



1986

A comparative study of positive versus negative polarity in the treatment of acute ankle sprains utilizing high voltage electrogalvanic stimulation

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A Comparative Study of Positive Versus Negative Polarity
in the Treatment of Acute Ankle Sprains
Utilizing High Voltage Electrogalvanic Stimulation

A Thesis
Presented to
the Graduate Faculty
of the
University of the Pacific

In Partial Fulfillment
of the Requirements for the Degree
Master of Science

by
Lauren Michelle Wells
January 1986

This thesis, written and submitted by

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ACKNOWLEDGEMENTS

I would like to thank Stockton Orthopaedic Medical Group for referring patients for treatment. Without their support, proper medical care and follow-up, the study would not have been possible. Also, I would like to thank Gerry Solberg for his assistance in developing the treatment protocol. My thesis committee has also been helpful in completing this study. In particular, I would like to thank Dr. Funkhouser for her support and understanding of sometimes difficult situations and circumstances. Last, but not least, I would like to thank my parents for the opportunity and encouragement necessary for me to complete this degree.

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Introduction

Electrical stimulation has long been used in the treatment of a variety of ailments. Its current uses range from muscle re-education and orthotic substitute to scoliosis management and edema control. I chose to study the effect of electrode polarity in high voltage electrogalvanic stimulation in the treatment of edema for several reasons. I had access to subjects because I was the only physical therapist at the Stockton Orthopedic Medical Group. High voltage electrogalvanic stimulation is a commonly used modality in treating edema (Brown, 1981). High voltage generators have a polarity switch, and the direction manual which accompanies the Electro-Med generator used by the Stockton Orthopedic Medical Group states that the negative pole should be used for edema reduction. (Instruction manual for high voltage Electrogalvanic Stimulator, 1977). However, the effect of electrode polarity on edema reduction has not been demonstrated, nor documented in the literature.

In clinical practice electrical stimulation can be provided by a variety of generators. Low voltage stimulation (using less than 150 volts) has been used for many years and the effects are well understood (Osborne and Holmquest, 1944). More recently, high voltage electro-

galvanic stimulation (using greater than 150 volts) has been introduced for clinical use (Instruction Manual for High Voltage Electrogalvanic Stimulator, 1977).

The major effects of low voltage electrical current include:

1) Iontophoresis, which is electrode and cellular polarization due to ion transfer. Clinically this technique can be used to drive compounds through the skin.

2) Electrophoresis, which is the movement of charged cells or particles. This technique is not used in the clinic; it can lead to caustic anion and cation accumulation at the electrode interface.

3) Electro-osmosis, which is a shift in cellular water content. This effect can be utilized in edema control.

4) A change in nerve sensitivity, which can be effective in controlling pain and spasm.

5) A mild heating effect in tissue between electrodes due to the conduction of current.

6) A slight increase in circulation due to the mild heating effect and the increase in O₂ and nutrient demand of contracting muscle (Wolf, 1981).

In most instances, the current form (AC or DC) as well as its magnitude and frequency determine the extent of these effects in low voltage electrical stimulation. The effects

of high voltage electrical stimulation are not as clear, but it is known that iontophoresis and electrophoresis do not occur, nor does temperature change (Killian, 1985).

Electro-osmosis may or may not occur. There does appear to be a change in nerve sensitivity and a slight increase in circulation resulting from high voltage electro-galvanic stimulation (Sohn, et al. 1982).

Treatment techniques can be either monopolar or bipolar. In monopolar treatment one electrode (positive or negative) is placed on the treated area and the other electrode is placed in a remote location. In bipolar treatment both the positive and negative electrodes are placed on the treated area and in relative proximity to each other (Killian, 1985).

The effects of current passed through tissues and the magnitude of the reaction depend on the tissue type, pulse duration, the type of waveform, current density through a specified area, and the frequency of the current used (Killian, 1985).

The perception of pain that many people anticipate prior to being stimulated is motivated by fear of high voltage generating a high average current of long pulse duration. Adjusting pulse duration and voltage so that there is a low average current and therefore a less painful

stimulus is the basis of both high voltage electrogalvanic stimulation (EGS) and low-voltage electrical stimulation (Benton, et al, 1981). If patients are to tolerate the treatment, they must not be stimulated with a higher amplitude (higher current) than their pain threshold. Therefore, high voltage electrogalvanic stimulation uses a short pulse duration (measured in micro-seconds) and high voltages to deliver an average current at non-painful levels; low voltage uses a longer pulse duration and lower voltages to deliver an average current at non-painful levels. The pulse duration on a high voltage stimulator range from 50-75 microseconds and may be delivered at 20-80 pps, while low voltage pulse durations last 300 microseconds and are usually delivered at a frequency of 35 pps (Killian, 1985; Benton, et al, 1981).

The waveform of the pulse is also important in how stimulation is perceived and how it functions in treatment. Most low voltage generators are battery operated and convert continuous direct current into rectangular pulsed direct current. The high voltage generator used in this study changes 120 volt, 60 cycle household current into dual spiked pulsed direct current. The interval between the pulses is much longer than the duration of the pulse. The current is off more than it is on. This allows for the low

average current (approximately 1.5 milliamps in typical treatment) delivered by high voltage generators despite peak currents between 300-400 milliamperes (Killian, 1985).

The short pulse duration of high voltage direct current stimulation permits selective stimulation of predominantly sensory and motor axons and much less stimulation of pain conducting fibers. Consequently, such stimulation is more tolerable.

The short pulse duration does not seem to cause the chemical and thermal effects seen with low volt, long pulse duration direct current stimulation. Therefore, high voltage electrogalvanic stimulation may be used for extended treatment periods with no apparent side effects. Because pulse duration cannot be shortened, any increase in pulse frequency rate will increase average current. Such an increase will cause a stronger contraction of the stimulated muscle and will increase the patient's perception of the current, but it will not increase the depth of penetration of the current.

However, increasing the amplitude of the peak current will increase the depth of penetration of current into tissues. Some electrogalvanic stimulators have maximum peak currents of approximately 300-400 milliamperes, compared to the 80 milliampere current of low voltage stimulators.

Even though the peak currents in electrogalvanic stimulation are 4-5 times that of low voltage electrical stimulation, they are safe because the average current is low (Smith, 1981). Because of the greater penetration of current with electrogalvanic stimulation, deep nerves and muscles can be stimulated effectively. As a result, muscle contraction is effective and can be achieved even without placement of the electrode on a nerve or muscle motor point.

The deep penetration obtained from high voltage electrogalvanic stimulation is not limited to peripheral nerves and skeletal muscles. It may be possible to stimulate sympathetic nerves and the smooth muscles of blood vessels directly or indirectly (Alon, 1981).

The current physiological theory concerning pain reduction through electrical stimulation is that stimulation can release endorphins and enkephalins from several sites in the central nervous system (Killian, 1985). However, the supposed release of endorphin/enkephalins is theoretical. This has not been demonstrated in humans, and the mechanism by which electrical stimulation of a peripheral body part can stimulate release of a substance from the central nervous system is not clear at present. Two different methods are presumed to achieve endorphin and enkephalin release (Francis, 1983).

One method is to use a supra-painful stimulus to a very narrow area at a trigger point, acupuncture point, or motor point. Trigger, acupuncture and motor points are often the same points, but they are defined differently. A trigger point is one where pain, numbness or tingling radiate from the point while being palpated. The name trigger describes the use of the index finger to press the point and "trigger" the radiation. An acupuncture point lies along the meridians of the body (Tappan, 1978). A motor point is where a nerve to a muscle lies close to the skin (Brobeck, 1979). The Western use of acupuncture points has been used for many years without realizing it, because many trigger, acupuncture and motor points are the same (Paris, et al, 1983).

The other method is to deliver non-painful stimuli to larger areas (usually to a painful area where a trigger point cannot be isolated, or to an injured area) in which pain fiber stimulation is minimized and sensory fiber activation is maximized. High voltage electrogalvanic stimulation can be used to deliver either a supra-painful stimulus or a non-painful stimulus (Killian, 1985).

By using larger electrodes, controlling intensity, and having very short pulse durations, selective non-painful sensory fiber stimulation can prevail. Both treatment methods appear to function equally well in most patients (Killian, 1985).

The mechanism by which high voltage electrical stimulation functions physiologically in increasing joint mobility is unclear. The use of long pulse duration, low voltage direct current is known to cause softening of some tissues under the negative electrode due to "liquification" of the protein (Osborne & Holmquest, 1944, p. 38). This softening is attributed to an increase in alkalinity due to the chemical effect of the stimulation (Alon, 1981). What tissues are affected by the current depends on the depth of penetration and tissue type. Current will flow more easily through substances with low impedance than through substances with high impedance. The relative conductivity of tissues is approximately equal to their content of water and available ions. Skin and bone are not softened by electrical stimulation because of their high impedance (5-16% water and ion content compared to 75% water and ion in muscle) (Benton, et al, 1981). In high voltage electrogalvanic stimulation such effects are practically non-existent, thus such a mechanism is unlikely to explain the result (Newton and Karselis, 1983).

With low voltage electrical stimulation peripheral circulation can be increased to the skin, muscles, core and any combination of these. High voltage pulsed direct current does not seem to increase the core circulation of normal subjects (Alon, 1981).

It may, however, have a electrical effect on blood vessels to increase blood circulation (clinical observation). When electrical stimulation is used to increase peripheral blood circulation the results that occur could be attributed to either a direct or an indirect effect on the circulatory system (Alon, 1981).

The direct effect could be direct activation of the sympathetic fibers of the autonomic nervous system. The indirect effect could be achieved by three different mechanisms. The first would be the circulatory response to the chemical changes of acid and alkaline under the positive and negative electrodes respectively. Such an effect would be unlikely to occur when high voltage pulsed direct current is used because the pulse characteristics eliminate the chemical changes under the electrodes (Newton and Karselis, 1983).

The second possible indirect effect is through the somatic sympathetic reflex. Such a response has been demonstrated on the sympathetics innervating a dog's kidneys. Whether such an influence affects the autonomic innervation of the peripheral arteries is still unclear (Alon, 1981).

The third possible indirect effect is from the pumping action of muscles. Effective venous return requires strong

intermittent contractions. High voltage pulsed direct current, because of its depth of penetration and relatively comfortable stimulation, can elicit such contractions (Alon, 1983).

Acute ankle sprains, strains and fractures develop post-traumatic edema which seriously limits recovery due to abnormal adhesion formation (Cyriax, 1978). The primary goals of treatment are to decrease pain and swelling and increase mobility. If range of motion limitation is associated with pain, then reduction of such pain should improve joint mobility (Nirschl and Sobel, 1981; Grava and Schelberg-Karness, 1983). Edema reduction would be a secondary gain because the patient would be more willing to move the affected part thus reducing adhesions.

Post-traumatic edema has traditionally been treated with compression, elevation and ice (Lehman, et al, 1974; Quillen and Rouillien, 1982). This treatment is currently being supplemented with high voltage electrogalvanic stimulation. The literature which accompanies the Electro Galvanic Stimulator (Electro-Med Health Industries, Miami, FL) used by the Stockton Orthopedic Medical Group suggests that negative polarity be used to treat edema. There is no literature available to verify that negative polarity is more effective than positive polarity.

The object of this study is to test the use of both positive and negative polarity of high voltage electrogalvanic stimulation in the treatment of edema in acute ankle sprains. The null hypothesis of this study is that polarity has no effect on edema reduction.

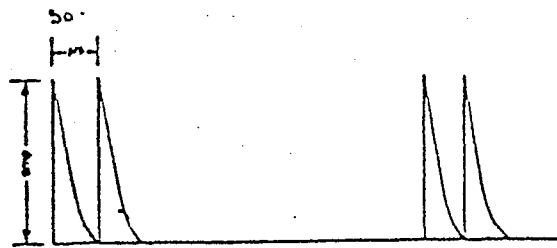


Figure 1.

(a) "High voltage" paired spikes are most frequently monophasic, resulting in ion flow in one direction. Because of their short duration, spikes are paired and required high amplitude to generate neural excitation (Benton, et al, 1981).

Materials and Methods

Patients participating in this study were seen initially by one of the physicians of the Stockton Orthopedic Medical Group, Stockton, California. These patients were then referred to Physical Therapy with a diagnosis of second degree ankle sprain. (In a second degree ankle sprain, the ligament is weakened, but there is no instability. Weight bearing is painful and range of motion is limited. Tenderness is marked over the anterior talofibular ligament. Swelling and ecchymosis are significant. X-rays are normal. Ice, elevation, taping and non-weight bearing are the immediate treatment (Floriani, 1976.)) All patients were treated according to the protocol shown below. The information from those who attended 75% of scheduled appointments was used as data in this study. Subjects whose information was included signed a release of information form after discharge.

Protocol:

Once a patient is referred to the clinic the stability of the ankle is re-evaluated according to the method of Hoppenfeld. The anterior talofibular ligament is checked for a positive drawer sign (when the talus slides anteriorly from under the cover of the ankle mortise). If the patient has a positive drawer sign, they are not a candidate for the

study. Only those patients with ecchymosis and swelling, without ligamentous instability may participate.

Once the stability of the ankle is verified, range of motion is evaluated. Dorsiflexion and plantarflexion are measured with a goniometer. (When the angle between the tibia and 5th metatarsal is at 90° the ankle is in neutral position and is re-defined as 0° for dorsiflexion-plantarflexion measurement. Normal dorsiflexion range is considered $0^\circ - 20^\circ$ and normal plantarflexion is considered $0^\circ - 50^\circ$. Many individuals do not have "normal" range of motion, (ROM), therefore the injured ankle is compared to the non-injured ankle (Esch & Lепley, 1974). The goal of treatment is equal ROM for both ankles.

Volumetric displacement measurements are taken to quantify the amount of edema. A plexiglass tank (30 x 45 x 15 cm) with a side spout was built for the study. The tank is filled to the base of the spout. The patient places their non-injured foot in the tank with the foot resting on the bottom. The water run-off is collected in a graduated cylinder and the volume of water is recorded. The same procedure is repeated with the injured ankle. The difference between the two measurements is the volume due to edema. The normal slight size or shape difference between left and right feet does not result in a significant volume difference (Paris, et al, 1983).

Following initial evaluation, treatment is begun. The patient begins by icing the injured ankle. When the ankle appears red the icing is stopped and patients begin the exercise program. The exercises consist of active plantarflexion and dorsiflexion with resistance added to patient tolerance, toe flexion and extension and toe abduction and adduction.

The ankle is then stretched by the patient with a towel, by the therapist, then again by the patient in a weight-bearing position.

Following stretching, patients are treated with high voltage electrogalvanic stimulation (Electro Galvanic Stimulator, Electro-Med Health Industries, Miami, FL). The dispersive pad (ground) is placed over the lower back and held in contact with an elastic belt. The toes are covered with a neoprene bootie and the injured ankle submerged in a bucket of cold water. The two active electrodes are submerged on the medial and lateral sides of the ankle (not in contact with the ankle) (Roy, 1983).

Treatment is delivered for 20 minutes. Switching rate is set on constant and the frequency at 80 pulses per second. The patient dorsiflexes and plantarflexes the foot during treatment. Ice cubes are added to the water 10 minutes into stimulation. Following stimulation, the ankle

is taped (Johnson & Johnson, 1983). The tape is to be kept on until the next clinic visit unless complications occur secondary to decreased circulation. Each patient is scheduled to be seen daily Monday through Friday.

Follow-up measurements for ROM and volume are taken, when possible during clinic visits.

The first group of patients were treated with negative pole, and the second group were treated with positive pole. During a year and a half only sixteen patients met the criteria of the study. The group treated with (-) polarity had an age range of 14 to 56 years, with 50% of the patients under 25. The group treated with (+) polarity had an age range of 18 to 45 years, with 25% under 25. Patient Cr, age 18, sprained both ankles at different times and was a subject in both groups.

In each group the younger patients (age 14-20) incurred their injuries in sports-related activities associated with competition for their high schools. They accounted for 6 of the 16 subjects participating in the study.

The other 10 patients sustained their injuries by tripping, and presented under a variety of circumstances: three (Gu, An and Do) were seen within 24 hours of injury, four (Sm, Fl, Ga, Br) were seen within 72 hours of injury, one (Bi) had circulatory insufficiency so could not ice or

exercise, one (Va) had been injured in New York, seen in the emergency room there and was not seen in the clinic until two weeks post-injury, and one (Th) sustained a fracture that required further follow-up.

Despite this difference in prior histories, all patients were begun on the same protocol.

Results

Range of motion (ROM) and volume data were analyzed for both groups. A total of 58 observations for ROM and volume were made for both groups. Thirty four observations were made on the negative group: 32 of these observations were made through the first 10 days, 1 at 20 days and 1 at 22 days. The other 24 observations were made on the positive group: 23 of these observations were made through the first 10 days and 1 at 14 days.

ROM versus time and volume versus time for both groups were considered separately and then together. ROM values were obtained by adding dorsiflexion and plantarflexion ranges together. The slope of the regression line for each subject was derived (Tables 1 and 2) then the average slopes for both groups were determined. The average slopes of both groups were compared using a t-test and found not to be significant at the 5% level.

Discussion

The ankle is one of the primary weight bearing joints. It must be both mobile and stable. Inversion or eversion sprains can tear the joint's supporting ligaments and produce instability. Excessive inversion stress is the most common cause of ankle sprains. There are two anatomical reasons for inversion ankle injuries: The medial malleolous is shorter than the lateral malleolous, and the talus can thus be forced to invert farther than it can evert and the ligamentous thickening on the lateral side of the joint are separate, and therefore not as strong as the massive deltoid ligament on the medial side (Hoppenfeld, 1976). The anterior talofibular ligament is the ligament most often involved in ankle sprains. The anterior talofibular and the calcaneofibular ligaments must both be torn to produce gross lateral ankle instability. All of the subjects participating in the study suffered from inversion sprains and Lu may also have had some stress applied to the deltoid ligament on the medial side. Th also had an associated fracture.

Each individual participating in the study suffered from various degrees of disability from their injury. Each had visible ecchymosis with swelling resulting in pain and stiffness, but the amount varied considerably between sub-

jects. The more severe the injury the more swelling present at the time of initial evaluation. The patient with the greatest amount of swelling was also the patient who suffered from an associated fracture.

The amount of post-traumatic edema is also influenced by the length of time between injury and initial treatment, the age of the individual and the number of complicating factors (i.e., fractures, tendon rupture, vascular compromise, other systemic disease). These factors acting in conjunction may lead to higher original volumes. For example, the second largest initial volume measured was that of a patient who had venous insufficiency and suffered from swollen ankles independent of trauma (Bi).

An acute injury is more likely to respond to treatment than a chronic injury because secondary damages caused by excessive bleeding, adhesion formation and weakness have not developed. The longer the tissues remain edematous the more stretched the tissues and associated vessels become. The accumulation of extracellular fluid is therefore greater in regions which are loose in texture and where the skin is readily stretched. In persons with firm resistant skin, edema makes its appearance later and is less pronounced. If edema can be prevented from forming and stretching the tissues, the initial volume is lower and there is less to

reduce. Those patients in the study who were seen immediately post-injury were those who participated in high school athletics (Cr, Mc, Ha, Lu, De, Cr), one seen in the emergency room (An) and two in the office (Gu, Do). In general younger patients have more elastic and resilient tissues than older patients.

There are some significant muscle changes that occur with aging that may affect an individual's recovery from injury. These include: "Gross muscular atrophy secondary to the loss of both number and size of muscle fibers" (Payton and Poland, 1983:43). This "muscular atrophy may be differentially attributed to a decrease in the number of red fibers" (McCarter, 1978:17). Muscle atrophy is always associated with weakness.

In the lower extremities, venous return is aided by the muscular pump. The stronger the contraction the more efficiently the muscular pump works to return blood to the heart (against gravity). When an individual becomes less active because of weakness they do not "pump" as effectively as an individual without weakness.

Weakness may also result from an increasing proportion of skeletal muscle free fat being replaced by fibrous tissue. Nevertheless, the primary cause of loss of strength appears to be a change in lifestyle and decreased use of the

neuromuscular system. Osteoarthrosis is a result of an imbalance between the stress a joint receives and the ability of the physiological shock absorbers in that joint to absorb the stress (Radin, 1976). This is accompanied by a marked decrease in tensile stiffness and fracture strength with increasing age. On the average, strength begins to decline around age 40 (Raven & Mitchel, 1980).

These musculoskeletal changes which occur with aging are accompanied by cardiovascular changes. Both the heart and the smooth muscle of vascular tissue decrease in sympathetic responsiveness in people of advanced age (Lakotta, 1980). Associated with the aging cardiovascular system is a decline in lipid catabolism, which generates lipid accumulation; this process may underlie the development of atherosclerosis (Kritchevsky, 1980).

These changes associated with aging coupled with the degree of injury account for some differences in recovery time and initial volumes. When these factors are considered in conjunction with the time between injury and first treatment, the test subjects are further divided into the following subgroups: young subjects with acute injuries; young subjects with chronic injuries; old subjects with acute injuries; old subjects with chronic injuries; patients with complicating factors. The sample size of each subgroup

is so small it is difficult to draw conclusions about the effect of the polarity of treatment.

The other major complicating factor is the amount of home follow-up. Each patient was instructed in a home exercise and icing program to be done three times per day. The amount of compliance cannot be verified. Each patient reported about their compliance level, which was frequently less than the amount prescribed. Standardizing and quantifying the amount of home follow-up is difficult. One patient may have overdone his home program in an attempt to get back to the football field faster, while another patient may have underdone her home program because the exercises may be painful and she does not tolerate pain well (by admission of patient). In essence, data from each patient can only be considered as an individual case study making it impossible to arrive at any significant conclusions from this study.

To help eliminate the variability which prevented conclusions from being drawn from this study, the following suggestions are offered.

It is essential to have (1) a large number of patients, (2) of similar age. The investigator should (3) adopt a set of standards related to the degree of injury (for example, decide on limits of talar-tilt), (4) obtain patients with

comparable injuries who are (5) seen no later than 24 hours post injury (6) before initial treatment.

One way to achieve most of these goals would be to perform the study out of an emergency room rather than from a private practice. The number of acute patients is greater during a given time period in an emergency room and they are less likely to have received previous treatment. They may then be referred to a physical therapist directly. A larger initial number of patients would facilitate selection for age and comparable degree of injury.

There is probably no reasonable way of insuring that patients keep their appointments and perform the home follow-up treatment and exercises. However, a large enough, initial sample might provide enough data to eliminate some of the individual differences.

Summary

The goal of this study was to determine whether one pole of high voltage electrical stimulation is more effective than the other in edema reduction of acute ankle sprains.

Patients were seen initially by a physician of the Stockton Orthopedic Medical Group and referred to Physical Therapy for treatment. Range of motion and volume of the affected ankle were measured during initial treatment, and follow-up measurements were taken until time of discharge. The first eight patients who attended 75% of their scheduled appointments were treated with negative pole and the second eight patients with positive pole.

Range of motion and volume data from each subject and from each group were analyzed by linear regression and t-tests. No significant difference was found between the range of motion and volume of individuals in the two treatments. The small sample size prohibits any conclusions being drawn regarding the relationship between polarity and edema reduction.

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Table 1. Ankle-foot volume and range of motion measurements of patients treated with negative polarity of electrogalvanic stimulation. Data presented as volume (ml), dorsiflexion-plantarflexion (degrees of movement).

| Patient/ diagnosis/ time post- injury | Sex/ age | TREATMENT DAYS | | | | | | | | | | Slope b | | |
|--|-------------|-------------------|-------------------|------------------|------------------|------------------|------------------|------------------|-----------------|------------------|------------------|-----------------------------|----------|-------|
| | | Initial | Day 2 | Day 3 | Day 4 | Day 5 | Day 6 | Day 7 | Day 8 | Day 9 | Day 10 | Volume | ROM | |
| Gu/acute 24 hours | M/17 | 180 ml 0-30° | | | | 100 ml 5°-30° | | 90 ml 10°-30° | | 20 ml 10°-35° | | | -18.71 | 1.8 |
| An/acute 24 hours | M/53 | 180 ml -3°-30° | 160 ml -3°-30° | 110 ml 0°-30° | | | | | 80 ml 5°-30° | 80 ml 5°-30° | | | -11.58 | 1.08 |
| Cr/acute 24 hours | M/18 | 80 ml 10°-45° | | | 80 ml 10°-40° | | 70 ml 10°-40° | | | | 50 ml 10°-45° | | - 3.51 | .058 |
| SM/acute 72 hours | M/27 | 80 ml 0°-30° | 60 ml 0°-30° | | | 0 ml 0°-45° | | 30 ml 0°-45° | | | | | - 4.5 | 2.967 |
| F1/acute 72 hours | M/43 | 220 ml -15°-30 | | | | | | 140 ml 0°-30° | | | 40 ml 0°-45° | | -19.05 | 3.214 |
| Mc/acute 24 hours | M/14 | 200 ml 0°-45° | | 140 ml 0°-45° | | | | 20 ml 0°-45° | | | | | -29.99 | 0 |
| Ha/acute 24 hours | M/18 | 60 ml 0°-45° | | 60 ml 0°-45° | | | | | | | 20 ml 0°-45° | | - 4.78 | 0 |
| Lu/acute 24 hours | M/18 | 140 ml 0°-30° | 140 ml 0°-30° | 120 ml 0°-30° | 90 ml 0°-35° | | | | 60 ml 0°-45° | 100 ml 0°-40° | | 20 days: 30 ml 0°-45° | - 5.34 | .8825 |
| | | | | | | | | | | | | 22 days: 20 ml 5°-45° | -12.1825 | 1.25 |

Table 2. Ankle-foot volume and range of motion measurements of patients treated with positive polarity of electrogalvanic stimulation. Data presented as volume (ml), dorsiflexion-plantarflexion (degrees of movement).

| Patient/ diagnosis/ time post- injury | Sex/ age | TREATMENT DAYS | | | | | | | | | | Slope b | | | |
|--|-------------|--------------------|-------------------|------------------|------------------|-----------------|------------------|------------------|-------|-------|--------|------------------|---------------------------|----------|--------|
| | | Initial | Day 2 | Day 3 | Day 4 | Day 5 | Day 6 | Day 7 | Day 8 | Day 9 | Day 10 | Volume | ROM | | |
| Ga/acute 72 hours | M/35 | 170 ml 0°-35° | | | | | 160 ml 0°-35° | 160 ml 0°-35° | | | | 100 ml 0°-40° | | - 6.9 | .476 |
| Bi/chronic vascular in- sufficiency | F/37 | 260 ml 0°-40° | | 100 ml 0°-45° | | | | 80 ml 0°-45° | | | | | 14 days 0 ml 0°-45° | - 16.50 | .2658 |
| Va/chronic 2 weeks | F/62 | 130 ml -5°-30° | 130 ml -5°-30° | 90 ml -5°-30° | 70 ml -5°-35° | 50 ml 0°-35° | | | | | | 80 ml 0°-40° | | - 5.93 | 1.8688 |
| Do/acute 24 hours | M/33 | 10 ml 5°-50° | | | | | | | | | | | | No Slope | |
| Th/acute fracture 24 hours | M/42 | 270 ml -15°-30° | | | 200 ml 0°-30° | | | | | | | 100 ml 0°-35° | | - 21.12 | 2.3468 |
| Br/acute 72 hours | F/28 | 160 ml 0°-30° | | | 80 ml 0°-35° | | | | | | | 40 ml 0°-45° | | - 14.28 | 1.8877 |
| De/acute 24 hours | M/18 | 60 ml 0°-40° | | | | 25 ml 0°-45° | | | | | | | | - 8.75 | 1.25 |
| Cr/acute 24 hours | M/18 | 60 ml 10°-45° | | | 50 ml 10°-45° | | | | | | | | | - 3.33 | 0 |
| | | | | | | | | | | | | | | - 10.97 | 1.564 |

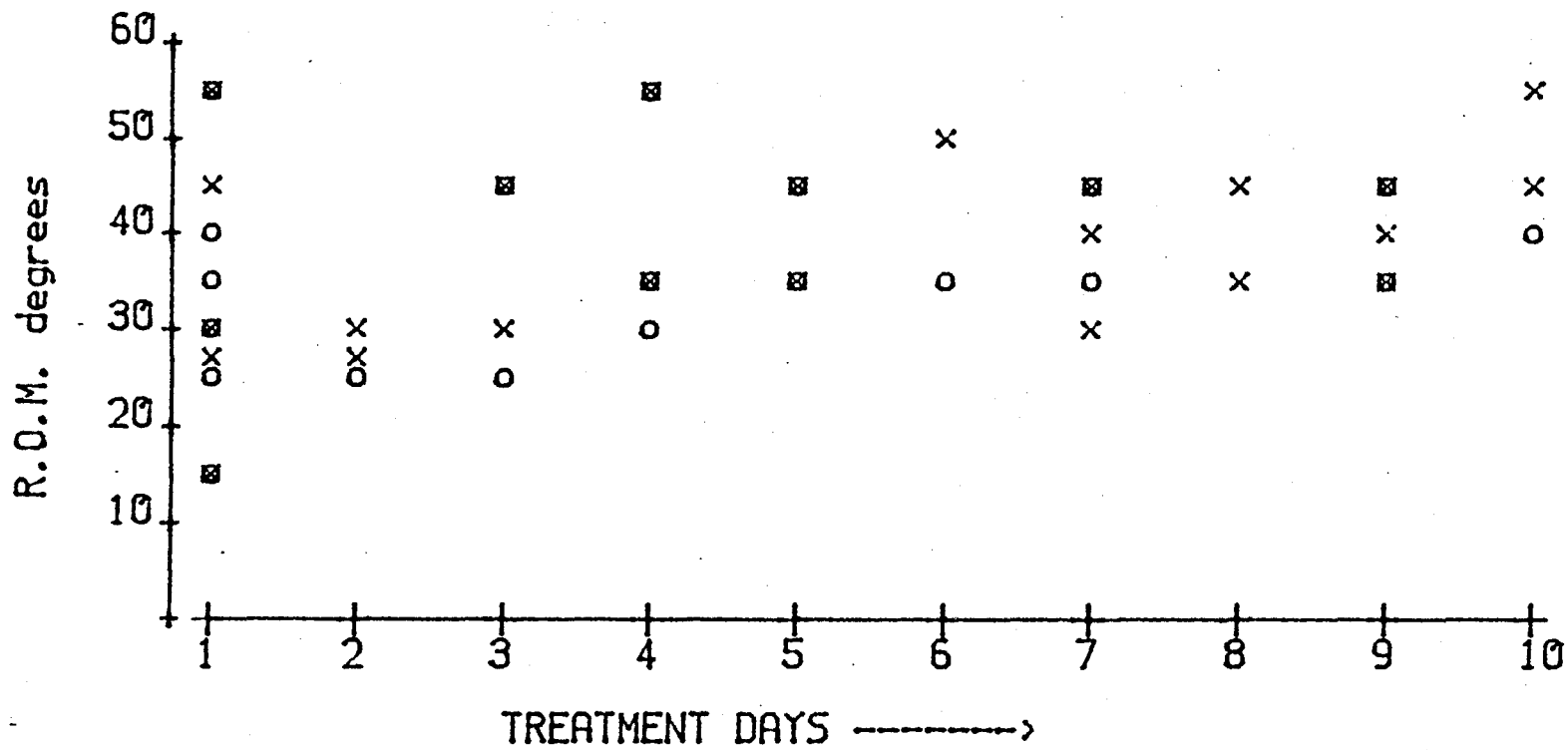


FIGURE 2 - RANGE OF MOTION

Change in range of motion (R.O.M.) of all subjects during treatment with either negative (x) or positive (o) polarity following acute ankle sprain.

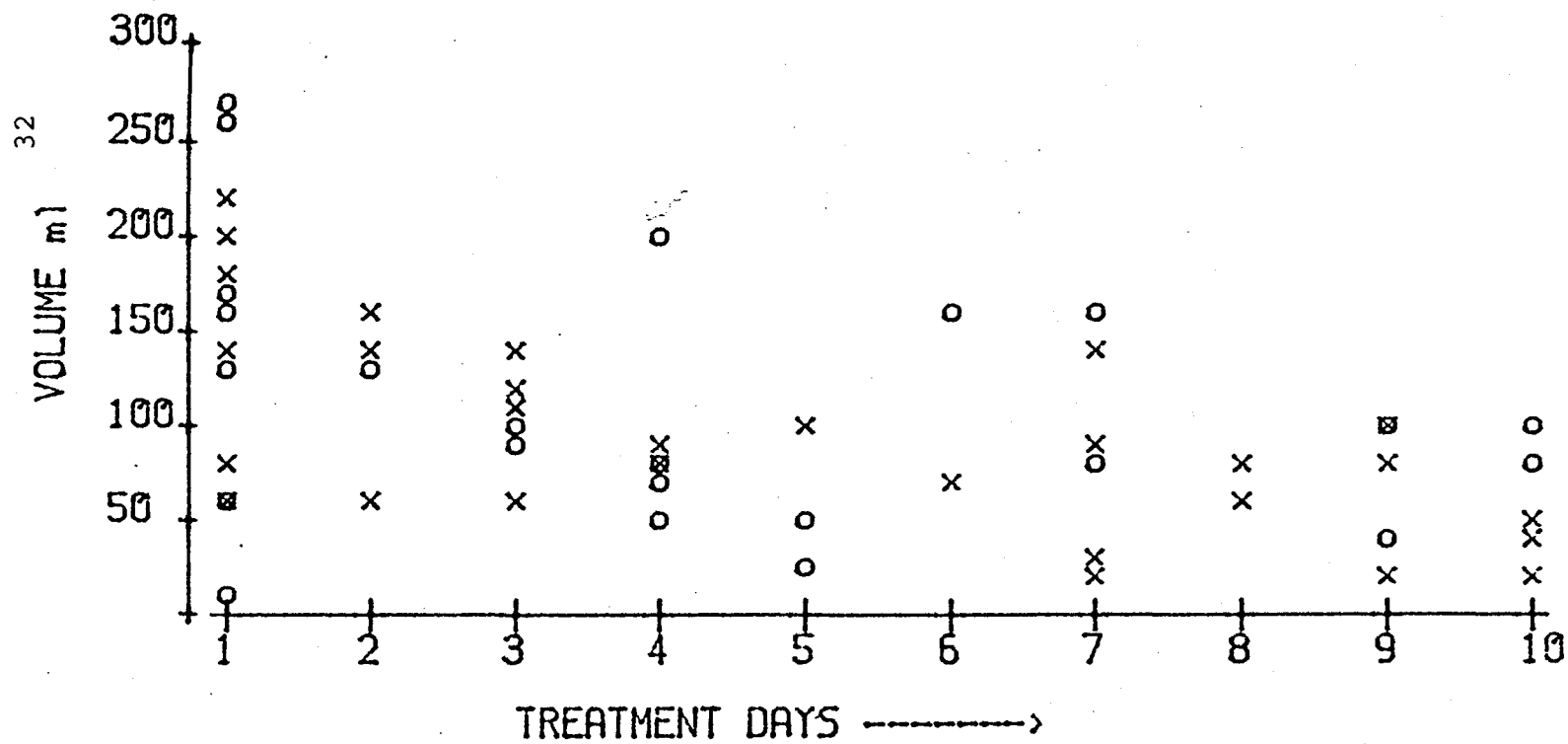


FIGURE 3 - VOLUME CHANGE

Change in ankle-foot volume (Volume - ml) of all subjects during treatment with either negative (x) or positive (o) polarity following acute ankle sprain.

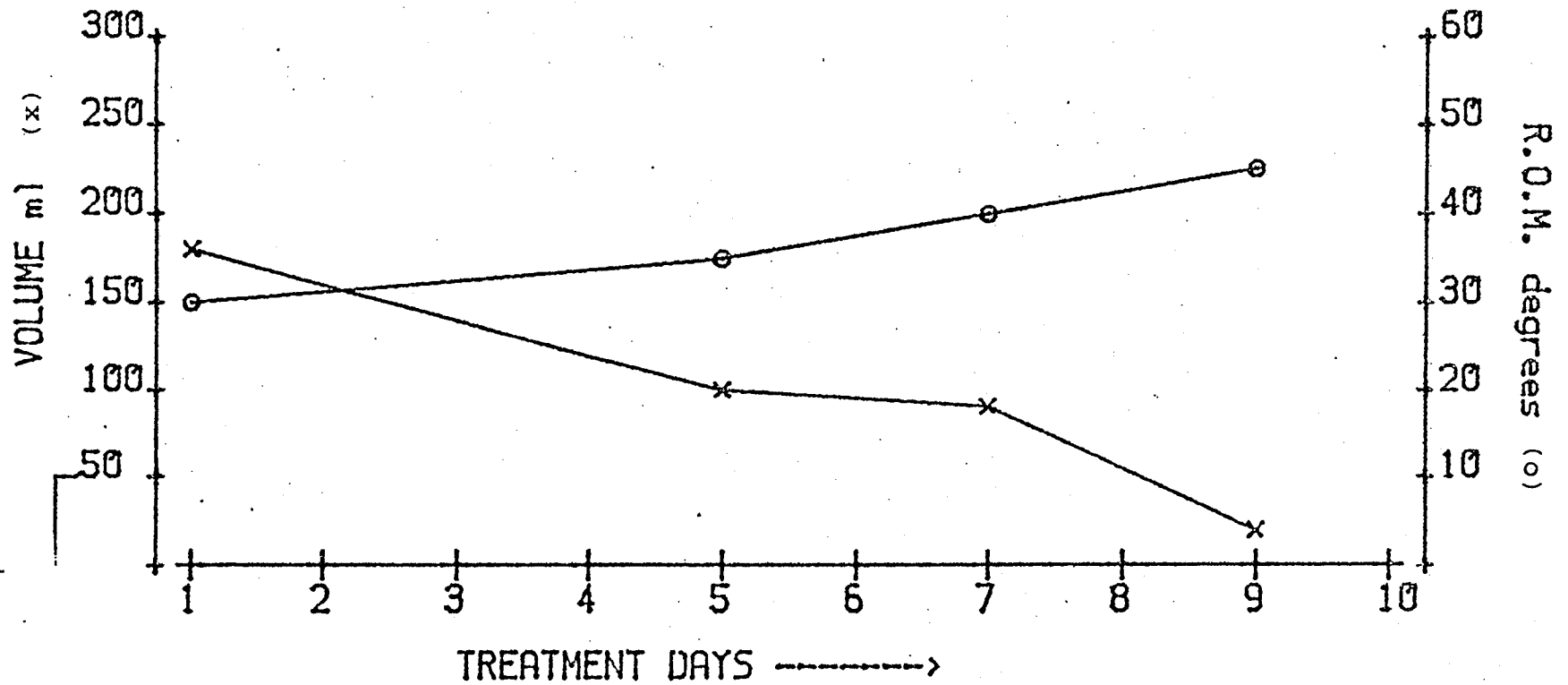


FIGURE 4 - SUBJECT 1Gu

Change in ROM and volume of subject GU during treatment with negative polarity following acute ankle sprain. X = volume, O = ROM

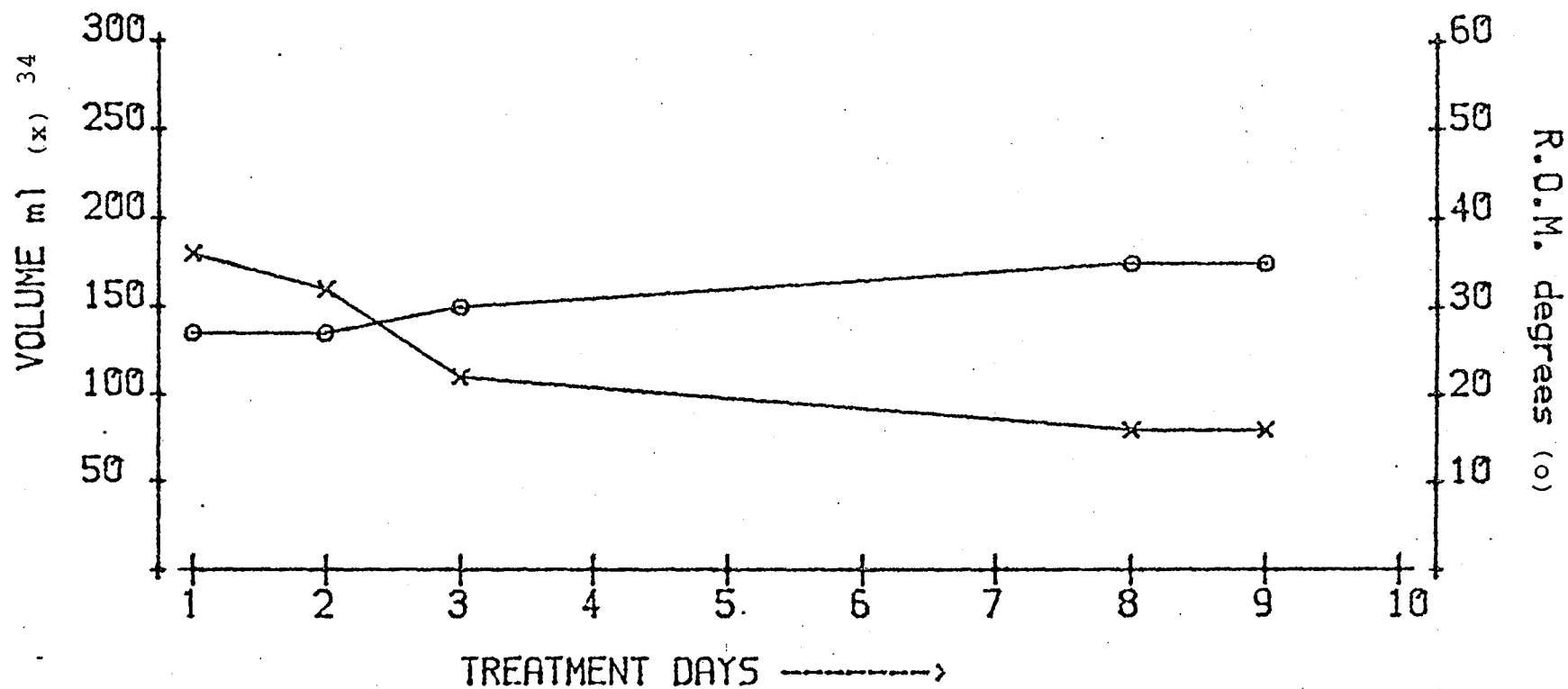


FIGURE 5 - SUBJECT 1An

Change in ROM and volume of subject An during treatment with negative polarity following acute ankle sprain. X = volume, O = ROM

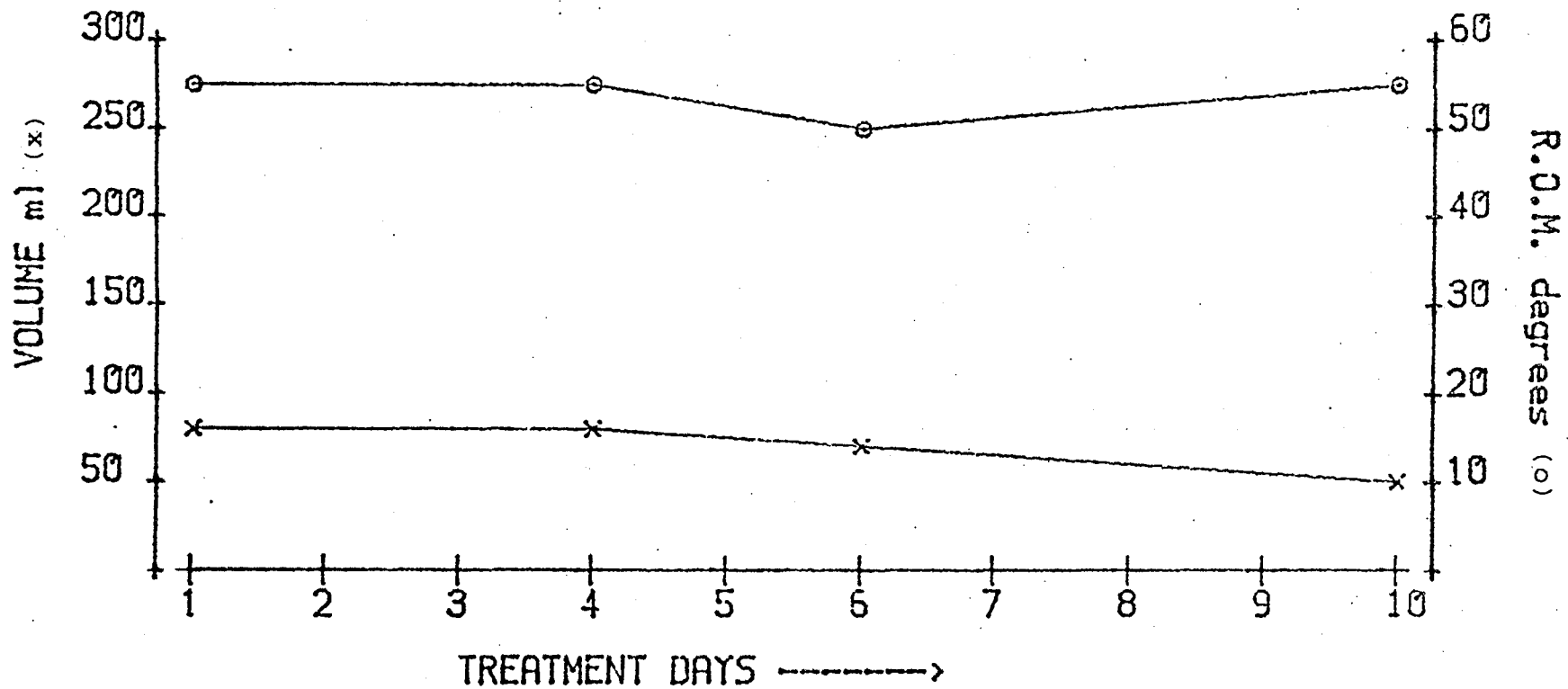


FIGURE 6 - SUBJECT 1Cr

Change in ROM and volume of subject Cr during treatment with negative polarity following acute ankle sprain. X = volume, O = ROM

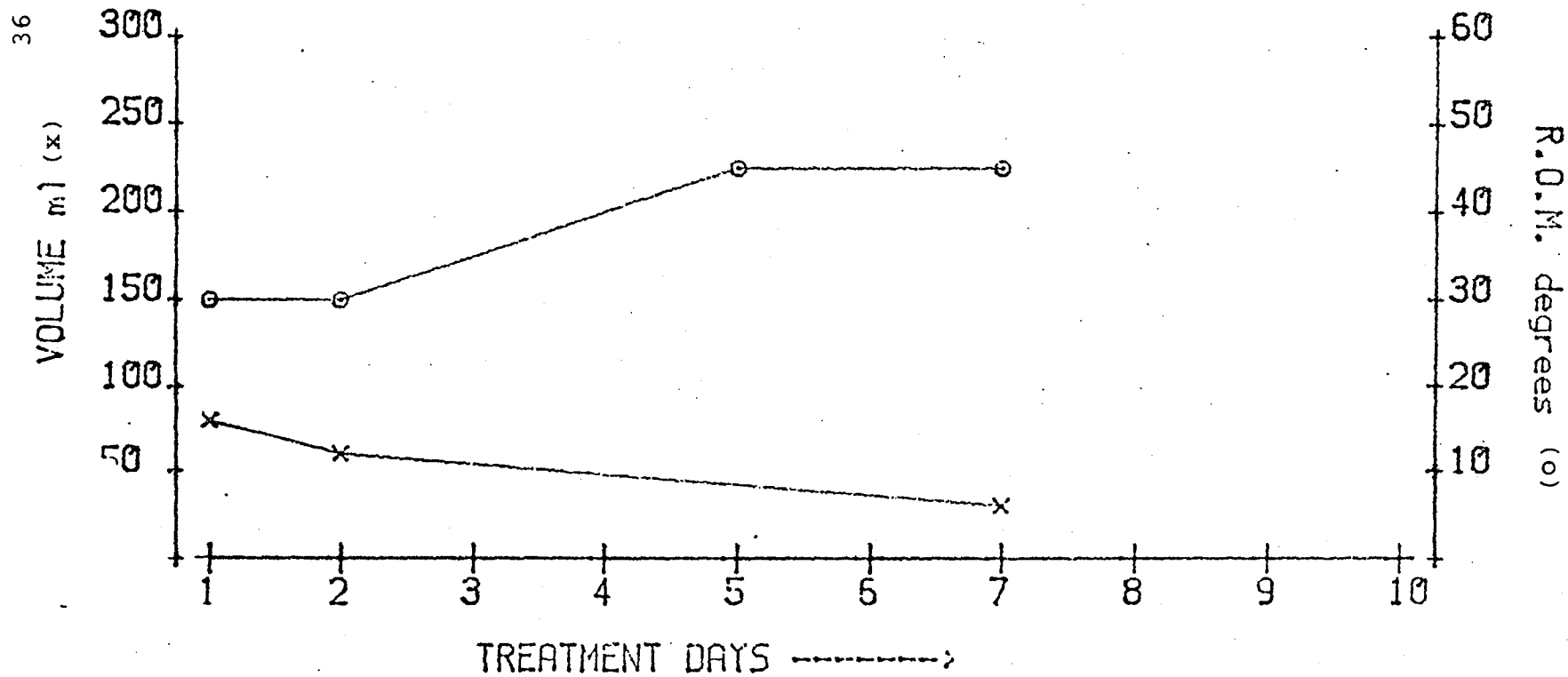


FIGURE 7 - SUBJECT 1Sm

Change in ROM and volume of subject Sm during treatment with negative polarity following acute ankle sprain. X = volume, O = ROM

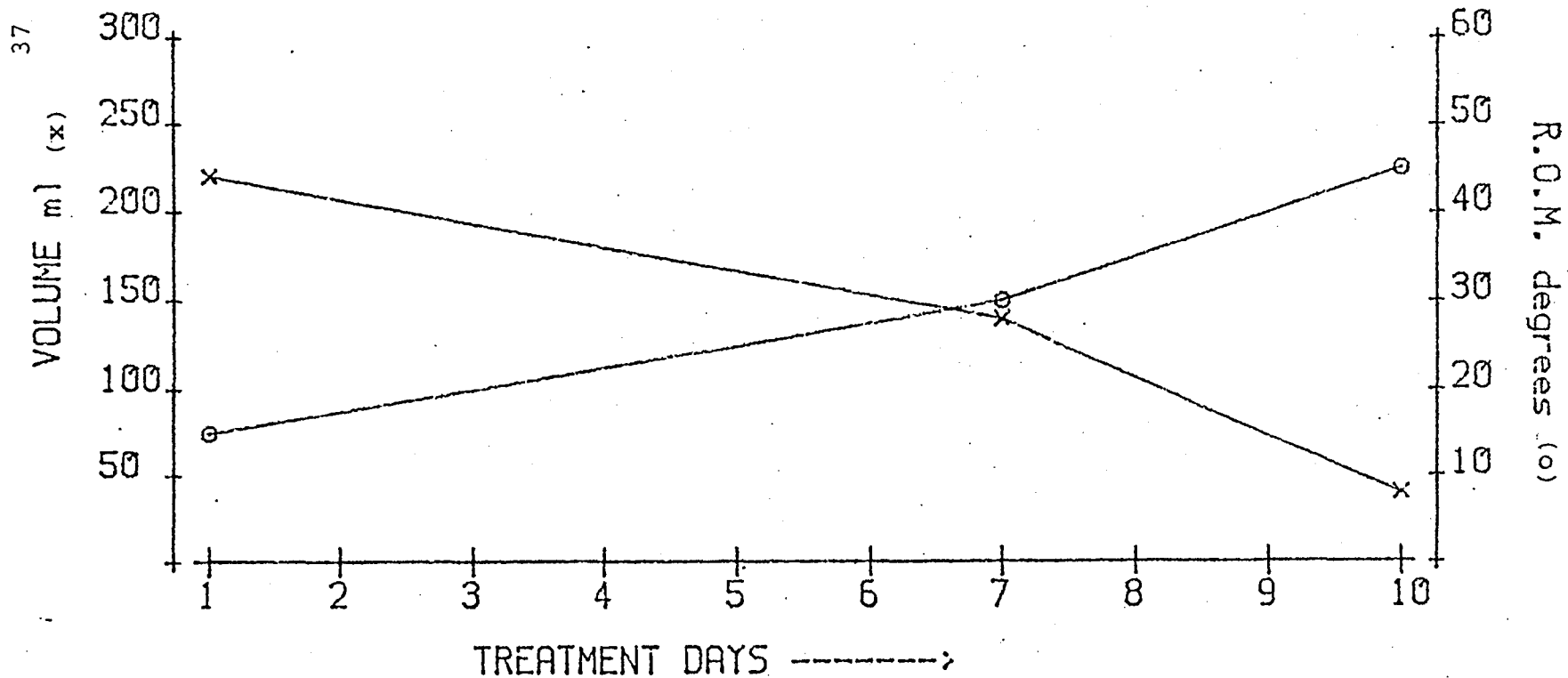


FIGURE 8 - SUBJECT 1F1

Change in ROM and volume of subject F1 during treatment with negative polarity following acute ankle sprain. X = volume, O = ROM

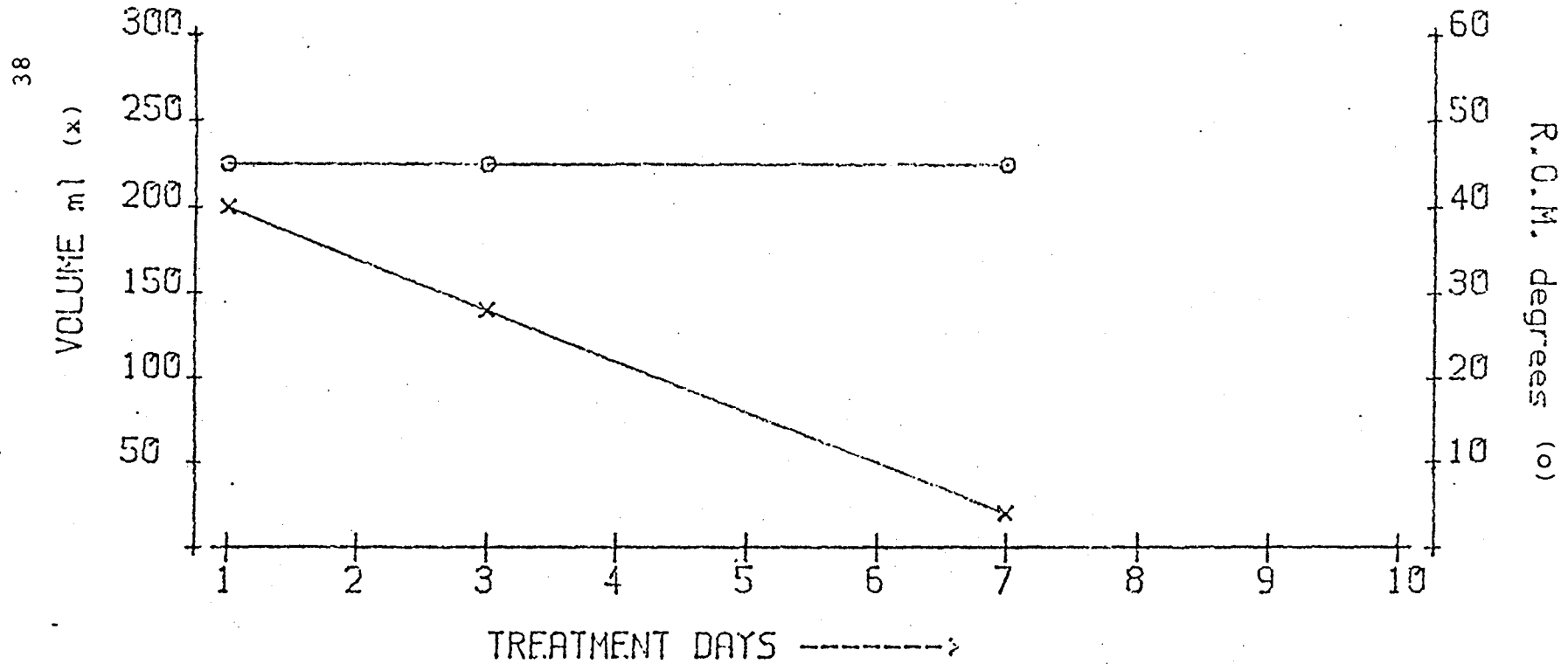


FIGURE 9 - SUBJECT 1Mc

Change in ROM and volume of subject Mc during treatment with negative polarity following acute ankle sprain. X = volume, O = ROM

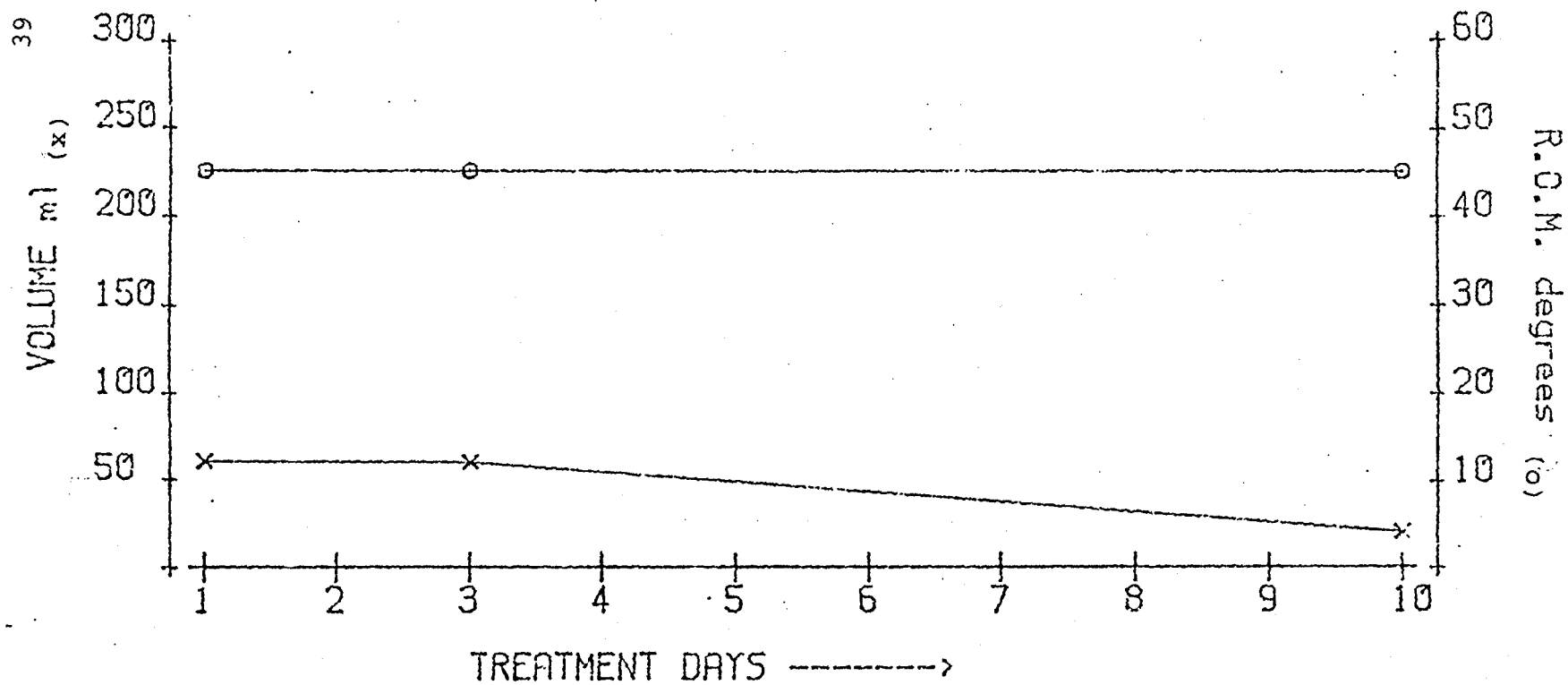


FIGURE 10 - SUBJECT 1Ha

Change in ROM and volume of subject Ha during treatment with negative polarity following acute ankle sprain. X = volume, O = ROM

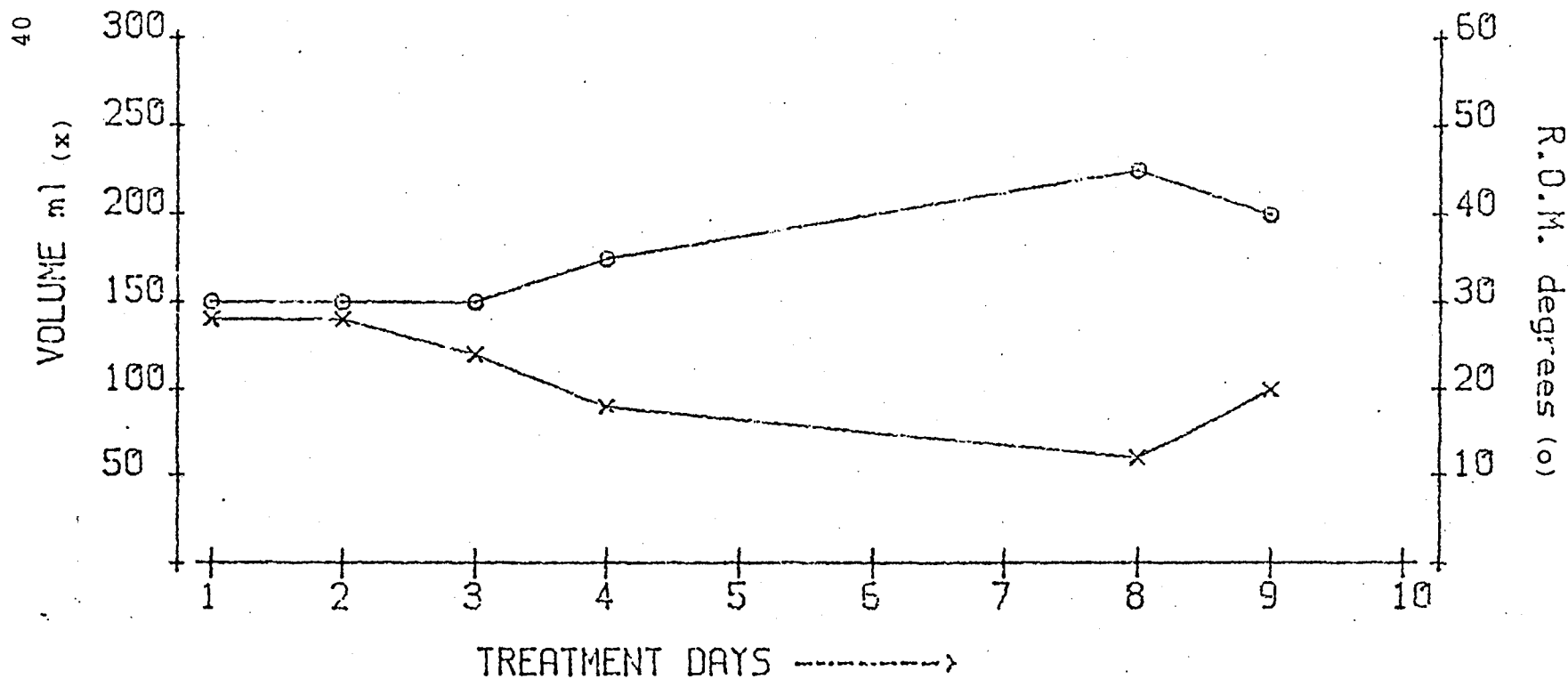


FIGURE 11 - SUBJECT 1Lu

Change in ROM and volume of subject Lu during treatment with negative polarity following acute ankle sprain. X = volume, O = ROM

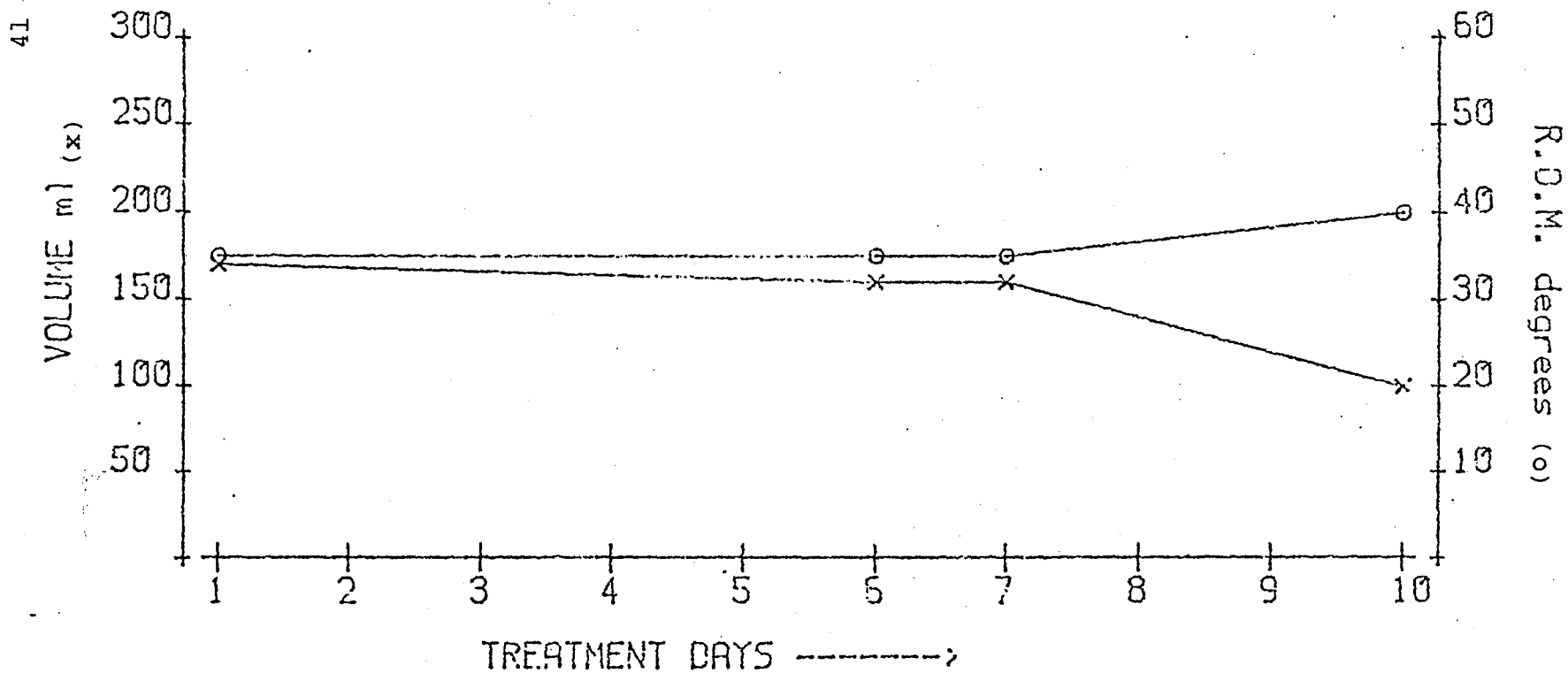


FIGURE 12 - SUBJECT 2Ga

Change in ROM and volume of subject Ga during treatment with positive polarity following acute ankle sprain. X = volume, O = ROM

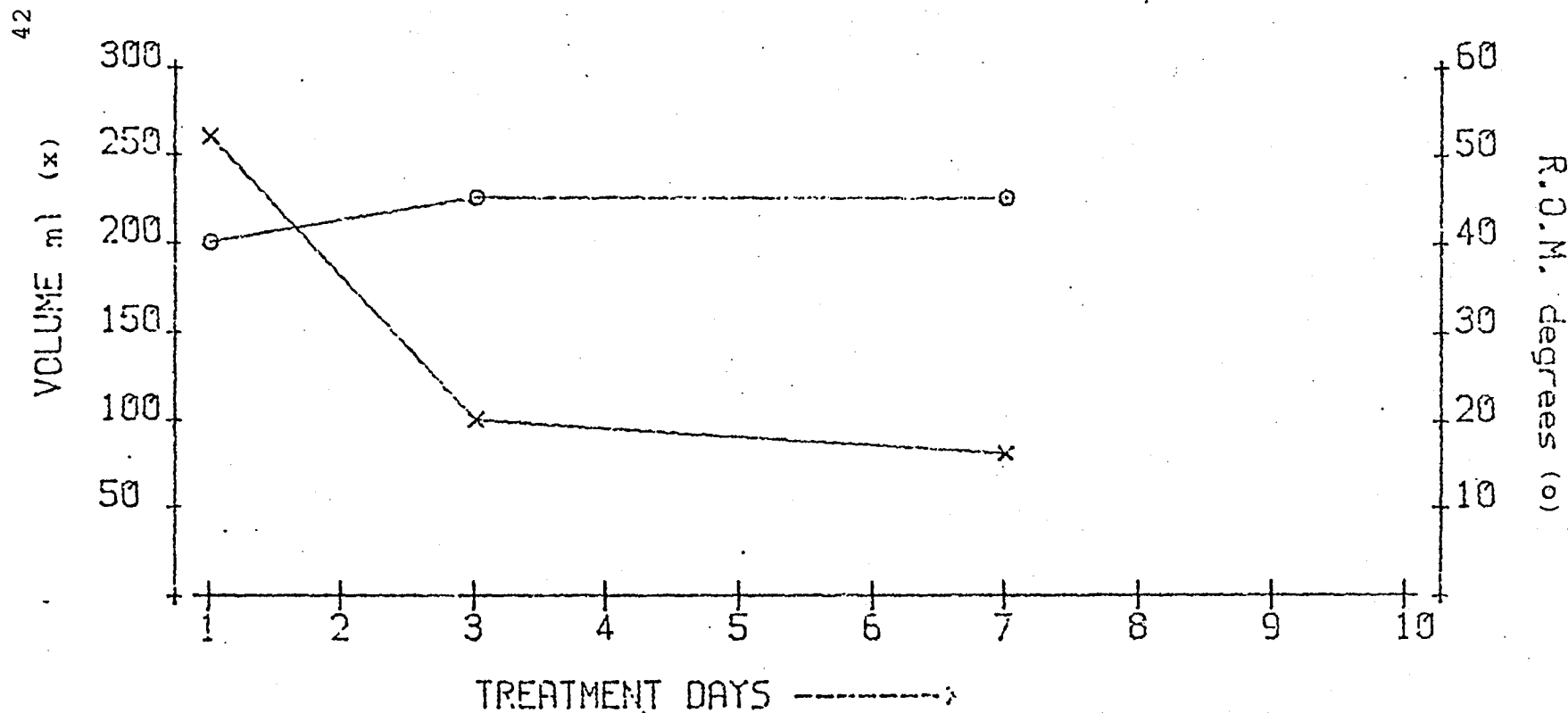


FIGURE 13 - SUBJECT 2Bi

Change in ROM and volume of subject Bi during treatment with positive polarity following chronic ankle sprain with vascular insufficiency. x = volume, o = ROM

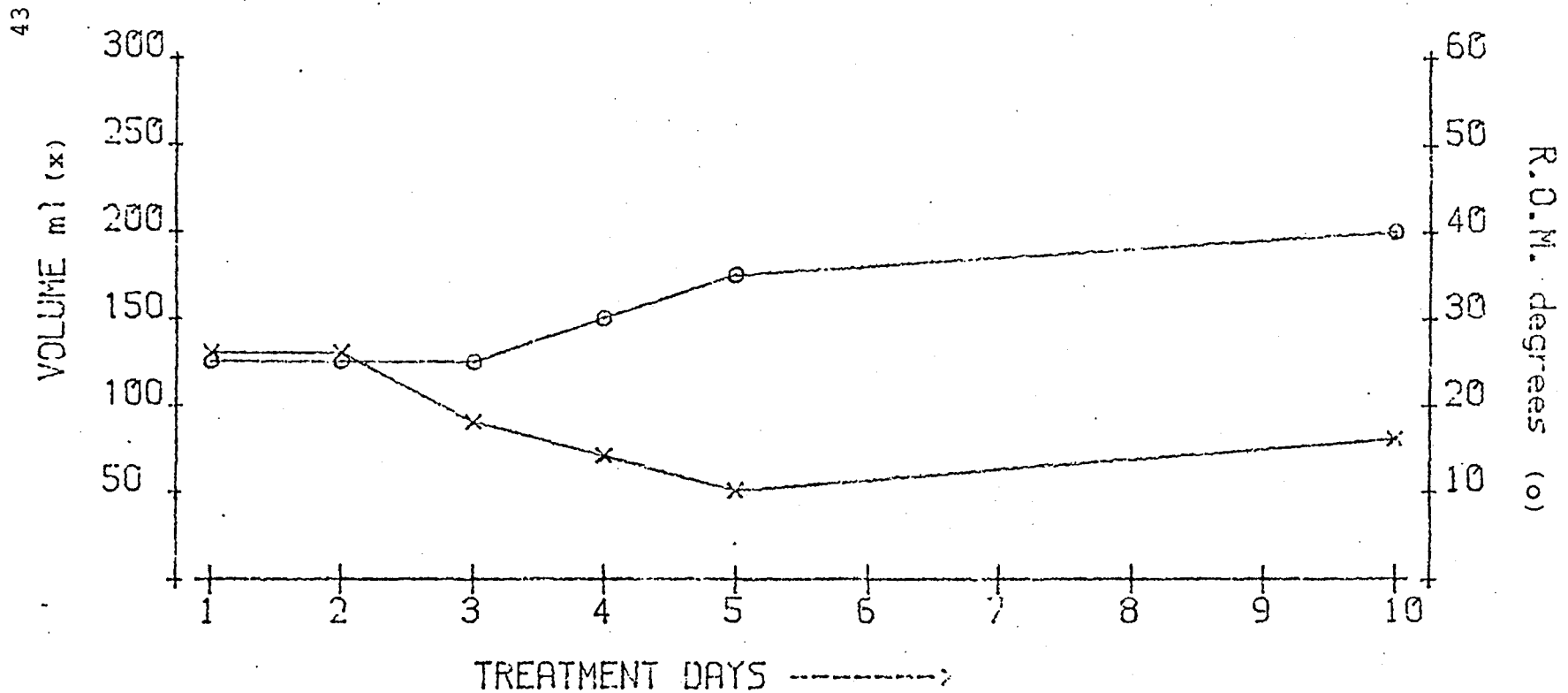


FIGURE 14 - SUBJECT 2Va

Change in ROM and volume of subject Va during treatment with positive polarity following chronic ankle sprain. x = volume, o = ROM

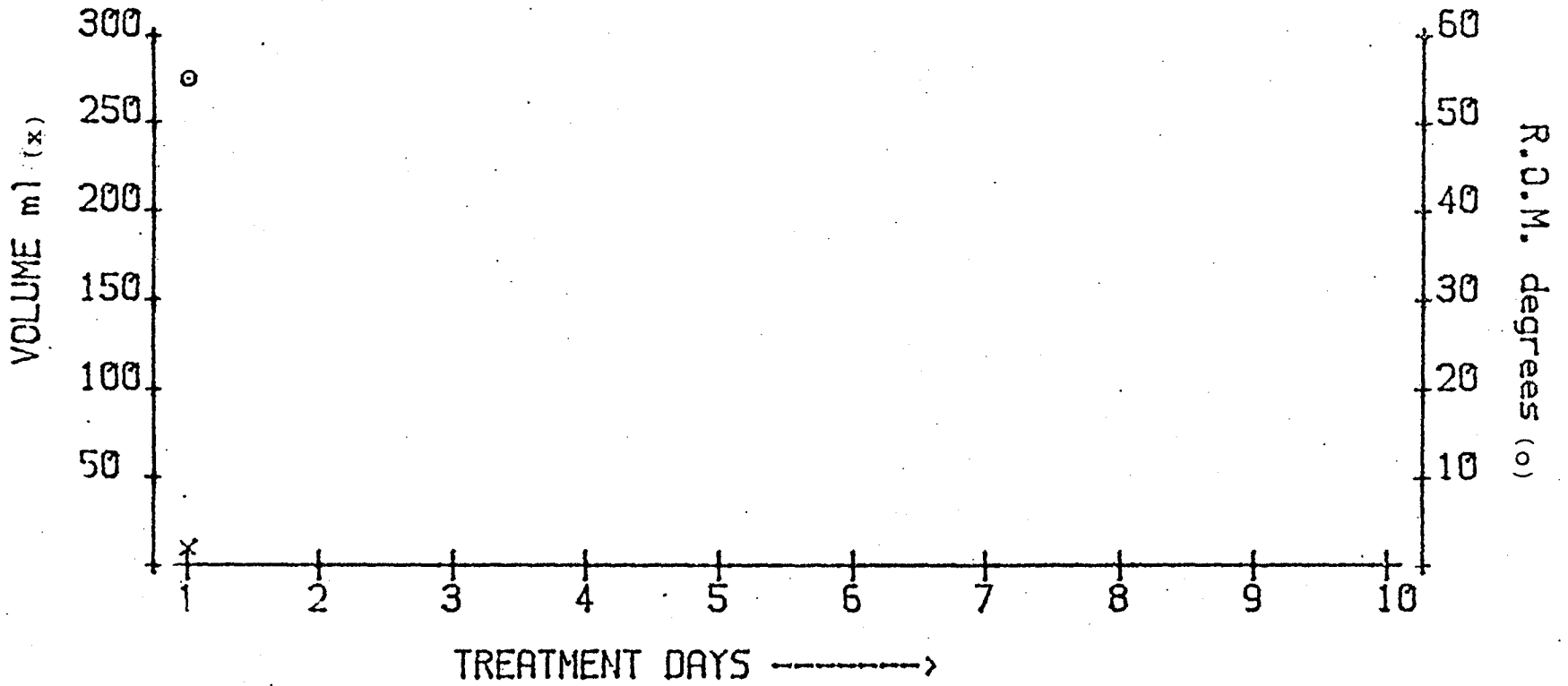


FIGURE 15 - SUBJECT 2Do

Change in ROM and volume of subject Do during treatment with positive polarity following acute ankle sprain. X = volume, O = ROM

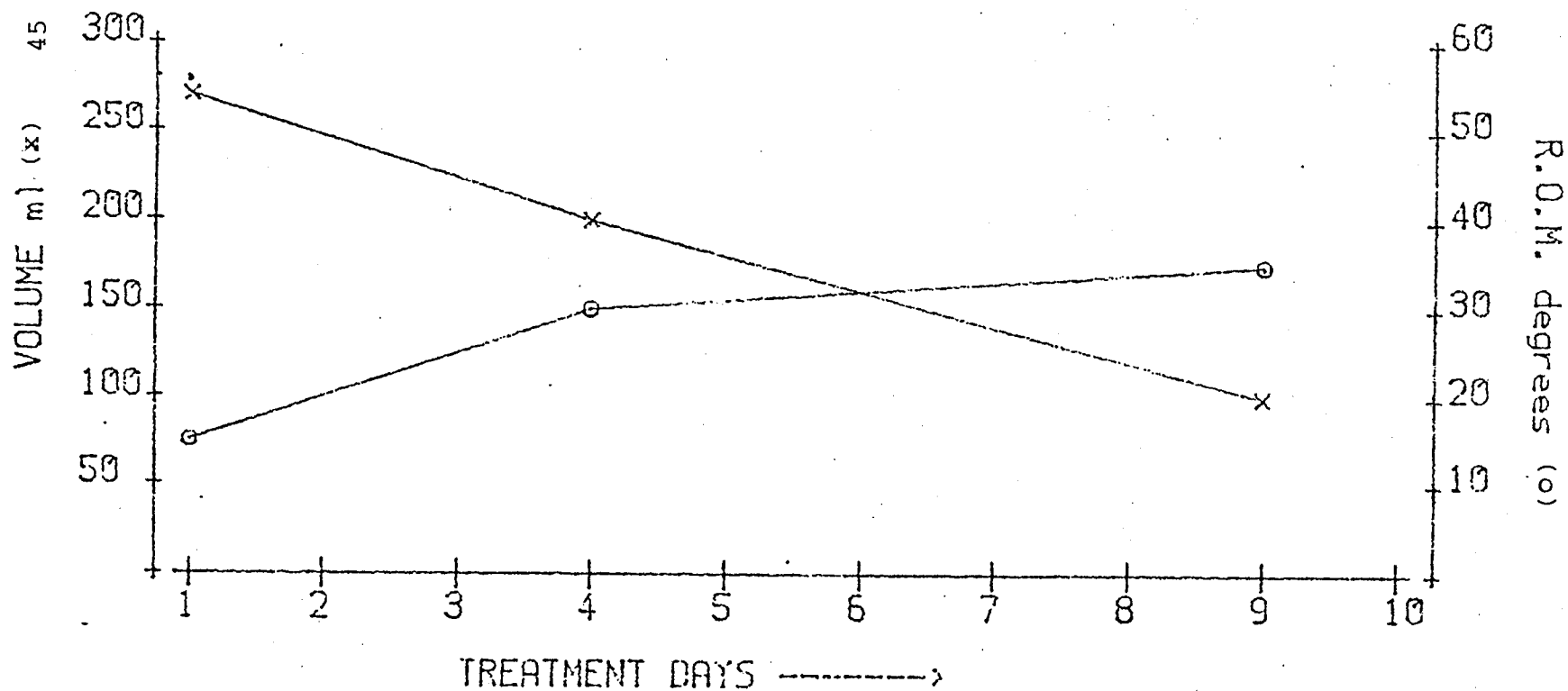


FIGURE 16 - SUBJECT 2Th

Change in ROM and volume of subject Th during treatment with positive polarity following acute ankle sprain with an associated fracture. X = volume, O = ROM

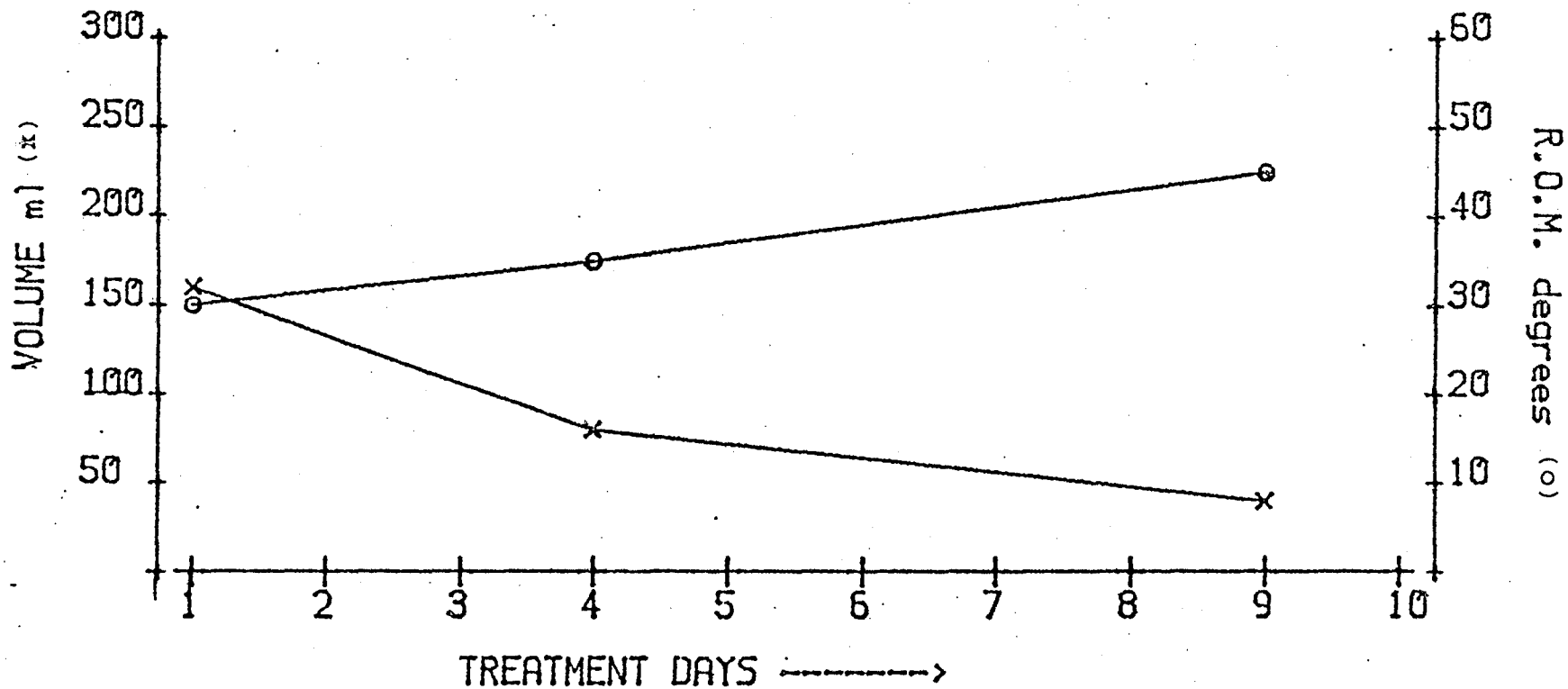


FIGURE 17 - SUBJECT 2Br

Change in ROM and volume of subject Br during treatment with positive polarity following acute ankle sprain. X = volume, O = ROM

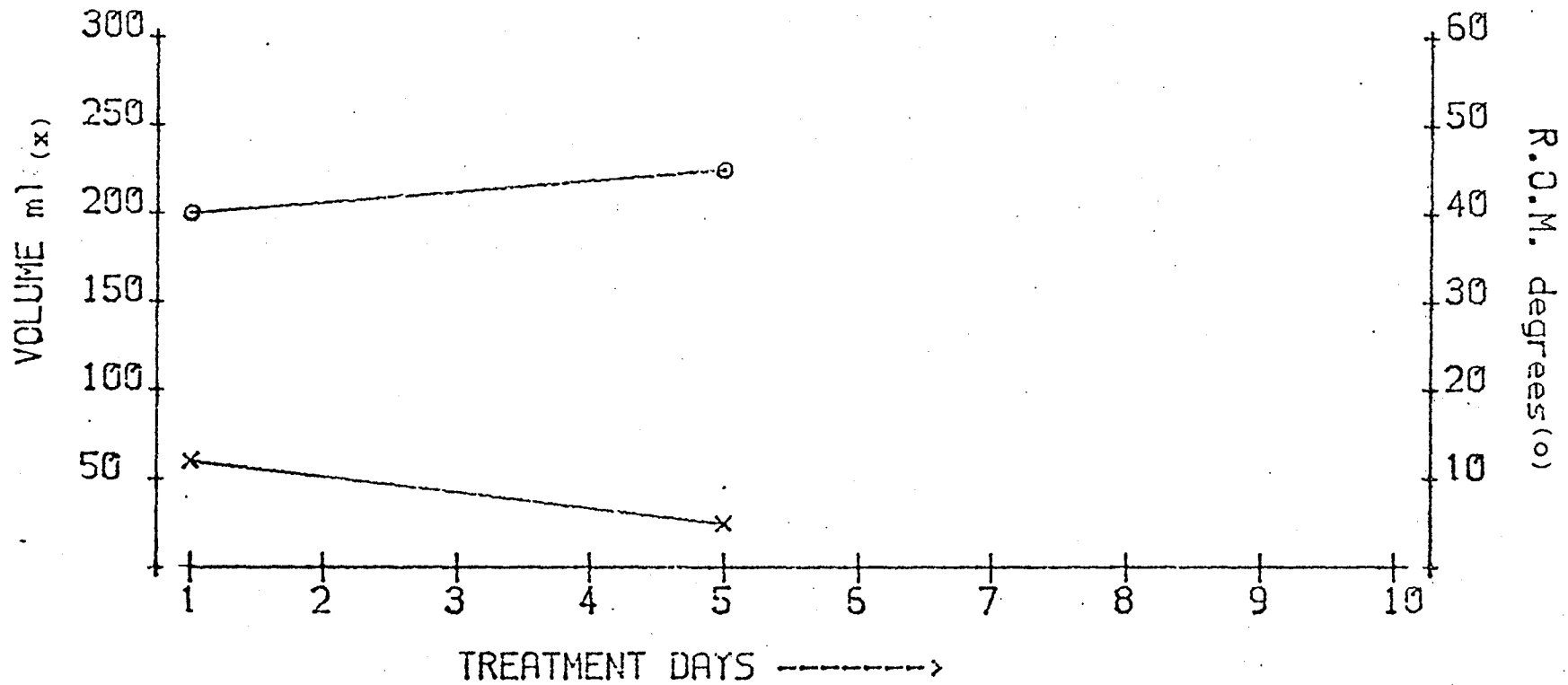


FIGURE 18 - SUBJECT 2De

Change in ROM and volume of subject De during treatment with positive polarity following acute ankle sprain. X = volume, O = ROM

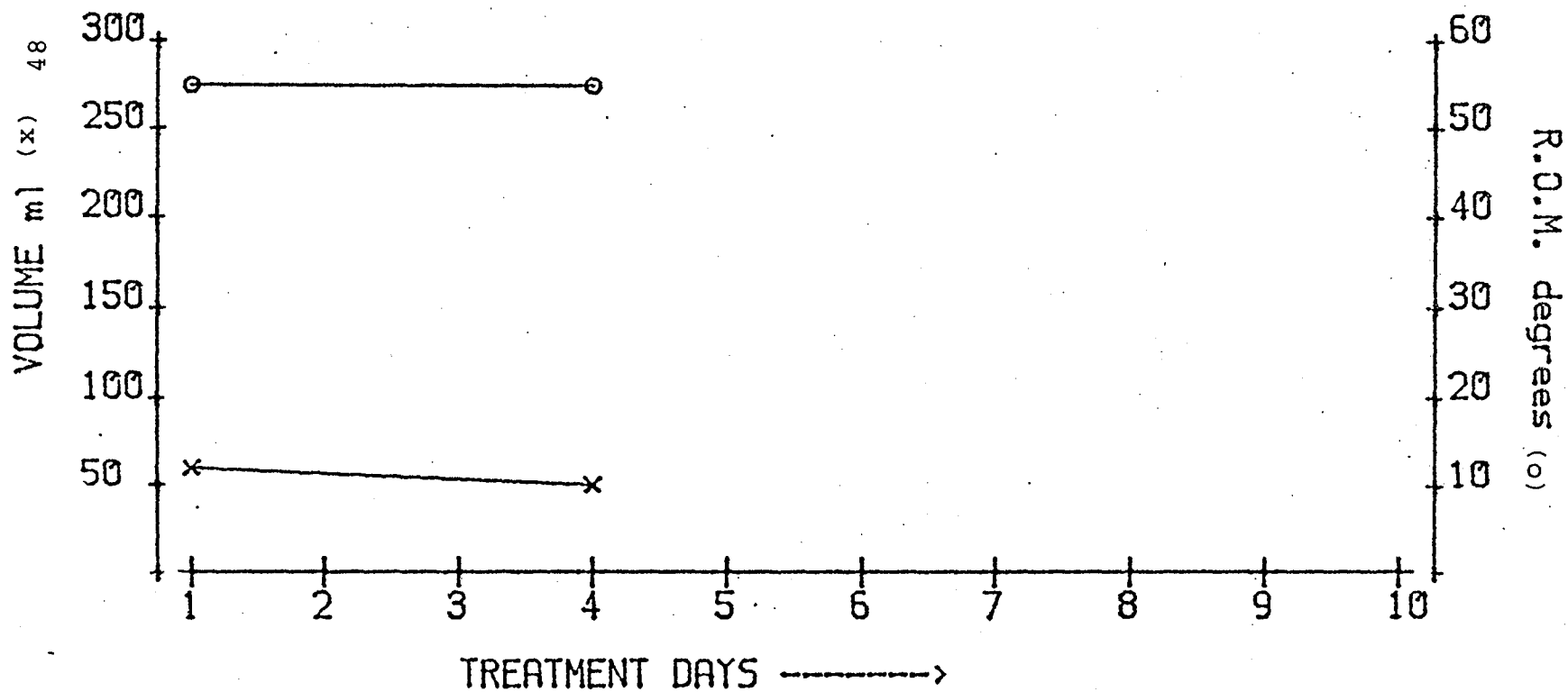


FIGURE 19 - SUBJECT 2Cr

Change in ROM and volume of subject Cr during treatment with positive polarity following acute ankle sprain. X = volume, O = ROM