THE EFFECT OF EMG BIOFEEDBACK ON CEREBRAL PALSY MUSCLE ACTIVITY.

by

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The Effect of EMG Biofeedback on Cerebral Palsy Muscle Activity

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Abstract.

One non-cerebral palsied and six cerebral palsied individuals were subjects in a study investigating the efficacy of electromyographic (EMG) biofeedback for improving movement control. Feedback was given with the intent to increase relaxation in specific antagonist muscles rather than induce global relaxation as in previous studies. During a forearm flexion task experimental subjects received auditory feedback proportional to EMG activity in the triceps muscle. Subjects were instructed to try to reduce the feedback signal and thereby diminish spasticity from unwanted triceps activity. Biceps EMG activity was monitored simultaneously.

Experimental subjects participated in one pre-training session (arm flexion without feedback); six to eight EMG biofeedback training sessions; and a post-training session (arm flexion without feedback). Sessions employed the same forearm movement task with integrated triceps and biceps EMG activity as the dependent measure. Control subjects completed similar sessions but received no EMG feedback during arm movements. They completed six non-feedback sessions as a control for the effects of movement practice without biofeedback.

The data was analyzed individually by subject because of extreme inter-subject variability discovered in EMGs of the C.P. population. Results indicated that all experimental subjects

significantly decreased triceps EMG activity from pre-training to post-training sessions; i.e., after biofeedback training, they could relax the antagonistic muscle to a greater extent when attempting an arm-flexion movement. All control subjects, except one, showed no such effect after practising the same movement without EMG feedback. The control subject who decreased his triceps EMG between pre-training and post-training sessions employed his own relaxation technique contrary to initial instructions.

A trend towards reduction in biceps EMG activity during flexions was also noted for some experimental subjects following triceps feedback. Decreased spastic antagonism may have reduced the effort needed to make the movement.

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This work is dedicated to a person who was a good friend and fan of mine. She supported me through some of the worst days of my university career. For this, and many other reasons, I would like to dedicate this thesis to the memory of Mary Bachelor.

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Introduction.

Cerebral palsy is an "umbrella" term for various, usually birth-related, effects of injuries to the brain, primarily affecting motor functions. It poses many problems for the afflicted individual and for the personnel who must help him or her cope with these dysfunctions. For example, a person with cerebral palsy suffers some degree of motor coordination impairment likely to cause difficulties in eating, dressing, communicating, and generally getting about in the world. These problems entail immense frustrations for the cerebral palsied individual, especially if he/she seeks a high level of independence. The primary task for the professional working with a cerebral palsied individual is to find methods for alleviating the client's coordination problems to facilitate his goal of functional independence. These methods have, to date, drawn from the following: standard physiotherapy (usually with an exercise routine of some sort), behavior modification techniques, electromyographic (EMG) biofeedback techniques, or any combination of the three. This thesis is a preliminary evaluation of a modification of the EMG biofeedback technique most commonly used with cerebral palsied clients, one that shows

promise as an alternative therapy for some patients. First, however, a basic question needs to be asked: What exactly is meant by the term "cerebral palsy"?

"Cerebral palsy" is defined variously by different authorities. For instance, Perlstein (1949) has defined cerebral palsy as "...a condition characterized by paralysis, weakness, incoordination, or any other aberration of motor function due to pathology of the motor control centers of the brain." This definition describes cerebral palsy in terms of physical symptoms only. Another definition, similar to the above "standard definition" but more limited in scope, defines cerebral palsy as "...a condition in which interferences with the motor system arise as a result of lesions from birth trauma." Both definitions can be criticized on two points. First, cerebral palsy is not only a "motor problem" because mental retardation and perceptual/sensory effects are also common (Cruickshank, 1976). Second, it is not always caused by birth trauma: cerebral palsy can also be caused by trauma to the motor cortex resulting from car accidents and/or other misfortunes which involved brain injury to the infant, infections such as meningitis or even increases in cerebral spinal fluid with hydrocephalis (Cruickshank, 1976). These criticisms suggest that the above definitions should be approached with caution. Currently, the term "cerebral palsy" is defined as

one aspect of a larger brain damage syndrome comprising neuromotor dysfunctions, psychological dysfunctions (including mental retardation), convulsions, and other behavioral disorders of organic origin (Cruickshank, 1976). The cerebral palsied individual may be affected by one or more of these factors according to the special nature of his/her condition. While there are psychological and intellectual dysfunctions which affect the cerebral palsied individual, in many cases the individual will have normal psychological and intellectual function but will not have normal neuromotor control over his body.

Motor Dysfunction and Cerebral Palsy:

Cerebral palsy, as one can surmise from the preceding discussion, can be caused by damage to the motor cortex, cerebellum, spinal cord and/or extrapyramidal regions of the brain before, at, or after birth. The damage results in a variety of dysfunctions ranging from mild tremors or speech impairments to severe motor coordination problems and mental retardation. The severity of the condition depends upon the extent and location of the damage.

There are many classifications of cerebral palsy based upon the type of neuromotor dysfunction exhibited. These include spasticity, dyskinesia (which encompasses athetosis, chorea, dystonia, tremor, and rigidity), ataxia, atonia and mixed types. Spastic and athetoid individuals are the most common of these subtypes.

Spasticity is a pathological condition characterized by disharmony of motor function between antagonistic muscle pairs (Denhoff, 1976). In general, it is caused by damage to upper motor neurons (motor cortex or pyramidal tract). It is characterized by an increase in deep muscle reflexes such as the startle reflex (hyperreflexia); hypertonia or increased muscle tone; and abnormal hand-held reflexes and slowness (Denhoff, 1976). The basic problem underlying this component of cerebral palsy is inappropriate activity in the antagonistic muscles during voluntary movement. In normal situations antagonistic muscles are inhibited during voluntary movement.

In contrast to spasticity, athetosis involves problems with involuntary movement. It is characterized by involuntary, exaggerated motor movements accentuated by emotional stress. Jerking, irregular, twisting movements, especially of the wrists and fingers, are apparent except in deep rest

or sleep and during periods of active voluntary motor effort. The extent of the incoordination caused may be profound (Denhoff, 1976). Athetosis is caused mainly by damage to the cerebellum as opposed to the pyramidal and motor cortex damage associated with spasticity.

Spasticity and athetosis do not necessarily appear in their pure form. The cerebral palsied individual often exhibits both spastic and athetoid symptoms. This combination for example may mean that the cerebral palsy patient may have to deal with both incoordination and spasticity.

Reflex Control of Voluntary Movement:

As discussed above, athetosis and spasticity result from damage to motor control centers in the brain, resulting in a loss of control over the muscles involved in voluntary movement. An understanding of how the muscles involved in voluntary movement function is therefore important. Cerebral palsy is basically a muscle control problem. Whatever the problem is (spasticity, athetosis, or other) there is a need to understand how a "normal" muscle contracts and compare that to the way in which a cerebral palsied individual contracts his muscle. This should help in understanding the physiological problem.

Voluntary movement is dependent upon communication between higher brain centers and the sensory and motor fibers of the muscles. This communication is bioelectric in nature and causes one motor unit to contract, or "fire". A motor unit is a single motor axon and the muscle fibers it innervates.

When enough of these units fire in synchrony, the muscle will contract. A simple voluntary movement requires the simultaneous activation of agonist muscles and inhibition of antagonist muscles. The inhibition of antagonist muscles is dependent upon a physiological reflex loop - reciprocal inhibition (Roland, 1978; Rosenzweig & Leiman, 1982).

Reflex control of muscle movement is dependent upon the activity of alpha and gamma motor neurons in the ventral horn of the spinal cord and muscle spindle organs (spindle fibers and golgi tendon organs) of the muscle's intrafusal fibers. These muscle spindle organs provide information on whether the muscle is stretched, contracted or in motion. This afferent information travels from the muscle to the ventral horn motor cells to synapse on alpha motor neurons which innervate the extrafusal fibers of this muscle. This loop forms the basis for the muscle stretch reflex which functions to maintain muscle tone. Gamma motor neurons in

the ventral horn of the spinal cord function to adjust the sensitivity of the muscle spindle fibers. Gamma motor neurons are controlled by pathways from higher brain centers (Rosenzweig & Leiman, 1982). It is the loss of inhibitory control from higher brain centers over the gamma motor neurons which results in some of the characteristic signs of cerebral palsy, namely, spasticity and hyperreflexia (Melyn and Grossman, 1976). The spasticity associated with some classes of cerebral palsy thus may be a result of motor cortex damage affecting the descending control of inhibitory gamma motor neurons.

Athetosis, on the other hand, results from the damage of extrapyramidal centers in the brain (particularly the cerebellum) which are responsible for unconscious movements and posture. Damage to this system is exhibited primarily as inappropriate, involuntary writhing and twisting movements of the distal musculature. Once again, motor disturbances can be thought of as a disruption of communication between higher brain centers and the sensory and motor components of the motor unit.

Partial verification of the communication breakdown between higher brain centers and the muscle's motor units may be found in a study by Harris, Spelman and Hymer (1974).

Harris et al. (1974), investigating a sensory aid design for athetoid cerebral palsied subjects, proposed the concept of "inappropriception" to explain the involuntary movements of their subjects. This concept referred to a defective proprioceptive feedback system resulting in faulty kinesthetic monitoring. The authors suggested that such incoordination problems could be caused by deranged muscle stretch receptor reflexes that distort the information sent to the brain centers (Harris et al., 1974). This suggests that alternative methods may be used to provide proprioceptive information to the brain. One possibility is that artificial proprioceptive information can be provided through electromyographic biofeedback.

Biofeedback:

One of the most frustrating problems faced by the cerebral palsied individual is that he/she has to be in constant battle with his or her muscles in order to perform a task. This problem is demonstrated in a study by Hallett and Alvarez (1983). Fourteen athetoid subjects were given an asynchronous rapid elbow flexion task to perform. EMG measurements were taken from the triceps and biceps muscle of the arm. These experimenters found that, along with the excessive muscle activity accompanying voluntary movement,

there was "... inappropriate activation of muscles both extraneous to the task and directly antagonistic" (Hallett & Alvarez, 1983, p. 745). Thus there was evidence that the triceps was working against the biceps in performing the task.

In the baseline component of their study, Cataldo, Bird and Cunningham (1978) reported similar results to those of Hallett and Alvarez (1983). Similarly, Neilson and O'Dwyer (1984), studying speech muscle activity in athetoid subjects, noticed excessive muscle activity during the voluntary activity component of their experiment.

EMG biofeedback is a technique by which electrical activity of muscles is quantified and fed back to the individual via a monitoring system. This monitoring system, which provides information about muscle activity through different sensory modalities such as auditory signals, may be used to replace or assist the disrupted proprioceptive system. During training the person attempts to learn to control this bio-electrical muscle activity, thereby - it is hoped - learning more precise control of the associated activity, whether it be relaxation or skilled movement (Basmajian, 1975; Love, 1975).

Interest in EMG biofeedback training, as a possible tool to help the athetoid cerebral palsied, was first aroused by an article by Finley, Niman, Standley and Ender (1976). Six athetoid cerebral-palsied individuals participated in this study. Each was affected to differing degrees by his/her condition as indicated by an evaluation of speech and motor functions administered before and after the biofeedback training. The training consisted of monitoring the frontalis muscles and asking the subject to try, by any appropriate means, to reduce the pulsation rate of a high frequency tone, thereby indicating decreasing muscle activity. Subjects received 12 sessions of training and the hope was that learned frontalis relaxation would generalize to other muscle groups, thereby benefitting voluntary movements.

All subjects in the study by Finley et al (1976) decreased their EMG activity levels significantly over the 12 sessions, and all except two of the more severely affected subjects showed significant improvement in the post-test evaluation. Thus Finley et al. (1976) concluded that EMG biofeedback is therapeutically effective. There is no reason to dispute this conclusion, but there remains some question, however, regarding the further conclusion of Finley et al. that these results were due to the general or full-body relaxation achieved by their subjects.

Davis, Brickett, Stern and Kimball (1978), studying electrode placement procedures, monitored EMGs from muscles in "normal" subjects. These researchers found there was no generalization of relaxation from the frontalis muscle so trained to the contralateral frontalis. This result is not surprising in that related studies in the field of learning have identified similar responses. Biofeedback is a highly discriminative procedure. Why should the learned response be expected to generalize as widely as therapists would desire? In other studies (Basmajian,1975; Brudny, Korein, Levidow, Grynbaum, Lieberman & Friedmann, 1974; Love, 1975; Skrotzky, Gallenstein & Osternig,1978; Fernando & Basmajian, 1978), EMG training was found to have more positive results if the motor problem was localized to a specific part of the body as in "foot drag", ankle movement problems, or spasmodic torticollis. Results were more uncertain if the problem was due to anxiety or other generalized relaxation difficulties. The subjects in these studies had different handicaps ranging from cerebral palsy to hysterical paralysis.

Durability of EMG biofeedback effects:

Another question that is frequently asked is: How long does the effect of EMG biofeedback last when compared with other biofeedback techniques? The effects of electromyographic biofeedback seems to last as long as or longer than other relaxation techniques. For example, certain biofeedback techniques need a maintenance program to retain the same level of relaxation because of a short retention period (Ontario Crippled Children's Centre, 1976; Finley, Niman, Standley & Wansley, 1977: Chen, 1983). However, this is not the case for EMG biofeedback . Chen (1983) found that subjects could maintain the same level of frontalis muscle relaxation eight weeks after an initial training period. He used 34 "normal" subjects and three groups: re-training after two weeks; retraining after eight weeks; and no re-training. All subjects retained their relaxation level after the eight-week period and the " two-week re-training" group improved their ability. This suggests that a maintenance procedure is not necessary and that EMG biofeedback is better for long term results.

Hypothesis

The foregoing studies led to the formulation of the following questions for the present research: Can EMG biofeedback training be used profitably as a specific muscle training tool for athetoid/spastic cerebral palsied clients? More specifically, does EMG training of a specific muscle group (triceps) decrease the antagonistic and inappropriate activity of this group during a simple forearm flexion in the cerebral palsied? This study was designed to answer the latter question.

Method

Design:

Subjects in this study were divided into a control and an experimental group, but data analysis was performed on each subject individually because of high intersubject variability. Each subject participated in a pre-training session, either a training or a control activity phase, and a post-training session. The pre-training sessions consisted of obtaining average EMG activity levels for both biceps and triceps muscles without biofeedback. The pre-training sessions usually took about 1.5 hrs for each subject and preceded the first training or control activity session by about 1 week.

Each of the training or control activity sessions were spaced about 2 days apart, depending on the availability of the subject. Control subjects were given no biofeedback during the training phase (control activity phase) and experimental subjects were given feedback during training.

The post-training and pre-training sessions were similar and were held about 1 week after the final training or control activity session. The purpose of the post-

training session was to compare any changes in EMG activity levels with those obtained before training or control activity sessions (i.e., the pre-training session). A diagrammatic representation of the experimental and control data gathering sequences is given in Figures 1 and 2, respectively.

Figure 1:
Experimental Group Data Gathering Sequence

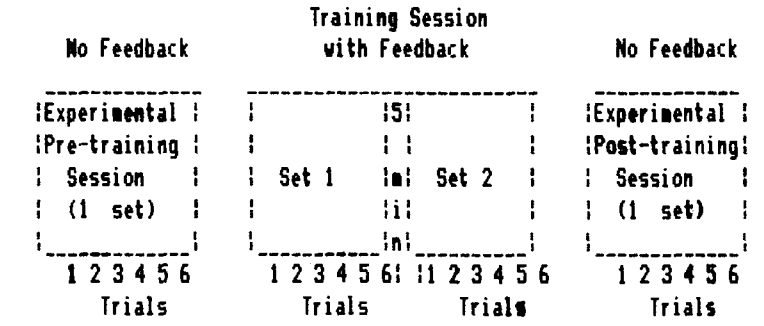
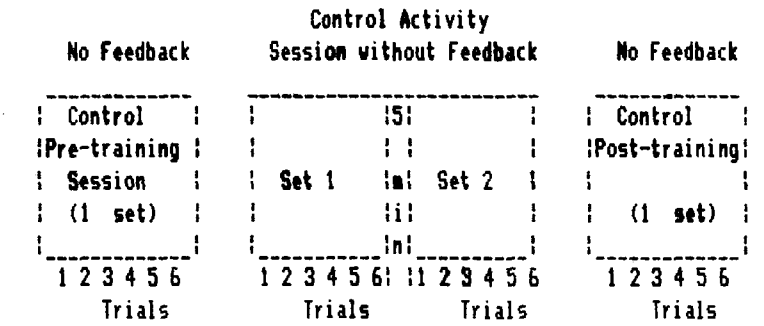


Figure 2:
Control Group Data Gathering Sequence



The purpose of the control group was to determine if any improvement over the baseline in the experimental group was due to the biofeedback training or to some other variable such as simple practice in performing the movement.

A "trial" consisted of a single 10-or a 30-second arm flexing exercise. Duration of a trial was determined by the stamina of the subject which varied considerably in this clinical population. A "set" included six of these trials. Each set was followed by a five minute rest period. "Session" refers to all of the trials completed in one day. The number of sets per session was determined, again, by the stamina of the subject.

The pre-training session for both experimental and control groups consisted of one set of trials; that is, a minimum of six arm flexing movements.

For the experimental group, the training phase consisted of six to eight sessions with subjects completing two to three sets per session. The control group completed six sessions with two sets per session. As stated before, the number of sessions and the number of sets per session was determined by the stamina of the subject.

The post-training session for both the experimental and the control groups consisted of one session with one set of a minimum of six trials.

Subjects:

Subjects for this study were volunteers from Simon Fraser University and from several cerebral palsy associations in the Vancouver area. The experimental group consisted of four subjects with cerebral palsy, ranging in age from 19 to 42 years. The control group consisted of one non-cerebral palsied subject and two cerebral palsied subjects, ranging in age from 23 to 30 years.

Experimental Group: Of the four subjects in the experimental group, three were confined to a wheelchair or scooter, and one could walk without difficulty. Self-classification of the severity of their disorder yielded two reports of severe, one of moderate to severe, and one of mild. The self-report method was used because other known scales of severity were standardized on children, and it was not known whether use of these scales on adults was appropriate. Types of cerebral palsy were also noted: three athetoids and one spastic. Guidelines used in making these determinations were provided by definitions in Cruickshank (1976) and

subjects' self-report. Two subjects were left-handed and two were right-handed. Two were female and two were male. Initials were used to identify subjects to ensure confidentiality. All experimental subjects - except RO - were volunteers from a recruitment campaign conducted at several cerebral palsy associations around the Vancouver area. RO was a student at Simon Fraser University. Further detailed information regarding subjects is listed in Table 1.

A total of eight subjects had originally been included in the experimental group. However, four individuals were subsequently eliminated from the study. One subject left the study for personal reasons. The other three were judged to be too severely affected to perform the training task.

Control Group: Subjects for this group included one non-motor-impaired individual and two cerebral palsied individuals. The non-motor-impaired subject was a psychology student at Simon Fraser University. She was an athlete, right-handed, and was recruited by personal contact. To preserve anonymity in this study she is refered to as SH.

Table 1: Subject's characteristics.

Subject	Sex	Age	Subject's Assessment of C.P. severity	Type of Cerebral Palsy	Arm Used For Study	Can Subject Use exp. Chair?	Does Subject Use a W.chair?	Comments
RO	male	n/a	mild	athetoid	right	yes	no	little extraneous movements Subject did not practise relaxation techniques at home.
TR	female	42	severe	athetoid	left	no	yes	Could not keep elbow on arm rest. Had prior experience with biofeedback general relaxation paradigm. She used techniques at home to relax.
RA	male	n/a	moderate/ severe	athetoid	right	yes	no	Had good muscle development, good stamina. Had up-and-down spasms in his movements. He tried to relax at home before coming in for his sessions. He was a wheel-chair athlete.
JE	female	30	severe	spastic	left	no	yes	Had trouble keeping her elbow on armrest. She used her body as a lever to accomplish movement. Did not practise any home relaxation techniques. She had much extraneous movements. A possible hearing loss may account for delays in reaction to commands.
SH	female	23	non C.P.		right			
PA	male	30	profound	spastic	right	yes	yes	Used visualization. Possible confounding factor.
DE	male	30	moderate	athetoid	right	yes	yes	Chief experimenter. Basic information sought so confound is minimized.

Of the cerebral palsied controls, PA, was male and was diagnosed as having profound (between moderate and severe) symptoms of spastic cerebral palsy. He was right-handed. He used a scooter-type wheelchair but could transfer from his scooter to the experimental chair with assistance. PA expressed a keen interest in the study but had no prior experience with EMG biofeedback. He was recruited from the membership list of a large cerebral palsy organization in Vancouver, B.C. He was recruited in person, and described himself as an "electronics specialist."

The other cerebral palsied control subject was a moderately-afflicted athetoid quadriplegic who used a scooter-type wheelchair. He walked with relative ease and could transfer from scooter to experimental chair unassisted. His hand of preference was the left, although use of his right hand in the experiment presented no problems with respect to the task required. He was quite familiar with EMG biofeedback, being the chief experimenter on this project. Because the control group only provided basic information for comparison and received no biofeedback, any confounds caused by his knowledge of and experience with the procedure would be minimal.

Apparatus and Materials:

A "trial" consisted of a single continuous arm flexion movement which took 10 or 30 seconds to complete depending on the stamina of the subject. A "set" contained six trials. Each set was followed by a five minute rest period. "Session" refers to all of the trials completed in one day. The goal at the outset was to run eight sessions - on different days - for each subject. The number of sets per session was determined, again, by the stamina of the subject. TR and RO completed six sessions each. RO completed three sets each session, TR was only able to complete two sets per session. RA and JE completed eight sessions with three and two sets, respectively. The control group completed six sessions with two sets per session.

Equipment: Two Grass Model 9 four-channel polygraphs and one Autogen 1700 biofeedback system were used in this study.

a)EMG Recording

The modules for recording electromyographs (EMGs) included eight Grass Model 79 7P5 Wide Band Pre-amplifiers coupled to eight 7DA DC driver amplifiers and associated pen channels of a strip chart recorder. The output from selected channels of the amplifiers was fed into two Grass

Summing Integrators (for triceps: model #7P 10E; for biceps: model #7P 10C) whose output was recorded on separate channels of the chart paper.

All units were calibrated according to the applicable Grass manuals at the commencement of each day's trials. Two Grass 4-channel chart recorders provided a hard copy of the data from which dependent measures were calculated manually.

The integrator averages the raw EMG activity and provides a peak summation of the data. The output of the integrator is a rectified summation of the bioelectrical activity of the monitored muscles. According to the Grass Instruction Manual(1978), each peak corresponds to nine microvolts as per the setting on the integrator. Each 9-microvolt peak constitutes one reset of the integrator and the slope of the reset is directly proportional to the output activity.

b)EMG Biofeedback.

One Autogen Model 1700 with battery pack provided subjects with EMG feedback from the triceps muscle. Two stereo headphones were used so that both experimenter and subject could monitor the Autogen signal. The feedback to the sub-

ject was a high frequency tone designed to pulsate more quickly when muscle activity was increasing and to pulsate more slowly when muscle tension was decreasing.

c)Electrodes and Connections.

Two pairs of disposable silver-silver chloride electrodes transduced EMG signals. They were utilized in combination with a fifth electrode of the same type used as a ground electrode (attached at the bony part of the wrist of the same arm used in the experiment).

Electrode sites were cleaned with alcohol scrub and skin abrasion prior to electrode attachment. Beckman silver-silver chloride disposable unit electrodes were used in conjunction with snap-on leads to pick up triceps and biceps muscle activity. After attachment of electrodes, impedance was measured between each active electrode and the ground electrode to ensure that impedance was no higher than 10K ohms.

Electrodes were placed in parallel with the line of the biceps and triceps muscle fibers. Since overall muscle tension/relaxation information was being sought, the electrodes were spaced as widely as the subject's arm size would permit.

The EMG electrode leads were fed to the 3-post 7P5 cables with the cables connecting to two junction boxes. The triceps muscle EMG signal was fed to one pre-amplifier of the first Grass Polygraph (GP1), and the biceps EMG signal was fed to one of the pre-amplifiers of Grass Polygraph 2 (GP2). The raw triceps EMG was also fed to the Autogen through a second pre-amplifier on GP2 while the Autogen feedback signal was recorded on a separate channel of GP1.

The remaining polygraph channels were dedicated to potentiometer readings from the arm flexion apparatus (to indicate onset and duration of movements), time indicators, and integrated EMG signals derived from the raw EMG channels. Some of the outputs were cross-referenced during the original pre-training experimental group sessions to provide the experimenters with assurance that the two Grass Units were recording exactly the same data. After this validation was obtained, and it was determined that extra channels would be needed for the post-training sessions, the practice was terminated.

d)Recording Site, Subject Positioning and Flexion Apparatus.

Subjects sat in a sound-reduced electrically-shielded room and were observed through a one-way mirror. The mirror permitted subject observation with minimal outside distraction. Communication with subjects was maintained through a wall-mounted intercom system. Subjects were seated in an upholstered chair with a wide right armrest through which a vertical hole had been drilled. The hole accommodated a one-metre nylon line to which a one-kilogram weight was fastened at the bottom end. Travel of the nylon pullcord during a flexion-relaxation movement caused the shaft of a potentiometer to rotate. The resulting change in current across the potentiometer indicated the onset of and duration of each movement. This signal was fed into one channel of each polygraph. To stiffen the wrist joint, experimental subjects had a flat metal plate strapped to the back of their right wrist and hand. The plate was then attached to the nylon cord and weight. For the same purpose control subjects had a towel wrapped around their right wrist which was also attached to the one-meter nylon cord and weight (the metal plate was uncomfortable for control subjects and so the restraining method was changed). This change should not effect the results since both methods restrained movement of

the subject's wrist equally well. This system allowed subjects to perform the desired arm movement against a load and for movement onset and offset to be recorded automatically.

The movement consisted of flexion of the biceps muscle, raising the forearm from resting horizontally on the armrest up to the shoulder. A record of the time of the start and end of each movement was derived from the voltage changes across a potentiometer whose shaft was rotated by movement of the pull line.

During the biofeedback test a Sankyo Stopwatch Model I-60 was used to time both the up-and-down arm movements and the rest periods. The Grass timers - used in other phases of the experiment - had been repositioned to accommodate the biofeedback equipment and were consequently inaccessible to the experimenter.

Measures:

The intent of this study was to measure changes in muscle relaxation following EMG biofeedback.

"Resets per second" of the EMG integrator channels was adopted as a dependent measure (rather than raw EMG voltage) for ease of calculation and because increases and decreases

in muscle activity are directly related to increases and decreases of resets per second (Grass Instruction Manual, 1978).

Procedure:

After giving the subject written and verbal instructions regarding the goals of the experiment (Appendix 1), and after obtaining written consent, the subject was either seated in the upholstered chair or placed beside the right armrest, seated in his/her wheelchair.

Electrodes were placed on either the left or right arm - whichever was closest to the armrest - and the subject was asked to place the back of his/her hand and arm on the armrest. The experimenter then immobilized the subject's wrist by wrapping it with a folded hand towel (control group) or metal plate (experimental group) and secured the weighted cord to the wrist. For the experimental group, the subject placed his/her hand and wrist on a metal plate and the wrist and hand were taped to the plate. Subjects were instructed to keep their elbow on the armrest as much as possible. The experimenter left the room and allowed the subject to relax for a few minutes while he checked the equipment .

The experimenter then gave the following sequence of commands to the subject: "flex", "hold", "down" and "relax". The experimenter waited 30 seconds between commands; the sequence was repeated six times. It had been explained to subjects that "flex" meant that, using as little effort as possible, the subject must bring the arm from a hyperextended horizontal position through approximately 120 degrees to bring his hand up to his shoulder. The subject was asked to hold this position for 10 or 30 seconds. "Down" meant to return the arm to the horizontal position. The experimenter reset the integrator before giving the next "flex" command. All subjects received identical sets of instructions whether they were in the control (no feedback) or experimental (feedback) groups. Debriefings were also given at the completion of the final post-training (Baseline 2) session.

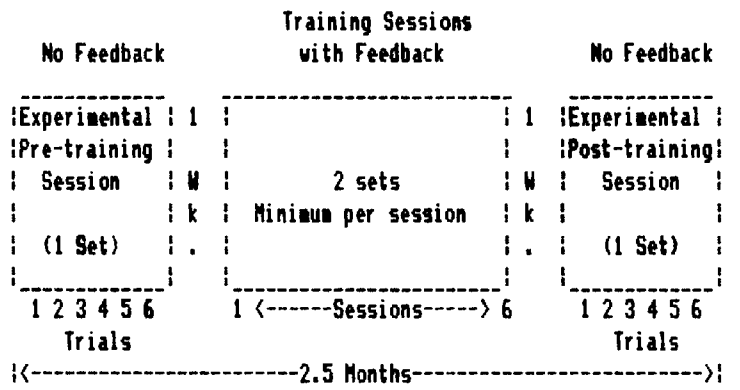
During all sessions (pre-training, experimental training and control activity, and post-training), biceps and triceps activity were being recorded by the Grass polygraph units. Both amplitude (voltage) changes and integrator resets (amplitude summation), were obtained from both muscles.

Feedback Training session: Earphones were placed over the subject's ears. The subject heard a high frequency tone designed to "beep" fast when the activity of the monitored muscle was high and to "beep" slowly when the activity was low. Subjects were asked to slowly flex and relax the arm a number of times on command while it was attached to the weighted cord. It was explained that the task during these trials was to reduce the frequency from a fast-beeping tone to a slow-beeping tone. Subjects were also asked to refrain from making any sudden movements or from talking during the test trials, as this would affect the results. See Appendix 1 for a more complete discussion of the purpose of the biofeedback trials and a summary of the instructions given to the subject regarding the auditory feedback signal.

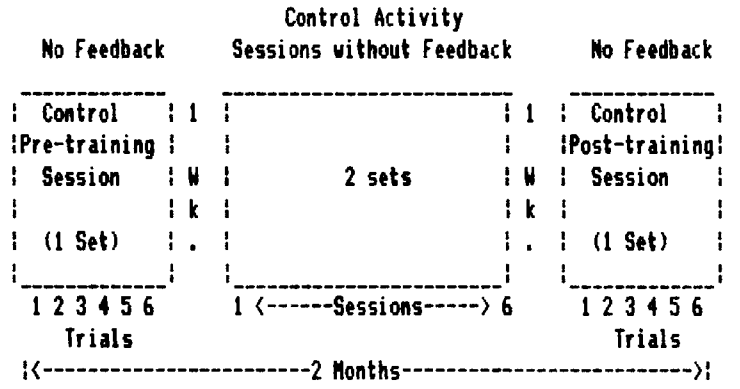
When the experimenter was satisfied that the system was working well, a second experimenter started the commands "flex", followed by "hold" (for 10 or 30 seconds, depending on the subject's stamina), then "down", and "relax" (for 30 seconds). The routine was repeated six times (six trials) followed by a five minute break, during which the equipment was checked again. This procedure was repeated for a maximum of three times (three sets). The experimenter recorded Autogen maximum and minimum readings for each movement and a third experimenter observed the subjects for extraneous movements. Time was monitored with a stopwatch.

The schedule for the feedback training sessions was originally set at two sessions per week (with at least one day between sessions). However, this schedule was flexible to accommodate each subject's varying time commitments. The training sessions were held over a two-month period. The entire feedback group completed their portion of the experiment within 2.5 months (Figure 3).

Figure 3:
Time Frame for Experimental Group (with Biofeedback)



Time Frame for Control Group (without Feedback)



Control (non-feedback) Group:

Pre-training Session: All subjects in the control group participated in a pre-training session which, like the experimental group, did not involve feedback. This session - which consisted of a single set (six trials) - was obtained approximately one week prior to the first control activity session. However, like that of the experimental group, the schedule for the pre-training recordings was made flexible to accommodate each subject (Figure 3).

Some subjects experienced much discomfort with the metal plate used to immobilize the wrist. For the control group, the plate was replaced with a folded towel secured to the wrist by several pieces of string. The change appeared to lessen the discomfort felt and did not seem to affect the other characteristics of the pull.

Non-feedback Control Activity Sessions: Control group subjects were given the same initial instructions as the experimental subjects regarding the arm flexion movements. However, since no feedback was given, they were instructed to not employ any form of relaxation technique and simply to attempt to produce a smooth, controlled arm movement. After equipment calibration and set-up, the commands "flex",

"hold" (for 10 or 30 seconds, depending on the subject's stamina), "down", and "relax" (for 30 seconds) were issued. The routine was repeated six times (six trials) followed by a five minute break during which the equipment was rechecked. This procedure was repeated for a maximum of two times (two sets). Subjects were also asked to refrain from making any sudden movements or from talking during the test trials, as this would affect the results.

As with the experimental group the schedule for the non-feedback control activity sessions was originally set for two sessions per week (with at least one day between sessions). However, this schedule was also flexible to accommodate each subjects own time schedules. The entire non-feedback control activity sessions was completed over a period of two months.

Post-training Session: The recordings for the control group's post-training session were obtained approximately one week following the last non-feedback control activity session. These sessions were identical to the pre-training sessions in the instructions and procedures followed. Once again the schedule for these recordings was made flexible to accommodate each control subject's schedule.

Results.

Due to problems of extreme intersubject variability that became apparent while running this study, analysis of the data had to be limited to single-subject pre-training/post-training Student's T tests; two-way repeated measures analyses of variance (ANOVAs) for the training sessions; and a qualitative discussion of interesting patterns within the data. Group analysis of the data was rejected due to high intersubject variability. This variability was impossible to avoid given the nature of the cerebral palsy subject pool available. This decision left a single-subject analysis for the training as well as for the pre/post-training sessions.

Pre/Post Training Triceps Comparisons -Experimental Subjects

As predicted, all of the experimental subjects showed a significant decline in triceps EMG activity between pre-training and post-training sessions for the triceps muscle group. T statistics for JE, RA, RO, and TR were 4.37, 7.47, 9.81 and 4.37 respectively, significant at alpha = 0.05, (Table 2). This indicates that there was a training effect for the triceps during the EMG biofeedback sessions for the four experimental subjects. "Training effect" refers to the decline of EMG activity, as indicated by the number of integrator resets, in the antagonistic muscle over the

course of training. Inability to relax antagonists, it will be recalled, resulted in spasticity of movements in these subjects.

Table 2: T Test Results of Mean EMG Differences in All Subjects' Triceps Activity.

Condition	Subject	Test Mean	df	T Statistic	Alpha	Significance
No Feedback (Control)	SH	0.0587	5	2.43	0.05	N. S.
	DE	0.0495	5	1.54	0.05	N. S.
	PA	0.0852	5	6.51	0.05	S.
Feedback Subjects (Experimental)	JE	0.2356	4	4.37	0.05	S.
	RA	0.4317	5	7.47	0.05	S.
	RO	0.5783	5	9.81	0.05	S.
	TR	0.1390	5	4.37	0.05	S.

Pre/Post Training Triceps Comparisons -Control Subjects.

Two of the three control subjects showed no significant difference between pre-training and post-training triceps EMG activity, which coincides with a priori expectations. SH and DE were non-significant at the alpha = 0.05 level. However, PA showed a significant difference at the alpha = 0.05 level. T statistics for these three subjects were 2.43, 1.54 and 6.51, respectively (Table 2). These results suggest that DE and SH had no practice effect due to interpolated activity between pre-training sessions and post-training sessions, while PA showed some practice effect, even though no biofeedback was given to any control subjects.

Pre/Post Training Triceps Summary.

All four of the experimental subjects decreased their triceps EMG activity which is consistent with a priori expectations. RA and RO exhibited the most improvement while JE and TR showed the least although their t statistics were still significant, ($p.< 0.05$). Although one of the control subjects (PA), showed an unexpected significant decrease, ($p.< 0.05$) in triceps EMG activity without any biofeedback, SH and DE were non-significant, ($p.> 0.05$).

Pre/Post Training Biceps Comparisons-Experimental Subjects.

Three experimental subjects showed a significant difference, ($p.< 0.05$), between the biceps activity of pre-training and post-training sessions which is contrary to a priori expectations. JE, TR and RA are significant at the alpha = 0.05 level. T statistics for these three subjects are 15.40, 13.22 and 7.02, respectively. RO showed an expected non-significant difference in biceps activity, ($p. > 0.05$), with a t value of 1.52. T statistics for these subjects are summarized in Table 3.

Pre/Post Training Biceps Comparisons-Control Subjects.

Only one control subject showed a significant, ($p. < 0.05$), t test difference between pre-training and post-training biceps EMG activity (Table 3). SH was significant at the alpha = 0.05 level with a t test value of -2.92. Once again, this is contrary to a priori expectations since no feedback was given for the biceps muscles in either the experimental or control groups. DE and PA were non-significant, ($p. > 0.05$), with t statistics of -0.19 and 1.53 respectively (Table 3). DE and PA's data are consistent with a priori expectations.

Table 3: T Test Results of Mean EMG Differences in All Subjects' Biceps Activity.

Condition	Subject	Test Mean	df	t Statistic	Alpha	Significance
No Feedback (Control)	SH	-0.0907	6	-2.92	.05	S.
	DE	-0.0120	6	-0.19	.05	N.S.
	PA	0.0348	6	1.53	.05	N.S.
Feedback Subjects (Experimental)	JE	0.7574	5	15.40	.05	S.
	RA	0.1815	6	7.02	.05	S.
	RO	0.0552	6	1.52	.05	N.S.
	TR	0.6795	6	13.22	.05	S.

Pre/Post Training Biceps Summary.

The foregoing results for the biceps EMG data are not completely consistent with a priori expectations since no feedback was given to the subjects for the biceps muscle

group in either the control or the experimental conditions. Although two of the control subjects did not exhibit a significant decrease in their biceps activity (as expected), SH increased her biceps activity significantly, (p.< 0.05), and RA, JE and TR, of the experimental group, showed a significant, (p.< 0.05), decrease in biceps muscle activity. RO was the only experimental subject who did not show a significant, (p.> 0.05), decrease in biceps activity.

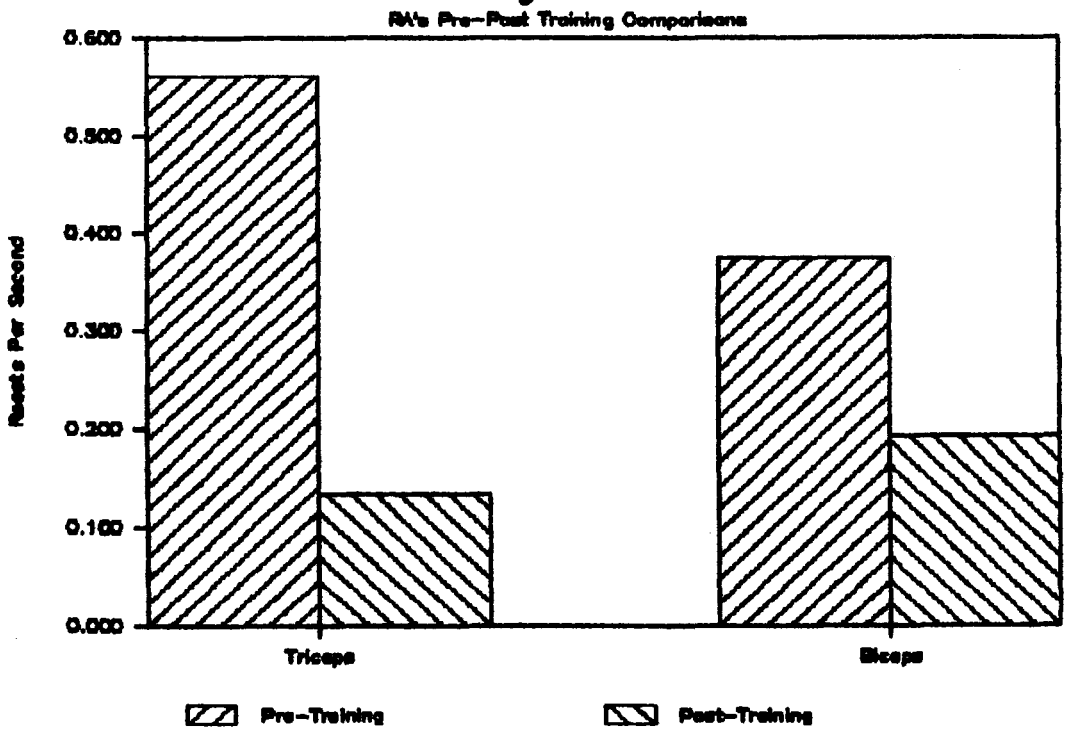
Since no feedback was given regarding biceps muscle activity, the biceps comparison was given primarily as an interesting interpretation of the data. The biceps pre/post training comparisons are difficult to interpret. One control subject and three experimental subjects showed a significant difference between the mean biceps activity of the pre-training session and the post-training session. Two controls and one experimental subject have non-significant differences in biceps activity for the pre/post training sessions.

Summary of Graphs

The foregoing results are visually depicted in the graphs comparing pre-training and post-training triceps EMG

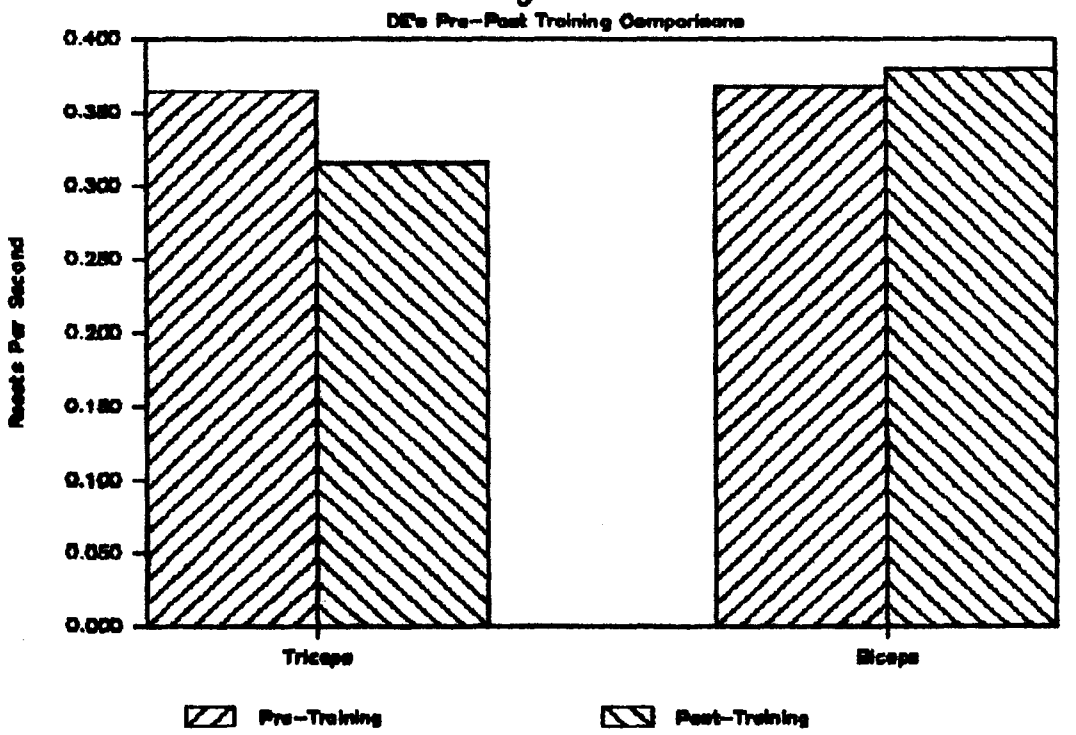
activity for both experimental and control groups (Appendix 2, Set 1). Selected subjects' results are reproduced here as representative observations. RA's (experimental group) triceps activity showed the largest significant decrease in the interval between the pre-training and post-training sessions. Mean resets-per-second averaged over the six trials (for triceps) were 0.563 for the pre-training session and 0.135 for the post-training session (Figure 4). RA's biceps results are also represented in Figure 4, indicating the unexpected significant EMG decrease (measured by resets-per-second) from a pre-training level of 0.375 to 0.193 for the post-training session.

Figure 4:



A representative graphic depiction of a control subject's pre/post training data (DE) is presented in Figure 5 below. DE's average EMG (measured by resets-per-second) during the pre-training session was 0.365 which showed a non-significant (p. > 0.05) decrease to 0.315 resets-per-second for the session. Biceps EMG activity for DE also showed an expected non-significant increase from 0.369 resets-per-second during the pre-training session to 0.381 for the post-training session. Comparison of DE's biceps data for the pre and post-training sessions are also represented graphically in Figure 5. Graphs for other control subjects are given in Set 1 of Appendix 2.

Figure 5:



Experimental subjects' triceps and biceps mean EMG activity for pre-training, training, and post-training sessions are summarized in Table 4, below. Although it is apparent that there is a certain degree of variability from one training session to the next, the t test results comparing the pre-training and post-training sessions were consistent with a priori expectations. As was described elsewhere, all four of the experimental subjects showed an expected decrease in triceps EMG activity from pre-training to post-training sessions. Mean biceps EMG activity for all experimental subjects are also depicted in Table 4.

Table 4: Mean Triceps and Biceps Activity for All Sessions.

Means of Pre-Training, Training and Post Training Sessions Experimental Group								
Subject:	JE	JE	RA	RA	RO	RO	TR	TR
Muscle:	Biceps	Triceps	Biceps	Triceps	Biceps	Triceps	Biceps	Triceps
Session:								
Pre Training:	0.876	0.366	0.375	0.563	0.795	0.351	1.082	0.425
Trn 1:	0.443	0.183	0.227	0.123	0.321	0.147	0.399	0.286
Trn 2:	0.663	0.373	0.254	0.115	1.015	0.396	0.973	0.262
Trn 3:	1.499	0.426	0.259	0.097	0.665	0.478	0.570	0.522
Trn 4:	0.829	0.417	0.413	0.135	0.969	0.296	0.458	0.118
Trn 5:	0.617	0.189	0.850	0.126	0.676	0.355	0.476	0.184
Trn 6:	0.681	n/a	0.602	0.114	0.707	0.420	0.602	0.127
Trn 7:	0.235	0.378	0.900	0.159	0.796	0.511		
Trn 8:	0.679	0.350	0.396	0.105				
Post Training	0.125	0.131	0.193	0.135	0.736	0.296	0.402	0.286

A summary of all control subjects' triceps and biceps EMG activity is given on Table 5, below. Although there is also a certain degree of variability from training session one through six for all subjects, an expected non-significant difference was still obtained between pre-training and post-training triceps EMG measures for subjects SH and DE (Table 5). Biceps EMG activity between pre/post-training sessions were also found to be non-significant (as expected) for subjects DE and PA (Table 5).

Table 5: Mean Triceps and Biceps Activity for All Sessions.

Means of Pre-Training, Training and Post-Training Sessions Control Subjects						
Subject:	SH	SH	DE	DE	PA	PA
Muscle:	Biceps	Triceps	Biceps	Triceps	Biceps	Triceps
Session:						
Pre-Training:	0.553	0.440	0.369	0.365	0.311	0.357
Trn 1:	0.536	0.750	0.449	0.409	0.264	0.360
Trn 2:	0.682	0.643	0.376	0.346	0.195	0.364
Trn 3:	0.632	0.400	0.353	0.322	0.341	0.331
Trn 4:	0.744	0.565	0.342	0.306	0.372	0.376
Trn 5:	0.560	0.589	0.189	0.233	0.224	0.254
Trn 6:	0.538	0.463	0.294	0.283	0.252	0.304
Post-Training:	0.643	0.381	0.381	0.315	0.276	0.271

Appendix 2 depicts the data in graphic form for all subjects. Set 1 shows pre-training and post-training comparisons for all experimental and control subjects. Bar

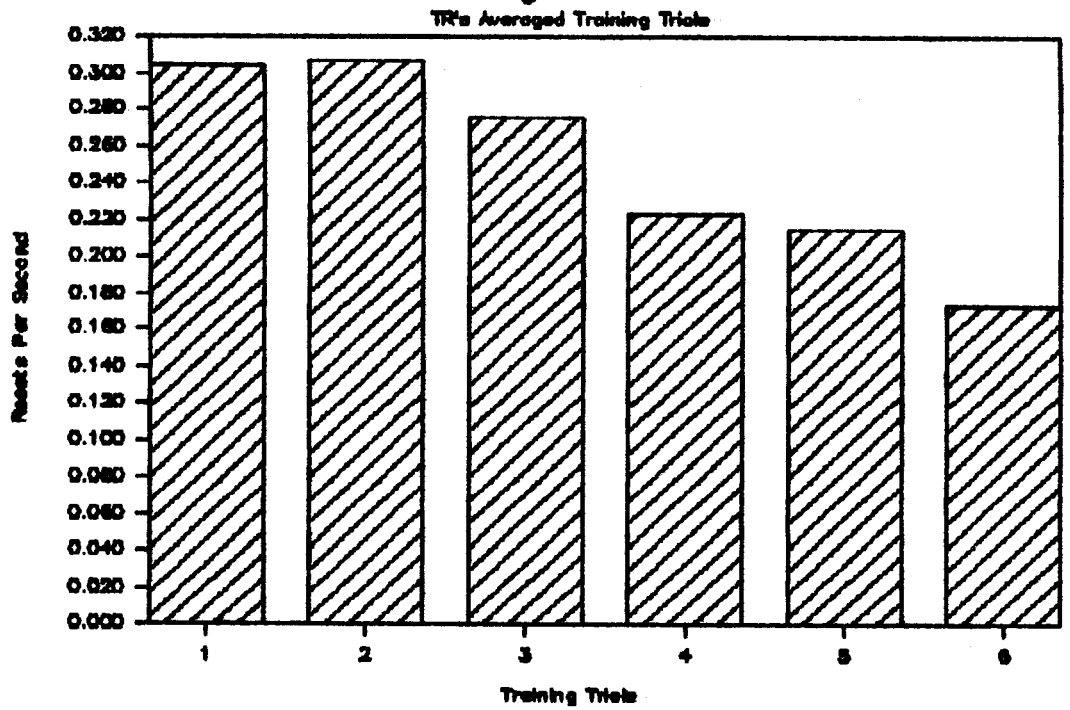
graphs depicting the mean EMG level (measured by resets-per-second) are used to compare both biceps and triceps EMG activity for all subjects.

Appendix 2, Set 2 pictures the triceps data collapsed across set and session to produce a bar graph depicting the average pattern of activity from the first trial to the last trial for any given training session. These graphs were obtained by averaging a subject's EMG activity for individual trials across all sets and training sessions. This was done for each trial until an average pattern of activity could be obtained for the subject's training sessions. An example is given in Figure 6, for TR's data. The resets-per-second measure for trial 1 (0.305), represents the average for trial 1 of all training sessions and sets. Trials 2-6 are calculated in the same manner for the purpose of visually depicting what a subject's average EMG activity is for any given training session.

If the subject showed a consistent decrease in EMG activity during a training session (as was expected with the experimental subjects), then this pattern should be apparent in an average of all trials across set and session. TR's data exemplified this expected pattern as can be seen by Figure 6. It can also be seen from the graphs of Appendix 2,

Set 2 that only the experimental subjects showed any sort of consistent pattern of decreasing triceps activity for any given training session.

Figure 6:



Appendix 2, Set 3 contains bar graphs which depict the mean EMG activity for pre-training, training, and post-training sessions. All subjects and both triceps and biceps muscle groups are depicted in the graphs of Set 3. The graphs represent the data from Tables 4 and 5. The high degree of variability from one training session to the next can be easily seen in these graphs.

Analysis of Variance

Tables six through ten show analyses of variance for the training data. They show F tests for triceps and biceps muscles and include the experimental and control subjects. Table 6 is the main effect for sessions; Table 7 is the main effect for trials; Table 8 is an analysis of the linear component (slope) for sessions; Table 9 is a test of the slope for trials; and Table 10 is an analysis of the interaction between muscle (biceps and triceps) and session, and muscle and trial.

Sessions ANOVA for Biceps and Triceps

The biceps main effect (sessions) for the experimental subjects is significant. F statistics for TR, JE, RO and RA are 30.76, 25.58, 209.68 and 23.32, respectively. The control subjects are also significant for a biceps main effect. F statistics for PA, SH and DE are 18.88, 39.67 and 12.53, respectively, at alpha = 0.05 (Table 6).

The triceps main effect (session) ANOVA yielded different results. JE and RO are significant at 0.05 with F ratios of 14.18 and 503.23 respectively. However TR and RA are non-significant with F's of 9.72 and 1.29.

Table 6: ANOVA for Main Effect of Sessions (Biceps and Triceps Muscles) for Seven Subjects.

Subject (Exper)	Muscle	Source	S.S.	DF	MS	F	Sig.
TR	Biceps	Sessions:	2.56821	5	0.51364	30.76	S.
		Error:	0.91846	55	0.01670		
	Triceps	Sessions:	1.35064	5	0.27013	9.72	N.S.
		Error:	1.52884	55	0.02780		
JE	Biceps	Sessions:	11.34717	7	1.62102	25.58	S.
		Error:	4.87919	77	0.06337		
	Triceps	Sessions:	0.75406	6	0.12568	14.18	S.
		Error:	0.58493	66	0.00886		
RO	Biceps	Sessions:	5.72429	6	0.95405	209.68	S.
		Error:	0.46428	102	0.00455		
	Triceps	Sessions:	1.62176	6	0.27029	503.23	S.
		Error:	0.05479	102	0.00054		
RA	Biceps	Sessions:	4.30664	7	0.61523	23.32	S.
		Error:	1.47715	56	0.02638		
	Triceps	Sessions:	0.01673	7	0.00239	1.29	N.S.
		Error:	0.11698	63	0.00186		
Subject (Contr)	**Muscle**	**Source**	**S.S.**	**DF**	**MS**	**F**	**Sig.**
PA	Biceps	Sessions:	0.28134	5	0.05627	18.88	S.
		Error:	0.16414	55	0.00298		
	Triceps	Sessions:	0.12763	5	0.02553	50.26	S.
		Error:	0.02794	55	0.00051		
SH	Biceps	Sessions:	0.43843	5	0.08767	39.67	S.
		Error:	0.12152	55	0.00221		
	Triceps	Sessions:	0.93972	5	0.18794	79.17	S.
		Error:	0.13057	55	0.00237		
DE	Biceps	Sessions:	0.45600	5	0.09120	12.53	S.
		Error:	0.40014	55	0.00728		
	Triceps	Sessions:	0.21188	5	0.04238	23.38	S.
		Error:	0.09968	55	0.00181		

Table 7: ANOVA for Main Effect of Trials (Biceps/Triceps) for Seven Subjects.

Subject (Exper)	Muscle	Source	S.S.	DF	MS	F	Sig.
TR	Biceps	Trials:	0.23804	5	0.04761	2.36	N.S.
		Error:	1.10793	55	0.02014		
	Triceps	Trials:	0.17430	5	0.03486	1.26	N.S.
		Error:	1.51930	55	0.02762		
JE	Biceps	Trials:	0.21086	5	0.04217	0.77	N.S.
		Error:	4.11857	75	0.05491		
	Triceps	Trials:	0.07038	5	0.01408	1.62	N.S.
		Error:	0.56488	65	0.00869		
RO	Biceps	Trials:	0.00738	5	0.00148	0.42	N.S.
		Error:	0.35004	100	0.00350		
	Triceps	Trials:	0.00077	5	0.00015	0.33	N.S.
		Error:	0.04630	100	0.00046		
RA	Biceps	Trials:	0.19954	5	0.03991	2.87	S.
		Error:	1.25402	90	0.01393		
	Triceps	Trials:	0.00828	5	0.00166	1.18	N.S.
		Error:	0.13342	95	0.00140		
Subject (Contr)	Muscle	Source	S.S.	DF	MS	F	Sig.
PA	Biceps	Trails:	0.01176	5	0.00235	1.06	N.S.
		Error:	0.12223	55	0.00222		
	Triceps	Trials:	0.00376	5	0.00075	1.48	N.S.
		Error:	0.02790	55	0.00051		
SH	Biceps	Trials:	0.00442	5	0.00088	0.34	N.S.
		Error:	0.14270	55	0.00259		
	Triceps	Trials:	0.00417	5	0.00083	0.37	N.S.
		Error:	0.12505	55	0.00227		
DE	Biceps	Trials:	0.04751	5	0.00950	1.26	N.S.
		Error:	0.41433	55	0.00753		
	Triceps	Trials:	0.02001	5	0.00400	2.07	N.S.
		Error:	0.10652	55	0.00194		

Table 8: Test of Significance of the Linear Component of Sessions (Biceps/Triceps) for Seven Subjects.

Subject (Exper)	Muscle	Source	S.S.	DF	MS	F	Sig.
TR	Biceps	Linear:	0.05905	1	0.05905	5.30	S.
		Error:	0.12260	11	0.12260		
	Triceps	Linear:	0.35461	1	0.35461	16.86	S.
		Error:	0.23133	11	0.02103		
JE	Biceps	Linear:	0.71710	1	0.71710	22.19	S.
		Error:	0.35557	11	0.03232		
	Triceps	Linear:	0.03175	1	0.03175	3.70	N.S.
		Error:	0.09446	11	0.00859		
RO	Biceps	Linear:	0.43150	1	0.43150	151.40	S.
		Error:	0.04848	17	0.00285		
	Triceps	Linear:	0.66548	1	0.66548	799.82	S.
		Error:	0.01414	17	0.00083		
RA	Biceps	Linear:	2.23016	1	2.23016	166.31	S.
		Error:	0.10725	8	0.01341		
	Triceps	Linear:	0.00116	1	0.00016	0.34	N.S.
		Error:	0.03114	9	0.00346		
Subject (Contr)	Muscle	Source	S.S.	DF	MS	F	Sig.
PA	Biceps	Linear:	0.00056	1	0.00056	0.13	N.S.
		Error:	0.04599	11	0.00418		
	Triceps	Linear:	0.05519	1	0.05519	66.45	S.
		Error:	0.00914	11	0.00083		
SH	Biceps	Linear:	0.01033	1	0.01033	5.30	S.
		Error:	0.02147	11	0.00195		
	Triceps	Linear:	0.35182	1	0.35182	106.22	S.
		Error:	0.03644	11	0.00331		
DE	Biceps	Linear:	0.31085	1	0.31085	27.46	S.
		Error:	0.12457	11	0.01132		
	Triceps	Linear:	0.16579	1	0.16579	78.33	S.
		Error:	0.02328	11	0.00212		

Table 9: Test of the Significance of the Slope of the Trials (Biceps/Triceps) for Seven Subjects.

Subject (Exper)	Muscle	Source	S.S.	DF	MS	F	Sig.
TR	Biceps	Linear:	0.08430	1	0.08430	4.09	N.S.
		Error:	0.22645	11	0.02059		
	Triceps	Linear:	0.16411	1	0.16411	5.85	S.
		Error:	0.30837	11	0.02803		
JE	Biceps	Linear:	0.01089	1	0.01089	0.11	N.S.
		Error:	1.43770	15	0.09585		
	Triceps	Linear:	0.01383	1	0.01383	1.82	N.S.
		Error:	0.09879	13	0.00760		
RO	Biceps	Linear:	0.00010	1	0.00010	0.02	N.S.
		Error:	0.09304	20	0.00465		
	Triceps	Linear:	0.00025	1	0.00025	0.58	N.S.
		Error:	0.00853	20	0.00043		
RA	Biceps	Linear:	0.16863	1	0.16863	4.35	N.S.
		Error:	0.69829	18	0.03879		
	Triceps	Linear:	0.00611	1	0.00611	2.39	N.S.
		Error:	0.04857	19	0.00256		
Subject (Contr)	**Muscle**	**Source**	**S.S.**	**DF**	**MS**	**F**	**Sig.**
PA	Biceps	Linear:	0.00536	1	0.00536	1.18	N.S.
		Error:	0.05019	11	0.00456		
	Triceps	Linear:	0.00305	1	0.00305	3.51	N.S.
		Error:	0.00956	11	0.00087		
SH	Biceps	Linear:	0.00095	1	0.00095	0.21	N.S.
		Error:	0.04910	11	0.00446		
	Triceps	Linear:	0.00163	1	0.00163	0.45	N.S.
		Error:	0.04008	11	0.00364		
DE	Biceps	Linear:	0.01240	1	0.01240	1.64	N.S.
		Error:	0.08296	11	0.00754		
	Triceps	Linear:	0.01554	1	0.01554	4.37	N.S.
		Error:	.03911	11	.00356		

Table 10: Test of the slope of "Sessions/Muscle" & "Trials/Muscle" Interaction for Seven Subjects

Subject (Exper)	Interaction Type	Source	S.S.	DF	MS	F	Sig.
TR	Sess/Muscle Interaction	Interaction:	0.06212	1	0.06212	3.86	N.S.
		Error:	0.35392	22	0.01609		
	Trl/Muscle Interaction	Interaction:	0.00658	1	0.00658	0.27	N.S.
		Error:	0.53482	22	0.02431		
JE	Sess/Muscle Interaction	Interaction:	0.36358	1	0.36358	15.51	S.
		Error:	0.51569	22	0.02344		
	Trl/Muscle Interaction	Interaction:	0.00021	1	0.00021	0.00	N.S.
		Error:	1.53650	28	0.05487		
RO	Sess/Muscle Interaction	Interaction:	0.01262	1	0.01262	6.85	S.
		Error:	0.06262	34	0.00184		
	Trl/Muscle Interaction	Interaction:	0.00033	1	0.00033	0.13	N.S.
		Error:	0.10157	40	0.00254		
RA	Sess/Muscle Interaction	Interaction:	1.22515	1	1.22515	150.49	S.
		Error:	0.13840	17	0.00814		
	Trl/Muscle Interaction	Interaction:	0.05737	1	0.05737	2.84	N.S.
		Error:	0.74686	37	0.02019		
Subject (Contr)	Interaction Type	Source	S.S.	DF	MS	F	Sig.
PA	Sess/Muscle Interaction	Interaction:	0.03341	1	0.03341	13.33	S.
		Error:	0.05513	22	0.00251		
	Trl/Muscle Interaction	Interaction:	0.00016	1	0.00016	0.06	N.S.
		Error:	0.05974	22	0.00272		
SH	Sess/Muscle Interaction	Interaction:	0.12080	1	0.12080	45.89	S.
		Error:	0.05791	22	0.00263		
	Trl/Muscle Interaction	Interaction:	0.00005	1	0.00005	0.01	N.S.
		Error:	0.08918	22	0.00405		
DE	Sess/Muscle Interaction	Interaction:	0.01130	1	0.01130	1.68	N.S.
		Error:	0.14785	22	0.00672		
	Trl/Muscle Interaction	Interaction:	0.00009	1	0.00009	0.02	N.S.
		Error:	.12207	22	.00555		

The control subjects were are all significant for a triceps main effect with F ratios of 50.26, 79.17 and 23.38 for PA, SH and DE (Table 6).

Trials ANOVA for Biceps and Triceps.

The main effect for trials are, for most subjects, non-significant at the alpha = 0.05 level. These results are observed for both muscle groups and both subject conditions. The only exception is in RA's data. His analysis yielded a significant biceps main effect. The F ratio is 2.87 (Table 7).

Sessions and Trials ANOVA for Linear Component.

The sessions linear component for the experimental subjects yielded significant F ratios for the biceps muscle. F statistics ranged from 5.30 for TR to 166.31 for RA. Two of the three control subjects were significant for the biceps while one was not. F values for these subjects ranged from 0.13 for PA to 27.46 for DE (Table 8).

The analysis of the triceps' linear component for sessions yielded two significant and two non-significant F values for the experimental subjects, and three significant

scores for the controls. F statistics varied from 0.34 to 799.82 for RA and RO and 66.45 to 106.22 for PA and SH (control), (Table 8).

The test of significance of the triceps and biceps linear components for trials yielded non-significant scores for both muscles for most subjects in the study. One exception did occur. TR had a significant triceps F value of 5.85. This significant linear pattern is depicted in Figure 6. Other F's ranged from RO's biceps value of 0.02 to RA's biceps value of 4.35 (Table 9).

ANOVA for Muscle/Sessions and Muscle/Trials Interaction.

The muscle by sessions and muscle by trials interaction ANOVAS complete the tests. Results showed three significant and one non-significant muscle by sessions interaction; and four non-significant trials by muscle interactions for the experimental subjects. The controls had two significant and one non-significant muscle by sessions interactions; and three non-significant muscle by trials interaction. F statistics varied from JE's non-significant trials by muscle interaction score of 0.00 to RA's significant session by muscle score of 150.49 (Table 10).

Summary of ANOVA's.

All these results were unexpected. Table 6 indicates there is a biceps main effect for the experimental subjects and two of them had triceps main effects for sessions. All the controls had main effects for triceps and biceps muscles as well. Table 7 indicated that main effects for trials (with the exception of one experimental subject's biceps) were non-existent for either muscle. It should be noted that the F's in these tables report only on general changes and do not test linear components.

It appears that there is a linear component (sessions) effect for the biceps for the experimental subjects and an effect for triceps in two of the subjects. Controls have main effects in triceps for all subjects but only two subjects have main effects for biceps (Table 8). It also appears that the linear component (trials)) effect does not exist with the exception of TR's results for triceps. (Table 9).

It would appear that a main effect for sessions does exist for subjects over the training phase. This conclusion is based on the significant session by muscle interaction, non-existence of a main effect for trials, and non-existence of a linear component for trials. This

conclusion should be approached with caution. Certain subjects did not show significance, and even when significance is shown, the subject's change in EMG activity may not be in the specified direction.

Discussion.

This study was designed to see if counterproductive activity of antagonistic muscles in a simple forearm flexion movement in cerebral palsied persons could be reduced with EMG feedback from the antagonistic muscles. The results of this study are of interest on a number of points.

First, electromyographic biofeedback decreased the activity of the triceps muscles significantly in all four experimental subjects with cerebral palsy. This conclusion should be approached with some caution, however, since one of the cerebral palsied control subjects (PA) achieved the same result with no feedback. This anomaly may be explained by his failure to follow the experimenter's instructions fully. Since the control condition was intended to provide a comparison level of triceps/biceps activity without feedback, all subjects were instructed not to use any relaxation techniques to help them relax during the sessions. PA admittedly misunderstood the intent of this part of the study and so used a "visualization" technique to relax his muscles. This unfortunate circumstance confounded his data. Since this study was a single-subject design, and the other two control subjects did not decrease their triceps activity over the pretest/post-test interval, PA's

data, while interesting on its own merits, should not be considered an invalidation of this study's overall conclusions.

Second, contrary to expectations, biceps activity decreased significantly in three out of four experimental subjects. Several explanations for this finding are possible. One possibility is that biofeedback from the antagonistic triceps muscle indirectly resulted in training of the biceps muscle. That is, while learning to decrease the inappropriate activity of the triceps muscle, the subject also acquired more control over the timing and force of contraction of the biceps muscle. Another possibility is that a biofeedback effect is not involved but repetition of the task resulted in motor learning of the flexion movement. This relaxation effect for biceps muscle may simply reflect the decrease in biceps activity needed to accomplish the pull brought about by the successful training of the triceps, and the reduction in antagonism between the muscles. The increase in antagonism between extensor and flexor, with the activation of one or other muscle, has been demonstrated by Hallett et al. (1983). The reverse might also prove to be true. The less activation of the flexor or extensor, the less antagonism between the two muscle groups.

The less the antagonism, the less activity is required to make the desired movement. Further research should be done to confirm or deny this proposition.

The third observation of interest in this study was the average decrease in triceps activity within a training session. Subjects did not show a decrease in average triceps EMG level over sessions. While there was a significant difference between post-training triceps activity and pre-training triceps activity, there was not a gradual improvement from session to session. Rather, there was considerable variability in performance from session to session. When trials are averaged over sessions (i.e., average of trial 1 for all training sessions, etc.), there is a consistent, although not significant, improvement in performance over trials within the session. This finding is not surprising, considering the severity of impairment in these C.P. subjects. The nature of their condition necessarily results in considerable within and between subject variability in performance. A subject's level of arousal, anxiety, and other factors has an especially large impact on performance in cerebral palsied people. However, within a training session, these factors are more nearly equal and a pattern of improvement can be discerned.

Although the pretest/post-test comparisons suggest a training effect for the triceps muscle, the factors responsible for the effect are still unclear. The non-significant ANOVAs for all but one subject suggest no interaction between session and trials as suggested by initial observation of the graphs. TR's significant sessions by trials interaction could be accounted for by her experience with other biofeedback procedures. Although the experimenters had requested that she use no relaxation procedures (or if she did use one, to describe it to us), her experience with other biofeedback procedures may have given her an advantage over other experimental subjects. PA's visualization technique certainly suggests the importance of psychological set on EMG biofeedback. It is difficult to observe and measure the psychological and physiological processes involved in learning to relax. These processes include mental relaxation, mood, fatigue, hypnotic states, and motivation (Lazar, 1977; Anderson, 1978; Scartelli, 1982). Scartelli (1982) has even found that soothing music playing in the background has a positive effect on finger extensor control in those with spastic cerebral palsy. His music group was compared to a group which received feedback but no music.

Although the majority of the experimental subjects decreased their biceps activity significantly during the pretest/post-test comparisons there is no way of telling whether or not the decrease was due to feedback generalization, psychological factors, physical factors or other variables. Thus the findings of this study should be approached with caution.

In summary, the results of this study indicate that EMG biofeedback can significantly reduce the inappropriate activity of antagonistic muscles in a simple forearm flexion movement in persons with cerebral palsy. This basic result should be interpreted with some caution given that the variability in performance in these subjects made it difficult to demonstrate unequivocally that there was learning over the sessions. Activity of the biceps muscle changed over training as well, although no feedback was received from this muscle. This finding is consistent with the hypothesis that improvement in performance of this particular task is dependant not only on decreasing the inappropriate activity of antagonistic muscles but on increasing the efficiency of activity in the agonist muscles.

These results, while not conclusive, are suggestive of an effect of biofeedback training on a specific, simple motor task in persons with cerebral palsy. The results of this paper indicate that future studies should aim to reduce the variability in performance of the motor task both between and within subjects in order to demonstrate unequivocally an improvement in performance over training sessions. This may be accomplished by selecting a more homogenous subject pool in terms of severity of disability or by selecting a motor task which shows less variability in performance. Additionally, the results of this study indicate that increases in performance of the task are dependent on the activity of both agonist and antagonist muscles. Future studies should include a condition in which feedback is provided from the agonist muscle simultaneously with that from the antagonist.

If the agonist/antagonist relationship can be refined to perform more "normally" in cerebral palsied individuals using EMG biofeedback, clinical applications of this specific muscle training technique would be exciting. For example training on the extensor/flexor muscles in the fingers might lead to better finger control and in turn to better writing skills for the cerebral palsied person. This study does indicate that within a specific muscle training

paradigm, EMG biofeedback does reduce unwanted muscle activity in one muscle of an antagonistic pair. However, this study cannot go beyond this point except to suggest that training of antagonistic pairs of muscles might be useful in understanding muscle control in the Cerebral Palsied and to provide a partial basis for other researchers to investigate the antagonistic relationship further.

Appendix 1.
Explanation of Experiment

In accordance with the ethics committee regulations, this explanation of the procedure of the experiment is provided to all subjects. Please read the following paragraphs carefully and if there are any questions the investigators will be happy to answer them for you. PLEASE BE ADVISED THAT SHOULD YOU, FOR ANY REASON, WISH TO WITHDRAW FROM THE STUDY, YOU MAY DO SO AT ANY TIME.

This study will assess the effects of EMG biofeedback training on specific muscle groups (tricep). There will be several sessions of approximately 60 minutes duration. The sessions will consist of having you seated in a specially designed chair while the investigators tape a number of electrodes on the biceps and triceps of your arm. If you are in the main study, you will be asked to listen to a biofeedback device which generates a tone in response to the activity of one muscle group. You will be asked to slowly flex and relax the arm a number of times on command while pulling on the weighted cord. Your task during these trials is to reduce the frequency of the tone from a "fast-beeping" tone to a "slow-beeping " tone. You will be asked to refrain from making any sudden movements or from talking during the test trials, as this will affect the results.

If you are in the control phase of this study, your instructions are the same as the main study except the feedback device will not be operational and thus you will not be asked to observe any muscle activity or to try to control it.

The weighted cord is provided so that there will be resistance to the movement of the arm. It is a light weight and requires little "effort" to pull. Therefore, you will be asked to pull "gently" on the cord using as little "effort" as possible to accomplish the required movement.

Specific information about your data will be kept confidential and will only be released upon your verbal or written request.

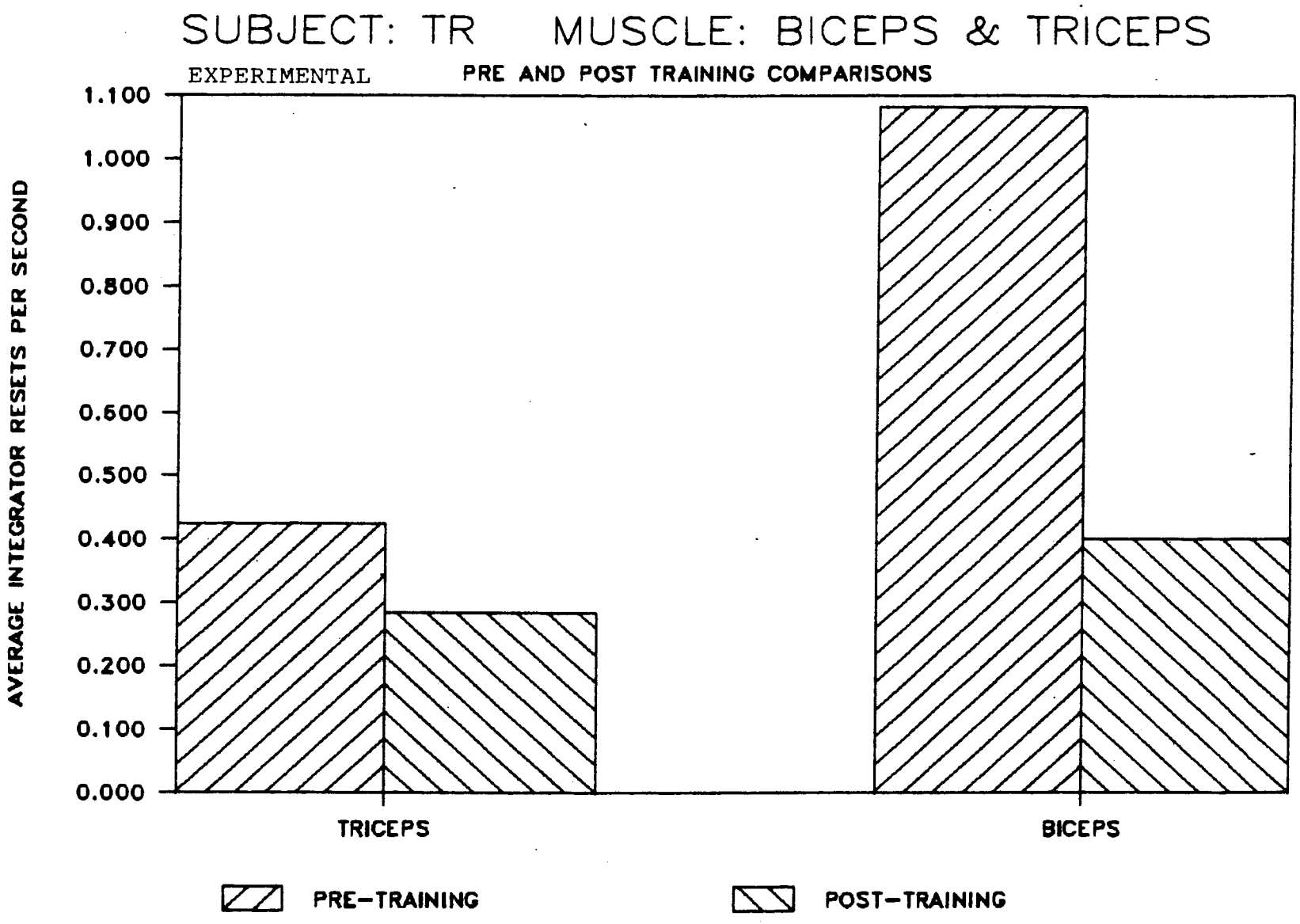
After the session, the electrodes will be removed and any problems you may have with the study will be discussed. If you understand this description and there are no questions or problems, please fill in and sign the following form and we shall begin the experiment. Please retain this copy of the description for your records.

Thank you for your participation,

Derek Isobe, Dave Dakin, Trevor Priest,
Alexis McIntosh, John Stork,
Blake Johnson, Elaine Furnel.

Appendix 2.
Set 1.

Pre-Training and Post-Training Comparisons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PRE AND POST TRAINING COMPARISONS
AVERAGE INTEGRATOR RESETS PER SECOND
1.100
1.000
0.900
0.800
0.700
0.600
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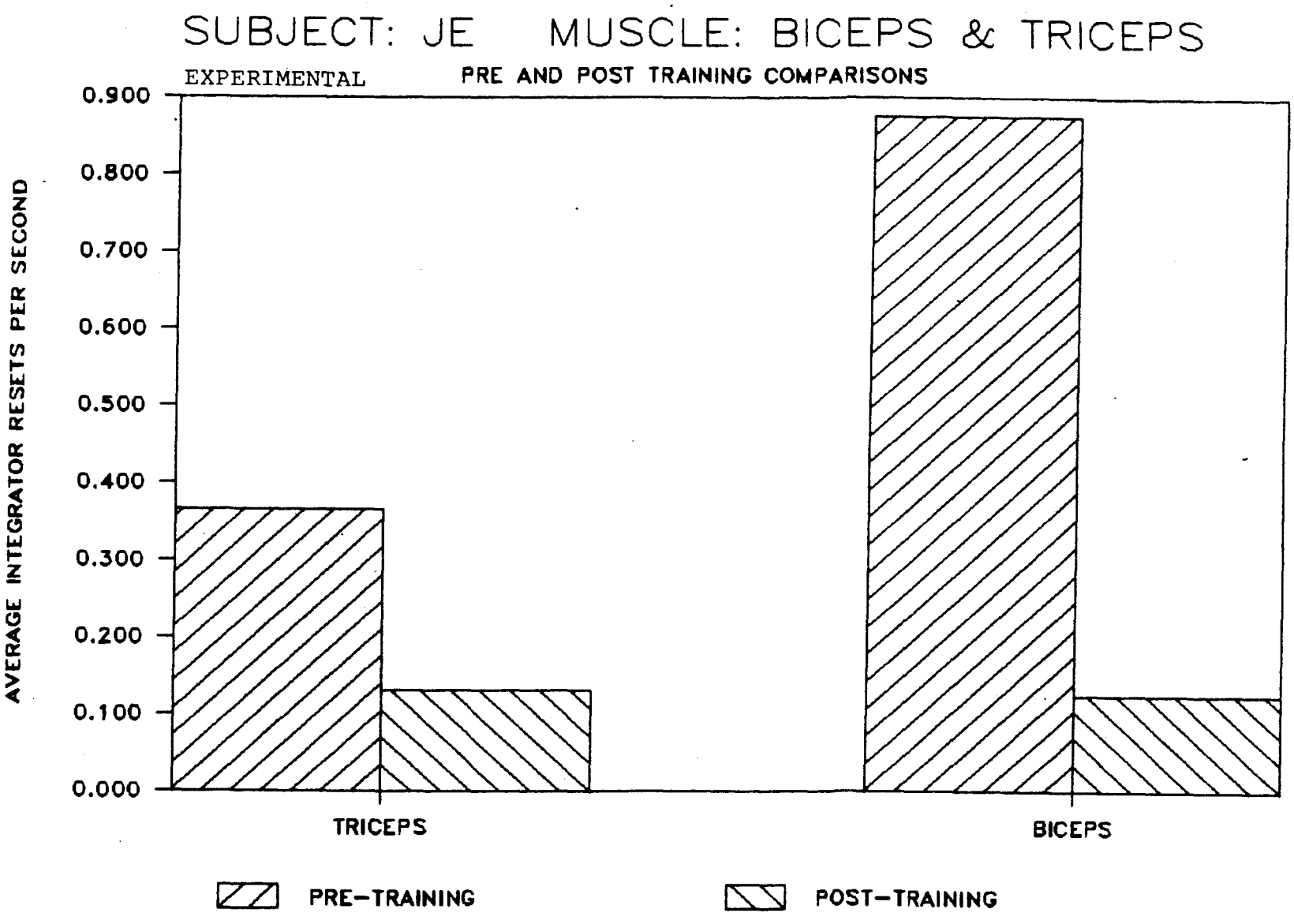
0.400
0.300
0.200
0.100
0.000
TRICEPS
BICEPS
PRE-TRAINING
POST-TRAINING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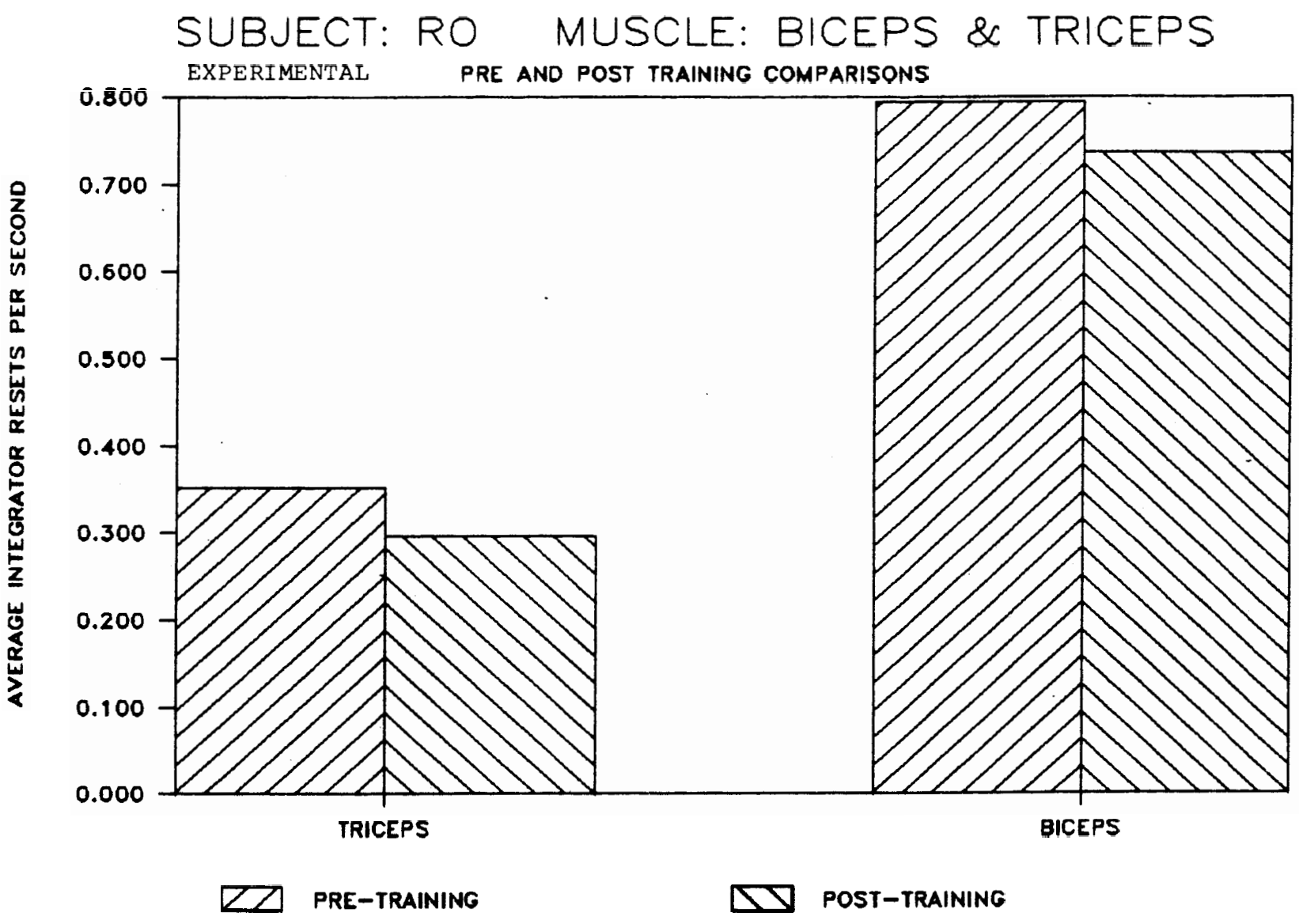
EXPERIMENTAL
PRE AND POST TRAINING COMPARISONS
AVERAGE INTEGRATOR RESETS PER SECOND
0.900
0.800
0.700
0.600
0.500
0.400
0.300
0.200
0.100
0.000
TRICEPS
BICEPS
PRE-TRAINING
POST-TRAINING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EXPERIMENTAL
PRE AND POST TRAINING COMPARISONS
AVERAGE INTEGRATOR RESETS PER SECOND
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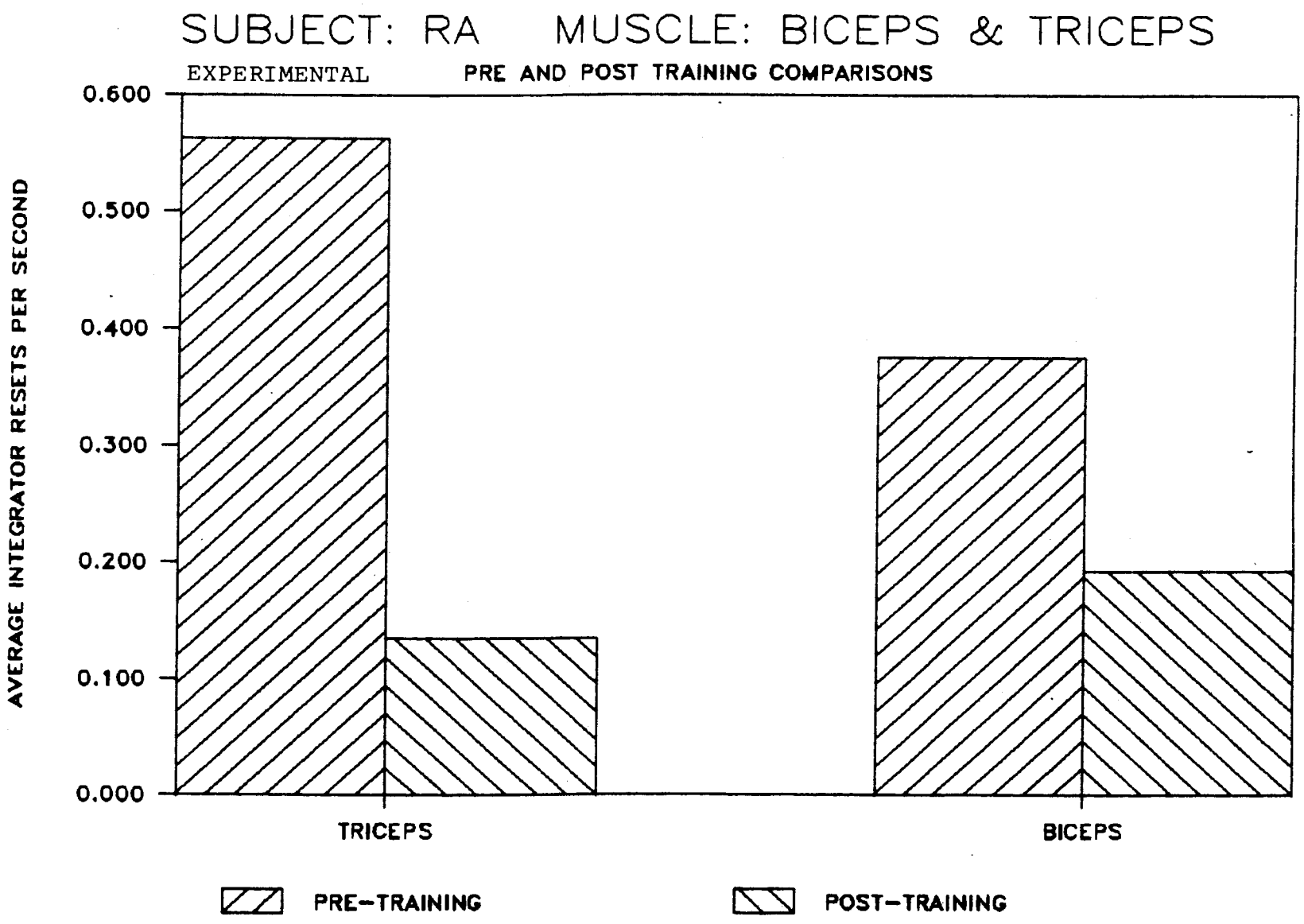
0.000
TRICEPS
BICEPS
PRE-TRAINING
POST-TRAINING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EXPERIMENTAL
PRE AND POST TRAINING COMPARISONS
AVERAGE INTEGRATOR RESETS PER SECOND
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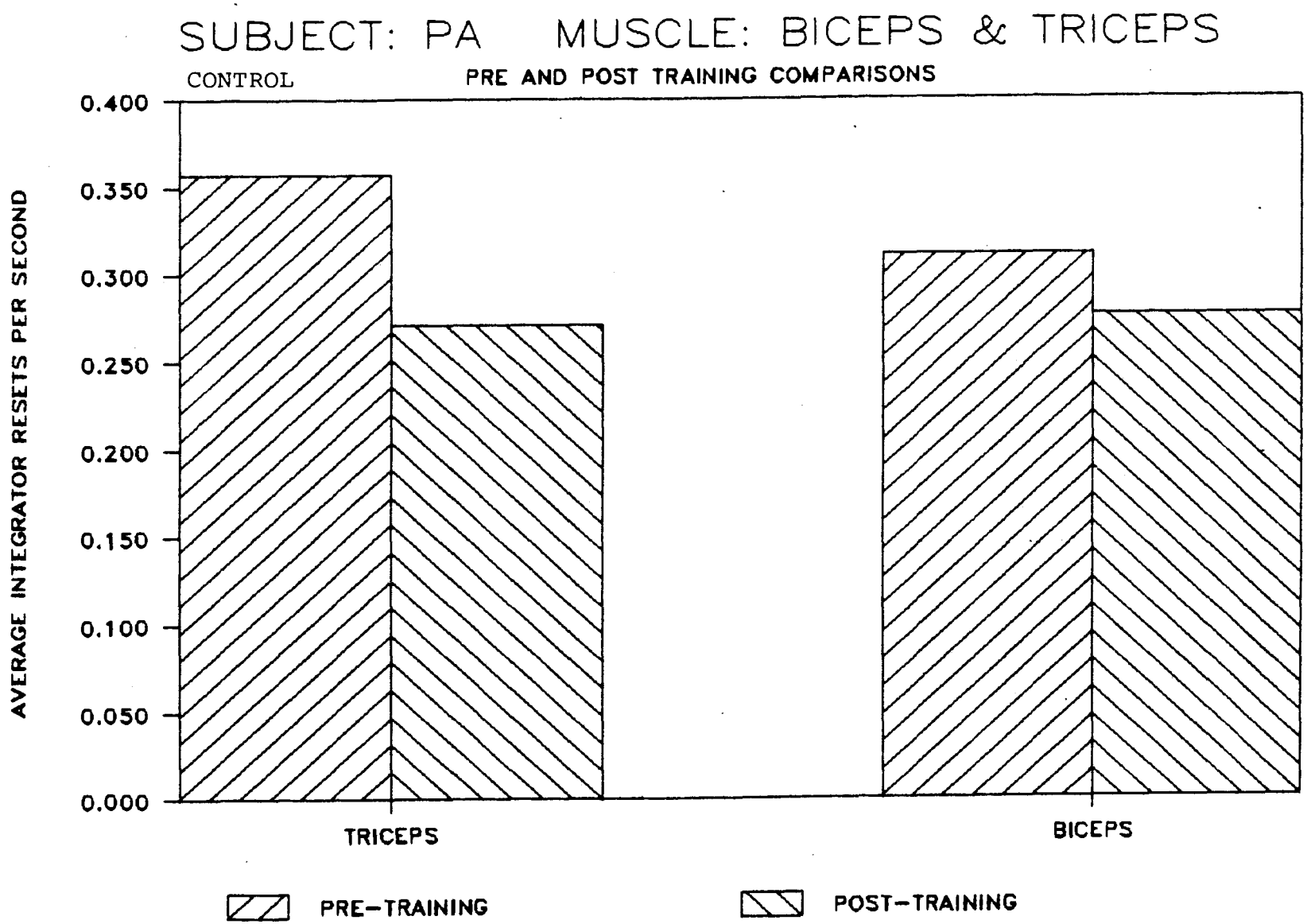
0.400
0.300
0.200
0.100
0.000
TRICEPS
BICEPS
PRE-TRAINING
POST-TRAINING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CONTROL
PRE AND POST TRAINING COMPARISONS
AVERAGE INTEGRATOR RESETS PER SECOND
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0.350
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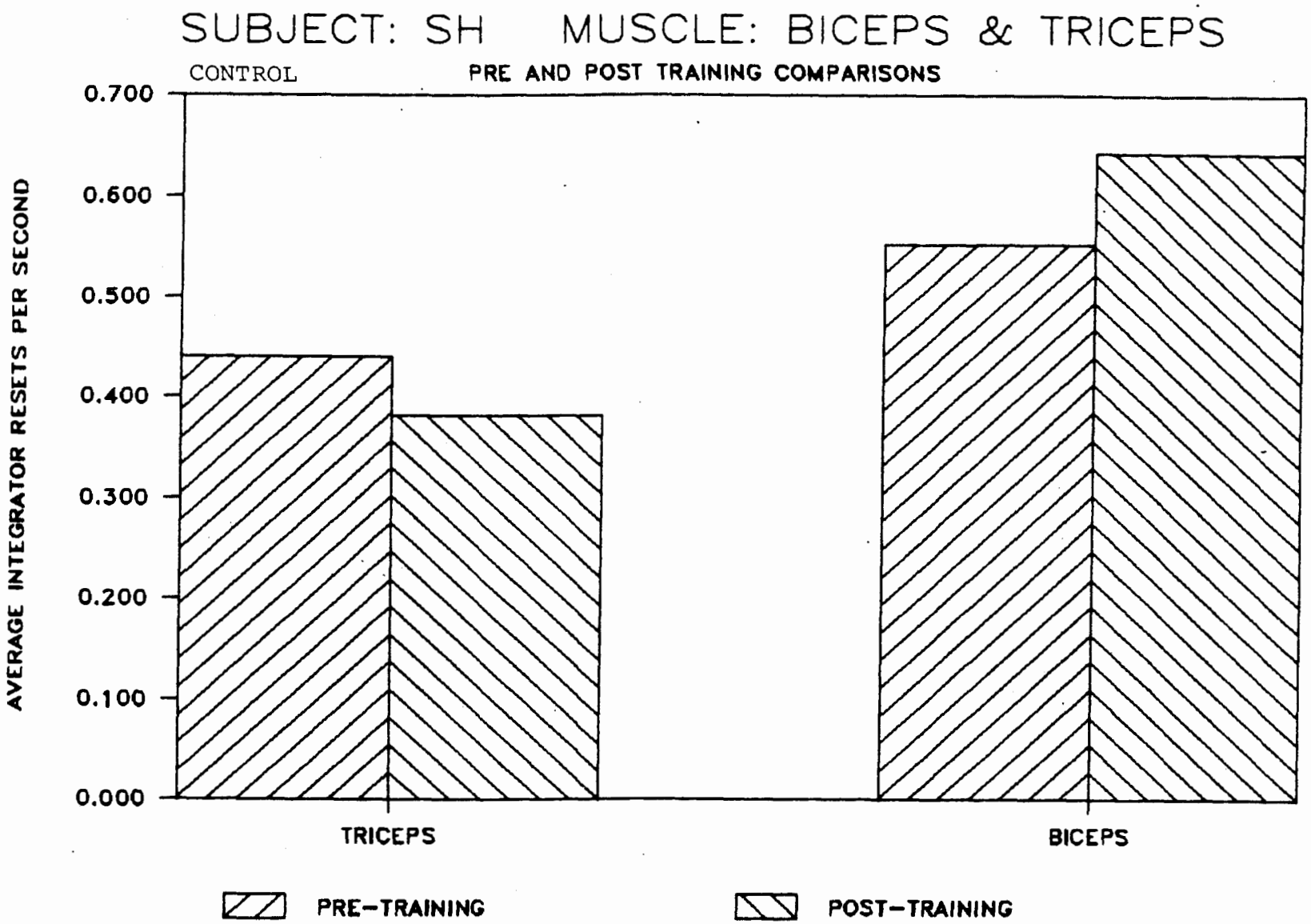
0.250
0.200
0.150
0.100
0.050
0.000
TRICEPS
BICEPS
PRE-TRAINING
POST-TRAINING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PRE AND POST TRAINING COMPARISONS
AVERAGE INTEGRATOR RESETS PER SECOND
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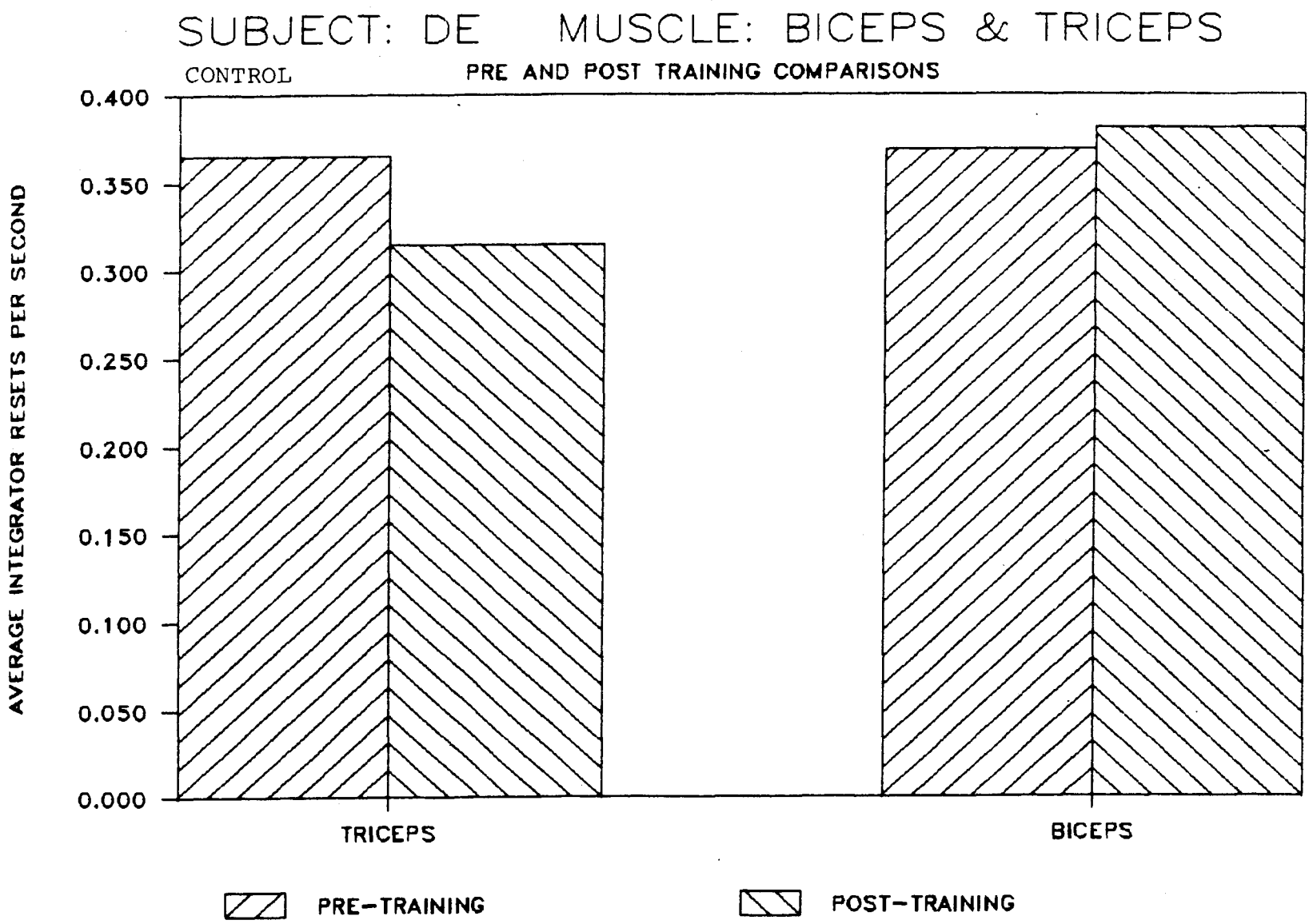
0.400
0.300
0.200
0.100
0.000
TRICEPS
BICEPS
PRE-TRAINING
POST-TRAINING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CONTROL
PRE AND POST TRAINING COMPARISONS
AVERAGE INTEGRATOR RESETS PER SECOND
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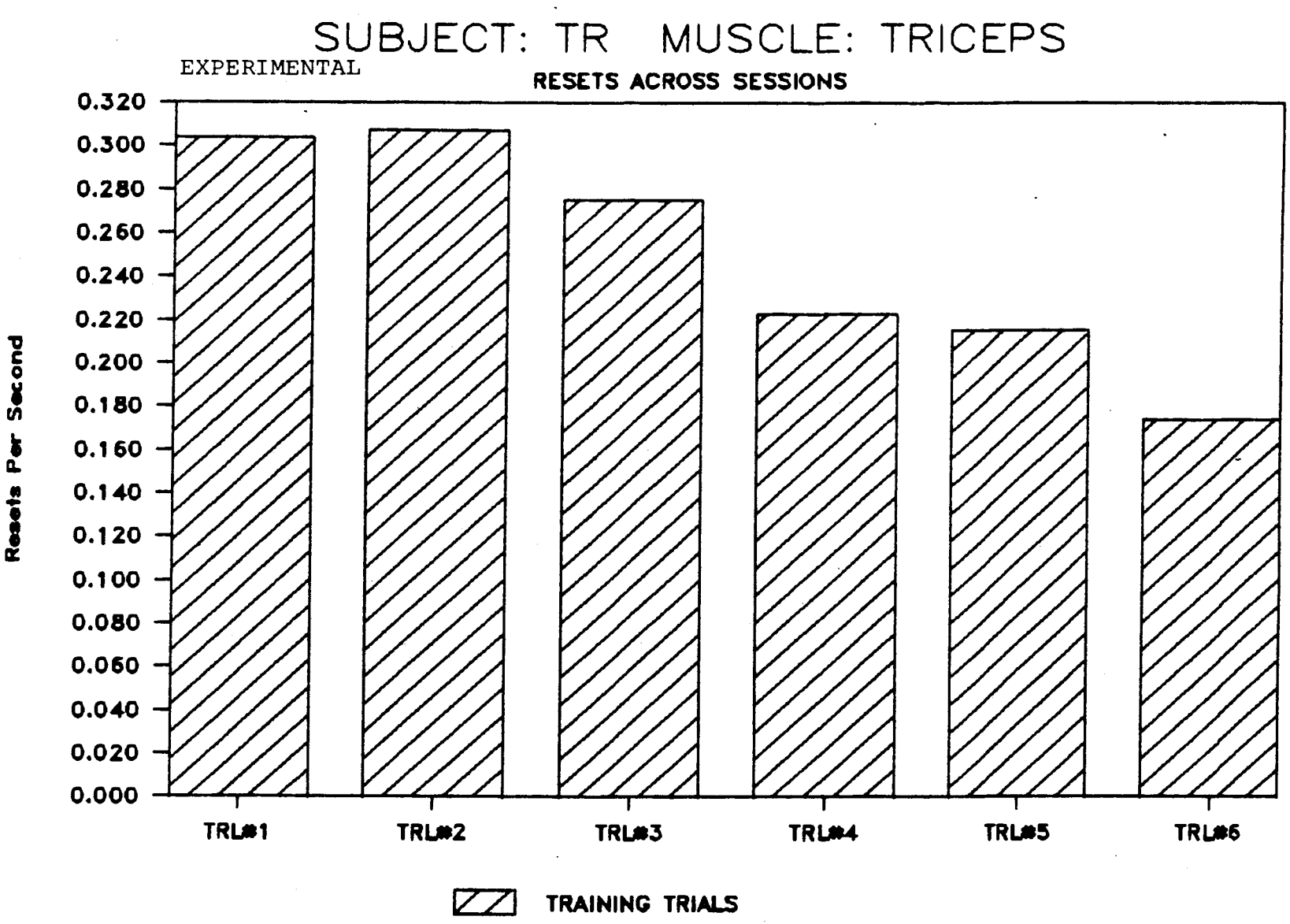
0.350
0.300
0.250
0.200
0.150
0.100
0.050
0.000
TRICEPS
BICEPS
PRE-TRAINING
POST-TRAINING

Appendix 2.
Set 2

Mean Triceps EMG for Trials Across Set & Session.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EXPERIMENTAL
RESETS ACROSS SESSIONS
Resets Per Second
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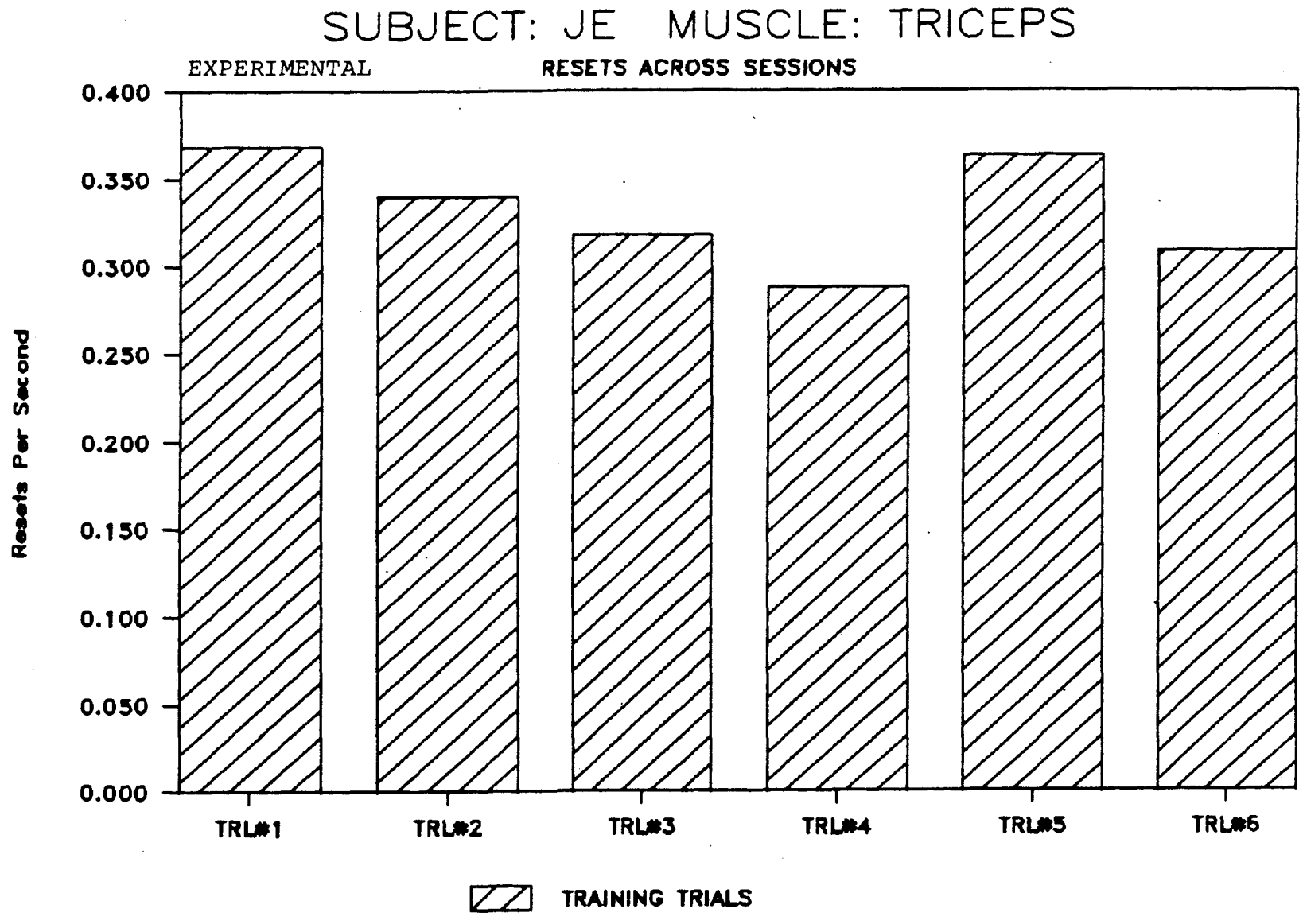
0.220
0.200
0.180
0.160
0.140
0.120
0.100
0.080
0.060
0.040
0.020
0.000
TRL#1
TRL#2
TRL#3
TRL#4
TRL#5
TRL#6
TRAINING TRIALS


SUBJECT: JE MUSCLE: TRICEPS
EXPERIMENTAL
RESETS ACROSS SESSIONS
Resets Per Second
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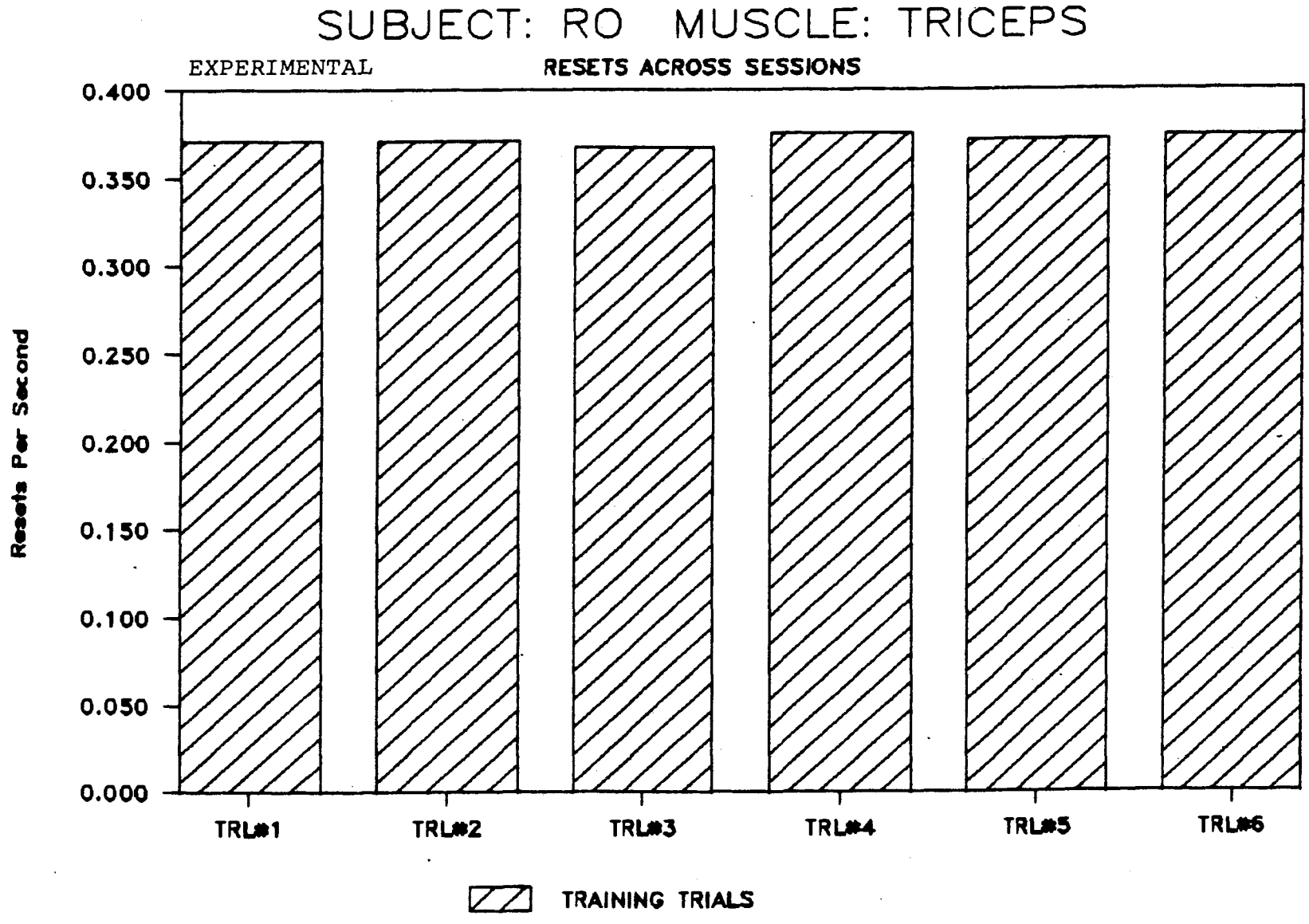
0.350
0.300
0.250
0.200
0.150
0.100
0.050
0.000
TRL#1
TRL#2
TRL#3
TRL#4
TRL#5
TRL#6
TRAINING TRIALS


SUBJECT: RO MUSCLE: TRICEPS
EXPERIMENTAL
RESETS ACROSS SESSIONS
Resets Per Second
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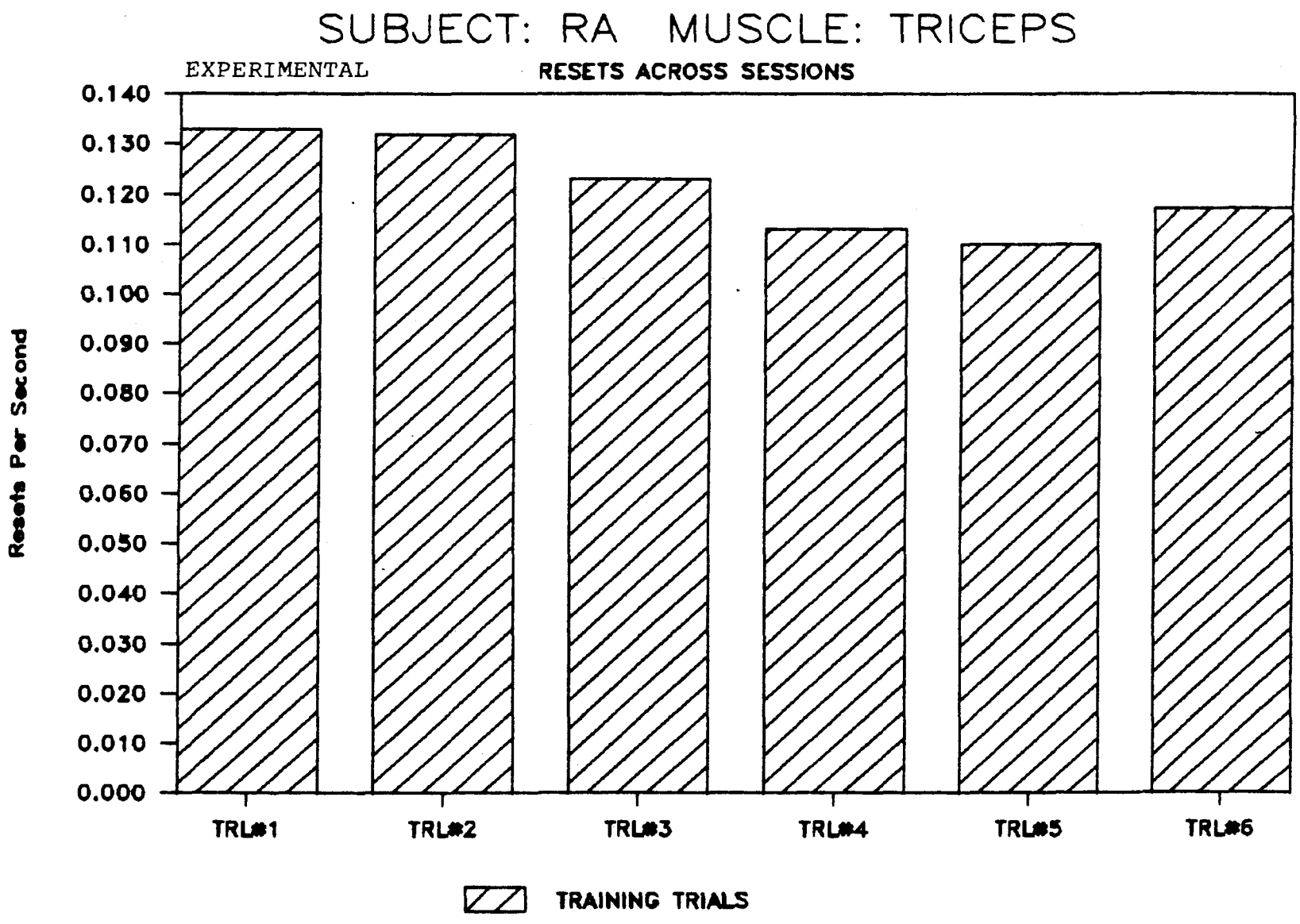
0.350
0.300
0.250
0.200
0.150
0.100
0.050
0.000
TRL#1
TRL#2
TRL#3
TRL#4
TRL#5
TRL#6
TRAINING TRIALS


SUBJECT: RA MUSCLE: TRICEPS
EXPERIMENTAL
RESETS ACROSS SESSIONS
Resets Per Second
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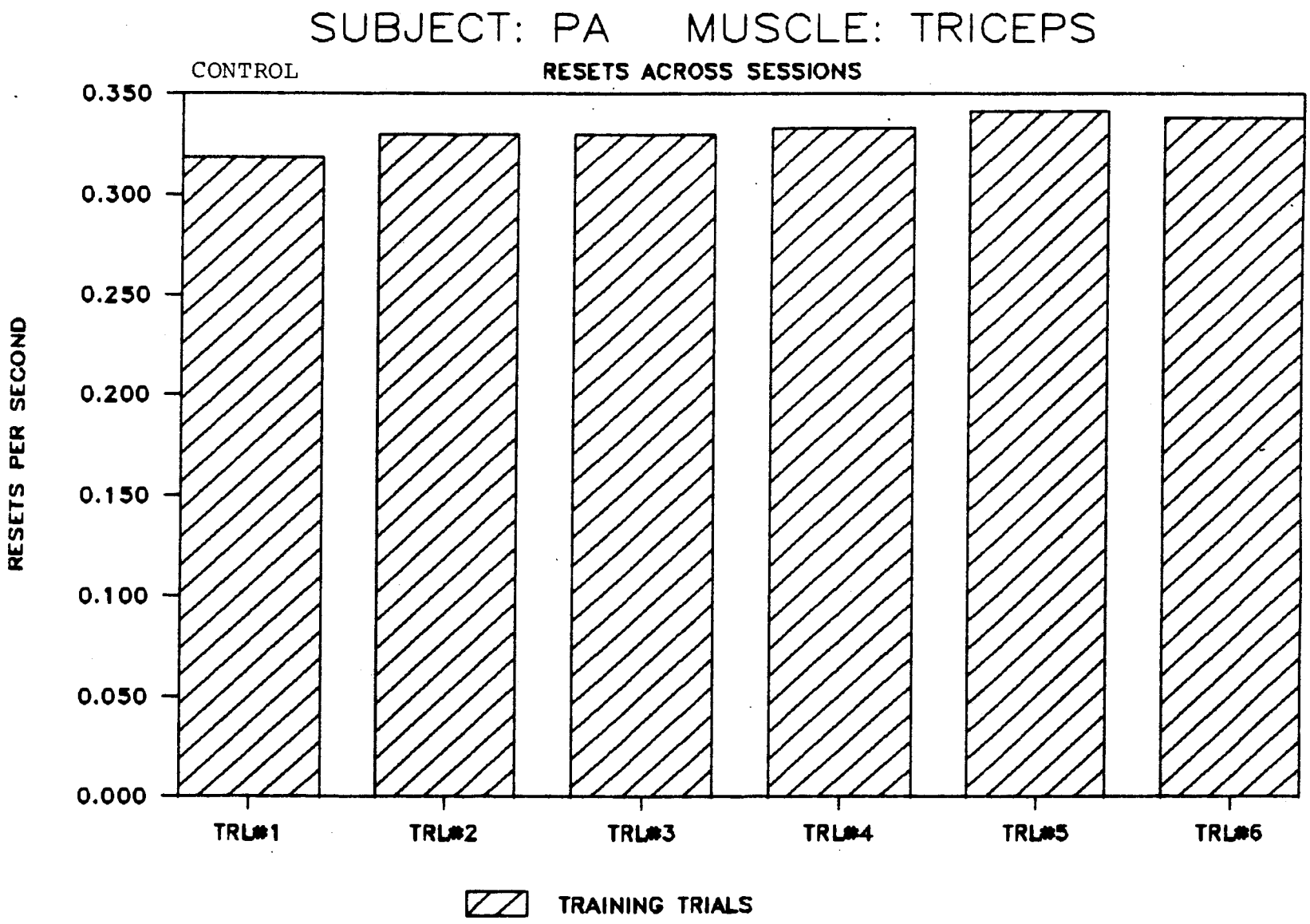
0.130
0.120
0.110
0.100
0.090
0.080
0.070
0.060
0.050
0.040
0.030
0.020
0.010
0.000
TRL#1
TRL#2
TRL#3
TRL#4
TRL#5
TRL#6
TRAINING TRIALS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CONTROL
RESETS ACROSS SESSIONS
RESETS PER SECOND
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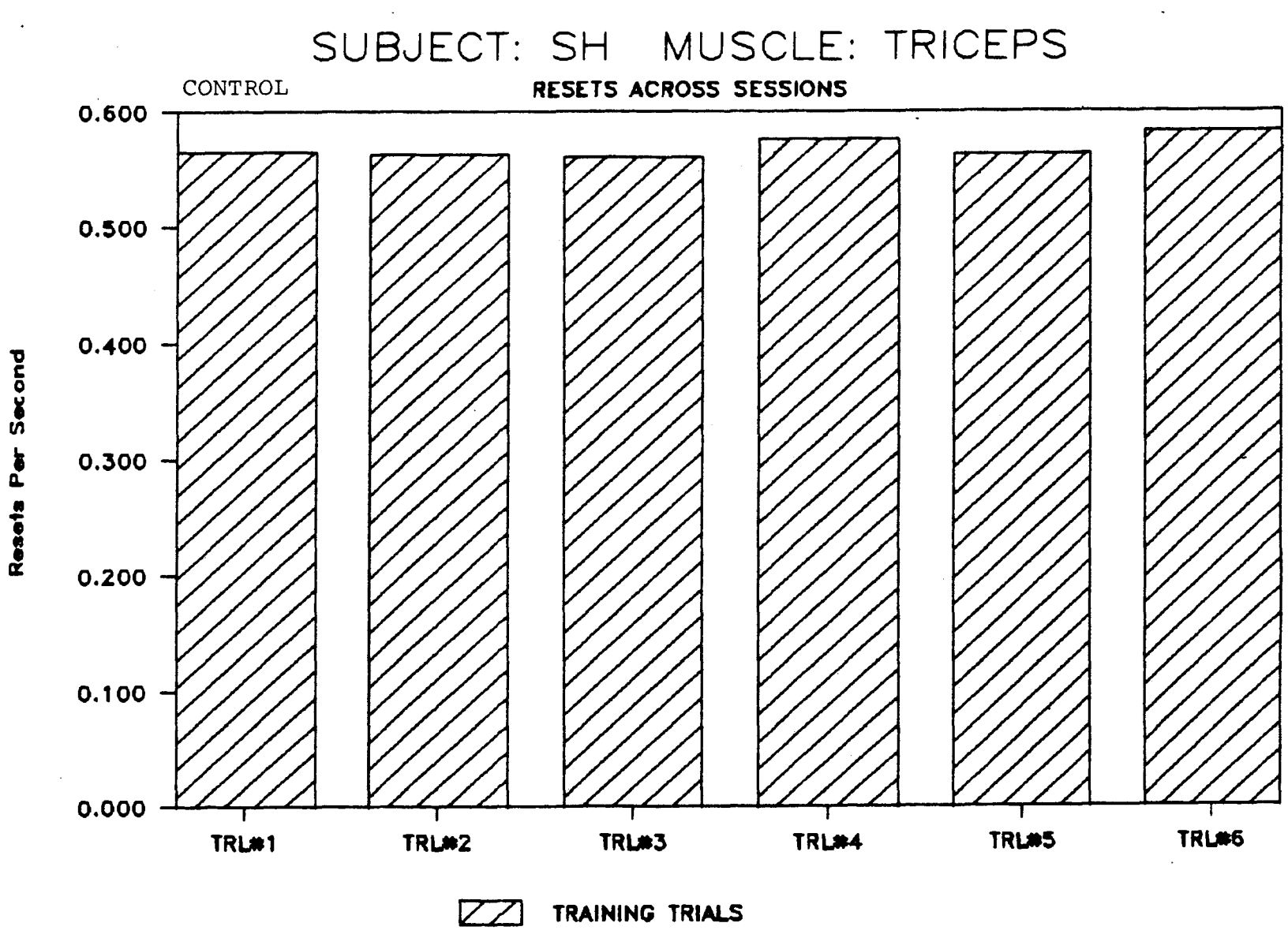
0.300
0.250
0.200
0.150
0.100
0.050
0.000
TRL#1
TRL#2
TRL#3
TRL#4
TRL#5
TRL#6
TRAINING TRIALS


SUBJECT: SH MUSCLE: TRICEPS
CONTROL
RESETS ACROSS SESSIONS
Resets Per Second
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0.500
0.400
0.300
0.200
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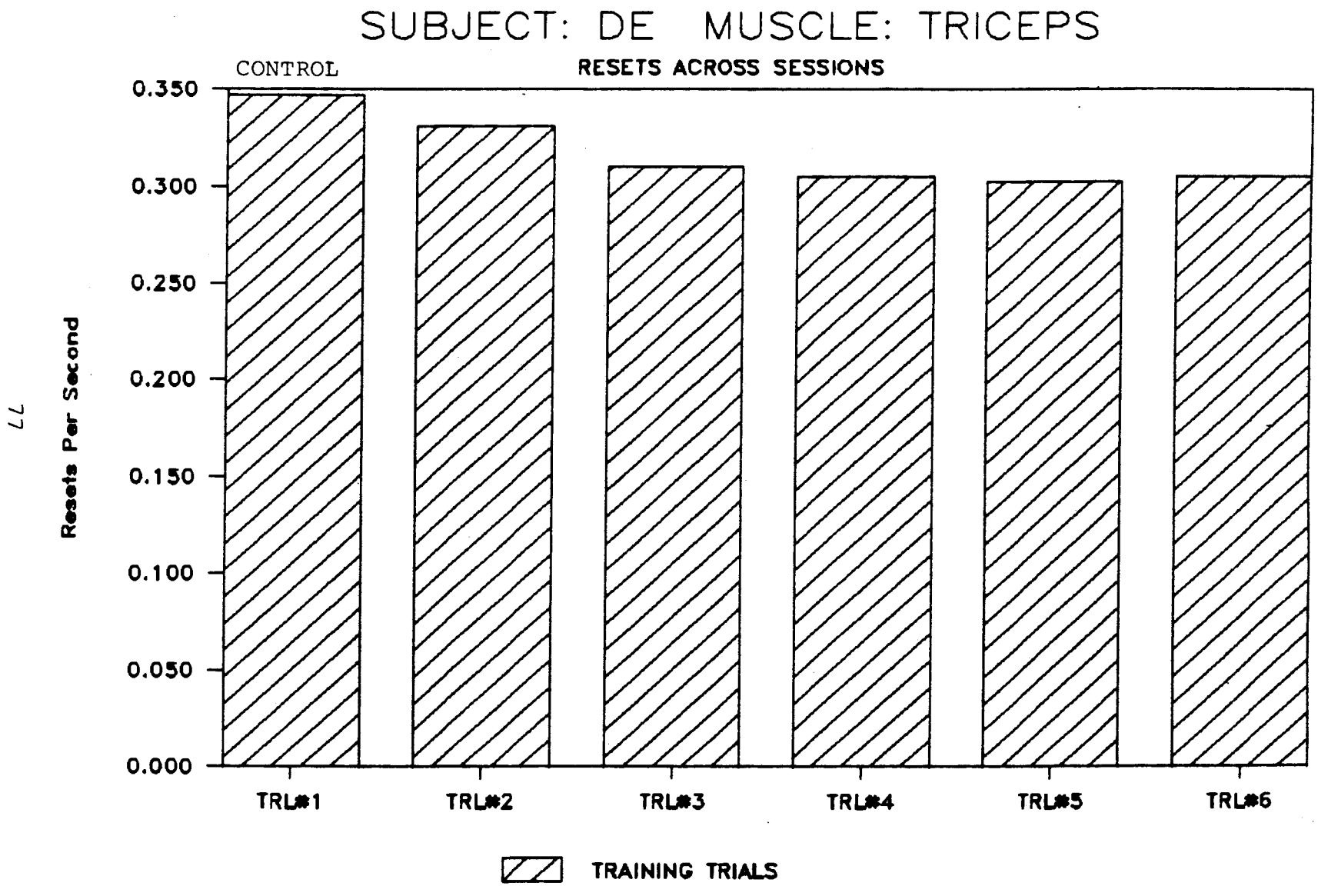
0.000
TRL#1
TRL#2
TRL#3
TRL#4
TRL#5
TRL#6
TRAINING TRIALS


SUBJECT: DE MUSCLE: TRICEPS
CONTROL
RESETS ACROSS SESSIONS
Resets Per Second
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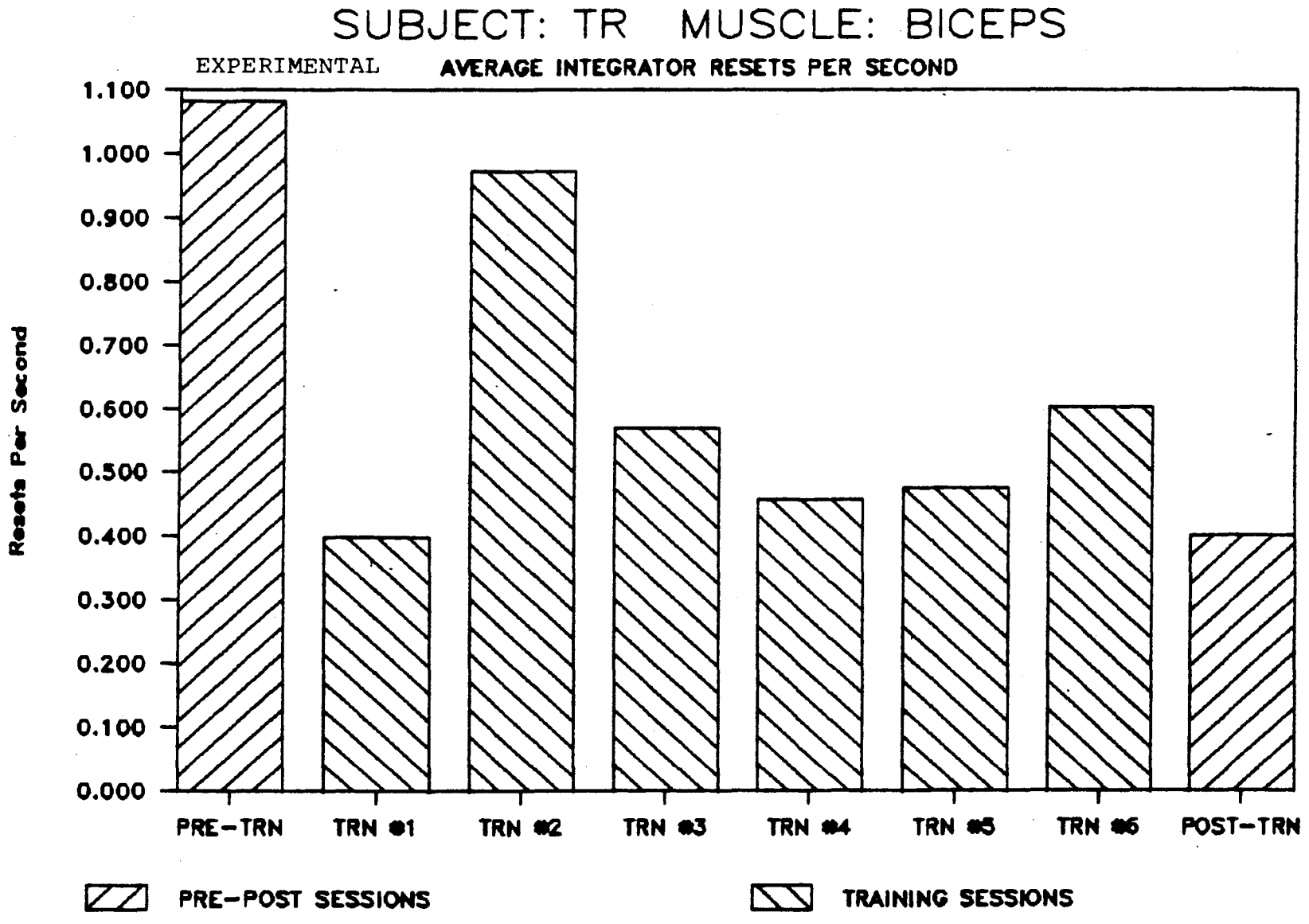
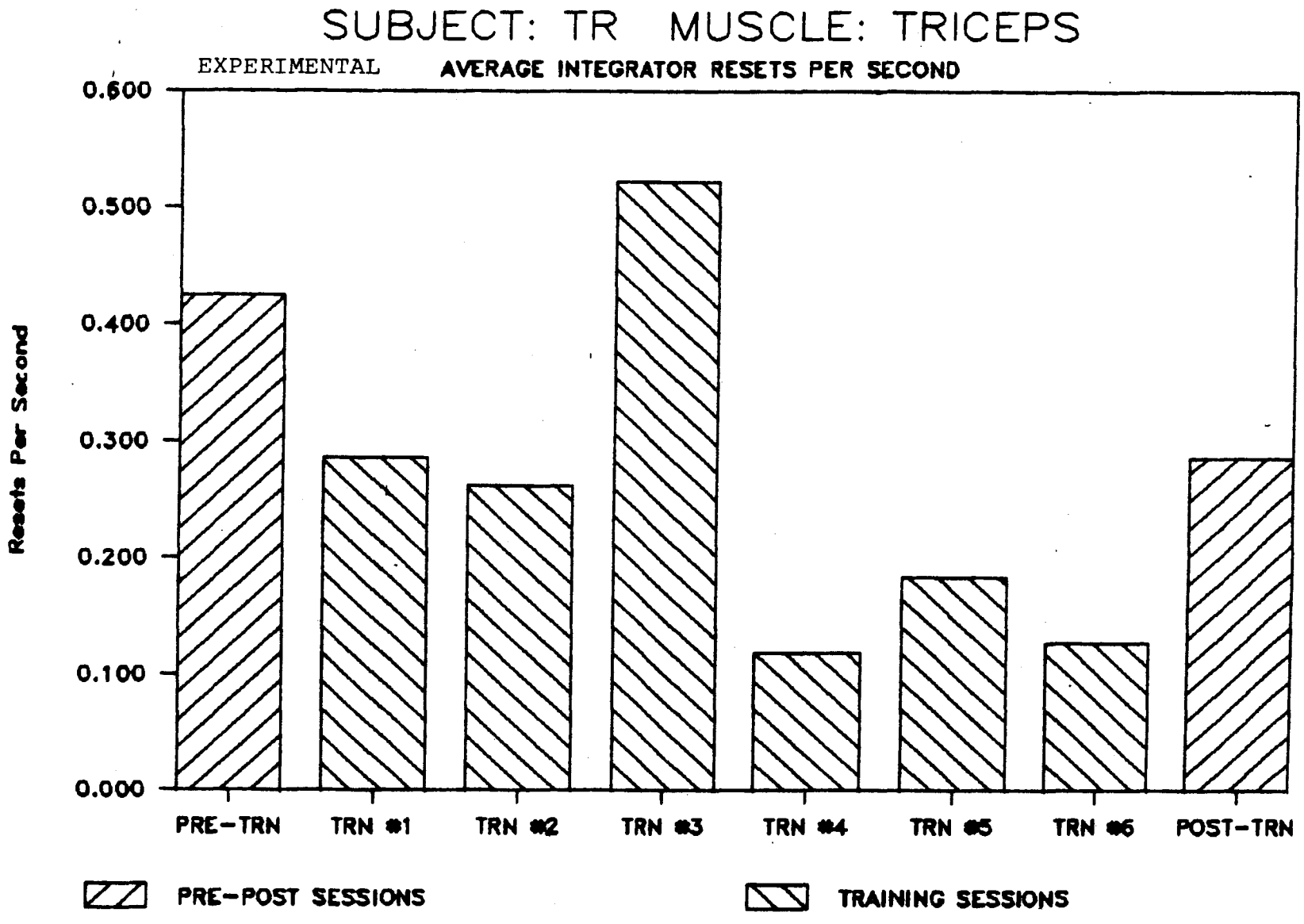
0.300
0.250
0.200
0.150
0.100
0.050
0.000
TRL#1
TRL#2
TRL#3
TRL#4
TRL#5
TRL#6
TRAINING TRIALS

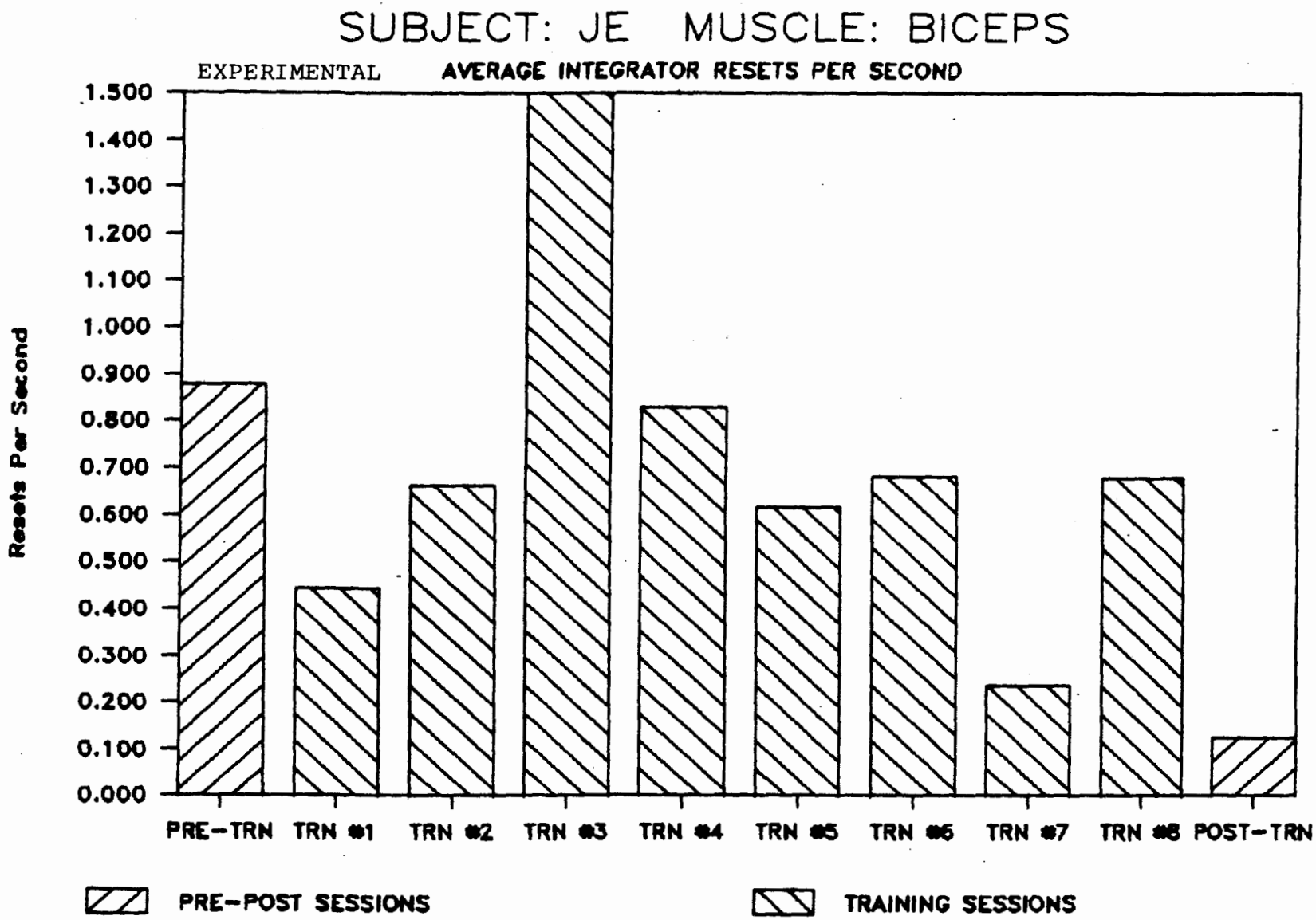
77

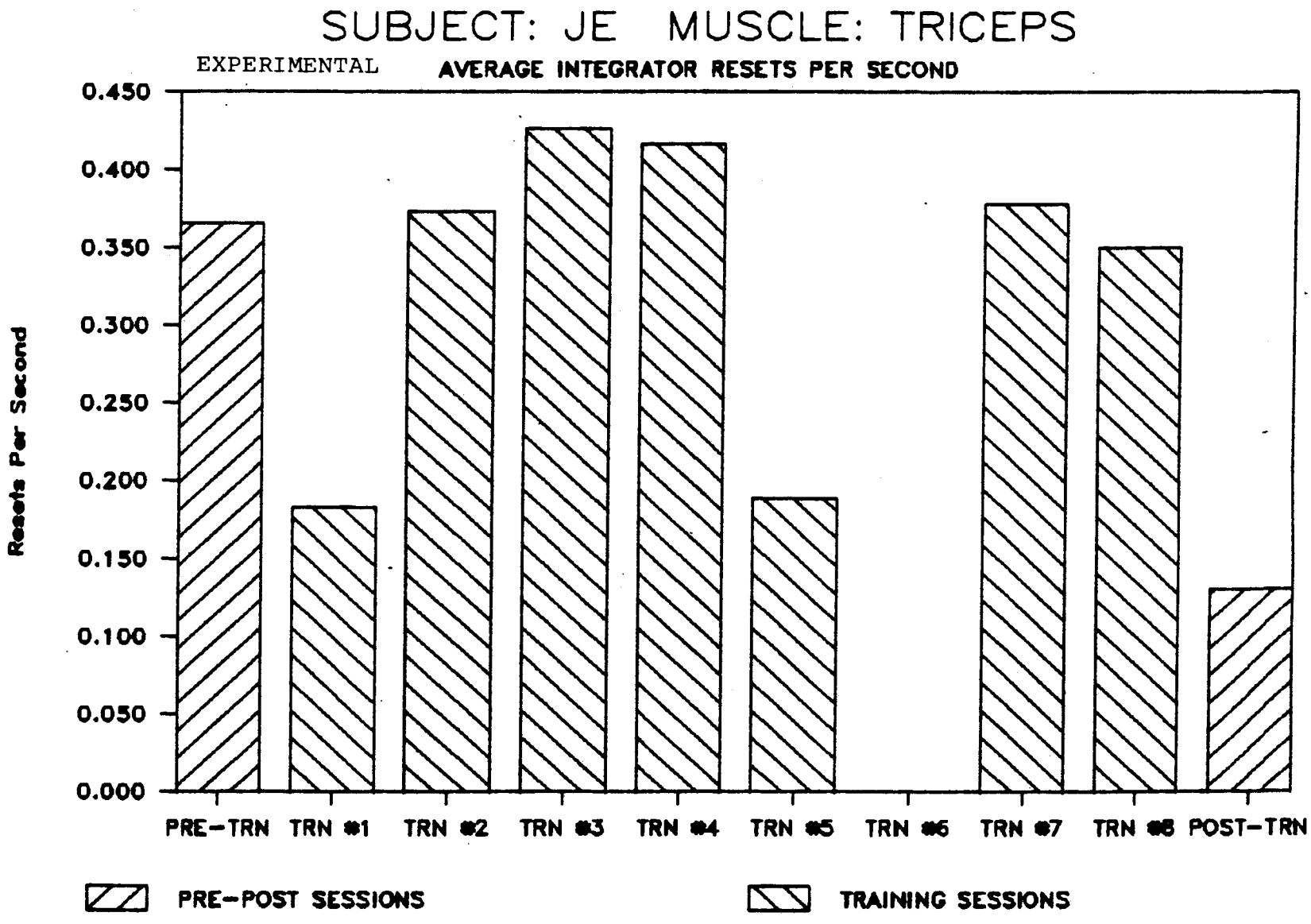
Appendix 2.
Set 3

Mean Pre-Training, Training and Post-Training Comparisons.


SUBJECT: TR MUSCLE: BICEPS
EXPERIMENTAL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
1.100
1.000
0.900
0.800
0.700
0.600
0.500
0.400
0.300
0.200
0.100
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS


SUBJECT: TR MUSCLE: TRICEPS
EXPERIMENTAL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
0.600
0.500
0.400
0.300
0.200
0.100
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS


SUBJECT: JE MUSCLE: BICEPS
EXPERIMENTAL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
1.500
1.400
1.300
1.200
1.100
1.000
0.900
0.800
0.700
0.600
0.500
0.400
0.300
0.200
0.100
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
TRN #7
TRN #8
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS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EXPERIMENTAL
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Resets Per Second
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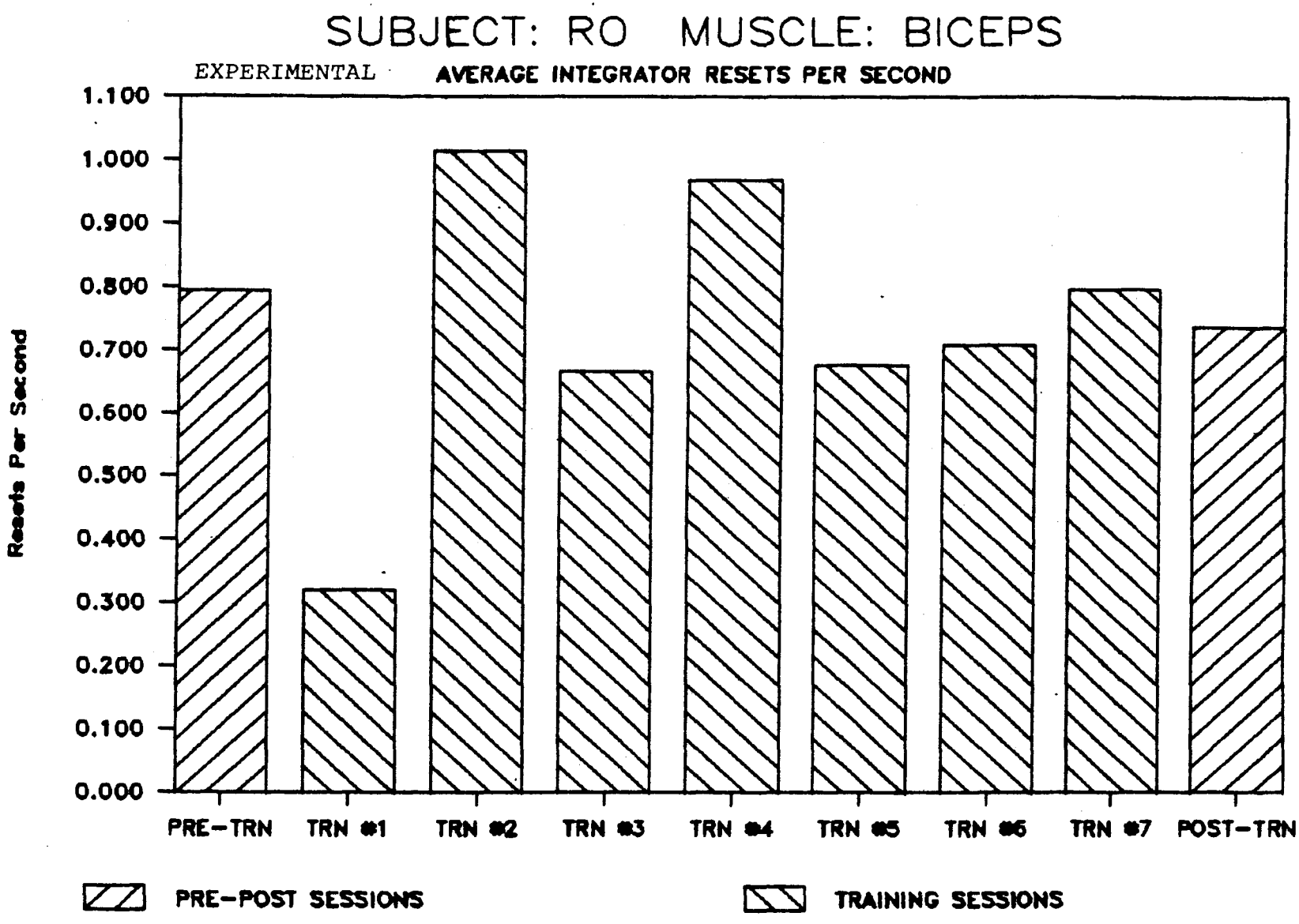
0.400
0.350
0.300
0.250
0.200
0.150
0.100
0.050
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
TRN #7
TRN #8
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS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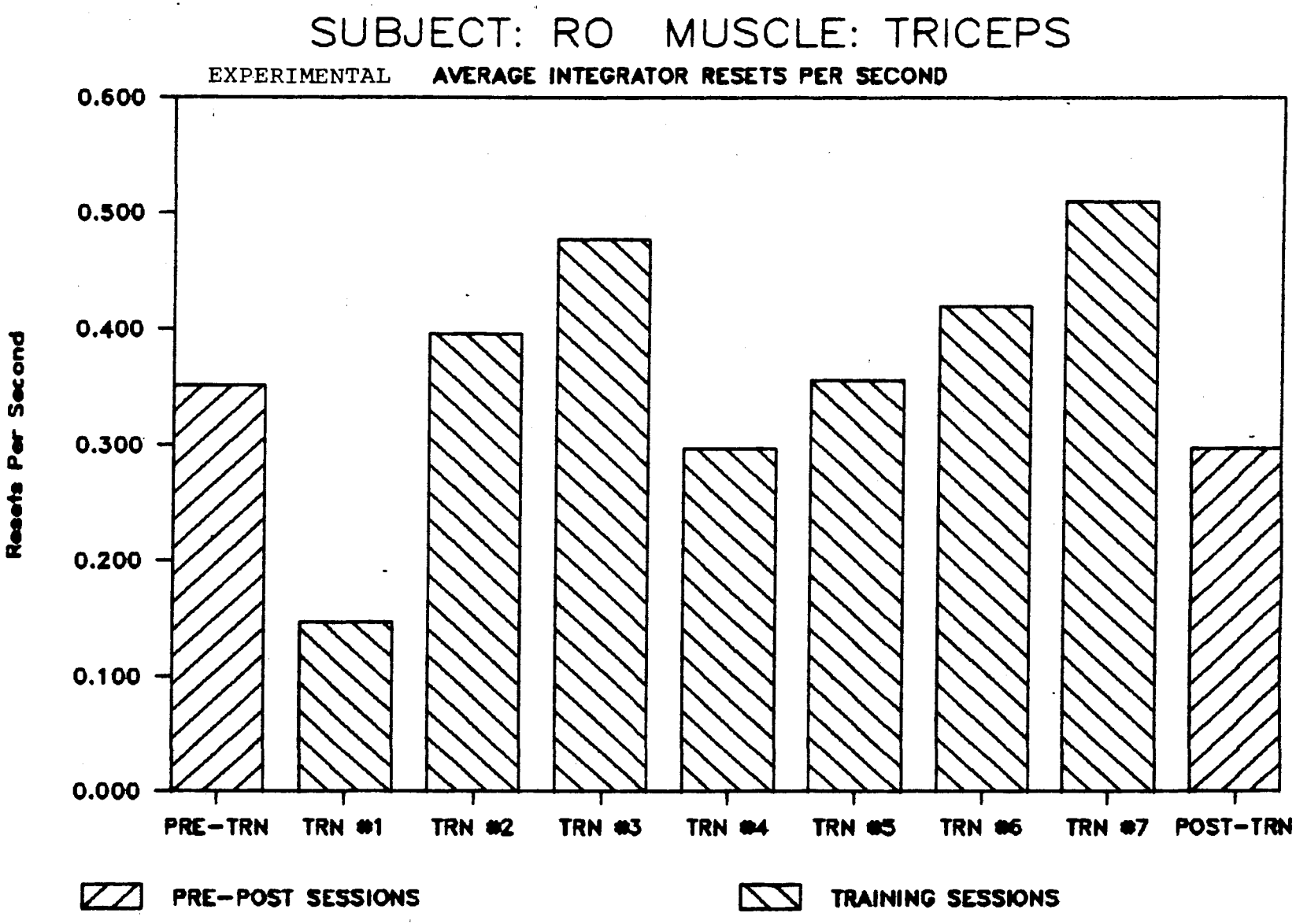
EXPERIMENTAL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
1.100
1.000
0.900
0.800
0.700
0.600
0.500
0.400
0.300
0.200
0.100
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
TRN #7
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS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EXPERIMENTAL AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
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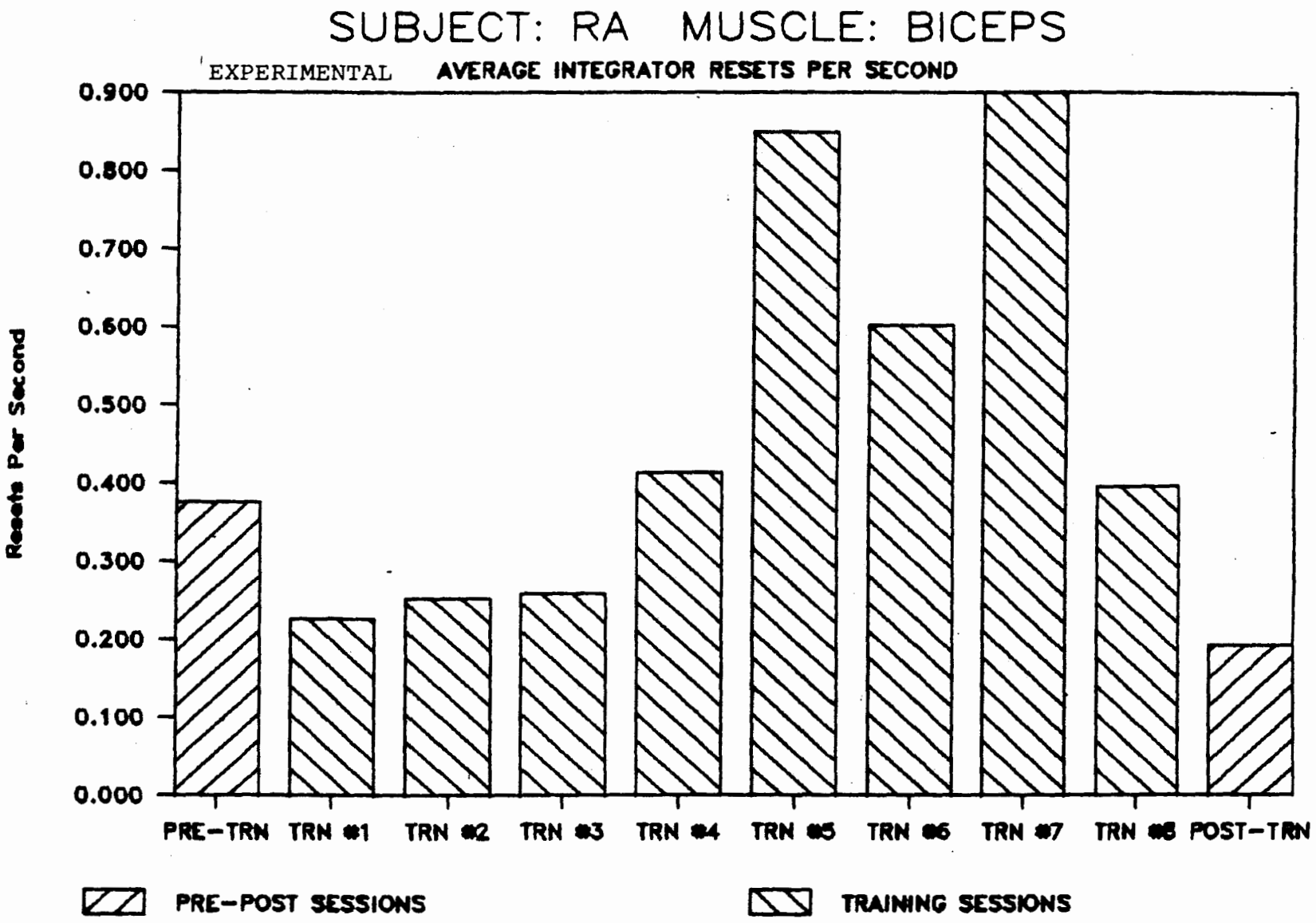
0.500
0.400
0.300
0.200
0.100
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
TRN #7
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS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EXPERIMENTAL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
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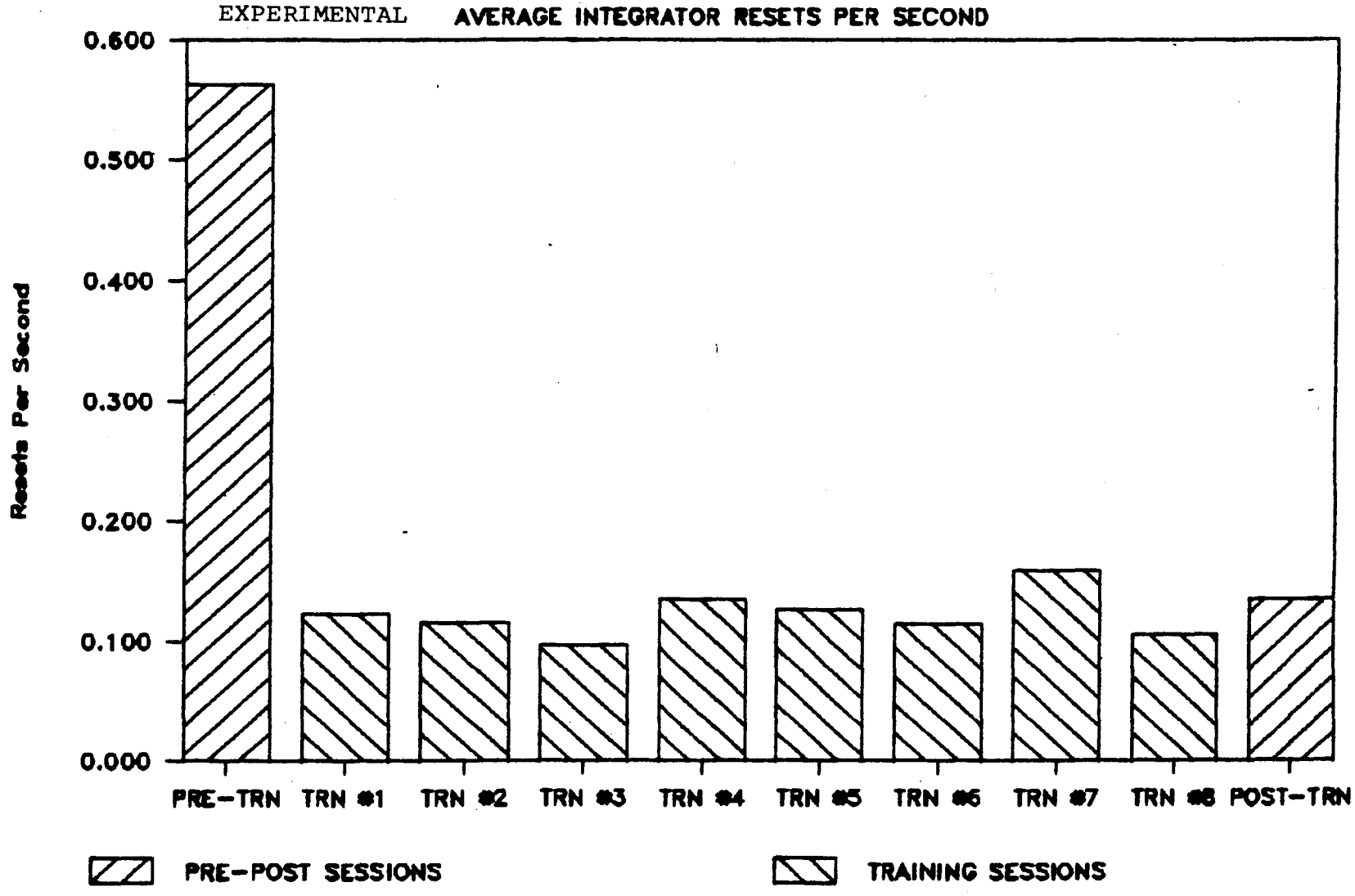
0.800
0.700
0.600
0.500
0.400
0.300
0.200
0.100
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
TRN #7
TRN #8
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS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EXPERIMENTAL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
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0.500
0.400
0.300
0.200
0.100
0.000
PRE-TRN
TRN #1
TRN #2
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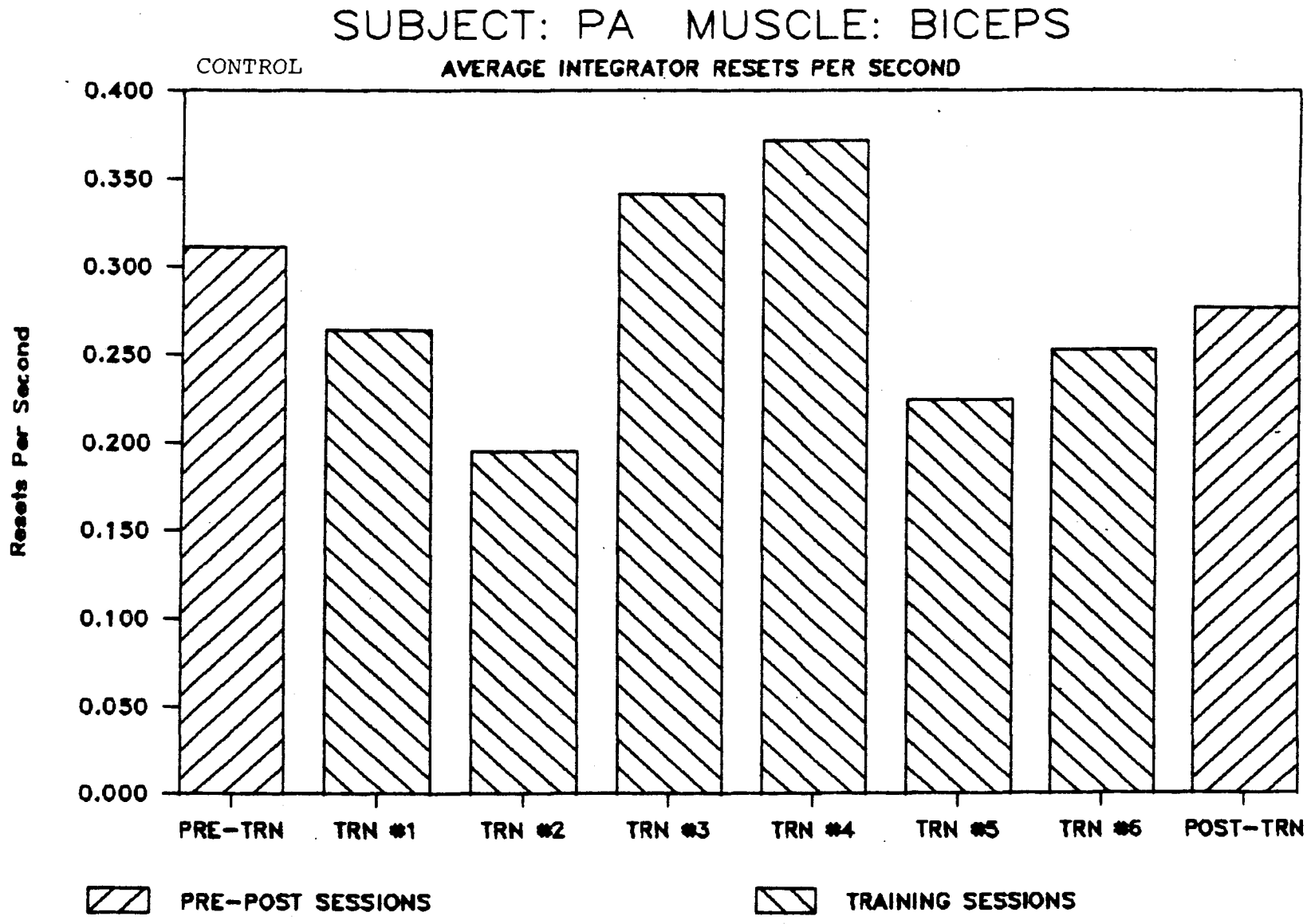
TRN #4
TRN #5
TRN #6
TRN #7
TRN #8
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS


SUBJECT: PA MUSCLE: BICEPS
CONTROL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
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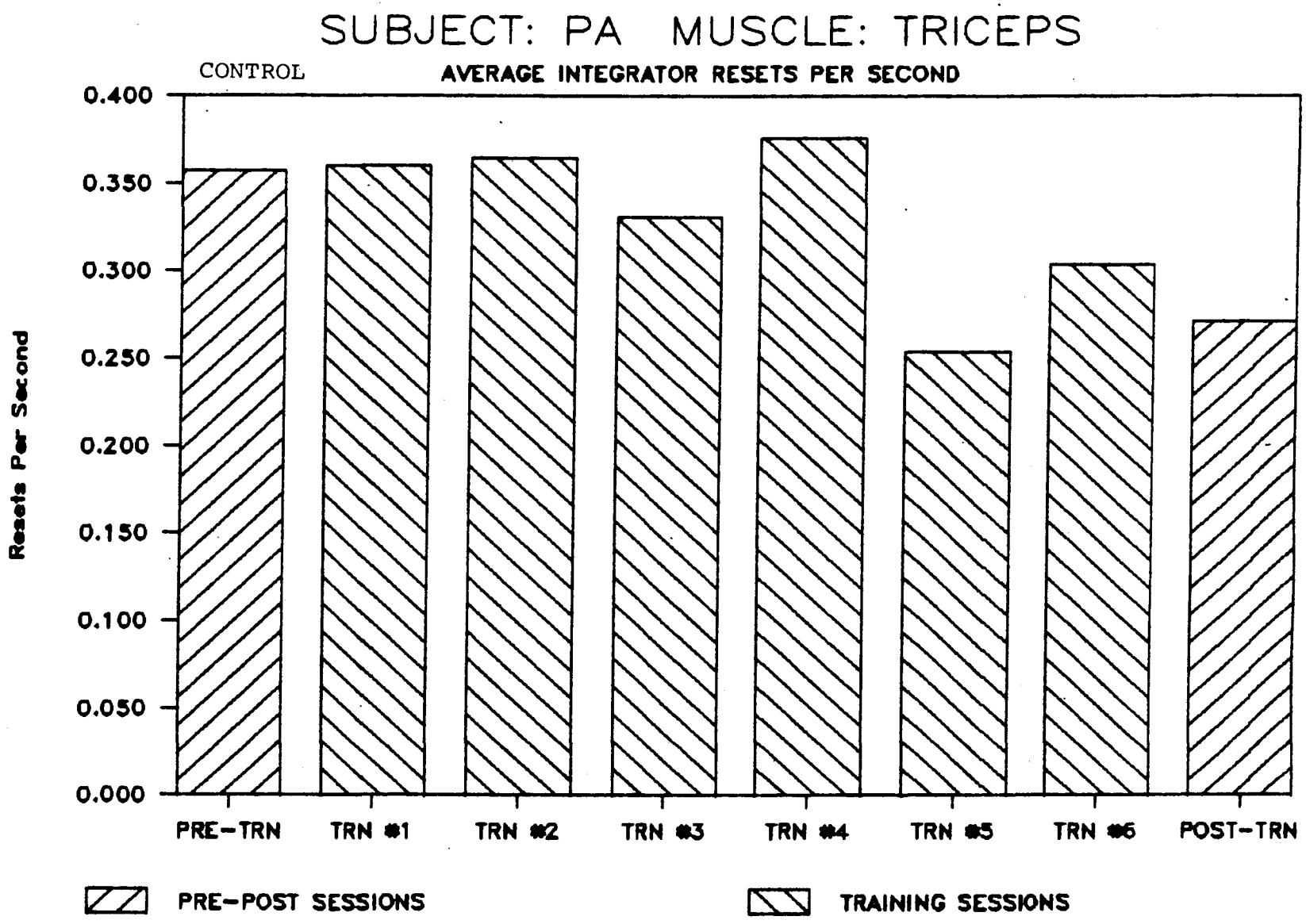
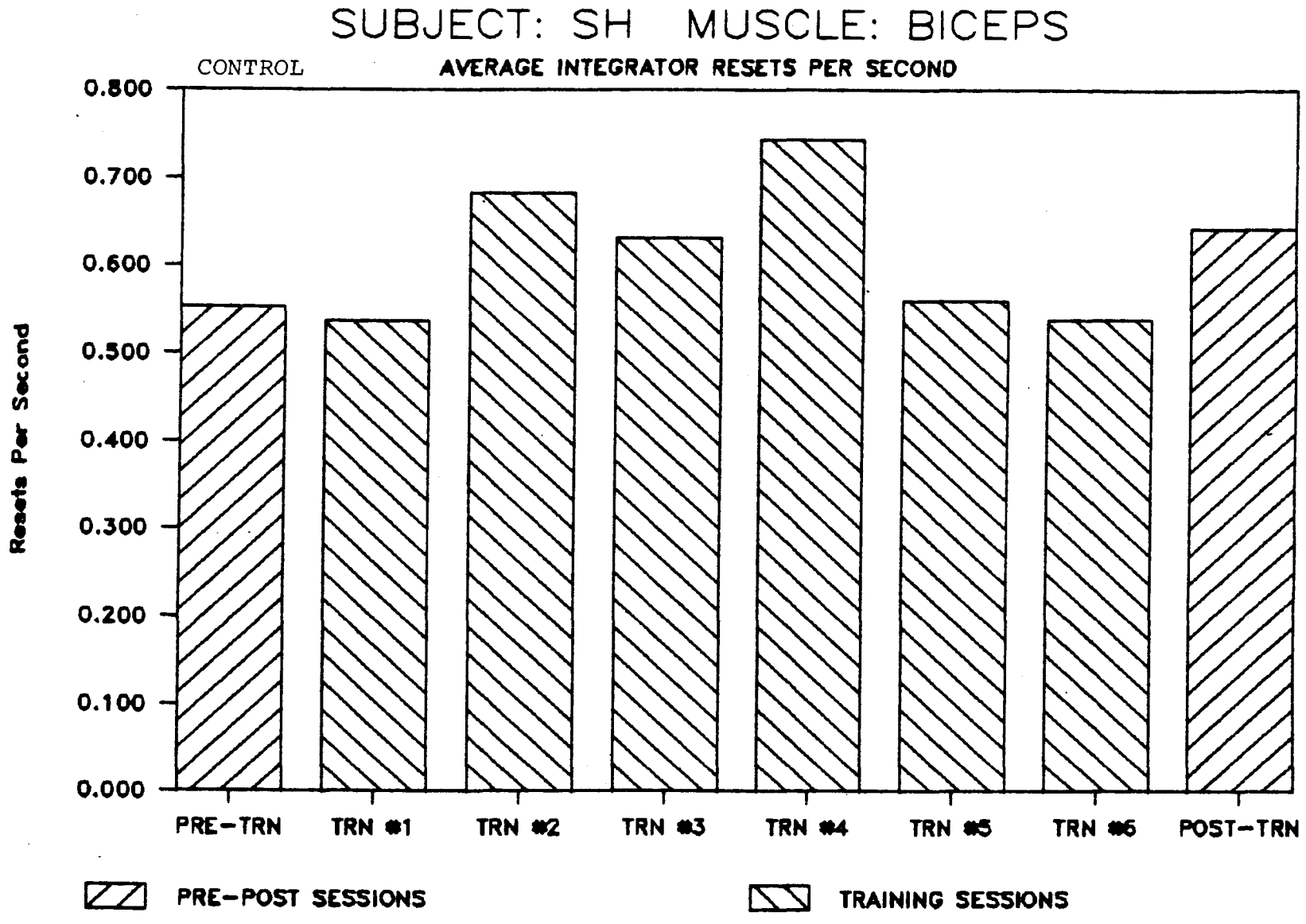
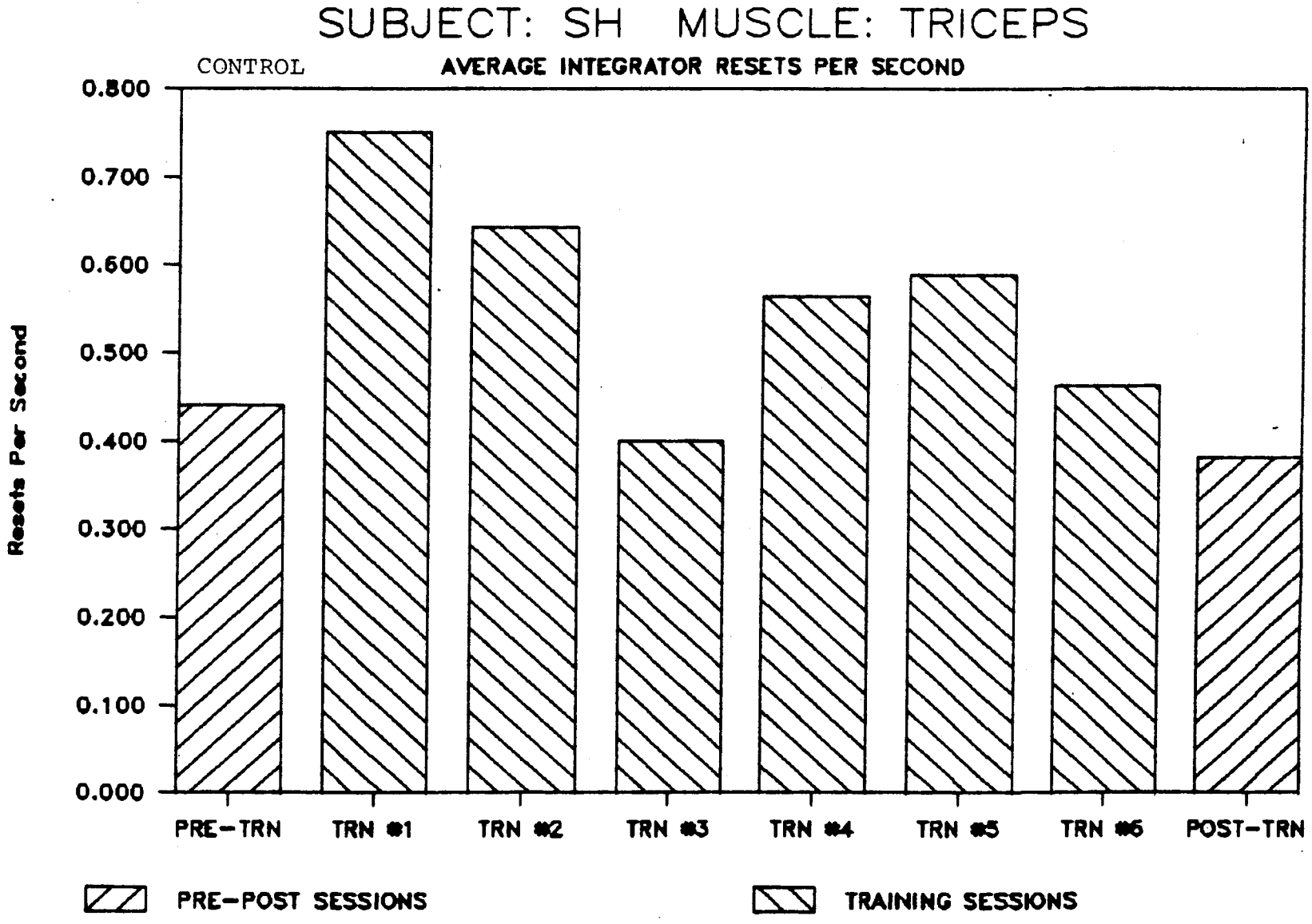
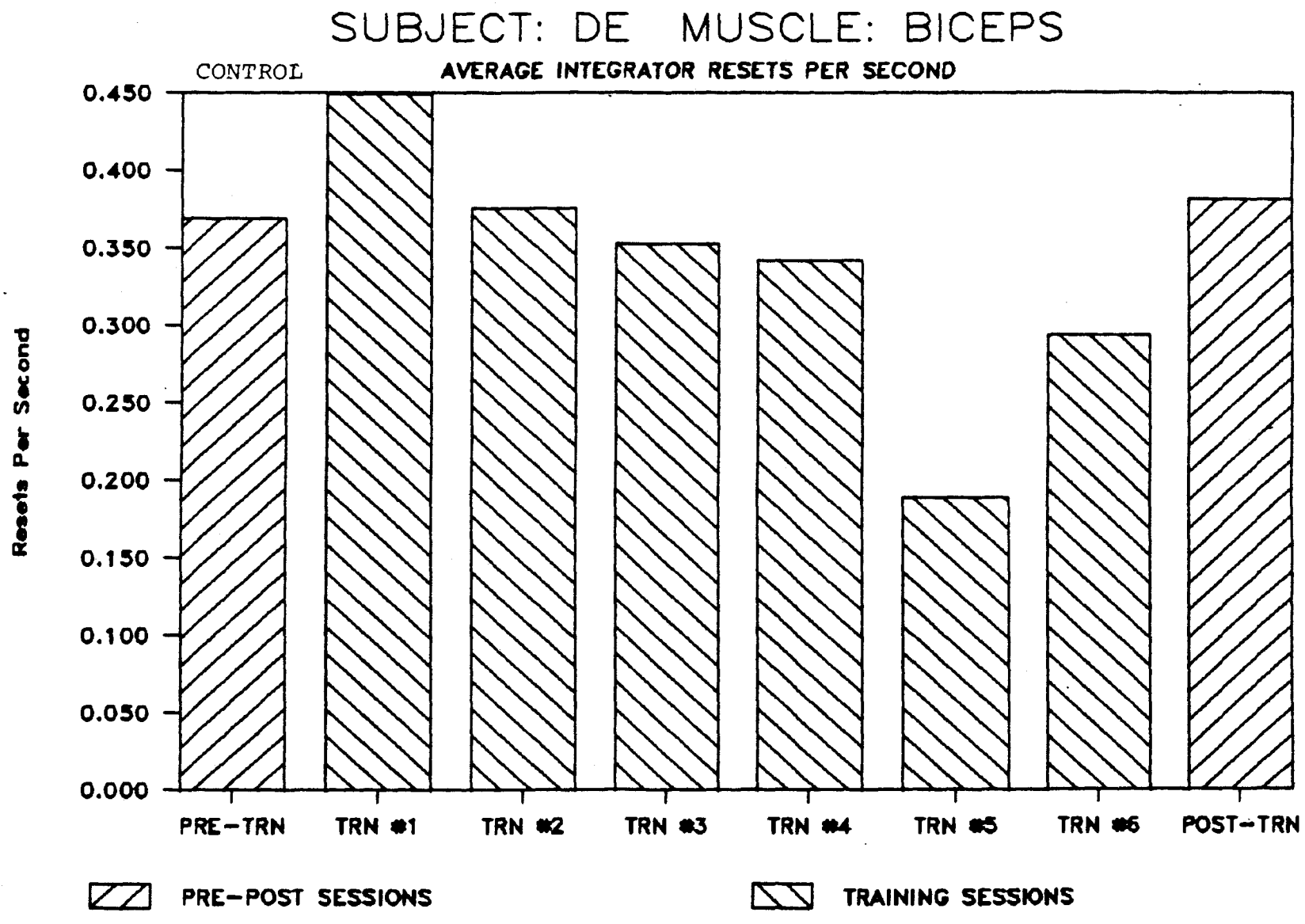
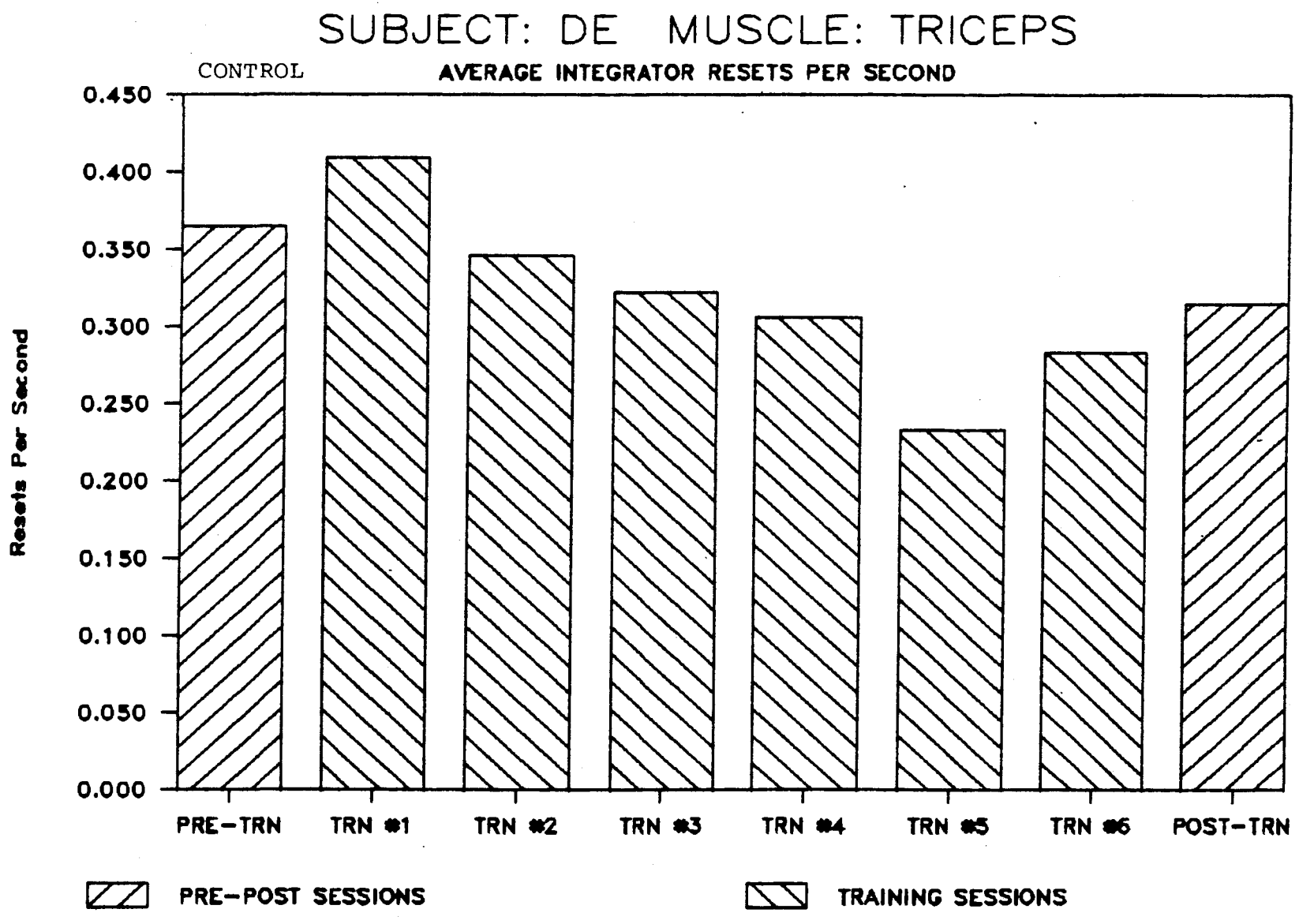
0.350
0.300
0.250
0.200
0.150
0.100
0.050
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS


SUBJECT: PA MUSCLE: TRICEPS
CONTROL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
0.400
0.350
0.300
0.250
0.200
0.150
0.100
0.050
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS


SUBJECT: SH MUSCLE: BICEPS
CONTROL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
0.800
0.700
0.600
0.500
0.400
0.300
0.200
0.100
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS


SUBJECT: SH MUSCLE: TRICEPS
CONTROL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
0.800
0.700
0.600
0.500
0.400
0.300
0.200
0.100
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS


SUBJECT: DE MUSCLE: BICEPS
CONTROL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
0.450
0.400
0.350
0.300
0.250
0.200
0.150
0.100
0.050
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS


SUBJECT: DE MUSCLE: TRICEPS
CONTROL
AVERAGE INTEGRATOR RESETS PER SECOND
Resets Per Second
0.450
0.400
0.350
0.300
0.250
0.200
0.150
0.100
0.050
0.000
PRE-TRN
TRN #1
TRN #2
TRN #3
TRN #4
TRN #5
TRN #6
POST-TRN
PRE-POST SESSIONS
TRAINING SESSIONS

References

Anderson, J.R. (1978). Arguments concerning representations for mental imagery. Psychological Review, 85, 249-277.

Basmajian, J.V. (1975). Biofeedback in physical rehabilitation: An overview. Biofeedback Technique In Clinical Practice: Volume 2. New York: Biomonitoring Applications. (Tape).

Brudny, J., Korein, J., Levidow, L., Grynbaum, B.B., Lieberman, A. & Friedmann, L.W. (1974). Sensory feedback therapy as a modality of treatment in central nervous system disorders of voluntary movement. Neurology, 24, 925-932.

Cataldo, M.F., Bird, B.L., & Cunningham, C.F. (1978). Experimental analysis of EMG feedback in treating cerebral palsy. Journal of Behavioral Medicine, 1(3), 311-322.

Chen, W. Retention of EMG biofeedback relaxation training. (1983). Perceptual and Motor Skills, 56, 671-674.

Cruickshank, W.M. (1976). The problem and its scope. In W.M. Cruickshank (Ed.), Cerebral Palsy: A Developmental Disability (3rd ed.), 1-28. Syracuse: Syracuse University Press.

Davis, C.M. Brickett, P. Stern, R.M. & Kimball, W.H. (1978). Tension in the two frontales: Electrode placement and artifact in the recording of forehead EMG. Psychophysiology, 15(6), 591-593.

Denhoff, E. (1976). Medical aspects. In W.M. Cruickshank (Ed.), Cerebral Palsy: A developmental disability (3rd ed.), 29-72. Syracuse: Syracuse University Press.

Fernando, C.K., & Basmajian, J. V. (1978). Biofeedback in physical medicine and rehabilitation. Biofeedback and Self-Regulation, 3(4), 435-455.

Finley, W.W., Niman, C., Standley, J. & Ender, P. (1976). Frontal EMG training of athetoid cerebral palsy patients. Biofeedback and Self-Regulation, 1(2), 169-182.

Finley, W.W., Niman, C., Standly, J. & Wansley, R.A. (1977). Electro-physiologic behavior modification of frontal EMG in cerebral-palsied children. Biofeedback and Self-Regulation, 2(1), 59-79.

Grass Instruction Manual. (1978). Polygraph Summating Integrator Model 7P10. Grass Medical Instruments. Quincy.

Hallett, A. & Alvarez, N. (1983). Attempted rapid elbow flexion movements in patients with athetosis. Journal of Neurology, Neurosurgery, and Psychiatry, 46, 745-750.

Harris, F.A., Spelman, F.A., Hymer, J.W. (1974). Electronic sensory aids as treatment for cerebral-palsied Children. Physical Therapy, 54,(4), 354-365.

Lazar, Billie S. (1977). Hypnotic imagery as a tool in working with a cerebral palsied child. The International Journal of Clinical and Experimental Hypnosis, XXV(2), 78-87.

Love Jr., W.A. (1975). EMG biofeedback: Therapeutic applications. Bio-feedback Technique in Clinical Practice volume 1. New York: Biomonitoring Applications. (tape).

Melyn, M.A., Grossman, H.J., (1976). Neurophysiological correlates. In W.M. Cruickshank (Ed.), Cerebral Palsy: A Developmental Disability (3rd ed.), 1-28. Syracuse: Syracuse University Press.

Neilson, P.D., & O'Dwyer, N.J. (1984). Reproducibility and variability of speech muscle activity in athetoid dysarthria of cerebral palsy. Journal of Speech and Hearing Research, 27, 502-517.

Ontario Crippled Children's Centre, Rehabilitation engineering research (1976). Bio-engineering in the Management of the Cerebral Palsied Child - Biofeedback Training. Final Report - 1972-1975. Toronto: Author.

Perlstein, M.A. (1949). Medical aspects of cerebral palsy. Nervous Child, 8, 125-51.

Roland, P.E. (1978). Sensory feedback to the cerebral cortex during voluntary movement in man. The Behavioural and Brain Sciences, 12, 129-71.

Rosenzweig, M.R., Leiman, A.L. (1982). Physiological Psychology. Toronto: D.C. Heath.

Scartelli, J.P. (1982). The effect of sedative music on electromyographic biofeedback assisted relaxation training of spastic cerebral palsied adults. Journal of Music Therapy, 19(4), 210-218.

Skrotzky, K., Gallenstein, J.S. & Osternig, L.R. (1978). Effects of electromyographic feedback training on motor control in spastic cerebral palsy. Physical Therapy, 58(5) 547-552.