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임상 전기진통치료의 개요

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Introduction of Clinical Electroanalgesia

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ELECTRICAL ANALGESICS

The analgesia obtained using clinical electroanalgesia(CEA) therapy is like an electrical analgesic such as non-steroid anti-inflammatory agents(NSAIDs) and/or narcotic agents. This analgesia occurs by activation of pain control mechanisms that cause an intracellular mechanism control and endorphins release 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 ELECTROANALGESIA

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.

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 pain-

ful 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. This high velocity enables the use of high pulse rates of 100 to 200 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

The introduced electrical stimulation activates neural potentials within small sensory fibers, which transmit at the slow rate of the ANS, to activate the bodies main pain defence mechanism. Small sensory fibers stimulation requires a longer application time of 20 minutes to 2 hours to reach a maximum level of endorphin release, but because 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. 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 hyper-activation of small sensory fibers. 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.

CLINICAL ELECTROANALGESIA MODULATION

Subsensory pain modulation

Definition;

Subsensory level threshold stimulation is the application of electrical stimulation below sensory perception. Subsensory stimulators are usually incorrectly referred to as microampare current, laser, and pulsed electromagnetic stimulation.

Mechanisms;

Theorized to alter or stimulate cellular metabolism(intracellular mechanism) as well as alter local vascular dynamics via stimulation of autonomic afferents.

Modalities & Procedures;

Microcurrent Electrical Neuromuscular Stimulation(MENS) Laser Pulsed Electromagnetic Stimulation(PEMS)

Clinical indications;

Acute musculoskeletal pain and inflammation, DOMS, wound healing

Nerve block pain modulation

Definition;

Stimulation applied first at sensory levels with progressive accommodation until the patient no longer perceives the current. A sensory and nerve block is created on the pain transmitting A delta and C fibers.

Mechanisms;

Electrical nerve block analgesia is effective in management patients with acute and chronic pain because of the temporarily interruption of nociceptive input at its source or the temporarily blocking of nociceptive fibers that course in peripheral spinal and cranial nerves or of afferent nerve fibers that accompany autonomic nerves. Medium frequencies inhibit nerve conduction based on the fact that they cause temporary nerve membrane depolarization while present. This effect is known as Wedensky inhibition. A brief suprathreshold current, if applied repetitively, causes conduction block at the site of cathodal stimulation after generation of one or more action potentials. This phenomenon, called cathodal block, occurs even when the interstimulus interval exceeds the absolute refractory period. Like the refractory period, it results from inactivation of sodium conductance. A hyperpolarizing current relieves this type of block by reducing depolarization and reactivating sodium conductance.

Medium frequency alternating currents(MFAC) or brief suprathreshold currents have an inhibitory effect on pain transmission and sensation within the field of treatment.

Modalities & Procedures;

Electrical Nerve Block Analgesia(ENBA)

Clinical indication;

Pain management
Reduction of muscle spasm and spasticity
To increase blood circulation

Sensory pain modulation

Definition;

Stimulation applied at sensory levels providing electroanalgesia at the local site of pain or its segmental origin. Also referred to as "Conventional" or "High Rate/Low Intensity" TENS. Brief Intense TENS or High Rate/High Intensity TENS is also included in this section since the parameters are the same but are at maximally tolerated sensory level.

Mechanisms;

Gate control stimulation of A beta fiber causes inhibition of noxious level pain transmission presynaptically on the C fiber.

Recently known to occur as a result of Met-Enkephalin and Dynorphin release presynaptically a the dorsal horn sensory neurons. They bind respectively to the Delta and Kappa receptors and inhibit stimulatory neurotransmitter release in the dorsal horn pain transmission neurons. Brief Intense TENS adds the additional dimension of diversion of patient attention or even counter-irritation to the above mechanisms.

Modalities & Procedures;

High Rate/Low Intensity TENS
Brief-Intense TENS

Clinical indications;

Comfortable modality used for symptomatic pain relief of acute and chronic pain from localized dermal or segmental definition. Brief Intense TENS is used during painful joint mobilization, vigorous stretching techniques, wound debridement, and deep transverse friction massage.

Motor pain modulation

Definition;

Stimulation applied at motor levels provid-

ing electroanalgesia.

Generally aplied to acupuncture points locally and away from the pain site. Sometimes referred to as "Low Rate/High Intensity" TENS.

Mechanisms;

Stimulation of the A delta fiber and muscle ergo receptor causes the release of Pituitary Beta Endorphin and Leu-Enkephalin as well as release of cortisol from the adrenal gland as a result of concomitant ACTH release from the pituitary. The site of action is segmental at Mu receptors(Leu-Enk) and Epsilon receptors (BE), peripheral at sites of inflammation, nerve fibers and their receptor field and throughout the autonomic and immune systems and within the CNS in the thalamus, PAG, and limbic systems(and potentially many more). Beta Endorphin is also produced locally by certain immune cells such as killer cells and T cells and may mediate anti-inflammatory effects during phagocytosis.

Modalities & Procedures;

Low Rate/High Intensity TENS
Burst TENS

Clinical indications;

Chronic pain, especially generalized or of a multi-segmental nature. Following initial inflammatory stage of acute injury to provide analgesia and accelerate tissue repair. Internal organ dysfunction, i.e., dysmenorrhea, abdominal distension, etc.

Motor-Sensory pain modulation

Definition;

Stimulation applied at sensory stimulation(intermediate frequency) followed by motor level stimulation(low frequency) followed by sensory level stimulation(high frequency). The technique takes advantage of all of the mechanisms of both techniques and the effect is further enhanced as a result of nerve receptor synergy.

Mechanisms;

Simultaneous activation of sensory and motor mechanisms acting in a synergistic manner. One should note that a greater analgesic effect is possible by combining sensory and motor stimulation than is obtainable with either modality individually.

Modalities & Procedures;

Sequential TENS

Clinical indications;

All indication of sensory(except brief intense TENS) and motor stimulation. This should be the treatment of choice over either sensory or motor stimulation alone.

Noxious pain modulation

Definition;

Stimulation applied at noxious sensory levels providing electroanalgesia. The sensation generally feels like a hot needle-like sensation. This modality is also referred to as hyperstimulation analgesia and is not the same as brief intense TENS.

Mechanisms;

Triggers central biasing system based on a serotonergic and potential noradrenergic descending inhibitory system. This descending system originates in the reticular formation in the PAG(periaqueductal gray) and the NRM (nucleus raphe magnus) and is triggered by as-

cending noxious signals. This descending signal causing the release of inhibitory neurotransmitters such as dynorphin and enkephalins in the dorsal horn. The relationship of the neural inputs to the areas of analgesia are mapped in the PAG and NRM. This may explain the relationship of various seemingly unrelated acupoints to their areas of action. Stimulation of autonomic sensory afferents produces strong autonomic effects, however the autonomic regulatory mechanisms remain unclear.

Modalities & Procedures;

Electrical Hyperstimulation Analgesia(EHA)

Auricular Electroacustimulation(AEAS)

Electrical Sympathetic Pain Modulation (ESPM)

Clinical indications;

Chronic and acute pain. Neuralgic and autonomically related pain such as reflex sympathetic dystrophy(RSD), phantom and memory pain, headaches, cervical and facial pain, chronic low back and joint pain, dysmenorrhea, epicondylitis and adhesive capsulitis.