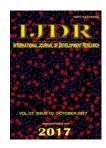


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# **ORIGINAL RESEARCH ARTICLE**

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# COMPARISION OF TWO TREATMENT TECHNIQUES: SHOCKWAVE THERAPY AND ISCHAEMIC COMPRESSION IN SUBJECTS WITH UPPER TRAPEZIUS MYOFASCIAL TRIGGER POINT

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# ARTICLE INFO ABSTRACT

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#### Keywords:

Myofascial pain syndrome (MPS), Myofascial trigger point (MTrP), Muscle Energy Technique (MET), Post isometric relaxation (PIR), Extracarporeal shockwave therapy (ESWT), Ischaemic compression (IC), Pressure pain threshold (PPT), Visual analogue scale (VAS), Range of motion (ROM).

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ischemic compression technique on subjective pain, pressure pain threshold and trapezius flexibility for subjects with upper trapezius muscle MTrP. **Methodology:** 60 subjects both males and females with the mean age of  $(23.46 \pm 2.31)$  were

Aim of the study: The aim of the study was to compare the effects of ESWT technique versus

randomly assigned into 2 groups. Subjects in Group A received shockwave therapy with MET and subjects in Group B received ischemic compression with MET. Treatment was given 5 times a week for 2 weeks.

**Data Collection:** Measurements of PPT by syringe algometer, pain on VAS and neck side flexion ROM by measure tape were taken prior to beginning of treatment (pre-test) and were repeated after completion of 2 weeks treatment protocol (post treatment).

**Data Analysis:** The dependent variables were analysed using a  $2 \times 2$  ANOVA. All pair wise Post- Hoc comparisons were analysed using a 0.05 level of significance.

**Results and Conclusion:** The overall results of this study shows that there was significant reduction in perception of pain in terms of VAS, increase in pressure pain threshold and increase in contra lateral neck side flexion ROM in both the shockwave and ischemic compression groups in subjects with myofascial trigger point in upper trepezius muscle. There wasn't any marked difference in reduction of pain on VAS between the groups, However the improvement in PPT and CROM was significantly more in shockwave group compared to ischemic compression.

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# **INTRODUCTION**

In contemporary society, people are spending increasing amounts of time sitting rather than standing, using the upper limbs more intensively than the lower limbs, and often suffer from pain and fatigue in the musculoskeletal system. Musculoskeletal diseases account for the largest share of functional diseases among people of working age and cause various problems for people in other age groups as well. (Cole and Edgerton, 1990) Especially, neck and shoulder pain are very common. Fricton JR *et al.* reported that myofascial pain syndrome was a primary cause for 55% of them. (Fricton *et al.*, 1985) During the past few decades, myofascial trigger points (MTrPs) and myofascial Pain syndrome (MPS) has received much attention in the scientific and clinical literature. Myofascial pain syndrome may be defined as the sensory, motor and autonomic symptoms caused by myofascial trigger points (MFTP), or the hyperirritable spots within skeletal muscles that are associated with palpable nodules in a taut band. These MFTP are extremely common and become a distressing part of nearly everyone's life at one time or another. (Lavelle *et al.*, 2007) Myofascial pain syndrome is very common in the general population and its incidence can be as high as 54% in women and 45% in men, although the

prevalence of patients with TrPs in the masticatory muscles does not exceed 25%. (Wright, 2000) The most common age at presentation is between 27.5 and 50 years, with preference in sedentary individuals. (Cummings and White, 2001) The majority of publications do not report significant differences between the two sexes, although a greater prevalence in females has been described. (Lavelle et al., 2007; Simons et al., 1999) Simons et al. assumed that the muscle fibres are shortened and taut bands are made by calcium influx in damaged fibres or acetylcholine secretion in motor end plates. In addition to this, the causes of myofascial pain syndrome include injuries or repetitive micro injuries, abnormal stresses due to overuse or mechanical overload of specific muscles and muscle groups, prolonged maintenance of incorrect habitual postures, and mental stress. (Simons, 2010) Recent studies have hypothesized that the pathogenesis results from the overloading and injury of muscle tissue, leading to involuntary shortening of localized fibre. (Mense et al., 2000; Travel and Simons, 1999) The areas of stressed soft tissue receive less oxygen, glucose, and nutrient delivery, and subsequently accumulate high levels of metabolic waste products. The most credible etiological explanation of muscle TrPs is the so called Integrated hypothesis. The end result of this cascade of events is the creation of altered tissue status, pain, and the development of TrPs. (Harden et al., 2000) Trigger Points have been associated with hyperalgesia and limited range of motion (ROM) and are therefore clinically important to identify as these possess the potential to restrict functional activities. Ge HY et al. have provided recent evidence of sympathetic facilitation of mechanical sensitization and facilitation of the local and referred pain reactions in muscle TrPs. (Ge *et al.*, 2006)

According to Travell and Simons, the formation of a trigger point may be due to acute and micro trauma to the muscle. This stress creates a disruption of sarcoplasmic reticulum and releases of free calcium ions. The calcium ions stimulate actin and myosin interaction and also metabolic activity. Increased metabolic activity causes an increase in the release of histamine, serotonin, kinin, prostaglandins, which raises the sensitivity and firing of group III and IV muscle nociceptors which converge with other visceral and somatic input creating perception of local and referred pain. (Schwatz, 1984) The TrP is a site of functional pathology where increased energy consumption is combined with decreased energy supply termed as Energy crises hypothesis. (Simons et al., 2002) This pain in turn stimulates motor unit inducing muscle spasm and splinting causing decrease of blood flow to muscle and decrease ATP, and calcium pump action, which in turns create pain-spasm cycle. This repeated pain-spasm cycle leads to sustained noxious metabolites in the area that build up in connective tissue, creating localized fibrosis nothing but trigger point. (Kuan, 2009; Skootsky et al., 1989) Myofascial trigger point mechanism is closely related to the spinal cord integration. When the input from the nociceptors in an original receptive field persists, central sensitization in the spinal cord may develop and the receptive field corresponding to the original dorsal horn neuron may be expended. Through this mechanism new MTrPs or satellite MTrPs may develop. (Hong, 1994) For a long standing untreated active MTrPs, the irritation from the peripheral nociceptors may be persistent and the expansion of nociceptive field may increase progressively. Finally spontaneous pain may spread to many distant regions in addition to the original reference zone through the mechanism of central sensitization in the spinal cord. (Simons

et al., 2002) Regardless of the underlying mechanism of trigger point origination, the treatment of MPS is usually directed to the trigger point in the palpable taut band aiming at reducing its sensitivity. A clinical relationship between TrPs and joint impairments has been suggested by several authors. (Fernandez-De-Las-Penas, 2009) Lewit emphasized the importance of the treatment of TrPs and joint dysfunctions when both were present. In clinical practice, therapists commonly use a treatment approach that includes different techniques directed at both muscle and joint dysfunction. (Lewit and Simons, 1984) MTrP treatment methods can be divided into invasive and non-invasive methods. Non-invasive method includes manual therapy, electrical treatment and exercise therapy. Manual approaches may include muscle energy techniques (METs), strain-counterstrain (SCS), myofascial release, and ischemic compression. (Victoria et al., 2010) Electrotherapeutic modalities include electrical muscle stimulation, TENS, LASER, Ultrasound, Extracorporeal shockwave therapy and Diathermy. Exercise therapy includes taping, stabilization exercises, PNF and positional release therapy. (Huguenin, 2004; Lee et al., 2013) Extracorporeal shock wave therapy (ESWT) is recently considered an effective treatment for myofascial pain syndrome. It is considered to reduce the pain of myofascial pain syndrome by pain signal alteration, promoting angiogenesis and increasing perfusion in ischaemic tissues induced by sensitization of nociceptors and muscle ischemia. The value of acoustic compression treatment for TrP therapy is that it appears to have very beneficial treatment effect duration, thus minimizing the time interval between subsequent treatments. (Maier et al., 2003; Kuo et al., 2009)

Muscle energy technique (MET) is commonly utilized method for achieving tonus release (inhibition) in a muscle before stretching. The approach involves the introduction of an isometric contraction to the affected muscle producing post isometric relaxation through the influence of the Golgi tendon organs (autogenic inhibition). It may also be applied to the antagonistic muscle group producing inhibition in the offending agonistic muscle(s). It is believed to improve range by causing length changes in the connective tissue structures which undergo shortening in patients with MTP's. (Chaitow, 2013) Sabby et al. conducted a study using MET and demonstrated improved range of motion and reduced pain in patients with upper trepezius MTrP. Simons et al. defined ischemic compression (IC) as "trigger point pressure release" and described it as "Application of slowly increasing, non painful pressure over a trigger point until a barrier of tissue resistance is encountered. Contact is then maintained until the tissue barrier releases, and pressure is increased to reach a new barrier to eliminate the trigger point tension and tenderness." The digital pressure applied to the trigger point causes a temporary ischemia thus depriving the area of oxygen, this causes a reduction in action potentials and blocks noxious sensory afferent input to the higher levels of the nervous system. (Simons et al., 1999) The mechanism employed by the static stretch due to ischemic compression would literally deform the muscle fibre, pulling apart the actin/myosin bridges, restoring the muscle fibre to full length. (Hains, 2002)

### Rationale of the study

Finally, it is currently unknown whether the benefits of ESWT exceed that of IC because no literature focusing on the specific comparison of ESWT and IC exists. There was a need for

further research to compare the effects between these two treatment methods so clinicians have an objective basis for selecting a particular method in treating MTrP. Hence the study was formulated to compare the effects of ESWT technique versus ischemic compression technique on subjective pain, pressure pain threshold and trapezius flexibility for subjects with upper trapezius muscle MTrP.

#### METHODOLOGY

Type of study: Comparative study

Study design: Pre Test and Post Test Study Design.

**Sample size:** A total of 60 subjects were recruited from the physiotherapy department of Swami Vivekanand National Institute of Rehabilitation Training and Research according to the inclusion and exclusion criteria.

#### Sampling method: Random sampling

**Inclusion criteria:** Age 15 - 30 years, Diagnosis of trigger point in upper trapezius (Unilateral) according to criteria given by Simons *et al.* a taut palpable band in the affected muscle, exquisite focal tenderness to digital pressure (the trigger point), in the taut band of the muscle, a local twitch response, elicited through snapping palpation or needling of the tender spot, reproduction of typical referred pain pattern of TP in response to compression, unilateral upper trapezius shortness. The ROM of the side contralateral to presence of TrP should be decreased than ipsilateral side.

**Exclusion Criteria:** Any contraindication to manual therapy, diagnosis of fibromyalgia syndrome according to American college of Rheumatology, history of any surgery in cranio-cervical region, history of chronic neck pain, history of whiplash injury.

#### Procedure

60 subjects both males and females were evaluated with the mean age of  $(23.46 \pm 2.31)$  recruited from department of physiotherapy in SVNIRTAR based on the fulfilment of the inclusion and exclusion criteria. The subjects were randomly assigned into 2 groups. The entire procedure was explained to them. They were given verbal instructions for the study and informed consent was taken from every subject before their participation in the study. Identification of the primary trigger point was done and measurement of baseline (pre intervention) - PPT, Contralateral cervical side flexion ROM and pain on VAS were carried out.

**Measurement of PPT:** Pressure pain threshold (PPT) is defined as the minimal amount of pressure that produces pain. A simple hand-held pressure algometer (PA) with a spring is commonly used, although more sophisticated electrical devices with a strain or pneumatic pressure gauge have been developed. Handheld pressure algometers have also been found to be highly reliable with repeated measures over time, when tested in pain-free muscles of either the hand or other body regions. The syringe algometer is easy to construct and performs reliably within a limit of 10% accuracy over the approximate range for clinical use. Calibration of the device can be assumed to be constant as the physical principle on which it relies (compression of air) will vary only slightly with

small changes in atmospheric pressure. Rolke *et al.* (2005) compared hand-held spring and electronic PAs and found no significant difference for clinical purposes. (Rolke *et al.*, 2005) The PA was placed perpendicular to the tissue surface and pressure applied steadily at a constant rate. Pressure was applied slowly enough to allow the subject time to react when pain is felt. When the subject reports feeling pain the action of pressure is stopped. 3 consecutive measurements of PPT at an interval of 30 seconds by the same examiner were taken. Mean of 3 readings was used for further analysis.

# Measurement of Cervical lateral flexion ROM using inch tape

**Subject position:** sitting erect, head vertical in respect to plumb line.

Examiner position: standing on the side of measurement.

**Procedure:** Head in neutral, plumb line passes through the midline which divide the body into right and left half equally. Distance between lateral tip of the acromion process and tip of mastoid process, referred to as initial measurement was taken. The subject was asked to actively flex the neck laterally bringing ear as close as possible to shoulder. No rotation, flexion or extension of cervical spine was allowed and the final reading was noted.

**Documentation:** the difference between initial and final measurement is the ROM lateral flexion ROM, which was recorded in cms.

Each group included 30 subjects (19 males +41 females) with a mean age of  $(23.46 \pm 2.3)$ . Subjects in Group A received shockwave therapy with MET and subjects in Group B received ischemic compression with MET. Treatment was given 5 times a week for 2 weeks.

#### Shockwave therapy

ESWT was conducted using ESWT (Physiotur-18005 Rostock) for the trigger points of upper trapezius using a 12 mm gun set to apply 2000 incidents of impact / beats at a frequency of 5Hz.

**Subject position**: Subject was made to sit on a chair near a couch on which he/she could rest his/her arms. The subject was made to relax. Trigger point was identified and marked with a skin marker. Shockwave gel was applied as a thin film over the tender point in the upper trapezius. Shockwave head was placed perpendicular to the trigger point identified. This position was maintained throughout the session. Subject was asked to inform any burning sensation if felt.

**Parameters:** Number of impulses / beats: 2000 beats, Frequency: 5 Hz

**Muscle energy technique**: Subject lies in supine position, arm by the side, the examiner stands at the head end of the subject. Head/ neck side bend away from the side being treated to just short of restriction barrier, while the examiner stabilises the shoulder with one hand and cups the ipsilateral ear/mastoid area, with the other. The three fibres of upper trapezius wee stretched. Posterior fibres by neck fully side bent and rotated contralaterally, middle fibres by neck fully side bent and half

rotated contralaterally, anterior fibres by neck fully side bent and slightly rotated toward the side being treated. The subject was asked to introduce a light resisted effort (20% of available strength) to take the stabilised shoulder towards the ear (a shrug movement) and the ear towards the shoulder. The double movement is important in order to introduce a contraction of the muscle from both ends simultaneously. The degree of effort should be mild and no pain should be felt. The contraction was sustained for 7-10 seconds and, upon complete relaxation of effort, the examiner gently eases the head/neck into an increased degree of side-bending and rotation, where it is stabilized, as the shoulder is stretched caudally. As stretching was introduced, the subject could assist in this phase of the treatment by initiating, on instruction, the stretch of the muscle ('as you breathe out please slide your hand towards your feet'). Subject participation in the stretch reduces the chances of a stretch reflex being initiated. Once the muscle was in a stretched position, subject relaxed and the stretch was held for up to 30 seconds. Repetition: 5 times or until no further gain is possible.

Ischemic compression: Subjects were placed supine on the couch with head fully on the surface of the couch, to reduce tension in the upper trepezius muscle. Arm was positioned in slight shoulder abduction with the elbow bent and their hand resting on their stomach. To perform IC to the upper trapezius, therapist stood at the head end of the couch. First, the muscle belly was palpated for a taut band by using a pincer grasp a trigger point was located. A common location of upper trapezius trigger points is in the middle of the muscle belly, approximately 1 to 2 inches medial to the acromion process of the scapula. Once trigger point was located, an IC was applied by gradually applying pressure to the trigger point with your thumb, during which the patient felt original referred pain within the limits of his pain tolerance. Hold this technique for approximately 20 seconds to 1 minute till the pain has diminished, or until the muscle fibres begin to relax under your pressure. Once the release was felt gradually release pressure. This was repeated five times for five sessions per week for 2 weeks.

#### **Data collection**

Measurements of PPT by syringe algometer, pain on VAS and neck side flexion ROM were taken prior to beginning of treatment (pre-test) and were repeated after completion of 2 weeks treatment protocol (post treatment).

#### Data analysis

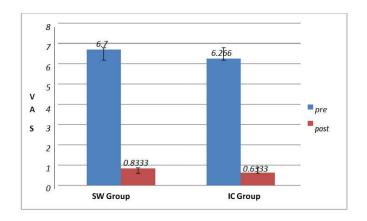
Statistical analysis was performed using SPSS version 16.0. The dependent variables were analysed using a  $2 \times 2$  ANOVA, repeated measures on second factor. There was one between factor (Group) with two levels (Group: shockwave, ischemic compression) and one within factor (Time) with two levels (Time: Pre, Post). All pair wise Post- Hoc comparisons were analysed using a 0.05 level of significance.

## RESULTS

#### Visual analogue scale

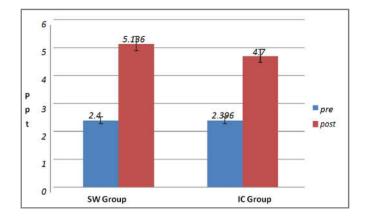
GRAPH 1 VAS (Mean ± SEM)

scores in both the groups from pre to post measurements after an intervention of 2 weeks. However there wasn't any significant difference between the groups. There was a main effect for time (F=1.681, df=1, p=0.000). There was a main effect also for group (F=2.601, df=1, p=.112). The main effect also qualified to interaction of time x group (F=.692, df=1, p=.409).



#### Pressure pain threshold

GRAPH 2 PPT (Mean  $\pm$  SEM)



Graph 2 illustrates that there was an improvement in PPT score in both group A (shockwave therapy and MET) and group B (ischemic compression and MET) following treatment from pre to post measurements but the improvement in shockwave group is more as compared to control group. There was a main effect for time (F=914.73, df=1, p=0.000). There was a main effect also for group (F=2.749, df=1, p=0.103). The main effect also qualified to interaction of time x group (F=6.762, df=1, p=0.012). Post hoc analysis reveals statistically significant difference and improvement in pre to post intervention PPT scores for both the groups after 2 weeks. However improvement in shockwave group.

#### Cervical side flexion rom

#### GRAPH 3 CROM (Mean $\pm$ SEM)

Graph 3 illustrates that there was improvement in contralateral side flexion ROM in both experimental and control groups following treatment for 2 weeks but the improvement in shockwave group was more compared to ischemic group. There was a main effect for time (F=137.723, df=1, p=0.00). There was also main effect for group (F=0.130, df=1,

p=0.720). The main effect qualified to time x group interaction also (F=6.736, df =1, p=0.012). Post hoc analysis reveals statistically significant improvement in ROM for both groups from pre to post measurements over 2 weeks of intervention. However shockwave group showed more significant improvement compared to ischemic group.



## DISCUSSION

The overall results of this study shows that there was significant reduction in perception of pain in terms of VAS, increase in pressure pain threshold and increase in contra lateral neck side flexion ROM in both the groups i.e (shockwave + MET) group and (ischemic compression + MET) group in subjects with myofascial trigger point in upper trepezius muscle. There wasn't any marked difference in reduction of pain on VAS between the groups, However the improvement in PPT and CROM was significantly more in shockwave group compared to ischemic compression.

#### Visual analogue scale

The VAS was used to quantify pain due to MTrP in upper trapezius in both the groups and it was found that there was a significant reduction in pain in both the groups. However there wasn't any statistically significant difference between the groups at the end of 2 weeks intervention. The results indicate that the interventions given in the study proved to be equally effective in reducing pain in subjects with MTrP in upper trapezius. There was common treatment MET given to both the groups. The difference was because of shockwave therapy and ischemic compression. Both groups showed significant reduction in VAS scores after 2 week of intervention; however the groups were not statistically different from each other at the end of 2 weeks. Mean change in shockwave group was 6.70 (pre) to 0.833 (post). Mean change in ischemic group was 6.26 (pre) to 0.633 (post). The most recent research by Shah JP et al. demonstrates increased concentrations of protons (Hb), bradykinin, calcitonin gene-related peptide, substance P, tumor necrosis factor-alpha, interleukin-1 beta, serotonin, and norepinephrine in the biochemical milieu of human trapezius muscle in patients with neck pain and active MTrPs when compared with controls. (Shah et al., 2005)

**Reduction in pain through Muscle Energy Technique:** In both the groups may be attributed to the improvement in blood circulation in the area of pain and increased extensibility of soft tissues. MET influence pain mechanisms and promote hypoalgesia, which involve central and peripheral modulatory mechanisms, such as activation of muscle and joint mechanoreceptors that involve centrally mediated pathways,

like the peri aqueductal grey (PAG) in the midbrain, or nonopioid serotonergic and noradrenergic descending inhibitory pathways. Animal and human studies have shown sympatho excitation and localised activation of the lateral and dorsolateral PAG from induced or voluntary muscle contraction. MET applications reduce pro-inflammatory cytokines and desensitize peripheral nociceptors. Additionally, MET may increase fluid drainage and augment hypoalgesia. Rhythmic muscle contraction increases muscle blood and lymph flow rates, and mechanical forces acting on fibroblasts in connective tissues change interstitial pressure and increase trans capillary blood flow changing intramuscular pressure and the passive tone of the tissue. (Chaitow, 2013) Lewit & Simons (1984), demonstrated that muscle lengthening utilizing post isometric relaxation appears to be successful in relieving pain due to myofascial trigger point. (Lewit and Simons, 1984)

Reduction in VAS in ischemic group: Ischemic compression produces its effects by reflex Vasodilatation – by temporarily occluding the trigger point's blood supply and then releasing the digital pressure from the trigger point causes a reactive increase in blood supply. This flushes any inflammatory exudates and pain metabolites such as serotonin and histamine out of the muscle, breaks down scar tissue, and reduces muscle tone. The extra flow of blood through the trigger point nourishes the muscle, the nerve endings are desensitized, and scar tissue is broken down so that the muscle fibres can move better. As the trigger point settles, there will be an accompanying decrease in referred pain. Ischemic nerve block - the digital pressure applied to the trigger point causes a temporary ischemia thus depriving the area of oxygen, this causes a reduction in action potentials and blocks noxious sensory afferent input to the higher levels of the nervous system. (Simons et al., 1999)

Reduction in VAS through shockwave therapy: The precise mode of action of shockwaves applied to muscular trigger points has not been defined to date. However, the shock wave mechanism attributed to reduction in pain can be explained on the basis of known theories. The biological basis for the analgesic effect of shockwave therapy is interaction with sensory neuropeptides such as substance P or calcitonin gene related peptide which contributes to musculoskeletal pain. (Mariotto et al., 2009; Takahashi et al., 2003) The pain modulation achieved with radial shock waves through the activation of A-beta fibres in the muscle providing pain inhibition through GABA interneurons in the dorsal horn. This phenomenon is more evident when shockwave therapy is applied repetitively which is linked to the desensitization of the exposure area and provides an explanation for the analgesic effects of shockwave therapy. This confirms the findings of Travel who observed many years ago that pain is relieved and trigger points are eliminated after pressure and stretching. (Travel and Simons, 1999; Hausdorf et al., 2008) The vibration of radial shock waves also seems to have a favourable effect on muscular structures. The physiological intrinsic oscillations of 15 to 30 Hz have been described by Nazarov SB et al. important for muscular blood circulation and lymphatic drainage. (Nazarov and Gorozhanin, 1988) Recently Wang et al demonstrated that shock waves induce neovascularisation associated with increased expressions of angiogenic growth factors, including endothelial nitric oxide synthase (eNOS), vessels endothelial growth factor (VEGF), and proliferating cell nuclear antigen (PCNA) at the tendon bone junction in rabbits. Neo-vascularisation play role in the

increase in blood supply that leads to repair. (Wang, 2003) Shockwaves can possibly influence the neuroplasticity of the human pain memory. Long-term fixation of pain impulses result in the development of a particular pain memory. By triggering minimal pain impulses, ESWT could break through this negative-conditioned pain memory by resetting the pain - an approach based on the neuron-holographic brain model. It defines the healing effects of ESWT by selective erasing of pathologic reflex patterns and might explain the possibility of influencing areas of pain localized at a distance from the treatment locus. (Niddam *et al.*, 2008)

### Pressure pain threshold

In the present study there was a significant increase in the PPT with treatment in both the groups. But at the end of 2 weeks the improvement in pressure point threshold was more in shockwave group as compared to ischemic group. Mean change in shockwave group was 2.400 (pre) to 5.136 (post). Mean changes in ischemic group was 2.396 (pre) to 4.700 (post). The local tenderness of the MTrP and its ability to refer pain to distant location depends primarily on the sensitization of nociceptors in the immediate region of the endplates that are associated electromyographically with abnormal endplate function. (Simons, 2001) The increase in PPT through MET in both the groups may be explained by the fact that PIR (Post Isometric Relaxation) lengthens the shortened muscle fibres as explained by Travell and Simons. (Travel and Simons, 1999)

**Increase in PPT through ischemic compression:** Ischemic compression is given for the purpose of eliminating point tenderness. (Travel and Simons, 1999) The increase in PPT due to IC is attributed to the fact that the digital pressure applied to the trigger point causes temporary ischemia thus depriving the area of oxygen which in turn causes a reduction in the action potential and blocks noxious sensory afferent input to the higher levels of nervous system, thus desensitizing the area. In addition, IC also flushes away any inflammatory exudates and pain metabolites such as histamine, serotonin out of muscle and breaks down the scar tissue and reduces muscle tone which eventually leads to decreased point tenderness on palpation. (Anderson *et al.*, 1997)

Increase in PPT through ESWT in the shockwave group can be explained by the selective degeneration of C-fibres, dilution of vasoneuroactive substances and release of NO. (Mense et al., 2000; Hausdorf et al., 2008; Schuppe and Newland, 2004) The shockwave group showed greater improvement in pressure pain threshold than ischemic compression group which may be due the fact that the high energy acoustic waves that are transmitted through the surface of the skin are spread radially (spherically) into the body and the body responds with increased metabolic activity around the area of the pain and tenderness This stimulates the reformation of blood vessels, stimulate and facilitate connective tissue recovery, supply adenosine triphosphate (ATP) to the bloodstream around the trigger points, and remove bodily wastes including the materials that cause pain and tenderness, thereby accelerates the healing process. There is some evidence to suggest that shockwave acoustic compression energy systems achieve their results from reducing the concentration of nociceptor stimulating substances inherent in TPs, which would in turn abort the cycle leading to possible plastic changes in the spinal cord by averting the sensitization phenomenon.

## Contra lateral neck side flexion rom

In this study contralateral side flexion significantly improved in both the shockwave group and ischemic compression group but at the end of 2 week treatment session improvement was greater in shockwave group statistically as compared to ischemic group. Mean change in shockwave group was 5.633 (pre) to 7.753 (post). Mean changes in ischemic group was 5.833 (pre) to 7.233 (post).

Improvement in Neck Side Flexion ROM through MET in both groups may be attributed to neurophysiological principle that account for neuromuscular inhibition that occurs during application of these techniques which states that after a muscle is contracted, it is automatically relaxed for a brief latent period. The improvement in ROM with MET can also be explained by the fact that regional cervical motion restrictions are caused by shortened, hypertonic long muscles of the cervical spine. Lengthening these muscle groups may help to restore gross physiologic range of motion in the neck. (Mitchell, 1995) Muscle lengthening brought about by PIR can be ascribed to its neurological effect, when a muscle is contracted isometrically, a load is placed on the Golgi tendon organ that, on cessation of effort, results in a period of hypotonicity, lasting in excess of 15 seconds. During this, a stretch of the tissues involved is more easily achieved than before the contraction. (Mitchell et al., 1979) Also, during and following an isometric contraction of a muscle, its antagonists are reciprocally inhibited, allowing tissues involved to be more easily stretched. (Liebenson, 1996) Light muscle contraction (15-20% of available strength) used is not only as effective as a strong contraction in achieving the desired effects but also is far less likely to provoke pain or cramping and easier to control. Rhythmic repetitive muscle contractions performed during MET may relieve passive congestion in the paraspinal muscles, as a result of fluctuating blood and lymph pressure gradients propelling fluid throughout the body. It has also been suggested that drainage of fluid from zygapophyseal joint and segmental muscles may achieve a change in ROM and end feel. (Lederman, 1997; Fryer and Fossum, 2008)

Improvement in Neck Side Flexion ROM through IC: Ischemic compression as applied follows a progression from light pressure to deep pressure over a period of 10 seconds 5 times at each treatment, therefore allowing for the treatment of both superficial muscle fibres as well as deep within the course of one visit. Following ischemic compression there would be resultant muscle lengthening and a decrease in trigger point activity, with a subsequent increase in the range of motion. The static stretch by ischemic compression would literally deform the muscle fibre, pulling apart the actin / myosin cross bridges restoring the muscle fibre to full length. (Schneider, 1995) It is noted that IC is an effective treatment, though the treatment effect is slower. It is hypothesized that this is directly related to the static nature of the stretch reflex employed during IC. In relation to the static stretch reflex the degree of neurological stimulation is less than that of a dynamic stretch reflex. (Korr, 2006)

**Improvement in Neck Side Flexion ROM through shockwave therapy:** Improved neck side flexion ROM by shock wave may be attributed to mechanical resolution of permanent actin/myosin contractures through local transverse stretching of the sarcomeres by the application of shock waves perpendicular to the fibre orientation up to the destruction of

abnormally contracted sarcomeres through the energy applied, reactive local tissue hyper perfusion and angioneogenesis, which would eliminate the ischemia responsible for the energy crisis, and eventually thinning of vasoneuroactive substances by the pressure exerted on the tissue by shock waves. All these mechanisms could explain the clinically observed reduction in muscle tension and muscular tightness and thus increase in the ROM. Muscle oscillation of 15 to 30 Hz have been described as important for muscular blood circulation and lymphatic drainage. (Nazarov and Gorozhanin, 1988) Dr. M. Gleitz, Luxembourg concluded that ESWT of muscle trigger points leads to a measurable improvement of range of motion of the cervical spine and to a significant reduction in pain. Shockwave therapy helps in mechanical resolution of permanent actin/myosin contractures through local transverse stretching of the sarcomeres by the application of shock waves perpendicular to the fibre orientation up to the destruction of abnormally contracted sarcomeres through the energy applied.

#### Conclusion

The results of the study demonstrates that shockwave therapy is superior to ischemic compression in terms of improving pressure pain threshold as well as cervical ROM in subjects with upper trapezius myofascial trigger point when combined with Muscle energy technique. However in terms of reducing pain shockwave therapy and ischemic compression are equally effective.

#### Limitations

Small sample size, lack of consideration of perpetuating factors like posture, psychological factors, no functional measure showing qualitative improvement in the patients status was taken, more reliable and sophisticated instrument to measure CROM and electronic pressure algometre could have been used. No follow up study was done.

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