T.R.
KIRŞEHİR AHİ EVRAN UNIVERSITY
INSTITUTE OF HEALTH SCIENCES
PHYSIOTHERAPY AND REHABILITATION
DEPARTMENT

THE EFFECT OF EXTRACORPOREAL SHOCK WAVE THERAPY(ESWT) ON SPASTICITY AND UPPER LIMB FUNCTIONALITY IN STROKE PATIENTS

SALAM KHLAIF JABER ALAASEMI

MASTER OF SCIENCE THESIS

KIRSEHİR / 2022

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ADVISOR

Assist. Prof. Dr. Anıl ÖZÜDOĞRU

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ACCEPTANCE AND APPROVAL

Kırşehir Ahi Evran University, Institute of Health Sciences, Department of Physiotherapy and Rehabilitation Master's thesis study named " The Effect of Extracorporeal Shock Wave Therapy (ESWT) on Spasticity and Upper limb Functionality In Stroke Patients " prepared by our student Salam Khlaif Jaber ALAASEMI. It was accepted as a Master's thesis in the Department of Physiotherapy and Rehabilitation by the following jury in 22/12/2022.

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THESIS STATEMENT

I declare that all the information in the thesis is obtained and presented within the framework of ethical behavior and academic rules, and in this study, which is prepared in accordance with the thesis writing rules, all kinds of statements that do not belong to me are fully cited to the source of the information.

Salam Khlaif Jaber ALAASEMI

According to Objects 9/2 and 22/2 of the Graduate Teaching and Training Rules printed in the Authorized Newspaper dated 20.04.2016; A report in agreement with the standards identified by the Institute of Health Sciences was got by using the plagiarism software package for this postgraduate thesis.

PREFACE

First of all, I thank God,

My esteemed thesis advisor, Assist. Prof. Dr. Anıl ÖZÜDOĞRU I would like to thanks.

I would like to thank all my teachers in my academic life for their efforts in my education from my childhood to this stage.

I would like to express my deepest gratitude to my mother, father, brothers and sisters.

I dedicate my thesis to my beloved wife Duaa, who was patient and endured with me the hardships of the path of knowledge.

I dedicate to the soul of my dead brother Ammar, who was supportive and supportive of me in completing this study

I dedicate to my dearest and nearest friend Pharmacist Hussein ALZUBAIDI

December 2022 Salam ALAASEMI

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LIST OF ABBREVIATIONS

ACH: Acetylcholine

ARAT: Action Research Arm Test

AS: Ashworth Scale

BTX: Botulinum Toxin

CP: Cerebral palsy

CVA: Cerebrovascular accident

ED: Emergency Department

EMG: Electromyography

EMG BF: EMG Biofeedback

ESWT: Extracorporeal Shock Wave Therapy

FME: Fugl-Meyer Evaluation

FME-UE: Fugl Meyer Evaluation Upper Extremity

FCR: Flexor carpi radials

fSWT: Focus Extracorporeal Shock Wave Therapy

ICF: International Classification of Functioning

JTHFT: Jebsen Taylor Hand Function Test

MAS: Modified Ashworth Scale

MFT: Manual Function Test

MRI: Magnetic Resonance Imaging

MT: Mirror Therapy

MTRP: Muscle Trigger Point

MTS: Modified Tardieu Scale

MVC: Maximum voluntary contraction

NO: Nitric Oxide

NSC: Neural Stem Cell

NTH-3: Neurotrophin-3

pSWT: Planar Extracorporeal Shock Wave Therapy

RCTs: Randomized Controlled Trials

rESWT: Radial Extracorporeal Shock Wave Therapy

SEA: Spontaneous Electrical Activity

SWMT: Semmes Weinstein Monofilament Test

TAS: Tone assessment Scale

TENS: Transcutaneous Electrical Stimulation

TS: Tardieu Scale

TSRT: Tonic stretch Reflex Threshold

UMN: Upper Motor Neuron

VAS: Visual Analog Scale

VEGF: Vascular Endothelial Growth Factor

WMFT: Wolf Motor Function Test

2PD: Two-Point Discrimination

9-HPT: 9-Hole Peg Test

ABSTRACT

MASTER SCIENCE THESIS

THE EFFECT OF EXTRACORPOREAL SHOCK WAVE THERAPY(ESWT) ON SPASTICITY AND UPPER LIMB FUNCTIONALITY IN STROKE PATIENTS

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Spasticity is a sign or symptom of a stroke, and it can last for days or even months. Studies show that spasticity affects about 38% of people who have had a stroke. People who have had a stroke can use Extracorporeal shockwave therapy to help with spasticity, pain, and improving the way their upper limbs work. Extracorporeal shockwave therapy (ESWT) is a series of single sonic pulses with high peak pressure (100 MPa), fast pressure rise (10 ns), and short duration (10 ls) that are sent to the target area by a generator and have an energy density of between 0.003 and 0.89 mJ/mm2. Radial ESWT (rESWT) has low to medium energy, a lower peak pressure (0.1 MPa), a longer rise time (50 ls), and lasts longer than focused ESWT (200–2000 ls). In Iraq, the Wasit Disabled Rehabilitation Center has 48 patients. The people who signed up were split into two groups (study and control). Each group had 24 people. In Control group, which used conventional physiotherapy, there were 20 men and 4 women. Study group used rESWT along with conventional therapy (16 men and 8 women). As ways to measure the outcome, we use the Modified Ashowrth Scale (MAS) score, the Fugl-Meyer Assessment-Upper Extremity (FMA-UE), and the Range of Motion.

In statistical analysis, there were significant improvement in both groups of patients whom treated with ESWT and without ESWT in FMA-UE. $P<0.001$.

It is found that ESWT has no effect on spasticity and upper extremity functionality in stroke patients.

December 2022, 89 pages.

Keywords: Stroke, Spasticity, Upper limb function, Extracorporeal Shock Wave Therapy, Fugl Meyer Assessment, Range of Motion.

ÖZET

YÜKSEK LİSANS TEZİ

İNMELI HASTALARDA EKSTRAKORPOREAL ŞOK DALGA TEDAVISININ (ESWT) SPASTISITE VE ÜST EKSTREMITE FONKSIYONELLIĞI ÜZERINE ETKISI

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Fizyoterapi ve Rehabilitasyon Anabilim Dalı

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Spastisite, inmeli hastalarda görülen semptomlardan biridir ve hastalığın prognozuna göre günler hatta aylarca görülebilir. Çalışmalar inmeli hastaların yaklaşık %38'inde spastisite görüldüğünü bildirmektedir. İnmeli hastalarda ekstremite fonksiyonelliğini arttırmak için spastisite ve ağrıyı azaltıcı etkisi olan ekstrakorporeal şok dalga tedavisi (ESWT) kullanılabilir. ESWT; yüksek atım güçlü (100 MPa), hızlı yükselen (10 ns), ve kısa durasyonlu (10 ls) ses enerjisinden oluşmaktadır. Bu cihaz hedef dokuya 0.003 and 0.89 mJ/mm2 enerji yoğunluğu sağlayan bir jeneratör yardımıyla uygulanır. Bu çalışma Irak'ın Wasit şehrindeki rehabilitasyon merkezinde 48 hasta üzerinde yapılmıştır. Hastalar, her grupta 24 kişi olacak şekilde iki gruba ayrılmıştır. Kontrol grubu 20 erkek ve 4 kadın hastadan oluşmaktadır ve bu gruba sadece konvansiyonel tedavi uygulanmıştır. Çalışma grubu ise 16 erkek ve 8 kadın hastadan oluşmaktadır ve bu gruba konvansiyonel tedaviye ek olarak rESWT uygulanmıştır. Çalışmamızda kullanılan sonuç ölçekleri; Modifiye Ashword Skalası, Fugl-Meyer Üst Ekstremite Değerlendirme Ölçeği ve eklem hareket ölçümüdür. İstatistiksel analiz sonucunda hem kontrol hem de çalışma grubunda Fugl-Meyer Üst Ekstremite Değerlendirme Ölçeği açısından anlamlı iyileşme bulunmuştur ($p<0.001$).

ESWT'nin inme hastalarında spastisite ve üst ekstremite fonksiyonelliği üzerine etkisinin olmadığı saptandı.

Aralık 2022, 89 sayfa.
Anahtar Kelimeler: İnme, Spastisite, Üst ekstremite fonksiyonu, Ekstrakorporeal şok dalga tedavisi, Fugl-Meyer Üst Ekstremite Değerlendirme Ölçeği, Eklem hareket ölçümü.

1. INTRODUCTION

A stroke is an illness with a rapid onset that alters local or global brain function. Stroke is a leading cause of the death and disability worldwide, especially in middle-income and low-income countries (1). Spasticity is a common symptom after a stroke, and it can continue for weeks or months. Studies indicate that approximately 38% of stroke survivors develop spasticity (2). If severe spasticity is not addressed, it can result in pain, poor posture, and disability, which is a significant issue. It can also hinder the patient's capacity to recover and reduce their quality of life and ability to do daily tasks (3). The precise brain pathways that lead to spasticity remain unknown. However, it has been suggested that the primary cause of spasticity is an issue with the stretch reflex arc. Spasticity may be caused by a variety of factors, including the dissociation or disintegration of motor responses to sensory input, the hyperexcitability of segmental processing in the central nervous system, the breakdown of inhibitory tracts, and the increase in excitability of motor neurons, according to some researchers (4). Physiotherapists who treat with stroke patients often have to deal with spasticity in these patients. As a result, it is absolutely necessary to exercise control over and seek treatment for spasticity as quickly as is practically possible. Oral medication, chemical neurolysis, injections of Botulinum Toxin (BTX), intrathecal therapies, and physical therapy are all examples of treatments for spasticity that do not involve the use of drugs. Physiotherapy is the initial treatment for spasticity and should be started right away. Managing spasticity can be accomplished using a variety of physiotherapy techniques (5). There are a range of neurological and musculoskeletal conditions, and it is believed that physical treatments, such as shockwave therapy, can help alleviate these conditions (6). For extracorporeal shock wave therapy, also known as ESWT, a generator will send a series of single sonic pulses to a target region. These pulses will have a high peak pressure, a quick rise in pressure, and a short duration (7). ESWT has found widespread use in the field of rehabilitation medicine, where it is used to treat a wide range of conditions affecting the muscles, bones, and joints (8). Focused, radial, and planar shock wave therapy (often shortened to fESWT, rESWT, and pESWT, respectively) are three types of shock wave therapy (9). The direct mechanical action on the treatment point, which is the most important of rESWT's two physical effects, and the indirect mechanical effect of cavitation, which is the second most important of rESWT's two physical effects, are both equally important (10). The

vast majority of studies have demonstrated that rESWT is only effective on spastic muscles. Patients who had suffered strokes saw improvements in their hand flexor digitorum tendon, forearm flexor spasticity muscles, and intrinsic muscles after undergoing rESWT (11). Extracorporeal shockwave therapy, is a sequence of single sonic pulses that have a high peak pressure (100 MPa), a rapid pressure increase (10 ns), and a short duration (10 ls). A generator transmits these sonic pulses to the area to be treated. The energy density of these pulses ranges from 0.003 to 0.89 mJ/mm2 of tissue. Especially in comparison to focused ESWT, radial ESWT (rESWT) has low to moderate energy, a lower peak pressure (0.1 MPa), a longer rising time (50 ls), and a longer duration (200–2000 ls). Additionally, the duration of radial ESWT (rESWT) is longer (12). According to the findings of two different published research, ESWT is effective in reducing spasticity in both children diagnosed with cerebral palsy (CP) and adult stroke patients (13). In recent years, there have only been a few review papers that have looked into whether or not ESWT is effective. On the other hand, they only take into account the tone of the muscles and pay no attention to the discomfort, limited range of motion (ROM), or activity constraints. In addition, recent randomized controlled trials (RCTs) have been conducted to explore the effects of ESWT on spasticity that occurs after a stroke (14).

Therefore, the goal of this (RCT) is to find out how ESWT affects spasticity, ROM, and how well the upper extremities function in people who have had a stroke.

Hypotheses of this RCS as follows:

1-H1: has an effect on ESWT spasticity, H0: ESWT has no effect on spasticity. 2-H1: ESWT has an effect on ROM, H0: ESWT has no effect on ROM. 3-H1: ESWT has an effect on upper extremity function,H0: ESWT has no effect on upper extremity function.

2. GENERAL INFORMATION

2.1. Stroke

In recent years, stroke has emerged as one of the primary causes of serious disability and mortality throughout the majority of nations. In the United States, over than 795,000 people have suffered a stroke (15). Ischemic stroke, also known as a classic stroke, transient ischemic attack, and hemorrhagic stroke are the three subtypes of stroke that can be diagnosed based on the symptoms and imaging of the brain. About twenty percent of all strokes are caused by bleeding in the brain, which can be caused when a blood artery bursts or releases blood (16). The intracerebral hemorrhagic stroke, also known as ICH, is associated with a high fatality rate as well as severe impairment in survivors (17). This particular form of stroke is responsible for around 42% of the disability-adjusted life years lost(18), and 50% of all stroke patients pass away within one year of their stroke diagnosis (19).Stroke is a clinical picture that can go from the loss of motor loss, sensory disorder, balance disorder, speaking and cognitive function losses due to loss of focal cerebral function without a visible reason other than vascular causes and characterized by sudden development of neurological deficits (20). Stroke is a neurological disease that usually occurs in two forms: ischemic stroke and hemorrhagic stroke (Figure 2.1).

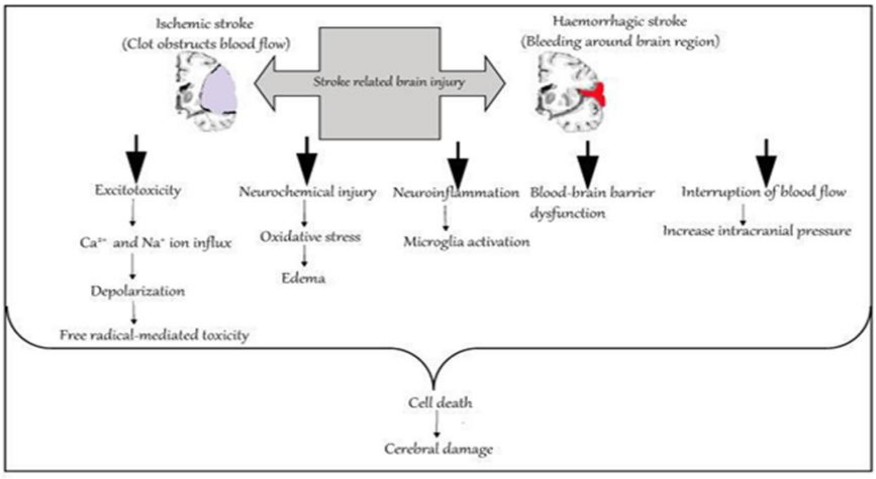

Figure 2.1: Stroke types (21).

2.2. Epidemiology

In the United States, stroke is the greatest cause of adult impairment that continues for an extended length of time, and it is also the fourth leading cause of mortality overall. It is estimated that seven million people in the USA aged 20 and older have suffered from a stroke. Over 795,000 people have a stroke each year, with roughly 610,000 being first-time occurrences and 185,000 being recurrences. Of these people, approximately 610,000 die from their strokes. When age was considered, it was shown that the risk of having a stroke was lower in women than in men. However, this trend is reversed as people get older; women have a greater risk than men until they reach the age of 85. It is estimated that African Americans have a likelihood of having their first stroke that is two times greater than that of whites. The chances are also much higher for American Indians, Mexican Americans, and Alaska Natives. The risk of having a stroke more than doubles in the decade following the age of 65. There are 28 percent of all strokes that occur in adults who are under the age of 65. Those who survive a first stroke have a 5% to 14% chance of having a second stroke within the next year. Within the next 5 years, the risk of having a second stroke increases to 24% for women and 42% for men. Recent studies have shown that there has been a decrease in the number of strokes that occur in adult populations that are predominantly white during the previous few years (21). Every year, more than 143,000 people pass away as a result of having a stroke. In the United States, a stroke is the underlying cause of one out of every 18 deaths. The type of stroke is a big factor in whether or not someone will live. Most people who have a stroke die from a hemorrhagic stroke, which has a death rate of 37% to 38% at 1 month. In contrast, only 8% to 12% of people who have an ischemic stroke die at 1 month. Age, high blood pressure, heart disease, and diabetes all make survival rates much lower. Loss of consciousness at the start of a stroke, the size of the lesion, severe hemiplegia that lasts for a long time, Multiple neurological impairments and a prior stroke are also significant mortality predictors. (22, 23). In recent years, epidemiological studies have been stated that stroke has fallen to fourth place in the United States and in Europe, among the most common causes of death (24). Although the death rate due to stroke is reduced; The stroke is still among the diseases that cause the most disability and addiction (25).

2.3. Etiology

Ischemic and hemorrhagic are the two main types of CVA. Ischemia is the cause of about 85% of CVAs, and bleeding is the cause of about 15%. 40% of all stroke deaths are caused by strokes that cause blood to leak out. Stroke is a neurological syndrome that is caused by problems with the blood vessels. Ischemic strokes cause 80% of all strokes. Ischemic, stubborn strokes are caused by cerebral thrombosis in 60% of cases and cerebral embolism, cerebral vasculitis, and lack of blood flow to the brain in 20% of cases. Thrombosis can be caused by atherosclerosis in the brain and blood vessels. Chronic hypertension can also cause atherosclerotic vascular changes in small, deep, penetrating arteries. The ventricular wall can result in chronic ischemic heart diseases with hypokinesis and arrhythmia and the rupture of cerebral artery aneurysm. 20% of strokes are of hemorrhagic origin. Hemorrhagic strokes; 15% develop as a result of intracerebral bleeding and 5% of subarachnoid hemorrhage. Intracerebral hemorrhages are caused by hypertension, arteriovenous malformation, brain tumor, and rupture of weak vessels. Subarachnoid hemorrhages are characteristic of the rupture of cerebral artery aneurysm (26). In embolic strokes, the clinical picture is quickly starting and completed within minutes. Thrombotic strokes may be fast or prolonged, usually in 1 to 2 hours slowly developing. Hemorrhagic strokes usually develop slowly in 1 to 2 hours. In strokes that develop as a result of the active calaboose of the major cerebral artery, a clinical picture may be characterized by the progressive development of neurological deficits (27).

2.4. Risk Factors for Stroke

Stroke risk factors can be split into two groups: modifiable and immutable. Age, gender, and race/ethnicity are risk factors for both ischemic and hemorrhagic strokes that can't be changed. On the other hand, hypertension, smoking, poor nutrition, and lack of physical activity are often mentioned as risk factors that can be changed. Recent descriptions of stroke risk factors and triggers include inflammatory diseases, infections, environmental pollutants, and problems with the heart's atria that are not caused by atrial fibrillation. Stroke may be the most common sign of rare inherited diseases that are caused by problems with just one gene. Recent research also shows that common and unusual genetic variants can affect the risk of common stroke causes like atrial fibrillation, which are caused by other risk factors and certain stroke processes. Some genetic factors, especially those that interact with the environment, may be more changeable than was once thought (28). Depending on their

gender, younger women are more likely to have a stroke than older men. For men, the risk of stroke goes up with age. Research shows that preeclampsia, hormone therapy, the use of contraceptives, and migraines with aura are all linked to the high rate of strokes in women (29). Women have a few things that make them more likely to have a stroke. Ischemic strokes are twice as likely to happen to women who go through menopause before age 42. Using estrogen alone or estrogen with progestin raises the risk of an ischemic stroke by at least 44 to 55%. Stroke risk goes up during pregnancy, giving birth, and the first six weeks after giving birth, especially for older women and African Americans. Preeclampsia is a risk factor for stroke on its own (22).

2.5. Prevention

For successful stroke prevention, it is necessary to raise awareness of the early warning signals of stroke. 60% of Americans can recognize at least one stroke warning sign, whereas 55% can recognize at least one stroke symptom (26). According to the American Heart Association and National Stroke Association, the symptoms described in table 1 serve as early warning signs as shown in (Table 2.1) (30). In accordance with the adage "time is brain," the importance of recognizing early warning symptoms is contingent on the rapid beginning of emergency care. Even if these symptoms disappear fast or are not unpleasant, patients and their relatives are recommended to dial 911 immediately. CT is utilized to discriminate at an early stage.

Table 2.1: Stroke Early Warning Signs (30).

Component	Description
Face	Numbness or weakness of the face, particularly on one side of the body.
Arm	Numbness or weakness in the arms, particularly on one side of the body.
Speech	Speech slurring or difficulty speaking or understanding.
Time	It is time to contact an ambulance.

between atherothrombotic and bleeding strokes. Clot-dissolving enzymes, such as tissue plasminogen activator, can be utilized in the process of thrombolysis in the event that the stroke was caused by atherothrombosis (tPA). Thrombolytic therapy, such as the

administration of tPA, must be performed within three hours of the onset of symptoms in order for it to be effective. However, thrombolytic therapy cannot be used in the event of a hemorrhagic stroke since the medication could make the bleeding worse. Within this window of opportunity, the patient must determine that their condition warrants being treated as a medical emergency, be transported to the proper hospital, be evaluated by emergency department (ED) personnel (including a CT scan of the brain), and be treated (31) Despite the fact that this treatment has been available since the middle of the 1990s, has been proven to be safe, and has been shown to greatly lower the risk of death and disability, The majority of stroke patients (55%) do not go to the hospital within the first two hours after experiencing symptoms (32). Women have a lower probability of showing up on time than men do. Only 65% of patients who presented to the emergency department after their symptoms had been present for more than two hours were imaged during the first hour of their ED appointment (33).

2.6. Upper Limb Motor Impairment

An impairment can be either 1) a change or loss in neuromusculoskeletal and movement-related function, such as joint mobility, muscle power, muscle tone, and/or involuntary movements, as defined by the International Classification of Functioning, Disability, and Health (ICF) model (34), or 2) a change in the structure of the nervous system or structures related to movement, as defined by the same model. In both cases, a stroke may be to blame. Having a good understanding of the underlying impairments is necessary for restoring upper limb function after a stroke, as these deficits are what make the affected upper limb difficult to use. However, there are two factors that make diagnosing issues with a patient's upper extremities challenging. First, the constraints shift with time. Some motor deficiencies may change in type and severity as rehabilitation continues. This necessitates adapting the treatment based on the specific impairment(s) in play at any particular point in time (2). A patient may be dealing with more than one health problem at once. When someone has a stroke, they often experience temporary impairments, such as arm and hand weakness, which may persist even after spasticity develops a few weeks or months later. Because of this, prioritizing care can be challenging. Reviewing Twitchell's (35) and Brunnstrom's (36) descriptions of the healing process is helpful for understanding the possible development of motor deficits over time (Table 2.2).

Table 2.2: Brunnstrom recovery stages (36).

Stage	Degree of Recovery
Stage 1	Paralysis with flaccidity
Stage 2	Spasticity emerges as a result of movement in the synergy pattern.
Stage3	Movements of many joints performed in unison are considered voluntary synergy movements, which can lead to an increase in spasticity.
Stage 4	Spasticity can be relieved through the use of non-synergistic, voluntary motions.
Stage5	Controlling individual or isolated movement
Stage6	Return to close to normal motor control

2.7. Spasticity

Post-stroke spasticity, also known as PSS, is a common consequence that is associated to various signs and symptoms of upper motor neuron syndrome. Some of these signs and symptoms include agonist/antagonist co-contraction, weakness, and a loss of coordination (37). In the year 1980, Lance was the first person to class spasticity as a type of motor dysfunction. He stated that those who suffer from spasticity have higher levels of muscular tone as a result of the stretch reflex. muscle spindle Ia rises as a result of hyperexcitability, which also contributes to increased motor neuron activity and, eventually, increased hyperexcitability of the stretch reflex. These three phenomena are all components of the motor neuron syndrome. This definition discusses spasticity in the context of passive movement; however, it does not address spasticity in the context of independent movement. An increase in the tonic stretch reflex that is brought on by abnormal intraspinal processing of velocity-dependent primary afferent inputs is the neurophysiological mechanism that defines spasticity, as described by Young in a paper that was published in 1994. This mechanism is the same for all different kinds of movement (38). Patients who have suffered a stroke often experience spasticity, which is a symptom that prevents them from moving. An increase in muscular tone as well as tendon reflexes that are speed-dependent are responsible for its induction. Spasticity in the upper extremities is commonly accompanied by pain, stiffness in the tissues, and joint contracture. Additionally, it may result in an aberrant position of the affected limb, a decline in overall quality of life, and an increase in the amount of work required of careers. Patients who have suffered a stroke are more likely to experience

spasticity in their upper limbs, which can make it challenging for them to return to a normal state of functioning (39). After an episode of spasticity, symptoms may persist for several days or even several months. According to studies, approximately 38 percent of those who have suffered a stroke develop spasticity (3). In the event that severe spasticity is not addressed, it has the potential to cause pain, poor posture, and disability, all of which are significant issues. Additionally, it can make it difficult for the patient to get better and can restrict both the patient's capacity to engage in activities and their quality of life (5). The particular brain mechanisms that are responsible for spasticity are not yet fully understood. On the other hand, malfunction in the stretch reflex arc has been determined to be the key factor contributing to spasticity. Dissociation or disintegration of motor responses to sensory information, hyperexcitability of segmental processing in the central nervous system, breakdown of inhibitory tracts, and an increase in motor neuron excitability are some of the potential explanations that have been hypothesized for spasticity (5). After a stroke, a person may experience spasticity in the stage that follows the elasticity stage. within the region of the Upper Limb Spasticity can have an effect on the function and functioning of the upper extremities, which can then lead to activity limitation, participation restriction, and decreased reliance, increasing the need for direct care services and costs in the first year after a stroke (40). Spasticity is one of the symptoms of UMN syndrome. People who have UMN syndrome, on the other hand, not only have reflex hypertonia, but they also have non-reflex hypertonia as a result of alterations in their connective tissues (6). Spasticity is more common in the upper extremities than it is in the lower extremities, and it worsens as the upper-limb impairment worsens. Spasticity can also occur in the lower extremities. In the first year after a stroke, there is an increase in the number of patients who experience spasticity (41).

2.7.1. Spasticity Epidemiology

More patients experienced upper-limb spasticity than lower-limb spasticity (42). The severity of spasticity in the upper extremities was correlated with the degree to which the upper extremities were unable to move on their own, but this was not the case for the lower extremities (43). These disparities between the upper and lower extremities could be the result of differences in supraspinal control. The upper limb often functions independently, whereas the lower limb is mostly controlled by spinal locomotor centers (44). Additionally, spasticity was most prevalent in muscles that functioned against gravity, such as the arm flexors and the leg extensors. This is comparable to what a prior report discovered (44). After 12 months (45), 5.4 days (mean), and 3 months (43), spasticity was more prevalent in patients younger

than 65 years of age. It is difficult to describe this observation. Aging is associated with a decline in reflexes, as observed in the tonic and tendon reflexes (46). This may be one of the reasons why the presented research revealed disparities between younger and older individuals. This may be due to the loss of people in the oldest age group (>85 years) who have more serious conditions. The difference was also evident when comparing the youngest group to those in the medium age range (65–74 years) (47). The percentage of individuals with PSS ranged from 4 to 46% in the first month, from 4.16 to 48% in the first three months, from 6.9 to 63% in the third to sixth months, and from 7.6 to 49% after six months. Within one month, 2%–2.6% of patients had severe or incapacitating spasticity, 5% within one to three months, 8–15.6% within six months, and 12.5–18% after six months (48).

2.7.2. Spasticity Etiology

Spasticity happens when the central nervous system is overstimulated and motor responses to sensory input are separated or broken down (CNS). More sensory input makes spasticity worse, but the location of the lesion also affects how bad spasticity is . Spasticity can happen in both muscles that are sensitive to stretching and muscles that aren't (49). Positive UMNS symptoms, also called spastic dystonia, are also caused by different pathways. Muscles that are always moving, can't be stopped by willpower, don't only use information from the edges, and don't stretch in waves are signs of this condition. In the hemiplegic posture, for example, a patient's fingers, elbows, and wrists tighten while their legs get longer. Pyramidal fibers might not be important in UMNS, but parapyramidal fibers are. Sensory impulses that cause spinal reflex activity are stopped by the dorsal reticulospinal pathway. Also, the brain stem stops the spinal cord from moving. The pyramidal or corticospinal tract and the dorsal reticulospinal tract are close to each other. Most of the time, the parapyramidal dorsal reticulospinal tract is the cause of spinal reflex activation symptoms. Also, the bulbopontine tegmentum, which is part of the medial reticulospinal tract, sends excitatory signals down from the brain stem. Different things happen to the brain stem, cortex, and spinal cord when these routes are taken. Cortical lesions often cause stiffness, hyperreflexia, and sometimes clonus, but these symptoms are usually much less severe than those caused by spinal cord injuries. Lesions that are both partial and complete have different effects. If a partial spinal cord lesion completely cuts off the inhibitory pathways but leaves the excitatory pathways alone, there will still be spinal activity, but it will be accompanied by a lot of stiffness and hyperreflexia. In contrast, a total lesion of the spinal cord that destroys both excitatory and inhibitory circuits will lead to the loss of all supraspinal control and hyperactivity (49).

2.7.3. Potential risk factors and predictors of spasticity post stroke

It is possible that early intervention or preventative treatment could lower the incidence of spasticity following a stroke if it were possible to understand the events or situations that precede spasticity. This is likewise true with regards to the formation of contractures. Although the predictive factors for spasticity have not been fully recognized as of yet, they are detectable to a certain extent in order to make adjustments to the therapy of spasticity. Patients who have just suffered a stroke must to be at the very least provided with improved education regarding potential complications. The one-year post-stroke spasticity study that was conducted in the United Kingdom and was cited earlier examined the same group of 106 post-stroke patients in an effort to identify risk factors for the development of spasticity. The researchers discovered a number of signs that could be used as predictors. The 7-day Barthel Index score of those who suffered from debilitating spasticity was lower than the score of people who did not suffer from spasticity. In addition to this, they had early onset of arm or leg weakness (50). The presence of left-sided weakness, a history of smoking, and the Barthel Index were all predictors of a more severe spasticity-induced impairment. Wissel et al. (51) found in a recent study that followed patients 6 days, 6 weeks, and 16 weeks after stroke that the typical patient at high risk for developing Table 1 severe spasticity had hemispasticity, and that TMS was used to determine the duration of the silent period 7 and 90 days after stroke. The study was conducted on patients who had suffered a stroke (52). At 90 days, a poorer functional outcome was associated with a shorter duration of the silent phase, as was an increased risk of spasticity (52).

2.7.4. Spasticity Evaluation

There are a variety of criteria that can be used to evaluate the effectiveness of spasticity therapy (53). Recent publications have included in-depth discussions of clinical, biomechanical, and neurophysiological approaches to the measurement of spasticity (54).

-The modified Ashworth scale

Is the clinical instrument that has received the most praise for its ability to detect an increase in muscle tone (55). In 1980, Jim Lance defined spasticity as a velocity-dependent increase in muscular stretch reflexes followed by an increase in muscle tone. Spasticity is a component of upper motor neuron syndrome. There are many different conditions that can lead to spasticity, such as brain damage, cerebral palsy, multiple sclerosis, trauma, and spinal cord injury. 42.6% of stroke patients acquired spasticity, with 15.6% experiencing severe spasticity, according to

a study that examined the prevalence of spasticity in communities of stroke survivors. According to the findings of a second study that looked at the prevalence of spasticity in cerebral palsy, ninety percent of the patients who were examined had spastic subtypes. The effects of severe spasticity on a patient's life are significant and can be seen in many aspects of their life, such as their day-to-day activities, mental health, and income. On the other hand, spasticity can be beneficial for people who have weak limbs, especially the lower extremities, because it makes it easier for them to walk or transfer from one place to another. Examining spasticity is essential for a number of reasons, the most important one being that it enables medical professionals to evaluate the effectiveness of the treatment methods they employ**.**

The Ashworth Scale was developed by Bryan Ashworth in 1964 for the purpose of evaluating spasticity in multiple sclerosis patients. The first version of the Ashworth scale was a numerical scale with five points that ranged from 0 to 4, where 0 indicated no resistance and 4 indicated a rigid limb in flexion or extension (56). In 1987, while Bohannon and Smith were researching the inter observer reliability of physical examinations of elbow flexor muscle spasticity, they added 1+ to the Ashworth scale in an effort to increase its sensitivity. This was done so that the scale could more accurately reflect the severity of the condition (57). As a method of gauging spasticity, the modified Ashworth scale (MAS) has been put to use in clinical settings as well as in research ever since it was developed. In order to categorize muscle spasticity, the modified Ashworth scale was developed. The weights are distributed as follows(58):

• 0: No increase in muscle tone.

• 1: Slight increase in muscle tone, with a catch and release or slight resistance at the end of the range of motion when a portion is moved in flexion or extension.

• 1+: Slight increase in muscle tone, exhibited as a catch, followed by minor resistance throughout the remainder (less than half) of the range of motion.

• 2: Significant increase in muscle tone over the majority of the range of motion, but the affected part(s) are still easily moved.

• 3: Significant increase in muscle tone, difficult passive movement

• 4: Affected limb or limbs are stiff in flexion or extension

- Isokinetic Dynamometers

Isokinetic dynamometers have been used to measure and test spasticity many times. The biggest benefit is that they make it possible to measure the applied stretch velocity and amplitude. This means that the velocity-dependent muscle resistance to passive movement can also be measured (59). Isokinetic dynamometers can be helpful when an objective and reliable measurement of resistance to passive movement is needed, such as for research projects or drug testing. The isokinetic dynamometers can measure how muscle resistance changes with speed, how much muscle resistance comes from the muscle itself, and how muscle resistance changes when the muscle is stretched (60).

-Pendulum test

Wartenberg came up with the pendulum test in 1951 (61), and many studies have looked at it since then (62, 63). In its most basic form, the patient sits or lies down on a couch with his or her lower leg hanging over the end. The examiner then moves the leg out to the side while telling the patient to calm down. The leg is then set free to move under the force of gravity. With the help of electrogoniometers, you can measure how far your knee joint moves. The swing is often smaller in people with spasticity. One way to measure this is to find the ratio between the knee joint's initial flexion and its final position, which can be done with goniometers after the leg has rested. This ratio has a strong relationship with how bad the spasticity is, as measured by the AS. The pendulum test is easier to do than the AS and gives a more accurate measurement of how bad the spasticity is. But it has a lot of bad things about it. It depends a lot on how the person is sitting and on how well they can relax. Also, it can only be used to measure spasticity in the muscles of the knee, and it doesn't seem to give useful information when spasticity is very bad. Lastly, the test can't tell the difference between increased muscle resistance caused by changes in viscoelasticity and resistance that depends on speed caused by spasticity. Most likely because of these problems, the test has not been widely used (64).

- Tone Evaluation Scale (TES)

TES is made up of 12 parts that are split into three sections. The patient's resting position is checked in the first section (items 1-3): Is the patient's hand on his or her leg? Is the shoulder straight? Does the foot touch the ground flat? In the second part (items 4-9), an ordinal scale is used to measure how the MAS reacts to passive movement in different joints of the body. (Are the lower limbs flexible? Can the knee be easily stretched while sitting down? Lastly, it

looks at how the person reacts when they try to control strange movements. For example, does the person's hand stay locked on their thigh while their other arm is raised above their head? (65).

- Tardieu Scale

The Tardieu Scale (TS) and the many different ways it can be used. The modified tardieu scale (MTS) measures how a muscle reacts to being stretched at different speeds, taking into account both the resistance to passive movement during the slow phase and the resistance to passive movement during the fast phase. The patient has problems with his or her legs and feet. He is put on his back for a test to see how stiff he is. Each joint passively moves at one of three speeds: V1 = as slowly as possible, V2 = the rate at which the limb falls against gravity, and V3 = by moving the limb as quickly as possible. Spasticity is judged by how strong this resistance is and at what angle it happens. The level of resistance is rated from 0 to 4. (or 5 depending on the version used). Zero means that there is no resistance to passive movement, and four means that the clonus lasts longer than 10 seconds. R1 is the angle where there is a capture or clonus during V3, and R2 is the angle where there is no capture or clonus during V3 (full passive range of motion in V1). The difference between R1 and R2 shows the muscle's active tone, which is different from the resistance from the muscle's passive components. TO/WTO is mostly a mental game It is used to measure how stiff children with cerebral palsy (CP) are. It has also been proven to work on stroke patients, though adults rarely take it. Even though TO and WTO can tell the difference between slow and fast resistance, they have flaws like AS and MAS. These only measure passive resistance and require a lot of training (66).

- King's Hypertonicity Scale

The Hypertonicity Scale made by King is a good way to measure spasticity. This scale measures muscle tone, the ability to move in different ways, the resistance to passive movement, and the range of motion while moving. Each part is looked at on its own and given a score between 1 (normal) and 5 (worst), for a total of 4 to 20 (67).

- Electrophysiological Measurement

The tonic stretch reflex threshold (TSRT) is discovered by using electrogoniometry and surface EMG to record the joint angle and myoelectric response of the spastic muscle as it is stretched by hand at different rates. Linear regression is used to figure out TSRT (stretch

reflex threshold angle and velocity). While the patient is hooked up to a wrist harness, the spastic muscle is manually stretched in a sinusoidal pattern at different speeds, following a target track on the monitor. As a measure of spasticity, the normalized maximum voluntary contraction (MVC) of the surface EMG is used (68).

2.7.5. Spasticity Treatment

Spasticity can be treated in several ways, including through physical therapy, splinting, oral medications, chemical neurolysis, and surgical techniques (69). When used orally, anti-spasticity medications like baclofen, tizanidine, and diazepam reduce muscular tone by acting on the central nervous system. However, these medications might have systemic side effects such lethargy and drowsiness (70). There is a risk of sensory loss and dysesthesia in the injected limb after chemo (e.g., phenol) treatment (71). Injections of botulinum toxin (BoNT), a protein neurotoxin derived from Clostridium botulinum, have emerged as one of the most effective anti-spasticity treatments due to their selective blockage of acetylcholine release at the neuromuscular junction. However, poor dosing of BoNT injections can lead to post-injection weakness, and repeated injections can lead to the development of neutralizing antibodies. Patients with significant spasticity despite non-invasive treatment may benefit from surgical treatments (such as selective peripheral neurotomy or intrathecal baclofen pump implantation) (71).

- Pharmacological Management

Drugs for PSS treatment are chosen based on a number of criteria, including the disease's severity, its anatomical distribution (Table 2.3), the existence of comorbidities, and the cost of the drugs in question. Many stroke survivors have cognitive abnormalities that could be made worse by the central effects of oral medications, or they are taking other drugs that shouldn't be taken at the same time as antispasticity drugs (for example, clonidine and tizandine act synergistically, resulting in hypotension; dantrolene sodium used concurrently with statins could cause hepatotoxicity) (42).

Table 2.3: Treatment algorithm for post-stroke spasticity (PSS) (42).

Focal	Multifocal	Regional	Generalized
•Botulinum toxins •Phenol/Alcohol neurolysis	•Botulinum toxins •Phenol/Alcohol neurolysis	•Intrathecal therapy •Botulinum toxins •Phenol/Alcohol neurolysis •may be used concurrently for different muscles in various regions	•Intrathecal therapy •Oral medication But if problem is focal, superimposed on a general presentation, Botulinum toxins or Phenol/Alcohol neurolysis can be considered

- Surgical Intervention

Several criteria, including severity, anatomic distribution (see Figure2), the presence of comorbidities, and drug cost, influence the selection of drugs to treat PSS. Many stroke survivors have cognitive abnormalities that may be exacerbated by the central effects of oral medications, or they are taking other medications that are contraindicated with specific antispasticity treatments (e.g., clonidine and tizanidine act synergistically, resulting in hypotension; dantrolene sodium used concurrently with statins may result in hepatotoxicity) (42).

- Physiotherapy Intervention

1. **Stretching**

Based on the systematic review, stretching is one of the effective treatments for spasticity in physiotherapy. Static stretching, such as orthotics , is more effective than intermittent stretching. Muscle spindles in the static method is inhibited , thus reducing the motor unit activity of the muscles. Stretching in association with other therapy modalities can improve the mobility of the joints, enhance the viscoelastic properties of the muscle-tendon unit, and reduce spasticity (74).

2. **Transcutaneous Electrical Stimulation (TENS)**

Modalities such as TENS It can alleviate spasticity and enhance the function of the upper extremities. The mechanism of action of this modality is reciprocal inhibition, presynaptic

increase inhibition, decrease stretch reflex excitability, corticomotor of the stimulated regions. It is based on reducing the excitability of the brain, ultimately modulating brain plasticity, and increasing sensory input and secretion of Beta endorphins due to the excitability of large-diameter AB fibers. TENS can be applied on the spastic muscle, on the antagonist muscle, along the nerve and distal to the spastic muscle as well as at acupuncture points (75).

3. **EMG Biofeedback (EMG BF)**

Myoelectric signals acquired from the muscles and converted into visual and aural signals in to inform the individual of muscle activity.. Based on this feedback, the patient learns how to alter the physiological characteristics of the activities and can minimize voluntary muscle tone to achieve cortical control in this method. Another method is to use EMG BF to activate antagonist muscles, thereby reducing muscle tone by reciprocal inhibition (76).

4. **Massage**

A simple and inexpensive method to reduce muscle tone. The mechanism of this method may be that manipulation stretches the muscle - tendon complex and Golgi, which may inhibit alpha motor neurons and reduce spasm. It stimulates the tendon organ. Supra , which can reduce the patient's stress and relax, eventually reduce spasticity. sensory input with spinal effect (76).

5. **Cold Application**

an easy and inexpensive method to reduce spasticity when used for a long time. As the effect of cold, it reduces the sensitivity of skin mechanoreceptors, decreases the conduction velocity of sensory and motor nerve fibers (alpha), or decreases neuromuscular It is a decrease in the tension sensitivity of the spindles and thus a decrease in motor nerve activity. Fibers (alpha) and gamma increase motor neuron activity and eventually reduce spasticity. On the other hand, The maximum amplitude of the H response increases in proportion to the maximum amplitude of the M wave (Hmax / Mmax), resulting in decreased reflex excitability and spasticity (77).

6. **Hot Application**

Hot application is commonly used to reduce spasticity. Heat can significantly reduce the F-wave parameters, thus reducing spasticity and causing local relaxation of the muscles. Heat may also increase the effect of stretching technique on spasticity due to increased collagen response to stress (77).

7. Dry needling

According to studies performed after spasticity, there is a change in muscle thickness, pennation angle and fascicle length of the spastic muscle. It is stated that dry needling applied to the spastic muscle changes the muscle thickness, pennation angle and fascicle length of the spastic muscle (78). Irritability of the trigger point (MTRP) of a muscle, the end plate in the MTRP region was highly correlated with the prevalence of fluctuation. Similarly, dry needling's mode of action is predicated on the mechanical disruption of the related defective end plate region. In addition, it has been demonstrated that dry needling increases blood flow and oxygen saturation in the stimulated region (79). Dry needling produces a local twitch response so it can alter the spontaneous electrical activity (SEA) of the muscles. The local twitch response that occurs when the needle is inserted into the end plate region reduces Acetylcholine (ACH) storage and results in lower SEA. Another mechanism of dry needling at the endplate is that dry needling causes muscle fiber discharge, thereby producing a local twitch response, resulting in changes in fascicle length, muscle thickness, and angle of pennation. Another mechanism is that it increases blood flow and decreases ACH and opioid or analgesic secretion, increases metabolism in the region, and accelerates its repair. Alternatively, When Aδ- nerve fibers are stimulated, the serotonergic and noradrenergic descending inhibitory systems can also be turned on. Even though there are no known specific experimental or clinical studies that support the proposed serotonergic and noradrenergic mechanisms of dry needling, it is thought that dry needling may have an effect on both systems (80).

8. Extracorporeal Shock Wave Therapy

Extracorporeal shock wave therapy (ESWT) with acoustic pulse has been used with lithotripsy on kidney stones, urinary calculi, and biliary calculi since 1982. (Wang et al.) (81). Salivary gland and pancreatic stone treatment is reportedly another application. ESWT is a non-invasive technique which involves sending a series of sound wave pulses (low-energy) into a wound through gel and skin. Focus shock wave therapy, radial shock wave therapy, and planar shock wave therapy are the three subtypes of extracorporeal shock wave therapy (ESWT). rSWT is almost often utilized in tandem with fSWT. Plantar fasciitis, lateral epicondylitis of, calcific tendinitis, and nonunion of long bones are some of the orthopedic disorders that have benefited from ESWT. Parameters for ESWT include an energy flux density (EFD) of 0.01-0.5 mJ/mm2, a pressure range of 10-100 MPa, a pulse rate range of

1200-4000 hertz (Hz), and a number of pulses of 1200-4000. Therefore, the depth of shock waves might theoretically vary from 3 cm to 12.5 cm. Low back pain is only one example of the many musculoskeletal issues that ESWT can alleviate (11, 82). Studies in the basic sciences demonstrate that the ESWT increases blood flow by causing a cascade of beneficial effects, including angiogenesis, neovascularization, and anti-inflammatory responses. By recruiting more fibroblasts and halting tissue necrosis, ESWT has demonstrated potent healing effects. Furthermore, studies have indicated that ESWT enhances the expression of neurotrophin-3 (NTH-3), which aids in the process of neuroregeneration, and enhances the activation of vascular endothelial growth factor (VEGF) and its neuroprotective effects. Additionally, the ESWT promotes neurogenesis by increasing the proliferation of brain stem cells (NSC). This has the potential to enhance the performance of the nervous system. How exactly ESWT works to lessen spasticity remains a mystery to researchers. There are, however, suggestions that attempt to explain the phenomenon. To begin, nitric oxide (NO) is believed to be produced by ESWT and is responsible for generating new neural and muscular connections. By applying continuous or intermittent pressure to the tendons, ESWT may reduce activation of motor neurons. Because ESWT temporarily blocks neuromuscular transmission by decreasing acetylcholine receptors in muscle connections, it may be useful for those with spasticity (83). Both fSWT and rSWT have similar biological effects, such as increasing permeability of cell membranes, stimulating microcirculation (of the blood and lymphatic systems), and releasing substance P (SP), a neurotransmitter involved in numerous neuronal signaling pathways and crucial to the regulation of pain. However, fSWT has its own unique mechanisms of action, for example cavitation, the release of nitric oxide (NO) to increase cellular metabolism and have anti-inflammatory, neovascularization, and angiogenesis effects, and the stimulation of growth factors like fibroblast growth factors and transforming growth factors as shown in (Figure 2.2) (83).

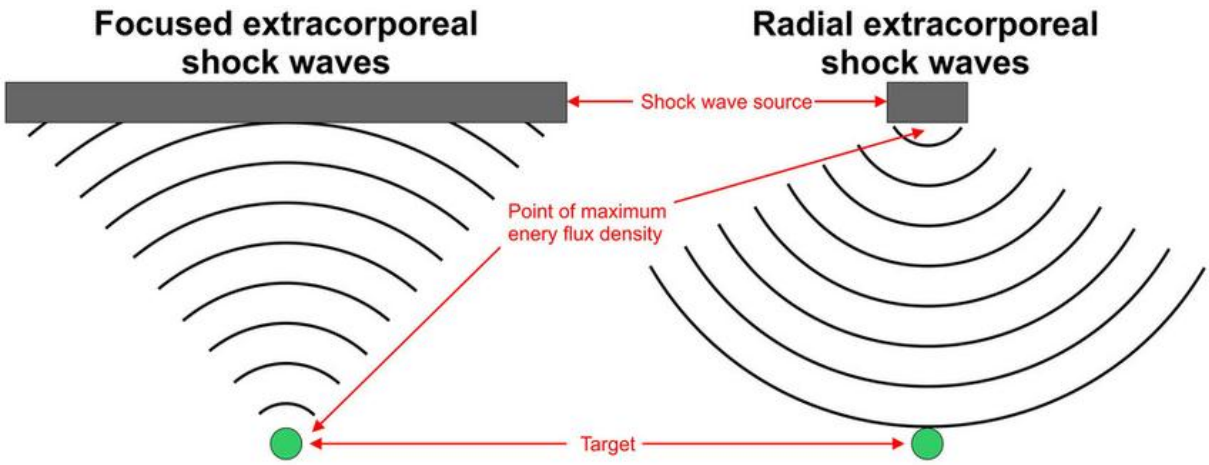

Figure 2.2: The main differences between focused extracorporeal shock waves and radial shock waves (83)**.**

Moreover, the biological process of Mechanotransduction, which explains cellular impacts, should be discussed while discussing the workings of ESWT. The ESWT is being explored as part of the emerging subject of mechanobiology because of the therapeutic benefits of its use of mechanical stimulation (also known as "mechanotherapy") (11). The effectiveness of a novel therapy for spasticity called (rESWT) has been demonstrated in recent studies (14). A meta-analysis of five trials found that after receiving rESWT, patients' ratings on the (MAS) improved significantly (84). Direct mechanical action on the treatment point is the major physical effect of rESWT, while the indirect mechanical effect of cavitation is the secondary result (12). Joint biomechanics should be altered by more than only spastic muscles and tendons. Tendons of opposing muscles can also develop adhesions and contractures. Most research indicates that rESWT can only be used to treat tight muscles (14). There were no distinctions in outcomes between children with cerebral palsy who presented with muscle stiffness and those who presented with muscle weakness when rESWT was used alone. Currently, there are no studies that evaluate the effects of rESWT on agonist and antagonist muscles in the context of treating spasticity caused by a stroke. For up to 6 months, rESWT reduced stiffness in persons with persistent hemiplegia (11).

2.8. Upper Extremity Function

After having a stroke, the muscles can be spasticity and weak. It hurts the quality of life of people who have had a stroke. In the first stage of a stroke, the affected area is both flexible and weak. So, the person can't move the affected limb, especially when it comes to fine motor skills like grasping, reaching, and manipulating objects. On the other hand, patients tend to

use the arm that isn't hurt. This means that the arm that is hurt isn't used, so the hand's function gets worse and the patient becomes dependent. Eventually, the person can't grasp or move objects, especially small ones, in a coordinated way (85). Extracorporeal shock waves and fake extracorporeal shock waves are compared. Two studies on spasticity were done in a forest plot with the Modified Ashworth Scale. The effects were small in both the very short term and the short term. It has been shown that fake shock waves have no effect on spasticity. A comparison between regular physiotherapy and physiotherapy with extracorporeal shock waves. The Modified Ashworth Scale was used to do seven spasticity studies in a forest plot. In two of these studies, fake extracorporeal shock waves were used, but they were not counted because they had no effect. The size of the effect in the very short term was very large, in the short term and medium term it was large, and in the long term it was medium. There are both short-term and long-term benefits, but there was a lot of variation. The Modified Ashworth Scale and the Modified Tardieu Scale were used to do a sub-analysis of elbow spasticity, and the results showed less variation. Four studies were done in a forest plot to see how well they worked, and the results showed that extracorporeal shock waves had a small positive effect. Using the visual analogue scale, two studies on pain showed a significant effect in the short, medium, and long term (86). Independence in day-to-day tasks is closely linked to being good at dexterity and functions, which means being able to hold and move things with your hands and fingers in a coordinated way. Hand and finger control is usually worse after a stroke than proximal upper extremity control, even though the two are closely related. It is also harder to recover. corticospinal and reticulospinal tracts on the same side, extremity It has been said that they can make up for motor problems better in the proximal muscles than in the distal ones (85).

2.9. Effect of Sensory Disorder on Function

Somatosensory impairment impairs control of movements and upper extremity function, and may also impair selective and goal-directed movements. Therefore, the patient may have activity limitation and participation limitation. Sensory input is impaired when there is somatosensory impairment. This impairment affects the ability to function in activities of daily living and participate in social life. Therefore , somatosensory recovery is clinically very important to aid rehabilitation (87). Functionally, problems arising from sensory deficits after stroke can be summarized as impaired perception of sensory information, impaired performance of motor tasks that require somatosensory information, and decreased

rehabilitation results for the upper extremity . Sensation is important for safety, even with adequate motor function. The development of secondary complications such as wounds, abrasions and shoulder-hand syndrome has been associated with sensory impairment. It has been found to be directly related to the development of shoulder pain and subluxation in sensory disorders (88). Impairment in sensory input and processing can disrupt the relationship between the patient and the environment. Van der Lee et al. According to the study, stroke patients with sensory impairment neglect their arm and do not use the affected arm in daily life, so the upper extremity functions and dexterity of the patient are impaired. of the upper extremity Spontaneous use has been noted to be significantly reduced. This lack of sustained use of the affected limb leads to a further reduction in dexterous movements, especially for functional activities that require sustained muscle contraction. This further adds to the model of learned disuse. In the presence of sensory disorders, the functional capacity of upper extremity movements is also impaired (86).

2.10. Sensory Tests Used in Upper Extremity Evaluation

- Tactile Sense

Semmes Weinstein The monofilament test (SWMT) is considered a simple test for somatosensory disorders. It is used by clinicians to evaluate somatosensory disorders in diseases such as diabetes, carpal tunnel syndrome, peripheral nerve disorders, and stroke . In this test, monofilaments of different diameters are used. The patient's eyes are closed, the filament is bent by half its length, and the filament is pressed against the skin at a 90º angle. The filament is held for 1.5 seconds and lifted. In the first step, the examiner tests from the thickest 6.65 to the thinnest 2.83 filaments . Maximum of 3 repetitions. At the start of the test, a trial test is administered to each volunteer once to introduce the test. The first value that cannot be felt from the thickness values of the applied filament is recorded. is a reliable sensory test used to evaluate the upper extremity of patients with stroke (89).

Rating of sensory test is as follows:

0: loss of sensation.

1: monofilament size (6.10-6.65) loss of protective sensation/decreased sensation of deep pressure.

2: monofilament size (5.07-5.88) reduction in protective sensory loss.

3: monofilament size (4.56-4.93) reduction in protective sensory loss.

4: monofilament size (3.84-4.31) decreased protective sensation.

5: light tissue with reduced monofilament size (3.22-3.61).

6: monofilament size (1.65-2.83) means the sense is normal.

-Light Touch

Light touch is another sensory test that examines the decrease in somatosensory. The examiner uses a cotton swab to touch the area to be tested on the upper extremity with their eyes closed and the subject is asked to say "YES" or NO. It is repeated 3 times for each region. At the start of the test, a trial test is administered to each volunteer once to introduce the test. If there is no loss of light touch sensation, “0 point” is given, if there is, “1 point” is given (90).

-Pain Sensation

Pain sensation is evaluated with a pointed- blunt test. Patients should tell whether the sensation they are feeling is pointed or blunt. At the start of the test, a trial test is administered to each volunteer once to introduce the test. If there is no loss in the sense of pain, “0 point” is given, if there is, “1 point” is given (90).

-Two Point Discrimination

Closed-eye testing with a discriminator is used to evaluate two-point discrimination (2PD). The examiner starts with the largest possible gap between the discriminators (100 mm) and works their way down to the minimum possible size (1 mm). The distance between the two points is given as its narrowest possible value. Each participant takes a practice test once at the beginning of the study to familiarize them with the format. In each area, the initial value experienced as a discrete point is recorded as one of the discriminator values. Three repeatable measurements give measurement reliability and reproducibility for assessing 2PD feeling in the fingers, hand, forearm, and arm on both sides of stroke patients (91).

-Vibration Sense

To assess the sense of vibration, the examiner should place his finger on the distal part of the patient. It places it under the interphalangeal joint and presses the vibrameter onto the joint. 100 frequencies are frequently used in studies. For test rating, 0 means normal, 1 means impaired sensory function. This test is a valid and reliable test (92).

- Streognosis Test

This test evaluates the patient's ability to grasp and recognize an object with his eyes closed. This test shows a normal tactile sense, tactile sense discrimination, etc. requires. Different objects are used to perform this test and patients are asked to guess what the objects are (93).

-Proprioception Test (Position Sense)

somatosensory and tactile proprioception , the assessor must position the joint at different angles with the patient's eyes closed. For accuracy of the test, you can use a designated board and place the joint twice at each angle, then the assessor records the angular value of the joint placed by the patient. Finally, the error angle is measured (93).

2.11. Upper Extremity Motor Performance Tests

-Purdue Pegboard Test

Purdue The pegboard test is a test that evaluates the function of the upper extremity . This test consists of nails, washers, and a perforated board assembly. There are two parallel rows of 25 holes on each side of the board. Nails and washers are inserted into the holes on the board. The test consists of 5 parts, 4 main subtests and the sum of three subtests, and finally a test result. These:

- ❖ Right hand fine grip
- ❖ Left hand fine grip
- ❖ Bilateral thin grip
- ❖ The sum of the first three points

consists of the performance of two extremities. Individuals will be briefed about the test and given time to practice before each test. The patient must insert a nail in 30 seconds for the first 3 tests. The maximum number it can do is the test result. First, the dominant hand, then the non -dominant hand, and finally both hands are evaluated at the same time. In the final subtest, individuals use both hands to create sets of nails and washers in a 60-second period. Finally, the total score is obtained by forming the number of nail and stamp sets collected (94).

-Hole Peg Test:

For an evaluation of finger dexterity, the 9-hole peg test (9-HPT) is commonly employed. The test is comprised of a 9-hole board and 9 sticks. The board is situated in front of the players, with the holes on the non-dominant side and the sticks on the dominant side. People are offered a practice exam to take before they fill out an official application, after which they are briefed about the test's procedures. The sticks must be placed on the board as rapidly as possible. One's performance is tracked by noting how long it takes them to go from touching the first stick to placing the last stick on the board. Three separate readings are obtained, and an average is then determined. Then, they are given 9 sticks and asked to remove them one by one using only one hand, with the average of the three times being recorded. The examination is repeated using the non-dominant hand. time units with a maximum of 120 seconds per point. The average time for a person of a similar age to finish the 9-HPT is recorded as 19.4 (2.68). Stroke, multiple sclerosis, brain damage, and tumor patients all have high levels of test-retest reliability (r = 0.98) (95).

-Taylor Hand Function Test

Jebsen The taylor hand function test (JTHFT) is used to measure the speed of performing tasks that consist of a series of activities that are frequently used in daily life. The test consists of 7 parts. First of all, all parts are explained to the individual in detail by the therapist, shown in practice, and individuals are allowed to experiment for better understanding. Each section is evaluated separately for both upper extremities of the individuals, first the non - dominant hand and then the dominant hand, and the time to complete this task is measured with a stopwatch and recorded in seconds.

1. For the page turning task, the person is given a booklet with A4 paper size pages and asked to turn 5 pages as quickly as possible.
2. covers, 2 book clips and 2 coins are used for the task of picking up and dropping small objects. These materials are placed on a plate, spaced apart on the table, just in front of the individual's hand to administer the test. The person will be asked to collect the ingredients in order and place them on an empty plate. The time will start when the person picks up the first object and the time it takes to drop the last object will be recorded.
3. In the task of stacking the backgammon pieces, he is asked to arrange 4 pieces of backgammon pieces spaced apart.

4. large bean grains are used for feeding simulation. People are asked to take the bean grains placed on a plate one by one with the help of a spoon and leave them on a different plate.
5. For the writing task, individuals are given a blank A4 paper and a pen and are asked to write the twenty-four-word sentence shown to them.
6. Five empty cans are used for the transport of light objects, and five full cans are used for the task of transporting heavy objects. Individuals are asked to move the boxes forward in order.

Illness, such as a stroke, brain injury, or rheumatoid Arthritis patients with stable hand problems have been observed to have high test-retest reliability (r = 0.90). According to reports, the average time for people of a similar age to finish this test is 30.4 (1.11 seconds) (95).

-Wolf Motor Function Test

Wolf Motor Function Test (WMFT) consists of 16 sub-steps to evaluate the function of the hand. A score of 0 to 5 is given for each test. The test consists of extending the arm to a specified line on the table, lifting the same pencil from a specified point on the table, abduction and abduction movements of the arm to the table level. Putting the forearm in a box, extending the elbow, extending the elbow with the weight, holding and folding the towel, lifting the basket, turning the key, etc. they are expected to do the tasks and are scored (96).

-Fugl-Meyer Upper Extremity Motor Evaluation Test

Fugl-Meyer upper extremity motor evaluation test is a scale that evaluates motor performance. This scale has 5 sections: motor function, range of motion, pain, sensory function, and balance. We have 3 scales in each area: (0 = unable to perform, 1 = partially achieves, 2 = fully performs). In this scale, grip, shoulder movement (retraction, extension), elbow, forearm, wrist, finger movements, etc. 66 points for the upper extremity , hip, knee, ankle and finger movements for the lower extremity and 34 points for the heel (97).

-Box and Block Test

In this test, patients sit in a chair facing a table with boxes and blocks on it. The patient must move the blocks one at a time from one place to another for one minute. The recorded score is the number of blocks moved from one compartment to another in 1 minute (97).

- Manual Function Test

Manual function test (MFT) is an assessment method used to evaluate upper extremity function. Eight tasks are performed in a standard way. For the testing of arm movements, four tasks are applied: flexion of the "upper extremity " abduction ", "palm touching the occiput " and "palm touching my back ". We use two tasks for the grip and bastard test: "grip" and " pinch ". We use two tasks to evaluate arm and hand activities: "carrying cubes" (CC) and “wooden block.” The maximum possible value for the total score is 32. MFT is a reliable and valid method for evaluating upper extremity functional disorders in stroke patients (98).

-Chedoke Arm and Hand Stroke Evaluation

The Chedoke assessment is a test used to evaluate functional limitation in the upper extremity in stroke patients. This test includes 13 items assessed using a 7-point quantitative scale: opening a coffee jar, calling 911, drawing a line with a ruler, putting toothpaste on the toothbrush, cutting medium paste, pouring water into a glass, squeezing the cloth, cleaning the lens , zipping up, buttoning up five buttons, drying with a towel, putting the container on the table, carrying the bag up the stairs (99).

The following scoring is applied for each task performed:

1. Complete independence (on time, secure).
2. With the help of the device can do a companion.
3. Able to do under supervision.
4. Gets minimal help (subject=75%).
5. She gets moderate help (subject=50%) Full dependency (helpful).
6. Can do with maximum assistance (subject=25%).
7. Totally dependent (subject=0%) (100).

-Action Research Arm Test

Action Research The Arm Test (ARAT) was developed by Lyle in 1981. This test evaluates upper extremity function and dexterity. This test contains 19 items. ARAT includes subtests; grasping (6 items; 0-18 points), grasping (4 items; 0-12 points), pinching (6 items; 0-18 points) and rough gestures (3 items; 0-9 points). This test starts with the less affected side. The patient receives a total score ranging from 0 to 57 points (101).

-Assessment of Pain

The visual analog scale (VAS) is a simple and frequently used method for assessing variations in pain intensity. Pain is assessed using a 10 cm horizontal axis. 0: no pain, 10: the worst possible pain. post-stroke spasticity is a very important and serious problem. Despite many studies on the efficacy of ESWT and dry needling on spasticity , these two modalities are not used in pain, upper extremity function and spasticity , especially biceps. There are no studies comparing it in the brachii muscle. Therefore, the aim of this study was to determine the upper extremity of dry needling with ESWT. To compare its effectiveness on spasticity, function and pain (102).

3. MATERIALS AND METHODS

3.1. Study Design and Participants

This study examined the effects of rESWT on the upper limbs of individuals with stroke. This is a two-group, pre- and post-test clinical trial with repeated measurements to determine the effect of rESWT on spasticity, upper extremity function, and range of motion.

On July 6, 2022, the Clinical Research Ethics Committee of the Iraqi Ministry of Health approved this study (code: 322). It was conducted in Wasit province, Iraq, at the Wasit Disabled Rehabilitation Center/physiotherapy and rehabilitation unit. 48 participants volunteered for the study. Participants were selected from Wasit Disabled Rehabilitation Center health clinics.

48 patients (female=12 and male=36), the participants were randomly split into two groups, A and B, with 24 patients in each group. Group A used conventional physiotherapy, contain (male=20 and female=4). Group B used rESWT with conventional therapy contain (male=16 and female=8).

Inclusion criteria for the study:

1. Aged 50–80 years.
2. A stroke diagnosed at least six months ago.
3. Having a stroke for the first time
4. Stable spasticity (no changes in the preceding two months) in the elbow and wrist.
5. Ability to understand commands.
6. Stable vital signs.
7. Unchanged drug doses that could affect its spasticity.
8. Not taking antispastic drugs.
9. Modified Ashworth scale (MAS) upper extremity score at least 1+.
10. No severe contracture of the elbow and wrist.

Exclusion criteria in the study:

1. Botox, alcohol, or phenol are treatments that can block.
2. Having any surgery in upper limb.

3. History of epilepsy, severe mental disorders, and cancerous tumors, as well as a history of blood clots in the veins of the limbs.

4. Having any problem in nervous system.

5. Receiving another management.

6. ESWT contraindications (pregnancy, on major vessels and nerves, pacemaker or other. implanted devices, open wounds, joint replacements, Pineal, blood clotting disorders including thrombosis, infection and cancer).

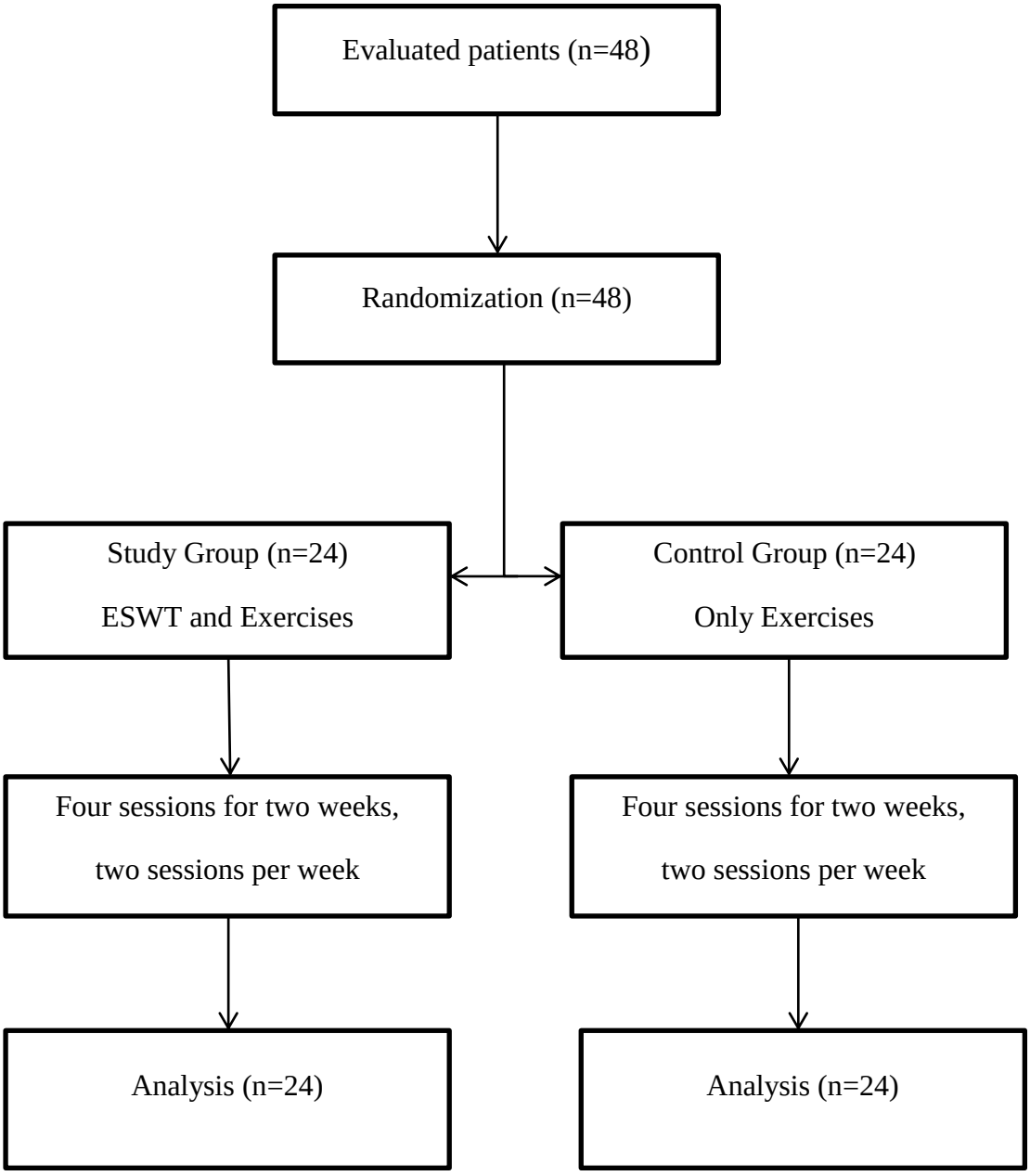

Figure 3.1: Diagram showing the number of patients included, the randomized study and the groups.

3.2. Sample Size

The sample size of the study was calculated as at least 21 individuals in a group, with a 95% confidence interval and 80% power. Analyses made in the G Power program (ver. 3.1.9.7) by looking at the literature (Ref). Against the probability of 15% drop out, this number was determined as 24 individuals for each group and totally 48 individuals for 2 groups.

3.3. Treatment Procedure

After patient distribution, demographic information and the initial assessment have been done for all patients in both groups (MAS, FMA-UE, and ROM).

All participants in this study received conventional physiotherapy (stretching exercises, ROM exercises, common MAT activities. strengthening exercises, gentle and controlled weight-bearing exercises, balance and coordination exercises) for ten sessions, five sessions per week, Perform all of these exercises once a day for 10 repetitions in two sets with a 10-second hold under the supervision of a physiotherapist.

Group (A) was treated with conventional physiotherapy only . The other group (B) got rESWT and conventional physiotherapy, rESWT was four sessions, with two sessions per week.

The way ESWT worked was to have the patient lie on his or her back on a comfortable bed with the hand on a medical couch in a supine position. All of the joints in the upper limbs were then stretched out. Each patient had 3000 shots, 3 bars, and a frequency of 5 HZ. The ESWT prop was put on the affected side's biceps and flexor carpi radialis muscles and moved on these muscles. and a gel that is used as a conductor.

After two weeks, at the end of the fourth session, the final evaluation is done on all patients in both groups (A and B).

3.4. Outcome Measurements

3.4.1. Evaluation of Spasticity

Modified Ashowrth Scale (MAS) Score: Using the MAS, the level of spasticity of the elbow (E) and radio carpal (RC) joints was clinically examined. This scale is a velocity-independent approach for physically assessing passive joint range of motion. It goes from 0 (normal muscle tone) to 4 (very restricted range of motion, limb rigid in flexion or

extension). A grade 1+ suggests a minor increase in muscle tone, shown by a catch, followed by moderate resistance throughout the duration of the range of motion (103).

General Specifics (103):

• Place the patient in the supine position " One Mississippi"

• When evaluating a muscle that primarily stretches a joint, insert the joint in its most extended position and move it to its most flexed position over one second (count) "a thousand and one"

• Score based on the following classification

Scoring(103):

0: No gain in muscle strength.

1: Slight increase in muscle tone, shown by a catch and release or minimal resistance at the end of the range of motion when the affected part is moved in flexion or extension.

1+: A slight increase in muscle tone is shown by a catch, and then there is very little resistance for the rest (less than half) of the ROM.

2: There was a more noticeable increase in muscle tone over most of the ROM, but the affected part(s) moved easily.

3: Significant increase in muscle tone, hard to move passively.

4: The part or parts that are hurt can't bend or straighten.

3.4.2. Evaluation of Function

Fugl-Meyer Assessment-Upper Extremity (FMA-UE): FMA-UE evaluation of functional recovery after UE impairment. The Five-Motif Approach (FMA) considers (motor, sensory, balance, range of motion, and pain). Movement, reflex, coordination, and speed are all evaluated by the FMA-motor UE's subtest. Many items in each domain are scored on an ordinal scale from 0 (does not perform) to 2 (completely performs). The total score on the FMA-UE is out of 66, with the upper arm receiving a score of 36 and the wrist and hand receiving a score of 30. All 33 elements from the FMA-UE were used in this analysis (104).

3.4.3. Range of Motion Measurement (ROM)

The articular ROM was measured with a standard manual goniometer at the joint's maximum active flexion and extension, and the active range of motion was found by adding the two numbers(105) (AROM). Both MAS and goniometric measurements were done by the same person.

Hemiparesis is a lack of active range of motion (ROM) and static or moving muscle strength on one side of the body. In this study, we showed the results of shoulder flexion, extension, and abduction, elbow flexion and extension, and wrist flexion and extension.

The patient sits in a chair or on a treatment bed, and the researcher measures how far the joints can move (shoulder, elbow and wrist joint).

3.5. Statistical analysis:

Descriptive statistics were presented in the form of numbers and percentages for the categorical variables, while mean and standard deviation were used for the numerical variables. Chi-square test and independent t-test were used to compare between group without ESWT and group with ESWT. Chi-square test was used for the categorical variables; gender, stroke, affected site, and hand dominance. Independent t-test was used for the numerical variables; age, height, weight, and BMI. Mixed ANOVA method was done to compare different measurements between group without ESWT and group with ESWT before and after physiotherapy.

IBM SPSS for windows version 28 was used for the analysis. A p-value <0.05 is considered statistically significant.

4. RESULTS

48 patients with upper limb spasticity (male = 36, female = 12) who met the inclusion criteria were randomly allocated to one of two management programs (Group A: conventional physiotherapy without ESWT; Group B: conventional physiotherapy with ESWT). In addition to other demographic information, age, sex, affected side, types of stroke, dominant hand, height, weight, and body mass index of patients were documented. Group A had a mean age of 59.21 years, while B had a mean age of 59.96 years. In group A, there were 20 men (83.3%), while in group B, there were 16 men (66.7%), and 8 women (33.3%). In both groups A and B, 14 (58.3%) strokes were hemorrhagic and 10 (41.7%) were ischemic. In group A, the right side was affected 13 (54.2%) and the left side 11 (45.8%), whereas in group B, the right side was affected 14 (58.3%) and the left side 10 (41.7%). In group A, the height was 171.9 ± 7.4 cm while it was 169.83 (6.34%) in group B. In group A, the weight was 81.1 ± 10.2 kg, whereas in group B, the weight was 82.5 ± 9.7 kg. A group's BMI was 27.4 ± 2.8, while B group's BMI was 28.6 ± 2.6 kg/cm^2. No statistically significant differences were found between the two groups. Characteristics of participants are presented in (Table 4.1).

Table 4.1: Characteristics of participants (N=48).

		Group without ESWT N=24		Group with ESWT N=24		P-value
		N	%	N	%	
Gender	Male	**20**	**83.3**	**16**	**66.7**	**0.182**
	Female	**4**	**16.7**	**8**	**33.3**	
Stroke type	Hemorrhagic	**14**	**58.3**	**14**	**58.3**	**0.999**
	Ischemic	**10**	**41.7**	**10**	**41.7**	
Affected site	Right	**13**	**54.2**	**14**	**58.3**	**0.999**
	Left	**11**	**45.8**	**10**	**41.7**	
Hand dominance	Left	**1**	**95.8**	**2**	**91.7**	**0.999**
	Right	**23**	**4.2**	**22**	**8.3**	
		Mean	SD	Mean	SD	
Age		**59.21**	**5.71**	**59.96**	**5.45**	**0.644**
Height (cm)		**171.92**	**7.42**	**169.83**	**6.34**	**0.301**
Weight (kg)		**81.08**	**10.29**	**82.58**	**9.74**	**0.606**
BMI (Kg/m^2)		**27.41**	**2.84**	**28.59**	**2.65**	**0.144**

Table 4.2: Comparison of motor function between the two groups before and after physiotherapy**.**

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**22.79±9.79**	**24.96±13.90**
After physiotherapy	**24.29±9.99**	**27.17±14.64**
	P-value	Partial eta squared
Time	**0.014**	**0.125**
Group	**0.627**	**0.011**
Time*group	**0.626**	**0.005**

A statistically significant difference with moderate effect size was found between motor function before and after physiotherapy, motor function after physiotherapy was higher than motor function before physiotherapy, F (1) =6.59, p-value=0.014. There was no statistically significant difference between groups with or without ESWT, F (1)= 0.528, p=0.60. p=0.626 indicates that there was no significant interaction between physiotherapy and groups with or without ESWT.

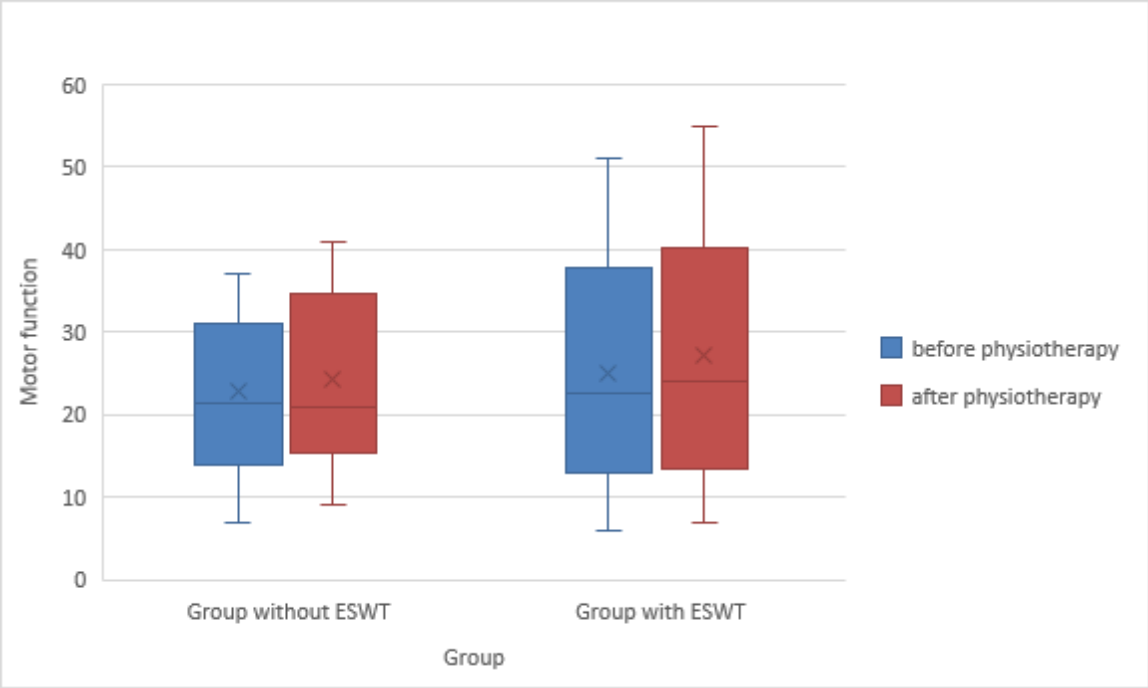

Figure 4.1: Estimated Marginal Means of Motor Function.

Table 4.3: Comparison of sensation between the two groups before and after physiotherapy**.**

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**8.83±2.036**	**8.08±3.189**
After physiotherapy	**9.83±1.761**	**9.25±2.418**
	P-value	Partial eta squared
Time	**<0.001**	**0.344**
Group	**0.318**	**0.022**
Time*group	**0.707**	**0.003**

A statistically significant difference with large effect size was found between sensation before and after physiotherapy, sensation after physiotherapy was higher than sensation before physiotherapy, F (1) = 24.14, p-value<0.001.

There was no statistically significant difference between groups with or without ESWT, F (1) =1.01, p-value=0.318. No significant interaction was found between physiotherapy and groups with or without ESWT, p-value = 0.707.

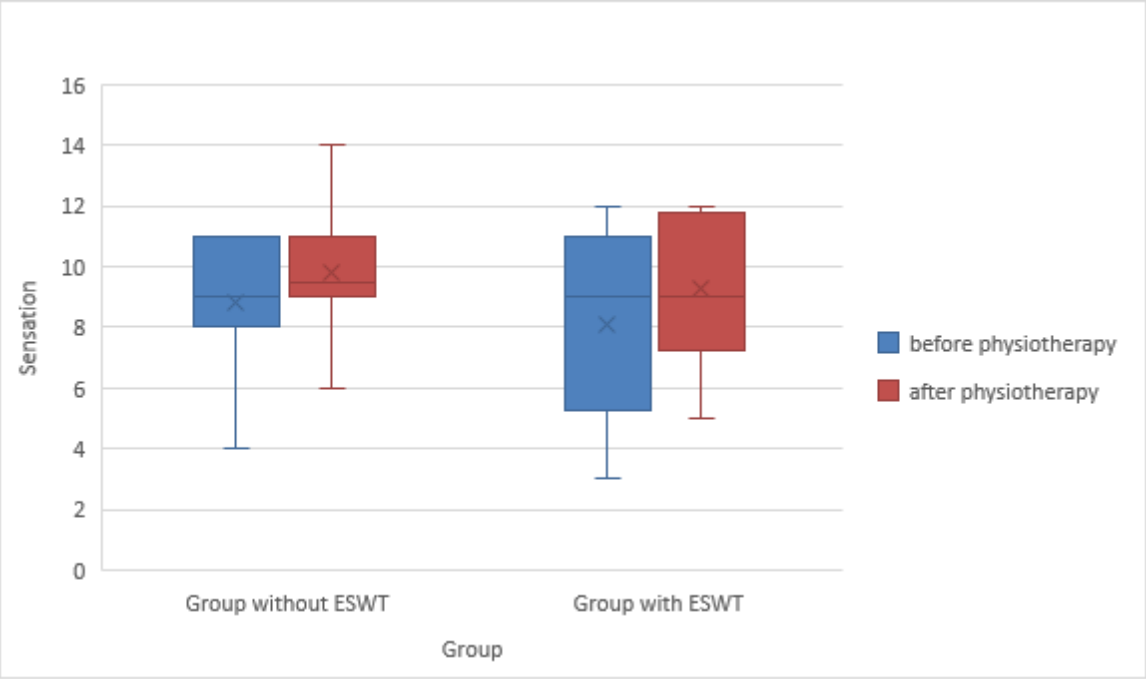

Figure 4.2: Estimated Marginal Means of Sensation.

Table 4.4: Comparison of passive joint motion between the two groups before and after physiotherapy.

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**17.63±4.00**	**16.38±3.94**
After physiotherapy	**18.83±3.57**	**18.42±3.93**
	P-value	Partial eta squared
Time	**<0.001**	**0.474**
Group	**0.447**	**0.013**
Time*group	**0.106**	**0.056**

A statistically significant difference with large effect size was found between passive joint motion before and after physiotherapy, passive joint motion score after physiotherapy was higher than passive joint motion score before physiotherapy, F (1) =41.37, p-value<0.001.

There was no statistically significant difference between groups with or without ESWT, F (1) =0.588, p-value= 0.447. No significant interaction was found between physiotherapy and groups with or without ESWT, p-value= 0.106.

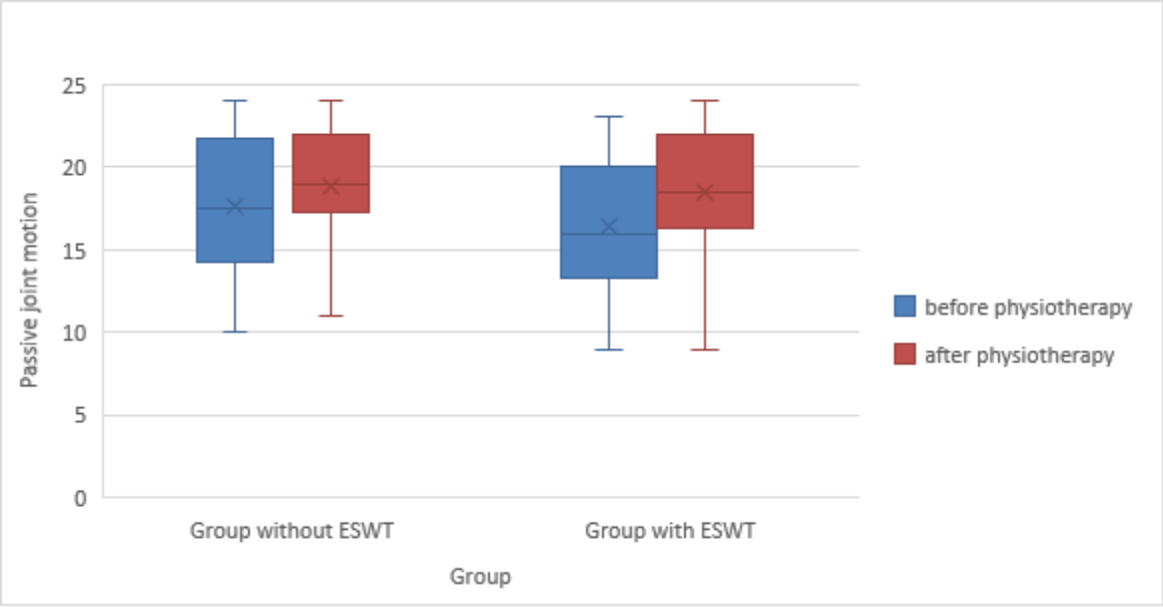

Figure 4.3: Estimated Marginal Means of Passive Joint Motion.

Table 4.5: Comparison of joint pain score between the two groups before and after physiotherapy.

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**10.50±4.21**	**11.38±4.38**
After physiotherapy	**11.42±4.06**	**12.96±4.50**
	P-value	Partial eta squared
Time	**<0.001**	**0.248**
Group	**0.318**	**0.022**
Time*group	**0.305**	**0.023**

A statistically significant difference with large effect size was found between joint pain score before and after physiotherapy, joint pain score after physiotherapy was higher than joint pain score before physiotherapy, F (1) =41.37, p-value<0.001.

There was no statistically significant difference between groups with or without ESWT, F (1) =1.02, p-value=0.318. No significant interaction was found between physiotherapy and groups with or without ESWT, p-value=0.305.

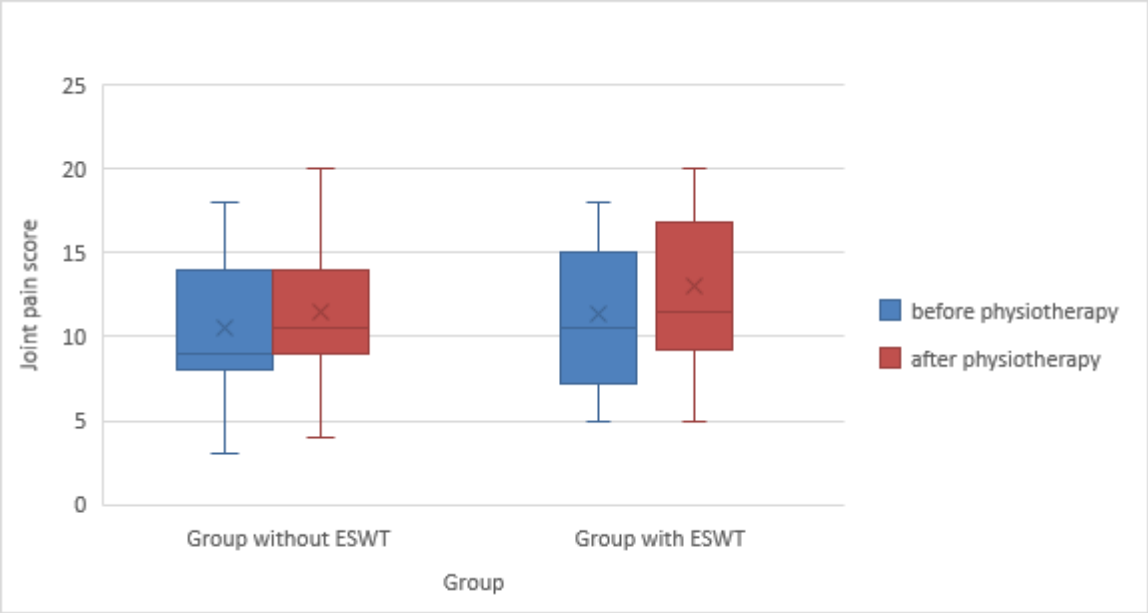

Figure 4.4: Estimated Marginal Means of Joint Pain Score

Table 4.6: Comparison of biceps flexor score between the two groups before and after physiotherapy in MAS.

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**1.88±0.90**	**2.04±0.95**
After physiotherapy	**1.67±0.76**	**1.58±0.72**
	P-value	Partial eta squared
Time	**<0.001**	**0.309**
Group	**0.858**	**0.001**
Time*group	**0.096**	**0.059**

A statistically significant difference with large effect size was found between biceps flexor score before and after physiotherapy, biceps flexor score after physiotherapy was lower than biceps flexor score before physiotherapy, F (1) =20.58, p-value<0.001.

There was no statistically significant difference between groups with or without ESWT, F (1) =0.033, p-value=0.858. No significant interaction was found between physiotherapy and groups with or without ESWT, p-value=0.096.

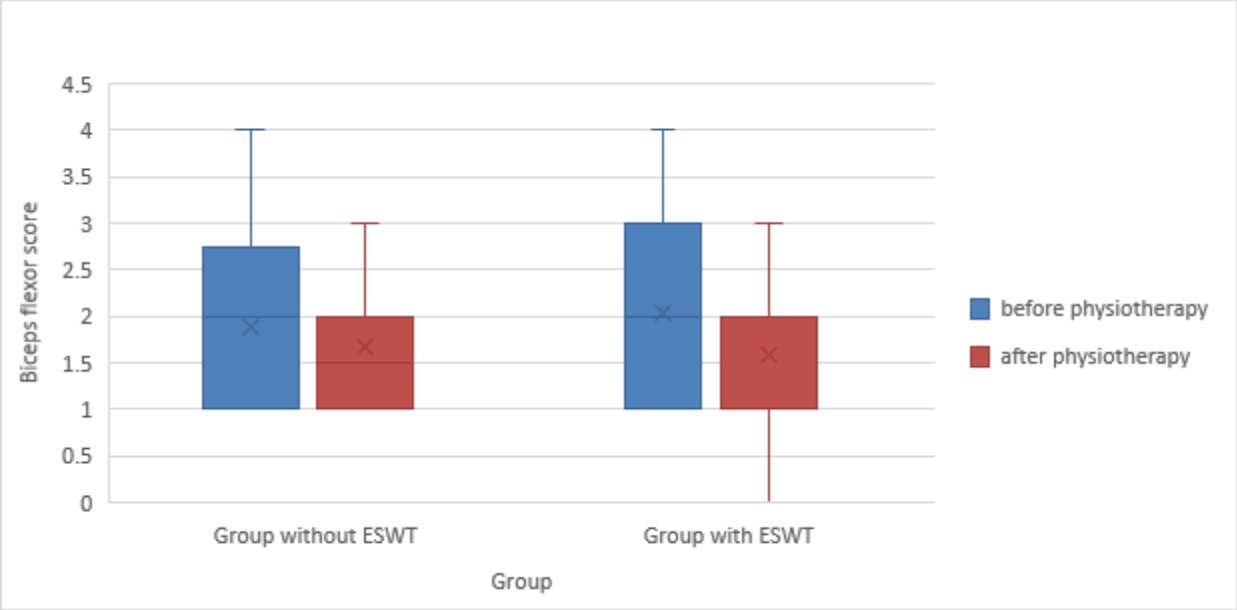

Figure 4.5: Estimated Marginal Means of Biceps Flexor.

Table 4.7: Comparison of wrist flexor score between the two groups before and after physiotherapy in MAS**.**

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**1.79±0.66**	**1.83±0.82**
After physiotherapy	**1.63±0.65**	**1.42±0.72**
	P-value	Partial eta squared
Time	**<0.001**	**0.308**
Group	**0.672**	**0.004**
Time*group	**0.059**	**0.076**

A statistically significant difference with large effect size was found between wrist flexor score before and after physiotherapy, wrist flexor score after physiotherapy was lower than wrist flexor score before physiotherapy, F (1) =20.49, p-value<0.001.

There was no statistically significant difference between groups with or without ESWT, F (1) =0.182, p-value=0.672. No significant interaction was found between physiotherapy and groups with or without ESWT, p-value=0.059.

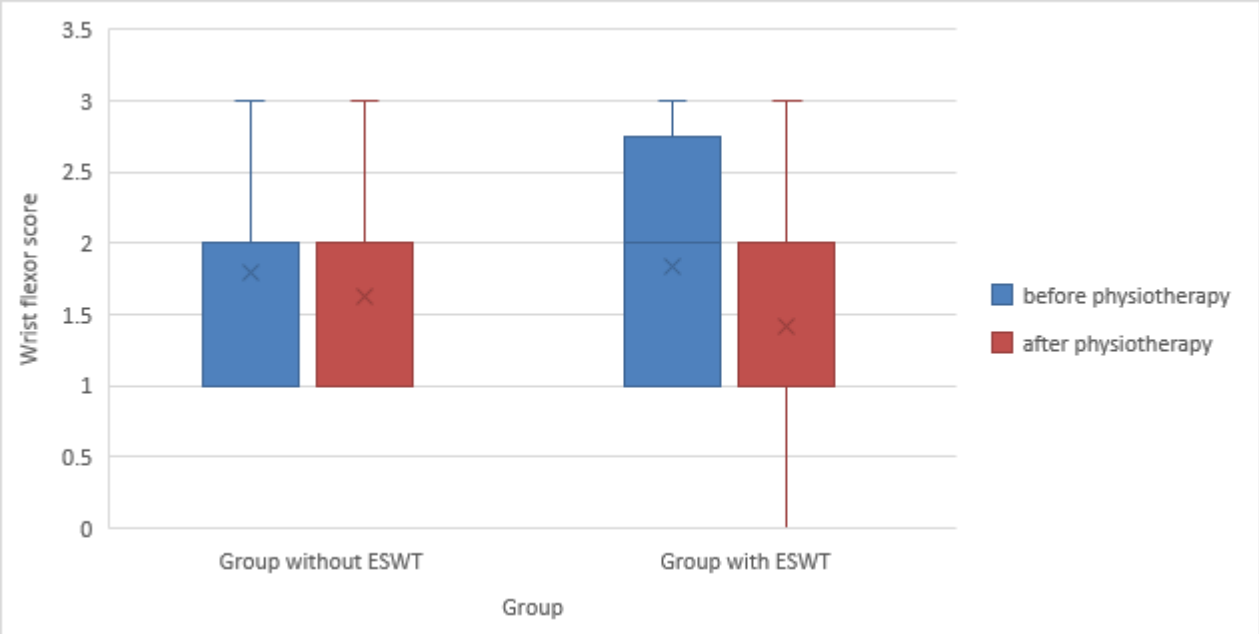

Figure 4.6: Estimated Marginal Means of Wrist Flexor.

Table 4.8: Comparison of shoulder flexion score between the two groups before and after physiotherapy in MAS.

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**31.04±24.58**	**45.83±45.48**
After physiotherapy	**33.33±25.40**	**50.83±46.38**
	P-value	Partial eta squared
Time	**0.003**	**0.157**
Group	**0.135**	**0.048**
Time*group	**0.251**	**0.029**

A statistically significant difference was found between Shoulder flexion score before and after physiotherapy shoulder flexion score after physiotherapy was higher than shoulder flexion score before physiotherapy, F (1) =9.78, p-value=0.003.

There was no statistically significant difference between groups with or without ESWT, F (1) =2.3, p-value=0.135. No significant interaction was found between physiotherapy and groups with or without ESWT, p-value=0.251.

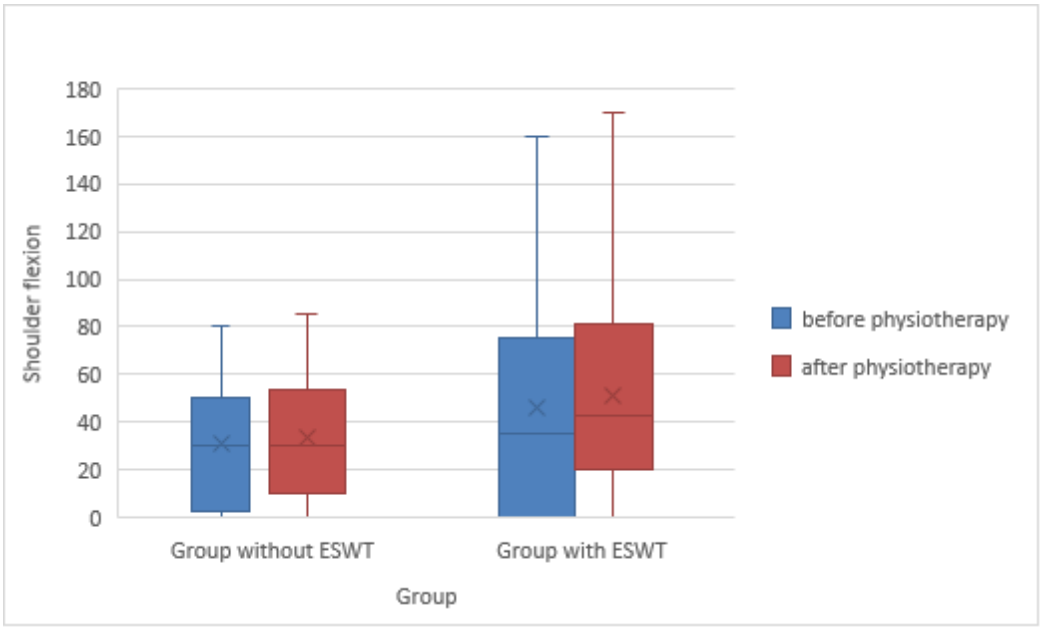

Figure 4.7: Estimated Marginal Means of Shoulder Flexion.

Table 4.9: Comparison of ROM of shoulder external rotation between the two groups before and after physiotherapy**.**

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**7.29±7.22**	**7.29±8.72**
After physiotherapy	**7.92±7.36**	**7.71±9.55**
	P-value	Partial eta squared
Time	**0.025**	**0.105**
Group	**0.965**	**<0.001**
Time*group	**0.645**	**0.005**

A statistically significant difference with moderate effect size was found between Shoulder external rotation score before and after physiotherapy, Shoulder external rotation score after physiotherapy was higher than shoulder external rotation score before physiotherapy, F (1) =5.73, p-value=0.025.

There was no statistically significant difference between groups with or without ESWT, F (1) =0.002, p-value=0.965. No significant interaction was found between physiotherapy and groups with or without ESWT, p-value=0.645.

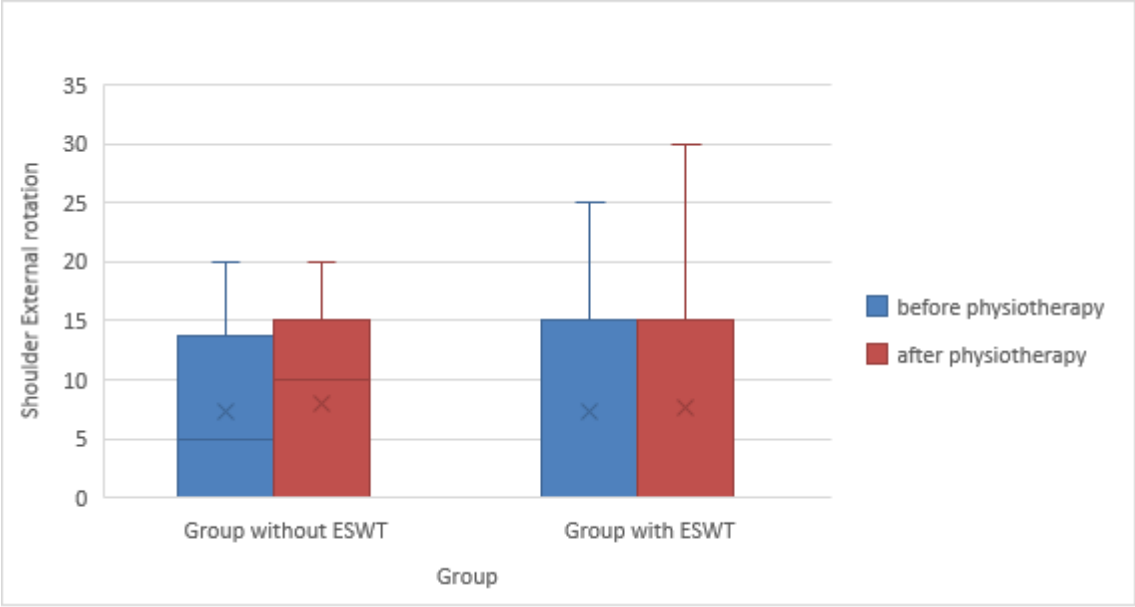

Figure 4.8: Estimated Marginal Means of Shoulder External Rotation.

Table 4.10: Comparison of ROM of shoulder abduction between the two groups before and after physiotherapy**.**

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**10.63±9.59**	**26.67±31.75**
After physiotherapy	**12.29±9.89**	**28.96±32.17**
	P-value	Partial eta squared
Time	**<0.001**	**0.303**
Group	**0.020**	**0.112**
Time*group	**0.484**	**0.011**

A statistically significant difference with large effect size was found between shoulder abduction score before and after physiotherapy, shoulder abduction score after physiotherapy was higher than shoulder abduction score before physiotherapy, F (1) =20.01, p-value<0.001.

There was no statistically significant difference between groups with or without ESWT, shoulder abduction score was higher in group with ESWT as compared to group without ESWT,

F (1) =5.77, p-value=0.020. No significant interaction was found between physiotherapy and groups with or without ESWT, p-value=0.484.

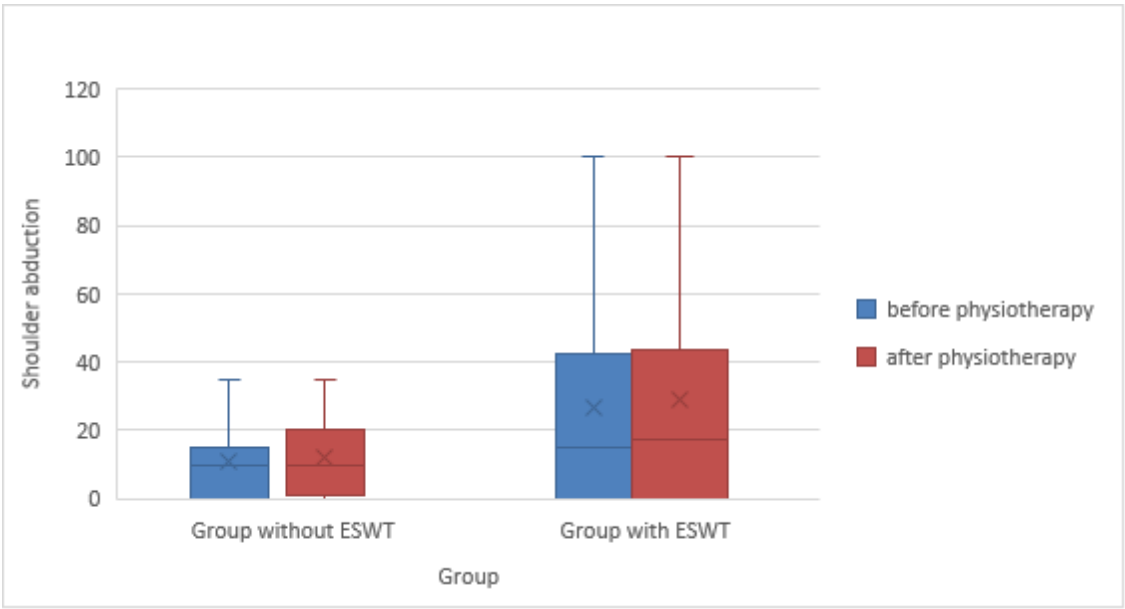

Figure 4.9: Estimated Marginal Means of Shoulder Abduction.

Table 4.11: Comparison of ROM of elbow flexion between the two groups before and after physiotherapy.

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**86.88±33.36**	**91.25±34.77**
After physiotherapy	**92.92±32.63**	**99.38±36.10**
	P-value	Partial eta squared
Time	**0.001**	**0.214**
Group	**0.578**	**0.007**
Time*group	**0.605**	**0.006**

A statistically significant difference with large effect size was found between elbow flexion score before and after physiotherapy, elbow flexion score after physiotherapy was higher than elbow flexion score before physiotherapy, F (1) =12.53, p-value=0.001.

There was no statistically significant difference between groups with or without ESWT, F (1) =0.313, p-value=0.578. No significant interaction was found between physiotherapy and groups with or without ESWT, p-value=0.605.

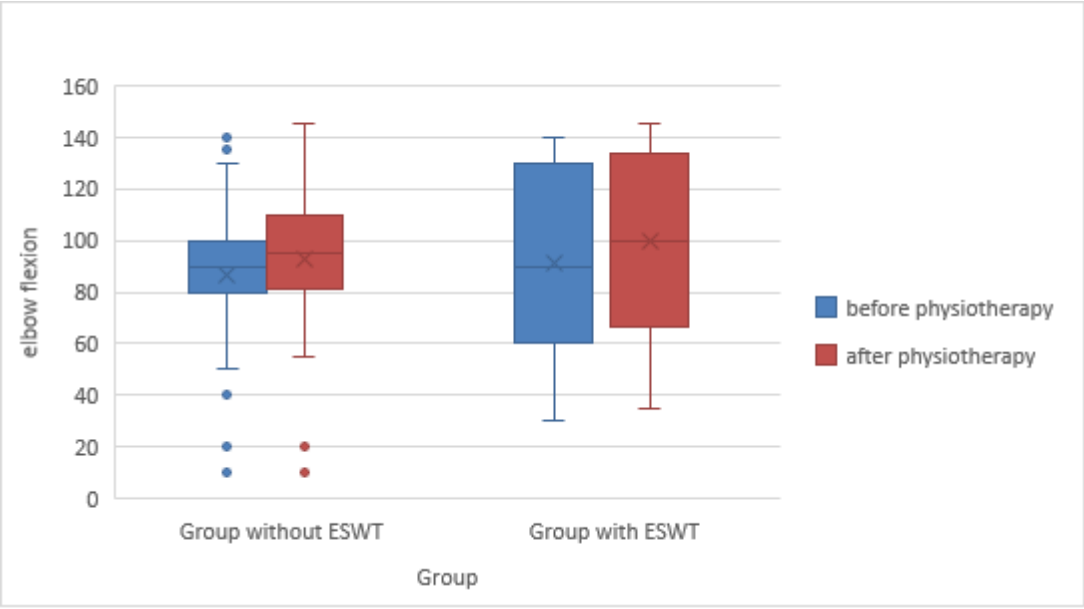

Figure 4.10: Estimated Marginal Means of Elbow Flexion.

Table 4.12: Comparison of ROM of elbow extension between the two groups before and after physiotherapy**.**

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**9.17±8.43**	**6.88±7.78**
After physiotherapy	**7.71±8.07**	**4.79±6.16**
	P-value	Partial eta squared
Time	**0.003**	**0.173**
Group	**0.229**	**0.031**
Time*group	**0.587**	**0.006**

A statistically significant difference with large effect size was found between elbow extension score before and after physiotherapy, elbow extension score after physiotherapy was lower than elbow extension score before physiotherapy, F (1) =9.61, p-value=0.003.

There was no statistically significant difference between groups with or without ESWT, F (1) =1.48, p-value=0.299. No significant interaction was found between physiotherapy and groups with or without ESWT, P-value=0.587.

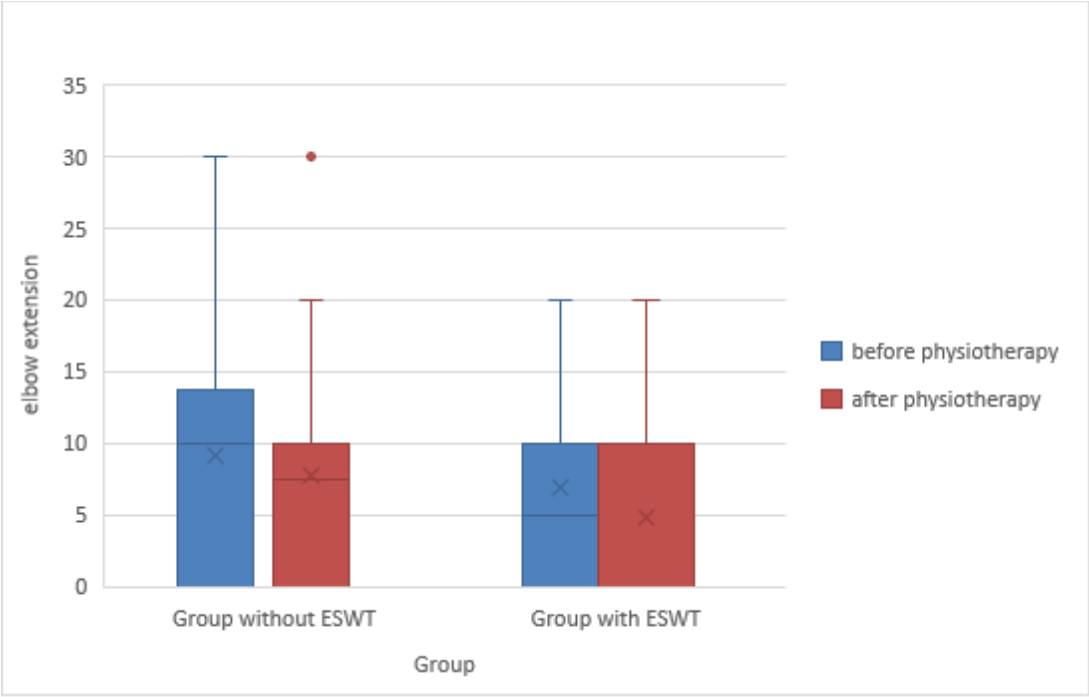

Figure 4.11: Estimated Marginal Means of Elbow Extension.

Table 4.13: Comparison of ROM of wrist flexion between the two groups before and after physiotherapy.

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**18.96±14.82**	**22.71±16.15**
After physiotherapy	**22.08±16.01**	**25.42±16.48**
	P-value	Partial eta squared
Time	**<0.001**	**0.327**
Group	**0.439**	**0.013**
Time*group	**0.737**	**0.002**

A statistically significant difference with large effect size was found between wrist flexion score before and after physiotherapy, wrist flexion score after physiotherapy was higher than wrist flexion score before physiotherapy, F (1) =22.73, p-value<0.001.

There was no statistically significant difference between groups with or without ESWT, F (1) =0.608, p-value=0.439. No significant interaction was found between physiotherapy and groups with or without ESWT, p-value=0.737.

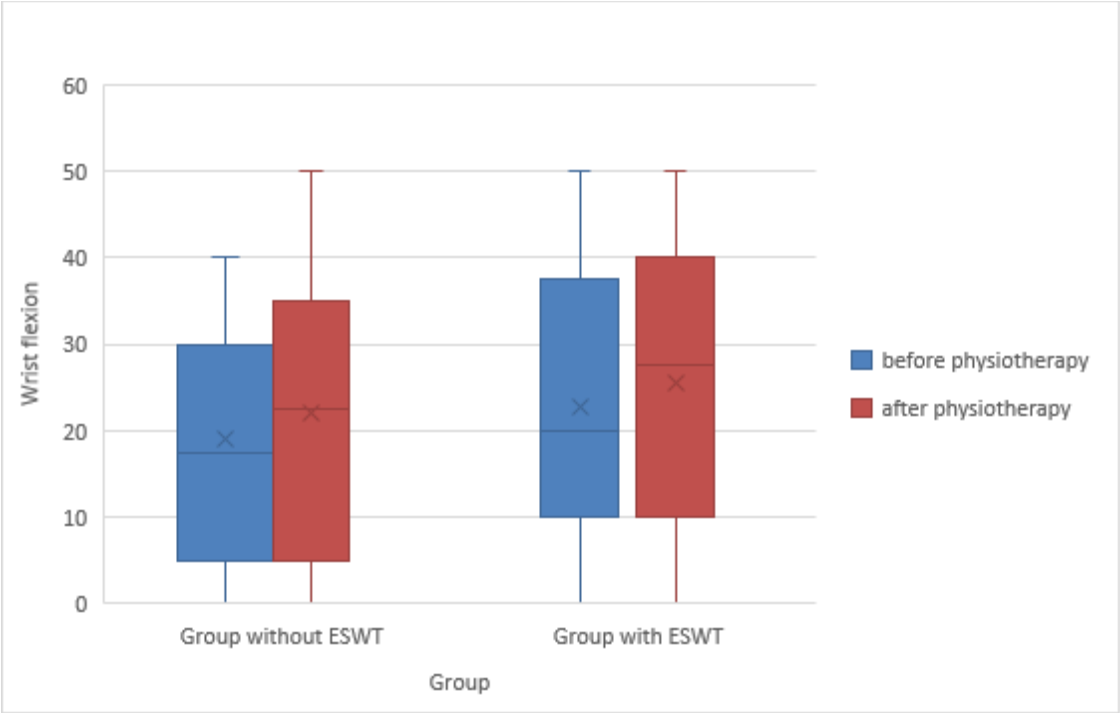

Figure 4.12: Estimated Marginal Means of Wrist Flexion.

Table 4.14: Comparison of ROM of wrist extension between the two groups before and after physiotherapy**.**

	Group without ESWT N=24	Group with ESWT N=24
Before physiotherapy	**13.33±13.88**	**13.54±14.41**
After physiotherapy	**15.00±14.89**	**14.79±15.21**
	P-value	Partial eta squared
Time	**<0.001**	**0.228**
Group	**0.439**	**<0.001**
Time*group	**0.737**	**0.006**

A statistically significant difference was found between wrist extension score before and after physiotherapy, wrist extension score after physiotherapy was higher than wrist extension score before physiotherapy, F (1) =13.75, p-value=0.001.

There was no statistically significant difference between groups with or without ESWT , P-value>0.999. No significant interaction was found between physiotherapy and groups with or without ESWT, p-value=0.601.

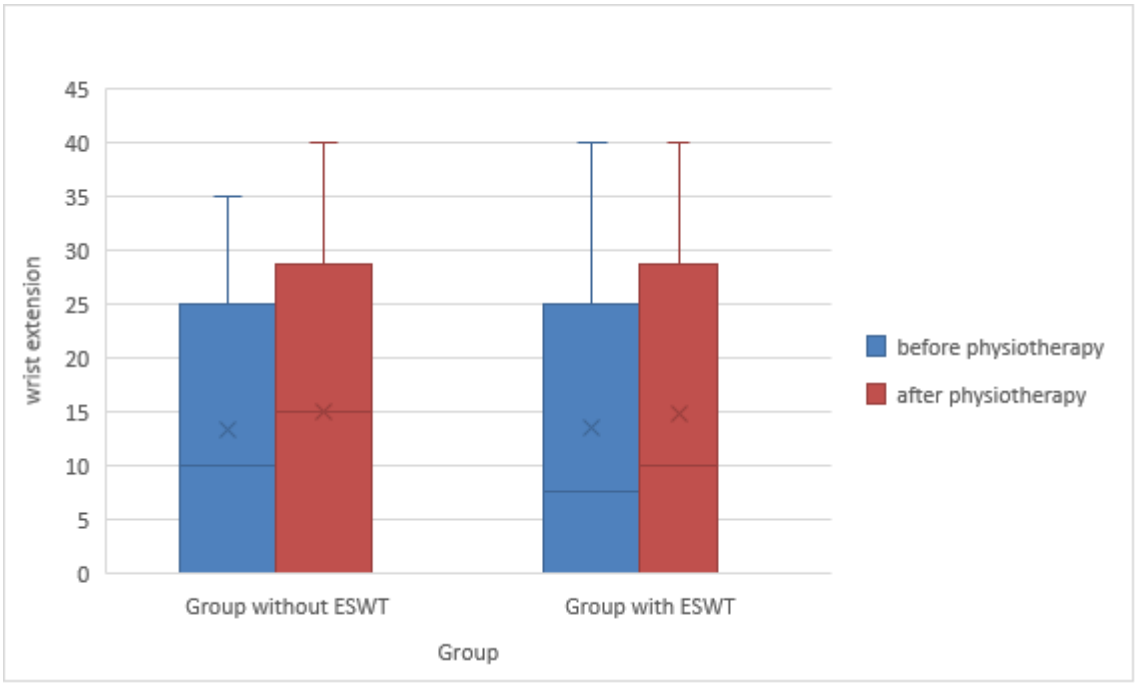

Figure 4.13: Estimated Marginal Means of Wrist Extension.

5. DISCUSSION

In this study, we wanted to find out how efficiently ESWT and a conventional physiotherapy program worked on spasticity and motor functions in the upper extremities of stroke patients who had developed spasticity in their elbow flexors and wrist flexors. In patients treated with ESWT and the conventional physiotherapy, the results showed that spasticity, functional status, and upper extremity range of motion (ROM) all got better. In patients treated with conventional physiotherapy alone, the results showed that spasticity, functional status, and ROM in the upper extremities got better. Therefore, we could say that ESWT has no effect on spasticity and upper extremity functionality in stroke patients.

Spasticity is a common problem for people who have had a stroke. The definition of this phenomenon is an increase in muscle tone after passive stretching that depends on the speed of the stretching. This is caused by the supraspinal disinhibition of stretch reflexes. Patients who have had their first stroke are 39% more likely to have spasticity after 12 months (39).

Motor function, sensation, joint pain, passive range of motion, active range of motion, and spasticity status all showed significant differences before and after treatment. According to the FMA-UE used for gross motor function assessment, there was a noticeable difference between before and after applying the treatment program in the two groups. There was no noticeable difference between the groups receiving ESWT and those who did not. No significant interaction was found between physiotherapy and groups with or without ESWT.

In Modified Ashworth Scale (MAS) in this study we have evaluated biceps brachii and wrist flexor muscles, there was significant difference between biceps score before and after physiotherapy, biceps score after physiotherapy is lower than biceps flexor score before physiotherapy, slightly significant difference was found between different groups with or without ESWT, no significant interaction was found between physiotherapy and groups with or without ESWT. While a significant difference was found between wrist flexion score before and after physiotherapy, wrist flexion score after physiotherapy is higher than wrist flexion score before physiotherapy, slightly significant difference was found between different groups with or without ESWT, no significant interaction was found between physiotherapy and groups with or without ESWT Li T-Y et al. significant reductions in spasticity observed in those that received rESWT. After three sessions of rESWT, this

reduction lasted a minimum of 16 weeks; after one session, it lasted between 8 and 12 weeks. Three sessions of rESWT had a greater and longer-lasting benefit than a single session, particularly in terms of wrist spasticity. In furthermore, the reduction in spasticity following three sessions of rESWT may be advantageous for hand function and wrist control, and the benefit was maintained for 16 and 12 weeks, respectively (12) . Wissel et al. elbow flexor in work emphasized that spasticity is the most common site where it develops, therefore, it is important to treat the elbow flexor in order to reduce pain and spasticity and ultimately improve upper extremity function in stroke patients (106). Li et al. studied the effect of treatment on agonist and antagonist muscles on spasticity in post-stroke patients with elbow flexor spasticity. It shows statistically that spasticity is significantly reduced regardless of the treatment in agonist or antagonist muscles, but that treatment in agonist muscle is more effective than in antagonist muscles (107).

It is very difficult to select the best modalities for the treatment of spasticity due to the interaction of spasticity with various components of the upper motor neuron syndrome, the heterogeneous patient population, and the lack of ideal criteria for spasticity management (108). In recent years, ESWT treatments have been widely used in the treatment of post-stroke spasticity and pain, but treatment is used in the upper extremity (108). Studies on the treatment of spasticity are limited (87), it was determined that ESWT treatment significantly reduced spasticity in the MAS score . Li et al. Although they stated in their study that applying more than one session of ESWT treatment(12) would be better, previous studies showed that one session of ESWT can reduce spasticity immediately after the treatment session, and this effect persists weakly. We used four session of ESWT treatment. Is not enough information about which ESWT wave is superior over the treatment of spasticity. It is not known whether radial shockwave therapy is better or focused shockwave therapy is better.

It is considered better to use a radial shockwave therapy for the treatment of spasticity, as radial shockwave therapy can affect a larger muscle area compared to focal shockwave therapy, which affects a smaller area of the spastic muscle (14). Our study is in agreement with previous studies. Park et al. stated in their study that ESWT treatment was effective in reducing spasticity in the wrist flexors (109). Yan et al. also showed in their study that there was a decrease in spasticity after ESWT (110). Radinmehr et al., a session of radial found that extracorporeal shock wave therapy reduced spasticity scores (111). Wu et al. At 4 weeks following treatment, ESWT was not inferior to BoNT-A in reducing MAS scores of the wrist

flexors. During the research period, both therapies produced comparable reductions in spasticity of the wrist and elbow flexors (112). Kamaluddin et al. demonstrated a trend toward improvement from spasticity prior to treatment, confirming that ESWT for spasticity of the upper limbs in chronic stroke patients has an immediate effect; however, the treatment effect did not persist after one week or four weeks. Also, the musculotendinous junction benefited more from therapy than the muscular belly did.

No association could be seen between the treatment's effectiveness and patient characteristics including age, baseline spasticity severity, or disease duration. More research into the treatment's features and other factors affecting its efficacy is necessary to improve ESWT's efficacy as a treatment for spasticity (113). ESWT therapy on spasticity is the physical effect of negative and positive phase forming waves. The positive phase is when there is direct mechanical compression on the tissue and the negative phase is when there is cavitation that explodes at high velocities and creates the second shock wave during a shock (76). Mariotto S. et al. ESWT has been recommended as an effective therapy for lowering stiffness in the upper limbs of stroke patients.

Among the potential processes responsible for this impact are the influence on nitric oxide generation, the change of spinal cord excitability, and the reduction in muscle fibrosis. ESWT can stimulate nitric oxide generation, which is essential for neurotransmission and synaptic plasticity in the central nervous system, modify interleukin release, and regulate inflammation and growth factor activation in spastic muscle. Based on research, this physical action can lessen muscle and connective tissue stiffness through altering fibrosis tissue (114). Another possible mechanism to reduce spasticity is Nitric Oxide (NO) secretion (NO) stimulates neuromuscular junction formation and its effect on the peripheral nervous system. NO also affects the physiological functions of the central nervous system, e.g. at the synapse. causing plasticity and acting on mono transmission. Excitability by direct continuous or intermittent pressure on the tendon or muscle at the neuromuscular junction and the Golgi causes a decrease in the action potential in tendon organ function (115). Another mechanism of reducing spasticity with ESWT is the change in neuromuscular transmission, such as acetylcholine (14). Acetylcholine is the neurotransmitter substance that causes blood vessel dilation, increased body secretion and smooth muscle contraction.

Guo J et al. This study was to examine the feasibility and potential efficacy of Mirror Therapy (MT) in conjunction with ESWT for the treatment of upper limb spasticity in

poststroke patients. MT combined with ESWT resulted in higher improvement in upper extremity motor performance and a considerable reduction in spasticity, and the effects lasted at least one year longer than those of MT or ESWT alone (116). Dymarek et al. demonstrated that a single session of rESWT resulted to significant improvements in spasticity and muscle tone as measured by electrophysiological studies (117).

In Fugl-Meyer Assessment-Upper Extremity (FMA-UE) There are few studies examining the effect of ESWT treatment on upper extremity function. Improvement in upper extremity function is critical for stroke patients. Fugl was used to evaluate upper extremity function in previous studies using ESWT in stroke patients. In our study, FMA test was used to evaluate upper extremity functions. Simpson et al. showed that the FMA score did not have sufficient sensitivity to show differences and changes after treatment. Troncati et al. reported that upper extremity function as assessed by FMA scores improved significantly after one session of ESWT treatment in post-stroke patients (118). Li T-Y et al. showed a significant improvement in upper extremity function as assessed by FMA scores with 3 sessions of ESWT in their study, and they stated that ESWT may be effective in reducing hand and wrist spasticity with increased wrist control and hand function in patients with chronic stroke, suggested that BONT-A and ESWT can improve the impairment of upper limb motor ability measured using the (FMA-UE) (12). Wu et al. reported that using ESWT to treat spasticity after stroke in upper extremity found reduction in the spasticity of the wrist and elbow flexors during the study period and suggested that ESWT yielded greater improvement in the PROM of the wrist and elbow joints and in the FMA-UE score. In our study, it was determined that upper extremity function tests improved after four sessions of treatment(112). Compared to the literature, the fact that the tests evaluating the upper extremity function were similar in our study makes our study valuable. Possible mechanism of improving upper extremity function may be due to the reduction of spasticity and pain. Post-stroke patients experience spasticity, pain, and muscle weakness on the hemiplegic side, which impairs coordinated and efficient movement patterns. Reducing spasticity can also increase range of motion and eventually improve upper extremity function (56). In our study, it was shown that there was no significant difference in improving function between the with ESWT and without ESWT groups. Since there was no significant difference between the two groups in reducing spasticity and pain, the decrease in pain and spasticity was associated with an increase in upper extremity function, which seems to be an expected result from our study.

The concept of stability was first developed by Kabat and Knott in the late 1940s and was used as a proprioceptive attributed to neuromuscular function. Core stability is the combination of passive subsystem (ligament etc.), active subsystem (muscles) and neural subsystem (corticospinal) (47).This is the proximal stability is important for better functioning of the hand and activities of daily living. The lack of proximal stabilization may limit patients' ability to exert maximum effort (119). One of the important muscles in shoulder stability is the biceps brachii, due to this muscle attachment, the upper extremity can stabilize the proximal part. Decreased spasticity biceps better performance of brachii and upper extremity. It leads to better stabilization of the proximal part and ultimately to improvement in functional tests. Somatosensory impairment impairs control of movements and upper extremity function, and may also impair selective and goal-directed movements. Correct sensory input and sensory function are essential for good motor performance. Impairment in sensory input and processing can disrupt the relationship between the patient and the environment. In the presence of sensory disorders, the quality of upper extremity movements also deteriorates. Kamaluddin K. et al. investigated the effect of RSWT as an additional therapy in chronic stroke patients getting infrared therapy and stretching exercises. Study results were as follows: first, both wrist and hand FMA scores in the experimental and control group after intervention were increased significantly; second, difference of increased wrist and hand FMA scores in the experimental group after intervention were more significant when compared with difference of increased wrist and hand FMA scores in the control group over six weeks (120).

In this study, the Active Range of Motion (AROM) score showed we did not find any clear improvement between the group of patients who were treated with ESWT and with patients who used a conventional physiotherapy without ESWT in all joints that we examined during this study(shoulder, elbow and wrist joint). To our best knowledge of literature, there are no previous studies that measured the AROM of the wrist joint.

This study has some limitations. First, the small sample size may limit the generalizability of the study's results. Second, we measured motor improvement using FMA and MAS but did not examine the Brunnstrom stages of motor recovery. Therefore, additional research is required to investigate the long-term therapeutic advantages of ESWT for the motor recovery of upper limb spasticity following stroke.

6. CONCLUSIONS

1- Our findings indicate that ESWT is a supportive and adjunctive addition to conventional physiotherapy application for the treatment of upper limb spasticity in chronic stroke patients.

2- The group of patients who got both conventional physical therapy and shock waves showed an improvement in their ability to function, spasticity is reduced more in patients who received ESWT and conventional physiotherapy than in patients who received only conventional physiotherapy.

3- The FMA score was significantly improved in patients who submitted for the ESWT intervention.

4- ESWT were simple to use and apply, so they could be combined with conventional physiotherapy to treat spasticity in stroke patients.

5- The MAS score was significantly reduced in both groups, but with a greater percentage of patients who received traditional physiotherapy and shock waves.

6- ESWT has no effect on spasticity and upper extremity functionality in stroke patients in short term. Improvements in the ESWT group are due to conventional physiotherapy.

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APPENDIXES

Appendix 1. Ethics Committee Approval

SAGLIK BAKANLIGI

WASET SAĞLIK İDARESİ

İNSAN VE EĞİTİM GELİŞİM DAİRESİ BAŞKANLIĞI

BİLGİ VE ARAŞTIRMA DAİRESİ BAŞKANLIĞI

ARAŞTIRMA KURUL KARARI

SAYI NO:327

KARAR TARİHİ:06.07.2022

KONU: **ARAŞTIRMA KURUL KARARI**

MERKEZ TIP İDARESİNDEN SELAMLAR

ELWASET SAĞLIK BAKALIĞI ARAŞTIRMA KOMİSYONUNDA EĞİTİM GÖRDÜM/EGİTİM VE İNSANİ GELİŞİM MERKEZİ/ARAŞTIRMA VE BİLİM MERKEZİ BÖLÜMÜ/ARAŞTIRMA TEZ PROJESİ.
TARİH İSE :06.07.2022 NUMARASI(322/2022 MİLADİ)
TEZİ VE ARAŞTIRMA ALANI(ŞOK DALGASI TEDAVİSİNİN VÜCUT DIŞINDAKİ ETKİSİ-**ESWT**-İNME VE BEYİN KRİZİ HASTALARINDA FELÇ SPASTİSİTESİ VE ÜST EKSTREMİTE FONKSİYONLARI).

ARAŞTIRMACI TARAFINDAN SUNULAN ARAŞTIRMA PROJESİ İNCELENDİ SAYIN(**SALAM KHLAIF JABER**)TÜRKİYE CUMHURİYETİNDE BULUNAN AHİ EVRAN ÜNİVERSİTESİ YÜKSEK LİSAN ÖGRENCİMİZİN SUNULAN DÖNEM TEZİDİR.

KURULUN KARARI:

BU ARAŞTIRMA TEZ PROJESİ KURULUMUZ TARAFINDAN KABUL GÖREREK SAĞLIK BAKANLIĞI TARAFINDAN ONAYLANDI.BU TEZİN ARAŞTIRMASINDA VE UYGULAMASINDA BİR ENGEL BULUNMAMAKTADIR.EKLER/DEĞİŞİKLİKLER/ONARIM VE ARAŞTIRMA KOMİSYON NOTLARI/YOKTUR.

ARAŞTIRMA BİLİMSEL KOŞULLARI KARŞILAR VE BİLİMSEL ARAŞTIRMA ETİGİNE UYGUNDUR.BİZE GÖRE ARAŞTIRMA YAPMASINDA ENGEL YOKTUR

ARAŞTIRMA KURUL BAŞKANI

EĞİTİM UZMANI/ MECİD HEVİR HALEF

İMZA- 08.07.2022 ARKA SAHİFE

ELWASET SAĞLIK BAKALIĞI ARAŞTIRMA KOMİSYONUNDA EĞİTİM GÖRDÜM/EGİTİM VE İNSANİ GELİŞİM MERKEZİ/ARAŞTIRMA VE BİLİM MERKEZİ BÖLÜMÜ/ARAŞTIRMA TEZ PROJESİ.TARİH İSE :06.07.2022 NUMARASI(322/2022 MİLADİ)ARAŞTIRMACI TARAFINDAN SUNULAN ARAŞTIRMA PROJESİ İNCELENDİ SAYIN(**SALAM KHLAIF JABER**)TÜRKİYE CUMHURİYETİNDE BULUNAN AHİ EVRAN ÜNİVERSİTESİ YÜKSEK LİSAN ÖGRENCİMİZİN SUNULAN DÖNEM TEZİDİR.KARAR KURULU İSİMLERİ:

kurul başkanı	üye	üye	üye
Dr.sadun muhsin hasan	Dr.abdulrezzag tüffah	haydar kerim katıa	Dr.ali hüseyin ali
imza	branş doktoru	biyokimya doktoru	baycezini doktoru kanun

dktoru üye:eczaneci esad nasif jasım üye:branş mühendisi ahmed abdülabbas kemmaz

İŞ BU FOTOKOPİ BELGE ARAPÇADAN TÜRKÇEYE TARAFIMDAN TERCÜME EDİLMİŞTİR.

Ömer ASLAN
Arapça Yeminli Tercüman
Tel : 0533 515 66 72

استمارة رقم/ :
رقم القرار/ ٢٢٢
تاريخ القرار/ ١٤/١٢/٢٠٢١

وزارة الصحة
دائرة صحة واسط
مركز التدريب والتنمية البشرية
شعبة ادارة المعرفة والبحوث
لجنة البحوث

م/قرار لجنة البحوث

درست لجنة البحوث في دائرة صحة واسط/ مركز التدريب والتنمية البشرية المشكلة" بموجب الأمر الأداري ذي العدد١٣٤٧٩ بتاريخ ٢٠٢١/١٢/١٤
البحث المقدم من قبل الباحث/ طالب الماجستير (سلام خليف جابر)
في جامعة (أهي أفران في تركيا)

قرار اللجنة:

رئيس اللجنة
الدكتور
سعدون محيسن حسن
طبيب اختصاص

عضوا
الدكتور
جلال عبد الرزاق تفاح
دكتوراه احياء مجهرية

عضوا
الدكتور
حيدر كريم كاطع
دكتوراه بايو جزيئي

عضوا
الدكتور
علي حسين علي
دكتوراه قانون

عضوا
الصيدلاني الاختصاص
اسعد نصيف جاسم
ماجستير صيدلة

عضوا
مهندس اكتصاص
أحمد عبد العباس كماز
ماجستير

عضوا
كيميائية
رشا كامل عبد الكاظم
مقرر اللجنة

الدكتور
علي حسين علي التميمي
مستشار قانوني مساعد

وزارة الصحة
دائرة صحة واسط
مركز التدريب والتنمية البشرية
شعبة أدارة المعرفة والبحوث
العدد // ٢٢٢
التاريخ ٧/٧/٢٠٢٢

جمهورية العراق

Republic of Iraq
Ministry of Health
Directorate Wasit of Health
Training and developing center

(محمد نبينا اقام دولة العدل والتسامح)

الموضوع // قرار لجنة البحوث

يهديكم مركزنا أطيب التحيات ...

درست لجنة البحوث في دائرة صحة واسط /مركز التدريب والتنمية البشرية/ شعبة إدارة المعرفة والبحوث/ مشروع البحث
بتاريخ ٦/ ٢٠٢٢/٧ ذي الرقم (٢٠٢٢/٣٢٢م)
والمعنون(تأثير العلاج بموجات الصدمة خارج الجسم –ESWT- على التشنج ووظائف الأطراف العلوية في مرضى السكتة الدماغية)
والمقدم من قبل الباحث (سلام خليف جابر) طالب الدراسات العليا/ ماجستير/ في جامعة آهي أفران/ الجمهورية التركية

قرار اللجنة:-
قبول مشروع البحث أعلاه كونه مستوفياً للمعايير المعتمدة في وزارة الصحة والخاصة بتنفيذ البحوث ومدونة أخلاقيات البحوث ولا مانع من تنفيذ البحث

للتفضل بالاطلاع.....مع الاحترام.

خبير التدريب
مجيد هوير خلف
مدير مركز التدريب والتنمية البشرية
٢٠٢٢/٧/ ٨

نسخة منه الى:-
- مكتب المدير العام / للتفضل بالاطلاع ..... مع الاحترام.
- مركز التدريب والتنمية البشرية مع الأوليات كافة
- التوثيق (اضبارة البحوث)

الكيمياوي:-رشا كامل

البريد الالكتروني لمركز التدريب والتنمية البشرية
مركز التدريب والتنمية البشرية / بناية مركز طيبة النموذجي التدريبي / الطابق الثاني

وزارة الصحة
دائرة صحة واسط
مركز التدريب والتنمية البشرية
شعبة أدارة المعرفة والبحوث
العدد //ع ٧\
التاريخ /ح/٣/٢٠٢٢

جمهورية العراق

Republic of Iraq
Ministry of Health
Directorate Wasit of Health
Training and developing center

(محمد نبينا اقام دولة العدل والتسامح)

إلى // المؤسسات الصحية كافة
الموضوع // تسهيل مهمة

يهديكم مركزنا أطيب التحيات ...

أشارة إلى الطلب الخطي المقدم الى السيد مدير عام دائرة صحة واسط بتاريخ ٢٠٢٢/٣/٢٢ للتفضل بتسهيل مهمة طالب الدراسات العليا/ الماجستير (سلام خليف جابر) طالب في جامعة كير شهير في الجمهورية التركية ماجستير العلاج الطبيعي لغرض جمع العينات الخاصة ببحثه

للتفضل بالاطلاع.....مع الاحترام.

وزارة الصحة العراقية
Iraqi Ministry of Health
Founded 1920 تأسست

خبير التدريب
مجيد هوير خلف
مدير مركز التدريب والتنمية البشرية
٢٠٢٢/٣/ح

نسخة منه إلى :-
- مكتب المدير العام / للتفضل بالاطلاع ..... مع الاحترام.
- مركز التدريب والتنمية البشرية مع الأوليات كافة
- التوثيق (اضبارة البحوث)

الكيمياوي:-رشا كامل

البريد لالكتروني لمركز التدريب والتنمية البشرية
مركز التدريب والتنمية البشرية / بناية مركز طيبة النموذجي التدريبي / الطابق الثاني

Appendix 2. Fugl-Meyer Assessment Upper Extremity (FMA-UE)

FUGL-MEYER ASSESSMENT **ID:**
UPPER EXTREMITY (FMA-UE) **Date:**
Assessment of sensorimotor function **Examiner:**

Fugl-Meyer AR, Jaasko L, Leyman I, Olsson S, Steglind S: The post-stroke hemiplegic patient. A method for evaluation of physical performance. Scand J Rehabil Med 1975, 7:13-31.

A. UPPER EXTREMITY, sitting position					
I. Reflex activity			**none**	**can be elicited**	
Flexors: biceps and finger flexors (at least one)			0	2	
Extensors: triceps			0	2	
Subtotal I (max 4)					
II. Volitional movement within synergies, without gravitational help			**none**	**partial**	**full**
Flexor synergy: Hand from contralateral knee to ipsilateral ear. From extensor synergy (shoulder adduction/ internal rotation, elbow extension, forearm pronation) to flexor synergy (shoulder abduction/ external rotation, elbow flexion, forearm supination). **Extensor synergy:** Hand from ipsilateral ear to the contralateral knee	Shoulder	retraction	0	1	2
		elevation	0	1	2
		abduction (90°)	0	1	2
		external rotation	0	1	2
	Elbow	flexion	0	1	2
	Forearm	supination	0	1	2
	Shoulder	adduction/internal rotation	0	1	2
	Elbow	extension	0	1	2
	Forearm	pronation	0	1	2
Subtotal II (max 18)					
III. Volitional movement mixing synergies, without compensation			**none**	**partial**	**full**
Hand to lumbar spine hand on lap	cannot perform or hand in front of ant-sup iliac spine		0		
	hand behind ant-sup iliac spine (without compensation)			1	
	hand to lumbar spine (without compensation)				2
Shoulder flexion 0°- 90° elbow at 0° pronation-supination 0°	immediate abduction or elbow flexion		0		
	abduction or elbow flexion during movement			1	
	flexion 90°, no shoulder abduction or elbow flexion				2
Pronation-supination elbow at 90° shoulder at 0°	no pronation/supination, starting position impossible		0		
	limited pronation/supination, maintains starting position			1	
	full pronation/supination, maintains starting position				2
Subtotal III (max 6)					
IV. Volitional movement with little or no synergy			**none**	**partial**	**full**
Shoulder abduction 0 - 90° elbow at 0° forearm pronated	immediate supination or elbow flexion		0		
	supination or elbow flexion during movement			1	
	abduction 90°, maintains extension and pronation				2
Shoulder flexion 90° - 180° elbow at 0° pronation-supination 0°	immediate abduction or elbow flexion		0		
	abduction or elbow flexion during movement			1	
	flexion 180°, no shoulder abduction or elbow flexion				2
Pronation/supination elbow at 0° shoulder at 30°- 90° flexion	no pronation/supination, starting position impossible		0		
	limited pronation/supination, maintains start position			1	
	full pronation/supination, maintains starting position				2
Subtotal IV (max 6)					
V. Normal reflex activity assessed only if full score of 6 points is achieved in part IV; compare with the unaffected side			**0 (IV), hyper**	**lively**	**normal**
biceps, triceps, finger flexors	2 of 3 reflexes markedly hyperactive or 0 points in part IV		0		
	1 reflex markedly hyperactive or at least 2 reflexes lively			1	
	maximum of 1 reflex lively, none hyperactive				2
Subtotal V (max 2)					
Total A (max 36)					

Approved by Fugl-Meyer AR 2010 1 Updated 2015-03-11

B. WRIST support may be provided at the elbow to take or hold the starting position, no support at wrist, check the passive range of motion prior testing		none	partial	full
Stability at 15° dorsiflexion elbow at 90°, forearm pronated shoulder at 0°	less than 15° active dorsiflexion dorsiflexion 15°, no resistance tolerated maintains dorsiflexion against resistance	0	1	2
Repeated dorsifexion / volar flexion elbow at 90°, forearm pronated shoulder at 0°, slight finger flexion	cannot perform volitionally limited active range of motion full active range of motion, smoothly	0	1	2
Stability at 15° dorsiflexion elbow at 0°, forearm pronated slight shoulder flexion/abduction	less than 15° active dorsiflexion dorsiflexion 15°, no resistance tolerated maintains dorsiflexion against resistance	0	1	2
Repeated dorsifexion / volar flexion elbow at 0°, forearm pronated slight shoulder flexion/abduction	cannot perform volitionally limited active range of motion full active range of motion, smoothly	0	1	2
Circumduction elbow at 90°, forearm pronated shoulder at 0°	cannot perform volitionally jerky movement or incomplete complete and smooth circumduction	0	1	2
	Total B (max 10)			

C. HAND support may be provided at the elbow to keep 90° flexion, no support at the wrist, compare with unaffected hand, the objects are interposed, active grasp		none	partial	full
Mass flexion from full active or passive extension		0	1	2
Mass extension from full active or passive flexion		0	1	2
GRASP				
a. Hook grasp flexion in PIP and DIP (digits II-V), extension in MCP II-V	cannot be performed can hold position but weak maintains position against resistance	0	1	2
b. Thumb adduction 1-st CMC, MCP, IP at 0°, scrap of paper between thumb and 2-nd MCP joint	cannot be performed can hold paper but not against tug can hold paper against a tug	0	1	2
c. Pincer grasp, opposition pulpa of the thumb against the pulpa of 2-nd finger, pencil, tug upward	cannot be performed can hold pencil but not against tug can hold pencil against a tug	0	1	2
d. Cylinder grasp cylinder shaped object (small can) tug upward, opposition of thumb and fingers	cannot be performed can hold cylinder but not against tug can hold cylinder against a tug	0	1	2
e. Spherical grasp fingers in abduction/flexion, thumb opposed, tennis ball, tug away	cannot be performed can hold ball but not against tug can hold ball against a tug	0	1	2
	Total C (max 14)			

D. COORDINATION/SPEED, sitting, after one trial with both arms, eyes closed, tip of the index finger from knee to nose, 5 times as fast as possible		marked	slight	none
Tremor	at least 1 completed movement	0	1	2
Dysmetria at least 1 completed movement	pronounced or unsystematic slight and systematic no dysmetria	0	1	2
		≥ 6s	**2 - 5s**	**< 2s**
Time start and end with the hand on the knee	at least 6 seconds slower than unaffected side 2-5 seconds slower than unaffected side less than 2 seconds difference	0	1	2
	Total D (max 6)			

			TOTAL A-D (max 66)	
H. SENSATION, upper extremity eyes closed, compared with the unaffected side		**anesthesia**	**hypoesthesia or dysesthesia**	**normal**
Light touch	upper arm, forearm palmary surface of the hand	0 0	1 1	2 2
		less than 3/4 correct or absence	**3/4 correct or considerable difference**	**correct 100%, little or no difference**
Position small alterations in the position	shoulder elbow wrist thumb (IP-joint)	0 0 0 0	1 1 1 1	2 2 2 2
			Total H (max12)	

J. PASSIVE JOINT MOTION, upper extremity, sitting position, compare with the unaffected side				**J. JOINT PAIN** during passive motion, upper extremity		
	only few degrees (less than 10° in shoulder)	decreased	normal	pronounced pain during movement or very marked pain at the end of the movement	some pain	no pain
Shoulder						
Flexion (0° - 180°)	0	1	2	0	1	2
Abduction (0°-90°)	0	1	2	0	1	2
External rotation	0	1	2	0	1	2
Internal rotation	0	1	2	0	1	2
Elbow						
Flexion	0	1	2	0	1	2
Extension	0	1	2	0	1	2
Forearm						
Pronation	0	1	2	0	1	2
Supination	0	1	2	0	1	2
Wrist						
Flexion	0	1	2	0	1	2
Extension	0	1	2	0	1	2
Fingers						
Flexion	0	1	2	0	1	2
Extension	0	1	2	0	1	2
Total (max 24)				**Total** (max 24)		

A. UPPER EXTREMITY	/36
B. WRIST	/10
C. HAND	/14
D. COORDINATION / SPEED	/ 6
TOTAL A-D (motor function)	/66

H. SENSATION	/12
J. PASSIVE JOINT MOTION	/24
J. JOINT PAIN	/24

Appendix 3. Modified Ashworth Scale Instructions

General Information (derived Bohannon and Smith, 1987):

- Place the patient in a supine position
- If testing a muscle that primarily flexes a joint, place the joint in a maximally flexed position and move to a position of maximal extension over one second (count "one thousand one")
- If testing a muscle that primarily extends a joint, place the joint in a maximally extended position and move to a position of maximal flexion over one second (count "one thousand one")
- Score based on the classification below

Scoring (taken from Bohannon and Smith, 1987):

0 No increase in muscle tone

1 Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension

1+ Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM

2 More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved

3 Considerable increase in muscle tone, passive movement difficult

4 Affected part(s) rigid in flexion or extension

Patient Instructions:

The patient should be instructed to relax

Name: Date:,

Muscle Tested _____ Score

1

2

3

4

5

Reference for test instructions:

Bohannon, R. and Smith, M. (1987). "Interrater reliability of a modified Ashworth scale of muscle spasticity." Physical Therapy 67(2): 206.

RESUME

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