THE EFFECTS OF VISUAL AND AUDITORY FEEDBACK ON THE ABILITY TO CONTROL A SPASTIC MUSCLE

by

Anne Scott

B.Sc.(PT), University of Western Ontario, 1971

A THESIS SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF SCIENCE (KINESIOLOGY)

in the Department

of

Kinesiology

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SIMON FRASER UNIVERSITY

January, 1973.

APPROVAL

NAME: Anne Scott

DEGREE: Master of Science (Kinesiology)

TITLE OF THESIS: The Effects of Visual and Auditory Feedback on the Ability to Control a Spastic Muscle.

EXAMINING COMMITTEE:

Dr. E. W. Banister. Senior Supervisor

Arthur E. Chapman

John M. Montgomery

Dr. A. C. Pinkerton

Louise McGregor. External Examiner.
Lecturer, University of British Columbia.

Date Approved: [illegible]

ABSTRACT

Clinical observations have suggested that visual and auditory feedback can be used to improve the functional capabilities of a hemiplegic patient. Neurophysiologists postulate that the improvement occurs through re-integration of the disturbed sensory and motor pathways. Other investigators have reported an improvement in functional abilities of hemiplegic patients following local cooling of the spastic limb.

This study, conducted on five hemiplegic patients, investigated the effects of visual and auditory feedback on the ability to produce and control a specific force (30% of MVC) at ambient temperature and after local cooling. Changes in muscular efficiency before and after cooling were also examined.

A strain-gauge dynamometer, used in conjunction with the electromyogram, provided the visual and auditory feedback. Data was collected on magnetic tape in analog form and converted into digital values by computer. The results of each subject were treated individually.

The ability to produce and control a force was determined by comparison of the variability of each attempt on each trial, and the mean response on each trial. The effect of temperature on muscular efficiency was considered

in terms of the relationship between the integrated electromyogram and force.

It was concluded that with practice, hemiplegic patients can learn to produce and control an isometric force in a situation where visual feedback is available. Local cooling of the spastic muscle did not significantly alter the level of control. Changes in the electromyogram after the muscle had been cooled prevented a quantified assessment of the changes which may have occurred in the functional state of the muscle.

ACKNOWLEDGEMENTS

The opportunity to conduct this clinical research was made possible by the co-operation and kindness of the staff and patients at the G.F.Strong Rehabilitation Centre. The author wishes to express appreciation to the Medical Director, Dr. A.C.Pinkerton and the Chief Physiotherapist, Miss Brenda Jones,for their interest and encouragement. Also to the five patients who acted as willing subjects for the experiment.

Extraction of the pertinent facts from the clinical information required the help of many people. Grateful thanks are extended to Rob Maskell and John Montgomery for their assistance with the computer programmes; to Dr.E.W. Banister for his cheerful optimism and overall guidance; and to Shirley Laing for the excellent preparation of the manuscript.

Expecial thanks are extended to Arthur Chapman who generously loaned, transported and assembled his equipment for my use. His constant guidance and counselling through all the stages of this study helped to reduce the obstacles to manageable proportions.

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CHAPTER I

INTRODUCTION

This study investigates the effects of visual and auditory feedback on the ability of five hemiplegic patients to control muscular force at both ambient temperature and after local cooling of the muscle.

A variety of methods have been developed to reduce spasticity and improve the functional abilities of the hemiplegic patient. These include facilitation and inhibition, patterning, local cooling of a limb, and visual and auditory feedback. These techniques are consistent with performance of voluntary movement.

Voluntary Movement

The performance and patterning of voluntary movement is the end product of sensory, emotional and thought processes (Schade, 1970), and every motor response must have an adequate stimulus (Denny-Brown, 1960). The complex interneural connections required for the normal control of voluntary movement are presently only partially understood. Traditional views which ascribe control of a specific motor function to an isolated area of the brain have been modified to account for the observed flexibility

manifest by the central nervous system (CNS) in normal and pathological conditions (Harris, 1971; Luria, 1966; Ruch, 1965). Current concepts of neuromuscular integration depict the CNS as a series of neuronal circuits interacting to form a functional entity (Schade, 1970; Harris, 1969; Paillard, 1960). As stated by Denny-Brown (1960):

> "A segmental reflex may be perfectly performed in terms of the segmental structures it serves, but for its integration into behaviour we have to examine the higher and higher levels of neural activity."

A variety of conditions arising from diseased states or traumatic injury interfere with the functional integrity of the sensory and motor pathways. One of these conditions is hemiplegia.

Hemiplegia

When the sensory-motor pathways are interrupted or disturbed there will be some disturbance of voluntary movement. The degree of impairment will depend both on the site of the lesion and the amount of tissue destroyed. A cerebral vascular accident (CVA), or head injury, which affects one cerebral hemisphere, may result in disturbance of voluntary movement and alteration of muscle tone on the contra-lateral side of the body. Clinically this condition is known as hemiplegia.

Barriers to the recovery of functional ability in the hemiplegic patient include: the severity of the

insult to the sensory and motor pathways, pre-trauma personality, age of patient, previous deficiencies in the sensory feedback mechanisms such as decreased visual or auditory acuity, and the presence of continuing disease (Hurwitz and Adams, 1972). In the hemiplegic patient the usefulness of residual movement may be hampered by disturbances in balance, muscular weakness and/or spasticity (Gersten, 1967).

Spasticity

Spasticity is a clinical term used to describe a muscle that shows increased resistance to passive stretch, hyperreflexia, and clonus (Bard, 1968). Neurologically this disturbance arises from the imbalance of excitatory and inhibitory impulses acting on the stretch reflex (Campbell, Dickinson and Slater, 1968). In the hemiplegic patient the principal muscle groups displaying spasticity are the anti-gravity muscles. These are the extensors of the lower limb and flexors of the upper limb. The presence of spasticity in some muscle groups restricts the opposing muscles from acting throughout their full range of movement which results in disruption of normal patterning and performance of movement.

Due to the widespread dysfunction and consequent distressing overt behaviour which is produced, a great deal of effort has been made to design successful methods of rehabilitation.

Rehabilitation Methods

Rehabilitation of a hemiplegic patient implies restoring as much functional ability as possible. To achieve this aim spasticity has to be reduced to allow re-training of voluntary movement (Dimsdale, 1964) and numerous techniques have been employed to this end.

Facilitation and inhibition. Rehabilitative personnel have recognised the importance of both sensory-motor integration and the concept of plasticity of the CNS in efforts to develop methods to overcome the deficits of a hemiplegic patient. Repetition of appropriate sensory information followed by purposeful motor output forms the basis of many techniques, including: proprioceptive neuro-muscular facilitation, reflex inhibiting patterns, the Rood method, and the Brunnstrom method. Examination of these treatment procedures reveals that the main emphasis is placed on the sensory input from the proprioceptive and tactile receptors in the muscles, tendons, joints, and skin. A combination of facilitation and inhibition of these sensory pathways decreases spasticity and allows purposeful voluntary movements. There are differences in the techniques employed and the philosophies supporting these methods which are discussed later in the review of literature.

The sensory input from the skin and muscle spindle can also be altered by decreasing the temperature of the limb.

Local cooling. The observed effect of local cooling on the reduction of spasticity and improvement in the functional ability of hemiplegic patients (Knutsson, 1970a and b; Hedenberg, 1970) has led to the inclusion of this modality in a number of treatment regimes. Cold anesthesia and raising the threshold of the muscle spindle have been suggested by a number of authors as possible mechanisms responsible for the reduction in spasticity (Hartviksen, 1962; Mead and Knott, 1966).

Recent work by Mecomber and Herman (1971) has added a new dimension to the applicability of local cooling. These authors utilized the relationship between the integrated electromyogram (IEMG) and the force of an isometric contraction (as described by DeVries, 1968). The results presented by Mecomber and Herman (1971) indicated that cooling a hemiplegic limb reduced the efficiency of muscular action when a voluntary movement was performed.

If this impairment of efficiency does occur the use of local cooling prior to an exercise programme requires careful evaluation.

Clinical workers have shown sporadic interest in exploiting the visual and auditory sensory information to achieve integration of the sensory-motor systems.

Visual and auditory input. Convincing demonstrations of the ability of normal subjects to exert control over spinal motoneurons using visual and auditory feedback (Basmajian,

1963, 1972) suggested that these sources of information could be of considerable importance in re-integrating disturbed sensory-motor pathways.

Electromyography has been used to provide visual and auditory feedback to patients with disturbed muscular function in a variety of disorders. The technique has been used to encourage muscular contraction (Marinacci and Horande, 1960) or to suppress unwanted muscular activity (Hardyck, Petrinovich and Ellsworth, 1966; Jacobs and Felton, 1969). Work by Andrews (1964) has shown that hemiplegic patients can obtain some measure of control over spastic muscle groups by selectively learning to activate a specific muscle. Harris (1970) noted that the information from the electromyogram could be used to increase functional abilities of hemiplegic patients.

Visual feedback of muscular activity can also be obtained by displaying the electrical signal from a strain-gauge dynamometer. While dynamometers have been used extensively to assess muscular performance in both normal (Clarke, Hellon and Lind, 1958; DeVries, 1968) and neurological (Lenman, 1959) conditions the application of this form of visual feedback in a therapeutic setting has not been investigated.

Limitations

When conducting research in a clinical setting a number of limitations have to be observed. Ethical

responsibilities have to be evaluated even more rigorously than in a laboratory. The desire of the researcher to reduce the variables to a minimum must not be allowed to jeopardize the health or rehabilitative prospects of the patient.

The subjects selected for this study were attending the G. F. Strong Rehabilitation Centre, Vancouver, British Columbia. A thorough review of both In- and Out-patient caseloads produced five patients who fulfilled necessary criteria of clinical evidence of spasticity, presence of voluntary movement, and sufficient comprehension to understand the requirements of the experiment. Each patient was fully informed of the nature of the experiment and both his and his physicians consent for his participation in the training and testing session was obtained.

The duration of the experiment was determined by the length of patient stay in the Centre and was limited to four weeks. During this time the patients maintained their regular rehabilitative programme with the exception of local cooling to the upper limb. As the group was so small a control group could not be established.

The use of an intramuscular thermoprobe to obtain true muscle temperature was rejected. Repeated puncture sites could provide a possible source of infection and pose a potential health hazard. Known literature sources were used to estimate the length of the cooling period required to

lower the mean internal muscle temperature.

Purpose of Study

Effective treatment procedures have been devised to improve the functional abilities of hemiplegic patients by manipulating the proprioceptive and tactile sensory input.

Local cooling has been employed to reduce spasticity. The question has been raised whether this form of treatment also reduces the efficiency of a muscle.

Visual and auditory feedback has been proposed as an alternate source of sensory input to re-train the functional abilities of the hemiplegic patient.

If feedback from visual and auditory sources can supplement the proprioceptive information an additional method of treatment would be available to rehabilitative personnel.

The present study investigates:

1. the degree of control of an isometric contraction that can be achieved by a hemiplegic patient using visual and auditory feedback.
2. the effect of local cooling on the degree of control achieved.
3. whether a training programme improves this level of control.
4. whether the functional state of a muscle does alter after cooling.

CHAPTER II

REVIEW OF LITERATURE

In the investigation of new rehabilitative methods it is necessary to reinforce empirical observations and opinions with knowledge gained from experimental work in physiology and the neurosciences.

The concepts of neuromuscular integration and the role of the muscle spindle are considered by Matthews (1971) and Schade (1970) to be an essential prelude to understanding the derangements of function that occur in spastic states.

Muscle Spindle

Muscle spindles are highly organised sense organs which lie in parallel to the extrafusal muscle fibres. The number of contractile fibres contained in a human muscle spindle ranges from two to fourteen (Swash and Fox, 1972). Two types of fibres have been identified: nuclear chain and nuclear bag.

Spindles are innervated by both sensory and motor nerves. The sensory nerves are divided into two categories: fast conducting primary sensory afferents which arise from both the nuclear chain and nuclear bag fibres, and smaller diameter secondary afferents which arise predominantly from the nuclear chain fibres (Matthews, 1971). The principle

motor axons originate from small cells, called gamma motoneurons, located in the anterior horn of the spinal cord. Matthews (1971) presented evidence that the nuclear chain and nuclear bag fibres are innervated separately. The existence of a third motor supply has been confirmed by Swash and Fox (1972). Collateral branches from the axon supplying the extrafusal fibres are found to terminate within the muscle spindle. The functional significance of this additional innervation has not yet been elucidated.

The role of the spindle in the stretch reflex mechanism is well established. The spindle lies parallel with the extrafusal fibres. When the muscle is stretched the spindle is distorted and an electrical potential is created in the non-contractile portion of the intrafusal fibres (Harris, 1969). This potential represents an impulse which is transmitted monosynaptically to the alpha motoneuron of the main muscle by the primary sensory afferents which enter the spinal cord through the dorsal root ganglion. The resulting twitch contraction of the extrafusal fibres removes the distortion from the spindle. This reflex mechanism enables the muscle to adjust automatically to changes in load without cortical involvement (Merton, 1972).

The sensitivity of the spindle can be 'biased' by the gamma motoneurons which are under the influence of the higher centres of the CNS. When the intrafusal fibres contract and deform the spindle the amplitude of the potential is proportional to the degree of distortion: this

alters the firing threshold of the spindle. Thus smaller external forces are required to initiate an impulse with partial tension on the spindle (Harris, 1969).

The role of the muscle spindle summarised by Werner, (1972) indicated that it regulates and controls movement and posture on a sub-cortical level by monitoring static and dynamic activity. The 'biasing' effect of the gamma motoneuron allows self-regulation of its sensitivity. The influence of many supraspinal centres on the stretch reflex is mediated through the muscle spindle.

Supraspinal influences, which set the level of excitability of the final common pathway, result from integration of sensory information from both the internal and external environment (Schade, 1970). A diffuse network of neurons located in the brain stem, called the reticular formation, is considered to be the main integrating centre (Harris, 1969; Moore, 1968) and all sensory and motor impulses are relayed through this area.

Armed with this basic knowledge of the muscle spindle and its importance in the performance of normal movement patterns, the functional changes observed in spasticity and methods designed to overcome the effects of these changes can be more readily understood.

Spasticity

Physiological definitions of spasticity vary with the orientation of the researcher. In a comprehensive

review of the literature Roasenda and Ellwood (1961) said "spasticity defied definition in the purely physiologic and technical sense". Levine, Kabat, Knott, and Voss (1954) described spasticity as an exaggeration of proprioceptive reflex function due to the absence of some normal factors which condition such functions. The exaggerated stretch reflex seen in spastic muscles was regarded by Herman (1962) as due to abnormal gamma efferent discharge on the muscle spindle. Rushworth (1964) pointed out that even at segmental level stretch reflexes have a very complex organisation, influenced by higher centres as well as segmental in-put: spasticity occurs when the stretch reflex is released from descending inhibition. Moore (1968) explained this 'release phenomenon' by tracing the development sequence of the CNS. As the nervous system develops the early reflexes are excitatory. Inhibitory influences are superimposed as the organism matures and a balance is attained. Disturbance of the mature CNS upsets the balance and releases the older excitatory reflexes.

Stolov (1966) and Herman (1970a) stressed the importance of non-reflex phenomena in hypertonia. In a detailed examination of the clinical physiologic aspects of the myotactic reflex in hemiplegic patients Herman (1970a) demonstrated the role played by the visco-elastic, plastic, and contractile properties of muscle in the total picture of altered reflex activity.

The functional basis for spasticity, as postulated

by Schade (1970), may be due to disturbance of the supraspinal pathways descending the spinal cord in the lateral column. Functionally two systems have been identified. The ventral system conveys excitatory impulses to the extensor mechanisms and inhibitory impulses to the flexor groups. The lateral system, composed of corticospinal, rubrospinal, and medulloreticulospinal tracts, inhibits the extensors and excites the flexors. Disturbance of the lateral system will cause a relative increase in the effects of the ventral system.

In summary it has been hypothesised that the manifestations of spasticity are caused by gamma bias on the muscle spindle due to disturbance of the supraspinal influences.

Evaluation of Spasticity

Attempts to measure spasticity have been frustrated by the elusive nature of the condition. As noted by Levine et al. (1954) spasticity is subject to autogenous neurologic and environmental influences. Any manoeuvre that is applied to the limb creates a response that could alter the degree of spasticity. Roasenda and Ellwood (1961) enumerated the devices used to measure spasticity, noting that a satisfactory method had not emerged. Sophisticated equipment has been developed by a number of researchers to measure the resistance of a limb to passive stretch. An electromechanical system developed by Webster (1964) included a programmed test movement and automatic digital printout.

However Webster stated that the disadvantages of the machine were its complexity, the unphysiologic testing position and its limitations regarding speed. The rotational joint apparatus (RJA) used by Herman (1970a) and Mecomber and Herman (1971) produced ramp and linear stretch of the calf muscles while driving the joint at various speeds. The torque generated at the ankle joint and the angular displacement of the foot was measured to obtain Length/tension curves. An electronic system used by Norton, Bomze and Chaplin (1972) moved the limb segment around a joint axis in a programmed manner and monitored the forces exerted by the subject in response to movement.

The electromyogram (EMG) has been used extensively to observe the changes which occur when a spastic limb is manipulated passively (Herman, 1970a; Long, Krysztofiak, Zamir, Lane and Koehler, 1968; Miglietta, 1964). Qualitative EMG was used by Hirschberg and Nathanson (1952) to identify the sequential recruitment of muscle groups in patients with a spastic gait. The value of EMG as an accessory tool to quantify spasticity in isometric or isotonic contractions is less well defined. Possibly the elusive nature of the condition has prevented detailed examination of this parameter.

Following the investigation of a variety of neurological and muscular diseases Lenman (1959) suggested that the ratio IEMG/muscular tension could be considered a measure of neuromuscular efficiency. The slope obtained from patients

with muscle weakness resulting from neurological diseases was within the normal range; whereas patients with primary muscular disease produced a steeper slope, indicating decreased neuromuscular efficiency. Mecomber and Herman (1971) suggested that the relationship between IEMG and tension represented a quantified value of muscular efficiency. After testing four hemiplegic patients under varying temperature conditions these authors reported that the efficiency of muscular effort was decreased.

The concept of expressing the relationship between IEMG and force as a determinant of the functional state of a muscle, proposed by Fischer and Merhautova (1961), was investigated in depth by DeVries (1968) who designated the slope of the line as 'efficiency of electrical activity'. The advantages of this measure over the conventional strength testing by cable tensiometer include the use of sub-maximal loads and the abolition of psychological factors.

If this method of quantification can be applied to determine the functional state of a spastic muscle comparisons of different therapeutic procedures designed to lessen spasticity could be made.

Rehabilitative Procedures Involving the Total Person

The problem of improving the functional capabilities of the hemiplegic patient has led to the development of a variety of therapeutic techniques. In a review of the

current trends in the treatment of neuromuscular disorders Brunton and Hunt (1969) listed the following techniques:

1. The Bobath reflex-inhibiting patterns.
2. Proprioceptive neuromuscular facilitation.
3. Rood's techniques of sensory stimulation (using ice and brushing).

These methods involve the utilization of total body movements to restore the functional ability of the patient.

Bobath (1969) interpreted the functional disability of a hemiplegic patient as follows:

> Abnormal patterning of muscles in posture and movement combined with abnormal qualities and distribution of postural tone.

The treatment devised by Bobath makes use of reflex-inhibiting patterns of movement which inhibit the abnormal patterns and facilitate more normal movements.

Proprioceptive neuromuscular facilitation (PNF) developed by Knott (1967), applies stretch, resistance, assistance and positioning to facilitate or inhibit a motor response. Knott proposed that the bombardment of a synaptic connection with impulses increases the excitability of that motoneuron. This can lead to more effective functioning. Application of PNF to spastic limbs uses the concept of reciprocal inhibition. Activation of the antagonists causes relaxation of the spastic agonists.

The Rood technique, as described by Goff, (1969) and Stockmeyer (1967), is based on applying the appropriate sensory stimulation to obtain a more normal movement or postural reaction. Cutaneous stimulation (brushing and

icing) is used to elicit the simple postural reflexes which are necessary for the development of the more complex reflexes. Working through the developmental learning patterns the effective functioning of a patient can be improved.

The treatment of the hemiplegic patient advocated by Brunnstrom (1964, 1966) is founded on the observation that all patients go through sequential recovery stages beginning with flaccidity and proceeding through a number of spastic stages, to diminishing spasticity. Treatment requires accurate knowledge of the status of the patient in this recovery sequence combined with movement patterns orientated to help the patient reach the next recovery stage.

Empirical observation indicates that these procedures are effective in rehabilitating many hemiplegic patients. However, there are some hemiplegic patients whose functional capabilities are severely limited by gross residual spasticity. For this group of patients treatment methods have been designed to reduce spasticity by altering the sensory input through the use of cooling or by adding visual and auditory feedback as a source of sensory stimulation.

Rehabilitative Procedures Using Local Cooling

Most clinical reports on the successful use of local cooling have been empirical. In many cases insufficient quantitative data has been presented to establish

a sound rationale for the use of cooling. The paucity of well controlled clinical studies may be due to the lack of objective standards of evaluation with which to assess functional improvement.

Hedenberg (1970) used a hand function test as a quantitative measure to establish the effectiveness of cooling. Twentyfour hemiplegics were tested before immersing their arms in water at twelve degrees centigrade for fifteen minutes. Retesting after cooling showed an improvement in scores for all patients; seventeen results being significantly different. Knutsson (1970b) specifically cooled the spastic muscles, leaving the antagonists at ambient temperature, and obtained one hundred and fifty percent increase in power in the antagonists. Evaluation of the long term effects of this procedure showed the increase in power in the antagonists ranged from 2.5 fold to 5 fold after two weeks of treatment with a concomitant increase in range of voluntary movements from ten to eighty degrees.

In contrast to the lack of studies dealing with functional improvement, a large number of researchers have investigated the effects of cooling on the reflex mechanism.

Using a mechanical hammer that could deliver repeated tendon taps at a constant speed Boes (1962) and Miglietta (1962) reported that the magnitude of the knee jerk was reduced by thirtyfour percent after seven minutes of cooling. Hartviksen (1962) could not elicit an Achilles tendon reflex (ATR) after three minutes of cooling.

Mecomber and Herman (1971) measured the intra-muscular temperature to relate the changes in ATR with the degree of cooling. Although they observed that the muscle temperature dropped at least four to five degrees centigrade they could not relate directly the degree of cooling with the reduction in reflex activity.

The effect of cooling on the tendon reflex in normal muscle has produced inconsistent results. Petajan and Watts (1962) found the mean half-relaxation time of the ATR was increased after cooling, and the reflex changes paralleled the changes in muscle temperature. When Knutsson and Mattson (1969) cooled the leg with commercial ice packs at minus twelve degrees centigrade for thirty minutes the ATR was depressed by thirtyfour percent. The H-reflex, elicited by electrical stimulation, was unchanged. These results are at variance with those of Urbscheit and Bishop (1970) who found the ATR unaltered and the H-reflex increased. The differences of time and temperature employed in these studies could account for the inconsistent results.

The physiological effects of cooling and the mechanisms responsible for the reduction in spasticity remain speculative. The experimental procedures have differed so much in method of cooling, temperature attained, time of application, and evaluation of results that only tentative conclusions can be drawn.

Hartviksen (1962) and Mecomber and Herman (1971)

suggested that as the reflex response of a spastic muscle was reduced before there was an appreciable change in the intra-muscular temperature, the sensory nerve ending in the skin must be involved. Cold anesthesia of the peripheral sensory end organs may change the balance of facilitatory-inhibitory influences on the alpha motoneuron in favour of inhibition. This would reduce the discharge to the extra-fusal fibres and reduce spasticity. This mechanism was also proposed by Mead and Knott (1966). Other factors must be involved however, as the reflex activity remained depressed when the skin was re-warmed (Hartviksen, 1962).

The effect of cooling on nerve conduction velocity has been well documented. Working with normal musculature DeJong, Hershey and Wagman (1966) determined that the conduction velocity of the peroneal nerve decreased linearly at the rate of 1.8 meters per second per degree centigrade fall in temperature. Henriksen (1956) found the decrease in conduction velocity of the ulnar nerve was 2.4 meters per second per degree centigrade fall in temperature. After cooling the forearm with ice for five minutes Zankel (1966) noted a change in conduction velocity of the ulnar nerve from 53.6 meters per second to 50.6 meters per second. Increasing the cooling time to fifteen minutes Petajan and Daube (1965) reported a larger decrease; median nerve conduction velocity dropped from 63 meters per second to 50 meters per second.

Hartviksen (1962) and Petajan and Daube (1965)

have suggested that the decrease in nerve conduction velocity could raise the threshold sensitivity of the gamma innervation of the muscle spindle and cause a reduction in spasticity. Experimental work involving direct cooling of mammalian muscle spindles by Newton and Lehmkuhl (1965) showed the frequency of firing was decreased when the muscle temperature was lowered to twentyfive degrees centigrade.

As Knutsson (1970b) pointed out the effects of cooling are complex and may involve reduced muscle spindle excitability, changes in spinal neurons, and peripheral effects on motor nerves and muscle fibres. Although the specific mechanisms activated by cooling remain unclear, the changes in sensory input from the skin and possibly from the muscle spindle indicate that it can be used to alter sensory-motor integration.

Rehabilitation Procedures Using Visual and Auditory Input

A number of clinical studies have reported the successful use of visual and auditory feedback from the electromyograph to improve the function of a spastic limb.

When a muscle fibre is activated the potential difference across the membrane alters, creating an action potential. Electromyography is a method of observing and recording these changes in electrical activity. Surface electrodes placed on the skin overlying the muscle belly will pick up the summation of action potentials from a

large number of motor units: needle or wire electrodes inserted into the muscle are used for more detailed study of selected motor units.

Myoelectric potentials can be amplified, displayed on a Cathode Ray Oscilloscope (CRO), and broadcast through a loud speaker to provide visual and auditory representation of muscular activity. Basmajian (1963) used these visual and auditory cues to train subjects to control consciously the activity of individual motor units. With practice a subject could initiate, maintain, alter the rate of firing, and stop the activity of a single motor unit. In a recent review of his work Basmajian (1972) reported that hundreds of subjects, using many different muscles, have mastered this technique. Carlsoo and Edfeldt (1963) and Harrison and Mortensen (1962) suggested that auditory stimuli were more important than visual information when a subject was achieving conscious control of motor units. Wagman, Pierce and Burger (1965) presented evidence that recall of a specific unit was highly dependent on the position of the joint.

The practical applications of this technique have been demonstrated by a number of investigators. When people with reading problems caused by sub-vocalisation were provided with the auditory information of the unwanted muscular activity in the laryngeal muscles they were able to eliminate this activity (Hardyck et al., 1966). Working with patients who were unable to relax the upper fibres of

trapezius due to a neck injury Jacobs and Felton (1969) used visual feedback from the electromyograph to teach relaxation.

The attainment of voluntary controlled activity in functionally paralysed muscles was shown to be possible by Marinacci and Horande (1960). Needle electrodes were inserted into a normal muscle to demonstrate to the patient the sight and sound of an action potential. This was followed by inserting the electrodes into a paralysed muscle and instructing the patient to contract the muscle voluntarily. When action potentials occured the patient was encouraged to repeat the effort. Repetition of this activity facilitated voluntary action. Using a similar technique with twenty hemiplegic patients Andrews (1964) obtained strong, voluntary, controlled muscle action in seventeen cases. An extensive clinical trial was conducted by Harris (1970) to assess the longterm effects of EMG-monitored muscle re-education in fourteen hemiplegic patients. Goniometric measurements of range of motion were chosen to evaluate the effectiveness of the procedure. Over a period of four weeks eleven patients showed significant improvement in the joints tested. The most consistent improvement was in the elbow joint.

Examination of current neurophysiological concepts of integration suggest that re-training of a spastic muscle by conscious control of motor unit action potentials could occur through improved integration of sensory stimuli.

Harris (1971) pointed out that as neural circuits become longer the number of synapses increase and the chances of alteration of the original input by other information also increases. This flexibility could be used to re-train a movement. When treating patients who have some disturbance of the neural pathways Harris suggested that cutaneous, visual, auditory and other sensory information could be used to obtain a motor response. When this response occurs the new sensory input from the muscle afferents will be relayed to the higher centres and integrated with the other sensory stimuli. This integration will rebuild the circuit and lead to a better control of movement. Motor learning will occur when correct responses are given to an effective stimuli.

This theoretical concept has been supported by experimental work. Herman (1970b) analysed the spatio-temporal patterns of EMG in the muscles involved in the acquisition phases of skilled motor performance. Herman concluded that visual feedback provided important sensory information to the cortex. This information was integrated with the peripheral sensory input to produce a high gain on the muscle spindle which improved performance. With repetition of the task the integration of information created an automatic skilled movement. Quantitative assessment of neuromuscular parameters in the acquisition of skill have been made by Finley, Wirta and Cody (1968) while investigating the design criteria for myoelectrically controlled

prostheses. The subject performed a simple movement of the forearm in response to a visual target. EMG records before and after the training period showed an increase in dominant activity in the agonists (prime movers). Finley et al. suggested this was due to apparent organisation of the neuromuscular system.

Summary

A large body of empirical observations exists which supports the use of local cooling to reduce spasticity. No definitive work has been found to support the suggestion that conscious control of spastic muscles can be achieved by the use of visual and auditory feedback. The suggestion by DeVries (1968) that the quantitative measures applied to determine the functional state of a normal muscle are applicable to spastic musculature has received little attention.

The present study investigated these questions.

CHAPTER III

METHODS AND PROCEDURES

METHOD

Materials

Five hemiplegic patients were the subjects for this study. The criteria used to select the patients were:

1. Clinical diagnosis of spasticity in the upper limb.
2. Evidence of voluntary movement in the upper limb.
3. The ability to comprehend the requirements of the experiment.

Relevant medical information is given in Table I.

Apparatus

The force lever[1] was a vertical, rigid steel bar incorporating two resistance strain gauges (Figures 1 and 2). It was mounted on a wooden platform which could be rotated through 180 degrees to accomodate either right or left forearms. A sponge rubber handgrip surrounded a metal bar which was bolted onto the lever. Force exerted on the handgrip created an out-of-balance voltage which was

[1]Designed and constructed by A. E. Chapman, Department of Kinesiology, Simon Fraser University.

TABLE I

Relevant Medical Information of Subjects

Subject	1	2	3	4	5
Age	48	26	36	28	19
Sex	M	M	M	M	M
Diagnosis	CVA	Aneurysm	CVA	Head Injury	Aneurysm
Time since onset	8 months	4 months	15 months	14 months	7 months
Side affected	L	R	R	L	L
Handedness	R	R	R	L	R
Spasticity	Moderate	Mild	Gross	Mild	Moderate
Function	Limited	Good	V.Limited	Moderate	V.Limited
Comprehension	Good	Good	Good (Aphasic)	Good	Reduced Attention
Hospital status	Out-patient	Out-patient	Out-patient	Out-patient	In-patient

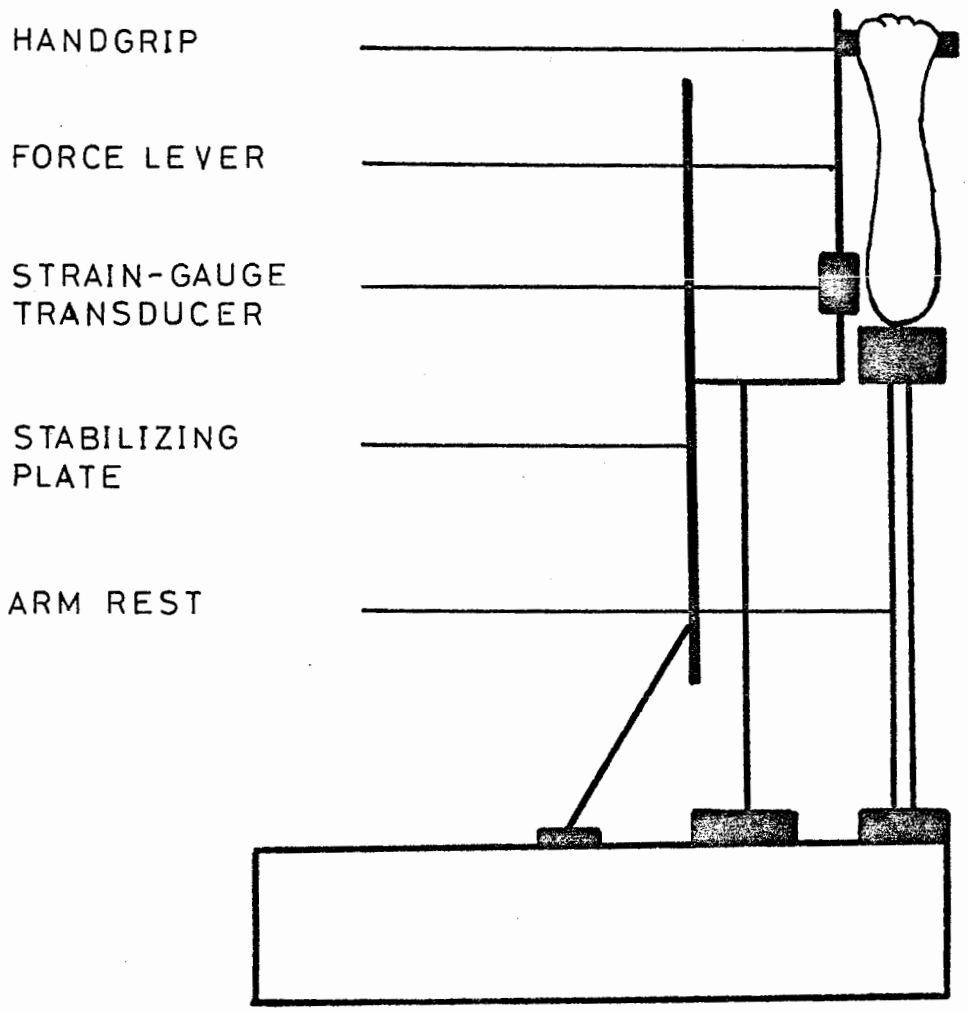

Figure 1: Diagrammatic representation of the frontal view of the dynamometer.

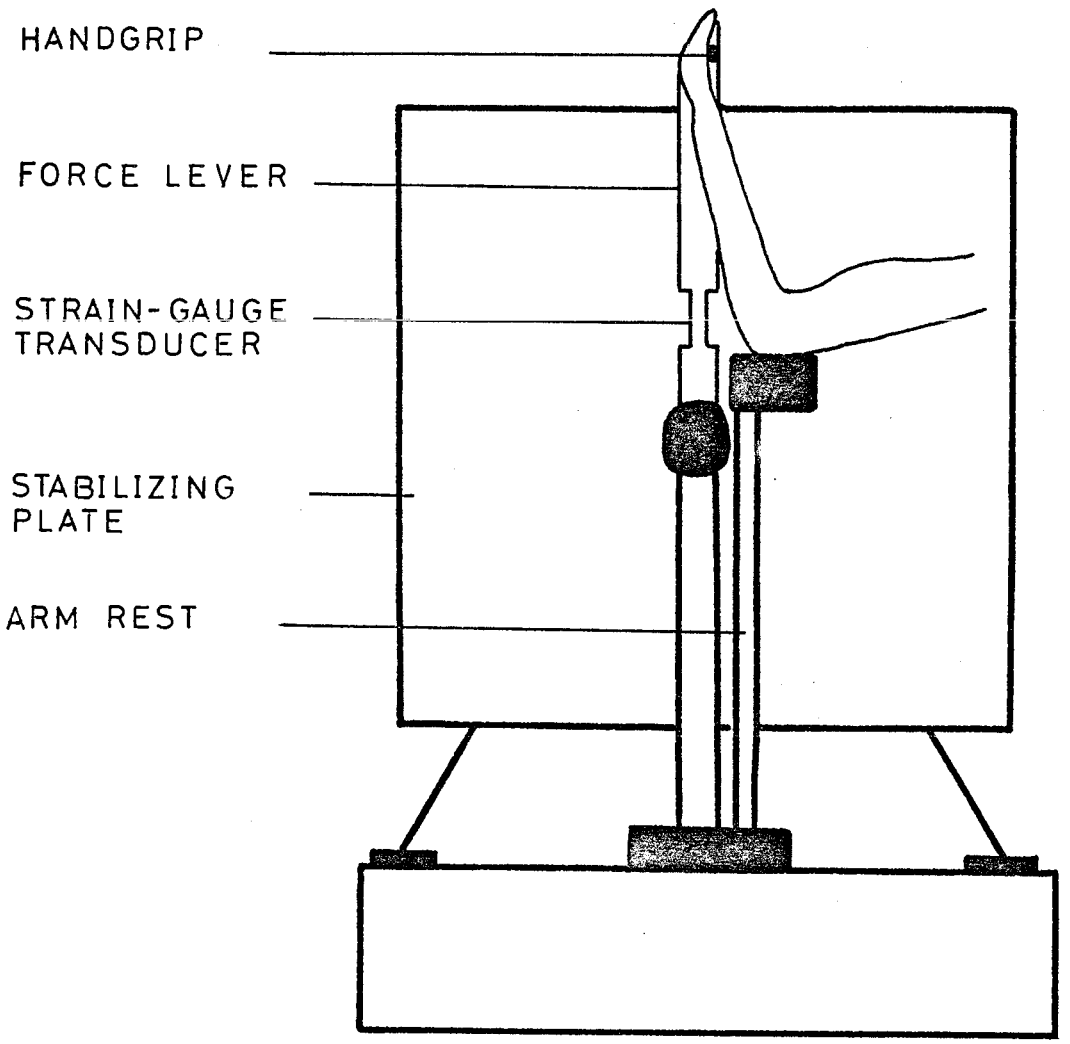

Figure 2: Diagrammatic representation of the side view of the dynamometer.

directly related to the deflecting force. This apparatus served as a dynamometer. Previous calibration verified that a linear response between output voltage and deflecting force existed throughout the entire range of the dynamometer.

A four-channel amplification unit[1] with plug-in modules was used to amplify the electrical signals from both the dynamometer and biceps brachii (Figure 3).

Signals from the dynamometer were amplified through a Strain Gauge Amplifier[2].

Electromyographic recordings were made from biceps brachii through two silver/silver chloride surface electrodes five millimeters in diameter[3]. The interference pattern of the electromyogram (EMG) was amplified in a low noise, high impedance pre-amplifier, located on a universally adjustable arm, connected by plug-in cable to an amplifier[4]. Random noise was reduced by screening and grounding the EMG wire connections. Good electrode/skin conductivity was ensured by filling the electrodes with electrode jelly[5], securing them with adhesive washers and abrading the skin prior to their attachment. A silver ground plate, five by three

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[1] TE4, Teca Corporation, White Plains, New York.

[2] AD6, Teca Corporation.

[3] IMI, Newport Beach, California.

[4] AA6, Teca Corporation.

[5] IMI Low Chloride Gel, Newport Beach, California.

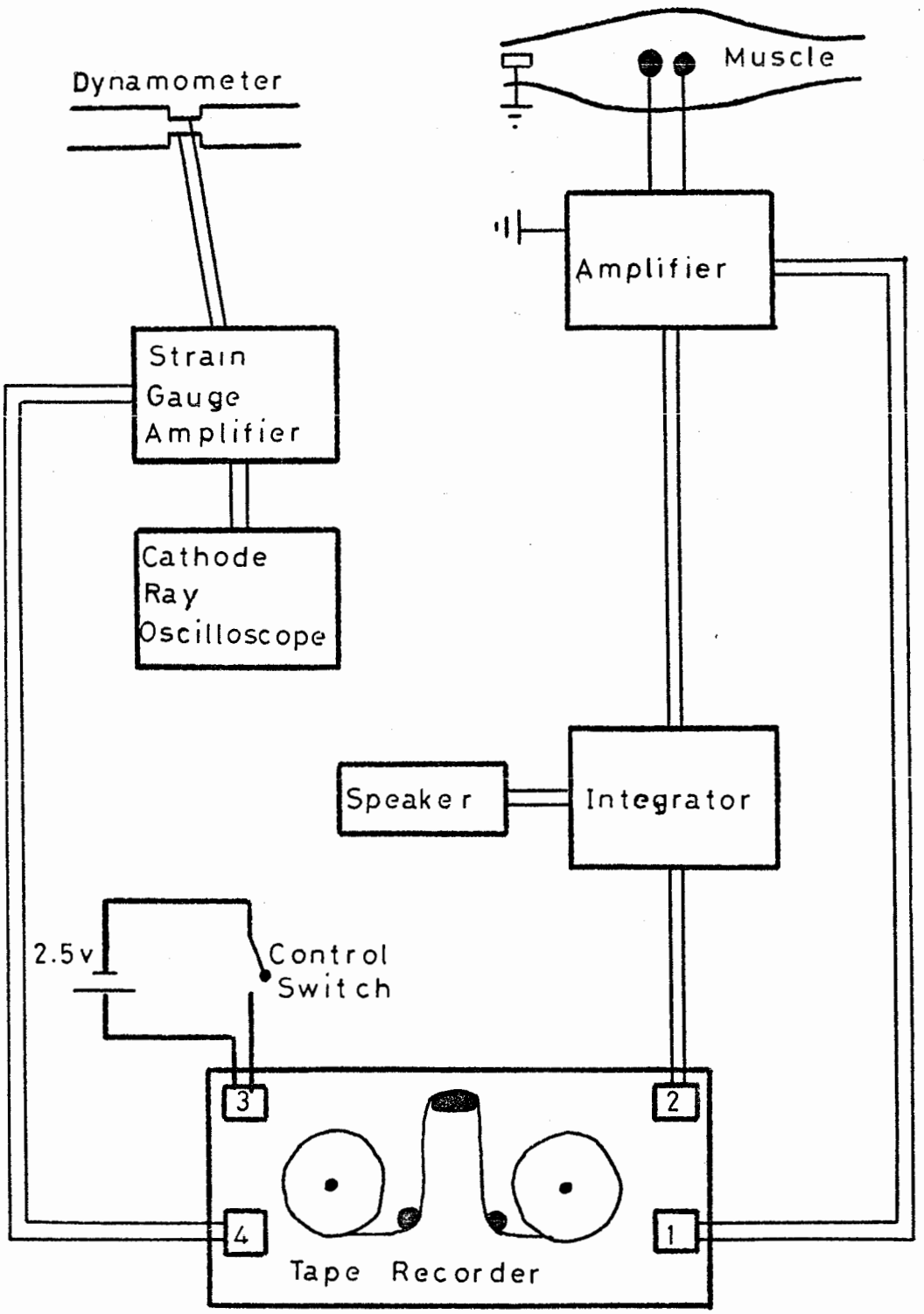

Figure 3: Block diagram of apparatus used for recording and display.

centimeters, was coated with electrode jelly and secured with a strap to the dorsal aspect of the same forearm close to the elbow. Skin resistance was reduced by cleansing the skin over the muscle belly with isopropanol prior to the application of the electrodes.

A quantitative measure of the EMG voltage was obtained by electrical integration[1]. The Integrator, an electronic device which rectifies and smooths the EMG signal, contains two methods of assessing the mean voltage of the complex wave form created during an isometric contraction. The Autoreset mode records pulses which are proportional to the rectified voltage. The number of pulses emitted per unit time are proportional to the area under the curve of the action potentials (i.e., the voltage-time integral). The second method is Mean Voltage recording where the EMG signal discharges through a capacitor at a pre-set rate. The recorded response from a pre-set base line is then proportional to the voltage discharge per unit time. For rapid changes in the size of the wave Mean Voltage recording does not respond as quickly as the Autoreset method. For this reason Autoreset was chosen as the quantitative measure of integration and also as the source of auditory feedback. Calibration of the Integrator on numerous occasions verified that a linear response existed between the voltage/

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[1]I6 Integrator, Teca Corporation, White Plains, New York.

time integral of the amplitude of the input signal and the number of resets per second as a measure of the output of the Integrator.

The active muscle was cooled with ice packs prepared by filling wet towelling with flaked ice and shaping it into a small pack. One layer of towelling separated the ice from the skin. Repeated checks established that the temperature of the pack when applied to the skin was two degrees centigrade. As the ice pack was placed directly over the electrodes an experiment was conducted on a normal subject, in an identical way, to determine whether the electrical properties of the electrodes were altered with this technique. Due to practical difficulties the result of this investigation were not subjected to the same form of analysis as used in the main study. Inspection of the results suggested that no change in the electrical properties of the electrodes occured.

Position of subjects. The subject was seated in a chair at right angles to the force lever, shoulder flexed approximately 45 degrees, elbow resting on a padded rest and flexed approximately 120 degrees (full extension representing 0 degrees). The forearm was supinated and parallel to the force lever with the hand resting on the handgrip. Figures 1 and 2 illustrate the position. The elbow rest was adjustable to accomodate the differences in length of each subjects forearm. All subjects tolerated the position with no discomfort.

Visual and auditory display. Two devices, incorporated into

the Electromyograph, were used to provide visual and auditory feedback.

A cathode ray display screen, a twelve by fifteen centimeter flat face, with a variable sweep trace velocity, displayed both the output from the dynamometer (force) and a non-input target line. The display screen was placed directly in front of the subject.

Audio amplification was obtained through a front mounted five by seven inch speaker. The integrated EMG (IEMG) signal was chosen as the most suitable form of auditory feedback as its auditory output was in the form of discrete pulses, their frequency was proportional to the intensity of electromyogram activity.

Recording of data. Data from the integrator, EMG amplifier and strain-gauge amplifier was recorded by a four channel F/M tape recorder[1] and stored on magnetic tape. The fourth channel was used to record a control signal required to activate the computer when the data was being converted.

Collection of data. All data was collected on tape in analog form. A digital computer[2] with 8K memory, interfaced with an analog to digital converter, was used to obtain digital values of force and IEMG. No attempt was

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[1] 3690 Instrumentation Recorder, Hewlett Packard.

[2] PDP8/E, Digital Equipment Corporation, Maynard, Massachusetts.

made to interpret the interference pattern of the EMG in this study.

An analog to digital conversion programme (A/DC, Appendix 1) retrieved two hundred values of force for one second of each pull. These values were converted and stored in the core memory of the computer. One thousand values of the corresponding IEMG were similarly retrieved, converted and stored.

A FOCAL programme (ASPROG, Appendix 2) was run to obtain a digital printout of the mean values and standard deviation of force for each pull. The IEMG values were converted into the number of resets per second.

PROCEDURES

The subject was positioned in the dynamometer and the electrodes applied to the skin. While the subject produced a maximum voluntary contraction (MVC) the sensitivity of the strain-gauge amplifier was adjusted to obtain a full-scale deflection. The non-input target line was then set at 30% of this deflection. The intensity of the trace from both the dynamometer and the target line was adjusted to ensure clear, visible signals. These signals provided the subject with the visual feedback of his performance. Force applied to the dynamometer caused an immediate deflection of the input trace. When a force of 30% of MVC was exerted the input trace was superimposed on the target line.

To establish a distinct audio signal from the integrator the gain on the EMG amplifier was adjusted while the subject produced a force of 30% of MVC.

These procedures were followed for each subject on each day.

To familiarize themselves with the equipment and the requirements of the experiments, all subjects undertook the full experiment once on a pre-test basis.

All subjects completed twelve trials (Monday, Wednesday, Friday for four weeks) with the exception of Subject 1 who could not complete the eleventh trial due to a sudden indisposition.

Two work tasks were performed. Work Task 1 (WT1) was executed on each day of the training period. Work Task 2 (WT2) was performed on the last five days on the training period.

Work Task 1 (WT1)

WT1 comprised twenty isometric contractions. For each contraction the subject was told to match the target line, maintain the contraction, and relax. The time required for one contraction sequence was five seconds. This was followed by a rest pause of fifteen seconds. The subject was encouraged to listen to the audio signal and use this additional information to help him sustain the correct isometric force. A recording was made of each pull for a period of one second. The tape recorder was switched on

and the control signal activated when the subject had produced sufficient force to match the target line.

Following this set of contractions the subjects hand was disengaged from the handgrip and the signal from the strain-gauge amplifier was set to match the target line. A recording was made of this target signal.

With the subject re-positioned in the dynamometer an ice pack was placed over biceps brachii. After fifteen minutes of cooling the ice pack was removed and the subject repeated the work task. Recordings were made of each pull and a second target signal completed the recording.

Work Task 2 (WT2)

A series of graded contractions were performed and recorded. The initial recording was made with the subject at rest, exerting no force. Six further recordings were taken with each successive isometric contraction increasing in magnitude by approximately 10%. The subject used the linear scale on the strain-gauge amplifier to estimate each increment. Immediately after cooling a second series of graded contractions were performed.

The time required for each subject to complete both work tasks was fortyfive minutes.

Problems

A number of uncontrollable variables were present throughout the experimental procedure.

The subjects were encouraged to maintain the same position throughout each daily session, however, some slight variability was unavoidable when the ice pack was applied. As the requirements of the subject was to match a target set at 30% of MVC and not to produce maximal force it was felt that small variations in posture were not detrimental.

The reproducibility of the subjects position from day to day was affected by a number of factors.

The physical and emotional well-being of the subjects underwent some fluctuations during the four week period. As both these factors can affect the degree of spasticity (Roasenda and Ellwood, 1963) it must be assumed that there was some variation in spasticity throughout the experiment.

The scheduling of the subjects required the dynamometer to be rotated twice a day to accomodate both right and left arms. As the chair was independent of the dynamometer slight changes in their relative positions was a possible source of variability. To ensure consistent positioning of the forearm in the dynamometer individual fixation (splinting) would have been required. As this type of fixation creates discomfort and distraction its incorporation into the apparatus was rejected.

As this experiment was conducted under clinical conditions these variables had to be accepted.

The experiment was conducted in an unused office at the G. F. Strong Rehabilitation Centre, Vancouver, B. C.

Due to the characteristics of the building two important sources of outside interference were unavoidably present. Electromyographic equipment is susceptable to extraneous electromagnetic and electrostatic forces. Short wave diathermy machines are a powerful source of external interference. On a number of occasions short wave diathermy was being used in an adjoining room while an experimental session was in progress. When this occurred the IEMG signal was distorted and unable to be used as a source of auditory feedback to the subject. As the output from the dynamometer was unaffected by this interference the trial continued using visual feedback only.

A further interference factor was present throughout the entire experiment. The force lever was a highly sensitive instrument with a low moment of inertia. Any vibration in the room created a detectable oscillation in the lever. Due to a major re-construction project on the adjacent property a constant vibration was present in the flooring of the room. This caused a variable degree of oscillation in the force lever. As this interference was of high frequency and low amplitude its effects were negligible in terms of both mean force and its contribution to variation of the output from the force transducer (standard deviation = 0.5% of its output at 30% of MVC). To prevent this affecting the visual signal to the subject a sweep trace time of 0.5 milliseconds was maintained on

the CRO for the entire experimental procedure.

Analysis of Data

Work Task 1

Conversion of the digital values of mean force ($\bar{F}$) per second per pull to percent MVC was effected from the values corresponding to 30% of MVC (target signal) and the resting level of the force lever for each subject.

Calculations were done by computer (Appendix 3) to obtain the following values for each subject on each trial.

a) mean force ($\bar{F}$)
b) mean standard deviation
c) coefficient of variance

These values were pooled for all subjects for an analysis of variance (Appendix 4).

The following graphical relationships were examined:

a) individual pulls of each trial for each subject under ambient and cooled muscle conditions compared to the target force.
b) mean force ($\bar{F}$) for each trial for each subject under ambient and cooled muscle conditions compared to the target force.

A non-parametric statistical test, the sign test (Siegel, 1956) was used to determine significant differences.

The considerable scatter that occurred between $\bar{F}$ and IEMG prevented further analysis of these parameters.

This relationship is illustrated in Figure 12.

Work Task 2

The graphical relationship between $\bar{F}$ and IEMG was examined for each subject on each day under both ambient and cooled muscle conditions.

CHAPTER IV

RESULTS

Ability to Produce an Isometric Force

The mean force ($\bar{F}$) for each pull on each trial was compared to the target force (30% of MVC). Figures 4 - 8 present these results for trial 1 (T1) and trial 12 (T12) under ambient conditions. The mean standard error of all pulls is included on the initial pull.

Subject 3 (Figure 6) presented the most obvious change in ability. The first four pulls of T1 caused the dynamometer to be deflected in the reverse direction. This indicated that the force was exerted by the extensors of the elbow instead of the flexors. This reverse effect occurred only once more throughout experiment (pull 6, trial 5).

The results from T1 indicated that none of the subjects matched the target force. Close approximation to the target force of 30% Of MVC was achieved by Subjects 4 and 5 with a mean force of 29.5% of MVC. Tables II - VI summarize the individual results for each subject on each trial.

Improvement in Control with Training

Inspection of the data indicated that the ability

of each subject to control a specific force had altered during the experiment. The sign test was applied to determine when this change had occurred. The difference between $\bar{F}$ and 30% of MVC for each pull was compared with the corresponding pull on the preceeding trial i.e. T2 - T1, T3 - T2,.... T12 - T11. These results showed that the variability of the response decreased in four subjects. As the variability of the response was considered to be an indication of the subjects ability to control a specific force it was concluded that Subject 1 improved his level of control by T3, Subject 2 by T2, Subjects 3 and 4 by T4. No significant improvement could be found for Subject 5.

To establish whether this improvement in control was maintained during the entire experiment the differences in the variability of the response on T1 were compared to the differences on T12. These results indicated that the learning effect was maintained in Subjects 2 and 3. The improvement in the ability to control a force that had been demonstrated by Subjects 1 and 4 was not evident by trial 12.

Effect of Cooling

Changes in the ability of the subjects to produce and control a specific force after local cooling were not significant either on individual trials or over the entire experiment. One significant result was found for Subject 3 on trial 1. As this change occurred while learning was still taking place the change could not be attributed solely

to the effect of cooling. To clarify this point comparisons were made between the variability of each pull T1 cooled and T2 ambient. This test yielded no significant difference. As similar comparisons of the remaining trials also were not significant it was concluded that this random event did not indicate that cooling had improved the ability of the subject to control a specific force.

Analysis of IEMG

Integrated EMG was used as a source of auditory feedback in WT1. Examination of the raw data indicated that considerable scatter occurred in the relationship between the mean force of each pull and the IEMG (example Figure 12). However there was a slight tendency for the IEMG to increase as muscular force increased. This scatter indicated that the level of IEMG used in this experiment had not provided a reliable source of auditory feedback to the subjects. This phenomenon existed at both levels of temperature, however, the values of IEMG produced was consistently higher after the muscle had been cooled. The results from WT2 allowed a more detailed examination of this trend.

IEMG/Force (WT2) (Figure 13, Tables VII - XI)

Twentyfour separate trials were conducted which compared the amount of IEMG (resets/second) produced in response to different levels of force (%MVC) at both ambient temperature and after cooling of the muscle. The values

obtained demonstrated that a trend to linearity existed between IEMG and force at both temperature levels employed.

In seventeen trials a significant increase in the amount of IEMG produced for the same force exerted was observed after cooling. These findings apparently indicated that the contractile properties of the muscle had altered with cooling. As technical difficulties prevented an analysis of the interference pattern of the EMG records of the subjects a separate experiment was conducted on a normal subject, using the same technique as in the main study, to allow detailed examination of the interference pattern of the EMG after cooling. The results from this experiment showed that while maintaining the same force as before cooling, a change in waveform was produced with an increase in the duration of the action potential.

Figure 4: Subject 1

Mean force (%MVC) for each pull compared to target force (30%MVC) under ambient conditions The mean standard error of all pulls is included on the initial pull. Trial 1 - solid line Trial 12 - broken line.

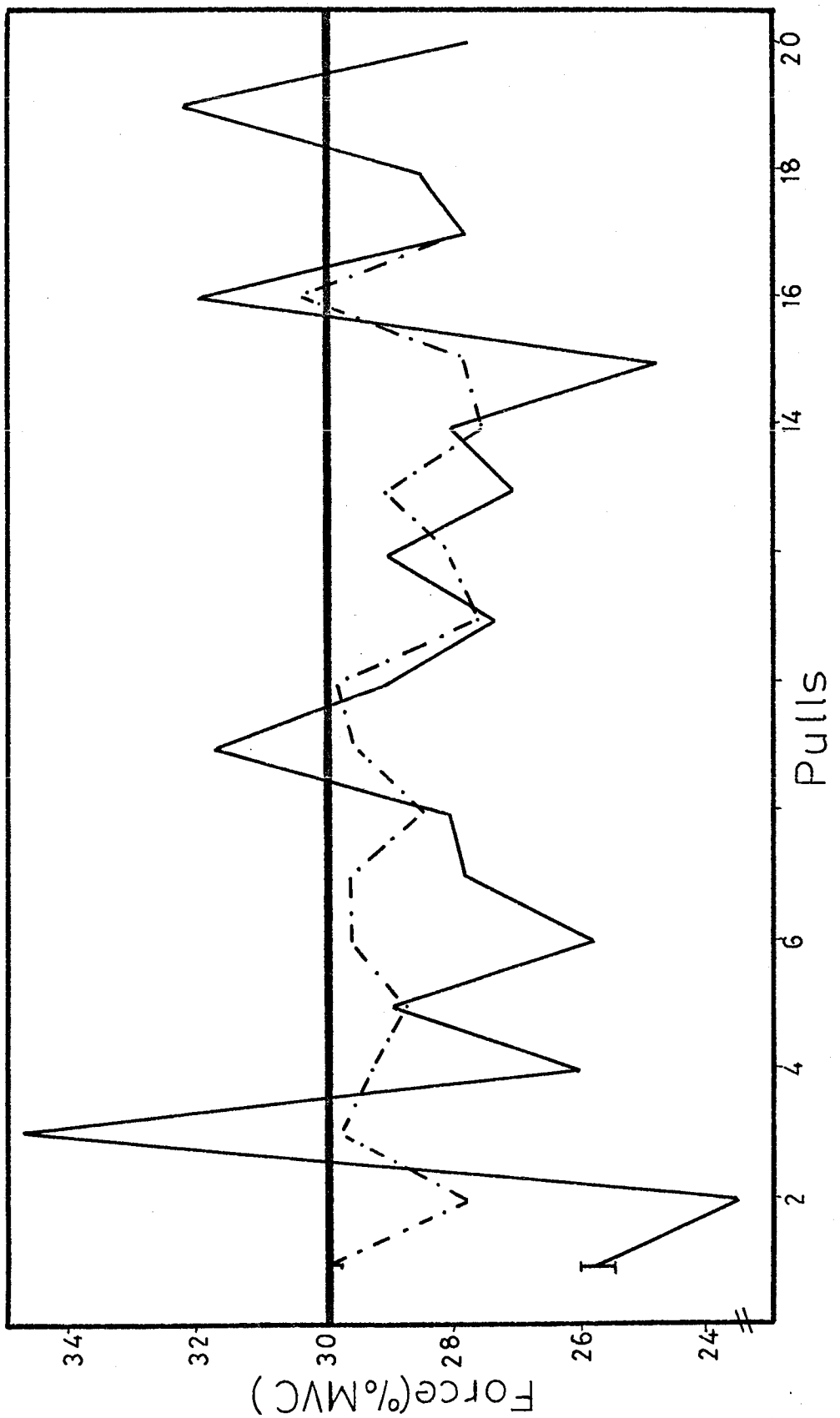

Force(%MVC)
34
32
30
28
26
24
2
4
6
14
16
18
20
Pulls

TABLE II. Showing the range and mean value of force (%MVC) corresponding to IEMG (resets/sec.) for each day of training under either ambient (A) or cooled (B) muscle conditions.

SUBJECT 1

DAY	TEMP.	RANGE	FORCE	IEMG
1	A	24.0-34.0	28.0	x
	B	23.5-34.0	28.5	x
2	A	24.0-29.5	27.5	29
	B	26.5-29.5	28.0	42
3	A	26.5-32.5	30.5	18
	B	28.0-31.0	30.0	34
4	A	28.0-32.0	30.0	16
	B	27.5-31.0	29.0	21
5	A	26.5-32.0	29.5	19
	B	28.0-31.0	30.0	19
6	A	28.0-30.5	29.0	17
	B	27.0-30.5	29.5	38
7	A	25.5-30.5	28.5	22
	B	25.5-31.0	29.0	32
8	A	26.5-30.0	28.5	x
	B	27.5-30.0	28.5	27
9	A	26.0-31.0	28.5	36
	B	27.5-30.5	28.5	43
10	A	27.0-31.5	30.0	14
	B	28.0-31.5	28.5	41
11	A	27.0-29.0	28.0	19
	B	not performed		
12	A	26.5-30.0	28.0	24
	B	27.0-30.5	28.5	28

x - IEMG invalid due to interference

Figure 5: Subject 2

Mean force (%MVC) for each pull compared to target force (30%MVC) under ambient conditions. The mean standard error of all pulls is included on the initial pull.
Trial 1 - solid line
Trial 12 - broken line.

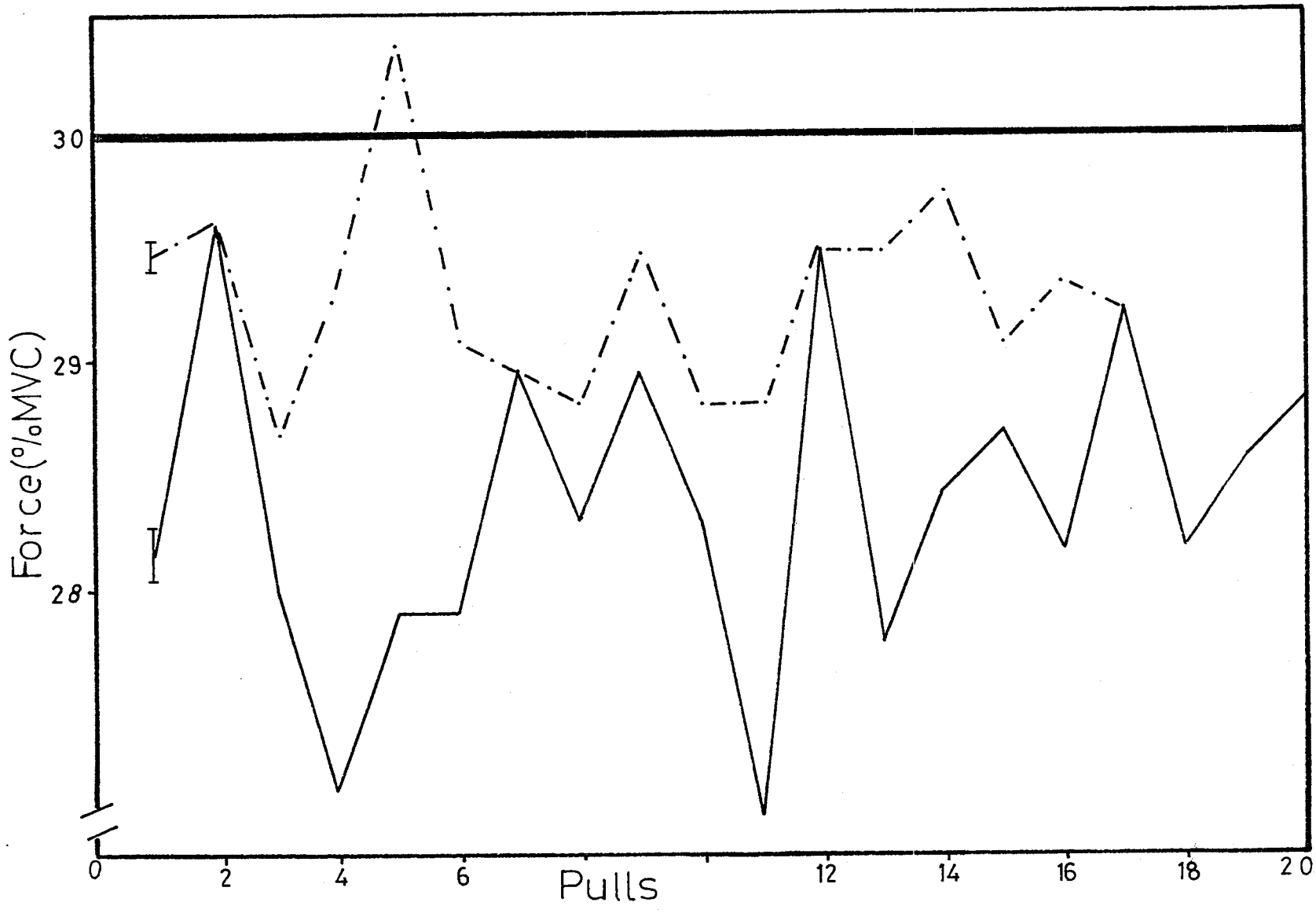

30
29
28
Force(%MVC)
0
2
4
6
Pulls
12
14
16
18
20

TABLE III. Showing the range and mean value of force (%MVC) corresponding to IEMG (resets/sec.) for each day of training under either ambient (A) or cooled (B) muscle conditions.

SUBJECT 2

DAY	TEMP.	RANGE	FORCE	IEMG
1	A	27.0-29.5	28.5	18
	B	27.0-30.0	28.5	20
2	A	27.0-31.0	30.0	10
	B	29.0-32.5	30.5	18
3	A	28.0-29.5	29.0	20
	B	26.5-30.0	28.5	34
4	A	26.5-31.0	29.5	21
	B	28.5-30.0	29.5	26
5	A	29.5-32.0	30.5	7
	B	29.0-31.5	30.0	11
6	A	29.0-30.5	30.0	16
	B	29.0-31.5	30.0	19
7	A	28.0-30.5	29.0	12
	B	28.5-30.0	29.5	17
8	A	28.5-30.5	29.5	14
	B	28.5-30.5	29.5	20
9	A	28.5-31.0	30.0	20
	B	30.0-31.5	30.5	29
10	A	28.5-31.0	29.5	16
	B	29.5-31.5	30.5	27
11	A	29.0-30.5	29.5	23
	B	28.5-30.0	29.5	20
12	A	29.0-30.5	29.5	12
	B	28.0-30.0	29.0	27

Figure 6: Subject 3

Mean force (%MVC) for each pull compared to target force (30%MVC) under ambient conditions. The mean standard error of all pulls is included on the initial pull.
Trial 1 - solid line
Trial 12 - broken line.

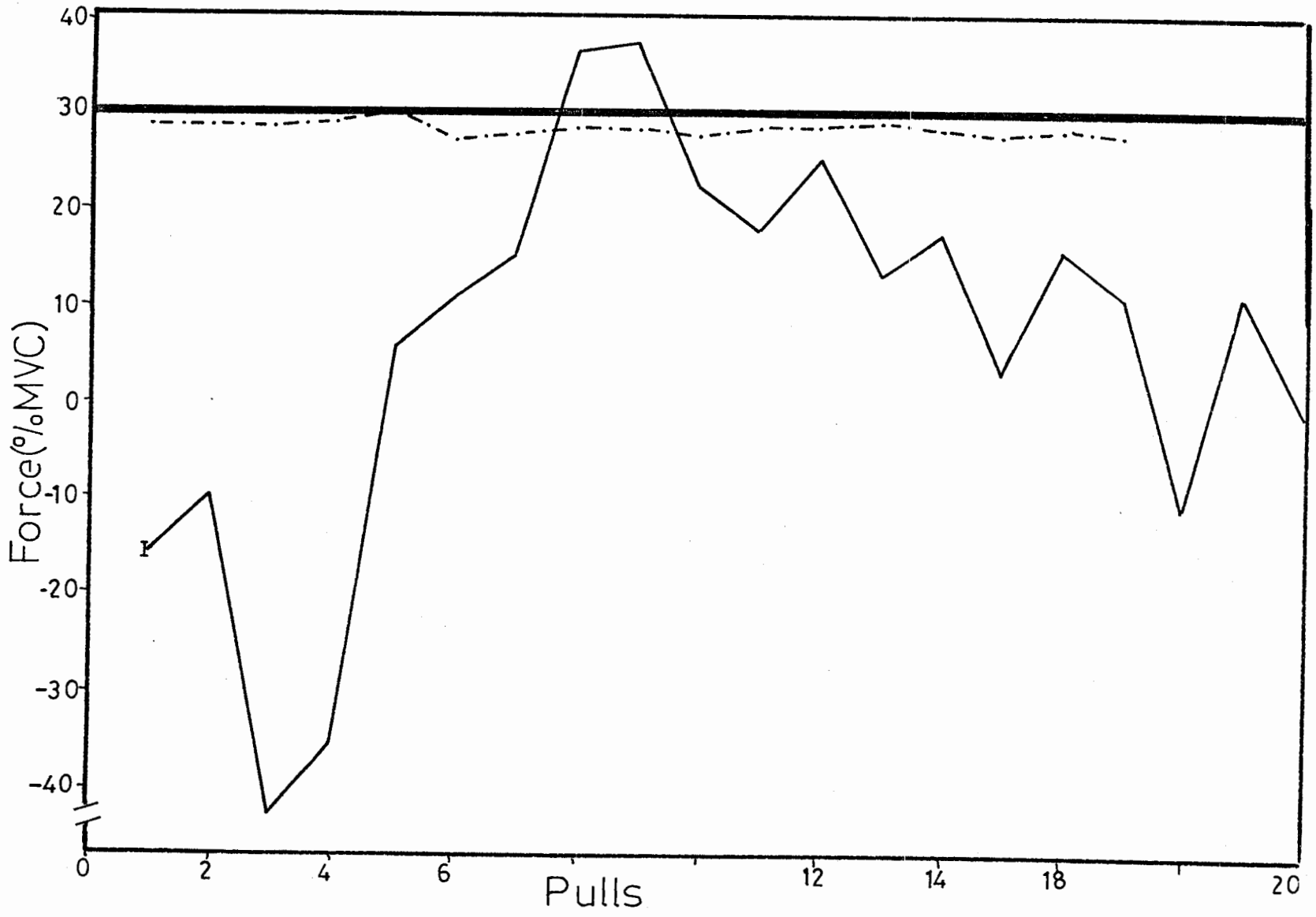

40
30
20
10
0
-10
-20
-30
-40
Force(%MVC)
0
2
4
6
Pulls
12
14
18
20

TABLE IV. Showing the range and mean value of force (%MVC) corresponding to IEMG (resets/sec.) for each day of training under either ambient (A) or cooled (B) muscle conditions.

SUBJECT 3

DAY	TEMP.	RANGE	FORCE	IEMG
1	A	*45.0-36.0	8.0	15
	B	18.0-29.0	25.0	31
2	A	5.0-28.0	22.5	20
	B	19.0-29.0	24.0	37
3	A	26.5-28.5	27.5	27
	B	16.5-29.0	25.5	26
4	A	28.5-33.0	31.0	16
	B	27.0-31.0	29.5	24
5	A	*15.0-34.5	25.5	6
	B	23.5-29.5	28.0	10
6	A	27.5-30.5	29.5	14
	B	27.5-31.5	30.0	28
7	A	21.5-30.0	27.0	14
	B	21.5-30.0	27.0	34
8	A	27.0-30.5	29.0	8
	B	24.5-30.5	28.5	12
9	A	26.5-30.5	29.0	28
	B	17.0-30.0	27.0	47
10	A	26.5-33.5	30.5	20
	B	29.5-32.5	31.5	37
11	A	30.0-33.0	31.5	13
	B	29.0-33.5	31.0	17
12	A	27.5-30.0	29.0	20
	B	27.0-30.5	28.5	20

* force exerted in opposite direction

Figure 7: Subject 4

Mean force (%MVC) for each pull compared to target force (30%MVC) under ambient conditions. The mean standard error of all pulls is included on the initial pull.
Trial 1 - solid line
Trial 12 - broken line.

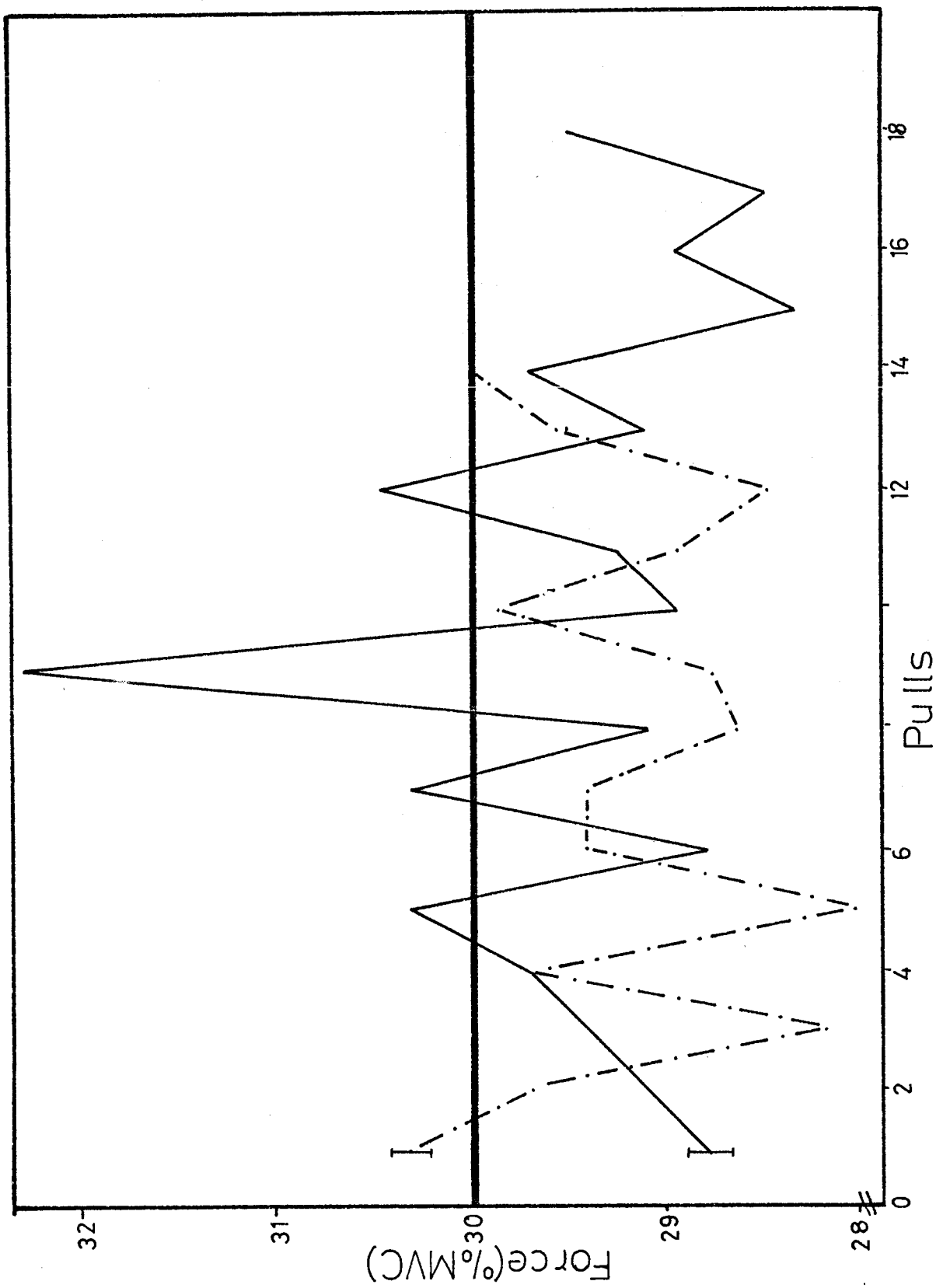

Force(%MVC)
32
31
30
29
28
0
2
4
6
12
14
16
18
Pulls

TABLE V. Showing the range and mean value of force (%MVC) corresponding to IEMG (resets/sec.) for each day of training under either ambient (A) or cooled (B) muscle conditions.

SUBJECT 4

DAY	TEMP.	RANGE	FORCE	IEMG
1	A	28.0-32.0	29.5	x
	B	27.5-31.0	30.0	x
2	A	28.0-31.0	29.5	21
	B	27.5-30.5	29.0	39
3	A	28.5-33.5	30.0	11
	B	28.0-30.5	29.5	16
4	A	29.0-31.0	30.0	25
	B	28.5-30.0	29.5	31
5	A	28.0-31.5	29.5	11
	B	28.5-30.5	29.5	19
6	A	29.5-30.5	30.0	12
	B	28.0-31.5	28.5	x
7	A	27.5-33.0	29.0	22
	B	29.0-36.5	30.5	32
8	A	28.0-30.5	29.0	23
	B	28.5-32.0	30.0	34
9	A	28.0-31.5	30.0	18
	B	29.0-31.5	30.0	28
10	A	27.5-30.5	29.0	11
	B	27.0-30.5	29.0	17
11	A	28.5-31.0	29.5	25
	B	28.0-32.0	29.5	39
12	A	28.0-30.5	29.5	27
	B	26.0-30.5	29.0	x

x - IEMG invalid due to interference

Figure 8: Subject 5

Mean force (%MVC) for each pull compared to target force (30%MVC) under ambient conditions. The mean standard error of all pulls is included on the initial pull. Trial 1- solid line Trial 12 - broken line.

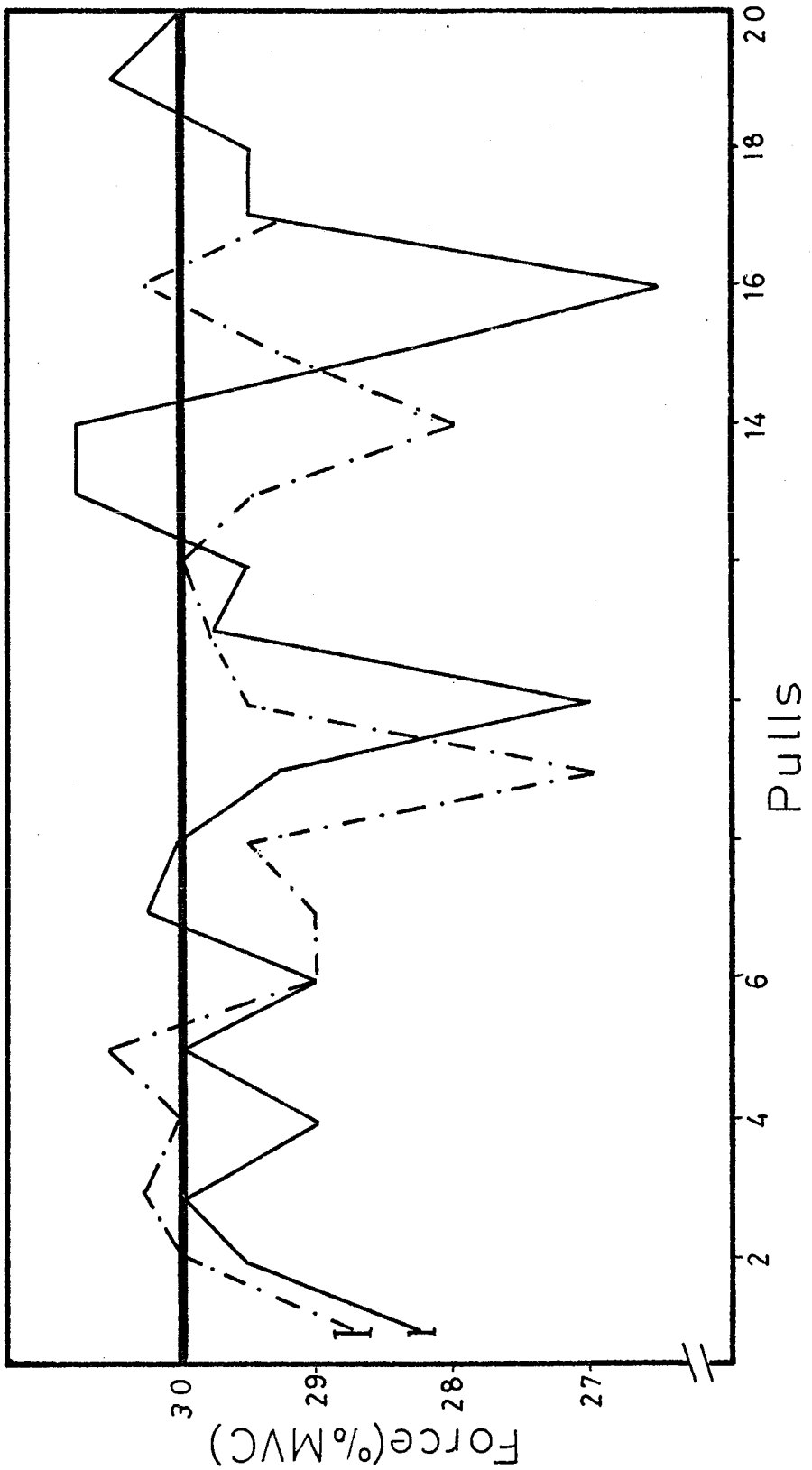

Force(%MVC)
30
29
28
27
2
4
6
14
16
18
20
Pulls

TABLE VI. Showing the range and mean value of force (%MVC) corresponding to IEMG (resets/sec.) for each day of training under either ambient (A) or cooled (B) muscle conditions.

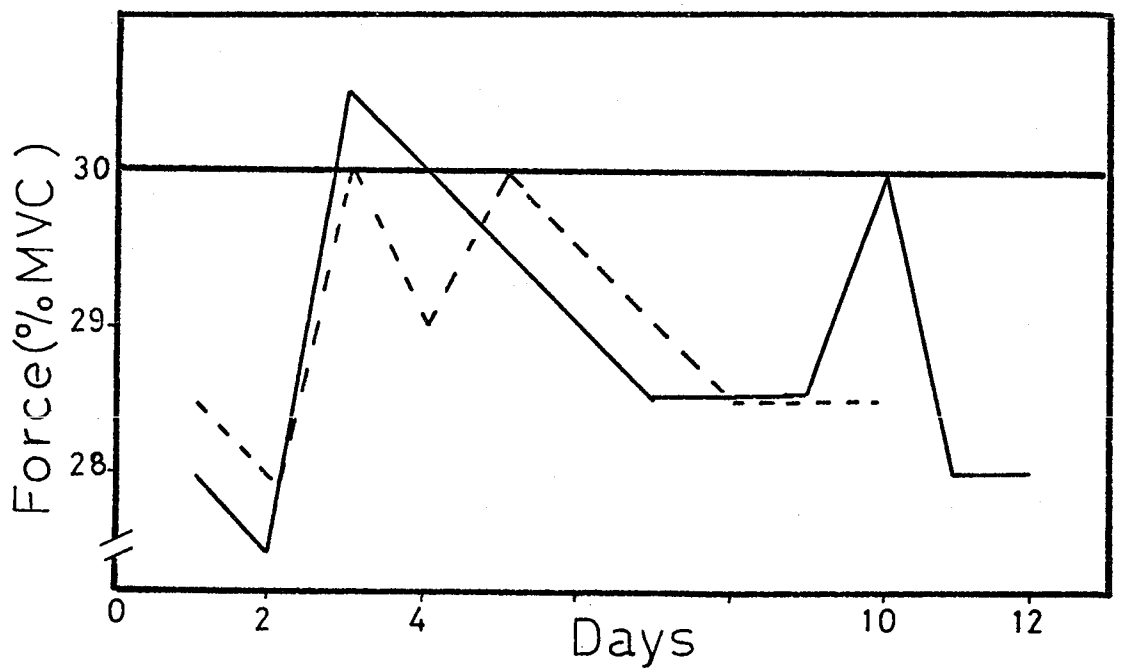
SUBJECT 5

DAY	TEMP.	RANGE	FORCE	IEMG
1	A	26.5-31.0	29.5	x
	B	24.0-30.5	28.5	x
2	A	22.0-30.0	26.5	14
	B	23.0-30.0	27.5	56
3	A	17.0-29.5	26.5	18
	B	4.0-31.0	27.0	34
4	A	18.5-32.0	28.5	29
	B	26.5-32.0	29.0	35
5	A	25.0-31.5	28.5	29
	B	22.5-29.5	26.0	52
6	A	28.0-31.5	30.0	13
	B	26.0-31.5	29.0	19
7	A	19.0-30.0	28.0	25
	B	28.5-31.0	30.0	37
8	A	26.5-30.5	29.0	23
	B	27.5-31.0	29.0	25
9	A	29.0-32.0	30.0	22
	B	27.5-32.0	29.5	34
10	A	27.5-30.5	29.0	13
	B	25.0-30.5	27.5	9
11	A	27.5-31.0	29.0	14
	B	27.5-30.0	29.0	27
12	A	27.0-30.5	29.0	17
	B	28.0-30.0	29.0	20

x - IEMG invalid due to interference.

Figure 9: Mean force (%MVC) for each day compared to target force (30%MVC). Ambient - solid line Cooled - dashed line.

Subject 1 (upper)
Subject 2 (lower)


Force(%MVC)
30
29
28
0
2
4
10
12
Days

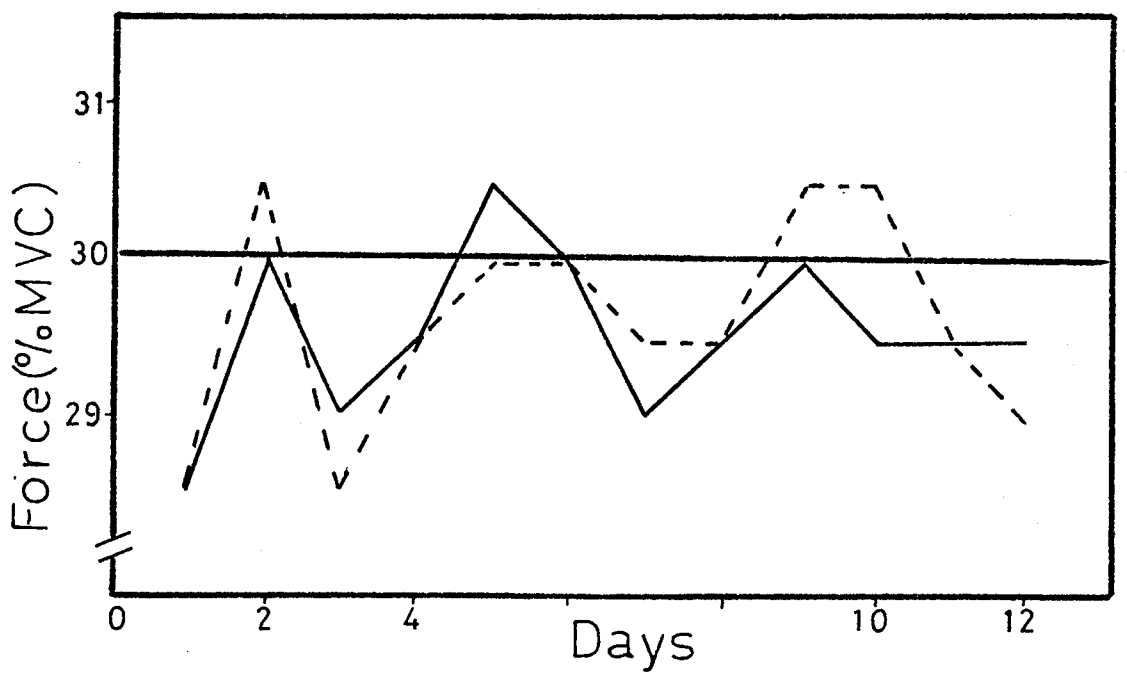

Force(%MVC)
31
30
29
0
2
4
10
12
Days

Figure 10: Mean force (%MVC) for each day compared to target force (30%MVC).
Ambient - solid line
Cooled - broken line.

Subject 3.

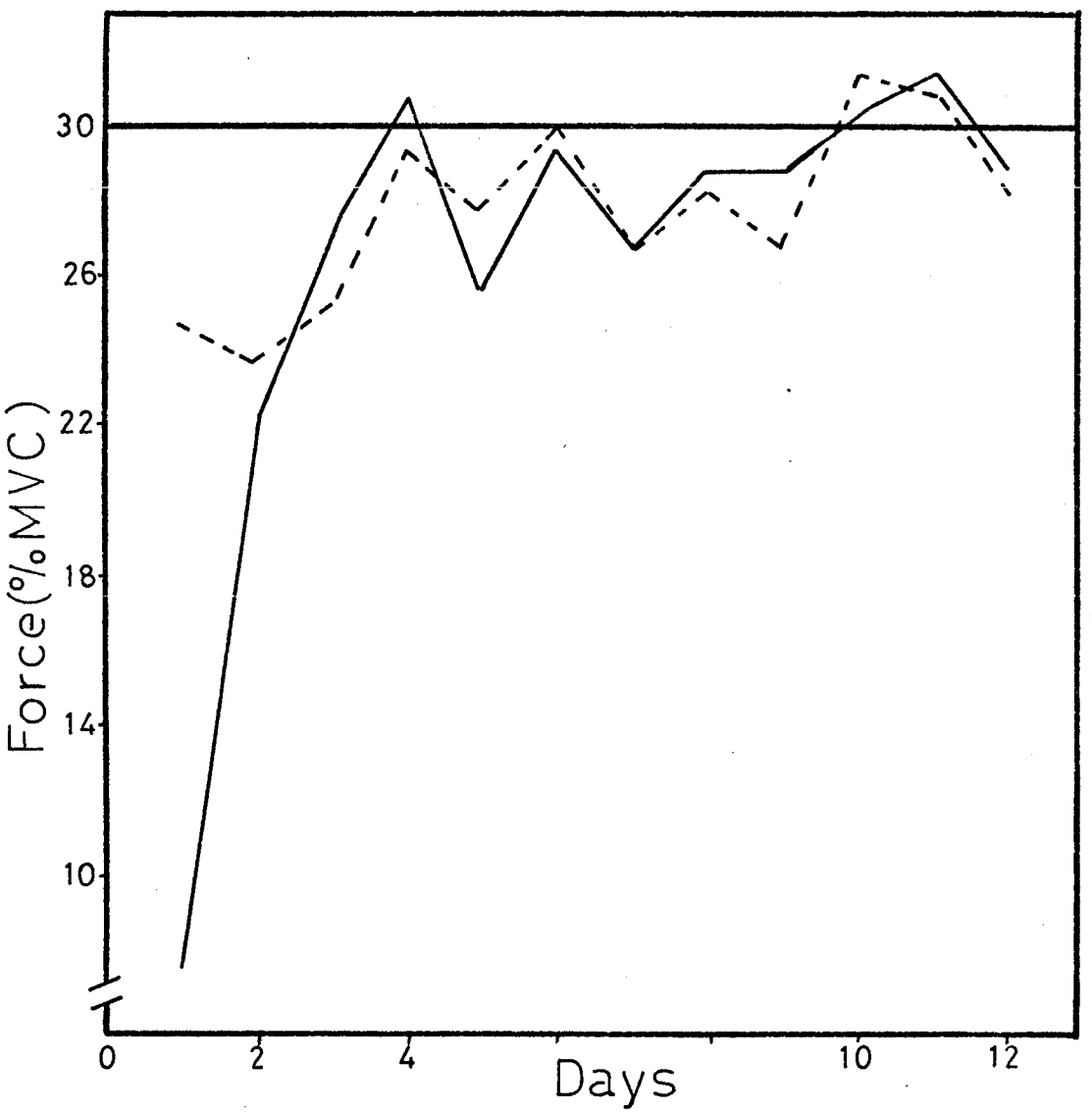
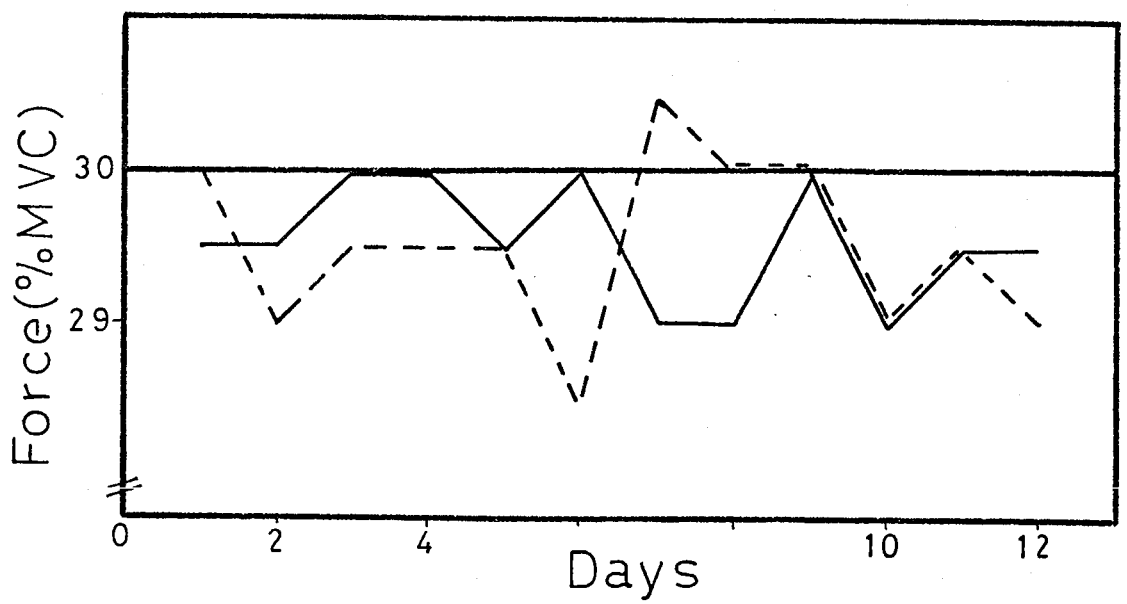

30
26
22
18
14
10
Force(%MVC)
0
2
4
10
12
Days

Figure 11: Mean force (%MVC) for each day compared to target force (30%MVC). Ambient - solid line Cooled - broken line.

Subject 4 (upper)
Subject 5 (lower).


Force(%MVC)
30
29
0
2
4
10
12
Days

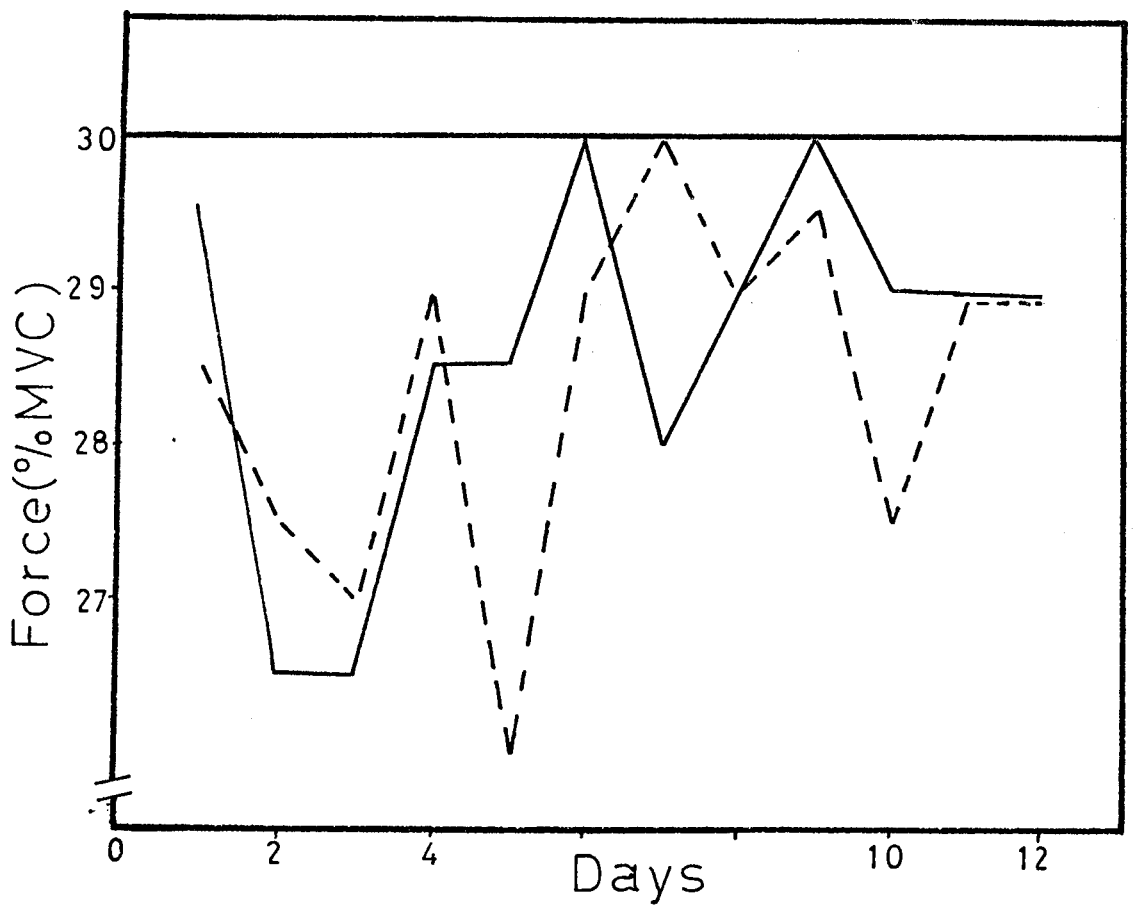

Force(%MVC)
30
29
28
27
0
2
4
10
12
Days

TABLE VII. Showing isometric force (%MVC) corresponding to IEMG (resets/sec.) for days (D) 8 to 12 under either ambient (A) or cooled (B) muscle conditions.

SUBJECT 1

D 8	A	%MVC	1.2	9.2	19.3	25.8	34.4	49.0	53.5	
		IEMG	1	16	16	15	17	26	24	
	B	%MVC	1.6	14.4	17.6	23.0	32.2	36.2	50.0	
		IEMG	5	30	24	27	24	26	30	
D 9	A	%MVC	2.3	13.6	17.5	23.5	36.2	46.0		
		IEMG	2	16	19	19	42	44		
	B	%MVC	1.2	16.0	21.0	22.2	31.0	35.0		
		IEMG	0	18	26	27	35	33		
D 10	A	%MVC	2.2	5.4	19.0	21.0	24.0	31.0	45.0	50.0
		IEMG	1	4	12	15	13	17	20	34
	B	%MVC	4.6	7.4	10.0	20.0	25.0	32.0	45.0	46.5
		IEMG	1	6	25	28	31	43	59	77
D 11	A	%MVC	1.0	9.0	14.0	26.0	29.6	36.0	41.0	
		IEMG	1	13	12	14	20	36	37	
	B	%MVC	1.8	2.4	10.0	13.6	27.5	33.0	42.0	46.0
		IEMG	1	5	30	33	44	43	56	69
D 12	A	%MVC	0.6	10.0	15.0	24.4	31.6	40.0	46.0	
		IEMG	1	9	11	13	26	24	30	
	B	%MVC	1.4	5.4	14.4	14.6	24.0	37.0	49.0	
		IEMG	1	27	35	28	54	53	60	

TABLE VIII. Showing isometric force (%MVC) corresponding to IEMG (resets/sec.) for days (D) 8 to 12 under either ambient (A) or cooled (B) muscle conditions.

SUBJECT 2

D 8	A	%MVC	0	9.5	17.4	27.0	35.6	45.0	54.0	
		IEMG	3	5	6	8	11	14	15	
	B	%MVC	2.0	10.5	18.7	25.8	35.0	46.5	54.5	
		IEMG	6	9	18	30	36	57	59	
D 9	A	%MVC	0	10.5	18.2	26.7	33.8	45.5	53.5	
		IEMG	6	10	12	21	34	41	58	
	B	%MVC	2.2	10.0	18.3	27.2	36.4	46.2	58.2	
		IEMG	7	12	18	37	63	51	61	
D 10	A	%MVC	0	9.1	17.8	26.3	34.0	43.0	54.0	
		IEMG	4	9	15	28	31	34	49	
	B	%MVC	0	11.9	18.2	26.7	37.0	50.8	56.0	
		IEMG	5	7	13	28	32	48	50	
D 11	A	%MVC	0	12.1	20.0	29.1	36.4	45.0	54.0	
		IEMG	9	14	16	25	32	47	66	
	B	%MVC	0	8.0	11.6	18.8	28.5	36.5	46.5	54.5
		IEMG	3	4	8	11	14	25	33	43
D 12	A	%MVC	1.0	11.3	18.4	28.1	35.0	47.4	53.5	
		IEMG	4	7	7	11	15	22	22	
	B	%MVC	0	9.2	19.3	28.0	35.8	46.0	53.4	
		IEMG	0	4	12	23	34	34	51	

TABLE IX. Showing isometric force (%MVC) corresponding to IEMG (resets/sec.) for days (D) 8 to 12 under either ambient (A) or cooled (B) muscle conditions.

SUBJECT 3

D 8	A	%MVC	4.1	5.5	19.8	33.4	37.4		
		IEMG	0	3	1	8	6		
	B	%MVC	2.5	5.5	12.7	23.2	27.0	32.8	
		IEMG	1	9	13	13	14	14	
D 9	A	%MVC	3.2	11.6	17.6	25.6	28.7	35.5	52.0
		IEMG	1	8	8	20	24	26	34
	B	%MVC	5.7	12.2	17.2	23.2	36.4		
		IEMG	5	16	51	56	75		
D 10	A	%MVC	0	10.0	15.7	30.1	30.3	35.0	47.5
		IEMG	1	12	13	19	26	25	28
	B	%MVC	4.4	11.8	17.5	21.0	27.3	40.3	56.6
		IEMG	1	14	15	23	35	43	45
D 11	A	%MVC	1.6	10.0	20.0	21.6	33.6	40.5	53.0
		IEMG	1	3	6	7	12	14	12
	B	%MVC	5.0	10.0	17.6	25.4	28.2	32.4	42.5
		IEMG	0	2	3	12	16	13	19
D 12	A	%MVC	3.2	12.8	17.5	25.0	30.2	32.6	36.0
		IEMG	2	9	18	10	17	24	21
	B	%MVC	2.0	9.2	12.8	14.9	20.4	15.2	
		IEMG	3	9	4	31	34	32	

TABLE X. Showing isometric force (%MVC) corresponding to IEMG (resets/sec.) for days (D) 8 to 12 under either ambient (A) or cooled (B) muscle conditions.

SUBJECT 4

D 8	A	%MVC	0.7	6.9	20.6	28.5	38.4	48.6	62.0
		IEMG	2	11	15	22	31	37	39
	B	%MVC	2.3	11.0	16.8	28.8	38.4	48.0	57.0
		IEMG	4	9	27	54	50	51	53
D 9	A	%MVC	1.0	9.5	21.2	29.1	42.8	57.0	
		IEMG	3	14	17	27	33	31	
	B	%MVC	1.0	7.9	18.7	31.8	38.8	49.7	59.0
		IEMG	1	12	31	31	43	47	48
D 10	A	%MVC	3.4	9.6	20.3	31.8	38.8	50.5	60.2
		IEMG	1	6	9	11	12	19	17
	B	%MVC	0	9.1	16.7	28.3	38.8	51.5	60.5
		IEMG	2	3	10	21	21	21	26
D 11	A	%MVC	3.3	9.4	18.6	28.2	37.0	46.8	59.8
		IEMG	2	6	13	20	22	27	37
	B	%MVC	2.0	9.2	17.4	28.0	39.0	47.5	
		IEMG	2	9	40	52	96	97	
D 12	not performed								

TABLE XI. Showing isometric force (%MVC) corresponding to IEMG (resets/sec.) for days (D) 8 to 12 under either ambient (A) or cooled (B) muscle conditions.

SUBJECT 5

D 8	A	%MVC	0	8.5	17.0	30.2	42.0	51.0	52.0
		IEMG	2	3	17	13	24	21	26
	B	%MVC	0	7.1	16.7	27.4	38.2	49.5	58.2
		IEMG	1	7	4	23	22	28	36
D 9	A	%MVC	0	12.4	18.6	24.0	35.2	49.0	54.6
		IEMG	2	7	9	17	17	23	29
	B	%MVC	0.5	9.7	21.5	36.2	34.4	47.2	
		IEMG	4	22	36	39	48	47	
D 10	A	%MVC	3.2	6.5	22.0	28.5	39.2	55.5	
		IEMG	2	2	6	8	11	16	
	B	%MVC	0.7	6.2	15.7	21.8	36.0	47.0	55.5
		IEMG	1	3	6	10	14	17	14
D 11	A	%MVC	2.4	8.2	19.0	27.7	38.8	40.0	41.2
		IEMG	1	8	10	16	15	18	16
	B	%MVC	1.2	12.5	19.8	39.4	40.4	47.0	55.4
		IEMG	1	7	10	26	21	27	35
D 12	A	%MVC	1.5	8.8	21.8	35.6	40.4	46.4	54.6
		IEMG	7	8	11	14	17	19	18
	B	%MVC	0	12.9	25.0	28.8	40.0	51.0	62.0
		IEMG	2	18	21	21	27	27	25

Figure 12: Mean force (%MVC) and IEMG (resets/sec.) for each pull under ambient conditions.

Subject 2, trial 4.

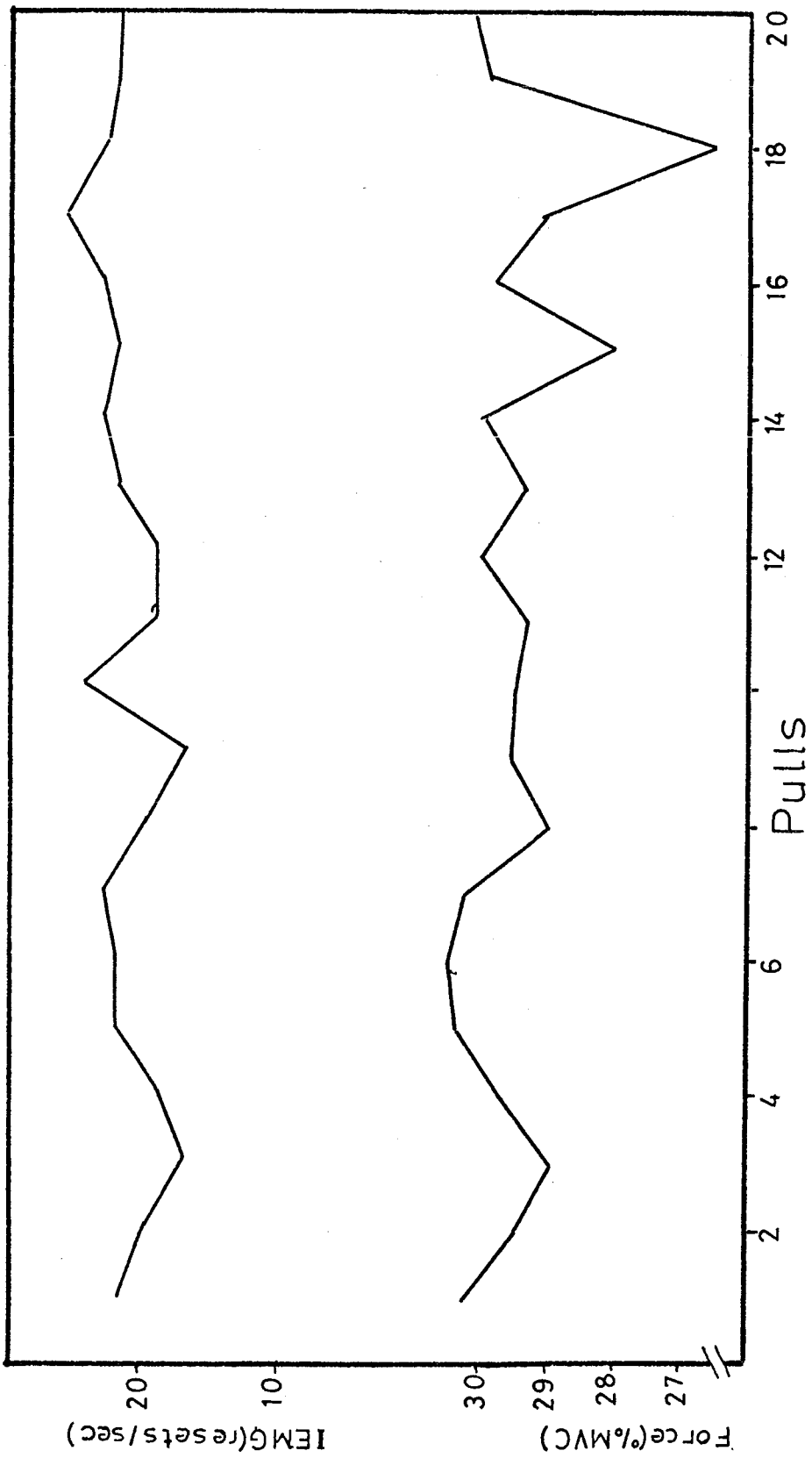
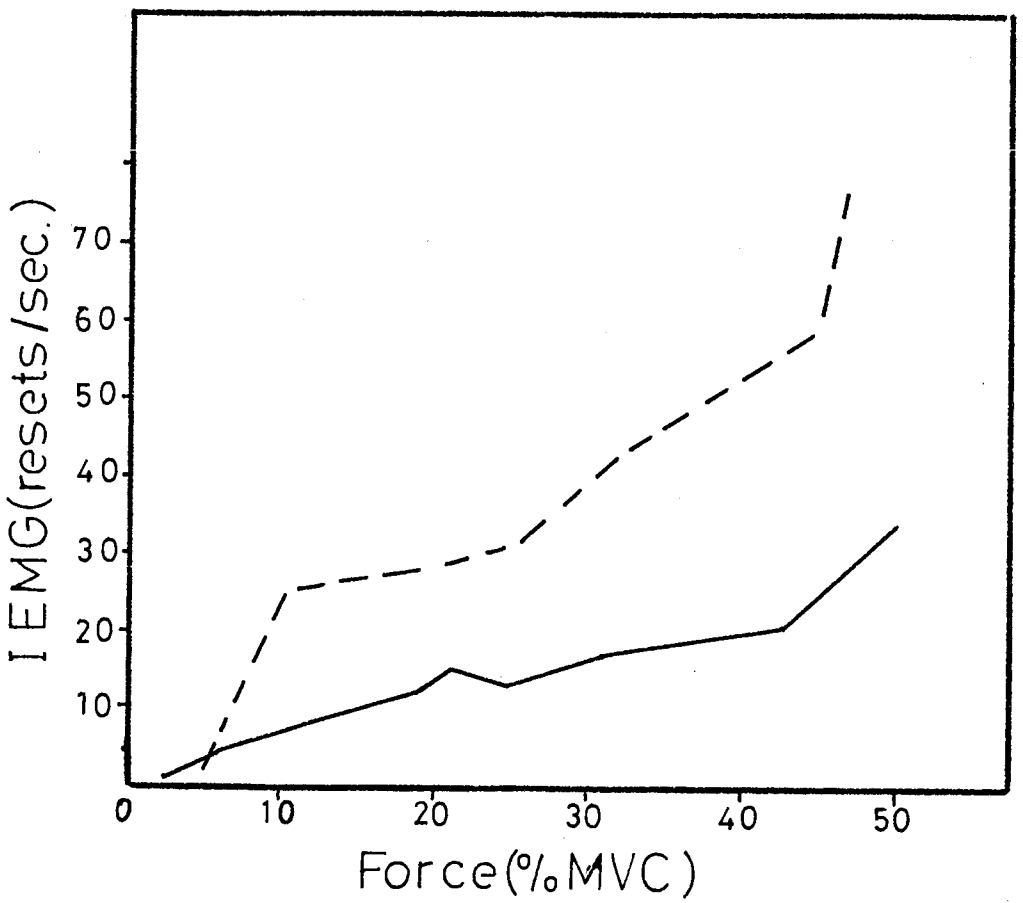

IEMG(resets/sec)
20
10
Force(%MVC)
30
29
28
27
2
4
6
Pulls
12
14
16
18
20

Figure 13: Mean force (%MVC) and IEMG (resets/sec.)
Ambient - solid line
Cooled - dashed line

Subject 1,WT2, Day 10.


IEMG(resets/sec.)
70
60
50
40
30
20
10
0
10
20
30
40
50
Force(%MVC)

CHAPTER V

DISCUSSION

Ability to Produce a Specific Force

Subject 1 (Figures 4 and 9; Table II). The medical information indicated that this subject had a moderate degree of spasticity with limited function. He was receiving intensive rehabilitation and was showing improvement.

In the initial trial this subject showed a range of variability of each pull of 10% of MVC with a mean response (i.e. mean value of all pulls) of 28% of MVC. Close approximation to the target force occurred on pull 5. This level was not maintained and the remaining pulls varied above and below the target force in an irregular pattern. Trial 2 showed a similar irregularity. On trial 3 the variability had decreased to 6% of MVC with a mean response of 30.5% of MVC. This significant improvement in control indicated that learning had occurred. Close approximation of the mean response to the target ($\pm$ 1.5% of MVC) was maintained until trials 11 and 12 when performance declined. The effects of a sudden physical indisposition, which occurred on trial 11 and was still evident on trial 12, could account for this deterioration in performance. As noted by Long et al. (1964) and Roasenda and Ellwood (1961) the management of

spasticity can be complicated by many physical factors. With the exception of these two trials Subject 1 showed an ability to produce a specific force using visual feedback with improvement in control occurring over time.

Subject 2 (Figures 5 and 9, Table III). This subject had a mild degree of spasticity with good function. He was receiving intensive rehabilitation with rapid improvement taking place.

In the initial trial this subject had a range of variability of each pull of 2.5% of MVC with a mean response to the target of 28.5% of MVC. Although close approximation to the target occurred on pull 2 the remaining pulls displayed an irregular pattern with the force produced being consistently below the target force. On trial 2 the subject matched the target force with a mean response of 30.0% of MVC. With the exception of trial 4 this subject produced a mean response of $\pm$ 1.0% of MVC throughout the twelve trials with trial 12 showing significant improvement over trial 1. These results indicate that subject 2 could produce a specific force using visual feedback with improvement in the ability to control that force occurring within one trial. No explanation can be offered to account for the inconsistent result on trial 4.

An interesting observation was made during the course of the experimental procedure. The subject was very conscious that the auditory feedback was an indicator of

muscular activity. In an attempt to relax biceps brachii and achieve a 'silent' muscle in the rest pauses, the subject exerted force in the opposite direction (i.e. contraction of triceps). This effectively silenced biceps brachii but the effect was only maintained while the triceps was being used. As soon as the extensors were relaxed the electrical activity in the flexors returned to the original level. This example of reciprocal relaxation can be compared to the findings of Levine et al. (1954). These authors stated that reciprocal innervation was an important element in reflex movement, especially spasticity. The experimental evidence presented to support this conclusion showed that electrical stimulation of the antagonist muscles was followed by an increase in range of passive movement of a spastic limb. This implied reduction in spasticity. While the reciprocal relaxation observed in the present study comfirmed the theoretical view of Levine et al. (1954) the transient nature of this relaxation conflicted with their experimental evidence. Further electromyographic investigation would be required to clarify this point.

The transient effects of reciprocal relaxation could be converted into a more useful and permanent form of relaxation by providing hemiplegic patients with EMG evidence of the presence or absence of electrical activity in both the spastic and antagonist muscles while isometric contractions were being performed. The work of Jacobs and

Felton (1969) and Hardyck et al. (1966) demonstrated the effectiveness of visual and auditory feedback in obtaining relaxation of normal musculature. As the present study has shown that patients with spasticity respond to these forms of feedback when learning to control gross motor output it is suggested that the same technique can be employed to encourage relaxation in these patients.

Subject 3 (Figures 6 and 10, Table IV). This subject had gross spasticity with very limited function. He had expressive aphasia with no impairment of comprehension. Physiotherapy and Occupational therapy had been discontinued.

The initial trial of this subject presented some very definite trends. As indicated in the previous Chapter the first four pulls were exerted in the opposite direction. This tendency to push instead of pull was also noted by Mecomber and Herman (1971). As the electrical activity was being recorded from biceps brachii it was possible to observe the sequence of events and evaluate this phenomenon. At rest the flexors were almost silent; as the subject attempted to match the target force the electrical activity in biceps brachii increased; visible contraction of the extensors (triceps) also occurred. The extensor contraction was of sufficient strength to overcome the force of biceps brachii causing the dynamometer to register a value for extension. As the subject increased his effort to match the target a corresponding increase in extension occurred.

On the fifth attempt to match the visual signal the force of the flexors was sufficient to counteract the extensors and a positive value for force was registered. This demonstration of co-contraction is at variance with the concept of reciprocal innervation (Levine et al., 1954) but is in agreement with the work of Bobath (1969) who stated that co-contraction occurred when the antagonists of a spastic muscle were stimulated. Bobath proposed that reciprocal innervation was different in a patient with an upper motoneuron lesion to one with a normal CNS. Further investigation involving patients with different levels of spasticity would be required to determine whether co-contraction is confined to patients with gross spasticity.

The results of trial 3, reinforced by the observations of the author, indicated that co-contraction had been reduced and the subject had learned to activate selectively the flexors of the elbow. Close approximation to the target force (mean value 31% of MVC) was achieved by trial 4 and was maintained within a range of $\pm$ 1.5% of MVC for the remaining trials with the exception of trials 5 and 7. The poor response on trial 7 might be attributed to physical causes as the subject developed a severe head cold on the following day. No explanation can be offered for the decline in performance on trial 5. The overall results of this subject demonstrated that visual feedback could be used to teach specific activation of a grossly spastic muscle, reduce co-contraction, and improve the accuracy of control.

Subject 4: (Figures 7 and 11, Table V). This subject exhibited a mild degree of spasticity with moderate functional abilities. Noticeable improvement was occuring with intensive rehabilitation.

The range in variability of each pull on trial 1 was ± 4.0% of MVC. The mean value of 29.5% of MVC constituted a close approximation to the target force. For the entire series of trials this subject maintained an accurate response (mean value ± 1.0% of MVC). The variability of each pull decreased through the first four trials which indicated that repetition of the task had improved the accuracy of production of a specific force. This level of control was held through trials 5 and 6: performance then declined and comparisons of trial 1 and trial 12 showed no significant difference. As the subject performed the task with ease and a high degree of accuracy from the beginning, the decline in performance might have been caused by the inaccurate auditory feedback. If the subject attempted to supplement the visual feedback with the additional information from the IEMG the accuracy of his response might have declined.

Subject 5: (Figures 8 and 11, Table VI). This subject had a moderate degree of spasticity with very limited function. Intensive rehabilitation was being conducted with some success.

This subject showed a variability of ± 4.5% of

MVC on the initial trial with a mean response (29.5% of MVC) in close approximation to the target force. The variability of control shown by this subject followed an irregular pattern on all trials with no significant improvement over the entire training period. Isolated improvement of the mean response was observed on trials 6 and 9. However, as the response on the remaining trials showed no improvement over trial 1 it was concluded that this patient could produce a gross motor response but this ability was not improved with repetition of the task.

In the initial selection of subjects it was noted that subject 5 had good comprehension but his attention span was reduced. The significance of this reduced attention became apparent after the experimental procedure had begun, as the subject had to be constantly reminded to keep his scheduled appointment. His results indicated that his performance of the work task was marred by his inability to concentrate.

Auditory Feedback

This detailed analysis of each subjects ability to produce and control a specific force has been confined to the effects of visual feedback. Inspection of the data indicated that auditory feedback had not provided accurate information.

Integrated EMG was used as the source of auditory feedback. To maintain a discrete signal within the ranges

of force used the signal was maintained between 10 to 40 resets/sec. At this level the IEMG did not accurately reflect the small variations in force produced by the subjects. The random relationship (Figure 12) between IEMG and force implied that the auditory feedback could not have contributed to the results of this study. As both forms of feedback were used simultaneously no attempt can be made to determine whether the inaccurate auditory information interfered with the overall response of the subjects.

IEMG also provided erroneous auditory information when co-contraction occured. The activity in biceps brachii did not correspond to the visual signal being produced. As this lack of correlation only occured on the initial trial of one subject its effects on the experimental procedure were considered negligible.

Auditory feedback may prove useful if the IEMG is presented in a different manner. By changing the gain on both the amplifier and the integrator (yet staying within the limits of linearity of the system) it is possible to produce a larger number of resets per second. If this number is increased to lie within the range of pitch of human beings the degree of muscular activation will vary directly with the note produced by playing the output of the integrator into a loudspeaker. Even if this proves to be a more suitable form of auditory feedback it can only prove useful if the activity in the antagonist muscles

is absent.

Summary of WT 1

From the results of this study it was concluded that visual feedback could be used by hemiplegic patients to produce and control a specific force. This conclusion corroborated the clinical findings of Harris (1970) and Andrews (1964). The use of a dynamometer enabled quantitative measurements to be collected which allowed the gross motor control and the accuracy of that control to be assessed as individual components.

The initial levels of control indicated that the subjects could be divided into two categories. Four subjects, with mild or moderate degrees of spasticity, could use the visual information to produce a gross motor output within 2.0% of MVC of the target force on the first trial. With repetition of the task three of these patients improved their performance by producing the required force with greater precision. This improvement in accuracy of motor output can be compared to the results obtained by Basmajian (1963) who reported that normal subjects could control and regulate the activity of individual motor units when visual and auditory feedback was provided. As the ability to control the motor response of a spastic muscle with a high degree of accuracy could facilitate re-training of fine co-ordinated movements, techniques using some form of visual feedback may be of value in the final stages of

rehabilitation.

The value of visual feedback as a method of obtaining gross motor control was clearly demonstrated in one subject. Co-contraction, evident in the initial trial of subject 3, interfered with the effective production of a specific force. Repetition of the task resulted in a decrease in co-contraction with a concomitant increase in specific activation of the spastic muscle. When control of gross motor output had been achieved this subject improved the precision of his response. The final level of accuracy achieved was within the same range as that shown by the other subjects.

As the gross spasticity and very limited function exhibited by this patient had resisted conventional methods of rehabilitation his response to visual feedback indicated that this technique could be of value in improving his control of motor output. If this control could be gained, the functional capacities of this patient, and other patients who had reached a similar plateau, may be increased.

The overall results of this section of the experiment supported the theoretical concepts of re-integration as presented by Harris (1971). The additional information gained from visual and auditory feedback enabled the subject to produce an accurate motor response. Integration of the sensory information from both internal and external sources can lead to better control of movement.

Effect of Cooling

The ability of the subjects to control an isometric contraction of 30% of MVC remained at the same level after muscle had been cooled. This result is in apparent conflict with the observations by many investigators that local cooling reduced spasticity.

As the procedure used in this study involved rhythmical isometric contractions it is suggested that an increase in blood flow, with a concomitant rise in the muscle temperature, introduced a variable that had not been present in previous investigations. Boes (1962); Hartviksen (1962) and Mecomber and Herman (1971) demonstrated the effect of cooling in reducing spasticity when the limb was in a resting state. Working with normal muscles Humphreys and Lind (1963) showed that muscle temperature decreased when the arm was cooled. During a contraction (30% of MVC) the muscle temperature rose by four degrees centigrade: as the muscle relaxed there was an immediate steep rise in temperature. These temperature changes were due to increase in blood flow. If this increased blood flow occurs when a cooled spastic muscle is actively contracted the different results found in this study compared to the previous reports could be explained. One of the mechanisms postulated for the reduction in spasticity with local cooling is the increase in threshold of the muscle spindle. This occurs when nerve conduction velocity is decreased by lowering the intra-muscular temperature. By increasing

the blood flow to the spastic muscles through contractions the internal temperature may remain near normal, thus counteracting the effect of cooling. As the intra-muscular temperature was not obtained in this study this explanation must remain theoretical.

These empirical observations implied that, as a therapeutic modality, local cooling may not effectively reduce low levels of spasticity when the limb is being actively exercised. As the initial effect of cooling noted with subject 3 could not be dissociated from the concomitant learning that was occuring further investigation would be required to determine the effect of cooling on a grossly spastic muscle.

Efficiency of Electrical Activity

Detailed examination of the interference pattern of the EMG of a normal subject indicated that Integrated EMG was not a suitable measurement to determine the changes in muscular efficiency when different temperature levels were employed.

The electromyogram is a continuous recording of voltage against time; of the electrical activity created when a muscle, or a muscle fibre, contracts. Integration of this action potential, either electronic or planimetric, measures the area between a baseline and the interference pattern. An increase in amplitude and/or duration of the action potential increases the area under the curve and

gives a higher integrated value. If the amplitude remains constant but the duration is increased (as indicated by fewer crossings of the baseline per second) the area under the curve would increase: i.e. when a change in waveform occurs the response of the IEMG will also change (Troup and Chapman, 1972a and 1972b).

Examination of the EMG of a normal subject showed a definite change in the waveform after cooling. At the same levels of force the duration of the action potential was increased. A similar change in waveform after cooling had been noted in both normal muscles (Clarke, Hellon and Lind, 1958) and spastic muscles (Mecomber and Herman. 1971).

Recently a number of techniques of quantification of the interference pattern of the EMG have been investigated (Rose and Willison, 1967; Troup and Chapman, 1972a and 1972b). The aim of these authors has been to describe more completely the waveform of the EMG in both normal and clinical conditions. Troup and Chapman (1972a and 1972b) have shown that measurements of the number of amplitude increments which the waveform makes per second correlates with muscular force equally as well as does the integrated EMG, while the number of spikes of the waveform per second shows an initial increase and then a decrease as muscular force increases. Both muscular fatigue and clinical myopathies have the effect of altering the waveform considerably according to these methods of quantification.

As the form of integration used in this study provided a value for the area below the curve, i.e. voltage/time integral, this change in waveform introduced a variable that prevented comparison of the electrical activity required to produce the same force before and after cooling. Further investigations of the efficiency of electrical activity under different temperature conditions will require a form of quantification of the EMG which interprets the change in waveform.

CHAPTER VI

CONCLUSION

Many rehabilitative techniques using sensory stimulation have been devised to improve the functional capabilities of patients with disturbed or damaged sensory-motor pathways. Based on a review of the current concepts of neuro-muscular integration this study investigated the use of visual and auditory feedback as additional sources of sensory stimulation.

From the results of this experiment it was concluded that under ambient conditions visual and auditory feedback can be used to improve the ability of a hemiplegic patient to produce a specific force.

As the auditory feedback did not provide accurate information to the subjects it was suggested that visual feedback was the main factor responsible for this result. An improved method of providing auditory feedback was noted which would allow the relative importance of these two forms of sensory information to be investigated more thoroughly.

The experimental procedure was conducted over a period of four weeks to determine whether the ability to control a force improved with training. Repetition of the

task enabled four of the subjects to improve their ability to control an isometric contraction. The effect of physical and emotional factors as disruptive influences on this learning pattern were noted.

Quantitative assessment was included in this study to clarify the changes in spasticity and muscular efficiency which have been reported to occur when spastic muscles are cooled.

The ability to produce and control a specific force was not changed by cooling the spastic muscles. It is suggested that the increase in blood flow which occurs when muscles contract rhythmically may have maintained the intra-muscular temperature near the normal level. This would prevent a decrease in nerve conduction velocity which has been postulated as one of the mechanisms responsible for the reduction in spasticity.

The apparent decrease in muscular efficiency with cooling, i.e., a greater amount of EMG required to produce the same force, was subjected to detailed examination. It was concluded that the method of quantification of the electromyogram used in this study was not a suitable method of assessing accurately the changes in waveform that occurred when the muscle was cooled. Alternate methods of quantifying the action potentials were noted which will allow this change in waveform to be analysed more effectively.

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APPENDIXES

```
        *200                            PAL8-V7         PAGE 1

        0200            *200
00200   7300             CLA CLL
00201   1030             TAD FFF1        /IS IT THIS CHANNEL
00202   7440             SZA
00203   5234             JMP CH2         /NO
00204   2034             ISZ FFA         /YES CONVERT THIS LOOP
00205   5234             JMP CH2         /NO
00206   1033             TAD FF1         /YES BUT REINITIALISE
00207   3034             DCA FFA         /CONVERSION SPEED
00210   1042             TAD FIELD1      /WHAT FIELD FOR STORAGE
00211   7440             SZA
00212   5222             JMP CHANGE      /FIELD 1
00213   1041             TAD A           /IS THIS THE END OF FIELD 0
00214   1062             TAD PATCH
00215   7440             SZA
00216   5223             JMP CONT        /NO
00217   1070             TAD DELAY6
00220   3041             DCA A   /YES
00221   2042             ISZ FIELD1      /SET UP FIELD POINTER

00222   6211  CHANGE,    CDF 10
00223   7300  CONT,      CLA CLL
00224   1054             TAD SLECT1      /SELECT CHANNEL+GAIN
00225   4020             JMS AD          /CONVERT AND PUT IN AC
00226   7000             NOP
00227   3441             DCA I A         /STORE
00230   2041             ISZ A           /INCREMENT POINTER
00231   2047             ISZ COUNT1      /COUNT CONVERSIONS
00232   5234             JMP CH2
00233   2052             ISZ COUNT1      /CONVERSIONS>>7777

00234   7300  CH2,       CLA CLL
00235   6201             CDF 0
00236   1031             TAD FFF2        /IS IT THIS CHANNEL
00237   7440             SZA
00240   5256             JMP CH3         /NO
00241   2036             ISZ FF3         /YES,CONVERT THIS TIME?
00242   5256             JMP CH3         /NO
00243   1035             TAD FF2         /YES REINITIALISE
00244   3036             DCA FF3         /CONVERSION SPEED
00245   1044             TAD FIELD2      /IS IT FIELD 1 OR 0
00246   7640             SZA CLA
00247   6211             CDF 10          /FIELD 1
00250   1055             TAD SLECT2      /SELECT CHANNEL+GAIN
00251   4020             JMS AD          /CONVERT AND PUT IN AC
00252   7000             NOP
00253   3443             DCA I B         /STORE
00254   2043             ISZ B           /INCREMENT POINTER
00255   2050             ISZ COUNT2

00256   7300  CH3,CLA    CLL
00257   6201             CDF 0
00260   1032             TAD FFF3        /IS IT THIS CHANNEL
00261   7440             SZA
00262   5300             JMP CH4         /NO
```

```
        *200                            PAL8-V7          PAGE 1-1

00263   2040            ISZ FFC         /YES CONVERT THIS TIME?
00264   5300            JMP CH4         /NO
00265   1037            TAD FF3         /REINITIALISE CONVERSION
00266   3040            DCA FFC         /SPEED
00267   1046            TAD FIELD3      /TEST FIELD
00270   7640            SZA CLA
00271   6211            CDF 10          /FIELD 1
00272   1056            TAD SLECT3      /ETC
00273   4020            JMS AD
00274   7000            NOP
00275   3445            DCA I C
00276   2045            ISZ C
00277   2051            ISZ COUNT3      /COUNT AQUIS+INCREMENT ADDRESSES

00300   6201  CH4,      CDF 0
00301   7300            CLA CLL
00302   1047            TAD COUNT1
00303   7041            CIA
00304   1057            TAD TOTAL       /SUBTRACT REQUIRED AQUISITIONS
00305   7440            SZA     /       FINISHED
00306   5122            JMP GO1         /NO
00307   1052            TAD COUTT1      /POSSIBLY CHECK MSH
00310   7041            CIA     /SUBTRACT MSH FROM TOTAL1
00311   1060            TAD TOTAL1
00312   7440            SZA
00313   5122            JMP GO1
00314   2061            ISZ TOTALG      /END OF GROUP
00315   5317            JMP TIME        /GO TO DELAY LOOP
00316   5777'           JMP 7600
00317   3047  TIME,     DCA COUNT1
00320   3050            DCA COUNT2
00321   3051            DCA COUNT3
00322   7300  LOOP,     CLA CLL
00323   1053            TAD SLECT0
00324   4020            JMS AD
00325   0072            AND MIN         /IS IT ZERO
00326   7450            SNA
00327   5122            JMP GO1 -
00330   5322            JMP LOOP


00377   7600
        0120            *120

00120   7300  GO,       CLA CLL
00121   3074            DCA FF          /SETUP CONTROL FLIPFLOP

00122   7300  GO1,      CLA CLL
00123   6001            ION
00124   1063            TAD DELAY1
00125   3064            DCA DELAY2

00126   7300  CONTROL,          CLA CLL
00127   1053            TAD SLECT0      /CHECK CONTROL CHANNEL LEVEL
00130   4020            JMS AD
```

```
        *200                            PAL8-V7         PAGE 1-2

00131   7000            NOP
00132   0071            AND MAX
00133   7440            SZA             /TO HIGH?
00134   5120            JMP GO          /YES
00135   1053            TAD SLECT0      /NO TRY FOR TO LOW
00136   4020            JMS AD
00137   7000            NOP
00140   0072            AND MIN
00141   7650            SNA CLA         /TO LOW
00142   5120            JMP GO          /YES
00143   2064            ISZ DELAY2      /GO INTO DELAY BEFORE SWITCHING FLB
00144   5126            JMP CONTROL     /DELAY NOT OVER YET
00145   1073            TAD FF4         /TURN ON
00146   3074            DCA FF
00147   5122            JMP GO1         /RETORN BUT DON'T TURN OFF
        0340            *340
00340   7300            CLA CLL
00341   1075            TAD START       /GET TRANSFER NO BY
00342   7041            CIA             /SUBTRACTING START
00343   1076            TAD FINISH      /FROM FINISH ADDRESS
00344   7041            CIA             /SET UP POINTER
00345   3077            DCA NUMBER
00346   1100  AGAIN,     TAD FIELD4      /IS IT FIELD 1
00347   7650            SNA CLA
00350   5353            JMP SKIP                /NO
00351   6211            CDF 10          /NO
00352   7410            SKP
00353   6201  SKIP,     CDF 0
00354   1476            TAD I FINISH    /GET DATA
00355   6211            CDF 10
00356   3501            DCA I NEW       /STORE DATA
00357   6201            CDF 0
00360   1076            TAD FINISH      /DECREMENT COUNTER
00361   1102            TAD F
00362   3076            DCA FINISH
00363   1101            TAD NEW
00364   1102            TAD F
00365   3101            DCA NEW
00366   2077            ISZ NUMBER      /COUNT TRANSFERS
00367   5346            JMP AGAIN
00370   5776'           JMP 7600



00376   7600
        0000    *0

00000   0000            0
00001   3066            DCA DELAY4      /STORE AC
00002   1074            TAD FF          /TEST ON OFF FLIP FLOP
00003   7440            SZA
00004   5777'           JMP 200
00005   1176            TAD (-7
00006   3065            DCA DELAY3
```

```
        *200                            PAL8-V7             PAGE 1-3

00007   2065  WAIT,   ISZ DELAY3
00010   5007          JMP WAIT
00011   1066          TAD DELAY4        /GET AC
00012   6001          ION
00013   5400          JMP I 0


        0020  *20


00020   0000  AD,     0
00021   6535          ADSC
00022   6531  BACK,   ADSF
00023   5022          JMP BACK
00024   6532          ADRB
00025   5420          JMP I AD
        0030  *30

              /CHANNNEL SELECTION 0=YES,1=NO
00030   0000          FFF1,0
00031   0000          FFF2,0
00032   0000          FFF3,0

              /CHANNEL CONVERSION SPEED=1/FFX*INTERRUPT PERIOD
00033   0000          FF1,0
00034   0000          FFA,0
00035   0000          FF2,0
00036   0000          FFB,0
00037   0000          FF3,0
00040   0000          FFC,0


              /ADRESSES FOR STORAGE OF DATA INCLUDING FIELDS

00041   7000          A,7000
00042   0000          FIELD1,0
00043   7000          B,7000
00044   0000          FIELD2,0
00045   7000          C,7000
00046   0000          FIELD3,0

              /COUNTER FOR CONVERSIONS CH1+CH2+CH3,2'S COMPLEMENT.COUTT1

00047   0000          COUNT1,0
00050   0000  COUNT2,0
00051   0000          COUNT3,0
00052   0000          COUTT1,0

              /CHANNEL + GAIN SELECT CODE

00053   0000          SLECT0,0
00054   0000          SLECT1,0
00055   0000          SLECT2,0
00056   0000          SLECT3,0
```

```
                /CONVERSIONS REQUIRED DOUBLE PRECISION,TOTAL=LSH,TOTAL1=MSH

00057  0000             TOTAL,0
00060  0000             TOTAL1,0
00061  0000     TOTALG,0 7777

                /CONTROL DATA


                /DELAY=LOOP TIME*DELAY(2'S COMPLEMENT)
                /FF=CONTROL ON OFF INDICATER
00062  0400             PATCH,0400
00063  0000             DELAY1,0
00064  0000             DELAY2,0
00065  0000             DELAY3,0
00066  0000             DELAY4,0
00067  0000             DELAY5,0
00070  2000             DELAY6,2000
00071  0000             MAX,0
00072  0000             MIN,0
00073  0001             FF4,1
00074  0000             FF,0

                /TRANSFER OF DATA
                /START=START ADDRESS FOR ARRAY
                /FINISH=FINISH ADDRESS FOR ARRAY
                /NUMBER=SIZE OF ARRAY
                /FIELD=FIELD OF ARRAY TO BE TRANSFERRED
                /NEW=LAST ADDRESS FOR NEW ARRAY

00075  0000             START,0
00076  0000             FINISH,0
00077  0000             NUMBER,0
00100  0000             FIELD4,0
00101  7000             NEW,7000
00102  7777             F,7777
                /AD CONVERTER IOT CODES
       6535             ADSC=6535
       6531             ADSF=6531
       6532             ADRB=6532


                $
03176  7771
00177  0200
```

```
A       0041        START   0075
AD      0020        TIME    0317
ADRB    6532        TOTAL   0057
ADSC    6535        TOTALG  0061
ADSF    6531        TOTAL1  0060
AGAIN   0346        WAIT    0007
B       0043
BACK    0022
C       0045
CHANGE  0222
CH2     0234
CH3     0256
CH4     0303
CONT    0223
CONTR0  0126
COUNT1  0047
COUNT2  0050
COUNT3  0051
COUTT1  0052
DELAY1  0063
DELAY2  0064
DELAY3  0065
DELAY4  0066
DELAY5  0067
DELAY6  0070
F       0102
FF      0074
FFA     0034
FFB     0036
FFC     0040
FFF1    0030
FFF2    0031
FFF3    0032
FF1     0033
FF2     0035
FF3     0037
FF4     0073
FIELD1  0042
FIELD2  0044
FIELD3  0046
FIELD4  0100
FINISH  0076
GO      0120
GO1     0122
LOOP    0322
MAX     0071
MIN     0072
NEW     0101
NUMBER  0077
PATCH   0062
SKIP    0353
SLECT0  0053
SLECT1  0054
SLECT2  0055
SLECT3  0056
```

```
.R ASADC

.R ASPROG
?00.00
*W
C-8K FOCAL 01969

01.01 D 31.03
01.02 A "SUBJECT CODE",SU,!
01.03 S S=0
01.05 F I=6144,6343;S S=FCOR(I)+S
01.07 S X=S/200;T %8.04,"X"X,
01.08 S E=0
01.09 F I=6144,6343;S E=(FCOR(I)-X)↑2+E
01.12 S Y=FSQT(E/200);T %8.04,"    Y"Y,

02.02 S N=0
02.03 F I=6464,7462;D 3
02.05 T %4.0,"    N"N,!
02.07 Q

03.03 S X=FCOR(I+1)-FCOR(I);I (X)3.09,3.09,3.05
03.05 I (X-385)3.09,3.09,3.07
03.07 S N=N+1
03.09 R

31.01 S Z=FCOR(1139,3564)
31.02 S Z=FCOR(1139,2409)
31.03 S Z=FCOR(3074,2409)
31.04 S Z=FCOR(3074,3968)
*
```

```
LEVEL 21.6 ( MAY 72 )                    OS/360  FORTRAN H

        COMPILER OPTIONS - NAME=  MAIN,OPT=02,LINECNT=54,SIZE=0000K,
                           SOURCE,EBCDIC,NOLIST,NODECK,LOAD,NOMAP,NOEDIT,NOID,NOXREF
ISN 0002        100 TOTY=0.
ISN 0003            TOTSD=0.
ISN 0004        150 READ(5,1)NSUB,NDAY,NTEMP,NTRIAL,YMEAN,YSD,DUMMY
ISN 0005            IF(NTRIAL.EQ.99)GOTO 200
ISN 0007            TOTY=TOTY+YMEAN
ISN 0008            TOTSD=TOTSD+YSD
ISN 0009            NT=NTRIAL
ISN 0010            GOTO 150
ISN 0011        200 XMEAN=TOTY/NT
ISN 0012            XSD=TOTSD/NT
ISN 0013            XCOVAR=XSD/XMEAN*100
ISN 0014            WRITE(6,2)NSUB,NDAY,NTEMP,XMEAN,XSD,XCOVAR
ISN 0015            WRITE(7,3)NSUB,NDAY,NTEMP,XMEAN,XSD,XCOVAR
ISN 0016            IF(DUMMY.EQ.99)GOTO 300
ISN 0018            GOTO 100
ISN 0019          1 FORMAT(4I2,1X,F3.0,2X,F4.1,59X,F2.0)
ISN 0020          2 FORMAT('0',3(5X,I3),10X,F5.0,10X,F5.2,10X,F6.2)
ISN 0021          3 FORMAT(3I2,F10.0,2(F10.2))
ISN 0022        300 STOP
ISN 0023            END
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LEVEL 21.6 (MAY 72)                    OS/360  FORTRAN H

        COMPILER OPTIONS - NAME=  MAIN,OPT=02,LINECNT=54,SIZE=0000K,
                           SOURCE,EBCDIC,NOLIST,NODECK,LOAD,NOMAP,NOEDIT,NOID,NOXREF
            C**  TWO-FACTOR WITHIN SUBJECTS ANALYSIS OF VARIANCE
            C**  PROGRAM WRITTEN BY J. MONTGOMERY
            C**  REF.  J.L. MYERS - FUNDAMENTALS OF EXP. DESIGN.  PP 82-111
ISN 0002            DIMENSION FORM(20),TITLE(20),FACTA(20),FACTB(20),Y(50,20,10),AB(20
                    1,10),ABSQ(20,10),ABM(20,10),ABVAR(20,10),ABSD(20,10),AS(50,20),BS(
                    250,10),A(20),B(10),ASQ(20),AM(20),AVAR(20),ASD(20),BSQ(10),BM(10),
                    3BVAR(10),BSD(10),S(50),ABSSQ(50,20,10)
ISN 0003            READ(5,1)NANA1
ISN 0004            NDEK=0
ISN 0005        10000 NDEK=NDEK+1
ISN 0006            READ(5,2)FORM
ISN 0007            READ(5,3)NA,NB,NS,(TITLE(I),I=1,17)
ISN 0008            READ(5,2)(FACTA(I),I=1,20)
ISN 0009            READ(5,2)(FACTB(I),I=1,20)
ISN 0010            DO 10010 I=1,NS
ISN 0011            DO 10010 J=1,NA
ISN 0012            DO 10010 K=1,NB
ISN 0013        10010 READ(5,FORM)Y(I,J,K)
ISN 0014            WRITE(6,5)(TITLE(I),I=1,17)
ISN 0015            WRITE(6,6)(FACTA(I),I=1,20)
ISN 0016            WRITE(6,6)(FACTB(I),I=1,20)
ISN 0017            SUM=0.
ISN 0018            SUMSQ=0.
ISN 0019            DO 10020 I=1,NS
ISN 0020            DO 10020 J=1,NA
ISN 0021            DO 10020 K=1,NB
ISN 0022            SUM=SUM+Y(I,J,K)
ISN 0023            SUMSQ=SUMSQ+Y(I,J,K)**2
ISN 0024            ABSSQ(I,J,K)=Y(I,J,K)**2
ISN 0025        10020 CONTINUE
                    C** AB SUMMARY MATRIX
ISN 0026            DO 10040 J=1,NA
ISN 0027            DO 10040 K=1,NB
ISN 0028            ABSQ(J,K)=0.
ISN 0029            AB(J,K)=0.
ISN 0030            DO 10030 I=1,NS
ISN 0031            AB(J,K)=AB(J,K)+Y(I,J,K)
ISN 0032            ABSQ(J,K)=ABSQ(J,K)+ABSSQ(I,J,K)
ISN 0033        10030 CONTINUE
ISN 0034            ABM(J,K)=AB(J,K)/NS
ISN 0035            ABVAR(J,K)=ABSQ(J,K)/NS-ABM(J,K)**2
ISN 0036            ABSD(J,K)=SQRT(ABVAR(J,K))
ISN 0037        10040 CONTINUE
                    C** AS MATRIX
ISN 0038            DO 10060 I=1,NS
ISN 0039            DO 10060 J=1,NA
ISN 0040            AS(I,J)=0.
ISN 0041            DO 10050 K=1,NB
ISN 0042            AS(I,J)=AS(I,J)+Y(I,J,K)
ISN 0043        10050 CONTINUE
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ISN 0044      10060 CONTINUE
              C** BS MATRIX
ISN 0045            DO 10080 I=1,NS
ISN 0046            DO 10080 K=1,NB
ISN 0047            BS(I,K)=0.
ISN 0048            DO 10070 J=1,NA
ISN 0049            BS(I,K)=BS(I,K)+Y(I,J,K)
ISN 0050      10070 CONTINUE
ISN 0051      10080 CONTINUE
ISN 0052            DO 10100 I=1,NA
ISN 0053            A(I)=0.
ISN 0054            ASQ(I)=0.
ISN 0055            DO 10090 J=1,NB
ISN 0056            A(I)=A(I)+AB(I,J)
ISN 0057            ASQ(I)=ASQ(I)+ABSQ(I,J)
ISN 0058      10090 CONTINUE
ISN 0059            AM(I)=A(I)/(NB*NS)
ISN 0060            AVAR(I)=ASQ(I)/(NB*NS)-AM(I)**2
ISN 0061            ASD(I)=SQRT(AVAR(I))
ISN 0062      10100 CONTINUE
ISN 0063            SSA=0.
ISN 0064            DO 10110 I=1,NA
ISN 0065            SSA=SSA+A(I)**2/(NB*NS)
ISN 0066      10110 CONTINUE
ISN 0067            DO 10130 J=1,NB
ISN 0068            B(J)=0.
ISN 0069            BSQ(J)=0.
ISN 0070            DO 10120 I=1,NA
ISN 0071            B(J)=B(J)+AB(I,J)
ISN 0072            BSQ(J)=BSQ(J)+ABSQ(I,J)
ISN 0073      10120 CONTINUE
ISN 0074            BM(J)=B(J)/(NA*NS)
ISN 0075            BVAR(J)=BSQ(J)/(NA*NS)-BM(J)**2
ISN 0076            BSD(J)=SQRT(BVAR(J))
ISN 0077      10130 CONTINUE
ISN 0078            SSB=0.
ISN 0079            DO 10140 J=1,NB
ISN 0080            SSB=SSB+B(J)**2/(NA*NS)
ISN 0081      10140 CONTINUE
ISN 0082            SSS=0.
ISN 0083            DO 10160 I=1,NS
ISN 0084            S(I)=0.
ISN 0085            DO 10150 J=1,NA
ISN 0086            S(I)=S(I)+AS(I,J)
ISN 0087      10150 CONTINUE
ISN 0088            SSS=SSS+S(I)**2/(NA*NB)
ISN 0089      10160 CONTINUE
              C** INTERACTIONS
ISN 0090            SSAB=0.
ISN 0091            DO 10170 I=1,NA
ISN 0092            DO 10170 J=1,NB
ISN 0093            SSAB=SSAB+AB(I,J)**2/NS
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ISN 0094     10170 CONTINUE
ISN 0095           SSAS=0.
ISN 0096           DO 10180 I=1,NS
ISN 0097           DO 10180 J=1,NA
ISN 0098           SSAS=SSAS+AS(I,J)**2/NB
ISN 0099     10180 CONTINUE
ISN 0100           SSBS=0.
ISN 0101           DO 10190 I=1,NS
ISN 0102           DO 10190 K=1,NB
ISN 0103           SSBS=SSBS+BS(I,K)**2/NA
ISN 0104     10190 CONTINUE
ISN 0105           X=SUM**2/(NA*NB*NS)
ISN 0106           SST=SUMSQ-X
ISN 0107           SSA=SSA-X
ISN 0108           SSB=SSB-X
ISN 0109           SSS=SSS-X
ISN 0110           SSAB=SSAB-SSA-SSB-X
ISN 0111           SSAS=SSAS-SSA-SSS-X
ISN 0112           SSBS=SSBS-SSB-SSS-X
ISN 0113           SSABS=SUMSQ-X-SSA-SSB-SSS-SSAB-SSAS-SSBS
ISN 0114           SSRES=SSAS+SSBS+SSABS
ISN 0115           NDFA=NA-1
ISN 0116           NDFB=NB-1
ISN 0117           NDFS=NS-1
ISN 0118           NDFAB=NDFA*NDFB
ISN 0119           NDFAS=NDFA*NDFS
ISN 0120           NDFBS=NDFB*NDFS
ISN 0121           NDFABS=NDFAB*NDFS
ISN 0122           NDFRES=NDFAS+NDFBS+NDFABS
ISN 0123           AMS=SSA/NDFA
ISN 0124           BMS=SSB/NDFB
ISN 0125           SMS=SSS/NDFS
ISN 0126           ABMS=SSAB/NDFAB
ISN 0127           ASMS=SSAS/NDFAS
ISN 0128           BSMS=SSBS/NDFBS
ISN 0129           ABSMS=SSABS/NDFABS
ISN 0130           RESMS=SSRES/NDFRES
ISN 0131           FA=AMS/ASMS
ISN 0132           FB=BMS/BSMS
ISN 0133           FAB=ABMS/ABSMS
ISN 0134           FAR=AMS/RESMS
ISN 0135           FBR=BMS/RESMS
ISN 0136           FABR=ABMS/RESMS
ISN 0137           WRITE(6,26)
ISN 0138           WRITE(6,9)
ISN 0139           WRITE(6,26)
ISN 0140           DO 10200 I=1,NA
ISN 0141     10200 WRITE(6,8)(AB(I,J),J=1,NB)
ISN 0142           WRITE(6,25)
ISN 0143           WRITE(6,10)
ISN 0144           DO 10210 I=1,NA
ISN 0145     10210 WRITE(6,8)(ABS(I,J),J=1,NB)
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ISN 0146            WRITE(6,25)
ISN 0147            WRITE(6,7)
ISN 0148            DO 10220 I=1,NA
ISN 0149      10220 WRITE(6,8)(ABM(I,J),J=1,NB)
ISN 0150            WRITE(6,25)
ISN 0151            WRITE(6,11)
ISN 0152            DO 10230 I=1,NA
ISN 0153      10230 WRITE(6,8)(ABVAR(I,J),J=1,NB)
ISN 0154            WRITE(6,25)
ISN 0155            WRITE(6,12)
ISN 0156            DO 10240 I=1,NA
ISN 0157      10240 WRITE(6,8)(ABSD(I,J),J=1,NB)
ISN 0158            WRITE(6,26)
ISN 0159            WRITE(6,16)
ISN 0160            WRITE(6,8)(A(I),I=1,NA)
ISN 0161            WRITE(6,25)
ISN 0162            WRITE(6,13)
ISN 0163            WRITE(6,8)(AM(I),I=1,NA)
ISN 0164            WRITE(6,25)
ISN 0165            WRITE(6,14)
ISN 0166            WRITE(6,8)(AVAR(I),I=1,NA)
ISN 0167            WRITE(6,25)
ISN 0168            WRITE(6,15)
ISN 0169            WRITE(6,8)(ASD(I),I=1,NA)
ISN 0170            WRITE(6,26)
ISN 0171            WRITE(6,17)
ISN 0172            WRITE(6,8)(B(J),J=1,NB)
ISN 0173            WRITE(6,25)
ISN 0174            WRITE(6,18)
ISN 0175            WRITE(6,8)(BM(J),J=1,NB)
ISN 0176            WRITE(6,25)
ISN 0177            WRITE(6,19)
ISN 0178            WRITE(6,8)(BVAR(J),J=1,NB)
ISN 0179            WRITE(6,25)
ISN 0180            WRITE(6,20)
ISN 0181            WRITE(6,8)(BSD(J),J=1,NB)
ISN 0182            WRITE(6,26)
ISN 0183            WRITE(6,21)
ISN 0184            WRITE(6,22)SSA,NDFA,AMS,FAR,SSB,NDFB,BMS,FBR,SSS,NDFS,SMS,SSAB,NDF
                   1AB,ABMS,FABR,SSRES,NDFRES,RESMS,SST
ISN 0185            WRITE(6,26)
ISN 0186            WRITE(6,23)
ISN 0187            WRITE(6,24)SSA,NDFA,AMS,FA,SSB,NDFB,BMS,FB,SSS,NDFS,SMS,SSAS,NDFAS
                   1,ASMS,SSBS,NDFBS,BSMS,SSAB,NDFAB,ABMS,FAB,SSABS,NDFABS,ABSMS,SST
ISN 0188            WRITE(6,26)
ISN 0189            IF(NDEF.LT.NANAL)GO TO 10000
ISN 0191          1 FORMAT(I4)
ISN 0192          2 FORMAT(20A4)
ISN 0193          3 FORMAT(3I4,17A4)
ISN 0194          4 FORMAT(10(F10.3,2X))
ISN 0195          5 FORMAT('1',T20,17A4)
ISN 0196          6 FORMAT('0',T20,20A4)
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ISN 0197          7 FORMAT('0','MEANS FOR AB MATRIX - ROWS= A, COLUMNS= B')
ISN 0198          8 FORMAT('0',T5,10(F10.3,2X))
ISN 0199          9 FORMAT('0',T10,'SUMS FOR AB MATRIX')
ISN 0200         10 FORMAT('0',T10,'SUMS OF SQUARES FOR AB MATRIX')
ISN 0201         11 FORMAT('0',T10,'VARIANCES FOR AB MATRIX')
ISN 0202         12 FORMAT('0',T10,'STANDARD DEVIATIONS FOR AB MATRIX')
ISN 0203         13 FORMAT('0',T10,'MEANS FOR LEVELS OF A')
ISN 0204         14 FORMAT('0',T10,'VARIANCES FOR LEVELS OF A')
ISN 0205         15 FORMAT('0',T10,'STANDARD DEVIATIONS FOR LEVELS OF A')
ISN 0206         16 FORMAT('1',T10,'SUMS FOR LEVELS OF A')
ISN 0207         17 FORMAT('1',T10,'SUMS FOR LEVELS OF B')
ISN 0208         18 FORMAT('0',T10,'MEANS FOR LEVELS OF B')
ISN 0209         19 FORMAT('0',T10,'VARIANCES FOR LEVELS OF B')
ISN 0210         20 FORMAT('0',T10,'STANDARD DEVIATIONS FOR LEVELS OF B')
ISN 0211         21 FORMAT('1',T50,'SUMMARY OF ANALYSIS OF VARIANCE',///,T40,'ADDITIVE
                   1MODEL - POOLING OF SUBJECT INTERACTIONS',///,T5,'SOURCE',T20,'SUMS
                   2 OF SQUARES',T40,'DEGREES FREEDOM',T60,'MEAN SQUARES',T80,'F-RATIO
                   3S')
ISN 0212         22 FORMAT('0',T5,'A',T25,F12.3,T45,I10,T65,F12.3,T85,F12.4,///,T5,'B',
                   1T25,F12.3,T45,I10,T65,F12.3,T85,F12.4,///,T5,'SUBJECTS',T25,F12.3,T
                   245,I10,T65,F12.3,///,T5,'A X B',T25,F12.3,T45,I10,T65,F12.3,T85,F12
                   3.4,///,T5,'RESIDUAL',T25,F12.3,T45,I10,T65,F12.3,///,T3,'TOTAL',T25,
                   4F12.2)
ISN 0213         23 FORMAT('1',T50,'SUMMARY OF ANALYSIS OF VARIANCE',///,T34,'NON-ADDIT
                   1IVE MODEL - TESTS USING SUBJECT BY FACTOR INTERACTIONS',////,15,'SO
                   2URCE',T20,'SUMS OF SQUARES',T40,'DEGREES FREEDOM',T60,'MEAN SQUARE
                   3S',T80,'F - RATIOS')
ISN 0214         24 FORMAT('0',T5,'A',T25,F12.3,T45,I10,T65,F12.3,T85,F12.4,///,T5,'B',
                   1T25,F12.3,T45,I10,T65,F12.3,T85,F12.4,///,T5,'SUBJECTS',T25,F12.3,T
                   245,I10,T65,F12.3,///,T5,'A X SUBJ.',T25,F12,3,T45,I10,T65,F12.3,///,
                   3T5,'B X SUBJ.',T25,F12.3,T45,I10,T65,F12.3,///,T5,'A X B',T25,F12.3
                   4,T45,I10,T65,F12.3,T85,F12.4,//,T5,'AB X SUBJ.',T25,F12.3,T45,I10,
                   5T65,F12.3,///,T3,'TOTAL',T25,F12.2)
ISN 0215         25 FORMAT('0','----------------------------------------------------
                   1----------')
ISN 0216         26 FORMAT('0','....................................................
                   1..........')
ISN 0217            STOP
ISN 0218            END
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