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Title: AN ELECTROMYOGRAPHIC APPROACH TO MOTOR UNIT ACTIVITY DURING SUSTAINED ISOMETRIC CONTRACTIONS

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Eight male college students served as subjects in an investigation to establish a procedural model for research of individual motor units during sustained isometric contractions. Teca TE-4 electromyograph system with needle electrode provided electrical potential recordings from active muscle fibers in the right biceps. Various experimental procedures were tested to produce necessary electromyogram clarity for identification of individual motor unit potentials and measurement of their frequency and amplitude. Established procedures utilized 7 to 10% maximum contraction with stabilization of subject's body position to prevent movement, elimination of electrical interference in and around testing room, and careful placement of needle electrode. Pre-fatigue electrical activity was recorded during the first minute into the contraction. Three post-fatigue recordings were taken at one-minute intervals beginning ten minutes after pre-fatigue recordings. Suitability of the model was determined by quality of electromyogram recordings. EMG traces allowing motor unit

evaluation by the application of Student's t-test for changes occurring in frequency and amplitude reflected model acceptability. A model was produced which permitted evaluation of motor units during sustained isometric contractions.

An Electromyographic Approach to Motor Unit Activity During Sustained Isometric Contractions

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CHAPTER I

INTRODUCTION

The human neuromuscular system is an intriguing and complex mechanism. Its many individual fibers may be called upon to perform a wide variety of tasks. Few fibers may be activated with very intricate, precise control for threading a needle. On the other hand, the system may utilize all or nearly all of the body's fibers for a major effort such as the Olympic clean and jerk of 562 pounds.

Furthermore, muscles of the body appear to have a tremendous potential for endurance. In activities which require less than maximal effort, muscles may operate for hours as classically demonstrated in the marathon run.

Physiological mechanisms which control the millions of fibers in the body for millions of body movements must have a capability beyond any computer. Equally astonishing is the system which allows a muscle to perform for a seemingly indefinite period of time.

Numerous scientific studies have been concerned with the neuromuscular system, yet many rudimentary questions remain

unanswered. Existing research provides contradictory evidence in the quest for solutions to basic questions. Motor unit utilization, as monitored by electrical activity, is one topic of concern and is the focus of attention in this thesis.

Statement of Problem

Published literature provides no universally accepted conclusions regarding utilization of individual motor units throughout a sustained contraction. Endurance qualities, rotational use, and fatigue sites of motor units are specific topics which exemplify the present unsettled state of research.

Evidence is available to suggest that muscle fibers weaken rapidly while other research indicates indefinite endurance. Experimentation supports the theory of motor unit rotation to prevent fatigue or to maintain tension as fatigue develops. Studies also provide evidence that rotation of motor units does not occur. Five different fatigue sites in the nerve-muscle track have been identified as the "weak link" during muscular effort. Identified sites are the central nervous system, neuronal synapse, neuromuscular junction, excitation contraction coupling, and the contraction mechanism itself.

Contradictory evidence in existing literature may be due to variations in testing parameters. Experiments have been conducted on a variety of test animals. Nearly all of the major muscles have

been examined. Muscles were studied under excised and in-vivo conditions. Great variations existed in the level of contractions studied. Contractions have been produced by voluntary effort and by artificial electrical stimulation introduced at various points. Motor unit electrical activity has been detected by surface and by needle electrodes. Such variations in experimental procedures produce results which are contradictory and do not lead to universally accepted conclusions.

The purpose of this investigation was to establish a procedural model for study of motor unit activity. Development of such a model required extracting meaningful muscle fiber information during contractions and identifying technique for data evaluation.

The hypothesis in this investigation was that a model can be created for research of motor unit activity during sustained isometric contractions. Verification of model suitability was established through successful evaluation of sample electromyograms. Student's t-test served as a basis for evaluating changes occurring in frequency and amplitude.

Significance of Study

Conflicts in theory observed in review of existing literature suggest the necessity for universally accepted procedures in research of skeletal muscle motor units. This investigation has identified a method of isolating electrical response of individual muscle fibers during sustained, fatigue-producing, isometric contractions. The

model isolated a specific muscle area and low force contractions under actual physiological conditions.

Procedures established in this study will provide information on amplitude and frequency of motor unit potentials and reveal rotation and fatigue characteristics. Discrepancies observed in existing literature may be resolved through the application of these procedures.

Consideration was also focused on three important concerns often omitted in muscle fiber research. These topics deal with the afferent nervous system, muscle fiber types and physical/chemical changes in intercellular and extracellular fluids.

Methodology

A Teca TE-4 electromyograph was utilized to monitor electrical potentials in human biceps brachii muscles during sustained voluntary contractions. Subjects were seated in a specially designed apparatus chair which stabilized the right shoulder and arm. Selected weights of 3 to 13 pounds were supported by the right arm at 90° flexion. Implanted needle electrodes were used to detect isolated electrical activity of individual motor units.

Eight male college students were used as subjects. All were active participants in physical conditioning programs and were in good health.

Experimentation was conducted in two phases. Phase one established procedures allowing identification and measurement of individual potentials recorded on the electromyogram. Measurements consisted of the distance between and height of individual potential spikes. Variations in weight, needle position and electromyograph settings were tested. Modification of the apparatus chair and reductions in electrical interference were accomplished. Identification of units was based on distinctive form, relatively uniform frequency and fairly constant amplitude of potentials.

Phase two established procedures for monitoring active units through a fatiguing condition. Continued isolation and identification of unit spikes with measureable characteristics was mandatory. Variations in weight were used to produce fatigue. Different sample recording systems provided motor unit potential information. Mean frequency and amplitude changes through fatigue underwent testing for statistical significance by Student's t-test.

Delimitations

Restrictions imposed on this investigation included subject population, muscle fibers tested, type of contraction and intensity of contraction. Such boundaries were established in order to eliminate as many variables as possible affecting recorded activity.

All subjects were in good physical condition. Population size

allowed multiple testing sessions for each subject.

Experimentation utilized the human biceps brachii muscle exclusively. Furthermore, the electrical detection area was limited to a few millimeters in diameter due to use of coaxial needle electrodes.

Testing procedures utilized only low tension contractions of 5% to 20% maximum over a moderate time period of 15 to 30 minutes. All contractions were isometric.

Limitations

Procedures developed in this investigation are applicable only for the biceps brachii during static contractions.

Results represent only low-tension contractions where blood flow is not occluded significantly. Conclusions apply to those units which are active at low tension and may not apply to those at high level contractions.

Minute detection areas, necessary to isolate individual motor units, established the possibility that significant activity existed in non-recorded units. Shifting of the subject's body, shoulder or arm position also limits application of results.

Significant changes in amplitude must be considered carefully since fluctuations in intercellular heat and chemical composition may affect transmission of impulses from the fiber to electrode tip.

Additionally, alteration of needle tip position with respect to active fibers affects spike height.

CHAPTER II

REVIEW OF LITERATURE

Over the years man has studied the neuromuscular system using animals and himself as subjects. He has isolated and experimented with all components of the system. He has tested nearly all muscles of the body over the entire range of tension. From this research comes important truths about the function of the human body and yet, unanswered questions still exist. Researchers have produced numerous cases of conflicting evidence which remain unsolved today.

This review presents some accepted beliefs regarding structure and function of the neuromuscular system. Electromyography is discussed along with its application to the subject of this thesis. Finally, conflicting theories concerning utilization of motor units and fatigue are surfaced.

Basic Neuromuscular System

The neuromuscular system consists of a complex computer center in the brain, a myriad of impulse channels down the spinal cord with separate nerves exiting at various segments, and countless muscle fibers attached at the distal end of the nerve branches. Muscle contractions result from successful transmission of impulses from the brain, over neuronal channels and onto the muscle fiber.

Anatomy and function of the biceps brachii neuromuscular chain are of special concern in this investigation.

Impulse Trace

Impulses for voluntary contractions of the biceps brachii originate in the motor cortex of the brain (38, 45). The corticospinal tract carries these impulses through the medulla and onto the spinal cord. In the gray matter of the spinal cord, internuncial neurons transmit these impulses from the descending tract to the anterior motor neurons. For the biceps, this transition is made to the musculocutaneous nerve of the brachial plexus (67). Internuncial cells also integrate afferent impulses with the impulses from the motor cortex (38). The musculocutaneous nerve is a group of large neurons exiting through the anterior horn of the spinal gray matter to innervate the biceps and other muscles of the arm (42).

A single motoneuron from the anterior horn branches off to innervate 10 to 200 individual muscle fibers and creates a motor unit (38, 53). According to Buchthal (13), as many as 1000 fibers may be associated with a single unit in large human limbs. The number of fibers per unit varies, depending on the degree of control needed by the muscle. Units requiring fine control (i.e., muscles of the eye) have few muscle fibers, while units used in gross motor movements (i.e., muscles of the leg) contain many muscle fibers (45).

Physiologists disagree concerning the placement of these individual muscle fibers in the overall muscle. Jensen (43) contends that fibers of a single unit are not grouped but rather are spread throughout the muscle belly. Contraction of a single unit will appear as a slight contraction of the entire muscle. Karpovich (45) reports that this "interspersed" characteristic is usually true. According to Buchthal (13), however, fibers of a single motor unit in the biceps are confined to an area 4-6 mm in diameter with other units intermingled in the same space.

Muscle Fiber Response to Stimuli

Single nerve impulses which reach the neuromuscular junction will cause a complete contraction of the fiber or no contraction at all. This phenomenon is known as the All-or-None Law (7, 22, 45, 73, 79, 81). No provision exists for gradation of the individual fiber contraction through the magnitude of its impulse. When the resting potential of the muscle membrane is depolarized sufficiently, the resultant action potential over the sarcolemma causes a fiber twitch. Impulses in excess of this threshold level yield no greater contraction.

The possibility does exist that an impulse will cause no response. Nerve action potentials may be of insufficient intensity to excite the muscle membrance (73). Additionally, a second impulse

may too closely follow (i.e., 3/1000 second) the previous impulse and produce no contraction (45, 81). Failure to respond to this second impulse is caused by a temporary "loss of irritability" and is called the refractory period. However, this consideration should not be a factor in large human muscles since impulse frequency does not approach that rate.

To say that these units contract or do not contract with each impulse does not however imply that the strength of the contractions will always be the same. Each fiber will contract to its "immediate ability" (42, 73, 79). Ability to produce tension is altered by initial length of the fiber, temperature and chemical environment (56). Stretched fibers yield stronger contractions than non-stretched fibers. Warmer temperatures create a greater force up to a certain point (near $40^{\circ}C$). An ideal chemical environment with adequate oxygen supply will likewise produce a greater contraction than will surroundings depleted of energy sources and oxygen with a buildup of waste products.

Muscle Contraction

Contraction strength of the entire muscle may vary from minimal muscle tone to complete 100% maximum voluntary contraction. Resultant force is a product of the number of motor units excited and the frequency at which those excited units contract (2, 7, 20, 38, 53,

54, 64, 68, 75, 77, 79). The number of excited motor units and their frequencies is dictated by the level of contraction demanded by the impulses from the motor cortex as altered by afferent activity from the muscle (31). Total contraction then, is a summation of all motor unit activity to produce a smooth muscular force.

Low level contractions excite only a small percentage of motor units (34, 45). Impulse frequencies increase and additional fibers (motor units) are brought into play to produce greater contractions (8, 12, 25, 34, 53, 54, 73, 77). Theoretically, during a maximal contraction, all units are firing at a rapid rate. Activation of additional motor units to increase force is referred to as spacial summation (45). Some physiologists (8, 31) believe the recruitment of additional units accounts for most of the tension increase in sub-maximal contractions.

Frequencies of neuronal discharge range from five to fifty or more impulses per second with each muscle fiber in that unit responding to every impulse (2, 3, 12, 39, 45, 53). According to Guyton (39), each motor unit responds only two to three times per second in a weak contraction. These low frequencies cause the muscle fibers to respond by separate "twitch" contractions. Asynchronous twitches from many operating units create a smooth muscular force (5, 34, 45, 53, 66). Neuronal circuits in the spinal cord control these twitches by distributing "the impulses evenly and

sequentially" among various motor units of the muscle (38). Higher frequencies cause individual contractions to build onto the previous contraction since its tension is not fully relaxed. The result is a contractile force greater than that at lower frequencies (52, 56). Such building of force is known as temporal summation or wave summation (42, 45). Continued increase in frequency causes the twitches to become fused and individual contractions are not visible. A muscle is then in a state of tetanus (42). Frequencies in excess of "certain limits" result in a decrease in tension (55).

Large skeletal muscles tetanize at about 35 impulses per second with maximum force developed near fifty impulses per second (42). Seyffarth (75) found that the maximum frequency specifically for the elbow flexors to be 55 to 60 impulses per second. Cooper and Eccles (20) established that impulse intervals of one-half the contraction time produced a contraction of nearly full tension. Merton's (58) findings regarding frequency and tension of the adductor pollicis are illustrated in Figure 1. His conclusion is in line with that of Cooper and Eccles. The arrow in Figure 1 indicates the point where contraction time and impulse interval are equal. Doubling that frequency produces nearly maximum tension. Forbes (30) believes that the impulse frequency is much higher for the forearm flexor. He states that the upper limit is between 300 and 1000, an assumption based upon the refractory period of motor neurons. According to Gellhorn

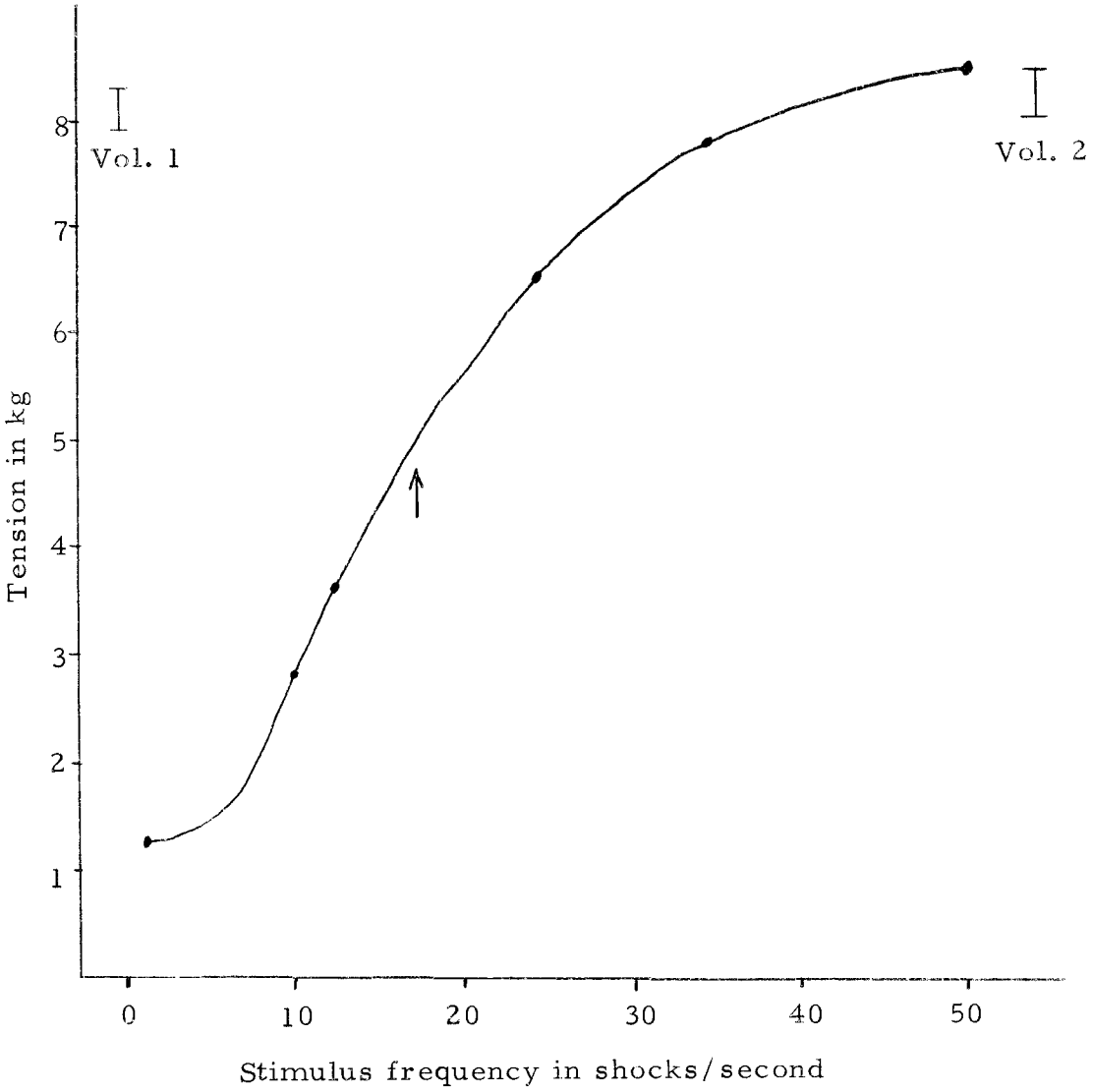

Figure 1. Relationship between tension and frequency and comparison with maximum voluntary contractions Vol. and Vol. 2. Zero frequency represents a single impulse. Arrow indicates the point where impulse interval and fiber contraction time are equal. Doubling that frequency produced near maximum force (58).

(32) the frequency will vary widely but does not approach its physiological limit, the refractory period.

Lindsley (53) studied frequency in various human muscles using a needle electrode. He found that units began responding with frequencies of five to ten per second in weak voluntary contractions. A few appeared with frequencies of 12 to 15 per second. Actual measurements of the biceps revealed the lowest possible frequency of five to six per second with the highest at maximal effort of 40 per second.

Adrian and Bronk (1) contend that increases in frequency have a greater effect on strength than does recruitment of additional units. Lindsley (53), on the other hand, concluded that frequency variation is a "more delicate method of grading" a muscular force, with recruitment being a quicker and more potent factor in building strength.

Merton (58) found a definite reproducible "ceiling" on voluntary muscular force which indicates a consistency on total recruitment of units and their frequencies. He also found that the "ceiling" was the same for both voluntary and artificially stimulated contractions, further indicating total involvement of units at maximal effort. Figure 1 illustrates this point by plotting single maximum voluntary contractions before (Vol. 1) and after (Vol. 2) artificially stimulated contractions to maximum.

Ikai, Yabe and Ischii (41), however, found that a maximum contraction resulting from artificial stimulation was about 30% greater

than that developed from a voluntary contraction. Their findings indicate that all motor units are not at the disposal of a voluntary effort for a given contraction.

Voluntary contractions are typically tetanic in nature responding to a "succession of closely spaced stimuli," according to Schneider (73). Other physiologists (34, 53, 65) however, believe that during sustained voluntary contractions, motor units discharge asynchronously at "relatively low frequencies" that rarely cause complete tetanus.

Although disagreement exists regarding specific details, research does establish that gradation of muscular force is a function of motor unit utilization. Activation of additional units and increase in frequency of stimulation will produce greater muscular tension. The fibers may or may not typically respond in tetanus. But evidence does indicate that individual twitches will occur during low force contractions.

Electromyography

Electrical impulses which pass over muscle fibers have measurable, finite characteristics. Since these impulses create the actual muscle contraction, a record of their activity reveals the operation of fibers involved in that contraction. Electromyography (EMG) is a tool used to provide information on a muscle in vivo by detecting

electrical changes which take place on the membrane of a contracting muscle (45). Changes in electrical potentials are transmitted away from the active membranes through the adjacent fluids and are picked up by electrodes. Signals are then amplified, displayed visually and audibly, and recorded for interpretation.

Surface and needle electrodes are available for use with the electromyograph. Surface electrodes are used to detect general or global electrical activity of the muscle within a few centimeter radius (65). They record the sum of many motor units. Implanted needle electrodes are used to detect activity in a more localized area of the muscle and allow a few units to be recorded individually. Needles may be concentric or monopolar. The concentric electrode is a needle within a needle while monopolar electrodes are separate, single pole needles insulated to near the tip. Concentric needle electrodes are best capable of isolating specific motor units due to their smaller area of reception (53, 56, 64, 65).

Since muscle fibers of various units are intermingled to some extent, several units may be detected by a single needle electrode (13). Criteria must be determined for identification of single motor units. Most researchers identify units by their distinctive form, fairly constant rhythm, and nearly uniform amplitude (34, 53, 75). Frequency may vary about ten percent of the average interval between responses or even greater in weak contractions (53, 75). Variation in

impulse intervals of a single unit is "more or less" independent of the intervals of other units (75).

With "carefully controlled minimal effort and precise placement of the active needle electrode" it is possible to detect and monitor a single motor unit (65). Potentials will be heard as "a sharp pop or click" when the needle tip is in direct contact with active fibers. Sounds from more distant units will be distorted to become a dull thump.

Resting potential across a membrane is 50 to 100 millivolts with the interior being negative (33, 65). As the action potential sweeps down a muscle membrane, the membrane is depolarized and repolarized in a very short period of time. This change in electrical stability is the variable that is recorded by the electromyograph.

A typical motor unit potential is triphasic (65) and is illustrated in Figure 2. Amplitude of the spike usually varies from 100 microvolts to a few millivolts (56, 65). Variations in amplitude are a result of several factors to include proximity of electrode to the fibers, number of active fibers in the unit, hypertrophy of the muscle fibers, and the degree of fiber synchronization (53, 65, 75). Distance between the fiber and electrode is altered by minute changes in needle position resulting from the muscle contraction (75). Additionally, changes in ionic composition around the fiber may affect the transmission of the electrical disturbance through the fluid (29).

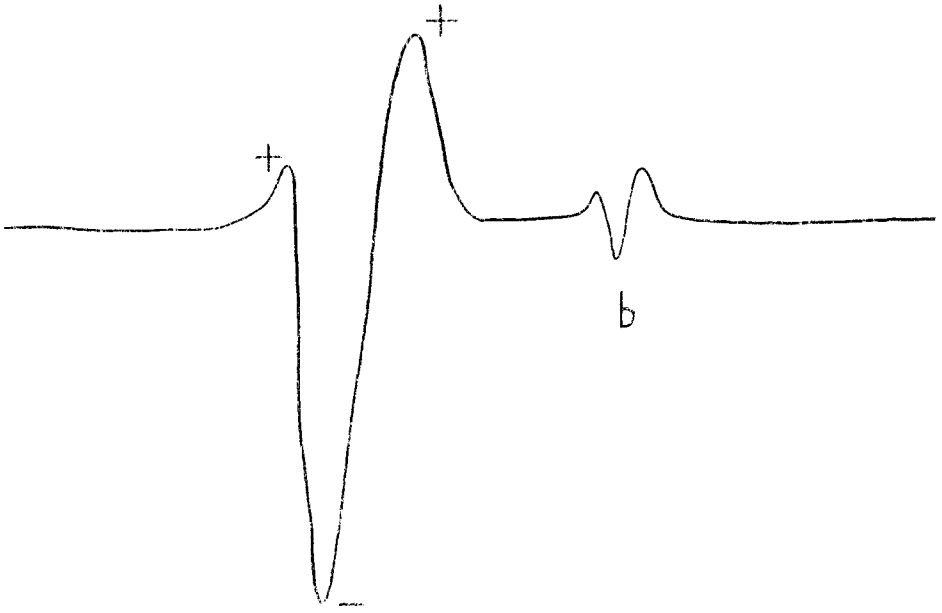

Figure 2. Typical appearance of a triphasic action potential as recorded on an electromyogram. Area "b" is another unit some distance from electrode.

Duration of an action potential is typically two to twelve milliseconds (65). This time period is shortened by more precise synchronization of fibers and by fewer active fibers in the unit.

"Quantitative electrical changes" recorded by the electromyograph have a direct relationship to the amount of force developed (8, 29, 31, 45, 49, 54, 55, 64, 72, 82). Increases in muscular tension are reflected by increases in recorded electrical activity. EMG records allow investigators to monitor changes in utilization of motor units and in their frequencies during muscular work.

EMG Patterns During Contractions

Electrical potentials sweeping over the muscle membrane during sustained contractions provide important information on performance of motor units. Existing literature presents basically three schools of thought concerning EMG patterns during muscular contractions. These beliefs call for 1) an increase, 2) a decrease, and 3) no change in electrical potentials with time.

Increase in Activity

Increases in electrical activity may be a function of increased frequency of impulse, amplitude of spike, or both. Loofbourrow (55) supports the basic premise that an increase in EMG amplitude is associated with an increase in force, the change being due to the

recruitment of additional units. Much of the research discussed in this subsection could be used in support of Loofbourrow. Although some studies did not utilize a force increase, an increase in effort was required to maintain the prescribed tension. The following studies use primarily surface electrodes for global activity and low force contractions.

Eason (25) tested hand grip during sustained contractions of 25%, 50% and 75% maximum. Surface electrodes revealed a progressive increase in activity with time. He suggests that the results indicate the progressive recruitment of new units to compensate for the declining force of others. Subjects in his experiment promote this assumption by stating that they used progressively more effort in order to maintain the prescribed force.

Other physiologists (23, 29, 49, 72), report similar results. Scherrer and Bourguignon (72) used low intensity isometric and dynamic contractions (near 25%) and found that the electrical activity increased in logarithmic fashion. However, they did find interestingly different results from intense work.

Edwards and Lippold (29) used surface electrodes over the soleus muscle during sustained 25% maximum effort. They discovered a slight falling off of activity during the first minute and then the increase to well over the starting level.

Kuroda, Klissouras and Milsum (49) used 25% and 50%

contractions maintained until complete fatigue. Surface electrodes detected an exponential increase in activity.

deVries (22) explains this phenomenon, as does Eason, by single unit fatigue. As active motor units fatigue, they contribute less force. In order to maintain the same tension, additional units must be activated, thus increasing general EMG response.

Karpovich and Sinning (45) found support for this theory. With the gradual increase in total EMG activity, they also found that individual motor units (needle electrode) diminished and became more synchronous. The decrease was in amplitude and was considered to be due to failure of some individual muscle fibers to respond. Their experiments were conducted using sustained submaximal effort. Synchronization along with addition of new active units produced the global increase.

Biceps brachii muscles were studied via surface terminals in Zuniga and Simons' experiments (82). As tension was increased a progressive non-linear increase in amplitude appeared. They attributed this charge primarily to greater synchronization of motor units which began at about 20% maximum effort. Additionally, some of the effect comes from the larger potentials of higher threshold units.

Kogi and Hakamada (48) studied large human limb muscles. Electrical activity was recorded by surface electrodes before and

during fatigue from sustained contractions. "Subjective local fatigue sensations" were apparent 18 minutes into the test, accompanied by a rise in amplitude and a lowering of frequency. After 30 minutes and a great degree of local muscle pain, the "impulse waves" became considerably larger and slowed to 10-15 per second from the original 50-60. Again, this change was attributed to greater synchronization and slowing of individual motor units discharges. Figure 3 illustrates their findings by plotting comparison change in amplitude per frequency at minute intervals. The greatest increase in amplitude took place at lower frequencies and was attributed to synchronization and lowering discharge rates.

Decrease in Activity

Considerable research supports the concept of declining electrical activity during sustained contractions. These results stem primarily from experimentation at or near maximum effort and generally concern specific motor units.

Scherrer and Bourguignon (72) conducted experiments using intense dynamic work to the point of fatigue and found different results from that at low intensity. During intense effort they observed an initial increase in EMG activity in relation to work done, and then a decrease in activity with unchanged mechanical work.

Seyffarth (75) also found a decrease in EMG activity with needle

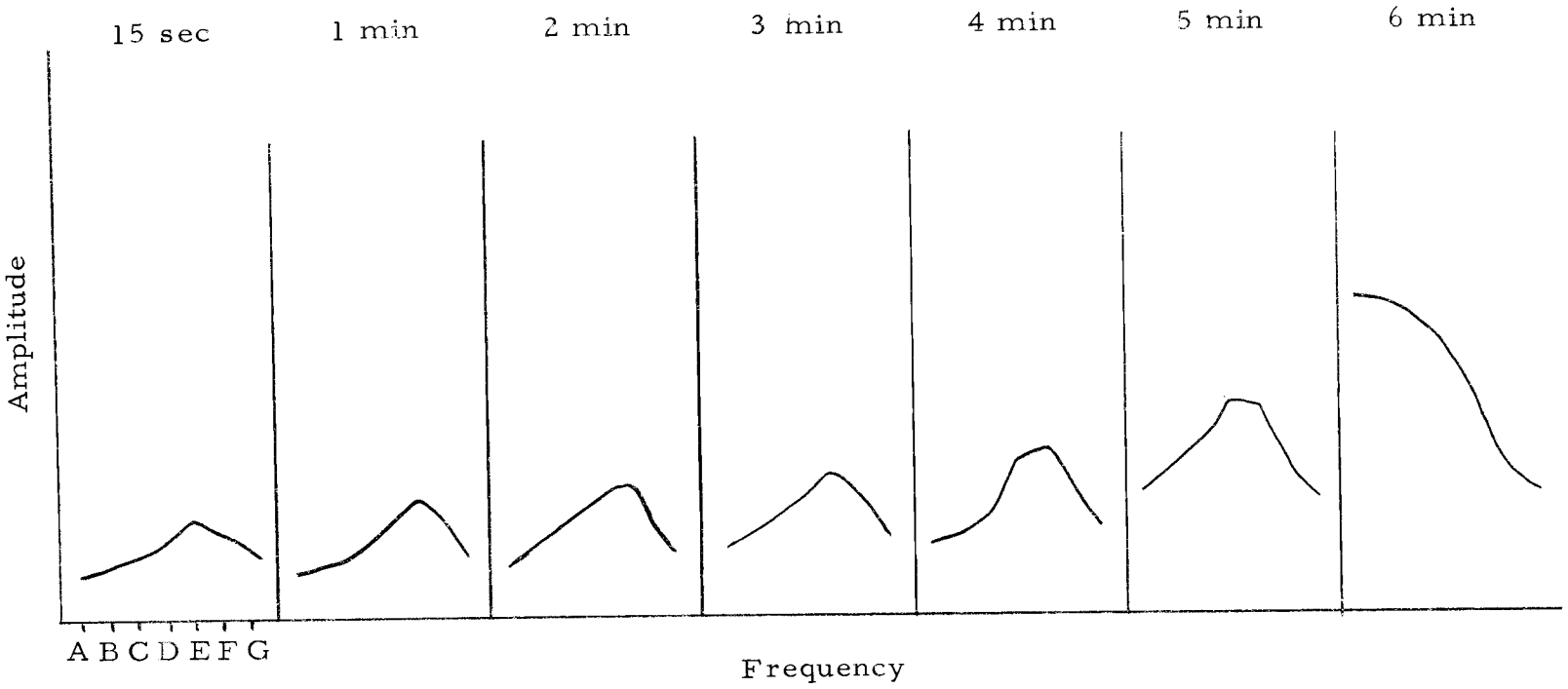

Figure 3. "Temporal change of frequency spectra of m. biceps brachii" with 6 kg load. Abscissae show frequency ranges as follows: A 8-13, B 13-210, C 20-30, D 30-40, E 40-60, F 60-80, G 80-120 impulses per second. Ordinates plot "relative integrated amplitudes. . . " (48).

electrodes during fatigue tests at maximum effort. Units reacted as if there was a gradual voluntary relaxation of the muscle. Frequency of individual units slowly decreased and then units gradually dropped out as strength fell off.

Lindsley (53) likewise found a drop in single unit amplitude during strong contractions that reached fatigue rapidly. These results were opposed to results at lower effort. He did not find an appreciable change in motor unit frequency, however.

Karpovich and Sinning (45) report a decrease in individual motor unit amplitude during submaximal effort. They attributed the decrease to failure of some muscle fibers to respond. Additionally, greater synchrony of motor units was associated with the amplitude drop.

Fatigue experiments were conducted on the tibialis anticus muscle in anesthetized cats (55). High intensity artifical stimulation was used at 38 impulses per second. The mechanical force reduction was accompanied by a decrease in EMG amplitude using needle electrodes. Loofbourrow (55) attributed this reduction to the failure of a number of muscle fibers to respond.

According to Gellhorn (31, 32), if muscle tension is maintained long enough to develop fatigue, there will be a progressive decrease in individual motor unit frequency and in the number of active units. Bronk (12) contends that the frequency parameter is the most likely

to decrease and is due to the slower contractions of muscle fibers with the onset of fatigue.

No Change in Activity

Evidence also supports the contention that electrical activity will not change through a constant voluntary contraction. These experiments utilized maximum and low effort contractions.

Experiments using needle electrodes revealed no change in amplitude or frequency of responding units for 30 continuous minutes. These results were associated with the tibialis anticus muscle during weak contractions which did not cause fatigue. This constant EMG activity is in contrast with results from the same study using strong contractions. During the low effort contractions active units were firing at the same frequency but for a much longer time than in greater effort contractions. According to Lindsley (53) this discrepancy needs further study.

Merton (58) found that amplitude remained unchanged even during extreme fatigue. His tests were conducted with artificial nerve stimulation before, during and after a maximum voluntary effort that extended through fatigue.

Low frequency twitch-producing stimulation resulted in a 50% to 75% strength reduction with no change in electrical activity as detected by needle electrodes (55). Results indicated that no change

occurs in the number of active units but that the ability of the units to contract does change.

Studies (75) of weak contractions on human subjects (needle electrodes) showed a "fairly constant frequency" and "fairly constant" number of units associated with constant tension.

Kuroda, Klissouras and Milsum (49) used surface electrodes and found that during maximum contractions the increase in EMG was not so obvious as at lower (25%-50%) levels, but "showed nearly a straight line. . . . "

Summary of EMG Patterns

Consideration of previously cited literature produces no firm conclusions regarding electrical activity during a sustained contraction. Evidence does suggest, however, a difference in motor unit utilization between high and low force contractions.

Electrical activity may tend to increase during maintained low effort, possibly due to progressive recruitment. Activation of new units may be required to compensate for possible lowering of fiber tension or total failure of some fibers to respond. Contraction of maximal effort may activate all motor units and may preclude further increase in amplitude. However, synchronization of unit impulses has demonstrated an increase in amplitude without recruitment.

Another interesting generalization can be made about low versus

high tension phenomenon. Experimentation in support of increasing electrical activity utilized surface electrodes. Recordings for global response are interpreted primarily from amplitude. Changing amplitude indicates motor unit recruitment but not individual motor unit fluctuations. Increased electrical activity is revealed by this global picture. Experimental support for decrease in electrical potentials, conversely, involved needle electrodes for minute area pickup. Needle recordings generally showed decreasing amplitude and in several cases, a decrease in motor unit frequency.

Evidence supporting no change in EMG makes it difficult to accept the previous points. These results were produced from surface and needle electrodes during weak and strong contractions. Difference in type of muscle fiber studied may be one explanation for this discrepancy. Review of Muscle Fiber Categories will pursue this aspect further.

Lindsley's (53) discovery of motor unit endurance time may be significant. He noted that units operating at low effort continued for a much greater time than at greater effort, even through frequency of stimulation was the same. Further reference to this matter is made in the review of muscle fatigue.

Motor Unit Rotation

Endurance quality of active motor units is of concern when voluntary isometric contractions are maintained. Existing research indicates that initially active units continue to perform indefinitely.

Evidence also supports the contention that motor units rotate their responsibility for maintaining a constant force. This section discusses both theories regarding rotation of motor units and uses experimental evidence as support.

Evidence Against Rotation

Scientific experimentation has revealed that "alternation of motor units, rotational activity or haphazard and irregular changes in unit activity do not occur in sustained or smoothly changing effort" (34). Researchers have discovered that single motor units remain active for periods up to 30 minutes with no evidence of dropping out (34, 53, 77).

"Constant threshold" relationships exist for a particular movement according to Gilson and Mills (34). This relationship indicates that units become active at very specific degrees of contraction. Threshold relationships may, however, change with a slight change in body position. Lower threshold units remain active as higher threshold units are fired with increased tension. All responding units continue to function as tension is maintained at a constant level. Low threshold units "repeatedly become active before other units" and continue through an entire series of contractions.

Seyffarth (75) found similar results during extensive studies of individual motor units through slow voluntary increase and decrease in biceps tension. Units become activated as their threshold is reached and rapidly increase in frequency to that near already active

units. As strength declines the last units to enter are the first to become deactivated. Seyffarth also found this order to remain constant through a series of contractions. Figure 4 provides a visual depiction of this behavior with three motor units.

Theory for control of motor units is explained by Seyffarth as a center of activation (Fig. 5). Expansion of the center triggers more units for increases in tension. Conversely, contraction of the center eliminates units for lowering the force of a contraction. Changes in the number of active units are "always closely connected" with changes in frequency. Through this mechanism low threshold units always remain active until the contraction drops below that threshold level.

Changes in movement pattern shift the center of activation to a different position. Center shifting is dramatized by Seyffarth's discovery that some units of the biceps appeared during very slight flexion effort with supination but only during very strong contractions with pronation. Experimentation which supports motor unit rotation is possibly explained by this shift in center position.

Smith (77) studied responses of individual motor units of the triceps and biceps during minimal and submaximal contraction. She attempted to view units continually even if a change in strength was necessary. Results prompted the conclusion that substitution does not take place but addition of other units may occur. Individual units

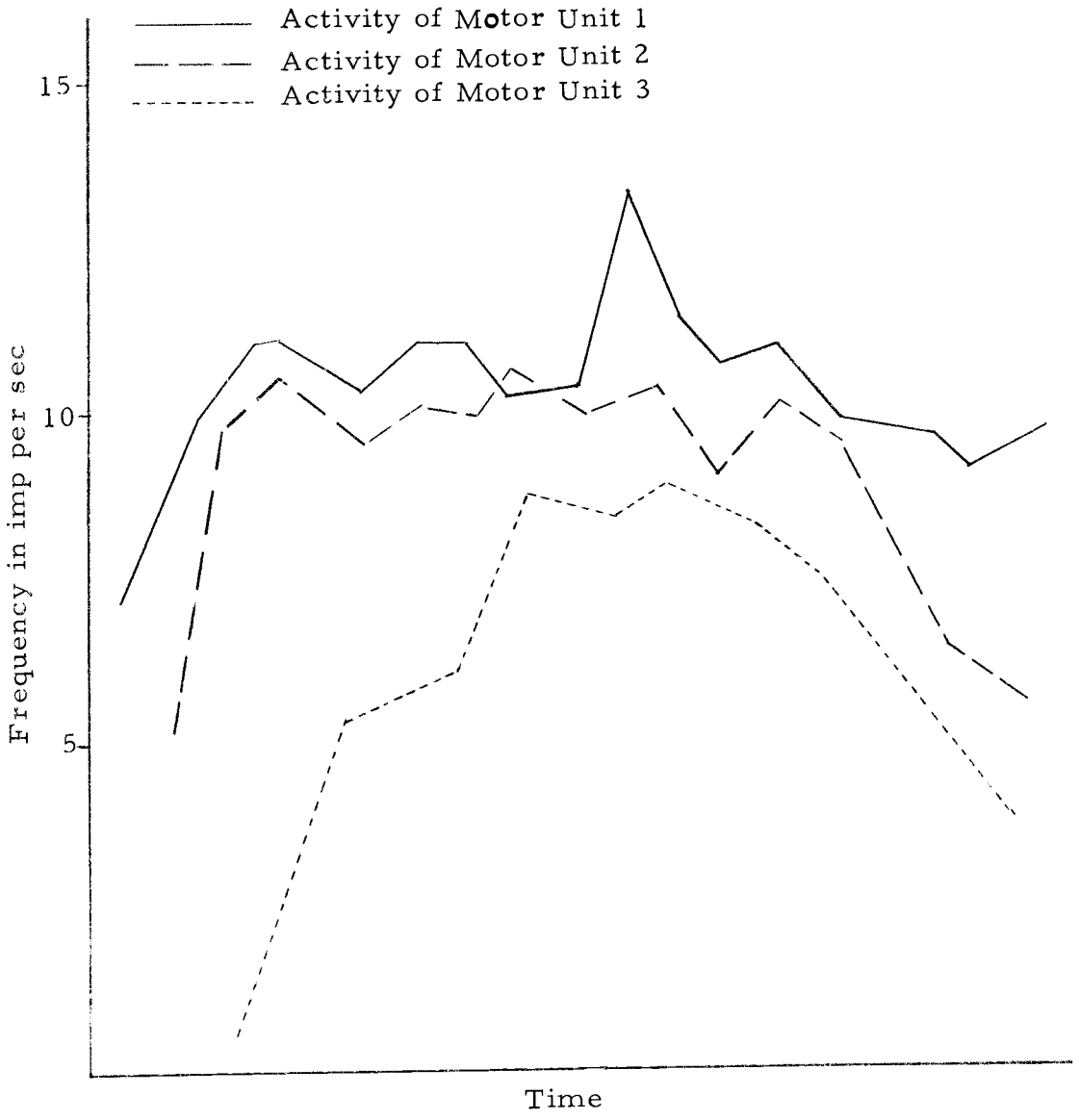

Figure 4. Activity of three motor units in the biceps during a slowly increasing and decreasing contraction (75).

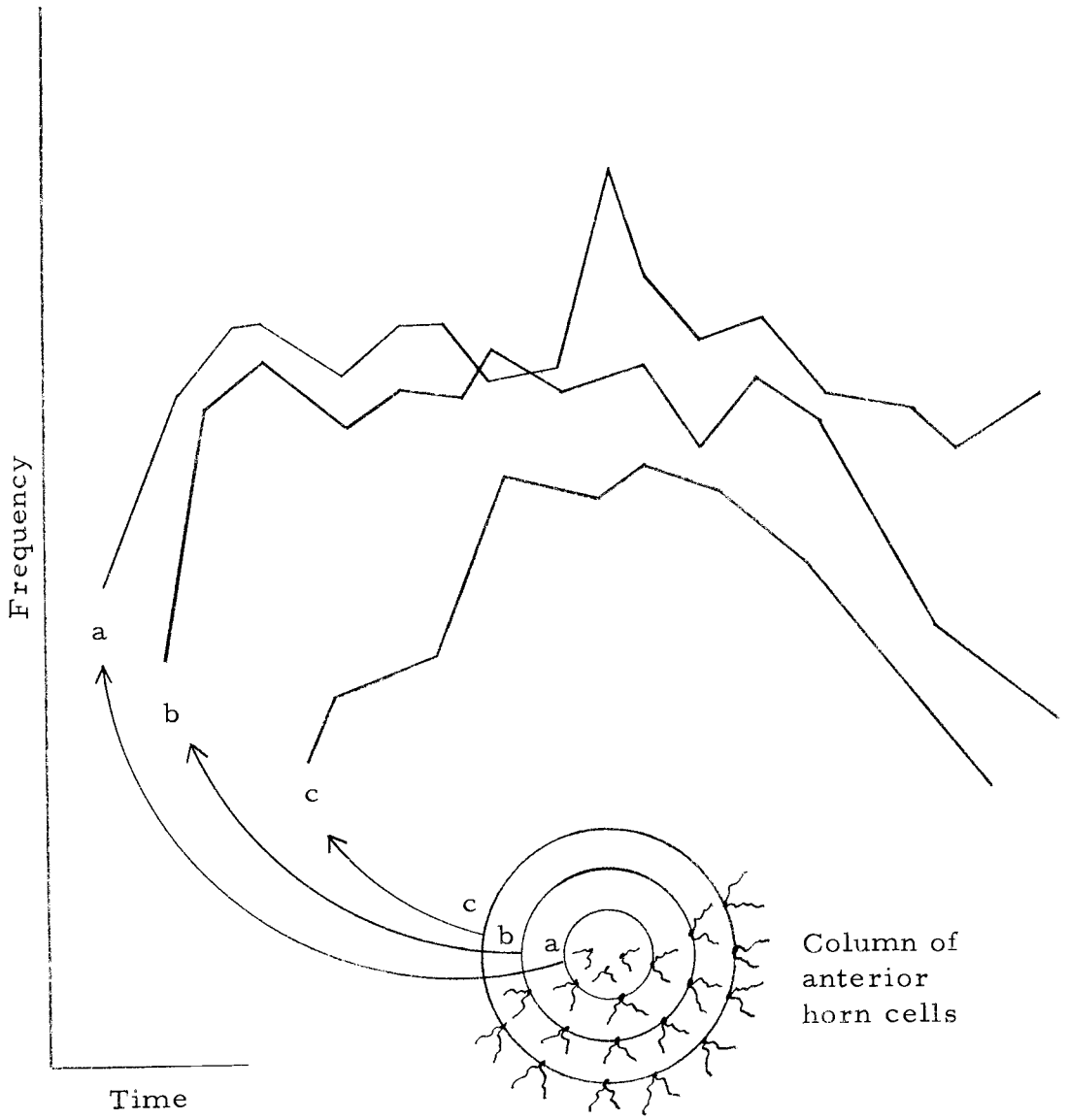

Figure 5. Illustration of motor unit behavior as explained by expanding and contracting center of activation in the column of anterior horn cells. As the center expands, new units are activated (75).

were found to last for 18, 21, 22 and 30 minutes with a few (2-4) momentary stops. These quiescent periods were near one second and much too short to be considered recovery periods. Smith believes that the slow rates of discharge explains the absence of fatigue in motor units.

According to Buchthal (13), Reid (68), and Lindsley (53), there is no evidence of rotational activity during weak to moderate contractions. Lindsley's (53) study showed single units continuing from 15 to 30 minutes with frequency and amplitude unchanged. Data were collected on the tibialis anticus during weak effort not causing fatigue.

Preceding evidence develops a substantial case against alternate utilization of motor units. According to this evidence, there is an exact point at which units become active. Motor units then remain active as long as that threshold is met or exceeded by cerebral cortex demands. Endurance characteristics of these units appear to be adequate for the time frame considered (up to 30 minutes) and at normal physiological frequencies.

Evidence Supporting Rotation

Considerable evidence is available to suggest rotational use of individual motor units in the maintenance of a required force (3, 13, 30, 36, 45, 56, 73). Evidence reporting an increase in global EMG will also support the rotation theory. Additionally, reports of

declining EMG amplitude of individual units should also favor rotation.

Some muscle fibers of an active motor unit become "apparently unable to respond" as the unit continues to fire "and more units are activated to exert the same amount of force" (45). Replacement is indicated by electromyograms showing an increase in global activity while individual unit amplitude decreases, as reported earlier in this chapter. Decreased amplitude suggests deactivation of certain muscle fibers.

Mathews, Stacy and Hoover (56) report that "motor units may become quiescent and another unit in another location will become active" during continual contractions. Some units are active at any one time while some are resting. Such "alternation of responsibility" between many units in the muscle produces a smooth force.

"Bursts" of activity with silent periods of up to two seconds have been found in single motor units during strong contraction of leg muscles (64). Rotational activity is suggested by these findings but only during strong contraction. Buchthal (13) also reports that certain units show a series of active and inactive periods during maximum effort, but not with weak or moderate effort.

Stimulation of the cortex by afferent impulses has been a point of consideration for Adrian (3). Afferent stimulation creates certain "regions of high and low excitability--due to previous activity which would direct the wave along particular channels. . . ." Directing

the impulse waves over different channels would rotate the responsibility between units. In essence, afferent stimulation would move the center of activation described by Seyffarth (75) in a manner similar to changing movement slightly.

During tetanic stimulation of the soleus muscle, Granit (36) found that most motor units oscillated between contraction and relaxation. He contends that when an "electromyogram fails to show signs of recruitment--as it occasionally does--it is always possible to argue that it has been selective and has failed to record from distant muscle fibers."

Forbes (30) describes a theory to explain "alternate periods of activity and rest" in "muscle groups" during sustained reflex contractions. Muscle fibers in a shortened state send "to a limited number of motor neurons the requisite proprioceptive impulses to establish reflex contraction." Motor units then contracted may be different from those first active. Synaptic fatigue (possibly at junction of afferent fibers with premotor neurons) would release "these muscle fibers" prior to their reaching fatigue. Contraction of this second group would cause the reflex contraction of a third group. Forbes does state that existing evidence would not warrant acceptance of such a theory, but it does offer a possible explanation for reflex rotation.

Summary of Rotation

Questions concerning motor unit rotation are obviously unsettled. Contradictory evidence may indicate that the neuromuscular system, in fact operates either way. Specific muscle fibers used and/or specific condition of the contraction may determine if rotation will occur.

Apparently, a given movement activates specific units, as indicated by Seyffarth's investigation. On this premise, very slight changes in position may produce the results interpreted as rotation.

Smith's evidence was evaluated to endorse an absence of rotation. However, her attempt to view units even if a strength change was necessary appears to detract from that assumption. Rotation may have been averted by the increase in effort.

Further investigation seems appropriate to completely evaluate the use of motor units in a sustained contraction.

Fatigue Sites

Empirical evidence suggests that a strong contraction cannot be maintained for an extended period of time. Reduction in the ability to manifest tension is regarded as muscle fatigue. Actual mechanism producing this decrement in tension is subject to controversy.

Sustained low force contractions activate a low percentage of motor units in that muscle. Previously cited reports are inconclusive

regarding the length of time those units remain active. Evidence indicating that motor units drop out or rotate generates concern about their controlling mechanism. Research suggesting that motor units continue to function indefinitely focuses consideration on why units fatigue during strong contractions but not during weak contractions.

Morehouse (63) defines muscle fatigue as "an impairment of the muscles' ability to respond to stimuli." Mathews, Stacy and Hoover (56) suggest four factors which accumulate and limit the contracting ability of a muscle. These factors include:

1. Accumulation of toxic waste products.
2 Exhaustion of needed raw materials.
3. Cumulative physical damage of muscle tissue.
4. Reduction in ability of the nervous system to supply impulses to muscle.

Isolated muscles which are artificially stimulated once per second fatigue after a "few dozen twitches" (56). Fatigue is manifested by declining strength, incomplete relaxation, longer twitch duration and eventually an inability to respond.

Positions of researchers vary regarding the actual cause of fatigue. Specific sites of fatigue which may affect the force of a muscular contraction include the motor cortex and central nervous system, nerve synapse, nerve fiber, neuromuscular junction, excitation contraction coupling, and the contraction mechanism itself (63).

Motor Cortex

Neuronal signals which initiate muscle contractions originate at the first possible fatigue site, the motor cortex (38, 45). Increased or decreased excitation in the cortex produces greater or lesser contraction force respectively. Graduated voluntary effort applied to any contraction then, will specify the amount of excitation and the contraction force.

According to Ikai, Yabe and Ischii (41), central control is a more pronounced factor in limiting force than is the actual muscle. They found that artificially stimulated muscle contractions are nearly 30% greater than maximum voluntary contractions. Fatigue tests indicated that strength decreased more with voluntary effort than with artificially stimulated contractions. Muscular force declined to 40% of original during voluntary and to 66% during direct electrical stimulation. Results precipitated the conclusion that the primary limiting factor in a voluntary contraction is in the motor cortex. Strength limits imposed are, therefore, a function of the voluntary effort which the subject is willing to exert. Peripheral aspects of fatigue were recognized but considered of secondary importance.

Reports indicate that a relationship exists between fatiguing muscle and motor cortex changes (63). Electroencephalograms have shown a decrease in activity of the cortex with the onset of fatigue.

This discovery is a very strong indication that the strength decrement in fatigue is a function of the central nervous system.

Seyffarth's (75) theory for control of motor units is also related to the central nervous system. He found that increases or decreases in the number of active units were very closely related to increases or decreases in frequency. Fatigue studies showed that active units decreased "in frequency and in number in the same manner as during voluntary relaxation of a contraction." Units which remained active were the same units that began first and had the highest frequency.

Seyffarth theorized that control of motor units rests with an expanding and contracting center of activation in the central nervous system. Declining force from voluntary relaxation or fatigue occurs as the center contracts to lower frequency and deletes units. Figure 5 graphically illustrates the operation of this center.

Physiologists (32, 45, 48, 82) have reported that motor units begin to fire synchronously with fatigue. Such reports may also implicate the central nervous system in fatigue since motorneuron impulses originate at the cord. Effects of afferent impulses, however, must also be recognized and are considered in the final section of this chapter. In any case, actual synchronization is implemented in the central nervous system whether or not it originates there.

Astrand and Rodahl (4) report that the subject's motivation and willingness to perform with discomfort are important factors in

maintaining a contraction. These factors would support the motor cortex theory in limiting contractions during fatigue. Previously presented results of Ikai, Yabe and Ischii also appear to be an example of central fatigue.

Nerve Synapse

Neuronal synapses to the motor nerves are next in order as possible fatigue sites. Basic understanding of synaptic transmission is necessary to evaluate this site.

Anatomically, the synapse consists of a presynaptic membrane at the distal end of axons, synaptic cleft or space between neurons, and the post synaptic membrane at the proximal end of motor neurons (81). A transmitter substance is released from the presynaptic membrane as an impulse reaches the synapse. This substance diffuses across the cleft to contact the post synaptic membrane and causes depolarization. Depolarization is very short-lived but does initiate the action potential over the motor neurons.

Contrary to the behavior of the neuromuscular junction, a nerve synapse rarely transmits impulses one-for-one to the next neuron (81). A series of impulses or a single impulse at many presynaptic membranes is required.

Nerve synapse fatigue occurs by exhaustion of the transmitter substance at the presynaptic terminal (39). Enough substance is

stored for only 10,000 normal transmissions, an amount which can easily be depleted in a few seconds. According to Forbes (30) endurance time of the synapse depends upon the fiber size. Small nerve branches will deplete their supply more quickly than will large nerves.

Mathews, Stacy and Hoover (56) report that with fatigue from physical exertion, muscles are still able to contract, nerve fibers are able to fire, and neuromuscular junctions are able to operate. They conclude the limiting factor to be fatigue of the synapse.

Forbes (30) drew a similar conclusion from flexion reflex tests on cats. With failure of the reflex response due to fatigue, direct stimulation of the motor nerve produced a normal contraction. Fatigue in a reflex arc occurs at the synapse which excites the motor nerve.

Nerve Fiber

Motor nerve fibers are the next link in the impulse trace. These fibers carry impulses from the anterior horn of the spinal column to the muscle fibers which they innervate.

Sufficient depolarization of the post synaptic membrane triggers the action potential. The myelin sheath of the axon allows extremely rapid transmission (50 meters/second) of action potentials to nerve endings at the neuromuscular junction (38).

Physiologists generally accept the concept that nerve fibers are

"indefatigable" (23, 30, 38). Forbes (30) found that a nerve stimulated at a rate of 40-50 times per second will continue to function for hours. For the purpose of this investigation, the nerve fiber is therefore discounted as a possible fatigue site.

Neuromuscular Junction

Impulses reaching the distal portion of the motor nerve fibers must be transmitted across a gap to the muscle fiber. Neuromuscular junctions are responsible for this transmission (7, 33, 81). Fatigue of this junction is considered next.

Neuromuscular junctions are similar to the synapse in operation. Transmitter substance is released, diffuses across the gap, and depolarizes the muscle membrane. Nerve endings at skeletal muscles are cholinergic (7), indicating the transmittal substance is acetylcholine. Production of acetycholine appears to occur in the nerve ending itself.

Nerve-muscle preparations show that the neuromuscular junction is more susceptible to fatigue than the muscle fiber (30, 56). When a motor nerve is stimulated through fatigue until no contraction response occurs, direct stimulation of the muscle causes a contraction. This phenomenon indicates failure of the neuromuscular junction (56). Forbes (30) believes however, that actual fatigue is a combination of synaptic and neuromuscular junction failure.

Support for neuromuscular junction failure is found in electromyographic experimentation (45). Decreased amplitude of individual motor units is considered due to failure of some muscle fibers to respond. Specifically, decreased amplitude indicates a failure of the nerve impulse to excite the muscle membrane of some fibers.

Loofbourrow's (55) experiments on anesthetized cats indicate failure of the neuromuscular junction at high frequencies. His rates of nearly 38 impulses per second are in line with actual physiological conditions (2, 3, 12, 39, 45, 53) and therefore, results are considered applicable. Tetanizing frequencies were found to produce a drop in EMG amplitude. Low frequency stimulation showed an absence of neuromuscular junction fatigue. His results could implicate only fatigue sites distal to the motor nerve, however, since artificial stimulation of the nerve was used.

According to Guyton (39) the neuromuscular junction has its transmission limits. Impulse frequencies in excess of 150 per second for many minutes can diminish the acetylcholine released at each impulse to a point where impulses will not be transmitted. Under actual physiological conditions, however, frequency rarely reaches this level and fatigue of the neuromuscular junction almost never occurs. Morehouse and Miller (63) report that a decrease in local production of acetylcholine may partly explain fatigue in prolonged contractions.

Excitation Contraction Coupling

Action potentials travel over the sarcolemma at 1 or 2 meters per second and must somehow initiate a contraction of that muscle fiber (33). Coupling of the membrane impulse to the contraction is still another possibility for the site of fatigue.

Actual mechanism of this coupling is not known for certain, but calcium liberation is considered a likely theory (33). A minute tubular system, termed the T-system, extends inward from the sarcolemma with two lateral extensions of the sarcoplasmic reticulum. Although the membrane action potential does not appear to extend into the tubule system, it does initiate a release of calcium. Free calcium in some way triggers the contraction. Upon repolarization of the membrane, calcium is bound again to the membrane and reticulum and the contraction ends.

Exact excitation contraction coupling mechanism is uncertain and therefore speculation is limited concerning fatigue in this area. DeVries (23) and Ebsterein and Sandow (26), do however report coupling as a site of fatigue in addition to the contraction mechanism. Furthermore, coupling may be a factor in cases where EMG activity leads the researcher to conclude the contraction mechanism has fatigued. Failure of either mechanism would produce the same decrement of strength with unchanging motor unit amplitude.

Muscle Contraction Mechanism

Physiologists and researchers have identified the contraction mechanism as the cause of fatigue (23, 25, 26, 29, 38, 55, 59). Fatigue is attributed to the breakdown or slowing of metabolic activity in the cells and/or waste accumulation.

The exact mechanism of the contraction is again unknown. Popular theory involves the breakdown of ATP (7, 33). Presence of ATP in the cellular fluid disassociates the myosin and actin filaments. Appearance of free calcium from the excitation contraction coupling apparently triggers a mechanism which enzymically breaks down ATP to ADP. Energy is released during this breakdown and the actin and myosin filaments are drawn together causing a contraction. Termination of ATP breakdown dissolves the filament link and the muscle relaxes.

Energy sources are an important consideration in maintaining the contraction mechanism. ATP itself is the immediate source of energy (7). Regeneration of ATP from ADP rests immediately with the high energy phosphate bond transfer from creatine phosphate with the influence of the enzyme creatine kinase. Further ATP production comes from the breakdown of carbohydrates either with or without the presence of oxygen. Greater quantities of energy are produced when oxygen is available.

Astrand and Rodahl (4) emphasize the importance of metabolic function in relation to fatigue. Ability to maintain a contraction is dependent upon the blood supply in that muscle. Muscles are able to work for short periods without blood flow due to existing ATP, creatine phosphate and metabolism utilizing myoglobin-bound oxygen. However, during a prolonged contraction an adequate supply of oxygen must be delivered and metabolites and heat must be removed. Specific factors which limit the contraction are not known but possibilities are "accumulation of lactic acid, of H^+ and/or heat."

Appropriate blood flow allows operation of necessary cell functions and muscular work may continue aerobically for extended periods of time (4). Blood flow is likely to be restricted during the contraction however. Intramuscular pressure may be as high as several hundred mm Hg during a maximum contraction. This level is higher than arterial blood pressure of 120 mm Hg at rest and 200 mm Hg in exercise, so blood flow may be partially or even fully occluded. Lind and McNicol (52) discovered that a contraction in excess of 70% maximum would occlude blood flow entirely from the muscle. Due to restriction of blood flow, contraction endurance decreases exponentially with the increases in tension (4, 52).

Merton (58) conducted extensive experiments involving voluntary and artificially stimulated contractions. Artificial nerve stimulation was incorporated with voluntary effort while global and single unit

potentials were monitored. Tension dropped evenly from the start with maximal effort and was not affected by artificial nerve stimulations at any point on the tension curve. Merton concluded that voluntary effort was still contracting the muscle completely and the drop in force was due to failure of "some part of the peripheral apparatus."

Low force sustained voluntary contractions were tested with occasional artificial nerve stimulation. Nerve shocks initially caused individual twitches. Twitch height decreased with the onset of fatigue and eventually disappeared. Subjects made a "progressively greater effort" to maintain the desired force. Twitch disappearance occurred at the same time that maximum voluntary effort was required. Tension dropped off as in the maximal effort curve following disappearance of the twitches.

Throughout these tests, including extreme fatigue, EMG amplitude did not diminish. Amplitude of the artificial stimulus spike likewise remained the same before, during and after fatigue. With cessation of voluntary effort, artificial spike amplitude remained the same throughout muscle recovery. Constant amplitude indicates passage of the impulse across the neuromuscular junction and over the muscle membrane.

Merton concluded that the large decline in contraction force during fatigue takes place "without appreciable impairment of the conduction from nerve to muscle." Decrease in force is due to

failure in the "biochemistry of the contractile process."

Additional evidence of contraction fatigue was found by Merton's experiments with restricted blood flow. Normal fatigue curves show tension dropping to about one-third of the original and then leveling out. Lower tension is maintained for a considerable period of time with some pain. However, tension continues to decline to zero with no appearance of leveling out when blood flow is occluded.

Ordinary leveling of tension is considered due to the lowering of "intramuscular pressure" below systolic blood pressure. Lower pressure allows return of the blood flow, less fatigue, and therefore the ability to maintain the contraction at that level. When circulation is occluded by a cuff, tension continues the drop toward zero since blood flow is not restored. Recovery does not take place without blood flow. Recovery is aerobic.

Evidence from ischemic muscles further indicates to Merton that fatigue is due to contraction mechanism failure. He contends that if fatigue were central, recovery would take place when effort is stopped since there is no circulation impairment for "central structures."

Merton strongly disagrees with the contention that a completely fatigued muscle contracts with artificial stimulation of the nerve or muscle. Artificial stimulation produces contractions only if all units are not used in the voluntary contraction and therefore all units are

not fatigued.

Guyton (39) attributes fatigue to inability of the "contractile and metabolic processes of the muscle fibers" to continue. Nerves still operate, impulses pass over the neuromuscular junction onto the muscle, normal potentials spread over the muscle membrane, but lower force is produced. Occluded blood flow produces severe fatigue very rapidly due to the "obvious loss of nutrient supply."

Kuroda, Klissouras and Milsum (49) also attributed the reduction in muscular force in fatigue to the reduced contractility of the muscle.

Apparent contraction failure was found by Loofbourrow (55) but only at low frequency stimulation. Low frequency stimulation produced a tension drop of 50%-75% with no change in EMG activity. A constant number of units remained active with constant spike amplitudes. Since impulses pass normally over the muscle fibers, decreased strength must be due to the "contractility of fibers" or a change in their ability to contract.

Further evidence suggests that the site of fatigue is dependent upon the frequency of stimulation (21, 44). del Pozo's (21) experiments on various muscles of anesthetized cats indicated neuromuscular junction failure during high frequency stimulation (over 30 per second) and contraction mechanism failure at lower frequencies (below 20 per second). Tension dropped from nerve stimulation

at 60 per second but direct muscle stimulation produced normal responses. Failure of this nature is attributed to junction fatigue or more specifically to a discharge rate of acetylcyoline greater than the production rate.

Continual nerve stimulation below 20 per second produces fatigue where direct muscle stimulation responses become diminished. Fatigue at this frequency was attributed to the contractile mechanism failure. The possibility does exist, however, for the failure to be excitation contraction coupling fatigue.

Furthermore, del Pozo discovered two types of recovery patterns. High frequency stimulation where tension decreased rapidly was associated with quite rapid recovery. Low frequency stimulation where tension declined slowly produced a gradual recovery. del Pozo concluded that recovery from junction fatigue was rapid while recovery from the contraction mechanism was slow.

Physiologists report different EMG patterns between high and low frequency stimulation (49, 53, 72). Different EMG patterns support the theory of alternate fatigue sites due to change in frequency. High frequency junction fatigue prevents transmission of impulses to the muscle membrane and therefore produces a drop in electrical response. Conversely, low frequency impulses cause fatigue of the contraction mechanism while excitation continues to be transmitted to the muscle and electrical activity does not decrease.

Summary of Fatigue

Identification of the actual site of fatigue is obviously unsettled. With the exception of the nerve fiber and excitation contraction coupling, each site has considerable scientific support. Each site has enough supporting evidence to establish it as the failure point when considered singularly. Each fatigue site may be involved at one point in the body under certain conditions. Wide variance in muscles used, intensity of contractions and methods of stimulation may explain the variation in previous results and conclusions.

Several authors referred to the importance of limited blood flow as a fatigue contributor (4, 39, 52, 58). Reference was made primarily to support the theory of contraction mechanism failure. Merton (57) specifically identifies contraction fatigue and rules out "central structures" from his studies with restricted blood flow.

Two other important considerations exist regarding blood flow occlusion. First, occluded blood must have some impact on the production of acetylcholine in the nerve ending. Adequate quantity of acetylcholine is essential for neuromuscular junction transmission and any impairment in its production would affect impulse passage to the muscle. Morehouse and Miller (63) recognized this factor as they partly attributed fatigue to a decrease in "local synthesis of acetylcholine-like substance" in sustained contractions. Secondly,

occluded blood flow has a great effect upon afferent activity. Afferent influence is considered in depth in a later section of this chapter. Suffice to say at this point that afferent impulses inform the central nervous system of muscle condition. "Central structures" then may act upon the blood-starved muscle even though not directly affected by fatigue itself.

Acetylcholine production and afferent factors implicated during ischemia are likely to explain Lindsley's fiber endurance question discussed earlier. Lindsley found that single units operated much longer during low effort than at higher effort even though stimulation frequency was the same in both cases. Previous evidence has established that more intense contractions restrict blood flow. Individual motor units may be hindered by multiple effects of ischemia during high effort contractions. These same units, however, may continue without this hindrance during low effort.

Frequency of stimulation appears to influence the site of fatigue. The primary contention is that failure occurs at the neuromuscular junction during high frequency and at the contraction mechanism during low frequency stimulation. EMG studies discussed in a previous section of this thesis appear to support this conclusion to some extent. Maximum or near maximum effort, represented by high frequency stimulation, produced a decrease in EMG activity. Interpretation of this decrease may suggest failure

of impulses to pass from the nerve to the muscle membrane. Low intensity contractions, represented by low frequency stimulation, produced unchanging or increasing EMG activity. Performance decrement in these cases may be interpreted as contraction failure. Several reports, however, discounted this theory. Merton specifically denounced neuromuscular junction failure based upon his EMG response.

Muscle Fiber Categories

Major differences exist between types of skeletal muscle fibers which require consideration. Two types have been labeled with a variety of names. One has been referred to as red, slow, tonic and heavy work muscle, while the other has been called pale, white, fast, phasic, and light work muscle (47). Muscle fiber types in this study will be referred to as tonic and phasic to indicate their characteristic function. Physiological differences between muscle fibers demand attention when muscle performance is being considered. This section discusses physical, chemical and functional properties of these fibers.

Skeletal muscles will contain a combination of the two fibers. Percentage of each depends upon that muscle's function (37, 47). Muscles used continuously in maintenance of body position, extensor muscles, will have a preponderance of tonic fibers. Those used primarily to change the body position, flexor muscles, will contain

more phasic fibers.

Position of these fibers in the overall muscle appears to be fairly constant (6, 47, 61). Tonic fibers tend to be located in the deep center portion of the muscle belly. Phasic fibers generally surround the tonic fibers.

Tonic muscle fibers are slow-contracting, slender, and reddish in color (45, 46, 47, 61). High myoglobin content in the cells is responsible for the color distinction. These fibers are well suited for oxidative metabolism with their enzyme activity, large surface area, and high quantity of mitochondria and myoglobin (6, 46, 47). Such characteristics create a fiber ideally suited for prolonged aerobic activity. Tonic fibers are innervated by small motoneurons with low thresholds and low frequency range of 10-20 impulses per second (27, 47).

Phasic fibers are large (three to four times the diameter of tonic fibers), pale in color and produce quick powerful contractions (26, 47). These fibers are chemically equipped for anaerobic glycolysis with their enzyme activity, limited mitochondrial content, high glycogen content, high creatine phosphate content, "lack of myoglobin" and small surface area (37, 46, 47). Phasic fibers contain a greater amount of calcium in the sarcoplasmic reticulum which likely effects the speed of contraction. They are innervated by large neurons with higher thresholds, faster conduction velocities, and a

higher frequecy range of 30-60 impulses per second (27, 47). Shorter contraction time requires a higher frequency to produce tetanus. Phasic fibers are suited well for short powerful contractions.

Keul, Doll and Keppler (46) also recognize a third type of fiber. These are referred to as intermediate fibers and appear to be a compromise between the extremes of tonic and phasic fiber characteristics.

Tonic and phasic fiber characteristics apparently are not highly differentiated at birth (16, 46, 47). Muscle fibers develop their specific characteristics from neural influence which may involve trophic or frequency factors. Trophic theory involves the passage of a substance down the nerve axon and over the muscle fiber which results in fiber specialization. Frequency theory proposes that muscle fibers receiving continuous low frequency stimulation develop features of tonic fibers while those receiving occasional high frequency stimulation evolve with phasic fiber characteristics. According to Gutmann (37) the difference in fibers is apparent within three days after birth in animals.

Cross-innervating tonic neurons to phasic muscle fibers and vice versa, tends to transform the characteristics of these fibers (16, 46, 63). Modification includes twitch speed, myoglobin content, enzymatic activity and resistance to fatigue. Transformation is not complete, however, even after two years with cross innervation.

Marked differences exist in the endurance capability of tonic and phasic fibers (26). Isolating single phasic fibers and small bundles of ten or less tonic fibers from frog muscle, Eberstein and Sandow were able to measure changes in tension with time. Slightly suprathreshold impulses at the rate of one per second were used as stimulation. Tension of phasic fibers decreased continuously in two phases. Initially a rapid drop of 60% took place within the first four minutes. Continued decrease in tension was gradual until no response was produced. Recovery of phasic fibers also occurred in two phases. Initial rapid recovery of 30% was observed in 15 seconds, followed by gradual return to full strength requiring one hour.

Tonic fibers produced a much more gradual fatigue curve. Tension decreased nearly 15% in four minutes and 35% after a full hour. Recovery tests showed a return of 15% strength in two minutes and continued gradual return to full strength.

Eberstein and Sandow's experiments and those of del Pozo (21) reveal a much greater endurance capability in tonic fibers. Endurance of phasic fibers is limited by stored glycogen and high energy phosphate reserves. Tonic fibers, on the other hand, may endure due to their capacity for oxidative metabolism (6). Experimentation of endurance time at low level tension utilizes tonic fibers almost exclusively (61).

Evidence appears to establish that individual muscle fibers lose

some degree of tension in sustained contraction. Fatigue of muscle fibers does apparently take place. Their decrement in performance would demand additional recruitment in order to maintain a prescribed force by the total muscle.

However, these experiments isolated only two possible sites, excitation contraction coupling and contraction mechanism, and do not necessarily lead us to conclude that in vivo fatigue occurs in this manner. Other sites in the neuromuscular chain may fail prior to fiber failure, especially with more appropriate stimulation frequencies. Possibility also exists that the site of fatigue may be different with respect to fiber type, since endurance quality of tonic and phasic fibers is substantially different.

Additionally, endurance characteristics actually measured may not be realistic for intact muscles. Physiological condition of in vivo fibers may allow continuation of the contraction well in excess of an excised fiber contraction. Availability of blood supply may also affect tonic and phasic fibers differently and may negate Eberstein and Sandow's results. This possiblity appears plausible in light of energy sources of the two fibers. Tonic fibers may gain markedly with hemoglobin-bound oxygen perpetuating oxidative metabolism. Phasic fibers may be affected very little due to their reliance upon anaerobic glycolysis.

Fiber characteristics may help explain the contradictive

evidence in previous sections. Needle electrodes in contact with aerobic tonic fibers are likely to detect a different activity picture than if in contact with anaerobic phasic fibers. Experimentation using extensor and flexor muscles should reflect this difference since they contain quite different proportions of each fiber type. Similarly, studies of high versus low effort contractions would likely contrast since low effort may only involve tonic fibers while both are involved in high effort.

Restoration of blood flow is used to explain leveling of the fatigue tension curve (58). However, the initial drop in tension may be due to phasic fiber failure and leveling of the curve due to continued work of tonic fibers. Actual physiological conditions may combine blood flow and fiber type factors in determining the tension curve.

Conclusions suggest that the type of fiber being considered is significant. Studies involving endurance must recognize this fact and the experimentor must interpret experimental results accordingly.

Afferent Influence on Contractions

An extensive afferent (sensory) neuronal system acts as a communication network from skeletal muscles to the central nervous system. Afferent neurons may perform either inhibitory or

excitatory functions for muscles at the level of the spinal synapse. Additional impulses are supplied by the system which are interpreted by the brain as fatigue and pain from peripheral areas (55).

Physiologists report that one-third to one-half of the nerve fibers in normal cat muscles are afferent (7). Other muscles may have an even higher percent afferent content. Generally, muscles which perform powerful body movements have a lower ratio of afferent to efferent neurons and muscles used in delicate movements have a higher ratio for more precise control.

The central nervous system exerts some degree of control over the afferent feedback it will receive. Scientific evidence indicates approximately one-third of all efferent neurons entering the muscle innervate intrafusal fibers of the muscle spindles (22). These nerve fibers are referred to as gamma efferents. Intrafusal fibers are a key to feedback of information to the central nervous system and do not function as a force-producing element. Muscle contractions involve excitation of intrafusal and extrafusal fibers.

Annulo spiral endings and flower spray endings in the spindle are sensitive to misalignment or a difference in length between extrafusal and intrafusal fibers (60). Afferent discharge from these endings will cause extrafusal fibers to contract and match the setting of the intrafusal fibers. This mechanism is commonly referred to as the stretch reflex but, according to Merton (60), also regulates

the force of a voluntary contraction. Merton suggests that the muscle spindles are associated with a certain group of muscle fibers and that they fire together in a contraction. Spindles may cause other fibers in their group to contract as previously active fibers weaken. Afferent impulses then, exert some degree of control over extrafusal fibers of that muscle.

Muscle spindles are an important regulator of motor units for body posture (23). The stretch reflex causes antigravity muscles to contract when they are subjected to stretch. Bell, Davidson and Scarborough (7) suggest that rotational use of motor units in maintenance of postural tone would account for the absence of fatigue. Motor unit rotation of this nature is apparently caused by the afferent system since the spindle detects and corrects shortcomings in muscle force. Similar control may exist in maintenance of force during sustained voluntary contractions.

Golgi tendon organs are mentioned in light of their inhibitory effects upon muscular contractions (7, 23). This organ is located at the junction of muscles and tendons and is sensitive to excessive tension, either passive or active. Golgi tendon organs discharge to inhibit the contraction and prevent possible damage to the muscle. Although the organs are generally considered to discharge only with high tension, Granit (35) did find low threshold inhibition. He speculated that this inhibition is due to Golgi tendon organs in muscle

spindle tendons having low stretch thresholds.

Muscle spindles and Golgi tendon organs work together for control of a muscle. Cooperatively they add to and subtract from motor neuron discharge in a self-regulating manner (35).

Other receptors in the muscles are sensitive to chemical and temperature changes (7). Impulse discharges from these receptors are returned to the central nervous system for interpretation and may affect efferent impulses which cause the contraction.

Experimental evidence indicates the importance of afferent influence on muscle performance. Molbech and Johansen (61) emphasized the importance of afferent feedback by chemically blocking gamma efferent impulses to the spindle. Results revealed reduced afferent discharge and decreased muscle endurance. They conjectured that the decreased endurance resulted when the declining afferent activity provided less facilitation of motor cortex impulses. Conversely, they postulated that chemical activation of the spindle would increase afferent activity, facilitate motor impulse transmission and therefore may prolong contraction endurance. Molbech and Johansen also emphasized the importance of afferent activity as a contributor to fatigue or at least to the subject's feeling of fatigue.

Afferent activity is greatly increased when blood flow is occluded (57). Frequency and number of responding afferent fibers increase and appear to be closely associated with metabolic activity

of the muscle. Conditions which cause increased sensory feedback also cause pain sensations. Great increases in afferent discharge may well affect muscle performance through its influence on the central nervous system.

Lowering of efferent impulse frequency in fatigue may be due to a decrease in proprioceptive feedback from the spindles (48). Decreased motoneuron discharge frequency is related to the appearance of local fatigue sensations and inability to maintain desired tension. Such evidence indicates an interrelationship of afferent and efferent activity.

Seyffarth (76) also suggests that a decrease in motor frequency during fatigue may be due to afferent activity. He attributes the decrease to greater inhibitory effects, however. Impulses may affect the central regulation of frequency causing a slowdown with the onset of fatigue. Seyffarth further supported this theory with results of ischemic experiments where increased afferent frequency was related to decreased motoneuron frequency.

According to Gellhorn (32), low frequency stimulation in a muscular contraction is due to inhibition by afferent discharge. Motor unit impulses then are always under the influence of afferent braking action to maintain appropriately low frequencies.

Greater synchrony of central nervous system discharge occurs in fatigue (32). The afferent neuronal system is apparently the only

network allowing muscle-to-central communications and its effect must be recognized as important in causing efferent synchronization.

Further evidence that afferent volleys are interrelated with muscle performance arises from reports of Morehouse and Miller (62). Thresholds of knee and ankle stretch reflexes gradually rise during work periods. Following work sessions, the thresholds lower as recovery from fatigue takes place. Rising thresholds with work suggest a reluctance to use fatigued muscle fibers.

Afferent discharge appears to play a significant role in controlling the frequency and utilization of motor units. Influence appears to be manifested in three ways. First, direct inhibitory effects upon motoneurons exist for maintaining low frequency impulses and preventing damage from excessive stretch. Secondly, direct excitatory effects on motoneurons exist which maintain the required tension during a contraction. Thirdly, afferent pathways transmit impulses to the brain concerning the physical and chemical status of that muscle. Impulses report common sensations of fatigue, heat, and pain. Motor cortex impulses then, may be modified in appreciation of the muscle condition. Reports of decreasing electroencephalogram activity in fatigue are likely an implementation of this factor.

Previously cited experiments utilizing restricted blood flow must have had considerable afferent influence. Great increases in

afferent discharge during ischemia may influence the contraction as much as energy depletion and waste accumulation.

Composite effects of afferent discharge are significant and must be considered in fatigue/endurance investigations.

Summary

This chapter has presented structural and functional aspects of the neuromuscular system and electromyography as an investigative tool. Current knowledge regarding the neuromuscular system is a cumulative result of dedicated and talented scientists through more than 50 years of research. Operation of this complex human system is now understood to a major extent. Conflicting evidence does exist, however, concerning utilization of motor units.

Generalizations concerning striated muscle fatigue and rotation have resulted from studies of specific muscle and under isolated conditions. Review of literature suggests that consideration must be given to a variety of factors in order to properly generalize results. Muscle fiber types possess different functional characteristics and would likely present contrasting results. In vitro performance may not represent in vivo activity due to absence of blood flow and afferent signals. Afferent activity has significant influence over operation of muscle fibers and requires consideration. Blood flow occlusion has been utilized in numerous studies of muscle performance; however,

rarely is its influence recognized on the afferent system or on possible effects on acetylcholine production. Other variables such as high vs. low tension contractions and surface vs. needle electrodes produce different results and require attention. Such variables appear to be reflected by contradictory evidence found in existing literature.

Electrical resistance is another important concern which has not been cited in the literature. Electrolytes conduct current more readily at higher temperatures than at lower temperatures (74). Sustained contractions produce heat in body tissues due to increased metabolism and therefore, will decrease electrical resistance. This phenomenon suggests that EMG amplitude would increase as transmitting fluids increase in temperature. Another factor, which is unpredictable, concerns leaking of positive or negative ions through the electrode puncture. Alteration of fluid ionic composition would affect recorded amplitude either positively or negatively.

Review of literature suggests a need for continued research in the area of motor unit performance. Appropriate attention however, must be directed toward implications of the previous factors.

CHAPTER III

METHODS AND PROCEDURES

Experimentation was conducted under medical supervision at the Corvallis Clinic, Corvallis, Oregon. An electromyograph utilizing implanted needle electrodes provided recordings of electrical activity from motor units in the right biceps brachii muscle. Information recorded by the EMG was used to establish a workable procedure and appropriate equipment operation for study of individual motor units. Procedures included designing and building a special apparatus chair. Electromyogram evaluation and interpretation technique was developed as part of the procedural model.

Subjects

Eight caucasian male college students served as subjects in this investigation. Subjects were selected based on their availability, willingness, and involvement in some form of physical fitness. A thorough briefing of testing procedures was presented to each subject prior to his volunteering. Numerous potential subjects declined involvement after becoming aware of needle implantation. Self-elimination process was considered favorable since strong-willed and determined individuals were desired. Such personality characteristics were regarded as prerequisites since precise muscular effort

was necessary under somewhat discomforting conditions. Subjects' ages ranged from 21 to 30.

Three of the seven subjects were actively involved in competitive varsity athletics while the remaining five were participants in other physical activities. All subjects were in good physical condition.

Equipment

Equipment utilized in this investigation included two basic instruments, the electromyograph and specially designed apparatus chair. Electromyograph system was made available by Dr. George Knox, neurologist at the Corvallis Clinic. Apparatus chair was designed and built by the investigator specifically for providing a stable platform for the subjects' right shoulder and arm.

Apparatus Chair

Apparatus chair shown in Figure 6 consisted of a standard desk-type school chair with the addition of a specially designed variable weight pivot arm platform. The chair and platform allowed a comfortable resting posture for prolonged testing periods and stabilized the right arm and shoulder at a specific position. Platform-to-chair attachment was accomplished by a railing system with locking wing nut providing suitable adjustment for subject size

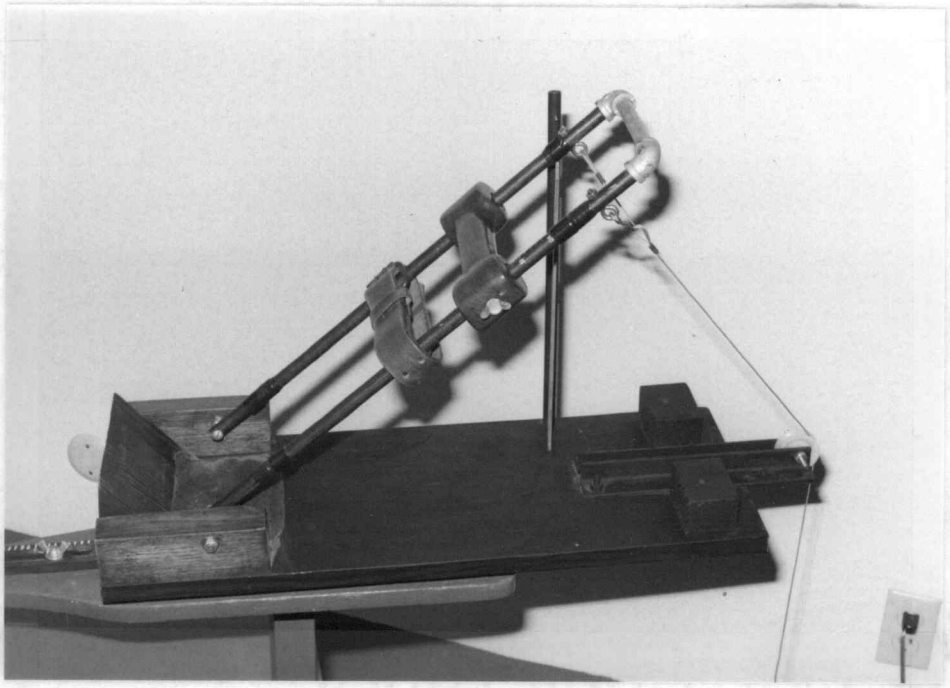

Figure 6. Apparatus chair consisting of pivot arm platform attached to desk-type school chair.

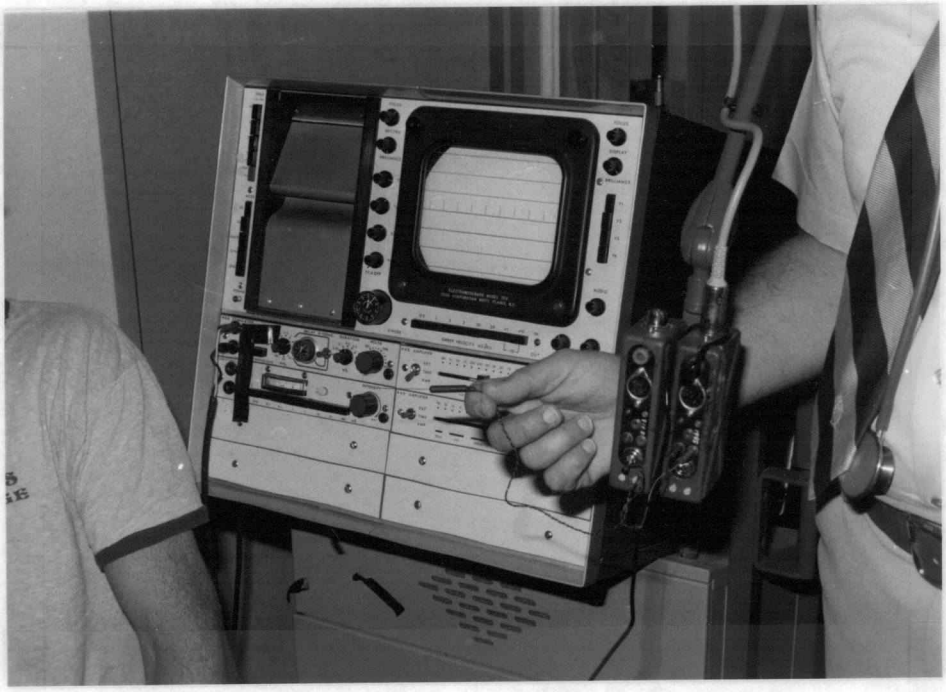

Figure 7. Electromyogram system utilized during the investigation.

variation.

Twenty-four inch by one-by-twelve fir board served as the platform base. Attached to one end of the board was a cable pulley and to the other end an elbow block and pad, and pivot arm supports. One-half inch pipe rectangular frame with adjustable hand grip served as the pivot arm. Supinated wrist position was maintained throughout testing sessions by the carved wood hand grip.

Weights were suspended on a cable connected to the end of the pivot arm and draped over the pulley. Suspended weights allowed constant force throughout the contraction. Wooden dowel marker was used to indicate the appropriate pivot arm position for desired 90 degree elbow flexion.

Two leather belts were added during the course of the experimentation for added shoulder and arm stability. One belt secured the shoulder position to the chair. The other belt was affixed midway on the pivot arm to prevent wrist flexion or extension.

Electromyograph

TE-4 Electromyograph System built by the Teca Corporation provided data on muscle potentials. This system, shown in Figure 7, was mounted on a "mobile console base" and had a "universally-adjustable patient electrode plugbox arm." A Cathode Ray Tube display, audio monitor loudspeaker and high speed direct recorder

presented electrical potential information. Upward deflections on the display tube and recorder indicated a positive change on inner electrode in relation to outer electrode. Electronic Time Ruler was printed below each potential trace as a time reference.

Primary instrument for evaluation was the direct recorder printout. Potential traces were recorded on 100 foot rolls of 100 mm Kodak Linagraph Direct Print paper type 1895. Patterns were recorded exactly as displayed on the cathode ray tube screen with no distortion due to the "inertialess" system. Direct print paper required no darkroom or chemical photographic processing. Photodevelopment occurred with exposure to fluorescent light for approximately four seconds. Trace recordings were printed continuously or selectively in strips by depressing a foot pedal.

Three electrical connections from the subject to the EMG were required. Two input terminal connections provided information to the EMG amplifier. The third connection was a metallic plate ground electrode.

CT37P and CF25P general purpose coaxial tapered needle electrodes, of 37 mm and 25 mm in length, were utilized for signal detection. The CF25P electrode was used exclusively following the first testing session. Coaxial needles consisted of a stainless steel cannula or outer tube conductor containing an insulated nickel-chrome alloy inner conductor. Both pickup electrodes are therefore housed in a

single structure. Contact with the muscle by the inner electrode occurred at the tip only and isolated small pickup areas. Needle electrodes were cleansed between sessions by autoclaving with temperatures of 250° to 260°F under 15 to 20 pounds pressure for 20 minutes.

Procedures

Subjects were seated in the apparatus chair with right elbow resting on the elbow pad as illustrated in Figure 8. The platform was adjusted to a comfortable position and locked into place. Hand grip was adjusted as required to match the forearm length. Platform and grip positions were recorded for duplication during subsequent sessions.

Ground wire plate was coated with electrode paste and strapped to the right forearm. Alcohol applications sterilized skin surface over the right biceps. Right elbow was flexed to 90° without added cable weight and the electrode inserted fully to the crown. Needle electrode lead wires were attached to the EMG plugbox arm and power was applied to the electromyograph system.

Elbow position was rigidly maintained at the specified angle for two reasons. First, movement from this set position would cause bending, possible breaking of the needle and much discomfort for the subjects. Secondly, movement of the arm may have produced misleading artifact on the EMG or may have modified motor unit

Figure 8. Photograph of subject and apparatus during testing session.

utilization.

Experimentation occurred in two phases. Phase one established procedures for identification and measurement of individual motor units. Phase two developed protocol for monitoring individual motor units through a prolonged, fatigue-producing isometric contraction.

Requirements for phase one included isolation of a small number of active motor units, identification of those individual units and measurement of impulse frequency and amplitude. Criteria for identification of unit potentials included distinctive form, fairly constant rhythm and nearly uniform amplitude. Frequency was expected to vary about ten percent or more since weak contractions were utilized.

Computations of frequency and amplitude were based on the following relationships. One centimeter deflection horizontally on the electromyogram equaled 100 milliseconds and one centimeter deflection vertically equaled 100 microvolts. Measurements were made with a centimeter scale.

Frequency was calculated from the distance between adjacent spikes of the same unit measured at their lower tips. Given the above relationship and the conversion factor of 1000 msec/sec, the following equation was developed:

$$F = \frac{1}{d(100\,\text{msec}/1000\,\frac{\text{msec}}{\text{sec}})}$$

F is frequency and d is the value representing distance between spikes. Mathematical simplification of the equation produces

$$F = \frac{1000 \text{ msec/sec}}{d\,(100 \text{ msec})}$$

or

$$F = \frac{10}{d \text{ sec}}$$

Utilizing the equation, a sample calculation is presented with a distance of 2.5 cm between spikes.

$$F = \frac{10}{2.5 \text{ sec}} = 4 \text{ imp/sec}$$

Amplitude measurements were taken from the highest positive to the lowest negative points on each spike. Multiplying this distance by 100 microvolts produced potential amplitude. Three centimeters distance, for example, would compute to 300 microvolts.

Frequency and amplitude data were evaluated for range, standard mean ($\overline{X}$) and standard deviation (s). Computations were made utilizing the following equations:

$$\overline{X} = \frac{\Sigma X}{n}$$

$$s = \sqrt{\frac{\Sigma\,(X - \overline{X})^2}{n-1}}$$

X is individual readings of frequency or amplitude.

n is the number of individual readings.

Weights added to the cable varied from zero to ten pounds during the course of this investigation. Pivot arm, grip, cable and weight holder totaled three pounds without attached weights. Initially, needle positions were tested throughout the muscle belly of the right biceps. Subsequent sessions utilized a specific area to maintain some degree of consistency. That target area was located centrally on the long axis of the short head of the biceps and distal to its midpoint one-quarter of the muscle length. Muscle length was considered to be the distance between muscle-tendon junctions and estimated by palpation. Frequent adjustment of needle tip position was made in an attempt to isolate single units. EMG recorder settings were tested in recording modes of Single sweep, Raster, and Continuous, at recording speeds of 5, 10 and 20 centimeters per second in Continuous and Raster modes, and at sweep velocities of 5, 10 and 20 milli-seconds per divisions.

Phase two required placing the muscle under a sustained fatig-fatiguing contraction under conditions allowing identification and measurement of individual motor units. Monitoring of constant muscle sites without changes in body position or muscle utilization was mandatory. Weights ranging from 0 to 10 pounds were added to cable and pivot arm assembly during the fatigue portion. Trace samples were recorded before and after fatigue.

Test Description and Procedures

Suitability of the model developed in this investigation was determined by successful evaluation and interpretation of motor unit activity during a sustained contraction. Student's t-test provided a basis for evaluating changes occurring in recorded potential amplitude and frequency for each active unit.

The null hypothesis was tested concerning changes in frequency and amplitude between pre- and post-fatigue recordings. Comparison of independent samples of unequal size required the use of the equation:

$$t = \frac{\overline{X}_1 - \overline{X}_2}{S_{\overline{X}_1 - \overline{X}_2}}$$

Probability of a greater t-value was found in the distribution of t table (78). $\overline{X}_1$ and $\overline{X}_2$ represent means of sample one and sample two, or more specifically, pre- and post-fatigue means. $S_{\overline{X}_1 - \overline{X}_2}$ represents the pooled standard error and is computed by the equation:

$$S_{X_1 - X_2} = \sqrt{S^2 \frac{n_1 + n_2}{n_1 n_2}}$$

n_1 and n_2 are sample sizes. S^2 is the pooled standard deviation computed by the equation:

$$S^2 = \frac{\Sigma(X_1 - \overline{X}_1)^2 + \Sigma(X_2 - \overline{X}_2)^2}{(n_1 - 1) + (n_2 - 1)}$$

Degrees of freedom (d.f.) for hypothesis testing is $(n_1 - 1) + (n_2 - 1)$.

Utilizing these equations, a sample calculation is presented with the following frequency data:

Sample One	Sample Two
$n_1 = 5$	$n_2 = 10$
$\Sigma(X_1 - \overline{X}_1)^2 = 0.255$	$\Sigma(X_2 - \overline{X}_2)^2 = 0.177$
$\overline{X}_1 = 1.951$ imp/sec	$\overline{X}_2 = 1.804$ imp/sec

$$S^2 = \frac{0.255 + 0.177}{4 + 9} = 0.03323$$

$$S_{\overline{X}_1 - \overline{X}_2} = \sqrt{0.03323 \ \frac{5+10}{(5)(10)}} = 0.09985$$

$$t = \frac{1.951 - 1.804}{.09985} = 1.472$$

$$d.f. = 4 + 9 = 13$$

The null hypothesis would not be rejected in this case since the probability of a greater t-value is near .20 (78). This slowing of frequency would be considered due to normal fluctuation and therefore, not significant.

Application of student's t-test to electromyogram data enabled evaluation of motor unit performance during sustained contractions.

Evaluation and interpretation of results reflected suitability of the established model.

CHAPTER IV

RESULTS AND INTERPRETATION OF DATA

This investigation was designed to establish testing procedures which allow research of individual motor unit performance throughout a sustained contraction. Electromyograms obtained from a variety of experimental procedures, electromyograph settings and equipment modifications provided the necessary information.

Results were interpreted in two phases. Phase one concerned isolation and identification of individual motor units. EMG potentials with measurable frequency and amplitude characteristics were required. Phase two concerned monitoring individual units through a fatigue-producing contraction. The capability of measuring changes in frequency and amplitude which would allow hypothesis testing was required.

Phase One: Identification of Units

Phase one requirements included recording motor unit potentials of sufficient clarity to allow identification and measurement as described in Chapter III. Electromyograms were analyzed in order to identify necessary procedural or instrumentation adjustments.

Initial testing sessions were conducted with $8\frac{1}{2}$ pounds supported by the right arm. Eight and one-half pounds represented 15-20%

maximum for subjects used. Figure 9 illustrates a series of recorded electrical potentials at that weight. Electromyograms in figure 9 and subsequent figures typify recordings presented in existing literature (48, 64). Time dot rows separate individual EMG strips recorded by depression of a foot pedal. Vertical deflections indicate membrane depolarization of motor unit muscle fibers. Identification of motor units required potentials to be recorded without interference from other units. However, numerous units recorded in Figure 9 caused potentials to alter characteristic form of one another and precluded positive identification. Amplitude of a given motor unit's spikes varied greatly due to positive or negative effects of other potentials recorded at or near the same moment. Excessive weight was determined to be a factor in recording such highly active traces.

Supported weight was reduced to 5 1/2 pounds, or 10-15% maximum. Electrical activity at this weight is illustrated by EMG strips in Figure 10. Reduction in number of recorded motor units took place as indicated by fewer vertical deflections. Potentials continued to interfere with form and alter amplitude. Results indicated the necessity to reduce contraction intensity further.

Movement of subjects produced significant effects during the investigation. Shifting of body or arm positions affected electrical activity as illustrated in Figure 11. Movement took place between strips 3 and 4 and is reflected by a marked increase in amplitude. Alteration of EMG amplitude may have been a manifestation of motor unit activation in close proximity to the electrode or a reduction in

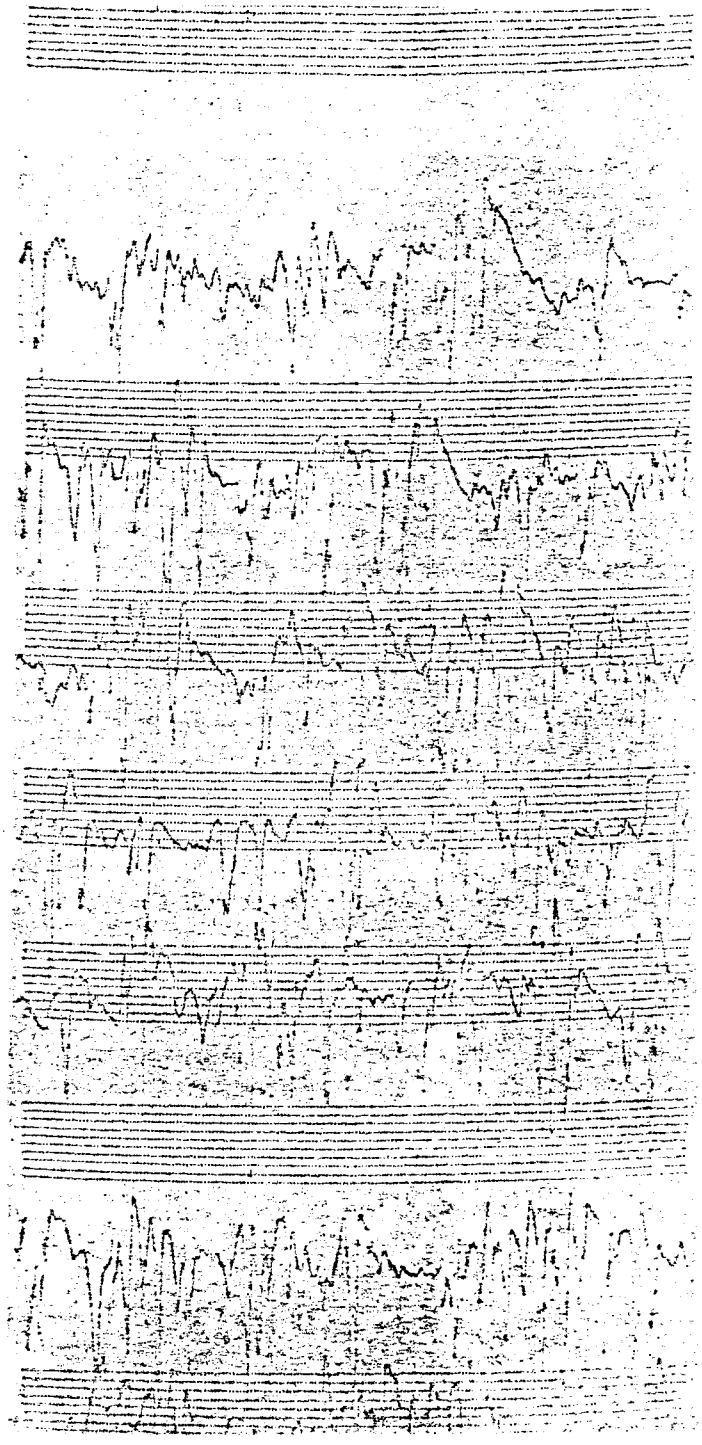

Figure 9. EMG trace with $8\frac{1}{2}$ pounds supported. Activity indicated numerous motor units. Potentials altered characteristic form and amplitude of one another.

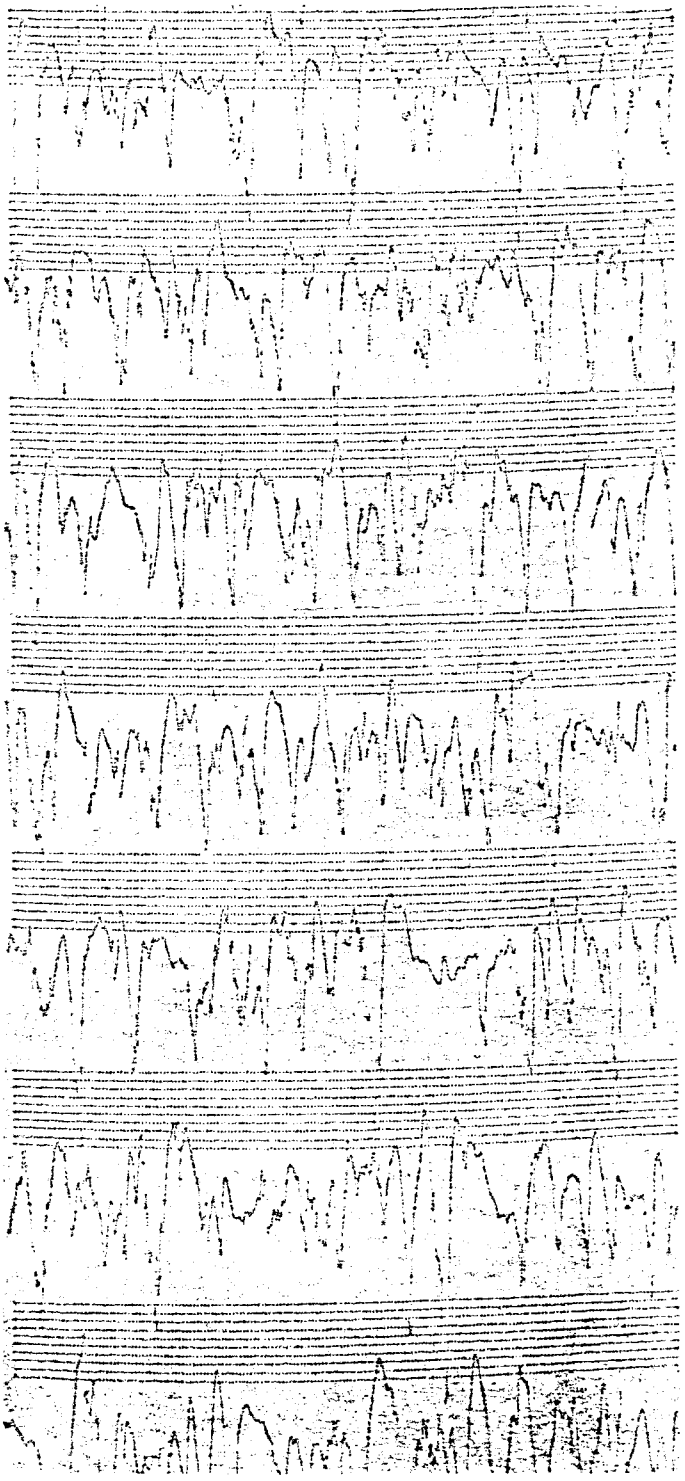

Figure 10. EMG trace with $5\frac{1}{2}$ pounds supported. Reduction in number of spikes had occurred; however, interference in form and amplitude continued.

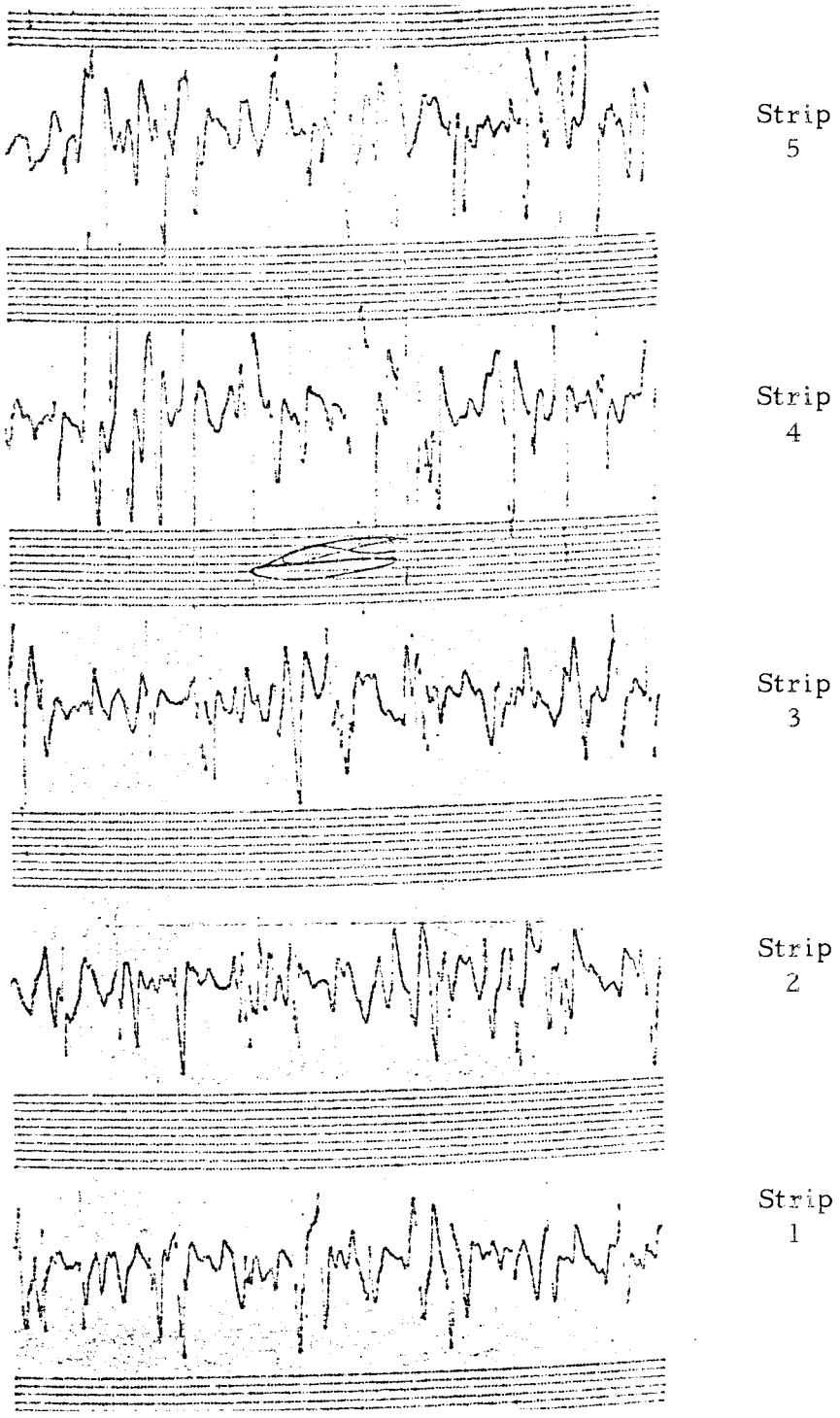

Figure 11. EMG trace illustrating effects of shifting body position. Movement took place between strips 3 and 4 and was indicated by an increase in amplitude.

needle tip to active fiber distance caused by the muscle movement. Either situation was considered undesirable since they were subject-induced. Testing session of 20 to 30 minutes, however, presented a difficult situation for subjects to rigidly maintain body/arm position and aids were considered necessary.

Modification of the apparatus chair provided a means to reduce movement artifact. Two leather belts and dowel marker were added. One belt was attached to the chair back and used to strap the right shoulder securely to the chair. A second belt was strapped around the pivot arm halfway between the pivot point and the hand grip. Addition of the two belts helped to prevent shifting of the subjects' back and right shoulder, and flexion or extension of the right wrist. One-half inch dowel marker with corresponding pins on dowel and pivot arm simplified maintenance of proper elbow angle. Without an exact marker, time and fatigue drew subject's attention away from arm angle and variations were more common.

Needle selection was considered important with respect to length. CT37P needle, 37 mm long, was used during the first testing session only. Low force contractions activate primarily tonic fibers which are clustered centrally in the muscle belly. Use of the CT37P electrode produced a detection point well past the muscle center and nearly beyond the muscle. Placement of the tip in the center left a portion of the electrode exposed outside the muscle, subject to

increased electrical interference. Either situation with the CT37P was undesirable. Subsequent testing utilized the 25 mm CF25P electrode.

Appropriate placement of needle electrodes was considered one of the most important and critical factors affecting results. Placement of the electrode tip in close proximity to an excessive number of active units produces a potential trace difficult to interpret. Results were similar to those found when using heavy weights. Figure 12 illustrates activity from numerous units which altered form and amplitude of one another and precluded identification. Movement of the needle to another site produces results in Figure 13. Large single spikes in Strips 1 and 3 indicate that one unit was near the electrode tip and could be easily distinguished from other units more distant. Adjustment of the tip position continued throughout the recording on Figure 13 and caused appearance and disappearance of the large spikes and changes in amplitude.

Background "noise" recorded was attributed in part to electrical disturbances in and around the building. Figure 14 reflects a large amount of electrical activity, part of which was considered artifact. Distinctive form of unit potentials was disturbed by numerous small spikes and produced uninterpretable results. Steps taken to reduce "noise" were:

1) Turned off all electrical lights where possible in testing room

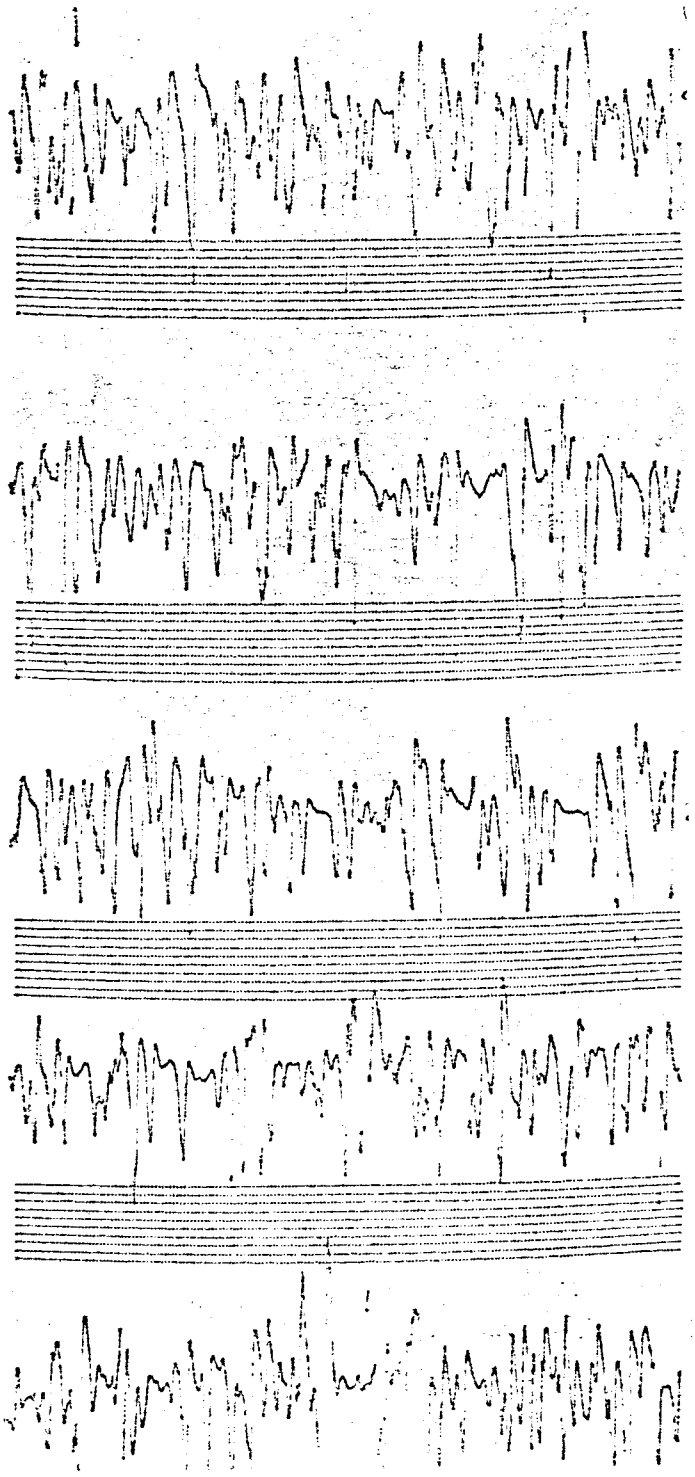

Figure 12. EMG trace with electrode tip positioned in close proximity to many active motor units. Excessive number of recorded spikes interfered with form and amplitude of one another.

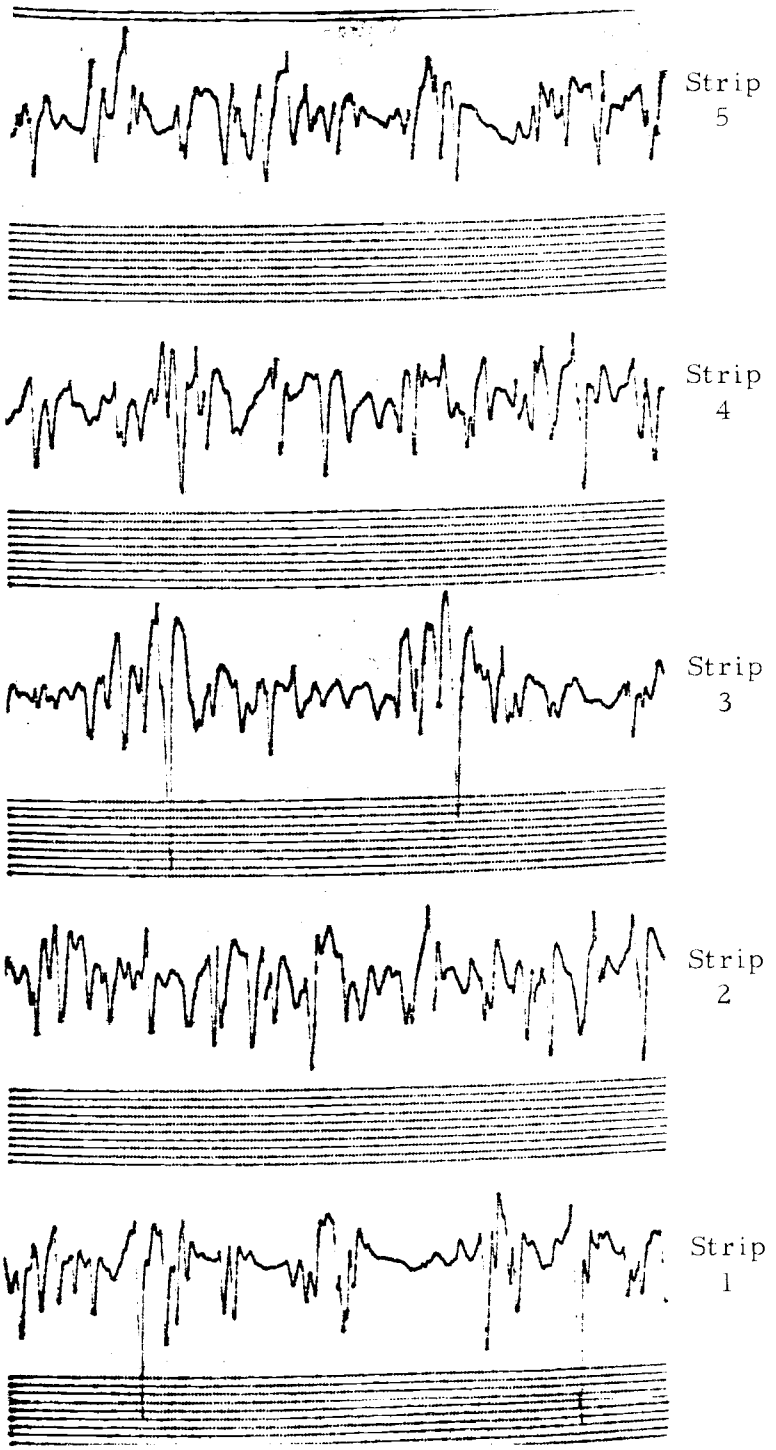

Figure 13. Movement of needle to another site reduced amplitude of all units except one which was near the electrode tip. Continued movement of needle produced variation in spike amplitude and appearance of large potentials in strips 1 and 3.

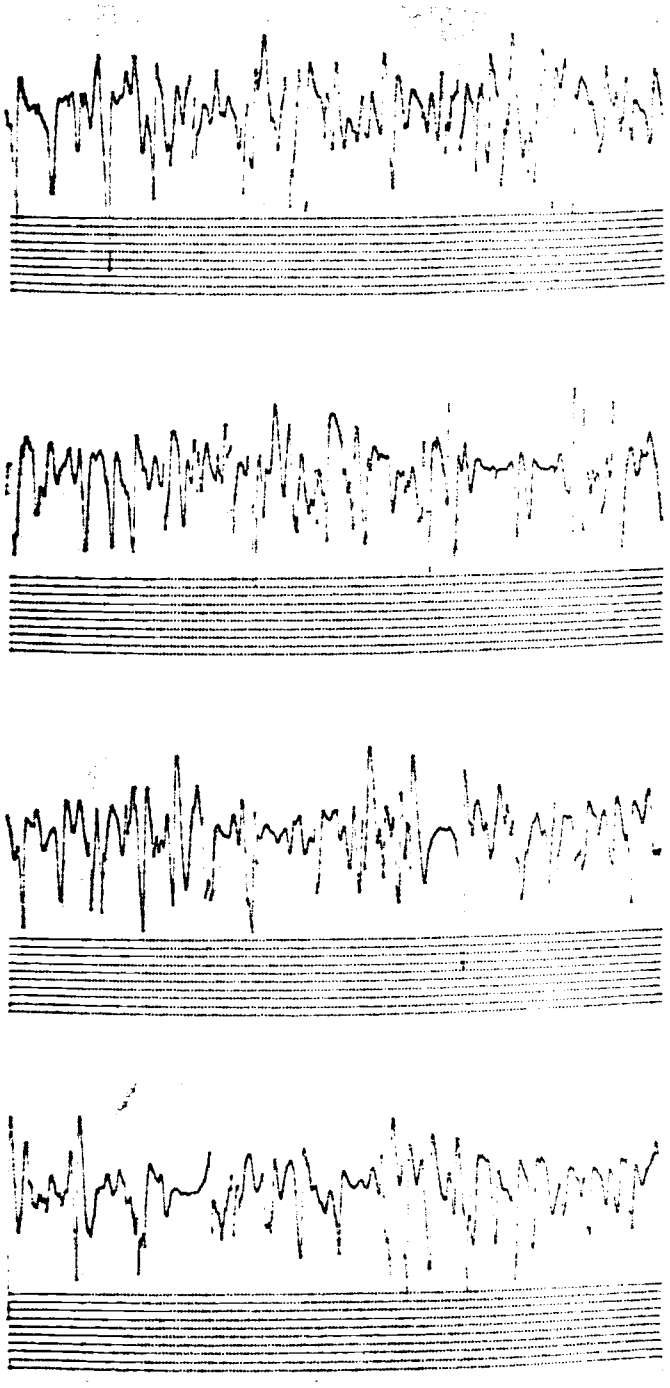

Figure 14. EMG trace which included considerable background "noise." Electrical current in and around the room was considered a significant contributor to the numerous small spikes recorded.

and adjacent rooms. Used flashlight to provide necessary lighting.

2) Fully inserted needle into muscle.

3) Verified that AC wiring in testing room and building was located in grounded metal conduit.

4) Turned off all heavy-load electrical items in building where possible.

5) Insured proper grounding of subject.

Electromyogram traces which allowed identification and measurement of individual motor units were recorded with careful attention to previously discussed factors. Samples of measurable results are illustrated in Figures 15 and 16. Both traces were recorded with $1\frac{1}{4}$ pound weight added to the cable and pivot arm assembly making total weight supported by the subject of $4\frac{1}{4}$ pounds. Figure 15 demonstrates activity of a single motor unit in close proximity to the electrode tip. Potentials of more distant units was not of sufficient amplitude to create difficulty in identifying primary unit spikes. Measurements and computations for frequency and amplitude are presented in Table 1.

A recording of two separate motor units is presented in Figure 16. For identification purposes, the unit with greater amplitude was labeled A and unit with lower amplitude was labeled B. Computations were made concerning frequency and amplitude of units A and B and

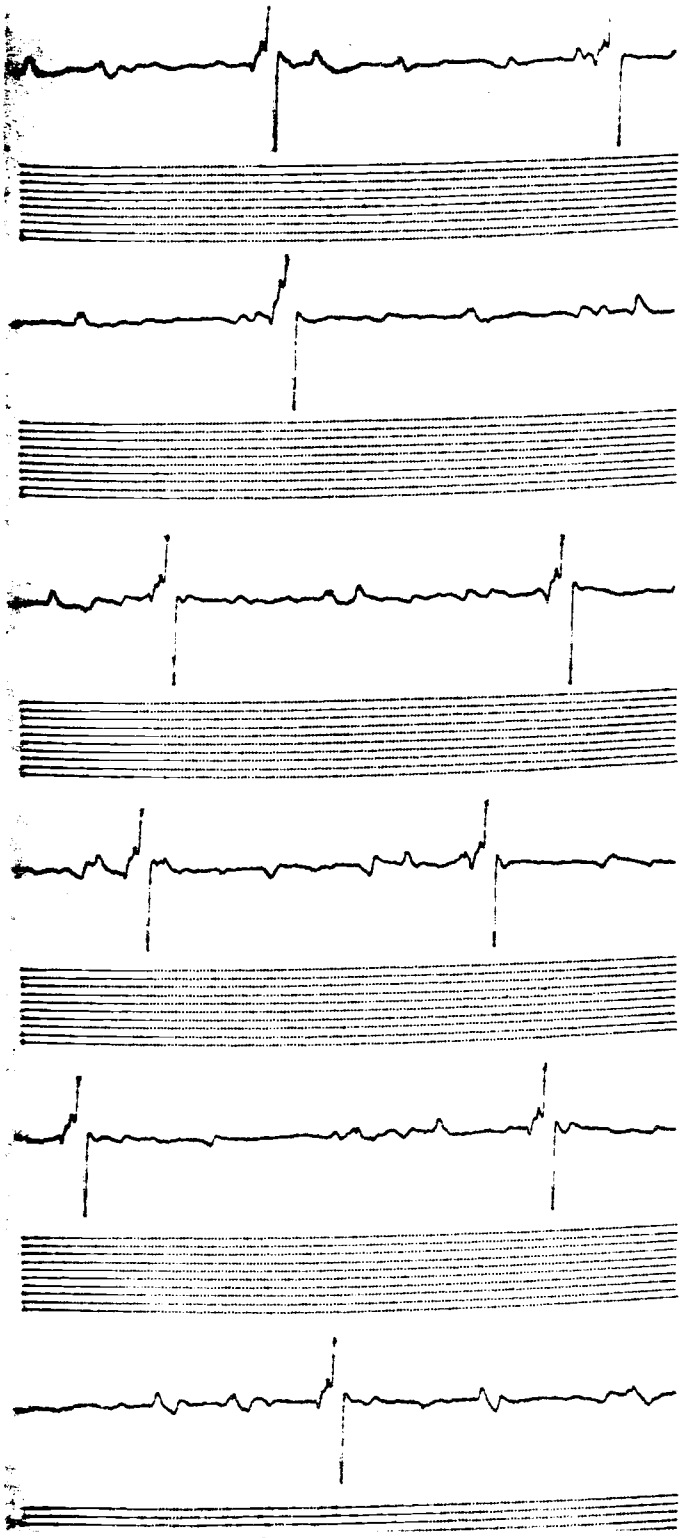

Figure 15. EMG trace with $4\frac{1}{4}$ pounds supported. Single unit potentials were recorded and easily measured for amplitude and frequency. More distant units were of low amplitude and did not interfere with measuring.

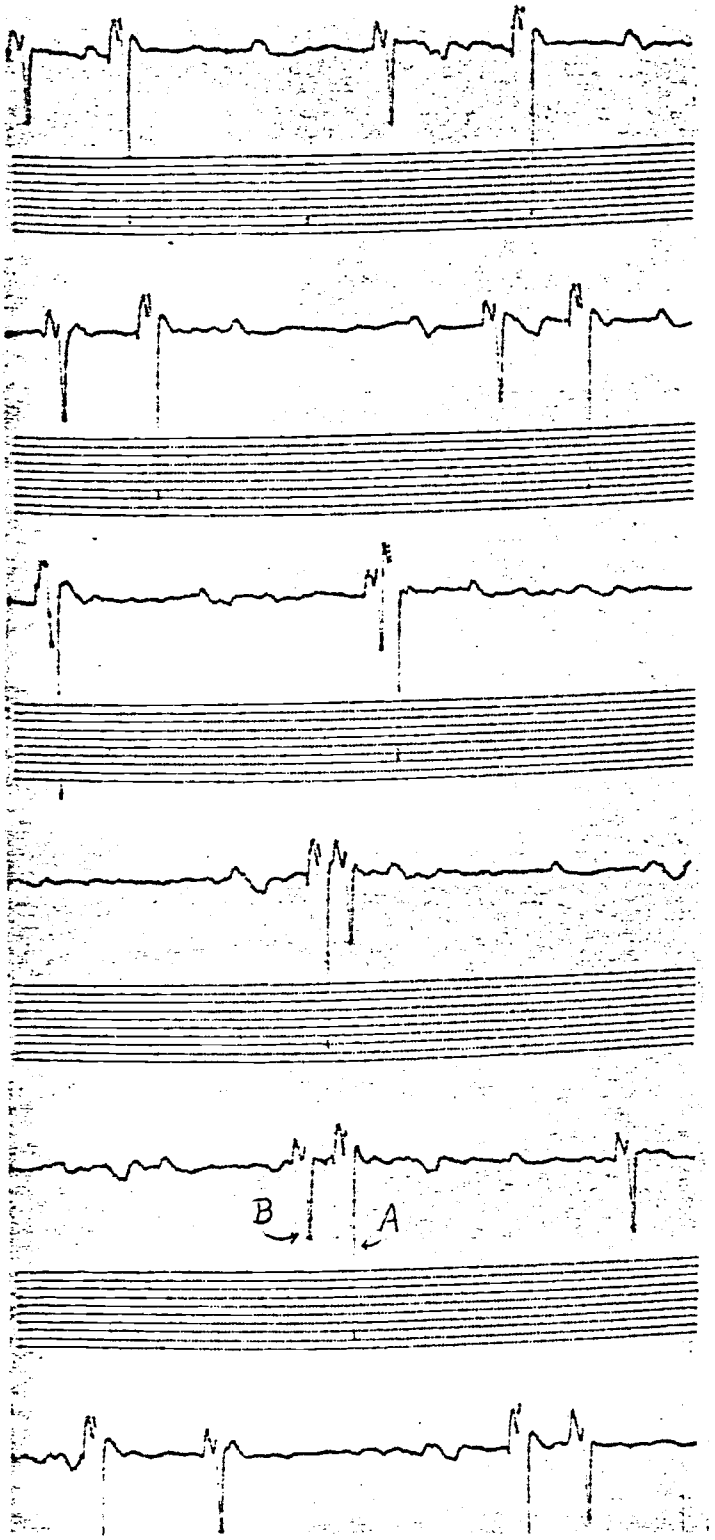

Figure 16. EMG trace with $4\frac{1}{4}$ pounds supported. Two motor units were recorded. Higher amplitude spikes were designated Unit A and lower spikes Unit B.

Table 1. Measurements and computations for frequency and amplitude of motor unit recorded in Figure 15.

Frequency Data: Distance between individual spikes in cm	Time between spikes in milliseconds (based on 1 cm=100 msec)	Frequency: Impulses per second	Amplitude Data: Spike height in cm	Microvolt equivalent (1 cm=100μv)
4.55	455	2.198	1.97	197
5.28	528	1.894	1.93	193
4.61	461	2.169	1.89	189
6.21	621	1.610	1.97	197
			1.92	192
			1.99	199
			1.99	199
			2.07	207
			1.99	199
			1.90	190

Frequency Range: 1.610 to 2.198 inpulses per second

Standard Mean: 1.968 impulses per second

Standard Deviation: 0.275 impulses per second

Amplitude Range: 189 to 207 microvolts

Standard Mean: 196.2 microvolts

Standard Deviation: 5.37 microvolts

are presented in Table 2. Measurements indicate consistency of motor unit amplitude. Frequency displayed greater variance than did amplitude. Combination of frequency and amplitude of each unit was rather specific and provided data to distinguish units in addition to distinctive form.

Procedures allowing electromyogram quality of Figures 15 and 16 were satisfactory for the purpose of this investigation. Motor units were identified and computations of frequency and amplitude were performed. Such EMG clarity allowed progression to phase two.

Phase Two: Monitoring of Sustained Contraction

The purpose of phase two was to develop procedures which would provide data concerning motor unit electrical potentials during sustained contractions. Phase one EMG quality was necessary for measurement and computation of spike frequency and amplitude.

EMG recordings were initially taken on a continuous basis throughout testing sessions of 15-30 minutes. Large quantities of EMG printout were acquired and considered excessive for manual evaluation techniques. Continuous recordings necessitated use of a computer for evaluation. Such provisions were not available to the investigator. Systematic sample recordings were therefore considered necessary.

Periodic single EMG strips were then recorded at 10, 15 and 20

Table 2. Frequency and amplitude data for motor units A and B recorded in Figure 16.

	Range	Standard Mean	Standard Deviation		Range	Standard Mean	Standard Deviation
Frequency (imp/sec)				Amplitude (microvolts			
Motor unit A	1.812-2.309	1.975	0.230	Motor unit A	260-305	273.6	13.28
Motor unit B	1.786-2.410	2.115	0.256	Motor unit B	107-141	129.8	9.99

second intervals. Excess quantities of printout were still recorded. Furthermore, isolated single EMG strips may not have reflected appropriate amplitude and frequency characteristics at that stage of fatigue. These parameters fluctuated and the possibility existed that a non-representative sample was recorded. Results indicated a requirement for reducing the quantity of data and for recording representative samples.

Weights of up to ten pounds were then added to produce fatigue more rapidly. Length of the fatigue periods varied with endurance of the individual. Subjective evaluation was used to discontinue fatigue before the subject began to lose control of the muscle or lose desire. Pre-fatigue recordings were taken under phase one conditions. Following "fatigue," pre-test weight was re-established and three post-fatigue traces were recorded at one-minute intervals.

Fatigue occurred much more rapidly with heavy weights than during previous sessions. Rapid onset of fatigue was usually accompanied by twitching or quivering of the muscle belly which often continued through post-fatigue recording. Figure 17 illustrates activity of units as twitching occurred. Two large spikes appear only in strip 4 and exemplify intermittent activation of muscle fibers. Motor units were not identifiable due to sporadic potentials and increase in total activity recorded. Characteristic form was masked

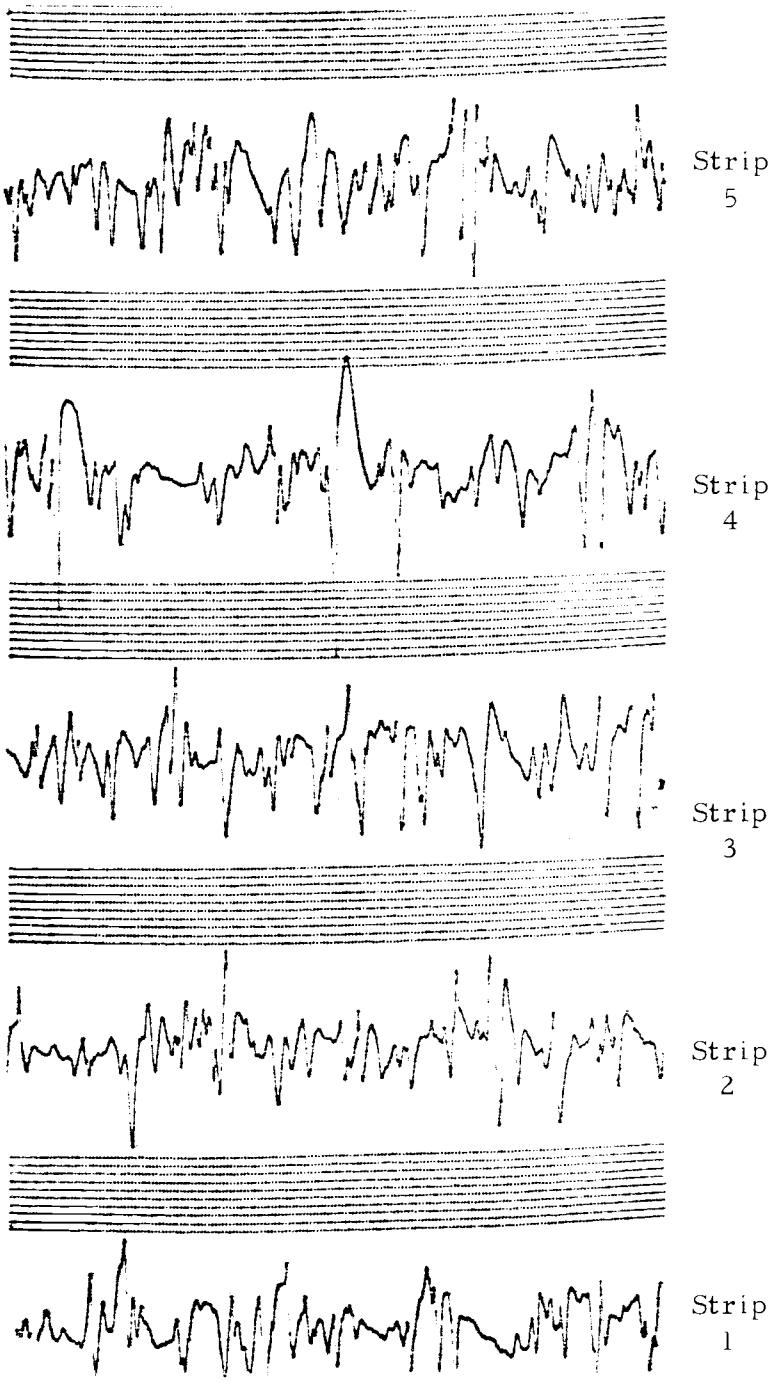

Figure 17. EMG trace during period of muscle twitching caused by excess fatigue. Fluctuating activity precluded identification of motor units. Two large potentials recorded in strip 4 illustrate sporadic activation of units.

and amplitude varied greatly. Fluctuating activity may have resulted from actual changes occurring in motor units or from electrode tip movements caused by muscle twitches.

Results may have been affected as weights were added and extracted before and after fatigue. Subjects could not maintain exact arm position as load changed. Arm movement at these moments may have produced a significant electrode shift or a rotation of units.

Acceptable results were found using a combination of the previous procedures. A constant load of $4\frac{1}{4}$ pounds ($1\frac{1}{4}$ weight added to cable and pivot arm assembly) was maintained for approximately 14 minutes. Pre-fatigue recordings consisting of ten strips were taken the first minute into the test. Ten minutes were then allocated to producing fatigue. Following the fatigue period, three post-test recordings were taken at one-minute intervals. Each recording consisted of ten individual strips of activity in rapid succession. Throughout the entire test, no change occurred in load or body-arm position.

Figures 18, 19, 20 and 21 present a portion of recordings from a single test session where procedures proved acceptable. Pre-fatigue activity is presented in Figure 18 while post-fatigue 1, 2 and 3 potentials are shown on Figures, 19, 20 and 21, respectively. EMG quality permitted measurement and computation of frequency and amplitude for each motor unit.

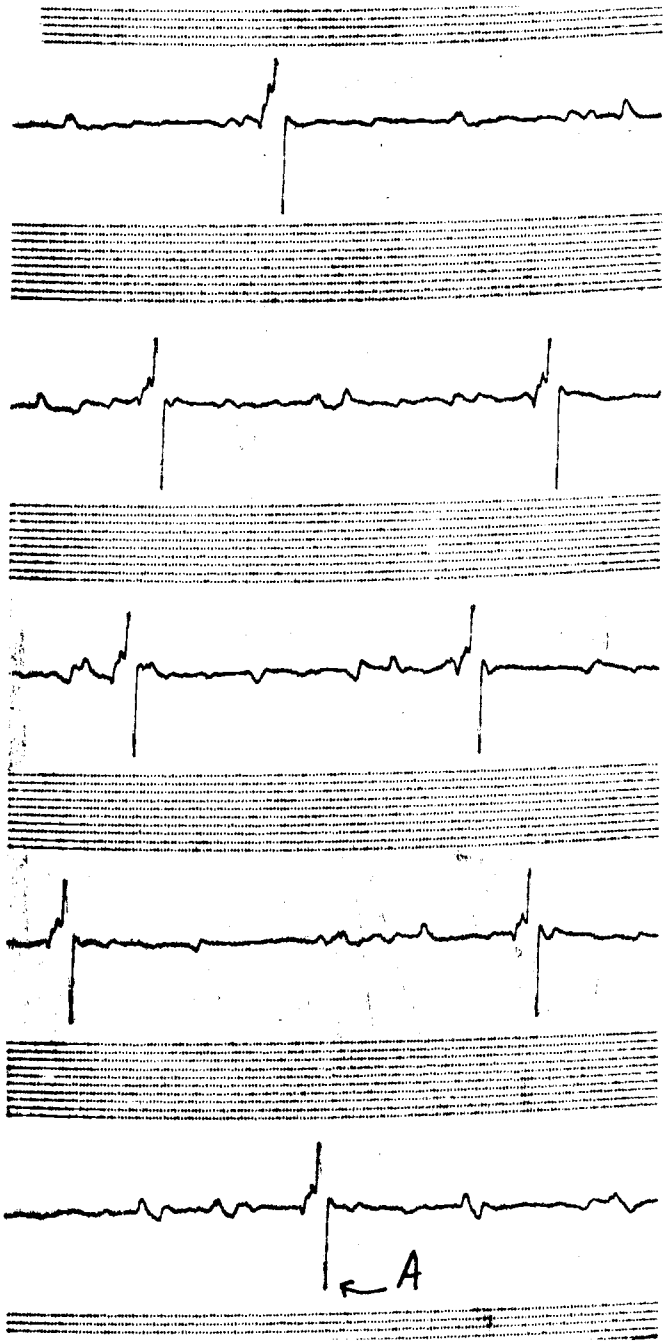

Figure 18. EMG trace during pre-fatigue period. One motor unit was recorded.

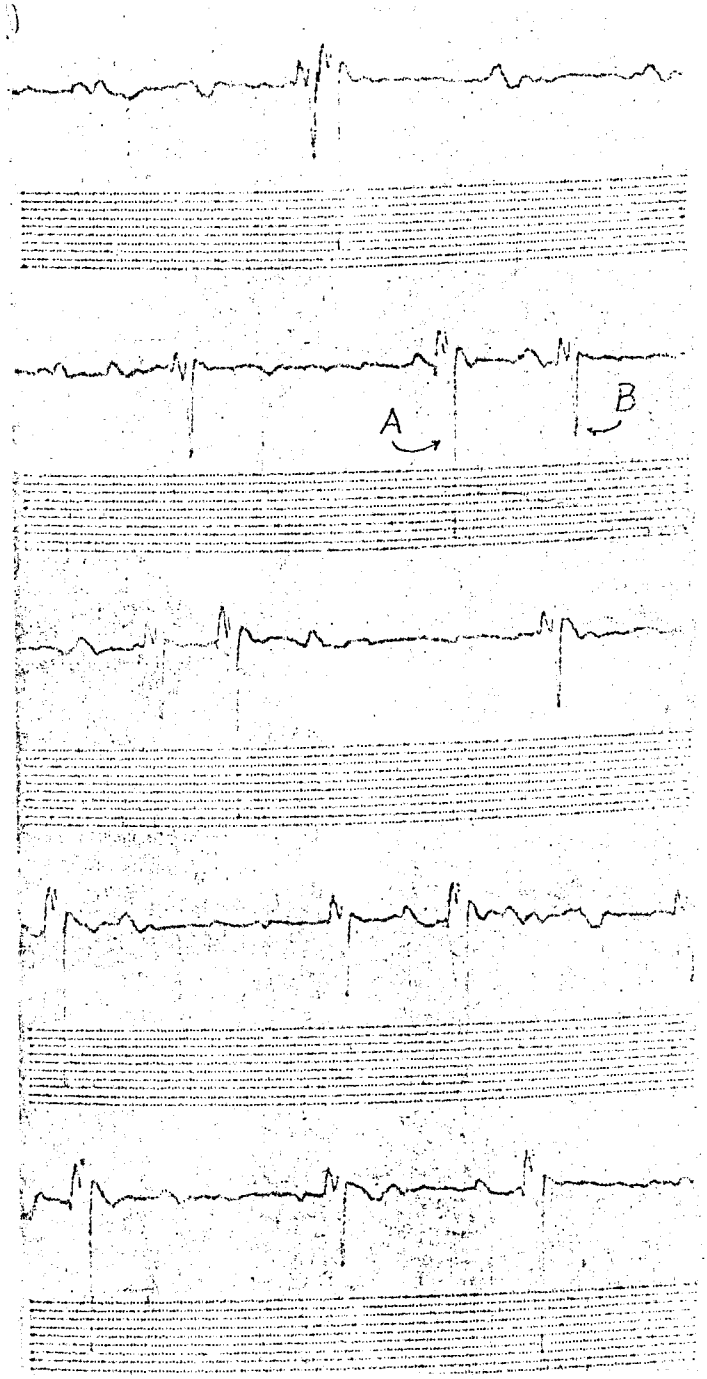

Figure 19. EMG trace from post-fatigue-1 period. Original unit A was joined by new unit B of lower amplitude.

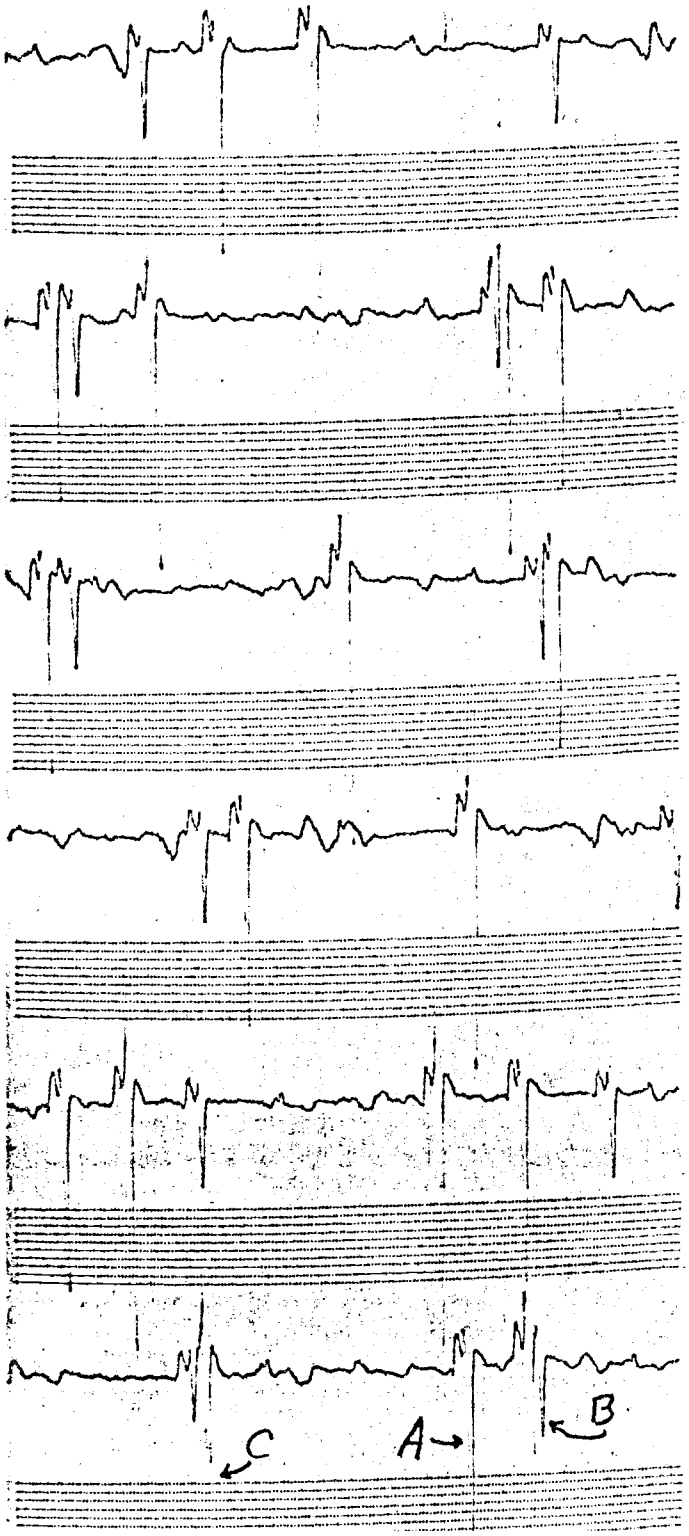

Figure 20. EMG trace from post-fatigue-2 period. Motor unit C had been activated while units A and B remained active.

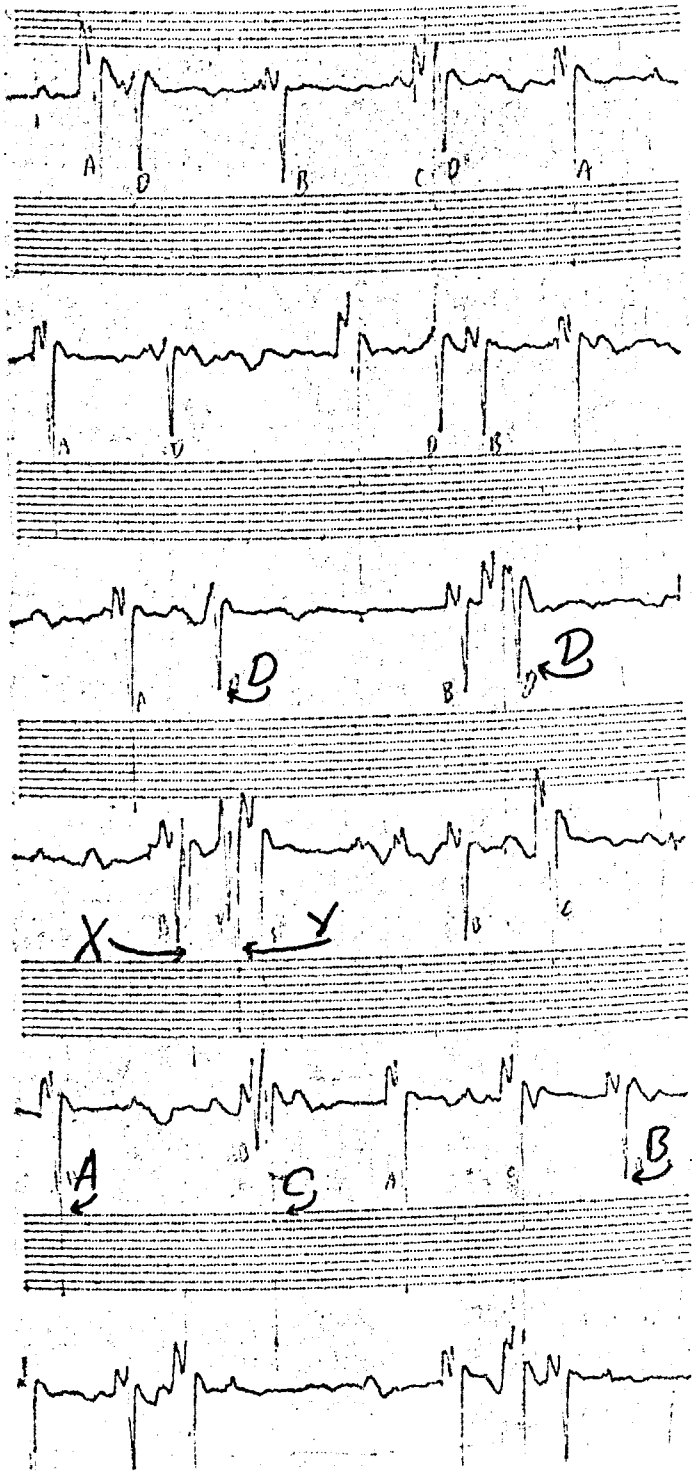

Figure 21. EMG trace from post-fatigue-3 period. Units A, B and C were joined in strip 4 by new unit D which remained active throughout post-fatigue-3. Spikes X and Y were not identified; however, one was presumed to be unit A and the other from another motor unit during a single twitch.

Pre-fatigue potentials were recorded from a single motor unit labeled A. Distinctive form of the unit remained consistent throughout the recording. Amplitude was 195.86 microvolts with a standard deviation of 6.011 microvolts. Unit frequency was 1.951 impulses per second with a 0.253 imp/sec standard deviation.

Figure 19 reflects activity of two units recorded during post-fatigue-1. Large spikes were identified as unit A from Figure 18 based on form and frequency. Amplitude increased approximately 35%. Smaller amplitude potentials indicated activation of a second motor unit B. Discharges from the new unit were slightly higher in frequency than from A.

Figure 20 reflects activity from post-fatigue 2. Motor units A and B were identified as the two lowest amplitude spikes. Appearance of a third unit C was indicated by the very large potentials of over 400 microvolts.

Post-fatigue 3 recordings are presented in Figure 21. Units A, B and C were again identified. Potentials of a fourth unit D appear during this recording. Amplitude and frequency of units B and D were relatively similar. Distinctive form of each unit served as the primary means of identification. Figure 21 also contains two spikes, X and Y, from units which were not identified. Amplitude of both was near that of unit A; however, interference of other unit potentials masked their form. One was presumed to be from unit A while the

other was considered a single potential from still another unit.

Tables 3 and 4 present frequency and amplitude data from recorded motor units A, B, C and D during pre-fatigue and post-fatigue 1, 2 and 3 periods. Figures 22 and 23 illustrate fluctuations in frequency and amplitude, respectively, of the four motor units.

Addition of new motor unit potentials occurred in each successive recording period. Frequency declined slightly and then increased. Amplitude increased from each recording session except for motor unit C which had a decline.

Phase two results indicate that procedures were developed to monitor motor units during sustained isometric contractions. Recorded electromyograms permitted identification of motor unit potentials and computation of their frequency and amplitude.

Model Testing

Hypothesis testing in this investigation was conducted to verify suitability of the established procedural model. Limited electomyogram samples underwent Student's t-test evaluation and established the certainty that results were testable. Experimentation was conducted to produce a workable model and not to gather sufficient data for conclusions regarding motor unit performance.

The null hypothesis was tested with respect to motor unit impulse frequency and amplitude variations between pre-fatigue and

Table 3. Frequency and amplitude data for motor units A and B during pre-fatigue and post-fatigue-1, 2 and 3.

	Range	Mean	Standard Deviation	n	$\Sigma (X-\overline{X})^2$
Motor Unit A					
Frequency (imp/sec)					
pre-fatigue	1.613-2.212	1.951	0.253	5	0.255
post-fatigue-1	1.558-2.070	1.804	0.140	10	0.177
post-fatigue-2	1.515-2.331	1.777	0.335	5	0.448
post-fatigue-3	1.471-2.237	1.900	0.289	5	0.335
Amplitude (micro v)					
pre-fatigue	185-206	195.86	6.011	14	469.71
post-fatigue-1	250-281	265.40	8.426	25	1704.00
post-fatigue-2	260-315	286.25	12.540	16	2358.96
post-fatigue-3	261-302	288.23	6.764	13	594.80
Motor Unit B					
Frequency					
pre-fatigue	not active				
post-fatigue-1	1.748-2.558	2.138	0.254	15	0.902
post-fatigue-2	1.563-2.439	1.876	0.275	10	0.680
post-fatigue-3	1.776-2.646	2.157	0.315	5	0.397
Amplitude					
pre-fatigue	not active				
post-fatigue-1	120-142	129.43	5.568	30	899.22
post-fatigue-2	130-148	137.11	5.301	18	477.74
post-fatigue-3	130-155	141.29	6.764	14	594.80

Table 4. Frequency and amplitude data for motor units C and D during their active periods.

	Range	Mean	Standard Deviation	n	$\Sigma(X-\overline{X})^2$
Motor Unit C					
Frequency					
pre-fatigue	not active				
post-fatigue-1	not active				
post-fatigue-2	2.151-2.584	2.335	0.183	6	0.168
post-fatigue-3	2.024-3.448	2.619	0.428	9	1.468
Amplitude					
pre-fatigue	not active				
post-fatigue-1	not active				
post-fatigue-2	400-473	433.47	21.394	15	6407.72
post-fatigue-3	375-470	421.21	31.376	14	12798.30
Motor Unit D					
Frequency					
pre-fatigue	not active				
post-fatigue-1	not active				
post-fatigue-2	not active				
post-fatigue-3	2.451-2.907	2.650	0.184	6	0.170
Amplitude					
pre-fatigue	not active				
post-fatigue-1	not active				
post-fatigue-2	not active				
post-fatigue-3	127-141	131.30	4.855	10	212.10

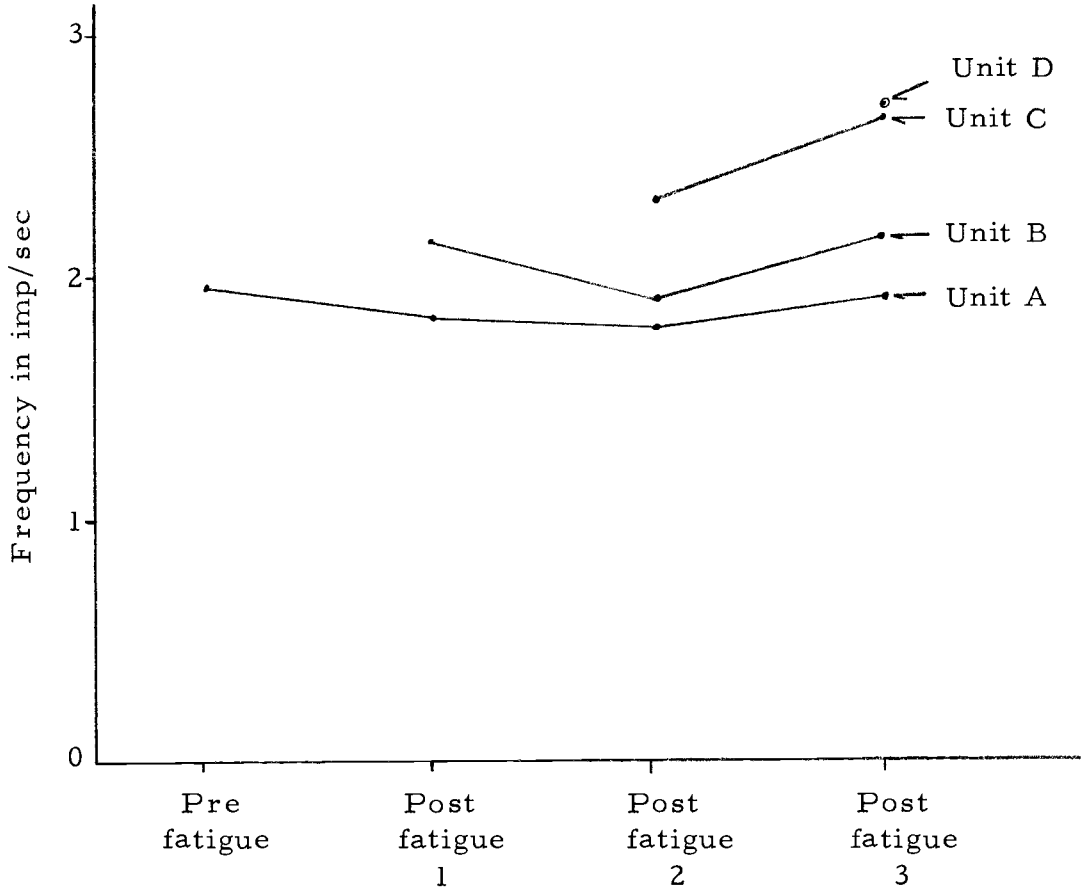

Figure 22. Frequency of motor units A, B, C, and D plotted for each phase in which they were active.

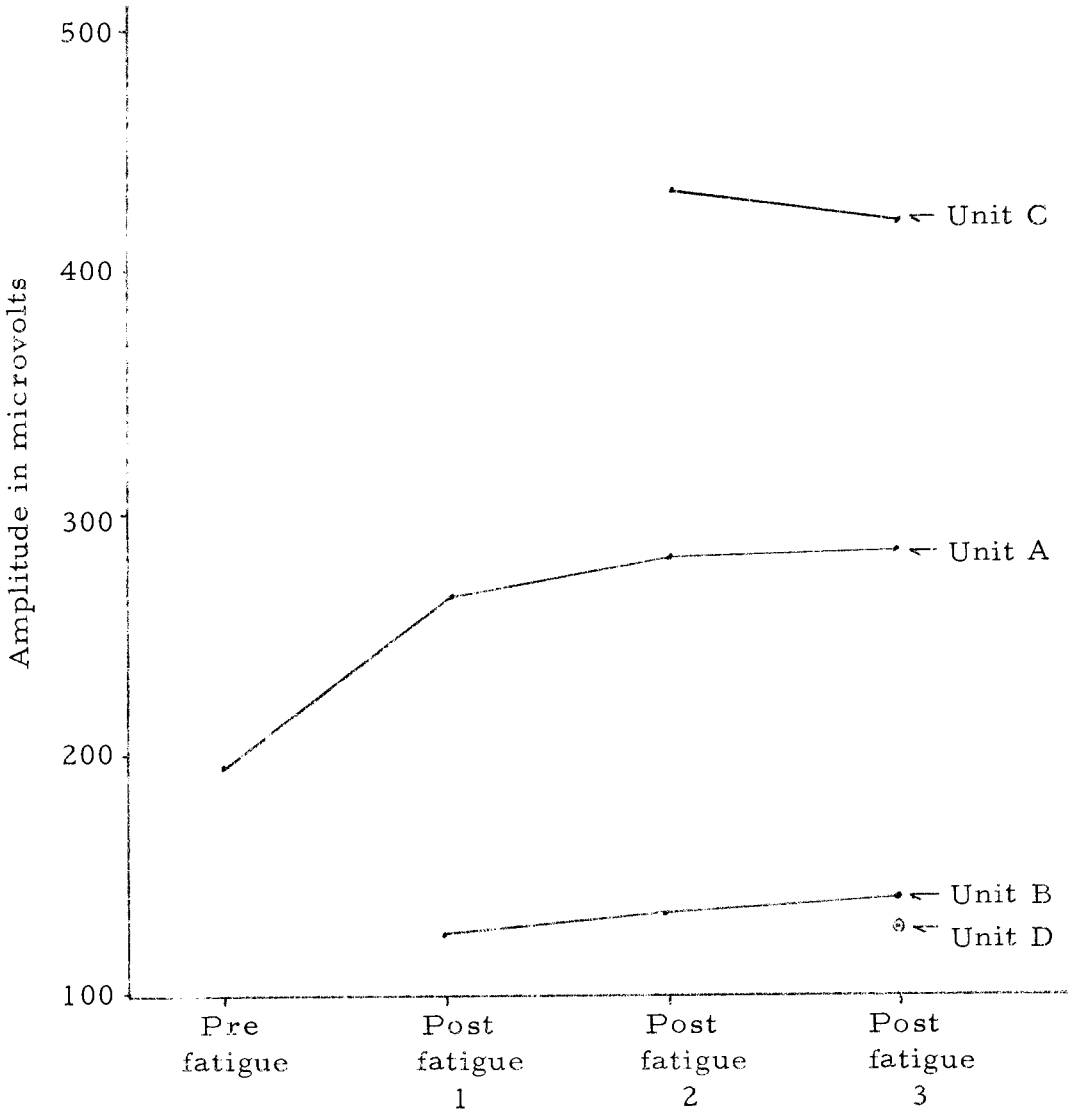

Figure 23. Amplitude of motor units A, B, C and D plotted for each phase in which they were active.

post-fatigue recordings. Additionally, testing was conducted for variations between each post-fatigue recording.

1) Hypothesis one: No change occurs in motor unit frequency between pre-test and post-tests 1, 2 and 3.

2) Hypothesis two: No change occurs in motor unit amplitude between pre-test and post-test 1, 2 and 3.

Motor unit A was the only unit detected in all four recordings and is discussed first. Hypothesis one was not rejected. Frequency generally decreased throughout the session; however, sufficient change for rejection of the null hypothesis at the .05 level did not exist. Additionally, no significant change in frequency occurred between first, second or third post-fatigue tests.

Hypothesis two was rejected at the .001 level for motor unit A. Significant amplitude increases occurred between pre-fatigue and each of the post-fatigue tests. The null hypothesis was also rejected, $p < .001$, for amplitude between post-1 and post-2 but was not rejected between post-2 and post-3.

Motor unit B, which became active during the ten minute fatigue period displayed similar performance. Frequency decreased between post-1 and post-2 but increased between post-2 and post-3. The decrease was significant at a .05 level; however, the subsequent increase was not. Post-1 to post-3 frequency was not significantly different.

Amplitude of motor unit B potentials increased from post-1 to post-2 to post-3. The null hypothesis was rejected, $p < .001$, for post-1 to post-2 and post-1 to post-3. Amplitude increase between post-2 and post-3 did not lead to rejection of the null hypothesis.

Motor unit C fluctuations in frequency and amplitude were not sufficient to warrant rejection of the null hypothesis. Activity of motor unit C was recorded only during the final two post-test periods, however.

Previous data indicates that appropriate procedures have been developed for evaluation of motor unit activity during sustained isometric contractions Electrical potentials have been identified and measured for frequency and amplitude during each recording phase of the contraction. Changes in these parameters have been tested for rejection or non-rejection of the null hypothesis.

CHAPTER V

SUMMARY AND CONCLUSIONS

This investigation was designed as a model for research of motor unit utilization during sustained isometric contractions. Procedures have been established which permit recording of electrical potentials from active motor units of the biceps brachii muscle. Electromyograms of sufficient clarity were recorded in conjunction with sustained isometric contractions. Sufficient clarity implies that action potential recordings allowed identification of motor units and measurement of their discharge frequency and amplitude. The model permitted comparison of frequency and amplitude between pre-fatigue conditions and three post-fatigue states by application of Student's t-test.

Established Procedures

Procedures utilized the Teca TE-4 Electromyograph system and specially designed apparatus chair described in Chapter III. Muscle fibers of the right biceps brachii were tested during 14 minute isometric contractions.

Subjects were seated in the apparatus chair with right arm flexed to 90°. Addition of a one and one-quarter pound weight to the cable and pivot arm assembly provided a 7-10% maximum

contraction. Ground wire plate was attached and CF25P needle electrode inserted fully into the muscle belly. Insertion point was centered on the long axis of the short head of the biceps and distal to its mid-point one-quarter of the muscle length.

Apparatus chair provided firm anchoring of subjects to prevent shoulder and arm movements. Subjects were instructed to avoid body movements and to maintain perfect pivot-arm pin alignment. Electrical interference from the testing room and adjacent rooms was reduced by specific steps outlined in this study.

Isolation of recording sites with few active units required fine adjustment of the electrode. Exact electrode, arm and body position was then maintained throughout the testing period.

Immediately upon securing a stabilized electormyogram, ten strips of activity were recorded in rapid succession by depression of a foot pedal. The contraction continued for ten minutes with strict attention to maintenance of position. Post-fatigue 1, 2 and 3 activity was then recorded at one-minute intervals. Each post-fatigue record consisted of ten individual strips as in pre-fatigue.

Recorded potentials were studied to identify potential spikes from specific motor units. Distinctive form and nearly constant amplitude and frequency served as identifying characteristics.

Following identification of potentials, measurements were taken and computations performed for frequency and amplitude of

each unit. Amplitude was measured from highest to lowest point for each recorded spike which had no interference from adjacent potentials. Frequency measurements were taken between adjacent spikes on each strip with two or more potentials of the same unit. Each centimeter of vertical or horizontal deflection represented 100 microvolts or 100 milliseconds, respectively.

Mean frequency and amplitude changes between each recorded phase were tested for statistical significance by Student's t-test for independent samples of unequal sizes.

$$t = \frac{\overline{X}_1 - \overline{X}_2}{S_{\overline{X}_1 - \overline{X}_2}}$$

Dividend is the difference between two means being tested. Divisor represents the pooled standard error of the two samples.

Interpretation of Sample Motor Unit Activity

Interpretation of sample motor unit activity based on measured and tested parameters was conducted to reflect suitability of the procedural model. Small quantity of data interpreted did not necessarily reflect actual motor unit characteristics.

Additional units were activated during each successive recording period. Minute recording areas in the muscle indicated that significant activity may have taken place outside the detection site.

Activation of additional units may have resulted, therefore, from rotation or fatigue factors. Central control systems may have rotated responsibility for force by deactivating units from non-recorded areas and activating those detected on the EMG. Fatigue, or a decrease in contractility, may have occurred among previously active fibers. Since total muscle force was maintained at a constant, activation of additional motor units would have been required. Deletion of units was not detected in this specific case and therefore either factor is suspect.

Hypothesis testing indicated the level of significance for changes occurring in frequency and amplitude. Sample data suggested little significance in frequency variations. Motor unit B possessed a statistically significant, $P < .05$, decrease in frequency from its first to second active period. Mean frequency increased, however, from second to third recorded period slightly greater than the original decrease. Fewer individual measurements of frequency and greater variance prevented statistical significance of this increase. Practical significance of the initial decrease was therefore reduced. Other motor units did not alter frequency sufficiently to reject the null hypothesis.

Motor units demonstrated a profound change in amplitude. Amplitude increased significantly, $p < .001$, from first recorded period to each successive period for each motor unit except C.

Motor unit C amplitude decreased slightly between its two active periods, but not sufficiently to warrant rejection of the null hypothesis. Rate of increase in amplitude of units A and B diminished progressively between successive recording periods.

Influences on spike height were evaluated in determining the physiological significance of amplitude changes. Greater motor unit synchrony occurs with the onset of fatigue. Distant units which produced small deflections on the EMG trace would increase recorded amplitude of near units if many began synchronous activity. Physical and chemical factors may also have as great an effect on amplitude. Amplitude increases should occur due to temperature increases in transmitting fluids, since electrical resistance is decreased. Undetectable shifting in electrode position toward active fibers will also produce greater spike height. Increased amplitude of motor units A and B at the same time C decreased may have reflected such a movement toward two units and away from the other. Changes in ionic composition of transmitting fluids due to the needle puncture may also effect conductivity. Consideration of previous factors suggests reduced physiological significance of amplitude changes throughout sustained contractions in relation to individual motor units. Actual changes in single motor units which would be reflected by amplitude changes is limited to firing of fewer or greater number of its attached fibers. According to existing literature this variable does not exist.

Sample data suggested that the initial contraction of 7-10% increased to a higher percent of effective maximum without a resultant force increase. Successive recordings detected activity of additional motor units. EMG appearance became progressively more similar to recordings of stronger contractions. If increased amplitude resulted from greater synchronization of motor units, greater percent of maximum is implicated. Synchronization does not occur until the contraction reaches 20% or greater. Sample data suggests that muscles must activate more units during sustained contraction in order to maintain a required force. If more total units were active, fatigue of previously active fibers must have taken place and resulted in a diminished contribution to total force. Decreased force of given muscle fibers without a parallel decrease in EMG amplitude implicates fatigue of excitation contraction coupling or the contraction mechanism.

Interference among closely spaced potentials of different motor units was demonstrated. As number of units increased and fired near the same time, characteristic form was affected and produced some identification difficulty. Furthermore, positive and negative components of single potentials added to and subtracted from amplitude of other close potentials. Several instances occurred where amplitude was significantly altered by such phenomenon.

Preceding interpretation of data indicates that the hypothesis in this investigation was substantiated. A procedural model was

developed for research of motor unit activity during sustained isometric contractions. Recorded electromyograms permitted identification of individual unit potentials and measurement of their frequency and amplitude. Evaluation of motor unit activity occurred through application of Student's t-test for variations in measured parameters. Interpretation of those results was conducted with regard to physiological significance as verification of model suitability.

Application of this model will reflect motor unit activity under specific conditions and conclusions must be drawn accordingly. When appropriate conclusions have been extracted, similar procedures could be developed to study various muscles and levels of contraction. Comparison studies could be developed to relate tonic and phasic fiber characteristics, male and female subject influences and various levels of subjects' physical condition.

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