MUSCLE CRAMPS AND MAGNETIC FIELDS: A REVIEW OF THE LITERATURE AND A CLINICAL CASE STUDY

A Senior Honors Thesis
Presented to
the Faculty of the Department of Psychology
University of Houston

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

By
Mary M. Brown Mauldin
August, 1976

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ABSTRACT

Systremma, or leg cramps, is a non-serious but common complaint usually associated with certain diseases or under certain body conditions. Whenever possible the underlying cause of the cramp is treated; otherwise numerous pharmacological treatments are prescribed to promote symptomatic relief but with limited success. Clinical reports propose an alternative physical treatment involving the use of magnetic fields to reduce systremma. As in other remedies based on folk medicine, this treatment warrants investigation to determine if its effectiveness has a physiological or psychological basis.

This paper presents a review of literature on systremma, noting the limitations of the present therapies. Included is a case study, an attempt to verify whether a reported reduction of cramping was due to a magnetic effect or to a placebo effect. The S was a 64-yr. old woman in relatively good health with a past history of thrombophlebitis. Five cramps were treated with a magnet; 5 more were treated with a steel bar (placebo). Magnetically-treated cramps and placebo-treated cramps were compared to each other and to baseline cramp measures, using mean cramp durations and mean cramp severities. Analysis of data yielded no conclusions.

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CHAPTER I

INTRODUCTION

Relatively little investigation has been done on the subject of muscle cramps, a common but non-serious complaint in clinical practice. The following paper presents a review of the literature detailing the probable mechanisms of cramping and the therapies presently in use. In addition to the pharmacological means of cramp relief, an alternative, biomagnetic therapy, is discussed. The latter part of the paper investigates the physiological versus the psychological aspects of magnetic field effects on muscle cramping.

Definition: Systremma

Muscle cramps are usually defined as involuntary, sustained, painful contractions of skeletal muscle, where the muscle is visibly and palpably taut. The discussion on cramps here will be limited to those in the leg and will, in addition, exclude "cramps" associated with intermittent claudication as these seem to be triggered by a different mechanism altogether. While muscle cramps may be treated as an entity, a review of the literature up through 1969 did not reveal a scientific name for this ailment. However, since then, Ayres and Mihan (1969) have submitted the term "systremma" as an appropriate name for leg cramps. Systremma, according to Ayres and Mihan (1969), is defined as

"cramps in the calf of the legs," from the Greek, meaning "anything twisted up together."

Incidence of Systremma

According to Ayres and Mihan (1969), "most current texts on internal medicine and orthopedic surgery give scant attention to leg cramps, despite the fact that it is recognized as a fairly common complaint." In addition, little agreement seems to exist as to how widespread this problem is.

Norris and his associates (1957) found that 16% of a sample of healthy young people had leg cramps. According to Mauer (1961) 50% of the adult population (ages 15 through 80) would have leg cramps during some period of their lives. Others, while not specifying the incidence, considered cramps among the most common complaints in clinical practice (Ayres & Mihan, 1969; Perchuk, Weinreb, & Aksu, 1961), especially among the chronically ill and elderly (Chesrow, Kaplitz, Breme, & Vetra, 1963; Stern, 1962).

Another segment of the population often affected are pregnant women. Page and Page (1953) found that 50% of pregnant women seen in private practice had leg cramps. Wolff (1954) confirmed the Pages' findings; the incidence of cramping for pregnant women on an unrestricted diet was found to be 51%. Murphy (1962) claimed an even higher incidence of 85%, with a third of these (28.3%) requiring treatment. On the other hand Misischia (1960) found that only 24.2% of pregnant women developed leg cramps. While

Salvatore (1961) found that 33% of his obstetrics patients had leg cramps, only 6.1% of the total sample had cramps severe enough to require treatment. The disparities in the incidences of cramping in pregnant women can probably be attributed to the fact that the symptom categories were based on the patients' subjective evaluations and the individual interpretations of each physician and only slightly to the differences in the population samples used. Except for the Murphy (1962) findings of 85%, the percentages of pregnant women who develop leg cramps do not appear to exceed the incidence of cramping in the overall adult population as reported by Mauer (1961).

Normal Muscle Physiology and Contraction

Systremma, present in a variety of conditions, are symptomatic of abnormal muscle states. Before one studies the abnormal, it may be advisable to become familiar with the normal muscle condition. The following is a brief review of skeletal muscle physiology and the mechanics of muscle contraction.

Physiology. A muscle fiber is a single cylindrical muscle cell. Each muscle fiber is surrounded by a thin membrane, the sarcolemma; within this is the true cell membrane of the muscle fiber, the plasma membrane which transmits action potentials. Within the muscle fiber are bundles of parallel myofibrils which run the length of the fiber. The myofibrils in turn are composed of actin and myosin protein

molecules which together constitute the contractile elements of the muscle; myofibrils have the same pattern of cross striations as the muscle fibers of which they are a part. These fairly wide light and dark bands are called I bands, or isotrophic bands, and A bands, or anistrophic bands, respectively, sue to the different refractive indices of these materials. The arrangement of the actin and myosin filaments in the fiber makes the light and dark bands; the A band corresponds to the myosin filaments and the I band corresponds to the segment between successive myosin filaments. The actin and myosin filaments interdigitate. Each I band is bisected by a dark, narrow line, the Z line, which appears to be attached to the sarcolemma; the H zone is a region of relatively low refractive index in the center of the A band; the H zone is sometimes bisected by the M line. The contractile unit from one Z line to the next is called a sarcomere (Dowben, 1969; Guyton, 1969).

The motor unit is the functional unit of the muscle, consisting of a single motor neuron and all the muscle cells innervated by its axon. The nerve fiber ends in a specialization of the muscle fiber called the motor end plate, which is a large area of contact between the nerve axon and the muscle fiber membrane (Dowben, 1969; Villee & Dethier, 1971).

Mechanics of Muscle Contraction. When a muscle is given an adequate stimulus, it responds with a single, quick twitch. If a second stimulus is received while the muscle

is still contracted, the superposition of the second contraction on the first results in a greater than normal shortening of the muscle fiber: this effect is called summation. Muscles do not usually contract in single twitches but in sustained contractions evoked by a rapid succession of stimuli, which do not allow the muscle to relax in between. The fusion of superimposed twitches is called tetanus or tetanic contraction and may be maintained until the muscle is fatigued (Rasch & Burke, 1967; Villee & Dethier, 1971).

A mild state of tetanus, tonus, is present at all times and involves only a few of the fibers in the muscle at a time; the individual fibers are believed to contract in turn so as not to become fatigued. Tonus allows the muscle to react more rapidly and contract more strongly than one that is completely relaxed (Villee & Dethier, 1971).

As in the transmission of an impulse over a nerve fiber, a muscle fiber that is excited contracts to the full extent of its immediate ability, demonstrating an all-or-none principle; the amount of contraction of a muscle fiber is independent of the strength of the stimulus (Guyton, 1969; Rasch & Burke, 1967).

Whole muscles do not follow the all-or-none principle. Instead, they have the ability to contract at varying degrees of strength. The strength of contraction or gradation results from the interaction of three factors: the number of motor units stimulated (recruitment), the frequency of

stimulation (summation), and the timing of the stimuli to varous motor units (synchronization). Recruitment, the major mechanism of gradation, is the ability of the central nervous system to send stimuli to more or fewer motor units (Rasch & Burke, 1967). This is sometimes called multiple motor unit summation. Summation (which is explained above) is sometimes called wave summation (Guyton, 1969). Ordinarily, impulses reaching different motor units are out-of-phase or asynchronous. When a strong contraction is required, impulses to many or all the motor units occur simultaneously; this is synchronization (Rasch & Burke, 1967). Asynchronous summation--the interaction of recruitment, summation, and synchronization--is caused by neuronal circuits in the spinal cord that automatically distribute impulses evenly and sequentially among the different nerve fibers to the muscle (Guyton, 1969). To strengthen a muscle contraction, impulse frequency is increased, more motor units are activated, and the timing of the impulses to the various motor units is synchronized.

Biochemistry and Energy for Contraction. As this is a complex process, only a summary version is given here. When a nerve impulse reaches the myoneural junction (motor end plate), acetylcholine (ACh) is released which acts on the plasma membrane to make it permeable to sodium ions. The rapid influx of sodium ions creates an electrical potential which, if strong enough, will travel along and through the

muscle fiber (Guyton, 1969; Villee & Dethier, 1971). One theory is that in a relaxed state, magnesium (Mg) ions are bound to a relaxing agent, the Marsh-Bendall factor; calcium (Ca) ions are bound to the actomyosin. When the muscle is stimulated, the Mg transfers to the actomyosin and Ca transfers to the Marsh-Bendall factor; this shift is supposedly responsible for the release of actomyosin to catalyze hydrolysis of adenosine triphosphate (ATP) to release energy for contraction (Rasch & Burke, 1967).

Another more likely theory proposes that when the action potential spreads inside the muscle fiber, the sarcoplasmic reticulum releases calcium ions into the fluids surrounding the myofibrils. When the action potential is over, no more stimulus is present for the release of calcium ions; calcium ions now recombine with the reticulum and the contraction ceases (Dowben, 1969; Guyton, 1969). But during the contraction, some of the calcium ions combine loosely with myosin molecules to form activated myosin which has the properties of the enzyme, ATPase. The activated myosin ATPase can now act with ATP to release energy for contraction (Guyton, 1969).

A steady supply of ATP is needed for contraction, so ATP is restored from creatine phosphate (CP) stored in the muscle. During muscle recovery, CP is resynthesized from ATP, using energy from glycolysis, the breakdown of muscle and liver glycogen to lactic acid. Lactic acid, a poison to

the muscle, must be removed. Part of it is oxydized, which in turn provides energy to convert the rest of the lactic acid back to glycogen (Rasch & Burke, 1967). Often lactic acid accumulates faster than it can be oxydized; the muscles have incurred an oxygen debt. The tissues need more oxygen to restore the energy-rich phosphate compounds and glycogen to their original conditions (Villee & Dethier, 1971).

Some controversy exists concerning this ATP theory; there is argument as to whether mechanical changes come first to elicit the biochemical events, or whether the biochemical changes come first and cause the mechanical activity to occur, or even whether neither one causes the other (Rasch & Burke, 1967).

Biophysics of Contraction. The exact physical mechanism whereby a muscle contracts is still unknown but the following theory is the prevalent one. The energy released from ATP is used to pull the actin filaments in the I band inward along the myosin filaments in the A band. The little cross bridges on the myosin are thought to attach themselves to reactive sites on the actin filaments. Movement of the cross bridges "pulls" the actin filaments in a rachet-like motion; the bridge is broken and reformed on the next reactive site. Each formation of new bridges is assumed to utilize the energy of one ~P bond; this corresponds to the rate of ATP utilization determined experimentally (Guyton, 1969; Rasch & Burke, 1967; Villee & Dethier, 1971).

In summary, from knowledge of the normal process of contraction, cramps may be due to excessive activity at a number of points in the neuromuscular apparatus. There may be (1) unstable polarization of the nerve fiber, as in hypocalcemic and hypomagnesemic tetany, or (2) hyperirritability of the motor neuron. (3) The threshold level, regulated by Ca ions, for mechanical activation or electrical reactivation of the sarcolemmal membrane may be reduced, or (4) a change may occur within the muscle fiber itself, which, once shortened may have insufficient energy for restoration to a relaxed state, relaxation being an active process requiring additional energy.

Muscle Cramps

Muscle cramps are known to be related to a variety of conditions. These include pregnancy (Fields, 1960; Mauer, 1961; Misischia, 1960; Murphy, 1962; Page & Page, 1953; Perchuk, 1964; Perchuk et al., 1961; Salvatore, 1961; Wolff, 1954), vascular diseases (varicose veins, arteriosclerosis, thrombophlebitis, vasospasm) (Chesrow et al., 1963; Fields, 1960; Mauer, 1961; Perchuk, 1964; Perchuk et al., 1961), static foot deformities (Fields, 1960; Perchuk, 1964; Perchuk et al., 1961), other orthopedic disorders (Turek, 1961), neurologic diseases (Chesrow et al., 1963; Norris, Gasteiger, & Chatfield, 1957), arthritic diseases (Chesrow et al., 1963; Perchuk, 1964; Perchuk et al., 1961), after unusual muscular effort (Fields, 1960; Norris et al., 1957),

old age (Chesrow et al., 1963; Perchuk et al., 1961; Stern, 1962), during sleep (Ayres & Mihan, 1969; Ayres & Mihan, 1974; Fields, 1960; Perchuk, 1964; Perchuk et al., 1961), and metabolic imbalances (Ayres & Mihan, 1969; Fields, 1960; Misischia, 1960; Murphy, 1962; Page & Page, 1953; Salvatore, 1961; Wolff, 1954). Perchuk and his associates (1961) found that mercurial diuretic administered to cardiac patients was also related to cramp incidence, most likely due to alteration of serum electrolytes.

For some patients the etiology of their cramps is unknown, but whenever possible, the underlying cause of the cramp is investigated before treatment; so many proposed underlying causes make investigation difficult. Even when a relationship between a particular condition and the cramp symptom is known to exist, there is still the problem of finding the proper therapy.

As an example, calcium deficiency had been suspected as a cause of systremma in pregnancy since the 1930's (Page & Page, 1953; Salvatore, 1961; Wolff, 1954), but the Pages (1953) found that deficiency to be enhanced by an excessive intake of phosphorus; the imbalance in turn caused a neuromuscular irritability that was thought to cause the cramp. Most of the authors consider neuromuscular irritability to be the chief cause of systremma (Perchuk, 1964). Irritability can be caused by any number of local irritants, including metabolic imbalances; the result is a painful cramp due

to the intense rate of contraction (300/sec.) compared to 10/sec. to 50/sec. during normal contraction (Perchuk, 1964). However, Norris and others (1957), investigating from a neurological view, found that muscle action potentials recorded during cramps reflected motor unit activity involving most, if not all, of the fibers of a motor unit and that the motor activity in cramps most likely originated in the central nervous system rather than in the peripheral nerves. Hyperexcitability of the spinal mechanism was suggested due to changes in cramp activity brought about reflex action.

On the other hand, Ayres and Mihan (1969) proposed that systremma is due to a vitamin E deficiency; in 1974 they revised the underlying cause to include muscle anoxia resulting in the accumulation of abnormal metabolites. As the causes of cramping are still being investigated, a direct cure for systremma is not yet in sight.

Pharmacological Therapy

Whenever possible, the specific cause of the cramp should be determined and treated, but since that is not always possible, symptomatic relief of the cramping and the pain may be obtained using analgesics, muscle-relaxants, vitamins, or magnets. The pharmacological means of therapy will be discussed first and will include the following drugs: carisoprodol, methocarbamol, tocopherol (vitamin E), Benadryl, quinine, and calcium lactate.

Carisoprodol

Carisoprodol appears to be particularly suited to treatment of cramps in that it has both muscle-relaxant and pain-relieving properties. This drug of the propanediol series is a derivative of meprobamate but surprisingly unlike it in pharmacologic properties. In animals, it depresses multineural spinal reflexes and acts more readily on complicated ones than on simple ones. Some of its relaxant properties differ from those of other centrally acting relaxants, however, so that it may have a different mode of action or act at different sites. It also modified central perception of pain, so that animals would tolerate movement of an inflamed joint at a time when the spasm was still present. The analgesic properties are thus independent of its muscle-relaxant properties. Carisoprodol has neither demonstrable anti-inflammatory properties nor significant antipyretic action. Thus it should be classed apart from the well-known analgesics. Its low toxicity suggests its suitability for clinical use (Turek, 1961).

Three clinical studies conducted with carisoprodol in dosages of two to twelve 350 mg. tablets per day yielded satisfactory to excellent results with some minor side effects (Chesrow et al., 1963; Stern, 1962; Turek, 1961). As the Turek study did not involve primarily leg cramps nor were drug side effects noted as in the other two studies, the details will not be discussed here.

Chesrow Study. Chesrow and his associates administered carisoprodol to two groups of patients (ages 26 to 79) having various neurologic, vascular, or arthritic diseases whose dominant complaint was leg cramps. The dosage was four 350 mg. tablets daily (taken at four different times). Of the total of 50 patients taking the drug, 26 were observed for two and a half months along with 19 others taking the placebo in the double-blind study; the remaining 24 patients, taking no other medication concomitantly, were observed for six weeks. Excellent results (complete or marked relief of pain, muscle spasm, and tenderness) were obtained in 35 patients (70%), slight relief in 12 (24%), and no relief in 3 (6%) compared to only slight relief for 21%, questionable relief for 26%, and no relief for 53% in the placebo group.

Side effects were experienced by 6 patients (12%). These included several episodes of drowsiness in one, itching sensations in another, mild nausea for three others, and mild gastrointestinal upset for the sixth. In all cases these occurred during the first two weeks, did not require treatment, nor necessitated discontinuation of the drug.

Stern Study. Sixty-one elderly patients (ages 59 to 93 years) with moderate to severe pain in one or both legs were treated with carisoprodol. Fourteen patients had cramps while walking (intermittent claudication) and 47 had cramps at night or during rest. All medication used previously for

these cramps was discontinued at least seven days before the trial with the carisoprodol. The initial dosage was one 350 mg. tablet, four times daily, but dosages were increased according to patients' requirements and responses to treatment. Excellent results (cramps completely controlled) were obtained for 15 (25%), satisfactory (marked reduction in frequency and severity of symptoms) for 43 (70%), and unsatisfactory (slight relief in frequency and severity) in 3 (5%). Seven patients received a placebo after two weeks of treatment while seven others received the placebo after three weeks. Pain of the original severity returned ten to fifteen days after discontinuation of carisoprodol taken for two weeks; the interval was seventeen to twenty-three days for those who discontinued the carisoprodol after three weeks of treatment. Relief was noted as soon as carisoprodol was resumed.

Side effects were noted in 7 (11%) of the patients. One patient taking 10 tablets (3500 mg.) daily reported severe nausea which persisted despite reduction of dosage; drug was discontinued. Four patients reported drowsiness and mental confusion; reduction of dosage from 10 to 4 tablets daily relieved these symptoms while still relieving the cramps satisfactorily. Another patient, taking 4 tablets daily, had a depressive reaction with crying spells; drug was discontinued immediately. The seventh patient, on the fourth day of therapy with 4 tablets daily, developed a skin

rash, erythema multiforme; the drug was discontinued immediately. There was no evidence of tolerance or of withdrawal symptoms on discontinuance. All laboratory studies yielded normal findings before and after treatment.

Methocarbamol

Methocarbamol is a derivative of guaiacol glyceryl ether. Pharmacodynamic studies have demonstrated that "methecarbamol (sic) produces a prolonged relaxing effect on skeletal muscles through a depression of spinal multisynaptic pathways with no effect on monosynaptic reflexes" (Perchuk et al., 1961).

> Therapy with methecarbomol (sic) is based on the drug's demonstrated ability to suppress reflex skeletal muscle spasm at the level of the spinal cord. Thus, proprioceptive stimuli arising out of ischemic conditions, imbalances in metabolism, or calcium-phosphorus imbalances are blocked before they are able to trigger clonic or tonic contractions (Perchuk et al., 1961, p. 104).

Methocarbamol's ability to relieve spasm of skeletal muscle suggested it as an agent for the relief of nocturnal leg cramps (Perchuk et al., 1961).

Perchuk Study. With this premise in mind, Perchuk and his associates conducted a study to test the drug's effectiveness in relieving nocturnal leg cramps. Included in the study were twenty women and seven men with nocturnal leg cramps of various origins: thrombophlebitis and other vascular disorders, pregnancy, profound diuresis after the administration of Mercuhydrin, other miscellaneous origins,

and unknown origins (seven patients). The majority of these subjects were between the ages of 40 and 80 years. Twenty-one patients received the drug, three received the placebo, and three alternated between drug and placebo. Medication was discontinued for seven in the first group to observe whether the cramps would recur. The dosages varied from four to eight 500 mg. tablets daily in divided doses to four tablets at bedtime. The evaluation of the drug was based on the frequency of cramps. Of the 24 patients receiving the drug, 19 had excellent results (night cramps completely controlled) and five had satisfactory results (a gross reduction in the severity and frequency of cramps). No reduction was reported while placebos were taken or when medication was suspended. Those who had improved while taking the drug experienced a return of symptoms within three to four weeks after the drug was discontinued. Symptoms disappeared when methocarbamol was resumed.

A mild gastric intolerance, reported by one patient, was the only known side effect. Methocarbamol was found to be non-toxic in recommended therapeutic dosages and to have no deleterious effect on normal muscle tone (Perchuk et al., 1961).

Tocopherol (Vitamin E)

Tocopherol is a fat-soluble vitamin composed of a number of fractions; the alpha fraction is the most physiologically active, containing nearly all of the active principle.

The "d" or natural form is more active than the synthetic form and the ester (acetate or succinate) insures stability. The type drug used in the treatment of systremma is d,alpha-tocopheryl acetate (Ayres & Mihan, 1969; Ayres & Mihan, 1974).

Vitamin E is important to the structural integrity and functioning of cells throughout the body. It tends to prevent or retard the peroxidation of unstable lipids in the lipoprotein membranes of cells and intracellular organelles which can result in a wide variety of pathologic changes; in addition, vitamin E facilitates oxygen use in normal metabolic processes through its catylyzing relationship with enzymes and other vitamins and minerals. While the exact mechanism is still unknown, vitamin E has been found to be essential to the proper functioning of muscles. Vitamin E deficiencies in animals resulted in muscular dystrophy and anemia (Ayres & Mihan, 1974). The use of vitamin E for certain dermatological conditions yielded the beneficial side effect of relieving muscle cramps, systremma. This led to the therapeutic prescription of alpha-tocopherol for this condition (Ayres & Mihan, 1969).

Ayres and Mihan Study. Vitamin E was administered to 125 patients with systremma; the majority of these were over 50 years old with cases of long duration, great frequency and severity. Dosages were usually 400 to 800 IU per day although nearly half were taking 300 or fewer IU per day. In 103 of the 125 cases the cramps were completely or almost

completely controlled; in 13 the treatment resulted in 75% to 95% improvement. In two cases the treatment was regarded as having little benefit; the other seven showed moderate improvement, 50% to 75%. Upon discontinuation of treatment, cramps returned but were promptly relieved when treatment was resumed. These findings were confirmed by others observing and treating nocturnal cramps with vitamin E (Ayres & Mihan, 1974).

No serious side effects were reported although certain precautions should be taken in some situations. Since vitamin E improves glycogen storage, patients taking insulin should begin tocopherol treatment at lower dosages, with a gradual increase of dosage while concurrently adjusting the insulin dosage to avoid an insulin reaction. Patients with cardiac conditions and hypertension should also begin treatment at lower dosages. Those not able to tolerate or absorb fat may take water-solubilized vitamin E; injections may be needed for those with severe intestinal malabsorption problems. Ingestion of organic iron (including iron-fortified foods) inactivates tocopherol; mineral oil or the excessive use of laxatives prevent absorption. Premarin reportedly counteracts the effectiveness of vitamin E (Ayres & Mihan, 1974).

Benadryl

Benadryl (diphenhydramine hydrochloride) has both antihistiminic and antispasmodic properties (Misischia, 1960);

the latter property suggests a suitability for the treatment of leg cramps during pregnancy since that condition precludes most stronger cramp relief treatment. Page and Page (1953) mention its use with equivocal results. Daide in 1950 reported the use of Benadryl in the treatment of cramps in the elderly (Mauer, 1961; Misischia, 1960).

Misischia conducted a study using 25 to 50 mg. of Benadryl nightly to treat systremma in 72 pregnant women. After one week 53 patients (73.6%) reported good results (complete and permanent relief); 16 (22.2%) reported fair results (partial relief or continuous need for medication for relief) and 3 (4.1%) reported little or no effect.

Reported side effects were complaints of undue morning drowsiness in two patients. A majority received some degree of sedation, enabling them to sleep better (Misischia, 1960).

Quinine

Quinine, one of the most commonly prescribed drugs for systremma, is the chief alkaloid of chinchona, the bark of the chinchona tree native to certain regions of South America. Its use for cramp relief was first described by Moss and Hermann in 1948 (Mauer, 1961). Quinine has a dual action on skeletal muscle: the drug acts by increasing the refractory period of the muscle so that the response to tetanic stimulation is diminished and it also decreases the excitability of the motor end plate, having a curare-like effect on neuromuscular transmission (occupies ACh receptor

sites) (Goodman & Gilman, 1970). Quinine has been found to be only partially effective in reducing systremma (Ayres & Mihan, 1969).

In some cases, especially in large dosages, quinine produces unpleasant side effects: tinnitis, deafness, visual loss; it can produce thrombocytopenia purpura (Perchuk et al., 1961). The use of quinine is contraindicated in cardiac patients and pregnant women due to its cardiotoxic and oxytocic effects (Perchuk, 1964). Prolonged use can result in quinine poisoning producing additional symptoms such as nausea, vomiting, abdominal pain, diarrhea, headache, fever, confusion, delirium, coma, renal damage, anemia, and abortion (Goodman & Gilman, 1970). Clearly, due to its debilitating properties, quinine is a drug to be used only after all else has failed and then only on certain populations.

Calcium Lactate

Calcium lactate and calcium gluconate are organic calcium salts often prescribed for cramp relief. Calcium is present in the A bands of the myofibrils and has a local inhibitory effect on neuromuscular irritability. Calcium ions act as a skeletal muscle relaxant so that a deficiency will cause an increased excitability of somatic nerves (Perchuk, 1964). Whether calcium ion deficiency causes cramps is not known for certain but in some cases, especially for pregnant women, increasing the calcium ion level reduces systremma. In severe cases of hypocalcemia, calcium gluconate

administered intramuscularly gives temporary relief. Studies demonstrating the effectiveness of calcium therapy have been conducted mostly with pregnant women as subjects; the details are as follows.

The Pages' Study. These authors' premise was that large quantities of milk or dicalcium phosphate (commonly used calcium supplement in pregnancy) caused a decrease in the concentration of diffusable calcium in the body and an increase in the concentration of inorganic phosphorus. One hundred and twenty obstetrics patients who complained of systremma were given one of three treatments: 1) a reduction of milk intake from over 3 pints daily to less than 1 pint daily, 2) the substitution of 2 gm. calcium lactate and vitamins for the previous supplement (dicalcium phosphate), and 3) a moderate reduction of milk intake together with 0.5 to 0.9 gm. of aluminum hydroxide gel with each meal (removes some phosphate ions from the gastrointestinal tract, permitting greater absorption of calcium). Symptoms were recorded the two weeks prior to and the two weeks following implementation of treatment. Symtoms for that same time period were recorded for a control group not receiving any cramp therapy. Significant results were found when each of the three therapies was compared to the control but the aluminum hydroxide group had the best results (Page & Page, 1953).

Blood serum analyses before and after 5 days' treatment with 0.6 gm. of aluminum hydroxide gel (at mealtime) were

conducted on ten pregnant women. In nine of ten cases there were significant differences in the diffusable calcium and inorganic phosphorus concentrations as the result of the dietary change; calcium increased while phosphorus decreased (Page & Page, 1953).

Wolff Study. This study was a demonstration of the value of a preventive dietary treatment for pregnant women using a phosphorus-free calcium salt and aluminum hydroxide gel. Rather than use subjects who had already developed systremma, Wolff administered this calcium treatment as early as the third month; cramping usually occurs from about the sixth month. Of these 120 patients only fourteen ever developed leg cramps during the pregnancy. Of the 14 nine did not take the tablets regularly as directed; all 14 ate large quantities of foods high in phosphorus. When milk intake was reduced and the supplement taken as directed, nine patients' symptoms were relieved. The incidence of cramping was compared to that of 200 patients used as controls; 51% of the controls, treated only by salicylates, rest and moderate exercise, experienced cramps at least once during the latter half of their pregnancies (Wolff, 1954). The study implies that cramps during pregnancy can be controlled and even prevented by taking the proper type of calcium supplement and restricting milk intake.

Salvatore Study. The study was conducted on 60 obstetrics patients between the ages of 21 and 38 years. Therapy

consisted of 10cc of a 10% solution (1.0 gm.) of calcium gluconate per day, for six days, slowly injected intravenously; during the following three weeks 3-5 gm. of calcium gluconate was administered orally three times per day. Symptoms were completely and permanently relieved for 36 (60%); eight patients' cramps (13.3%) were temporarily relieved and recurring cramps were not severe enough to warrant repeat therapy. In 11 cases (18.3%) calcium therapy did not cure the cramps but did offer some relief; the remaining five patients (8.4%) had severe recurrences of cramps and had to undergo a new series of treatment.

Although none of these studies reported any adverse effects, Perchuk (1964) cautioned about the use of calcium where a hyperirritable myocardium is suspected, because calcium enhances the toxicity level of digitalis.

In summary, the following results were obtained from the pharmacological studies of cramp-relieving drugs. Carisoprodol, a centrally acting muscle-relaxant and pain reliever, given in dosages of 1400 mg. or more per day was totally effective for 25% to 70% of the sample. Various side effects were experienced by 11% to 12%. Placebo groups experienced recurrence of cramps after carisoprodol was discontinued. Methocarbamol, another centrally acting muscle-relaxant, given in dosages of 2000 to 4000 mg., was totally effective for 79.1% of the sample. Side effects were experienced by 4.2%. Placebo groups experienced no cramp relief.

Tocopherol, administered in dosages of 300 to 800 IU per day was totally effective for 82.4%. No side effects were reported; no placebo group was used as control. Benadryl, an antihistamine with antispasmodic properties, in dosages of 25 to 50 mg. per day, relieved cramps completely in 73.6% of a sample of pregnant women. Sedation and drowsiness were reported by 2.8%. No placebo group was employed as a control. No controlled study of quinine was included, although the drug is frequently prescribed for systremma and manifests the most deleterious side effects of any drug used for cramp relief. Calcium therapy administered to pregnant women was 60% effective in totally relieving cramps; no side effects were reported. The Salvatore study employed no control group. Certain studies (Ayres & Mihan, 1974; Misischia, 1960; Salvatore, 1961) may be of questionable value due to the omission of placebo groups.

Disadvantages of Drug Therapy

While the presently available methods of treatment for systremma appear to provide adequate relief to the majority of the subscribers, certain questions have not been sufficiently answered. Very often when cramps are relieved by drug therapy, they recur upon discontinuation of medication. To maintain a relieved state, the drug must be taken nearly continuously, a certain disadvantage. What are the long-term effects of taking a certain drug for prolonged periods? None of the studies cited here addressed the question; no

long range investigation was attempted. Quinine's deleterious effects over extended periods of use are the only known long-term effects of any of these drugs. What is the total cost to the patient, both in money spent and in tissue damaged, two more disadvantages?

Another of the more serious drawbacks of drug therapy is the delay of effect. Carisoprodol requires at least 48 hours to be effective (Stern, 1962); methocarbamol and quinine's delay intervals are both about 72 hours (Perchuk et al., 1961). Vitamin E can take from one to two weeks before symptoms are relieved, as reported by Ayres and Mihan (1969). Benadryl's effectiveness can be noted after one week's use (Misischia, 1960); calcium gluconate has a three to six day delay of effect (Salvatore, 1961). These treatments may not take effect for two days up to two weeks after therapy was first begun, an interval of no relief to which every patient is subjected.

As if these first four drawbacks were not sufficient to warrant a search for a swifter acting, less drug-dependent therapy for systremma, a fifth disadvantage to these treatments are the side effects, both the deleterious and the merely uncomfortable. As was pointed out earlier, side effects can range from nausea to hearing loss, from rashes to insulin reaction. Furthermore, according to Goodman and Gilman (1970), treatments using methocarbamol resulted in additional side effects noted in a number of cases:

headache, anorexia, nausea, vertigo, fever, skin eruptions, and drowsiness. Up to 12% of a typical sample of cramp sufferers develop some side effect from treatment. Table 1 presents a summary of drug therapy for systremma. Clearly this situation demonstrates a need for better alternative methods for cramp relief.

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Insert Table 1 about here

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Magnetic Therapy

A possible alternative treatment for systremma was suggested by Reid (1972) in a clinical report indicating that magnetic fields have a therapeutic effect on muscle cramping. The doctor tried the magnetic therapy herself in place of quinine treatments which had become unsatisfactory. The form of treatment used was the placement of the magnet flat against the skin surface of the affected muscle; the reported result was nearly immediate cramp relief (in a matter of seconds). Thus biomagnetism appears to offer a simple, inexpensive, non-drug therapy for systremma.

Biomagnetism

In brief, biomagnetic theory proposes that the magnetic susceptibility of a substance arises from its electrons. An electron spinning around an axis behaves like a magnet. Electrons, forming a closed electron shell in an atom, are grouped in pairs of opposite spin and orbital movements.

TABLE 1

Summary of Studies of Pharmacological Therapies for Systremma

Drug	N	% Ss completely relieved	% Side effects	Side effects	Delay of effect
Carisoprodol	50	70	12	drowsiness, itching, nausea, gastro-intestinal upset, depressive reaction, skin rash	48 hrs.
	61	25	11		
Methocarbamol	24	79.1	4.2	gastric intolerance	72 hrs.
Tocopherol	125	82.4	none reported; precaution advised in some cases	-	1-2 wks.
Benadryl	72	73.6	2.8	morning drowsiness	1 wk.
Quinine	-	-	-	tinnitis, deafness, visual loss, thrombo-cytopenia purpura	72 hrs.
Calcium lactate,	120	-	none reported; 1 precaution	-	3-6 days
Calcium gluconate	60	60			

All dosages were within clinically recommended limits.

The magnetism produced by the spin of one electron is cancelled out by that of another electron spinning in the opposed direction; the substance is said to be diamagnetic. When unfilled electron shells exist, as in an ion or in compounds containing transition metal ions, the substance has a magnetic moment and is considered paramagnetic. Paramagnetic substances display a strong attraction toward magnetic fields masking the underlying diamagnetic property of very slight repulsion (Berg, 1975). The magnetic field effect on living tissue is thought to be based on the paramagnetic property of ions and ionic compounds within the tissue.

While biomagnetism, the study of biological effects of magnetic fields, is a relatively new science, a rather extensive bibliography of numerous aspects of the topic exists (e. g., Barnothy, 1964; Barnothy, 1969; Presman, 1970). Nevertheless, very little information is available concerning the effects of a magnetic field on muscle tissue. In time the situation may improve, providing information as to specific effects and mechanisms for those effects; application of this knowledge may be the therapeutic answer to systremma. Already magnetic fields are being used with increasing frequency for medical applications in other areas. Diagnostic applications include magnetocardiograms (Plonsey, 1972) and magnetoencephalograms (Cohen, 1968) which resemble electrocardiograms and electroencephalograms, respectively. Therapeutic uses include the control of tumor growth

(D'Souza, Reno, Nutini, & Cook, 1969) and the treatment of circulatory diseases (Kordyukov, 1969).

In reviewing the various treatments presently used to treat systremma, it appears that cramping is a complex interactive mechanism involving circulatory (anoxia, metabolic imbalance), neural (CNS and motor unit activity), and muscular (shortening) aspects. Only piecemeal information is available as to the effects of magnetic fields on blood circulation, nerve and muscle tissue; this evidence must then be interpreted in conjunction with the available theories of muscle cramping.

Blood circulation, the component of systremma to be examined first, is traditionally thought to be the causal mechanism of cramping by most physicians and the general public. Most home remedies (heat, massage, foot elevation) involve increasing blood circulation; cramps very often accompany cardiovascular diseases. Circulation contributes to muscle functioning in two ways: input, bringing in O_2, glycogen, needed metabolites; and output, removing lactic acid and excess metabolites. Another minor function is removal of excess ACh at the motor end plate. Failures at any of these points could result in cramping. If a magnetic effect on cramping is circulatory, it could conceivably act on the circulatory abnormalities.

Some evidence does exist to support a circulatory hypothesis. In 1969, Kordyukov reported that patients,

suffering from obliterative diseases of the blood vessels of the lower extremities, showed improved hemodynamics after exposure to a magnetic field. Toroptsev (1968) found that a 13-hr. exposure to a strong magnetic field (7000) gauss induced disturbances of hemodynamics and lymph circulation in guinea pigs, frogs, and fish. Histology of some of these animals revealed a paretic dilation of capillaries; however, after 30 days morphology of the other animals had returned to normal. Edlinskiy (1969) reported that patients with acute thrombophlebitis, exposed to a magnetic field of 200 to 400 gauss, showed a generally improved clinical picture.

Whether the magnetic effect is on the blood vessel or on the components of the blood is not known. Magnetic fields have reportedly decreased blood sugar levels (Cassiano, Carta, & Tronconi, 1967), increased Fe content and lowered Cu content in the blood (Edlinskiy, 1969), increased blood coagulation (Degen & Potashnik, 1970; Piruzyan, Rosenfel'd, Glezer, & Lomonosov, 1969), increased erythrocytes (Chachava, Charkviani, Zhgenti, Kintraya, Nishianidze, Lominadze, & Chachava, 1969), increased hemoglobin content (Ivanov-Muromskiy & Lukhachev, 1967; Likhachev, 1969), increased the number of leucocytes (Ivanov-Muromskiy & Lukhachev, 1967), and increased the number of platelets in mice by up to 28% (Barnothy & Barnothy, 1970). The relationship of these changes in blood components to cramping, if such a relationship exists, is yet unknown.

Further confusion arises when one considers the reports that magnetic fields actually decrease blood flow (Berg & Chute, 1976; Bresson and Bellosi, 1969; Likhachev, 1968). Clearly this does not easily fit the commonly accepted notion that merely increasing blood circulation relieves cramping.

Contradictory reports such as these may be evidence that blood circulation, while a possible mechanism through which a magnetic field acts, is not the sole component of muscle cramping which is thus affected. Another consideration is the muscle tissue itself. The sarcoplasmic reticulum's re-uptake of the Ca ions signals the end of a muscle contraction. Should this not occur, the contraction continues, resulting in a cramp. Magnetic fields may facilitate the re-uptake of Ca ions should this mechanism develop inconsistencies due to Ca-Mg imbalance, lactic acid buildup which interferes with Ca re-uptake, or insufficient supplies of ATP. Again this effect is merely a matter of conjecture. Bücking, Herbst, and Piontek (1974) reported that exposure of isolated skeletal muscle to a strong magnetic field (50 kilogauss) reduced the contraction of the muscle approximately 8%; the evidence indicates that magnetic effects on muscle tissue alone is probably insufficient to achieve the clinically reported cramp reductions.

A third component of muscle cramping which may be affected by magnetic fields is the nerve tissue. The nervous

system being electrical in nature could conceivably be affected by a magnetic field. The generation of an action potential depends on the permeability of the membrane to certain ions. These ion concentrations (Na^+, K^+, Cl^-, Ca^{++}) may be altered by magnetic fields. The likelihood that some nerve malfunction precipitates systremma is borne out by the existing drug therapies; most act on the nerves and the spinal reflexes. The possibility exists that magnetic fields may act on the motor unit to return it to a normal state and thus reduce the cramp.

Another proposed site of magnetic effect is the motor end plate. Young in 1969 suggested that the likely site of magnetic action is the acetylcholinesterase activity at the myoneural junction. If ACh is not removed from the receptor sites at the motor end plate, a muscle cramp may result. If an excess of ACh is the problem, then the magnet's action increasing the acetylcholinesterase level would effectively reduce the cramp.

Many speculations could be made about the possible mechanisms whereby magnetic fields could reduce systremma, but until research provides more information, determination of a site of action or a definite mechanism would remain a matter of conjecture.

One such study by Berg in 1975 investigated the effects of steady state magnetic fields on induced muscle tension in normal college age subjects, finding supportive evidence for

Reid's observations of magnetic effects on striate muscle tissue. A magnet was found to reduce muscle tension induced by cold pressor task (circulatory effect) significantly better than an iron bar which the subjects were told was also a magnet.

Statement of the Problem

Since research in this area of biomagnetism is relatively recent and since the Berg study involved muscle tension, not severe muscle cramping, this study attempted to determine if magnetic fields do reduce muscle cramping significantly more than steel bars which look like magnets and whether this reported phenomenon has a physiological or a psychological basis.

CHAPTER II

METHODS

Subjects

Twenty subjects were solicited from a private medical practice. Only one subject, a 64-year old woman, participated through the requested duration. She was selected, as were the others, to meet two criteria: she had frequent leg cramps and was in general good health for her age.

Apparatus and Materials

The commercially available General notched bar magnets No. 394-C (2 3/8" x 5/8" x 3/16") distributed by General Hardware Mfg. Co., Inc. (New York) having a magnetic field of approximately 100 gauss was used. A corresponding steel bar was made by the University of Houston Physics Department Machine Shop. The bar, having the same dimensions as and a similar physical appearance to the magnet, was used as a "dummy" magnet.

Procedure

Throughout the experiment, subject was blind as to the presence of the real magnet. The subject recorded information on the next fifteen consecutive cramps. Prior to commencing the experiment, the subject completed a pre-treatment questionnaire detailing past cramp history and treatment as well as other medical history information.

Condition I - Baseline. Subject experienced her first five cramps using no magnetic treatment, recording the following information on the provided data sheet: date, time of onset, time cramp was relieved, severity of cramp, activity prior to cramp, location of cramp, and any medication ingested in the last 24 hours. Subject was allowed to use non-magnetic means of cramp relief, if necessary. The relief mechanism used was described under "REMARKS" on the data sheet.

Condition II - Magnet Present. Subject applied magnet A to the cramped area for cramps #6-10, recording the same information as in Condition I.

Condition III - Non-magnet Present. Subject applied non-magnet B to the cramped area for cramps #11-15, recording the same information as in previous conditions. Subject was told the non-magnet was in fact a magnet but one of a differing strength from magnet A.

CHAPTER III

RESULTS

As Table 2 indicates, the mean cramp durations in Condition I (3.8 min.), Condition II (4.0 min.), and Condition III (3.0 min.) do not vary significantly for this S. The mean cramp severity (on a 5-point scale with 5 being the most severe) steadily and progressively increased from 2.8 in Condition I to 3.6 in Condition II to 4.4 in Condition III.

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Insert Table 2 about here

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In addition the cramp frequency increased from approximately five cramps per month in Condition I to ten cramps per month in Conditions II and III. See Figure 1.

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Insert Figure 1 about here

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The following information was obtained from the pre-treatment questionnaire.

Case History. A woman of 64 with a past history of migrating thrombophlebitis had the complaint of severe, exhausting and incapacitating nocturnal cramps in both legs and feet. These cramps have persisted for the past 10 to 15 years and have progressively become more severe. From 1965

TABLE 2

Summary of Data for Subject #1

	$\bar{x}$ Duration (min.)	$\bar{x}$ Severity (1 to 5 scale)
Condition I - Baseline	3.8	2.8
Condition II - Magnet Present	4.0	3.6
Condition III - Non-magnet Present	3.0	4.4

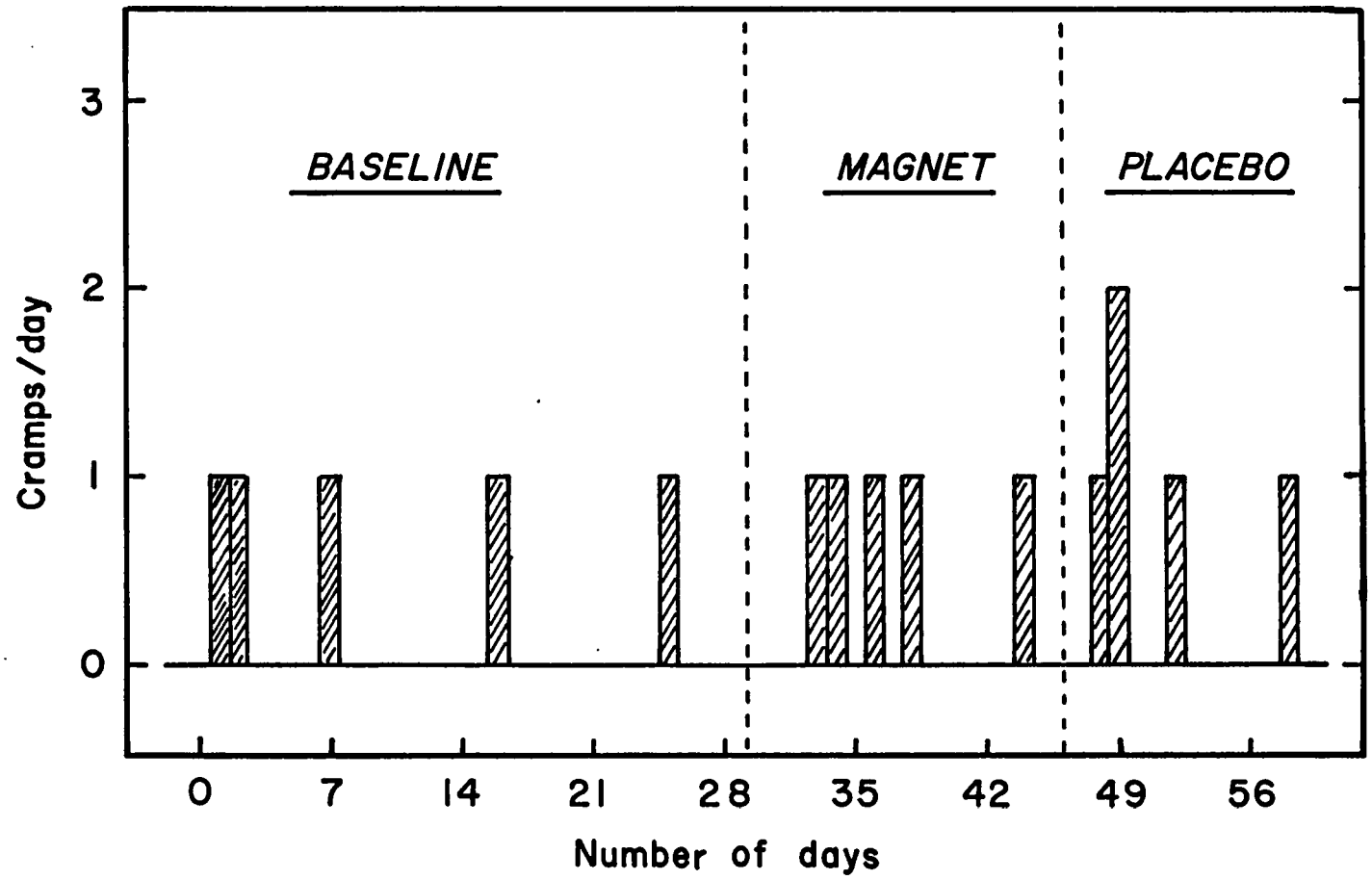

Fig. 1. Cramp Frequency for Subject #1

through 1970 quinine had been taken to help relieve the cramps, but the medication was not completely effective; heat, massage, hot baths, forceful breakdown of the contraction were also tried, all with limited success. Since January 1, 1971, quinine has been discontinued and relief has been derived from magnetic therapy. The cramps have been reported to have lessened both in severity and frequency; relief has been reported to occur almost immediately after the application of the magnet on the cramped muscle.

CHAPTER IV

DISCUSSION

While it was expected that a magnetic field would be effective in reducing cramp time and intensity, this hypothesis was not supported by the results obtained from this study. Differences in cramp duration were marginal enough to attribute these to random error; the null hypothesis that the reported therapeutic effects of magnetic fields to relieve muscle cramps is due to a placebo effect could not be rejected.

A critical review of the design would seem to be a logical starting point of investigation of the problems presented by the study. In an experiment such as this, one would ordinarily control for an order effect of the magnetic treatment and for an expectancy effect on the part of the subject by counterbalancing. Using a latin-square design, one S would recieve the magnetic treatment first and placebo, second, while another S would receive the treatments in reverse order. Another suggestion would be to use a reversal of treatment, ABA for some Ss and BAB for others (A = magnet, B = placebo).

Under the assumption that a magnetic field effectively reduces muscle cramps, the following events may be considered. In the AB component of the latin-square design, the

possibility that both the magnetic treatment and the placebo would show an effect beyond the baseline measure may be the result of expectancy, long-term magnetic effect, or an interaction of both. Using the BA order, especially on subjects who believe the magnet's effectiveness, one would anticipate an expectancy effect but no order effect. On sceptics one would expect little subject bias toward a reduction of cramping in the B condition but a reduction would be expected for both sceptics and believers due to the magnetic field in the A condition.

If the reversal was used, reduction may appear in all conditions of ABA due to a possible long-term magnetic effect but will appear only in A and the second B of BAB. Again groups of sceptics and believers may be employed. The long-term magnetic effects could be investigated in the AB or the ABA components by measuring the period of time required for cramp frequency, duration, and intensity in B to return to baseline levels. If the magnetic effect of reduction with no long-term effects does exist, the reversal offers more information than the latin-square design. A treatment effect would be shown that would discontinue as the placebo was used but which would return when the magnetic field again was applied. In most of the other situations presented, reversal offers little more advantage and does take more time. In addition the ethics of removing an effective treatment merely to determine if painful symptoms

reoccur may be in question.

As was mentioned above, comparisons of sceptics (non-users of magnets) and believers (users) would further validate the experimental hypothesis. Another possibility to consider would be the comparison of groups whose muscle cramps are associated with specific diseases or conditions which may yield clues as to the sites of magnetic action on systremma but which would add little to the prior establishment of a reduction of cramping due to magnetic effect.

If several of the aforementioned comparisons were used, greater numbers of subjects would obviously be required. While increasing the N may facilitate achieving results of statistical significance, the data required for confirmation of a magnetic effect, due to the general reserve of the scientific community concerning unorthodox methods, would have to be of an observable magnitude. While several subjects would be required, many more subjects would only serve as further confirmation, not as a statistical necessity.

Magnetic effects over time is another area of experimental consideration. A long-term study of magnetic effects on cramping over several years may be needed to determine if any deleterious effects exist and what, if any, changes occur in the nature of the systremma.

In reality all these factors could not be included in the study due to the constraints involved. First, certain ethical considerations needed to be observed. Under the

premise that magnets do relieve cramps, the subjects would be denied relief under baseline and placebo treatment conditions; in the reversal (BAB) or in the latin-square (BA) designs, subjects would be required to undergo no treatment during baseline and during at least one placebo condition, a minimum of 10 cramps. Asking a subject to endure such discomfort would be unethical.

Another reality to be confronted was that, in spite of the 50% incidence of cramping reported, finding a sufficient number of subjects who regularly and frequently have leg cramps would be difficult. Only a limited number of subjects would be available and willing to participate (without pay) in such a study. Formulating and matching groups under these conditions would be formidable.

However, in the interval since this study was designed, information from the Norris (1957) study suggested that subjects who have had cramps at one time or another could voluntarily induce muscle cramps by passively shortening a specific muscle and then producing a maximum voluntary effort until the cramp developed. The voluntary effort usually lasted less than one minute before the involuntary cramp developed; the muscles used (for convenience) were the biceps and the rectus femoris muscles. Induced cramps were found to be no different electromyographically than spontaneous cramps. A population sample having this ability to voluntarily induce cramps would facilitate the study of magnetic

effects on systremma; the experimenter would no longer be dependent on the cramps occurring spontaneously or on the subjects' self report. Again finding this population would necessitate a search as Norris (1957) found that only 18% to 26% of the subjects tested were able to voluntarily induce a cramp.

Time was another limiting factor; the study had a specific deadline to be met, thus an upper limit of 2 mos. was placed on the duration of the experiment. Due to the expectancy on the part of the experimenter (based on clinical reports) of obtaining robust results during the magnet treatment condition (anything less would not be acceptable) and due to the clinical reports that no long-term magnetic effect had been observed, the AB within-subjects design was adopted. This design would in fact favor a placebo effect, so that if magnetic effects were shown to result in significantly shorter cramp durations and lessened severities, the null hypothesis supporting a placebo effect could then be rejected.

The data presented some interesting problems. Not only were cramp durations for all three conditions very similar but the shortest mean duration occurred during the placebo condition rather than during the magnet condition as expected. Another curious point was that as time passed during these three conditions, the mean cramp severities increased steadily and progressively from 2.8 to 3.6 to 4.4 (on a

5-point scale where 5 is most severe). Cramp frequency increased as well over the experimental period. The baseline rate was approximately five cramps per month; in the next month, during Conditions II and III, the rate climbed to 10 cramps per month. Certain intervening events occurred about the time that Condition II began. The subject's children and grandchildren arrived for an extended visit, no doubt changing her lifestyle for that month. Another confounding factor is that treatment under Condition III did not exclude magnets; several cramps were so severe that the S applied her own magnets in addition to the placebo magnet. These reasons may help to explain why the experimental findings demonstrate effects nearly opposite to the expected ones.

A closer inspection of the data suggested that perhaps by looking at the use of alternate means of cramp relief (denoted under "REMARKS" on the data sheet) as another dependent variable (DV), the experimenter could determine which treatments were ineffectual. The effectiveness of the treatment could be determined by whether or not other relief was sought. In this case during baseline, other relief was sought 80% of the time. During Condition II alternate methods may have been used for 20% or 40% of the cramps; the comments are not clear on the data sheet. In Condition III again it is not clear; other magnets were used for at least one cramp (20%) and possibly for others. If the use of alternate methods of cramp relief is to be used as another DV,

a more clear-cut designation on the data sheet must be devised to avoid this kind of ambiguity.

While evidence for a physiological effect of magnetic fields on systremma appears to exist, the psychological effect of the use of magnets cannot be discounted. In spite of the confounding data, the subject continues to use magnets for therapeutic relief, believing them to be effective. Further research involving a large sample size will be necessary before the issue of whether a strong psychological factor influencing cramp relief by magnetic fields exists or not, can be resolved.

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APPENDIX A

Subject Information and Instructions

While muscle cramps of unknown origins have reportedly been relieved by the strategic placement of a magnet or magnets on the site of the cramp, the mechanism for this relief of discomfort is not known. Dr. Douglas Chute and I are conducting a research project at the University of Houston investigating the nature of the magnet's effect on cramps. Your participation in this experiment is much appreciated and you will have the satisfaction of knowing you contributed in the effort to reduce the discomfort caused by muscular cramping.

In joining us in this research, you will be asked to do the following:

(1) Keep a record of the next 5 cramps (without magnet).

(2) Keep a record of cramps 6-10, treated with Magnet A.

(3) Keep a record of cramps 11-15, treated with Magnet B.

(4) Promptly return the record sheet and magnets. (These magnets are reserved for the next subject; however, if you would like our assistance in obtaining your own magnet, we would be glad to oblige.)

The experiment will only last the time period it takes to have 15 cramps or 2 mos., whichever is sooner. It will only take a moment to jot down the requested information at each occurrence. You will need easy access to a watch or clock at these times and you must have your record sheet

handy - carry it in your pocket or purse; leave it at your bedside at night. Before we begin the experiment, you will also need to complete a pre-treatment questionnaire and sign a consent form. Please read them carefully and if you should have any questions, please feel free to ask them. You may discontinue this procedure if at any time you no longer wish to participate. In that case, contact Dr. _____ and return all the materials to him. The following information is a more detailed explanation of the procedure.

KEEPING THE RECORD

In this study you are being asked to keep a record of your next 15 cramps. You are to print:

(1) date

(2) time of onset of the cramp

(3) time the cramp was relieved

(4) severity of the cramp

(5) activity in which you were involved just prior to the cramp (e. g., sleeping, walking, etc.)

(6) location of the cramp

(7) medication taken in the last 24 hrs. (include the time)

(8) remarks (non-magnetic cramp relief techniques used)

A sample has been done for you on the data sheet.

SEVERITY OF CRAMPS

The severity of the cramp is to be rated on a scale of 1 to 5 with 1 being just a mild cramp and 5 being a severe

cramp.

TREATMENT OF CRAMPS

If you are already using magnets to relieve cramps, you must put your own magnets away for the duration of this experiment. Throughout this experiment use only our magnets and in the order described. This procedure is critical to the experiment and must be strictly followed.

We are needing a baseline of the frequency and nature of your muscle cramps, that is, how often and how severely they normally occur. In order to obtain knowledge of this, we are asking you to record the first 5 cramps without the use of the magnet. You may use any other non-magnetic methods of relief, such as massage, heat, elevation; if you should use such techniques, please note them on the cramp record under "REMARKS". If you need more space, write on the back indicating the cramp number involved.

For the remaining 10 cramps, use the magnets as follows: For cramps 6-10 use Magnet A. Leave the other magnet in its box in the packet until after the 10th cramp. Open the magnet box carefully; use every care not to drop the magnet. Use only Magnet A; have it readily available (in its box) but in a safe, protected place away from metal objects because these magnets are very sensitive. For cramps 11-15 use Magnet B. Replace Magnet A (in its box) in the packet; take Magnet B (in its box) out of the packet heeding the same precautions as for Magnet A.

USE OF MAGNET

At the onset of the cramp, place the magnet directly on the affected muscle. At night keep the magnet in a convenient place where you have ready access to it, for example, on the nightstand.

STORAGE OF MAGNET

When the magnet is not in use, it must remain in the case. These are extremely sensitive magnets whose use must be restricted to treating your muscle cramps. This part of the experiment is extremely important; the results could be adversely affected if this caution is not heeded.

You may also have been wondering why there are two different magnets. We are also investigating whether the strengths of the magnets used affects their property to relieve cramps. Magnets A and B are of two different strengths. Our belief is that the particular strength of the magnet within limits has little bearing on whether it will relieve the cramp. We believe that so long as a magnet has any "pull" it will be sufficient to bring relief. (The magnetic strength at these low levels can only be successfully measured by sensitive detecting devices.)

COMPLETING THE EXPERIMENT

After the record has been completed with all 15 cramps recorded or when 2 mos. have passed, please promptly return the record sheet and the two magnets as other subjects will be needing to use the magnets. Place the magnets (already

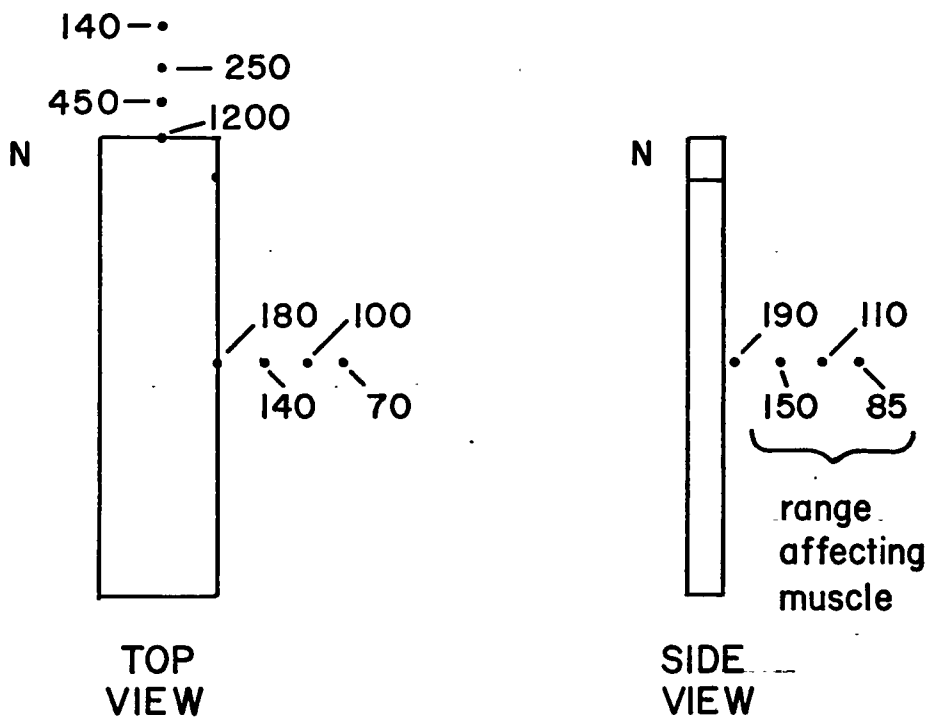
in their respective boxes) back into the corresponding small envelopes. Then put the smaller envelopes into the larger envelope for mailing back to Dr. ____. Thank you for your willing participation and your patient cooperation in helping us to collect the cramp data. You can feel proud of your contribution to the search for relief of the discomfort of muscle cramping.

Mary M. Mauldin

APPENDIX B

Full Scale Diagram of Magnet A

Denoting Field Strength in Gauss


140
250
450
1200
N
180
100
140
70
TOP
VIEW
N
190
110
150
85
range
affecting
muscle
SIDE
VIEW

MAGNET A

APPENDIX C

Cramp Data - Subject #1

Condition I - Baseline

Cramp #	1	2	3	4	5
Date	4-7-76	4-8-76	4-13-76	4-21-76	5-1-76
Time Onset	3:52 am	3:58 am	3:29 am	8:34 pm	9:10 pm
Time Cramp Relieved	3:54 am	3:59 am	3:32 am	8:38 pm	9:19 pm
Severity (1-5)	2	1	2	4	5
Activity Prior to Cramp	sleeping	sleeping	sleeping	sitting	sitting
Location of Cramp	L foot, back calf	L inside thigh	L calf, front & back	L thigh, inside & back	L foot, back calf
Medication Taken in Last 24 Hrs.	none	none	none	none	none
Remarks	standing on foot & massage	standing, massage	boot distribution, standing, massage	massage, forceful extension	none

Condition II - Magnet Present

Cramp #	6	7	8	9	10
Date	5-9-76	5-10-76	5-12-76	5-14-76	5-20-76
Time Onset	9:44 pm	6:35 am	11:15 pm	9:45 pm	5:03 am
Time Cramp Relieved	9:47 pm	6:36 am	11:22 pm	9:50 pm	5:07 am
Severity (1-5)	3	3	5	2	5
Activity Prior to Cramp	sitting	sleeping	lying down	sitting	lying down
Location of Cramp	L back thigh	R back calf	L calf, front & back, foot	L back calf	R back calf & foot
Medication Taken in Last 24 Hrs.	none	none	none	none	aspirin 10 gr.
Remarks	-	-	-	cramp continued so switched to my own magnet; relieved in 10 min.	

Condition III - Non-Magnet Present

Cramp #	11	12	13	14	15
Date	5-24-76	5-25-76	5-25-76	5-28-76	6-3-76
Time Onset	3:27 am	3:05 am	5:27 am	9:45 pm	11:41 pm
Time Cramp Relieved	3:28 am	3:10 am	5:28 am	9:49 pm	11:45 pm
Severity (1-5)	4	4	5+	5++	4
Activity Prior to Cramp	lying down	lying down	lying down	sitting	lying down
Location of Cramp	R back calf	L front calf	R foot	R inside thigh	L back calf
Medication Taken in Last 24 Hrs.	none	none	none	none	none
Remarks	twice relieved	twice relieved		so severe several magnets used intermittent relaxation & cramping at regular intervals of 10-15 seconds	