Novel Approaches for Chronic Pain

Areerat Suputtitada
Faculty of Medicine, Chulalongkorn University & King Chulalongkorn Memorial Hospital, Bangkok, Thailand

*Corresponding author: Areerat Suputtitada, Physiatrist, Faculty of Medicine, Chulalongkorn University & King Chulalongkorn Memorial Hospital, Bangkok, Thailand, Tel: 66814888549; E-mail: prof.areerat@gmail.com; sareerat1111@gmail.com

Received date: May 26, 2016; Accepted date: June 27, 2016; Published date: July 04, 2016

Abstract

‘Chronic pain’ describes a syndrome lasts for more than 3 months. characterized by persistent physical pain, disability, emotional disturbance, and social withdrawal symptoms, existing together and influencing one another in what Bandura termed ‘reciprocal determinism’. If original pain is not treated completely, the repeat pain signals lead to change in the central nervous system becomes hypersensitivity. This is the novel concept of central sensitization for chronic pain. Desensitization of the segment consists of local injection with anaesthetic agents for blocking posterior branch of the dorsal spinal nerve along the related paraspinal muscles, together with local anaesthetic injection directly to the trigger points, Extracorporeal Shock Wave Therapy (ESWT) and High intensity laser therapy for desensitization of trigger point foci and area of sensitization. All are novel therapies for chronic pain.

Keywords: Chronic pain; Central sensitization; Desensitization; ESWT; HILT; Diagnostic ultrasound

Introduction

‘Chronic pain’ describes a syndrome lasts for more than 3 months [1]. Chronic pain management is challenge since complex etiology and inadequately treatment. The duration of ongoing pain is not only the diagnostic criteria. Some physicians advise that any pain that persists longer than the expected healing time for the pathological tissues could be considered as chronic pain [2,3]. "Biopsychosocial systems model" is used for management of chronic pain patients [4]. With the novel of MRI imaging for demonstration of brain area which is activated by stimuli, the pain effect on emotion and the emotion effect on pain can be revealed.

The recently understanding of "central sensitization" is as if initial pain from an injury is not adequately treated, those pain signals are repeatedly sent, which leads to changes in the central nervous system, making it hypersensitive. Sensitization of spinal segments is the main cause of continuous hypersensitive pain in the associated part of the body. The recent concept of chronic pain is the sensitization of spinal nociceptive neurons, with or without the original provoking stimuli. The clinical presentation of dorsal horn sensitization is including dermatomal hyperalgesia, slerotome pressure pain sensitivity and myotome myofascial trigger points, which are all supplied by the sensitized spinal segment. There is significant elevation of the levels of nociceptive pain substances as substance P serotonin, and norepinephrine, calcitonin gene-related peptide (CGRP), bradykinin, tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β) in the area of active myofascial trigger point. There is significantly low pH in the active trigger point. Chronic pain management need to concern all these information, especially in chronic patients with amplified pain responses. The segmental sensitization consists of the nociceptive stimuli in the sensitized areas with sending the sensitization signal to bombard the dorsal horn in the spinal cord. This causes central nervous system sensitization with resulting of dermatomal and slerotome hyperalgesia and spreading from the sensory component of the spinal segment to the anterior horn cells, which control the myotome resulting of myotome myofascial trigger points within the territory of the segmental sensitization [5-10].

The segmental desensitization treatment consists of injection of local anaesthetic agents in the involved dermatome to block the posterior branch of the dorsal spinal nerve along the involved paraspinal muscles. Certainly, local injection with anaesthetic agents is applied peripherally directly into myofascial trigger points. Stretching exercises, local heat application and additional transcutaneous electrical nerve stimulation (TENS) treatment will completely relax the muscles after the injections.

Extracorporeal Shock Wave Therapy (ESWT) also plays a role as desensitization [10-12]. Extracorporeal Shock Wave Therapy (ESWT) can be nowadays approved as an effective, safe, noninvasive therapy for many musculoskeletal diseases and many conditions where regenerative effects are desirable. It is the novel therapy for applications in the fields of regenerative medicine, tissue engineering and cell therapies [11-14].

ESWT consists of biphasic acoustic energy that arises from positive high peak pressures to negative phase with the short rising times and short duration. The generation of focused and radial shockwaves are different. The focused shock waves will generate single acoustic pulses by electrohydraulic principle, electromagnetic principle or piezoelectric principle. The acoustic pulses are converted into a focused acoustic pressure wave with the highest pressure at the target pathological tissue. The radial shock waves will generate a projectile pressure wave within a guiding tube that attacks a metal applicator targeted on the pathological tissue. The energy of focused shock waves decreased within the target tissue consists of bone, calcifications, water, etc., more than 50% in occasionally, whereas consistent energy flux density was found in radial shock wave [12-14].

Shock waves effect on soft tissue and musculoskeletal tissue are as the follows; (1) increase cell membrane permeability and microscopic circulation of tissues which enhance the metabolism, healing and dissolution of calcification; (2) cavitation bubbles which are the result
of high pressure energy wave will expand to a maximum size, then collapse, like a bubble popping creating the high force for breaking down the calcification deposit in soft tissues; (3) the micro jets which are the smaller secondary energy force after cavitation bubbles collapse also breaks down the calcification; (4) thousands of cavitation bubbles formed from several thousand shockwaves being treated at the injured tissues can breakdown calcification deposit in joints, soft tissues and spur; (5) enhance the healing process of bone by stimulation of osteoblasts; (6) enhance the healing process of connective tissues such as tendon, ligaments, and fascia by stimulation of ESWT has also been shown to stimulate fibroblasts [13,14].

In myofascial fascial pain syndrome, the mechanotransduction effect of ESWT may increase perfusion and promote angiogenesis. Moreover, there are evidences of free nerve endings degeneration and transient dysfunction of nerve excitability at the neuromuscular junction, a pure mechanism of breaking-up the actin myosin links with the overstimulation lead to a diminished transmission of pain signals to the brainstem. For pain transmission, in animal studies revealed substance P, calcitonin gene-related peptide (CGRP) expression in the dorsal root ganglion and on neurovascular sprouting [12-22].

There are evidences of improving motor dysfunction, slower the joint degeneration and amelioration of pain in osteoarthritis by ESWT. The mechanisms are as (1) Inhibition of nitric oxide production in knee synovia and reduce chondrocyte apoptosis; (2) Reduction of cartilage degradation biomarkers as shown in Mankin score and Safranin O stain with reduction of matrix metalloproteinase 13 (MMP-13), type II collagen, nitric oxide, DKK-1 (Dickkopf WNT Signaling Pathway Inhibitor; (3) Promote bone healing and tissue repair with ingrowth of neovascularization and upregulation of angiogenic and osteogenic growth factors, such as vessel endothelial growth factor (VEGF), proliferating cell nuclear antigen (PCNA), and bone morphogenetic protein 2 (BMP-2), osteocalcin.

In spastic pain, the mechanism of ESWT in relief of spasticity might be as follows: (1) the generation of nitric oxides play important role in neurotransmission at neuromuscular junction, memories, and synaptic plasticity in the central nervous system; (2) the effect on excitability of the spinal cord; (3) the effect of mechanical vibration; (4) the effect on the Golgi tendon organ; (5) the passive stiffness of muscles determined by inactive connective tissues [12-14].

Established PRM indications are as the follows; Myofascial pain/trigger points, Tendinopathies, Calcifying tendonitits of the shoulder, Sub acromial pain syndrome, Primary long bicipital tenosynovitis, Tennis elbow (epicondylitis humeralradialis), Golfer’s elbow, Greater trochanteric pain syndrome, Patellar tendinopathy, Achilles tendinopathy, Plantar fasciopathy, Idiopathic low back pain/ pseudoradicular syndrome, Idiopathic cervical pain, Osgood Schlatter disease, Pseudarthrosis/nonunion. Emerging PRM indications are persistent pain after partial or total joint replacement, primary osteoarthritis, painful neuropathy, secondary lymphedema, spasticity, acute and chronic soft tissue wounds [12-14].

The advantage for treatment with ESWT are as the follows: (1) effectively relieves pain in more than 80 percent of patients even after just three treatments; (2) can replace surgery in many cases of diseases of the musculoskeletal system; (3) requires compliance by the patient that can easily be achieved (three times five to ten minutes treatment, usually once a week); (4) can be fully performed on an outpatient basis; (5) can be combined with other PRM treatments; (6) No medication; and (7) Gentle and effective [12-14]. Contraindications of ESWT are treatment over air-filled tissue (lung, gut), tissue with local tumors or local bacterial and/or viral infections, pre-ruptured tendons, pregnant women, patients under the age of 18 (except of the treatment of Osgood-Schlatter), patients with blood-clotting disorders (including local thrombosis), patients treated with oral anticoagulants, patients treated with local cortisone injections [12-14].

High Intensity Laser Therapy (HILT) is another one novel therapy for chronic pain. The term laser originated as “light amplification by stimulated emission of radiation.” The basic principle of laser devices are the amplification of electron spin rates by passing photon energy through a particular medium to produce a single directional laser beam having a different wavelength than the original light beam. Recently, high-intensity laser therapy (HILT) is increasing evidences. The privilege of HILT over Low Laser Light Therapy (LLLT) is able to reach and stimulate the large and/or deep joint [23,24]. Previous studies describe the anti-inflammatory, anti-edematous, and analgesic effects of HILT [23,24]. It has been used to provide relief from the symptoms of shoulder pain [23], chronic ankle pain [24], and low back pain [25]. In the present study, HILT results were superior to those of LLLT in pain relief and improvement of function.

This is supported by the belief that the effect of laser, which can alter cellular and tissue function [25]. Ultrasound guidance nerves block is another novel intervention which increasing practices and evidences in diagnosis of facet joint pain. Therapeutic blocks with only local anesthetics (greater occipital nerve and sphenopalatine ganglion) are effective in headache, whereas their efficacy for other chronic pain conditions have not been adequately [26-28].

Introducing the functional context of movement early in musculoskeletal rehabilitation may lead to greater movement gains and earlier cortical recovery, as CNS changes are greater with task specific training [29-31]. Repetitive movement can induce cortical changes such as the use of shoulder taping to facilitate pain-free repeated use of upper limbs during functional tasks throughout the day. When pain prevents a patient from performing movements, physical repetitions may be too limited to induce neuroplasticity changes and mental imagery may be used. These novel treatments being used in musculoskeletal rehabilitation will induce CNS plasticity [29,32].

Diagnostic ultrasonography (US) has shown promising results to identify and differentiate musculoskeletal pain. Diagnostic US has recently been used to identify trigger points because of its ability to characterize viscoelastic properties of myofascial tissue and identify high resistance arterial flow at trigger point sites [33-35].

In conclusion, novel approaches in chronic pain could be of any individual treatment. The underlying neurophysiological mechanisms that enhance the adaptive sensorimotor changes associated with musculoskeletal dysfunction are essential part in clinical practice. Novel of Desensitization techniques and the potential for neuroplasticity with specific interventions are well evidences support.

**Conflicts of Interest**

The author declares no conflicts of interest.
References

1. International Association for the Study of Pain. Classification of chronic pain: introduction. Pain 1986; 24: S3–S8.
2. Bandura A (1978) The self-system in reciprocal determinism. Am Psyche 33: 344-358.
3. Hyland’s-White N, Duarte RV, Raphael JH (2016) An overview of treatment approaches for chronic pain management. Rheumatol Int.
4. Jacobson L, Mariano A (2001) General considerations of chronic pain: Loser J Bonica’s management of pain. (3rd edn), Williams and Wilkins pp: 241-254.
5. Shah JP, Thaker N, Heimur J, Aredo JV, Sikdar S, et al. (2015) Myofascial Trigger Points Then and Now: A Historical and Scientific Perspective. PMR 7: 746-761.
6. Zhuang X, Tan S, Huang Q (2014) Understanding of myofascial trigger points. Chin Med J (Engl) 127: 4271-4277.
7. van Wilgen CP, Keizer D (2012) The sensitization model to explain how chronic pain exists without tissue damage. Pain Manag Nurs 13: 60-65.
8. Shah JP, Gilliams EA (2008) Uncovering the biochemical milieu of myofascial trigger points using in vivo microdialysis: an application of muscle pain concepts to myofascial pain syndrome. J Body Mov Ther 12: 371-384.
9. Lavelle ED, Lavelle W, Smith HS (2007) Myofascial trigger points. Med Clin North Am 91: 229-239.
10. Suputtitada A (2015) Spinal segmental sensitization and myofascial pain syndrome: Evidences and experiences. Int J Phys Med Rehabil 3: 4.
11. Saggini R, Di Stefano A, Saggini A, Bellomo RG (2014) Clinical application of shock wave therapy in musculoskeletal disorders: part II related to myofascial and nerve apparatus. Eur J Phys Rehabil Med 50: 217-230.
12. Suputtitada A, Schmitz C (2016) Extracorporeal Shock Wave Therapy (ESWT) in Musculoskeletal Disorders.
13. Schmitz C, Nikolaus BM, Stefan M, Matthias S, Nicola M, et al. (2015) Efficacy and safety of extracorporeal shock wave therapy for orthopedic conditions: a systematic review on studies listed in the PEDro database. Br Med Bull 116: 115-138.
14. Suputtitada A (2016) Extracorporeal Shock Wave Therapy (ESWT) in Physical Medicine and Rehabilitation conditions.
15. Ramon S, Glezit M, Hernandez L, Romero LD (2015) Update on the efficacy of extracorporeal shockwave treatment for myofascial pain syndrome and fibromyalgia. Int J Surg 24: 201-206.
16. Zhao Z, Jing R, Shi Z, Zhao B, Ai Q, et al. (2013) Efficacy of extracorporeal shockwave therapy for knee osteoarthritis: a randomized controlled trial. J Surg Res 185: 661-666.
17. Wang CJ (2012) Extracorporeal shockwave therapy in musculoskeletal disorders. J Orthop Surg Res 7: 11.
18. Notarnicola A, Moretti B (2012) The biological effects of extracorporeal shock wave therapy (eswt) on tendon tissue. Muscles Ligaments Tendons J 2: 33-37.
19. Speed J (2014) A systematic review of shockwave therapies in soft tissue conditions: focusing on the evidence. Br J Sports Med 48: 1538-1542.
20. Zhang D, Kearney CJ, Cheryian T, Schmid TM, Spector M (2011) Extracorporeal shockwave-induced expression of lubricin in tendons and septa. Cell Tissue Res 346: 255-262.
21. Schmitz C, DePace R (2009) Pain relief by extracorporeal shockwave therapy: an update on the current understanding. Urol Res 37: 231-234.
22. Louwerens JK, Veltman ES, van Noort A, van den Beikerom MP (2016) The Effectiveness of High-Energy Extracorporeal Shockwave Therapy Versus Ultrasound-Guided Needling Versus Arthroscopic Surgery in the Management of Chronic Calcific Rotator Cuff Tendinopathy: A Systematic Review. Arthroscopy 32: 165-175.
23. Santamato A, Solfrizzi V, Panza F, Tonioli G, Frisardi V, et al. (2009) Short-term effects of high-intensity laser therapy versus ultrasound therapy in the treatment of people with subacromial impingement syndrome: a randomized clinical trial. Phys Ther 89: 643-652.
24. Saggini R, Bellomo RG, Cancelli F (2009) Hillarapia and chronic ankle pain syndromes. International Journal of Information and Scientific Culture 3: 22-25.
25. Alayat MS, Alia AM, Ali MM, Shosha TM (2013) Long-term effect of high-intensity laser therapy in the treatment of patients with chronic low back pain: a randomized blinded placebo-controlled trial. Lasers Med Sci 29: 1065-1073.
26. Zhou R, Gordon DB, de Leon-Casasola OA, Rosenberg JM, Bickler S, et al. (2016) Management of postoperative pain: a clinical practice guideline from the American Pain Society; the American Society of Regional Anesthesia and Pain Medicine, and the American Society of Anesthesiologists’ Committee on Regional Anesthesia,Executive Committee, and Administrative Council. J Pain 17: 131-157.
27. Gordon DB, de Leon-Casasola OA, Wu CL, Sluka KA, Brennan TJ, et al. (2016) Research gaps in practice guidelines for acute postoperative pain management in adults: findings from a review of the evidence for an American Pain Society Clinical Practice Guideline. J Pain 17: 158-166.
28. Michele C (2016) Regional anesthesia in pain management. Curr Opin Anesthesiol.
29. Pelletier R, Higgins J, Bourbonnais D (2015) Addressing Neuroplastic Changes in Distributed Areas of the Nervous System Associated With Chronic Musculoskeletal Disorders. Phys Ther 95: 1582-1591.
30. Nils J, Van Houdenhove B, Oostendorp RA (2010) Recognition of central sensitization in patients with musculoskeletal pain: Application of painneurophysiology in manual therapy practice. Phys Ther 15: 135-141.
31. Saab CY (2012) Pain-related changes in the brain: diagnostic and therapeutic potentials. Trends Neurosci 35: 629-637.
32. Snodgrass SJ, Henehan NR, Tsao H, Stanwell PT, Rivett DA, et al. (2014) Recognising neuroplasticity in musculoskeletal rehabilitation: a basis for greater collaboration between musculoskeletal and neurological physiotherapists. Man Ther 19: 614-617.
33. Kumbhare DA, Elizab AH, Nowsorthy MD (2016) Assessment of Myofascial Trigger Points Using Ultrasound. Am J Phys Med Rehabil. 95: 72-80.
34. Sikdar S, Shah JP, Gebreab T, Yen RH, Gilliams E, et al. (2009) Novel applications of ultrasound technology to visualize and characterize myofascial trigger points and surrounding soft tissue. Arch Phys Med Rehabil 90: 1829-1838.
35. Turo D, Otto P, Shah JP, Heimur J, Gebreab T, et al. (2013) Ultrasonic characterization of the upper trapezius muscle in patients with chronic neck pain. Ultrasound Imaging 35: 173-187.