

**TensCam Low-back Pain
and Scalar Wave Therapy
Clinical Study**

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Orlando, Florida
2002

STUDY DESIGN

A. Treatment groups

This clinical study included 98 subjects, divided equally into an experimental (test) group and a control group.

- Subjects in the test group were treated with the actual TensCam device.
- Control group subjects received a sham treatment procedure using a placebo TensCam device.

Apart whether the actual or placebo TensCam device was used, all subjects received the same treatment protocol.

B. Double-blind study design

In this double-blind study, neither the subjects nor the investigators knew which group (test or control) each subject had been assigned to until after the study was complete.

Each subject was randomly assigned to group A or B by the study monitor. The investigator administered the treatment to each subject using a TensCam device labeled A or B according to this group assignment.

Only the study sponsor knew which label (A or B) corresponded to the actual TensCam device until after the study was complete. The study sponsor ensured that this information was stored and maintained confidentially at the sponsor's work site. This knowledge was not shared with any of the principal investigators or the subjects until the study's final subject data file was completed and submitted for analysis. The monitor also remained unaware of the relationship between the A and B devices and the test and control groups until data analysis became necessary.

Principal investigators and subjects could not detect any difference between the actual and placebo TensCam devices because the two looked identical; they were the same size, shape, color, weight, and emitted the same visible output (a blinking light during activation). The difference between the two units was that in the placebo unit, a "C" cell battery was substituted for the piezoelectric crystal array in the actual therapeutic device. This substitution ensured that no therapeutic output occurred on activation of the placebo device.

C. Randomized treatment group assignment procedure

Subjects were randomly assigned to treatment groups. Once a subject was found eligible to participate in the study and had voluntarily signed the informed consent form, the principal investigator notified the study monitor, who assigned the subject to a group.

Subjects were assigned to the test group or the control group by alternating assignments to each group as subjects completed the eligibility process.

This method guarded against the possibility of disparity between the number of subjects who received the test treatment and those who received the placebo treatment in case the study was terminated early. The alternating group assignments also accounted for any variation in the clinical characteristics of the subjects entering the trial during different time periods.

SUBJECT SELECTION AND GROUP ASSIGNMENT

A. Recruitment of subjects

Subjects were recruited by means of flyers distributed to members of an organization of retired University of Central Florida employees, and Palm Valley, a retirement community in Orlando, Florida.

B. Compensation of subjects

Each volunteer who met the eligibility criteria received \$25.00 for participating in the study.

Subjects were not charged for the evaluation process in the on-site investigator's office or for the treatment procedure with the TensCam device, nor were they charged for the cost of any other directly related evaluations or measurements related to their participation in the study.

C. Sample size

Ninety-eight subjects successfully completed the clinical study:

- 48 in the test group
- 50 in the control group

Testing was done at two locations with an approximately equal number of subjects participating at each site.

D. Rationale for sample size

The following parameters were established for assessing the efficacy of the TensCam device in this clinical study.

- Overall study success criteria of at least a 30% difference between groups, comparing the proportion of individual successes in each group.

Individual subject success criteria were defined as a 30% reduction in Degree of Pain rating on the Visual Analog Scale (VAS) for the low-back region from just before starting the treatment procedure to 10 minutes after completing treatment.

It was anticipated that about 50% of subjects in the test group and about 20% in the placebo group would meet the individual success criteria.

REPORTING OF ADVERSE REACTIONS AND EVENTS

No adverse reactions or events were reported.

PRIVACY AND CONFIDENTIALITY

Records for each subject in the clinical study were maintained in a locked filing cabinet at the principal investigator's office. The principal investigator was responsible for ensuring that all subjects' records pertaining to their participation in the study were stored in the file at all times other than when information was being recorded.

Subjects' identities were kept confidential by assigning each subject an identification number when accepted for the study.

MONITORING OF THE CLINICAL STUDY

The monitor made sure each test site and investigator executed the protocol exactly as outlined and intended. This included ensuring that each subject had submitted a signed informed consent form before beginning the protocol, that the protocol was administered as specified, and that all pre- and post-procedure evaluation and measurements were taken using the specified methods and correctly and fully recorded on the appropriate clinical case forms.

DEVICE DESCRIPTION

The Crosby Advanced Medical Systems, Inc. TensCam is a medical device being evaluated for the indication of applying therapy for providing temporary relief of the symptom of low-back pain arising from the musculoskeletal condition of low-back sprain and strain. The TensCam consists of two primary parts:

- The LISS Body Stimulator Model SBL502-B, which has been cleared by the FDA under 510(k) #K902976; and
- A transducer (figure 1) that incorporates a specially selected silicone diode crystal connected electrically to the LISS Body Stimulator.

The TensCam unit is powered by the LISS Body Stimulator, which is essentially a signal generator, through a cable to the transducer in the non-contact mode 18-24 inches from the skin. The electronic waveform of the TensCam is a 15 KHz square wave carrier, which is rectified, varying from zero to a maximum of 4 mA. The first modulating signal of 15 Hz provides an “on” time of 50 msec. and an “off” time of 16.7 msec. The second modulating signal of 500 Hz changes the “on” time series of 15 KHz carrier pulses (750 pulses in 50 msec.) into 25 smaller bursts of 15 pulses each of the 15 KHz carrier (375 pulses in the same 50 msec.).

The subject service is a bipolar version of a Cutaneous Electrical Stimulation (CES) device, wherein the first major burst of energy (50 msec. is positive, followed by 16.7 msec. of “off” time), is followed by a second major burst of energy (50 msec. is negative, followed by 16.7 msec. of “off” time). Thus, the consecutive positive burst and off time is followed by an equal and opposite negative burst and off time, balancing the direct current component to zero.

The pulse period for the basic carrier waveform of 15 KHz is 66.7 usec (50% duty cycle).

The pulse period for the first modulator of 15 Hz is 66.7 msec. (75% duty cycle).

The pulse period for the second modulator of 500 HZ is 2 msec. (50% duty cycle).

The output is variable from zero to 40 volts and then voltage limited, first positive and then negative. Therefore, load impedances of up to 10 KO will be able to have constant current up to 4 mA. However, beyond 19 KO, the constant current is limited inversely with the load. The load in the TensCam is the coil around the crystal. No direct conduction path is established in the no-touch mode.

The TensCam unit, powered by the LISS Body Stimulator Model Sbl502-B, has only one control, which encompasses both the on/off switch for turning the device on or off, as well as the intensity control for adjusting the level of current output of the SBL502-B.

There are two electrode receptacles, both of which are black. Since the polarity is shifting automatically during every major burst, there is no concern polarity.

There is a red LED indicator, which flashes for the total time the device is turned on, regardless of the intensity of the output setting.

There are four amber LED indicators that flash according to the intensity of the output setting; these are related to the circuit and cable that power the transducer. When the four amber LEDs are ON, the circuit to the transducer is working correctly. If the amber LEDs are not lighted, there is an open circuit.

DETERMINATION OF DEVICE SAFETY

An application for non-significant risk was written to determine whether the use of the device presents a potential for serious risk to the health, safety or welfare of a subject. The device is not intended as an implant, it is not purported or represented to be for use in supporting or sustaining life, and it is not for use of substantial importance in diagnosing, curing, mitigating, or treating disease, or otherwise preventing impairment of human health. Therefore, it has been determined that this will be a non-significant risk clinical study.

To further support the position of the Company that the TensCam is safe, the electrical output of the device is reduced by the transducer to 1/1000 of the output of the LISS Body Stimulator without the transducer.

PRODECURE

ETIOLOGY: INCLUSIVE CONDITIONS

To be considered eligible for participation in this clinical study, a subject must have satisfied each of the following “Inclusive Conditions” criteria.

A. Clinical Condition: Low-Back Pain Originating from Strain/Sprain Injury

All subjects in this clinical study will have low-back pain origination from the musculoskeletal condition of low-back sprain/strain injury, according to the criteria outlined below in “Symptomatology”.

Definition

The majority of low-back pain is caused by strains and/or sprains of the back’s muscles, ligaments and tendons that result when the soft tissues of the lower back are stretched or torn. When these tissues become overstretched or torn, they become inflamed, causing pain. This can result from a sudden injury or from more gradual overuse. The back is

prone to this strain because of its weight-bearing function and involvement in moving, twisting and bending.

Causes

Low-back sprains or strains can be caused by a single event or can be due to repeated small injuries to the back.

Identifying the specific cause of the sprain or strain can often be very difficult. The muscles of the low back provide power and strength for activities such as standing, walking and lifting. A muscle strain can occur when the muscle is poorly conditioned or overworked. A sprain of the low back can occur when a sudden, forceful movement injures a ligament that has become stiff or weak through poor conditioning or overuse. Heavy lifting and twisting, sitting for prolonged periods, and general poor health and fitness also contribute to back sprain.

Symptomatology

For this clinical study, the criteria for determining that a subject's low back pain was a result of sprain/strain injury were:

- a. Subject history attained verbally from the patient.

The investigator interviewed subjects to gather as complete a history as possible on their presenting low-back pain. Issues covered included, but were not necessarily restricted to:

- When the pain began
- How the pain might best be described
- If there had been any changes in the degree or type of pain over time
- Where the pain was greatest
- When the pain was most severe
- What caused the pain to worsen or be relieved
- Whether an injury or surgery had occurred to the lower back region at any time
- Any other factors, such as illness or disease, that affected the pain

The subjects' history information was used to identify sprain/strain injury as the origin of their low-back pain as well as to rule out any other similar potential causes such as a ruptured disk, degenerative disk disease, or fracture of a vertebrae.

The primary subject history symptoms that indicate sprain/strain included:

- Low-back pain matched with an inciting incident (i.e., trauma or accident) or previous occurrences that resolved quickly
- Low-back pain that radiated into the buttocks, but not to the legs
- Stiffness or tenderness in the low-back area, which limited motion

- Inability to maintain normal posture due to stiffness and/or pain
- Low-back pain made worse by activity and usually improved with rest
- Incidence of muscle spasms either with activity or at rest

The primary subject history symptoms that suggested exclusion of sprain/strain injury as the etiology of low-back pain included:

- Low-back pain radiating to the legs
 - Weakness in the legs
 - Low-back pain accompanied by fevers or chills
- b. Medication use: Optimal relief of low-back pain attained through the use of over-the-counter and/or prescription muscle relaxants and/or NSAIDs (if any medications have been taken).

Based on the information collected from a. and b. above, the investigator determined whether a subject's low back pain was caused by sprain or strain.

B. Location of Pain

To participate in this clinical study, a subject must have presented with pain in the low-back region, on the right side or left side, or both sides.

C. Degree of Pain

To be an eligible study participant, a subject must have presented with an initial pre-procedure, self-reported degree of pain rating for the low-back region of 1 or greater on the Visual Analog Scale (VAS) that ranges from 0 (no pain) to 10 (worst pain imaginable).

D. Medication Abstinence

Subjects must not have taken:

- Any over-the-counter medication indicated for pain relief beginning 12 hours before the treatment phase of the study, up to and including the time when their participation in the 24-hour follow-up-to-treatment phase of the study was complete.
- Any prescription medication prescribed by a physician for pain relief beginning 12 hours before the treatment phase of the study, up to and including the time when their participation in the 24-hour follow-up-to-treatment phase of the study was complete.

E. Age

To participate in the clinical study, male subjects must have been 18 years or older and female subjects must have been 45 years or older and not pregnant.

ETIOLOGY: EXCLUSIVE CONDITIONS

Subjects were considered ineligible to participate in this clinical study if they met any of the following exclusive conditions:

A. Clinical Condition

A subject whose symptoms suggested a primary origin for his or her low-back pain that was not sprain/strain injury, as determined by the criteria outlined above in the “ETIOLOGY: INCLUSIVE CONDITIONS” section, was considered ineligible.

B. Location of Pain

A subject who presented with pain not located in the right and/or left side(s) of the lower back was considered ineligible

C. Degree-of-Pain Rating

A subject who presented with an initial pre-procedure, self-reported Degree of Pain rating of less than 1 on the VAS pain scale was considered ineligible.

D. Medication Abstinence

A subject was considered ineligible if unwilling to abstain from over-the-counter medications for pain relief and/or any prescription medications prescribed by a physician for pain relief beginning 12 hours before the treatment phase of the study, up to and including the time when the 24-hour follow-up-to-treatment phase of the study was completed.

E. Age

A subject was considered ineligible to participate in the clinical study if he or she was:

- A male less than 18 years of age
- A female less than 45 years of age
- Pregnant

F. Previous Surgery

A subject with any prior surgical intervention to the lower-back region was excluded from participation in the study. These criteria included any past surgical treatment for the presenting condition of sprain/strain or any other related or non-related disease or injury, or for the insertion of metallic or other hardware in the region.

G. Litigation/Disability/Worker's Compensation

A subject involved in litigation and/or a worker's compensation claim and/or receiving disability benefits related to lower back pain was excluded from participating in the study.

BACKGROUND AND THEORY

The Mechanism of Pain

1. The *Academic Press Dictionary of Science Technology* defines pain as “a relatively localized sensation of discomfort, distress, or agony, resulting from the stimulation of specialized nerve endings.”
2. *Classification of Chronic Pain, Second Edition, IASP* (International Association for the Study of Pain) Task Force on Taxonomy, Merskey and N. Bogduk, editors. IASP Press, Seattle, 1994 defines pain as “unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.”
3. *The Merck Manual, 17th Edition*, describes pain as “an unpleasant sensation signaling that the body is damaged or threatened with an injury.” It further explains, “Pain begins at special pain receptors scattered throughout the body. These pain receptors transmit messages as electrical impulses along nerves to the spinal cord and then upward to the brain. Sometimes the signal evokes a reflex response when it reaches the spinal cord; when this happens, a signal is immediately sent back along motor nerves to the original site of the pain, triggering the muscles to contract. An example of a reflex response is the immediate pull-away reaction upon inadvertently touching something very hot. The pain signal is also relayed to the brain. Only when the brain processes the signal and interprets it as pain does a person become consciously aware of it.”
4. The National Institute of Neurological Disorders and Stroke, *Theories of Pain*, states that “For some time, neuroscientists had known that chemicals were important in conducting nerve signals (small bursts of electric current) from cell to cell. In order for the signal from one cell to reach the next in line, the first cell secretes a chemical, called a ‘neurotransmitter,’ ”

from the tip of a long fiber that extends from the cell body. The transmitter molecules cross the gap separating the two cells and attach to special receptor sites on the neighboring cell surface. Some neurotransmitters excite the second cell, allowing it to generate an electric signal. Others inhibit the second cell, preventing it from generating a signal. When investigators injected morphine into experimental animals, they found that the morphine molecules fit snugly into receptors on certain brain and spinal cord neurons. Why, the scientists wondered, should the human brain ... come equipped with receptors for a man-made drug? Perhaps there were naturally occurring brain chemicals that behaved exactly like morphine.

“Numerous studies around the world led to the discovery of not just one pain-suppressing chemical in the brain, but a whole family of such proteins. The smaller members of the family were named enkephalins (meaning ‘in the head’). In time, the larger proteins were isolated and called endorphins, meaning the ‘morphine within.’ The term endorphins is now often used to describe the group as a whole.

“The discovery of the endorphins lent weight to an overarching theory of pain: endorphins released from brain nerve cells might inhibit spinal cord pain cells through pathways descending from the brain to the spinal cord. Laboratory experiments subsequently confirmed that painful stimulation led to the release of endorphins from nerve cells. Some of these chemicals then turned up in cerebrospinal fluid, the liquid that circulates in the spinal cord and brain. Laced with endorphins, the fluid could bring a soothing balm to quiet nerve cells.”

5. Arthur C. Guyton, MD, Professor Emeritus, Department of Physiology and Biophysics, University of Mississippi Medical Center, a renowned expert on the physiology of pain, states in his book, *Basic Neuroscience, Anatomy and Physiology*, 2nd Edition, published by W.B. Saunders Company, that there are two leading causes of pain: muscle spasm and tissue ischemia:

“Muscle spasm is a very common cause of pain and is the basis of many clinical pain syndromes. This pain results partially from the direct effect of muscle spasm in stimulating mechanosensitive pain receptors. However, it possibly also results from the indirect effect of muscle spasm to compress the blood vessels and cause ischemia. Also, the spasm increases the rate of metabolism in the muscle tissue at the same time, making the relative ischemia even greater, creating ideal conditions for release of chemical pain-inducing substances.

“When blood flow to a tissue is blocked, the tissue becomes very painful within a few minutes. In addition, the greater the rate of metabolism of the

tissue, the more rapidly the pain appears. For instance, if a blood pressure cuff is placed around the upper arm and inflated until the arterial blood flow ceases, exercise of the forearm muscles can cause severe muscle spasm within 15 to 20 sec. In the absence of muscle exercise, the pain will not appear for 3 to 4 minutes.”

One of the suggested causes of pain in ischemia is accumulation of large amounts of lactic acid in the tissues, formed because of the anaerobic metabolism (metabolism without oxygen) that occurs during ischemia. However, it is also possible that other chemical agents such as bradykinin, proteolytic enzymes, and so forth, are formed in the tissues because of cell damage and that these, rather than lactic acid, stimulate the pain nerve endings.”

RESULTS

- The test group of 48 subjects receiving actual TensCam treatments showed 60% improved at 10 minutes and 63% improved at 24-hour evaluation.
- The control group of 50 subjects receiving the sham treatments showed 36% improvement at 10 minutes and 22% at 24-hour evaluation.
- There were no complications or side-effects.

REVIEW OF LITERATURE

Clinical Applications of TensCam-Related Therapies to the Relief of Low-Back Sprain and Strain Pain

Following is a collection of abstracts from clinical studies and other research evaluating the use of therapies similar to TensCam for the relief of low-back pain symptoms predominantly originating from sprain and strain injury.

Transcutaneous electrical nerve stimulation and acupuncture-like transcutaneous electrical nerve stimulation for chronic low-back pain. Gadsby JG, Flowerdew MW. Cochrane Database Syst Rev 2000;(2);CD000210, 47Milton Crescent, Leicestershire, UK, LE4OPA, Joseph, gadsby@virgin.net

One-shot percutaneous electrical nerve stimulation (PENS) vs. transcutaneous electrical nerve stimulation (TENS) for low-back pain: comparison of therapeutic effects. Hsieh RL, Lee WC Am J Phys Med Rehabil 2002 Nov;81(11):838-43.

Dept of Physical Medicine and Rehabilitation, Shin Kong Wo Ho-Su Memorial Hospital, Shin Lin District, Taipei, Taiwan, Republic of China

The effect of stimulus frequency on the analgesic response to percutaneous electrical nerve stimulation (PENS) in patients with low-back pain. *Anesth Analg* 1999 Apr;88(4):841-6
Ghoneim ES, Criag WF, White PF, Ahmed HE, Hamza MA, Gajraj NM, Eugene
McDermott Center for Pain Management, University of Texas Southwestern Medical
Center at Dallas 75235-9068.

Transcutaneous electrical nerve stimulation (TENS): nonparallel antinociceptive effects on chronic clinical pain and acute experimental pain. Cheing GL, Hui-Chan CW. *Arch Phys Med Rehabil* 1999 Mar;80(3):305-12. School of Physical and Occupational
Therapy, Faculty of Medicine, McGill University, Montreal, Quebec, Canada.