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Review article

Microcurrent therapy and the treatment of soft tissue injury

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Abstract

Electrotherapy is a form of rehabilitative treatment where electrical stimulation is used as a form of therapy. Examples of electrotherapy date back to 2500 BC with stone carvings in tombs in ancient Egypt showing patients being treated with catfish capable of producing an electrical charge. Microcurrent therapy, also called "microcurrent electrical neuromuscular stimulation" (MENS) is one of several forms of electrotherapy. A characteristic of microcurrent therapy is that the stimulating current is less than 600 μA and does not cause a contraction in skeletal muscle. Microcurrent therapy presumes the principle that injured tissue produces abnormal electrical potentials, termed "injury potentials" which are associated with a disturbance in homeostasis. In accordance with this theory, microcurrent therapy re-establishes "normal" electrical balance in the tissue and minimizes this disruption, resulting in a more rapid regeneration and return of normal function. Studies have investigated the efficacy of microcurrent therapy treatment on wound healing, and have generally shown that treatment can accelerate the healing process. However, a weakness of many of these studies has been the poor explanation of the treatment modalities, making comparisons between studies difficult. The effect of microcurrent therapy on soft tissue injuries is less well defined. A double-blind placebo controlled clinical trial investigating the efficacy of microcurrent therapy on soft tissue injury showed that treatment of the elbow flexor muscles immediately after the injury, and for four days thereafter reduced the severity of the symptoms of exercise-induced muscle damage. The mechanism explaining these effects is not well understood. Further laboratory and clinical trials are needed to explain the mechanism of action and the evidenced-based prescription of microcurrent therapy for tissue injury. **Keywords**: electrotherapy, muscle, soft tissue injury, sport

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Introduction

Electrotherapy can be defined as the treatment of pain and soft tissue injury with electrical means[1]. The earliest evidence for electrotherapy goes back as far as 2500 BC, with stone carvings in tombs in ancient Egypt showing a species of catfish found in the Nile being used to treat a patient. The catfish (*Malapterurus electricus*) contains organs that produce an electric charge. In 47 AD a Roman physician, Scribonius Largus, reported on a treatment for gout which involved making the patient have physical contact with an electric ray [2].

A more conventional form of electrotherapy was first used clinically in 1745, coinciding with the development of the electrostatic generator. However, the medical profession was generally sceptical about the efficacy of electrotherapy until the 1960s when the "gate control theory" of pain management was published[3]. This theory proposed that selective stimulation of large diameter afferent fibres (group II) could "close the gate" and inhibit incoming nociceptive information, resulting in a suppression of the sensation of pain. The publication and popularisation of this theory re-kindled the interest in electrotherapy and led to the commercial production of electrical stimulators for the treatment of certain muscle injuries [4, 5].

Definition of electrotherapy and evidence for endogenous electrical currents

An electric current is a flow of electric charges. In the body the electrical charges are comprised of electrons, protons, ions and other subatomic particles. An ion is an atom which gains or loses an electron. Positive ions move towards the negative pole which is the source of additional electrons, while negative ions move towards the positive pole where additional electrons are removed. Body tissues are organised systems of cells bathed in a fluid which contains electrolytes (sodium, chloride, and bicarbonate ions) [6]. Therefore electrical charges can be moved and distributed around the body with electrotherapy.

Cells are negatively charged in the central region [1]. The resting potential difference varies in the cells of different tissues, being anything from –60 mV to–90 mV. Scaled up to more familiar household units, this is equivalent to applying 50 000 to 100 000 V across a 1 cm thick insulator! The maintenance of the electrical charge of cells uses about 30% of the metabolic energy of the cell[7].

A continuous current of several microamperes (μA) will flow through an experimental circuit made between an electrode inserted into the dermis and an electrode placed on the surface of the skin. If the dermal electrode is pushed through the successive epithelial cells, the current will rise as the number of cell layers between the electrode tip and the surface increases, becoming maximal as the dermis is reached [8]. There are many other examples of electrical activity in the body. For example, electrical activity can be measured as evoked potentials, in the brain (electroencephalography), in the heart (electrocardiography) and in the skeletal muscles (electromyography). The electrical signals range from 0.1 – 10 μV (evoked potential), 1 – 5000 mV (electroencephalography), 0.1 – 10 mV (electromyography) and 0.5 – 4 mV (electrocardiography) respectively [6].

Types of therapy applied to injured tissue

There are several types of electrotherapy. For example, direct current therapy utilises the principle that particles on or near the skin are given an electric charge, causing ions in the tissues to move. The current passes continuously in the same direction. Muscle stimulating currents and transcutaneous electrical nerve stimulation (TENS) is a therapy where currents are varied either in intensity or direction, at a suitable frequency (50 – 150 Hz) and with a current of 20 – 30 mA. This treatment can disturb the ionic balance across a nerve or muscle membrane causing a nerve impulse or muscle contraction. During interferential therapy, another type of electrotherapy, two currents are passed into the tissue slightly out of phase. They interfere to produce an amplitude modulated current of low frequency which will stimulate nerve and muscle [1].

Microcurrent therapy is sometimes called "microcurrent electrical neuromuscular stimulation" (MENS) or more accurately, "low-voltage pulsed microamperage stimulation". Early microcurrent therapy devices were high-volt, monophasic stimulators (> 150 V), typically with short pulse durations and frequencies of 20-100 Hz. More recent devices are low voltage (about 60 V), with a pulse duration of between 0.5 – 5000 milliseconds, frequencies between 0.5-30 Hz and an average root mean square current of 25-600 μA [9]. Another microcurrent therapy device, developed at Beijeng University in 1985, consists of an electrostatically charged membrane, 8.5 X 15 cm; (Acustat®, TC Corporation, Tustin, CA) which is placed over the skin of the damaged area[10]. The polymer of the membrane stores a strong negative electrostatic charge (approximately 8×10^{-8} coulomb.g^{-1}) which, when in contact with the skin discharges over a 48-h period inducing a flow of electrons into the skin and subcutaneous tissues. The total current flow during this period is 20 μA. Therefore the current for microcurrent therapy is a thousand-fold less than the more conventional TENS treatment, and does not stimulate nerve endings to induce a muscle contraction as occurs with TENS treatment.

Theories in support of microcurrent therapy

The cells within the body can be compared to tiny electrolytic batteries each with a potential current of about 4 pico-amps [11]. It is this intricate electrical system that allows one cell to communicate with another through electromagnetic signalling. In healthy, undamaged tissue the bioelectrical function of these cells is regulated. During injury or disease, it is hypothesised that the bioelectrical function of the cells changes as the injured tissues produce abnormal electrical potentials, termed "injury potentials", which are associated with a disturbance in homeostasis [9]. This is supported in the adaptation of amphibians, such as salamanders, which are able to regenerate new limbs after amputation [12]. For regeneration to occur the wounded cells undergo a systematic restructuring which is initiated by a powerful wound current. Initially this is positive and then becomes strongly negative, before declining after about 35 days, coinciding with a completed regeneration of the new limb [13]. Frogs do not have the capacity to regenerate their amputated limbs; however, if a negative current is applied to the wound, remnants of a stub start to develop. In accordance with this theory a microcurrent applied to the injured tissue corrects the altered bioelectrical communication between injured cells and non-injured cells. A theoretical basis for microcurrent therapy is that the current is designed to mimic the body's own bioelectrical system. Re-establishing "normal" electrical balance in the tissue, through microcurrent stimulation, is intended to minimise this disruption, leading to a more rapid return of function. This theory has not been systematically subjected to rigorous scientific scrutiny and therefore the validity of the arguments supporting microcurrent therapy remains to be verified.

Miscellaneous research on microcurrent therapy

There are not many controlled experiments on the efficacy of microcurrent research and the studies that do exist have variable modes of microcurrent transmission [9]. Most of the research on microcurrent therapy has addressed the efficacy of wound healing after treatment with microcurrent therapy. Microcurrent stimulation resulted in 1.5 to 2

times faster healing and absence of infections in patients with skin ulcers [14]. Byl et al. studied wounds induced in Yucatan mini pigs which were treated either with microcurrent (100 μA, 60V, 0.1Hz administered for 1 hour per day for 5 days) or sham treatment which served as the control [15]. The electrodes (2 X 4 cm) were placed directly over each wound. There were no differences (control vs. microcurrent) in tensile strength, collagen density, collagen maturity, collagen deposition (hydroxyproline), wound size or visual appearance of the wound. These authors concluded that further research is needed to determine whether there is a critical interaction between the size of the electrode relative to the wound, density of the current, and the duration of treatment. A more recent meta-analysis of electrical stimulation on chronic wound healing showed that the rate of wound healing was significantly faster with electrical stimulation (22%) compared to controls (9%), with the results being most effective for pressure ulcers [16]. The study concluded that further research is needed to identify which electrical stimulation devices are most effective.

An *in vitro* model has also been used to study the efficacy of microcurrent therapy [17]. In this study, skin samples from Wistar rats were stimulated with a range of currents (1 – 30 000 μA). A marker for amino acid incorporation into protein (2-^{14}C glycine) increased when the stimulation current was between 10 – 1000 μA. This study also showed that adenosine triphosphate (ATP) concentrations in the skin increased when the stimulation was within 10 – 1000 μA [17]. Although this study has interesting applications and offers some suggestions on the mechanisms of the action of microcurrent therapy, there are no similar studies against which this study can be compared.

There are only two published randomised controlled trials on the efficacy of microcurrent therapy on symptoms of exercise induced muscle damage[10, 181]. In the first study, forty untrained female subjects were randomly assigned to a massage group, microcurrent stimulation group, exercise group or control group after being exposed to a bout of exercise designed to induce muscle damage. The microcurrent treatment consisted of stimulation (30 μA) for 8 minutes through two 4.57 X 4.57 cm electrodes. Treatments were applied immediately after the exercise and again after 24 hours. The control group rested after the exercise. Although there were symptoms of muscle damage in all groups, none of the treatments caused a reduction in any of these symptoms [18].

The next study on the efficacy of microcurrent therapy on exercise-induced muscle damage was done in the authors' laboratory [10]. This was a double-blind placebo controlled clinical trial in which thirty male subjects underwent a series of eccentric actions of the elbow flexor muscles in their non-dominant arm (25 repetitions X 5 sets at 80% of maximum voluntary contraction). Thereafter they were randomly divided into a group which received microcurrent therapy continuously for four days via an electrostatically charged membrane (Acustat®), or a group which received an indistinguishable placebo patch for the same period. Subjects in both groups experienced severe pain and swelling of the elbow flexors, peaking at 48 hours after the exercise. The microcurrent group had less shortening of the elbow flexors after treatment than the placebo group. The maximum voluntary contraction decreased by 25% in the placebo in contrast to the microcurrent group which did not have any decrease in muscle function. The serum creatine kinase activity, a marker of the change in permeability of the muscle cell membrane, was significantly lower in the microcurrent group at 4, 5, 6, and 7 days after the exercise respectively [10].

Discussion

The results of this study clearly show that the treatment of exercise-induced muscle damage with microcurrent therapy reduces the severity of the symptoms. These findings are in contrast to the findings of Weber et al. [18], perhaps as a consequence of the duration of treatment (total of 16 minutes: 8 minutes after exercise and 8 minutes at 24 hours vs. 4 days (5760 minutes) of continuous treatment after the exercise-induced muscle damage). The mechanism of action causing a reduction of the symptoms of muscle damage are unknown but may be related to the maintenance of intracellular calcium

homeostasis. Uncontrolled increases in intracellular calcium may result in the activation of calcium sensitive proteases and phospholipases [19] which can cause an increase in membrane permeability [20]. Sustained concentration of intracellular calcium may also activate nonlysomal cysteine proteases, such as calpain, which cleaves cytoskeletal and myofibrillar proteins [21]. The degradation of these proteins may explain the impaired muscle function which occurred in the placebo group but not the microcurrent group [10]. An *in vitro* study showed that thermal fibroblasts and U937 human monocytic cells, stimulated with microcurrent, secreted the transforming growth factor ß1. These are important regulators of cell-mediated inflammation and tissue regeneration [22]. If these data can be extrapolated to an *in vivo* model, they may provide evidence for a mechanism through which microcurrent therapy may exert its effect in reducing the symptoms in exercise-induced muscle damage.

Summary

1. Microcurrent therapy is one of several forms of electrotherapy.
2. A collective weakness of the studies on the efficacy of microcurrent therapy is the poor explanation of the treatment modalities.
3. There is evidence to suggest that microcurrent therapy administered continuously for four days reduces the symptoms of exercise-induced muscle damage. The mechanism explaining the effects of microcurrent therapy is not well understood.
4. Further laboratory and clinical trials are needed to explain the mechanism of action and the evidenced-based prescription of microcurrent therapy.

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