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임상 전기진통치료의 개요

포항성모병원 물리치료실

김 태 열

Introduction of Clinical Electro-Analgesia Therapy

Kim, Tae Youl, M. P. H., R. P. T.

Dept. of Physcial Therapy, Pohang St. Mary's Hospital

PAIN

Definition

Pain is always sujective. Each individual learns the application of the word through experiences related to injury in early life.

Biologists recognize that stimuli that cause pain are likely to damage tissue. Accordingly, pain is the experience that we associate with actual or potential tissue damage. It is unquestionably a sensation in a part or parts of the body, but it is also always unpleasant and therfore also an emotional experience.

Causes

The phenomenon known as pain, wether it be acute, chronic, superficial, deep, visceral, somatic, or referred, has numerous etiologies but generally falls into three categories : physical(trauma, heat, and cold), physiological (inflammation, spasm, ANS dysfunction), psychologic(emotional).

Pain-Spams-Dysfunction cycle

When trauma is sustained, a painful response is frequently noted. This pain precipitates measures by the body to protect itself by guarding, frequently noted as muscle spasm. Guarding promotes subsequent dysfunction of the soft tissue and the joints of the area being guarded.

PAIN MECHANISMS

Algesic substances

Tissue damage caused by injury, disease, or inflammation releases endogenous chemicals, called algogenic, algesic, or pain-producing substances, into the extracellular fluid that surrounds the nociceptos. These substances include H^+, K^+, seotonin, histamine, prostaglandins, bradykinin, substnace P, and many others.

Nociceptive primary afferents

Tissue damage activates distinct types of receptors, called nocicpetors, which are at the termination of free nerve endings of A-delta and C afferents, located in various body tissues. The skin is supplied by A-delta high threshold mechanorecpetors(HTMs) activated by mechanical noxious stimuli;by A-delta myelinated mechanothermal nocicpetors(MMTNs) activated by noxious heat and noxious mechanical stimuli;by C polymodal nociceptors(CPNs) activated by mechanical, thermal, chemical noxious stimuli;and by a miscellameous group consisting of C mechanical nociceptors and cold nociceptors. Deep somatic structures are supplied by C(group Ⅳ) and also probably by A-delta(group Ⅳ) fibers.

Viscera are also supplied by C afferent fibers, and some also by A-delta afferents, which activated by disease, inflammation, contraction under isometric conditions, ischemia, rapid distention, and other adquate visceral nociceptive stimuli. These nociceptors, aciva-ted by noxious stimuli and endogenous algogenic substances, transduce the stimuli into nociceptive impulses that are transmitted to the dorsal horn of spinal cord or the medulla.

Transmission of painful impulse

The activation of nociceptors results in generalized activity in finely myelinated A-delta and unmyelinated C fibers that project to the dorsal horn of the spinal cord or to the medulla. Peptides such as sP and calcitonin-gene-related peptide(CGRP) have been strongly implicated as neurotransmitters of primary nociceptive afferents.

Nociceptive afferents and the dorsal horn

Nociceptive afferents contact second order neurons in the dorsal horn of the spinal cord or medulla. Inhibitory interneurons are present in the dorsal horn;they play an important role in modulating the effectiveness of nociceptive afferent input from the periphery. CPN afferents synapse exclusively in laminae Ⅰ, $Ⅱ_o$, and Ⅴ of the dorsal horn;A-delta nociceptors terminate in laminae Ⅰ and $Ⅱ_o$, but also penetrate deeper to end in laminae Ⅴ and X. The synaptic endings of primary afferents in the dorsal horn contain various types of vesicles and excitatory neurotransmitters.

Ascending pain pathways

The ascending pathways in the anterolateral fasciculus(ALF) are the primary pathways for transmission of nociceptive information from the body to the brain(spinothalamic tract[STT], spinoreticular tract[SRT], spinomesencephalic tract[SMT]). The trigeminal system has similar anatomic and physiologic characteristics, the neotri-geminothalamic tract(nTTT) projecting to the ventroposteromedial thalamic nucleus(VPM) and the paleotrigeminothalamic tract(pTTT) projecting to the same medial thalamic nucelei, where they make contact with neurons that project to limbic forebrain structures and have diffuse projections to other parts of the brain. In addition to transmitting nociceptive impulses, these tracts also transmitt other sensory information. There is evidence that the dorsal column postsynaptic system(DCPS), spinocervical tract(SCT), and multisynaptic ascending system(MAS) may also have a role in nociception.

Supraspinal mechanisms

Pain is a multidimensional experience that includes somatic sensory events in terms of space, time, intensity, and submodality and is associated eith aversive motivational-emotional mechanisms leading to escape and other forms of aversive behavior.

Three major psychologic dimensions of pain;

a) Sensory-discriminative dimension
b) Motivational-affective dimension
c) Cognitive-evaluative dimension

Descending pain control system

The models of the descending inhibitory systems have evolved in four regions of the central nervous system: a)cortical and diencephalic level, b)mesencephalic level(PAG and PVG), c)parts of the rostroventral medulla level(NRM), and d)The spinal and medullary dorsal horn level. These descending fibers are serotonergic and terminate among nociceptive transmission cells in laminae I, II_0, and V, and thus selectively inhibit nociceptor neurons, including interneurons and the rostrally projecting STT, SRT, and SMT. There is also evidence that norepinephrine-containing neurons originating in the locus coeruleus and other brainstem sites contribute to this endogenous pain system.

Neurochemistry of the endogenous opiate system

The discovery by Hughes and collaborators of the two penta amino acid peptides, leu-enkephalin and met-enkephalin, gave rise to the suggestion that these ligands, found in high concentration at the spinal and medullary dorsal horn and in various other parts of the CNS, might be the agents that are released to act on intrinsic opioid systems. β-endorphin was later isolated from the periphery, and in 1797 the discovery of dynorphin and related opioid peptides, three classes of opioids are currently knwon : the enkephalins, dynorphins and β-endorphins, representing three distinct families of opioid peptides, each class cleaved from a different precursor and each having a distinct anatomic distribution.

Non-opiate endogenous inhibitory system

The agents and terminals of descending systems that originate in raphe nuclei and medulla are primarily monoaminergic and release serotonin(5-HT), norepinephrine(NE), and, of course, less importantly, enkephalin and other peptides.

ELECTRICAL ANALGESIC

The analgesia obtained using clinical electro-analgesia(CEA) therapy is like an electrical analgesic such as non-steroid antiinflammatory agents(NSADs) and/or narcotic agents. This analgesia occurs by activation of pain control mechanisms that cause an endorphins release and gate control effect, to suppress or shut off pain signals from reaching the brain. This has a neurochemical and neurophysiological effect of suppressing pain conducting signals at nerve junctions and to unlearn the feeling of pain.

PAIN CONTROL MECHANISMS IN CLINICAL ELECTRO-ANALGESIA

Intracellura mechanism

There are some trillion cells in the body. The chemicals, potassium and calcium, cause cells to act as miniature batteries. Body cells are the basic building blocks of our body, with each cell acting with others to provide specific interlocking and/or discrete functional activities for our bodies well being. Any malfunction of the body such as injury or disease upsets this co-operation between the cells, causing a shift to a higher positive charge level of bio-electrical activity in the tissues surrounding and within the injured or debilitated regions. The correction of this positive potential imbalance requires the corrective functions of exercise, an increase in blood supply and/or rest to the region. The supply of nutrients to, and removal of waste productis from body cells, occurs across the cell menbranes through channels. This movement in the channels occurs from the ionisation of calcium with potassium acting as the conducting medium for the exchange of nutrients and removal of waste products.

Electrical nerve block mechanism

Medium frequencies inhibit nerve conduction based on the fact that they cause temporary nerve menbrane depolarization while present. This effect is knwon as Wedensky inhibition. Medium frequency currents have an inhibitory effect on pain transmission and sensation within the field of treatment. This effect is responsible for the decreased sensation under the stimulation electrodes.

Medium frequencies are also selected due to their excellent tissue penetration. This occurs as a result of the decreased tissue resistance at higher frequencies.

Gate control mechanism

A gating effect obtained using electrical stimulation is a quick acting effect. It occurs because the introduced stimulation acts as a counter to the stimulus causing the pain, by blocking it from resistering. It switches off painful sensations at hypothetical pain control gates in the CNS, therby achieving a pseudo mechanical effect knwon as gating or blanketing. The theory of gating is that a pain control gate is closed by the hyper-activation of neural sensory potentials within A fibres, which overrides the slow velocity pain conducting neural potentials transmitted in the C and A fibres. The function of A fibres is to transmit non-painful sensation at high velocity. This high velocity enables the use of high pulse rates of 100 to 400 pps to maximise a strong gating effect. A gating effect is achieved using short periods of strong stimulation. This is the most common from of electrical induced analgesia, but it is not necessarily the best, because it only controls pain for a short period.

Descending pain control mechanism

During the 1970s, exciting discoveries were made concerning the biochemistry of the descending inhibitory system. In this system, the midbrain's PAG, which has inputs from the thalamus, the hypothalamus, the amygdala and the frontal cortex, projects to the medullary situated nucleus raphe magnus(NRM) and nucleus reticularis gigantocellularis (NRG). Serotinergic axons from these latter structures descend in the dorsolateral funicu-

lus to end in synaptic contact with enkephalinergic(ENK−nergic) interneurons situated on the dorsal horn. The ENK−nergic inhibitory interneurons situated on the border of laminae I and II of the dorsal horn not only block C afferent transmission as a result of A−delta afferent activity causing the descending inhibitory system to come into action in the manner just described. These interneurons also do this because A−delta nerve fibres make direct intraspinal contact with them in the dorsal horn. In addition, ENK−nergic interneurons present on the terminals of C afferent fibres exert a presynaptic inhibitory effect. It therefore follows that the non−invasive acupuncture technique of stimulating A−delta nerve fibres with electrical currents relieves C afferent transmitted tissue damage type pain as a result of this kind of stimulus evoking activity in opioid peptide mediated pain modulating mechanisms situated at both supraspinal and spinal levels.

The medication effect an β endorphin release has a slow acting effect. Electrical stimulation is applied at a low pulse rate of less than 10 pps. The introduced electrical stimulation activates neural potentials within A δ fibres, which transmit at the slow rate of the ANS, to activate the opioid mediated analgesia system mechanism. Aδ fibres stimulation requires a longer application time of 20 minutes to 2 hours to reach a maximum level of β endorphin release, but because β endorphins remain at effective levels in the blood stream for extended periods, a pain relief period of up to 36 hours may be achieved. Sustained stimulation at low levels of pulse intensity has the strongest effect on managing chronic pain. β endorphins flow through the circulatory system acting like pain medication, inhibiting pain message transmission at nerve junction throughout the body.

An enkephalin release also occurs in response to hyperactivation of Aδ fibres. Enkephalins act similarly to endorphins by flowing within the blood stream and have a short medication type life cycle of less than two hours. A enkephalin release is required for strong acute pain management an in exceptional circumstances the condition may need ongoing pain mangement.

MODALITIES AND PROCEDURES

New modalities & procedures

Auricular Electroacustimulation(AEAS)

Minor energy system(Odontons, Hand & Foot, Orbit, etc.)

Pain control mechanism : Descending pain control mechanism ECIWO biology

Wave form : DC monopolar or bipolar square wave

Pulse rate : 1 to 160 Hz

Pulse width : 0.5 sec.

Output intensity : 1 to 200 μA. Increase intensity to a slight burning, but not painful sensation

Application technique : Dry metal special probe on the bio−holographic points

Point selection : The best results are obtained by stimulation of a few, well chosen, points exhibiting hyper−sensitivity to electrically or pressure

Stimulation time : 30 to 60 seconds per point

Treatment interval : 1 to 2 weeks between treatments in most conditions. 2 to 3 times per weeks(acute).

Total number of treatments is 3 series of 10 treatments

Onset of pain relief : Very rapid or immediate post treatment response

Duration of pain relief : Continued improvement in the hours and days following treatment

Electrical Hyperstimulation Analgesia(EHA) /Electroacustimulation(EAS)

Pain control mechanism : Descending pain control mechanism

Wave form : DC monopolar or bipolar square wave

Pulse rate : 4, 100, or 1,000 Hz

Pulse width : 125 ms

Output intensity : 1 to 2,500 μA. Increase intensity to hot, needle-like sensation

Application technique : Dry metal trigger probe on acupuncture and trigger points or painful area or points

Stimulation time : 30 to 60 seconds per point

Treatment interval : 3 times weekly or as determined by patient response

Onset of pain relief : 50~100% reduction of pain immediately post treatment

Duration of pain relief : days, weeks, and sometimes permanently Electrical Nerve Block (ENB) analgesia

Pain control mechanism : Electrical nerve block mechanism

Wave form : AC biphasic square wave

Pulse rate : 10,000 Hz continuous

Pulse width : 15 ms

Output intensity : 1 to 5 mA with intensity increased to maintain the desired perception od stimulation

Sensation : Mild, almost impercevable tingling sensation

Electrode placement : Over the local peripheral nerve(s) innervating the painful area

Stimulation time : For as long as relief is desired

Onset of pain relief : Very rapid

Duration of pain relief : Brief. lasting only during stimulation

Cerebral Electrotherapy(CET)

Electroacusleep therapy

Pain control mechanism : Stimulation-produced analgesia(SPA)

Wave form : DC monopolar or bipolar square wave

Pulse rate : 1 to 120 Hz(usually 8 Hz)

Pulse width : 62.5 ms

Output intensity : 25 to 100 μA

Sensation : Imperceivable(subsensory level)

Electrode placement : Ear clips on lobules bilaterally or head belt on front or occipital region

Stimulation time : 15 to 30 minutes

Onset of pain relief : Rapid. 20~30 minutes

Duration of pain relief : 3 to 8 hours

Transcranial Electrical Stimulation(TES)

Pain control mechanism : SPA

Wave form : DC monopolar or bipolar square wave

Pulse rate : 8 to 20 Hz

Pulse width : 15 usec.

Output intensity : 1 to 5 mA

Sensation : Imperceivable(subsensory level)

Electrode placement : Electrodes positioned on the forehead and GV-16 for the first 10 minutes. Follow with a bilateral transcranial application for the second 10 minutes

Stimulation time : 10 to 20 minutes

Treatment interval : Twice daily for 2 weeks or at initial onset of symptoms as needed

Onset of pain relief : Rapid. 30 minutes

Duration of pain relief : 3 to 8 hours

Microcurrent Electrical Neuromuscular Stimulation(MENS)

Pain control mechanism : Intracellular mechanism

Wave form : DC monopolar or bipolar square wave

Pulse rate : 0.1 to 30]0 Hz

Pulse width : 1.25 second to 0.5 ms

Output intensity : 1 to 600 μA

Application technique : Enhancement of Muscle Re-education(EMR)

Enhancement of Tissue Repair(ETR)

Onset of pain relief : Rapid or immediately post treatment

Duration of pain relief : 2 to 4 hours

Classical modalities & procedures

Convesional TENS Analgesia

Pain control mechanism : Gate control mechanism

Wave form : Variform

Pulse rate : 80 to 120 Hz

Pulse width : 80 to 100 usec.

Output intensity : Comfortable intensity (Submotor threshold)

Application technique : Surface electrodes on dermatome, paraspinal, peripheral nerve pathway, painful area, etc.

Onset & duration of relief : Rapid acting analgesia

Acupuncture like TENS Analgesia

Pain control mechanism : Descending pain control mechanism

Wave form : Variform

Pulse rate : 1 to 5 Hz

Pulse width : 300 to 600 usec.

Output intensity : Adjust for slight motor contraction

Application technique : Surface or spot electrodes on trigger, motor, acupuncture points

Onset & duration of relief : Long lasting analgesia

INDICATIONS AND CONTRAINDICATIONS

indications

1. The CEA system is used for the symptomatic relief and management of chronic intractable pain and as an adjust treatment in acute and post traumatic injury.
2. To increase blood supply through stimulation of muscle contraction causing vasodilation.
3. Reduction of muscle spasm through the analgesic properties of the current.

Contraindications and Precautions

1. For external use only.
2. Interference with pacemakers
3. Keep from chidlren.
4. Skin irritation.
5. Special cases(Pregnant, cardiac disease, etc.).
6. Underlying pathologies.
7. Electrode application.