>, H<sup>+</sup>, Ca<sup>++</sup> channels in the peripheral sensory nerves, generating an ectopic excitation and augmented sympathetic activity [13]. The LTP-mechanism of neuropathic pain is the theoretical base for the therapeutic use of drugs with membrane-stabilization activity, some anti-depressants and sympaticolytics [14].

Probably, preformed physical modalities execute their influence exactly on a membrane level (plasmalema and neurolema). We consider that preformed factors control the algesic type of stimulation of nociceptors (closing ion channels and thus inhibing the generation of action potentials). Presumably, peloids and physiotherapy (kinesitherapy in Bulgarian nomenclature) regulate the hyperalgesic type of stimulation (reducing the probability of irritation of nociceptors by mechanical, chemical and thermal stimulation) [5, 15].

# Traditional methods for pain treatment

#### Drug therapy

Traditionally, in clinical practice different medicaments are applied [3, 4, 5, 15-22].

Opioid analgesics = opioids (Morphine, Codeine, Fentanyl, Meperidine, Methadone, Propoxyphene, Levorphanol, Hydromorphone, Oxycodone hydrochloride, Oxymorphone, Pentazocine) have a lot of limitations due to their side effects and the high risk of addiction.

Non-opioid analgesics are steroids (corticosteroids) and non-steroidal anti-inflammatory drugs (classical NSAIDs salycilates, diclofenac, ibuprofen, naproxenic acid; and the modern COX-2 inhibitors) (Table 1). Some authors consider acetaminophen (Tylenol, the most common antipyretic drug used in the US) as belonging to this group too. The use should be very carefully evaluated (risks of gastro-intestinal events, cardio-vascular side effects, heart failure, etc.). The dose titration is important.

Adjuvant therapy includes drugs that are not usually used for pain relief but may relieve pain in certain circumstances. When used to relieve pain, they are usually combined with other analgesics

Table 1. Non-opioid analgetics

NONSTEROIDA	L ANTHINFLAMMATORY DRUGS)
GROUPS	DRUGS (commercial name)
Salycilates	Aspirin
	Choline magnesium trisalicylate
	Diflunisal Salsalate
COX-2 inhibitors	Celecoxib
(Coxibs)	Valdecoxib
Other NSAIDs	Diclofenac
	Etodolac
	Fenoprofen
	Flurbiprofen
	Ibuprofen
	Indomethacin
	Ketoprofen
	Ketorolac
	Meclofenamate
	Mefenamic acid
	Meloxicam
	Nabumetone
	Naproxen
	Oxaprozin
	Piroxicam
	Sulindac
	Tolmetin

or non-drug pain treatments. This group includes: tricyclic antidepressants (TCAs like amitriptyline, clomipramine, desipramine, imipramine, maprotiline, nortryptyline, etc.); selective serotonin reuptake inhibitor (SSRI like fluoxetine); anticonvulsants (ACs like gabapentin, pregabaline, phenytoin, carbamazepine, clonazepam, divalproex, lamotrigine, topiramate, oxcarbazepine). The main effect of TCAs and SSRIs on pain is explained by the blockade of norepinephrine and serotonin reuptake resulting in an increased availability of these transmitters in the synapses of the descending pain-modifying pathways. The main effect of ACs on pain is explained by bind to synaptic Ca channels resulting in reduced release of neurotransmitters responsible for afferentation of pain [21, 22].

Local anesthetics have different mode of application: peroral (e.g. the antiaarrhytmic Mexiletine); topical (e.g. paravertebral blockade with lidocaine); local (local injection or application of crème containing Capsaicin).

Local nerve destruction is applied in some cases of pain (e.g. trigeminal neuralgia): local injection of a nerve-destroying substance (phenol), nerve freezing by cryotherapy, nerve burning by radiotherapy.

# Physical analgesia

In physical analgesia we apply different physical modalities [4, 5, 15-31]:

## Preformed modalities

Low-frequency currents and low frequency modulated middle-frequency currents (sinusoidal-modulated, interferential, Kots currents);

Ttranscutaneous electroneurostimulation (TENS);

*High-frequency currents* (diathermy, ultrahigh frequency currents, decimeter and centimeter waves);

*Ultra-sound* and *phonophoresis* with NSAIDs:

Low-frequency magnetic field.

## Natural modalities

Cryo-factors (ice, cold packs, cold compresses);

*Thermo-agents* (hot packs, hot compresses);

Hydro- and balneo-techniques (swilling, rubbings, showers, baths, piscine); hydro and balneo-physiotherapy techniques (underwater massage, under water exercises, etc.);

*Peloidotherapy* (fango therapy, thermal mud, sea lye compresses);

Physiotherapy techniques - stretching, postisometric relaxation, manual therapy (traction, mobilization, manipulation); massages (manual and with devices; periostal, connective tissue massage, etc.).

**Reflectory methods**: electrotherapy, thermotherapy and physiotherapy in reflectory points and zones; acupuncture, laserpuncture, acupressure, etc.

## Mechanisms of physical analgesia

In physical medicine, we applied the principles of gate-control theory of Melzack & Wall [1] for central nociceptive influence. Investigations of Gacheva [32, 33, 34] have demonstrated that selective electrostimulation of tactile Aβ-nerve fibers (with high velocity of conduction) provokes a preliminary stimulation of suppressive neurons, that inhibit tardily occurring nociceptive stimuli of Aδ and C-fibers (with lower conduction velocity). It is assumed that a closer suppressive transfer mechanism exists at spinal level. At the peripheral level, direct anti-adaptation electrostimulation of the receptors probably provokes a hyperpolarization with an increase of the sensibility of nociceptors. A direct low frequency electrical stimulation of the A $\delta$  and C fibers may have an analgesic effect.

We propose our own theory to explain the mechanisms of action of physical modalities on nociceptive and neuropathic pain - we introduced

the notion *physical analgesia* or *anti-pain effect* of physical modalities [5]. Our hypothesis is based on the traditions of Bulgarian neurorehabilitation school, on analysis of scientific rehabilitation literature (including electronic media), on our modest 20-year experience (1986-2008) - clinical observations, scientific and applied investigations [5, 17, 26-28]; and on the results from systematic interviews with in-patients (treated in the National Physical Therapy and Rehabilitation Hospital, Sofia and in the Rehabilitation Clinic of Pleven University Hospital) and with outpatients (of different Medical centers of Sofia and Pleven).

The physical complexes used may provoke an analysesic effect by the following mechanisms (Fig.1):

By influencing the cause for irritation of pain

Probable mechanisms of action of pre-formed and natural physical modalities

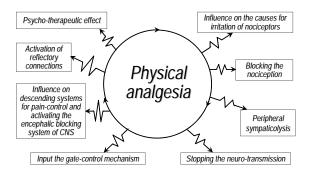


Figure 1. Mechanisms of physical analgesia

receptors - a consequence of stimulation of circulation, metabolism and trophy of tissues (by low and medium frequency electric currents, magnetic field, ultrasound, He-Ne laser; massages; manual techniques);

By blocking nociception (low frequency currents, including transcutaneous electrical nerve stimulation or TENS; lasertherapy);

By peripheral sympaticolysis (low frequency currents like dyadinamic currents, peloids);

By stopping the neural transmission (by C and A\delta fibers) to the body of the first neuron of general sensibility (iontophoresis with Novocain in the receptive zone the region of neuroterminals);

By input of the gate-control mechanism (TENS with frequency 90-130 Hz and interferential currents with a relatively high

resulting frequency - 90-150 Hz);

By activation of the reflectory connections: cutaneous - visceral, subcutaneous-connective tissue-visceral, proprio-visceral, periostal-visceral and motor-visceral (classic manual, connective tissue and periostal massage, post-isometric relaxation and stretching-techniques);

By influence on the pain-translation in the level of posterior horn of the spinal medulla using the root of activation of encephalic blocking system in the central nervous system (increasing the peripheral afferentation) and influence on the descending systems for pain control (TENS with frequency 2-5 Hz and interferential currents with low resulting frequency 1-5 Hz, acupuncture and laserpuncture; reflectory and periostal massage, zonotherapy, acupressure, su-dgok massage; preformed factors in reflectory zones /palms of hands, plants of feet, paravertebral points; zones of Head, of Mackenzie, of Leube-Dicke, of Vogler-Krauss);

By influence on the psychic state of the patient - the "doctor" drug and the "procedure" drug.

During last years, the development of physical medicine has proven the existence of some reflectory connections in the human body, based on the theory of the metameric structure of the embryo during the intra-uterine development. In physical analgesia, we apply the following groups of reflectory connections: *cutaneous-visceral*, *subcutaneous-connective tissue-visceral*, *proprio-visceral*, *periostal-visceral* and *motor-visceral* (Fig.2).

### **GROUPS OF REFLECTORY CONNECTIONS**

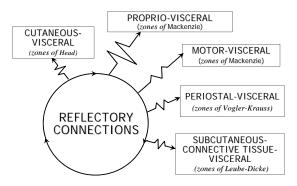


Figure 2. Groups of reflectory connections

The construction of a complex physical and rehabilitation programme is needed, because the mechanism of action of different procedures is diverse (Fig.3). This opinion is in agreement with

modern tendencies in drug treatment of neuropathy (during the last ten years). Moreover, contemporaneous studies prescribe a combintion of symptomatic and pathogenetically oriented therapy [21].

Synergy between physical modalities

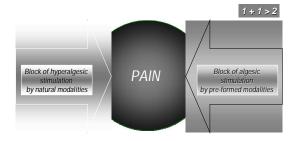


Figure 3. Synergy between physical modalities

Physical modalities have an effect on the interstitium modulating the intern compartments (milieu interieur of Claude Bernard) and this way creating an optimal medium for the influence of medicament substances. This is the theoretical base for therapeutic application of a combination of drugs and physical modalities. The synergy between different physical modalities is the logical base for prescription of a complex rehabilitation program [1, 13].

In conclusion we should not underestimate the use of methods of physical analgesia in clinical practice, which can improve the quality of life of patients and accelerate their returning to work and social activities.

#### References

- 1. Melzack R, Wall P. Pain mechanisms: A new theory. Science. 1965;150:971-9.
- 2. Noordenbos W. Pain: Problems Pertaining to the Transmission of Nerve Impulses Which Give Rise to Pain. Amsterdam: Elsevier; 1959.
- 3. Lewit K. Postisometric relaxation in combination with other methods of muscular facilitation and inhibition. Manual Medicine. 1986;2:101-4.
- 4. Kotzeva R, Georguiev G. Acupuncture points for influence pain. In: Shotekov P, editor. Pain pathogenesis and treatment. Sofia: Lieder press; 1998. p.191-6. (In Bulgarian)
- 5. Koleva IB. Physical analgesia and stimulation (including algorithms and methods for neurological patients). Sofia: SIMEL; 2006. (In Bulgarian)
- 6. Terenius L. Profiles of CSF neuropeptides in chronic pain of different nature. In: Sicuteri F,

- Terenius L, Vecchiet L, Maggi C, editors. Advances of pain research and therapy. 1992;20:93-100.
- 7. Shotekov P. Anatomical and pathophysiological bases for treatment of pain. In: Shotekov P, editor. Pain pathogenesis and treatment. Sofia: Lieder press; 1998. p.27-46. (In Bulgarian)
- 8. Casey KL, editor. Pain and central nervous system diseases. The central pain syndromes. New York: Raven Press; 1992.
- 9. Evans R, Bronfort G, Nelson B, Goldsmith Ch. Two-Year Follow-up of a Randomized Clinical Trial of Spinal Manipulation and two types of rehabilitative exercise for Patients with Chronic Neck Pain. Spine. 2002;27(21):2383-9.
- Ferreira SH. Prostaglandins: Peripheral and Central Analgesia. In: Bonicca JJ, editor. Advances in Pain Research and Therapy. New York: Raven Press; 1983. p.627-34.
- 11. Krogstad BS, Jokstad A, Dahl BL, Soboleva U. Somatic complaints, psychologic distress, and treatment outcome in two groups of TMD patients, one previously subjected to whiplash injury. J.Orofac.Pain. 1998;12(2):136-44.
- 12. Weissenberg M. Pain and pain control. In: Daitzman RJ, editor. Diagnosis and intervention in behavior therapy and behavioral medicine. Vol.1. New York: Springer; 1983. p.90-149.
- 13. Merskey H, Bogduk N, editors. Classification of chronic pain descriptions of chronic pain syndromes and definition of pain terms. Seattle: IASP press; 1994.
- 14. Vlaskovska M. Pharmaco-biochemical principles in pain treatment. In: Shotekov P, editor. Pain pathogenesis and treatment. Sofia: Lieder press; 1998. p.47-59. (In Bulgarian)
- 15. Nalty T, Sabbahi M. Electrotherapy Clinical Procedures Manual. Nalty T, editor. New York St.Louis San Francisco Bogota Caracas Lisbon London Madrid Mexico City Milan Monreal New Delhi Singapoore Sydney Tokio - Toronto: McGraw Hill; 2001.
- Gutenbrunner C, Ward AB, Chamberlain MA, editors. White Book on Physical and Rehabilitation Medicine in Europe. Europa Medicophysica. 2006;42(4):289-332.
- 17. Koleva IB. Peripheral radicular pain and physical analgesia in neurorehabilitation clinical practice shifting the pain management paradigm from pure pharmacological interventions. Neurorehabilitation.2008;2(1):51-62.(In Bulgarian)
- 18. Boureau F, Willer JC. La Douleur exploration, traitement par neurostimulation et electro-acupuncture. Paris New York Barcelone Milan: Masson; 1979.
- 19. Gildenberg PL, editor. The Chronic Pain Patient: Evaluation and Management. Vol.7 of Pain and Headache, Gildenberg Houston Texas Medical School; 1985.
- 20. Hayes KW. Manual for physical agents. New

- Jersey: Prentice Hall Health; 2003.
- 21. Varkonyi T, Kempler P. Diabetic neuropathy: new strategies for treatment. Diabetes, obesity and metabolism. 2008;10:99-108.
- 22. Duby JJ, Campbell RK, Setter SM, White JR, Rasmussen KA. Diabetic neuropathy: an intensive review. Am J Health Syst Pharm. 2004;61:160-173.
- Johnson M. The clinical effectiveness of TENS in pain management. Critical Reviews in Physical and Rehabilitation Medicine. 2000;12(2):131-149
- 24. Johnson M, Tabasam G. An investigation into the analgesic effects of different frequencies of the amplitude-modulated wave of interferential current therapy on cold-induced pain in normal ubjects. Archives of Physical Medicine and Rehabilitation. 2003;84(9):1387-94.
- 25. Kahanovitz N. Diagnosis and treatment of Low Back Pain. New York: Raven Press; 1991. 145 p.
- 26. Koleva YB, Yoshinov RD, Edreva V, Kouyoumdjieva M. Examens nosometriques chez des patients lombalgiques au cours d'une cure thermale. In: Proceedings of the XI Congress of World Hydrothermal Organization. Istanbul, Turkey, 1992. p.15.
- 27. Koleva IB, Lishev NS, Iochinov RD. Manual-Therapeutic and Kinesitherapeutic Techniques in Patients with Cervically Related Headache. In: Proceedings of 2<sup>nd</sup> World Congress of the International Society of Physical and Rehabilitation Medicine (ISPRM), Praga, Czech Republic, 2003. P.345-50.
- 28. Koleva IB, Milanov IG, Ioshinov RD, Goranova Z. Complex rehabilitation in patients with tension type headache. In: Abstracts of the 36<sup>rd</sup> International Danube Symposium for Neurological Sciences and Continuing Education, Sofia, 2004. p.51.
- 29. La Freniere JG. Le patient lombalgique (techniques de traitements kinesitherapiques). Paris New York Barcelone Milan Mexico Sao Paulo: Masson; 1983.
- 30. Walsh D. TENS: Clinical applications and Related Therapy. Churchill Livingstone; 1997.
- 31. Walsh DM, Foster NE, Baxter GD. Trancutaneous electrical nerve stimulation. Relevance of stimulation parameters to neurophysiological and hypoalgesic effects. American Journal of Physical Medicine and Rehabilitation. 1995;74(3):199-206.
- 32. Gacheva J. Electrophysiological observations on ultrasound effect in some neurological diseases. Ph.D. thesis. Sofia; 1955-1960. (In Bulgarian)
- 33. Gacheva J. Excitomotory electrodiagnostics. In: Ganev G Editor. Clinical electrophysiology. Sofia: Medizina & Fizkultura; 1970. p.193-222. (In Bulgarian)
- 34. Gacheva J. Diagnostics and therapy with low frequency currents. Sofia: Medizina & Fizkultura; 1980. (In Bulgarian)