An Investigation and Evaluation of Acoustic Myography

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by

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Le chemin est long du projet à la chose.

Molière (Le Tartuffe)

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Abstract

The study of vibrations from muscles (AMG) has recently been used to monitor force production from skeletal muscle both *in vivo* and *in vitro*.

It has been found that the amplitude of the vibrations increases with an increase in force, similar to the relationship between the electrical activity (EMG) from the muscle and force. During fatiguing isometric contractions the amplitude of the vibrations have been found to follow the force of the contraction more accurately than the amplitude of the EMG signal. It has been proposed that the vibrations from muscle would be an easy and convenient method of monitoring contractions of skeletal muscle.

The series of experiments described in the following pages investigated the relationships of force, EMG and AMG, using isometric contractions of biceps and triceps brachii, first dorsal interosseus, adductor pollicis and rectus femoris.

It was found that in all cases increases in force produced increases in both EMG and AMG, although the results from the fatiguing contractions were less conclusive, with the AMG amplitude being very variable.

The median frequencies of the EMG and AMG signals were also analysed. The EMG frequencies were much higher than the AMG frequencies, being around 60-100 Hz compared with 12-15 Hz, and much clearer changes in EMG frequency were seen with changes in force. The EMG median frequency increased with the increase in force, and showed decreases during the fatiguing contractions. The AMG median frequency showed no distinct trends remaining around the same value throughout the contractions.

In conclusion, AMG is probably not as useful as was originally thought, due to the variability of the signal. However, it may prove to be a diagnostic aid when used in conjunction with the more conventionally used EMG.

List of Publications

1. Rouse, M.E. and Baxendale, R.H. (1990) A technique to record the sounds emitted from triceps brachii in man during voluntary contractions. J.Physiol. 429:8P.

2. Rouse, M.E. and Baxendale, R.H. (1991) A comparison of the frequency content of muscle sounds and electromyogram during voluntary isometric contractions in man. J.Physiol. 435:80P.

3. Rouse, M.E. and Baxendale, R.H. (1992) Acoustic myogram recorded during isometric contractions of rectus femoris in man. J. Physiol. 446:462P.

INTRODUCTION

The introduction is divided into 7 sections, in order to introduce a fairly recent area of interest, to clarify the three main areas of current research and to introduce the main structure and function of muscle and the nervous system, with reference to the recording of AMG.

These areas are:

1. The historical background to acoustic myography.
2. Recent studies of acoustic myography.
3. AMG studied *in vitro*.
4. The study of fatigue and AMG.
5. Muscle and nerve - structure and function.
6. Muscle Fatigue
7. Analysis of EMG and AMG

The Historical Background to the Study of Acoustic Myography

Acoustic Myography, as the title suggests, is the study and recording of sounds from muscle. To be more specific, it is the recording of low frequency vibrations from actively contracting skeletal muscle. It is an area of muscle physiology about which little is known, but an area which is rapidly expanding, both in the amount of work being done and in the interest it is generating in the scientific world. These sounds can be easily demonstrated, either by pushing thumb and forefinger together close to the ear or by clenching the teeth together. As the transmission of sound is much better in water than in air, the quality of the sounds, or more correctly, the vibrations can be improved by putting your head under water and performing the same movements.

The first chronicled report was by Francesco Maria Grimaldi, an Italian Jesuit priest. He is more often recognised for his treatise on the diffraction of light, in his book - *Physicomathesis de Lumine* (1665). He was also actively involved in the study of acoustics and noticed the presence of low 'rumblings' when he flexed a limb. He attributed this to the emanation of 'animal spirits' from the brain, an idea popularised by Galen, a Greek physician of the 2nd Century AD.

It was nearly 200 years later, in 1810, that the next report appeared. William Hyde Wollaston an eminent physicist, chemist and physician gave a lecture to the Royal Society in which he reported, that when he placed his thumbs in his ears and clenched his fists, he could hear a low rumbling sound. The loudness appeared to increase, as the force exerted increased. To him, this sound resembled a distant carriage travelling over the London cobblestones on a cold, clear morning. Taking this very literally, he asked his cabman to drive at various speeds across the stones until the sounds resembled those that he heard in his ears. The speed that corresponded most closely was 8 miles per hour which is equivalent to 11.7 feet

per second. Luckily, the London cobblestones were a uniform 6 inches across and so the frequency was approximately 23 per second, or a frequency of 23Hz. Wollaston verified this by rubbing a notched stick at various speeds and comparing the sounds from that with those from the muscles. The frequency recorded here was also calculated to be about 23 Hz.

This first published report was followed by an increase research on the sounds from muscles. One of the earlier reports was by the German physiologist and biophysicist, Helmholtz in 1864. He reported that the rumblings seemed to accompany muscle contraction and his calculation of the frequency of the sound agreed with Wollaston. Marey (1874) recorded the sounds from jaw muscles and found that they could be heard on clenching of the teeth, and with increased force of biting there was an increase in intensity. Herroun and Yeo (1885) compared the sounds from stimulated and voluntary contractions, concluding that the sounds during both types of contraction were identical in pitch and quality. They also reported a similarity in the sounds from muscles and heart sounds.

It was almost 100 years later that any further work was published. One of the earlier papers this century was published in the Journal of Physiology (Gordon and Holbourn, 1948). They recorded from the muscle controlling the eyelid, orbicularis oculi, and the muscles controlling the jaw, masseter and temporalis. They compared the firing of single motor units when amplified through a loud speaker system to the clicks of sound that could be determined from the contracting muscle. The muscle action potentials were recorded using silver/silver chloride surface electrodes and the AMG was recorded in one of two ways. Either a crystal microphone was placed over the muscle or a piezo-electric crystal was directly connected to the skin to record both the clicks and any movement of the skin. The records obtained from the eyelid (Figure 1.1a) show very clearly the coincidence of muscle action potentials and clicks. These were recorded with the eye closed throughout but the subject making movements as if to close the eye. The click from the muscle was diphasic

with a duration of around 5-15 msec. They postulated that the sounds must be made by minute movements of the motor units lying underneath the microphone. They suggested that the firing of an action potential in the muscle was accompanied by a mechanical event which was sudden enough to cause a sound. They also acknowledged that when a motor unit fired it was accompanied by contraction of the fibres, which in turn was accompanied by some lateral movement of the muscle which might lead to an increase in contact with the microphone and then a relaxation away from the microphone reducing the contact pressure. This was suggested as one cause of the biphasic waves. By expanding the time scale on their recording oscilloscope, they could estimate the interval between start of the action potential and start of the sound wave, or mechanical wave as they termed it. They reported that this was extremely brief, between 0.4 and 2ms and although variable, was consistent within the same experiment. This type of measurement relies on the presence of synchronously firing units, thus the action potentials and the mechanical events may be being recorded from different units, which would lead to erroneous calculation of any time intervals. They concluded by suggesting that the rumble heard from large contracting muscles might be due to the asynchronous activation of motor units and the clicks heard from single motor units were the basis of the rumble.

Until recently, these papers formed the basis of the work reported on sounds from muscle. It took almost 40 years for any further work to be initiated.

2. Acoustic Myography studied from human skeletal muscle contractions.

The study of muscle sounds and their association with force and electromyography was started by looking at isometric contractions of the biceps muscle (Oster and Jaffe, 1980). It was found that when EMG and AMG were plotted against force

there was a linearly increasing relationship (Figure 1.1b). Thus, as the percentage of maximal voluntary contraction (MVC) increased the amplitude of the sounds produced increased. The sounds recorded from voluntary and stimulated contractions were found to be similar, if not identical. When the blood flow to the muscle was reduced no change in quality of the sound was detected, although when the temperature of the muscle was decreased there was an accompanying decrease in intensity, although no change in the frequency of the sounds. Conventionally, electromyograms (EMG) were used to monitor the force and state of the muscle, but problems were encountered when the relationship between force and EMG became complex, for example in high force fatiguing contractions, where the EMG amplitude rose even though force was constant or decreasing. Interest in AMG grew with the possibility that it reflected force more accurately than EMG and the possibility of using AMG to monitor force during contractions .

In an early review article the initial observations were detailed (Oster, 1984). During the study of chicks before and after birth, and the development of the muscle in their backs, m. latissimus dorsi, the electrical activity of this muscle was being recorded before, during and after hatching. In order to monitor the hatching activity a microphone was attached to the outside of the shell. It was noticed that while the movements from within the shell could be heard, other quite distinct noises could also be detected, sometimes as a 'click' and at others a 'rumble'. On further investigation, by removing the shell to reveal the membranous sac below, it was seen that as the chick moved there was a coincident rumble. A search through the literature found that these sounds had been reported, not for their own worth, but as an interference to the recording of other body sounds such as those of the heart.

Some other observations on the occurrence of the sounds during muscle contraction were also made. It was reported that in a comparison of the sounds from a ballerina's gastrocnemius and from an untrained individual, the sounds were much

more intense from the dancer (Oster, 1984). This suggested that the measurement of sound could be used as an indicator of the muscles operating in a particular posture. It was also found that the sounds from the soleus muscle were 10 times more intense than the signals from gastrocnemius while standing. Some rather interesting, if perhaps speculative, facts about sounds from biological sources were also reported. As water is a much better medium for the transmission of sound than air, it was suggested that the 'purring' of the gourami fish (Trichopsis vittatus) which can be heard when the fish makes violent and rapid motions associated with mating could be the sounds from the muscular contraction as they do not appear to have any other origin. It has also been found (Nelson and Gruber, 1963) that lemon sharks are attracted by sounds of frequency 20-50 Hz. Grey sharks were also shown to be attracted to intermittent low frequency signals and to recordings of fish caught on hooks struggling. It was therefore suggested that it was the muscle sounds from the fish that caused the sharks to react.

The detection of low frequency sounds from the eggs of Japanese quails before hatching was reported (Oster, 1984). The sounds emitted by the hatching eggs had been recorded using a transducer attached to the outside of the shell. The sounds started to appear about four days before hatching, and in order to see inside the shell, a portion of the shell was removed and covered with plastic. It was found that the sounds accompanied muscle movements of the chicks and it was postulated that this was a form of communication between the chicks and perhaps between other eggs.

The first heart sound also came under scrutiny. Although thought to be due to the reverberation of blood within the ventricular chambers as the walls contract ejecting blood, this sound can be heard slightly before any ejection of blood. It was proposed that the first heart sound could be attributable to the cardiac muscle contracting and causing vibrations detectable at the skin surface. By suggesting

this, the whole area of clinical measurement and the uses of AMG within this field was expanded and interest grew amongst physiologists, clinicians and engineers.

Recently, acoustic myography has been used as an indicator of force from small muscles in the hand (Goldenberg et al, 1991) in particular abductor digiti minimi muscle, which controls the abduction of the little finger. By using an electret microphone encased in foam to stop extraneous noise they recorded the AMG from the muscle during sustained fatiguing contractions at 15, 25, 50 and 75% of the maximal contraction. The amplitudes of the AMG signals at the beginning, middle and end of the contractions were compared. The subject was seated with the arm pronated. The microphone was placed half way between the posiform bone of the wrist and the head of the fifth metacarpal bone of the hand. Subjects performed a maximal contraction with the finger in 5° of abduction. The maximal contraction was determined and then the subject asked to maintain the percentage levels for as long as possible. The subject received visual feedback and the experiment was stopped when the force level fell by more than 10% for 5 seconds. Data was filtered (low pass filter, <14Hz.) to eliminate physiological tremor and the effect of it on the vibration signal. They found that during a 75% MVC, from beginning to end, there was no change in the signal measured thus it reflected the force production very clearly. However, during the 50% contraction there was a significant ($p<0.05$) fall in the root mean square of the signal from beginning to end. At 15% and 25% MVC, the acoustic amplitudes increased over time and were significantly ($p<0.05$) higher at the end compared with the beginning. The frequencies of the AMG were analysed and found to be low frequency with the main power being below 30Hz. Thus they suggested that the acoustic signal did not accurately reflect the force record over the time of the contraction. The increase in AMG throughout the 15% and 25% MVC was thought to reflect the continued recruitment of motor units, especially the large diameter fast twitch fibres, as the smaller fibres fatigue. At 50% MVC the drop in the acoustic signal was considered

to be due to the fatiguing of the larger fast twitch fibres. At a contraction level of around 50% MVC the majority of the fibres are fully activated (Basmajian and De Luca, 1985) and so the fast fatiguing units will drop out and the smaller fatigue resistant units will fire more frequently to maintain the force level. They postulated that the steady acoustic signal during the 75% contraction may be in part due to the short duration of the contraction which was terminated before any significant changes could be seen in the electrophysiology of the muscle. In conclusion they suggested that although the acoustic signal may not reflect the force level accurately, it may be dependent on the activity and recruitment of the motor units.

The surface EMG and the AMG signals were recorded for comparison from a normal population and a group that had been diagnosed as having muscle disease (Barry et al,1990; Barry and Gordon, 1988; Barry et al, 1987). The subjects were asked to lift and support weights using the biceps brachii muscle with the arm flexed to 90° and with the body supine. Signals were recorded for 20 seconds with rest periods of 40 seconds in between. They found that there was no significant difference in root mean square AMG (rmsAMG) levels between the two groups although there was a significant difference in root mean square EMG (rmsEMG) levels with the control group slope being much less steep than the group of patients. This was suggested as showing the acoustic signal being a better monitor of the force output of the muscle. The EMG signals were higher and exhibited a larger variability at each force level. By plotting rmsEMG against rmsAMG, the relationship for the control group was at a much lower level than that for the patient group. They proposed that although AMG had not proved to be useful on its own, in conjunction with EMG, it provided a clearer division between the two groups. Thus, they proposed that the accuracy of diagnosis would improve by measuring both levels.

The acoustic myogram signal was measured during voluntary isometric contractions of the quadriceps muscle (Stokes and Dalton, 1991). The subjects were seated with

the angles at the knee and hip being 90°. Surface electrodes were placed over rectus femoris together with an electronic condenser microphone and the force was measured using a strain gauge. They found that there was a close linear relationship between force and the integrated AMG signal recorded. This differed from the relationship described for other muscles, biceps brachii muscle (Orizio et al, 1989a) and erectores spinae (Stokes et al, 1988) in which at forces greater than 80% the AMG signal was reduced, even though the force and the EMG signal continued to rise. There is an optimal muscle length and hence angle for maximal force production, and it may be that the angle chosen was not the optimal angle and so the signals recorded were not maximal. In contractions of biceps brachii it was found that EMG and AMG were proportional to each other and that both exhibited a quadratic relationship to muscle force (Maton et al, 1990). It was also noted that there was a greater variabilty within the AMG signal than there was with the EMG signal. It was also found that the mean power frequency increased with EMG and AMG up to a force level of 30%MVC and then showed no further changes. With these rather contradictory reports it appears that there is no clear mathematical relationship between force, EMG and AMG although there is no doubt that increases in force are accompanied by increases in AMG amplitude. Indeed, all reports concluded that the AMG signal should be studied further and could possibly be used to study muscles when force could not be measured accurately.

Recently there has been an increase in both the interest, and the amount of work being done using AMG. Various methods were employed to detect and record the sounds, or vibrations, that were experienced during contractions of skeletal muscle. One group (Barry et al, 1992) tried to standardise recording techniques by using an accelerometer, and using the units of acceleration ($m.s^{-2}$) to describe the vibration amplitude. However, this does not clarify the results in any way, as there must be some sort of calibration procedure which will vary between laboratories. A much more meaningful interpretation, is one in which the results are normalised to the

maximal value obtained within one experiment. Thereby, the results can be directly compared rather than allowing a comparison of actual values. In the following description of the work using accelerometers, the units are given as reported, but it is easy to see the importance of normalizing the results, as only the investigators can have any comprehension of the data and its relevance.

Much of the recent work has concentrated on stimulated contractions rather than maintained voluntary isometric contractions. By stimulating the median and ulnar nerve supplying abductor pollicis brevis and abductor digiti quinti respectively, the AMG wave form and the compound action potential from the muscle was recorded (Barry, 1991a,b). The amplitudes of the waves ($5.7\pm0.6 m.s^{-2}$ and $5.1\pm0.6 m.s^{-2}$ respectively) were reproducible for each muscle and the latencies, or time from stimulation to appearance of the wave, were also extremely reproducible being 6.5 ± 2.4 ms and 7.2 ± 2.0 ms respectively. These pressure waves are due to the lateral acceleration of the muscle under the skin. The pressure waves were biphasic rising sharply from the base line, peaking, then declining slowly to cross the base line and then return to the previous level.

A detailed study of the acoustic signal during the period between stimulation and the appearance of any force record, or the latent period, was performed (Hufschmidt, 1985). It was found that during stimulation the sound wave is often preceded by a smaller wave of the opposite polarity. This initial wave was around a thousand times smaller than the main wave, and if there was very strong stimulation another small wave of the same order of magnitude was seen preceding the 'initial wave'. Shortening of the muscle is accompanied by an increase in the diameter of the muscle. The delay of muscle thickening and of force development are exactly the same (Rauh, 1922) and so it was proposed that the latent period of muscle contraction could be determined by the lateral acoustic registration. It was suggested that the initial wave could be caused by the drop in tension after stimulation and prior to force development (Rauh, 1922). This phenomenon was termed 'latency

relaxation' and would appear to correspond to the period where there is a decrease in stiffness in the muscle as cross bridges detach from the binding sites in readiness for reattachment. It also appears to tie in with the evidence from laser diffraction techniques that the sarcomere lengthening that must be the cause of this relaxation occurs in both active and completely slack fibres. The smaller wave that occurs before the initial wave was thought to be a result of the strong stimulation causing a release of calcium (Ca^{2+}) from the lateral sacs which would cause a localised contraction causing shortening in some areas resulting in a thickening of the fibres.

Stimulation of the ulnar or median nerve produces twitches of the abductor digitii minimi or adductor pollicis muscles respectively. These contractions were measured using a microphone with a frequency response of 0.1-1500 Hz attached to the skin over the motor point at the same site used for the EMG recording electrode (Barry, 1990). By stimulating the nerve, biphasic acoustic signals were recorded. These were found to appear after the electrical action potential (3.6±1.0ms for median nerve stimulation and 3.9±1.1ms for ulnar nerve stimulation) but before the force record was seen. It was suggested that the delay in the appearance of the acoustic signal is the time for electromechanical coupling to occur and further, that the acoustic signal is a better measure of this delay than the appearance of force, due to the necessity for the stretching of the series elastic component before any force is seen. It was deduced that there are two components in the sound signal, a low frequency biphasic wave and smaller higher frequency oscillations superimposed on the first. The large low frequency wave was due to the bulk movement of the muscle when it is stimulated and the higher frequency vibrations were due to the resonant vibrations of the muscle.

In a further paper (Barry, 1991a) stimulated twitches of adductor pollicis brevis and abductor digiti quinti were used to compare the twitches from concentric contractions and those under isometric conditions. These vibrations were measured using an accelerometer and the recordings were typically bi or triphasic. Preceding

this main wave there was a small dip, similar to that recorded in twitches of tibialis anterior (Hufschmidt, 1985). The waveform recorded during isometric contractions was larger in amplitude and higher in frequency than with the muscles allowed to contract freely. Isometric contractions produced waves with more cycles and with a form that was similar to those recorded from *in vivo* frog muscle (Barry, 1987). The frequencies of each waveform were slightly different although not significantly so. Isometric contractions revealed that 90% of the energy lay between 20 and 390 Hz, 70% of the energy was between 20 and 100Hz, whereas for the concentric contractions 90% of the energy lay between 2 and 330Hz, 70% between 16 and 90Hz.

Acoustic myography has been said to have many uses as a measure of the force being produced by the muscle, a measure of the activity within the muscle and an indicator of the fatigue state of the muscle. The use of AMG is still in an early stage but an indication of its potential was given when the AMG signals were used as the control signal to power a prosthesis (Barry et al, 1986). The signals from the flexor and extensor forearm muscles were detected using a phonocardiograph microphone and then amplified before being fed to the prosthetic hand. Initial trials were performed on normal individuals without amputation, and they could learn to control the hand with very little difficulty. Further tests were performed on two below elbow amputees who had no experience of the more common myoelectric prosthesis. They needed very little instruction to become competent with the hand, and very quickly, within ten minutes were able to perform quite delicate movements to pick up objects from smooth surfaces. Figure 1.2 shows the acoustic signal recorded from the wrist flexors and extensors. As can be seen there is virtually no crosstalk, or interference between the two signals, and so this allows good discrimination between the two opposing movements. Unlike the more conventional use of the EMG signal to power these prostheses, there was no need to implant electrodes or detectors, selection of a suitable site for placement of the

microphone was limited to placement over the muscle and if the choice of first site was unacceptable, it was extremely easy to replace the microphones. Also reported was the low interference factor from external sources such as doors shutting and vibrations from people and vehicles passing. This was a very positive report highlighting the benefits of acoustic myography. A report which has not yet been expanded or improved upon, and so the practical aspects are not well documented. Recently, the same group has been exploring the use of the AMG signal as a feedback signal for FES (functional electrical stimulation) in paraplegic patients (personal communication). This work has not been published because of its commercial possibilities, but could prove to be very important in further AMG work.

3. AMG studied using *in vitro* preparations.

Work done in vitro has seemed to concentrate on the mechanism of the sound production and the more mathematical aspects of the muscle sound. The first report (Brozovitch and Pollack, 1983) used frog sartorius muscle to investigate the sounds. The muscles were stimulated to shorten under loaded conditions and by placing a piezoelectric transducer beside the muscle they found that discrete bursts of sound were generated. The duration of each sound burst was in the order of 400μs. In some of the muscles used, single bursts of sound energy were detected during shortening or lengthening, while in the rest, discrete bursts were recorded throughout the contraction. The pattern of burst was repeatable within the same muscle, although they did find that some bursts disappeared, of which a further fraction remained silent and the rest produced another pattern of sound. The amplitude was not consistent and varied throughout the whole series of muscles. They concluded that shortening muscles generated sounds which could be detected from within the recording medium, which in this case was saline. In the stimulated

contractions the sounds were not continuous, but more discrete spikes. Thus, they decided that this indicated that the muscle did not contract smoothly, but rather in discrete steps agreeing with the observations by other muscle physiologists in single muscle fibres (Delay, 1981; Jacobson et al, 1981; Pollack et al, 1977) and whole muscles (ter Keurs et al, 1978,1979) who found that there were pauses during contraction when the sarcomere length remains constant. Because the muscle has a constant volume, its width must increase during shortening, therefore a stepwise change in muscle length would result in a similar change in muscle radius. It was proposed that if enough fibres changed length in synchrony then this could lead to the bursts of energy.

The mechanism of the production of the muscle sounds was studied in frog gastrocnemius muscle (Frangioni et al, 1987). The muscle was enclosed within a sound proof tank and the sounds were recorded using a hydrophone suspended in the bath pointing towards the muscle belly, 2mm from its surface. The fluctuations in pressure were recorded along with the force exerted by the muscle and the action potentials from the muscle. The muscle was stimulated via the sciatic nerve, with one tendon held rigidly and the other tendon fixed to the transducer. The muscles were held over a range of different lengths from $0.8L_0$ to $1.1L_0$, where L_0 is the length of the muscle at which the maximum tension is recorded, or optimal length. The change in the sounds were recorded as the hydrophone was moved along the length of the muscle, as the muscle was plucked with a probe and as the muscle was stimulated repeatedly at 10 second intervals to investigate fatigue. It was found that the only significant sound was at the beginning of a contraction. If the muscle was stimulated as in an unfused tetanic contraction there was most sound at the first twitch, with a decrease in amplitude at every successive contraction. During a fused tetanic contraction there were only traces of any activity throughout the contraction, although there was activity within the first 50 milliseconds of the contraction. Changes in length of the muscle showed that the pressure wave from the muscle

roughly followed the tension record of the muscle and had a peak level which occurred at $0.95L_o$ decreasing with both shorter and longer lengths. The frequency of the wave increased throughout the length increase, until L_o after which it seemed to plateau. The frequencies measured were significantly higher than other reports, at a maximal level of 100Hz. The method of stimulation lends itself to the oscillation of the muscle rather like a string and indeed the pressure wave exhibited a damped oscillatory tendency.

The pressure field produced by isometrically contracting frog gastrocnemius muscle was found to be described by the fluid mechanics equations for a vibrating sphere (Barry and Cole, 1988b). They stimulated the muscle via the sciatic nerve and varied the length of the muscle with a servomotor set to provide minimal resistance to the muscle. The muscle was suspended in between the servomotor and the force transducer, from which the length of the muscle could be altered. The muscle length was measured relative to the optimal length at which the maximum force was obtained. The vibrations were recorded with hydrophones calibrated and found to be capable of measuring in the range 1.0Hz to 30kHz. The vibration recorded had a characteristic pattern with an initial rise in amplitude and then decaying. They found that if the hydrophone was rotated about the long axis of the muscle, there were two azimuths 180° apart. At the first a maximum signal was recorded and at the other a minimum signal was recorded. Fluid mechanics were used to measure the pressure field produced by an arbitrary function of muscle movement. They report that these pressure waves were generated by lateral movements of the muscle during isometric contractions. Thus, isolated muscles in a tank radiate as a dipole, however they also acknowledge that this is probably not the case in muscles *in vivo* . It has also been found that the vibrations emitted are at the resonant frequency of skeletal muscle (Barry and Cole, 1988a, 1990). Using the same experimental setup as described previously, isometric contractions of frog gastrocnemius were investigated. It was found that there were two types of movement, a slow movement due to the lateral

expansion of the muscle axially and faster movements superimposed on the first signal. It was proposed that the fast AMG oscillations corresponded to the resonant frequency of the muscle.

4. The study of fatigue and AMG.

Changes in the sounds from muscle have been measured up to exhaustion (Orizio et al, 1989b) in human muscle. Again the biceps muscle was used in healthy subjects and they were asked to maintain percentages of their maximal contraction for as long as they were able. EMG and AMG were both recorded and converted to the rectified integrated signal (iAMG and iEMG). They found that at the beginning of the exercise both the rectified integrated AMG and EMG signals increased linearly with time. There was then a variable period (253 seconds for the 20%MVC to 0 seconds for the 80%MVC) of constant amplitude after which there were changes in iAMG signal level. The iAMG during the 20%MVC increased until the end of the contraction, whereas at 40%MVC the level fluctuated and for 60 and 80%MVC the levels fell. The iEMG signal rose throughout the contractions, thus they concluded that, at least in the higher force contractions, the iAMG signal reflected the rate of fatigue onset within the muscle.

The effects of fatigue on AMG has been studied using evoked muscle twitches (Barry et al, 1992). Subjects maintained contractions of the first dorsal interosseus muscle at 30, 50 and 70% MVC for as long as possible. During short, 5 second, rest periods the muscle was stimulated percutaneously via the ulnar nerve. Hand temperatures were maintained at 32°C using a silicone heat pack if necessary. The force measured was that exerted by abduction of the second digit. The subjects were asked to contract maximally and from this percentages of the maximal contraction were calculated. The subject maintained contraction levels of 30, 50 or 70 % MVC for periods of 25 seconds and then there was a 5 second rest period

during which the muscle was stimulated to evoke a twitch. The subject then voluntarily contracted to the percentage level again. When they could no longer hold the percentage MVC, they were asked to contract maximally, this was maintained for three more recording periods and then the test was ended. It was found that the subjects showed reductions in twitch potentiation and in force produced during each twitch. This was accompanied by parallel decreases in the amplitude of the AMG wave although the compound action potential (CAP) did not change in amplitude. Thus, they felt that the AMG wave showed a higher degree of correlation with force and the fatigue state of the muscle than the CAP wave.

5. The general structure and function of muscle and the central nervous system.

Skeletal Muscle

The muscles attached to the skeleton which cause limb movement are termed skeletal muscles. These muscles have a fibrous appearance and this structure can be dissected down to the individual fibres in which the constituent molecular chains lie. Muscle fibres are multinucleate cells varying in length from a few millimeters to several centimeters. The fibres have diameters in the range 50-60μm, and in the most simple arrangement of muscle run the whole length of the muscle, although in large muscles it is quite common to find two or more fibres in series. In order to increase the force production of the muscle, the arrangement of the fibres can be changed to allow more fibres without increasing the bulk of the muscle, as in pennate muscles. This arrangement often reduces the range of movement of the muscle, and so a compromise is reached. Increasing the number of muscle fibres and, therefore, the size, allows the range of movement to remain and the force production from the muscle is increased. The whole muscle is arranged in bundles of muscle fibres or fascicles covered with a fibrous sheath called the perimysium.

Each fascicle is arranged in smaller bundles called myofibrils which are the muscle cells and within the cytoplasm of these cells are the myofilaments, myosin and actin. The regular arrangement of these filaments gives rise to the characteristic striated appearance of skeletal muscle. The thick myosin filaments interdigitate with the thin actin filaments. The myosin is composed of bundles of short molecular chains with specialized endings or head groups. These head groups protrude from the main filament allowing the head groups to attach to the binding sites which are present on the actin, which has a double stranded helical array. There are two other components associated with the actin chain, tropomyosin and troponin. Tropomyosin has a filamentous structure and lies over the binding sites on the actin filament. Troponin is a protein attached to the tropomyosin filament, and has a calcium binding site. When calcium binds to troponin there is a conformational change which pulls the tropomyosin away from the binding sites, leaving them available for the myosin head groups.

The filaments are arranged in sarcomeres, the contractile units of skeletal muscle, with the overlap of the filaments causing differences in the reflection of light when viewed under the light microscope. A schematic diagram of this is shown in figure 1.3. The A-band is the extent of the myosin filaments, the I-band is the extent of the actin filaments and the Z-lines, which are the areas where the actin filaments are held together, show the extent of the functional unit, the sarcomere.

Surrounding the myofibrils is the sarcoplasmic reticulum. This is arranged in a lace like structure around the myofibrils with sacs at either end of the myosin, at the junction between the A and I bands. The sacs are arranged around a transverse tubule (T-tubule). These T-tubules are in direct connection with the membrane surrounding the muscle, the sarcolemma. As the muscle is stimulated to contract via the nerve, either from the CNS or by artificially stimulating the nerve, the action potentials spread across the muscle membrane surface from the neuromuscular junction and invade the T-tubules. This causes a depolarisation of the lateral sacs

which causes them to release their stores of calcium. This diffuses into the muscle cell to the filaments and binds to the troponin molecule, revealing the binding site on the actin filament by pulling away the tropomyosin molecule. Contraction will continue if there is also energy for the movement of the myosin head group. Adenosine triphosphate (ATP) binds to the head group, is split into a highly energetic form, ADP-P_i (P_i = inorganic phosphate), which powers the movement as it splits, and is lost from the head group as ADP and P_i.

Energy for the contraction as stated previously comes from the splitting of ATP into the constituent parts ADP and inorganic phosphate. Very little of the ATP required is actually stored in the muscle and it is the rephosphorylation of the ADP that provides the ATP. Creatine phosphate, of which rather more is stored within the muscle, is the main source of P_i start of the contraction. As the muscle continues to work it changes to another source of ATP. Fatty acids and glucose are the fuels for oxidative phosphorylation and glucose is the source for anaerobic glycolysis. Within the cells themselves are large stores of glycogen which can be converted to glucose if required. The mitochondria required for oxidative phosphorylation are numerous around the periphery of the fibre and are gathered around the area close to the motor end plate. The capillaries are in close connection with the filaments and the diffusion of oxygen is assisted by the presence of myoglobin. This has a very similar structure to haemoglobin but with a higher affinity for oxygen at any given partial pressure of oxygen (P_{O_2}).

The type of metabolism depends on the type of muscle fibres that are activated and whether the oxygen supply is keeping pace with the demand. Basically, there are 3 types of muscle fibre, fatigue resistant slow twitch fibres or type I, fatigue resistant fast twitch fibres or type IIa and fatiguable fast twitch fibres or type IIb. The type I fibres are red in appearance, relying solely on oxidative phosphorylation and if there is an adequate blood supply, and therefore oxygen, these fibres can contract for extended periods. The IIb fibres are more white in appearance, and rely on

glycolysis for their energy source. They are not able to maintain their contraction for as long as the type I fibres due at least in part to the build up of the metabolic product, lactate. The type IIa fibres are intermediate having both glycolytic and oxidative capacity. As the type I fibres are lower force and slower to contract than the type IIb and IIa, the proportions of these fibres within a muscle determine how fatigue resistant it is, how fast it will contract on stimulation and what the maximal force able to be produced is. Therefore, postural muscles have a higher proportion of type I fibres than muscles which are used more intermittently and produce higher forces.These muscles tend to have more type II fibres. The table (Table 1.1) below summarises the fibre type content from some human muscles. This data comes from cadaverous tissue but myosin ATPase activity remains unaltered for at least 24 hours.

Table 1.1: Table showing the fibre type content of some human muscles. Data was taken from post-mortem tissue. (Johnson et al, 1973).

Muscle (location of sample)	% Type I	% Type II
Biceps brachii (surface)	42	58
(deep)	51	49
Triceps brachii (surface)	32	68
(deep)	33	67
Adductor pollicis	80	20
1st Dorsal Interosseus	57	43
Rectus Femoris (medial head)	42	58

Isometric Contractions

In isometric contractions the length of the muscle remains constant and it is the force generated by the contractile mechanism that is being measured. In an *in vitro* condition when the muscle is activated the action potential measured occurs almost instantaneously and lasts for around 5ms, whereas the tension takes longer to develop, around 5-10ms and is also longer lasting, with peak tension taking from 10-100ms to develop. If the muscle is repeatedly stimulated a summated contraction develops, and if the stimulation frequency is high enough, a smooth tetanic contraction will be seen. The tension developed during a tetanic contraction may be 10 times the force seen during a single twitch. The sequence of events producing the force is the same for a twitch as for a tetanic contraction. The difference is that instead of the cross bridges on the myosin heads being stimulated to attach and then detaching when the stimulation is removed, the cross bridges attach and then continue to develop force until the stimulation is removed when the muscle will relax. Thus, because the muscle does not relax in between stimulations a greater force can be developed. Voluntary contractions are different because the number of contracting fibres is under central control and it is only at high forces, 50% MVC and above, that all the fibres will be active (Basmajian and De Luca, 1985). Thus, it is important to remember the differences when comparing results from stimulated and voluntary contractions.

The Motor Unit

The definition of the motor unit is the innervating nerve fibre and the muscle fibres that are supplied by it. Depending on the type of muscle under investigation, the number of fibres supplied by the nerve varies. If the muscle is a small, fine control muscle there are a small number of fibres innervated by one nerve fibre and if the muscle is a large high force muscle, such as quadriceps, a larger number of muscle

fibres are supplied by any one nerve fibre. This is shown diagrammatically in figure 1.4. The two motor nerve fibres shown leaving the anterior horn of the spinal cord innervate the muscle fibres. Each motoneurone innervates several muscle fibres and no muscle fibre is innervated by more than one motoneurone branch. In reality, the fibres from the two motor units would be homogenously mixed within the whole muscle, rather than the two separate areas as shown here.

Relationship between muscle and the central nervous system.

Voluntary contractions are under the control of the central nervous system via neurones that have their cell body within the ventral horn of the spinal cord. These fast conducting neurones, α motoneurones, leave the ventral horn of the spinal cord forming a spinal nerve, and travel to the muscle as a bundle of neurones, a peripheral nerve. At the muscle, the nerve branches to innervate muscle fibres. This, as previously described, is termed the motor unit. At the end of the nerve fibre there is a specialised ending, the nerve terminal, that contains acetylcholine, the neurotransmitter. The neurotransmitter is manufactured in the cell body, packaged into vesicles, and transported along the axon by neurofilaments and microtubules. These vesicles are released in response to nerve stimulation. This is only one side of the neuromuscular junction, the events that happen subsequently have been described previously and initiate muscular contraction.

Recording of muscle activity.

The action potentials from the active motor units can be recorded either at the skin surface using surface electrodes or from electrodes within the muscle introduced via a needle. This composite signal is the electromyogram, or EMG. The EMG is the algebraic summation of all the motor unit action potential trains from all the active motor units within the area of the electrode.

Within the EMG signal there are many features that can be observed and subsequently analysed. Recording from the surface, the EMG is a very complex record that requires detailed decomposition. Electromyography is the most common way of studying muscle and muscle generated electricity has been documented since the 17th Century when Francesco Redi, an Italian, deduced that the shock from an electric ray fish was muscular in origin. A Frenchman, Du Bois-Reymond (1849) was the first to report the voluntarily elicited electrical signals from human muscle. He found that if he immersed one hand in saline and contracted it while gripping a metal bar connected to a recording device, he could get minute deflections. Realizing that the impedance of the skin was in fact reducing the signal, he induced blisters on his hands, removed the skin and placed the open wounds in contact with the saline. This time he got much larger deflections and the results were repeatable. It was not an easy recording procedure at this time and required careful preparation before any work could be done. It was in the early part of this century that the clinical work started with introduction of the needle electrode (Adrian and Bronk, 1928). Many studies were carried out with the quality and availability of the equipment improving, and there was increasing use of electromyography clinically. The real changes were seen with the production of the electrically stable silver/silver chloride electrode in the 1960's. As with many studies in clinically related science, the real interest comes when an area of work ceases to be purely academic and shows the potential to change and improve the life of the patient. During the 1960's

a group of engineers in the Soviet Union, used the electrical signals from the forearm muscles to control a prosthesis (Basmajian and De Luca, 1985). This created wide interest in the world as people rushed to use this new technique. This is being mirrored today with the growing interest in AMG and its possible uses.

Muscle Fatigue

Fatigue is used to describe the condition of reduced force from muscle and can be thought of as peripheral fatigue with causes distal to the motor end plate or central fatigue with causes proximal to the motor end plate. Peripheral fatigue is thought to be a result of metabolite levels changing, for example increases in potassium, hydrogen and lactate ions or decreases in blood supply and therefore oxygen, or neuromuscular fatigue, where the transmission of nervous impulses is no longer able to be maintained. Central fatigue is thought to have a wide range of causes and includes motivational problems in subjects. Fatigue is often studied during sustained isometric contractions. These types of contraction produce a decrease in force output, an increase in muscle tremor and gradually increasing localised pain. This localised muscular fatigue along with the accompanying EMG signal have been recorded from many muscles. Both the signal level and the frequencies have been examined. An early study (Cobb and Forbes, 1923) noted that there was a shift towards the lower frequencies from the beginning to the end of the contraction. They also recorded an increase in the EMG amplitude over the same contraction and this has been confirmed by many investigators (Stephens and Usherwood,1975; Stulen and De Luca, 1978; Hagberg, 1981). These two observations have been linked (Lindström et al, 1970; De Luca, 1979) and it was proposed that the explanation lay in the fact that during a sustained contraction the low frequency components of the EMG signal increase and therefore more of the EMG signal passes through the low pass filtering effect of the tissue. Although this

is an important part of the detection of the EMG signal clearly increases in EMG are rely more heavily on the recruitment and firing rates of the motor units active. This brings up many other issues which must always be taken into account when considering the detected and recorded EMG signal. It is always dependent on themotor units active, the type of electrodes used, their array over the muscle, the thickness of the subcutaneous tissue, the level of contraction, and a number of other less important factors.

It has been proposed that motor unit recruitment, motor unit synchronisation and changes in the conduction of the muscle fibres, account for the increase in EMG and downwards shift in frequency. As a contraction progresses more motor units are recruited and it would seem reasonable that this would cause an increase in the signal recorded (Edwards and Lippold, 1956; Maton, 1981). However, increases in amplitude and shifts in frequency have been seen during 80% of the maximal contraction in first dorsal interosseus muscle (Merletti et al, 1984) which is known to have no further muscle fibre recruitment at this level (Milner-Brown et al, 1972, 1973b). Synchronisation of motor units, or the tendency for them to fire simultaneously tends to occur more frequently as the contraction progresses (Lippold et al, 1957). Therefore, as the shift in frequency is most pronounced at the beginning of the contraction (Basmajian and De Luca, 1985) synchronisation would not appear to be the main cause of this change in frequency.

If the conduction velocity of the action potentials in the muscle decreased, the time duration of the action potential would increase, due to the increased time to traverse the separation of the electrodes. Thus, this would be seen as an increase in the low frequency components of the signal and a decrease in the high frequency.

During sustained contractions, a build up of metabolites causes a decrease in force. The motor end plate does not fatigue in a normal contraction and this was shown by examining the force produced during sustained isometric contractions (Merton,

1954). It was shown that when maximal contractions of the adductor pollicis were used, the force fell with time. The force could not be recovered when the muscle was stimulated via the ulnar nerve, and as the action potential from the stimulation remained unchanged throughout the experiment, it was deduced that the fatigue seen was peripheral rather than central, and not due to a lack of effort from the subject. It was also found that if the muscle was not perfused after the experiment the force could not be recovered, which agreed with the fatigue being a peripheral effect. It was also found that during voluntary contractions to fatigue there was no change in the amplitude of the M-wave thus the decline in force was not due to neuromuscular blocking (Bigland-Ritchie et al, 1982).

In a completely different situation, during stimulated contractions, at high stimulation rates, it is the muscle end plate that fatigues (Kugelburg and Lindegren, 1979). Rat tibialis anterior muscles were used and enzyme levels were measured within the motor unit stimulated. It was found that at high stimulation rates, there was failure of both the electrical and mechanical response with time, whereas, at low frequencies of stimulation there was a decrease in the twitch and tetanic force with time but no change in the action potential recorded. It was concluded that endurance depended on a chain of events including the neuromuscular junction and the excitation-contraction coupling mechanism and under aerobic conditions, was matched to the oxidative enzyme capacity.

The metabolite levels were measured during fatiguing contractions using nuclear magnetic resonance (Dawson et al, 1978). It was found that the force level was closely related to the levels of metabolites within the muscle, especially the rise in inorganic phosphate. Thus, it was proposed that fatigue was at least partly due to a build up of metabolites within the muscle causing a disruption of the contraction process and the energy supply to the fibres.

7. Analysis of EMG and AMG

The EMG signal is the sum of all the electrical activity of the motor units during a contraction. The series of action potential waves from all the muscle fibres active at the time of measuring can be recorded either at the skin surface or using electrodes within the muscle. It is an extremely complex signal that is affected by the instrumentation, the central nervous system, and the properties of the muscle itself. In recording the EMG, two electrodes are commonly used, being termed bipolar, the signal passes first one and then the other, as a differential recording.

Amplitude of the action potentials is dependent on the diameter of the fibre, the distance from the active fibre to the recording electrode and the filtering properties of the electrode. The amplitude increases with an increase in fibre radius and decreases as the distance from the detection electrodes increases.

It has been common practice to position the electrodes over the motor point, the surface projection of the centre of the innervation zone. Presumably this procedure arose from the fact that the motor point is the source for the spread of excitation across the muscle. However, it is for this very reason that it is not always the best place for positioning the electrodes. Around the innervation zone the activity is only beginning, and recording conditions are better at a distance from this point. The signal is extremely variable as the orientation of the electrodes across the fibre changes. This variability was investigated (Basmajian and De Luca, 1985) in rabbit gastrocnemius, by keeping the detection electrode surface in line with the fibres and moving it to various locations along the muscle, near the achilles tendon, between the tendon and the innervation zone, over the innervation zone and between the innervation zone and the origin of the muscle. The ankle was moved to different angles and this was found to alter the compound action potential recorded as well as the position of the electrodes. However, when the electrodes were firmly attached or had external pressure applied, the most unreliable position was found to be over

the innervation zone. The conclusion from the results was that the preferred location of the electrodes was between the innervation zone and the far tendon.

The recording of the EMG signal is altered by all of these factors, and obviously this must be borne in mind when interpreting the data.

Analysis of the EMG signal is complicated by many factors, the most important being that the EMG signal is a rapidly changing one. Perhaps the most meaningful technique to reduce the data into something more meaningful is the calculation of the root mean square (rms) of the data. This operation is performed, as the name suggests, by squaring the data sampled at intervals, taking the mean level, and then calculating the square root of the mean squared data, according to the equation :

$$\text{RMS } \{m(t)\} = \left(1/T \int_{t}^{t+T} m^2(t)\, dt\right)^{1/2}$$

Where **m** is the datum point,

t is the starting time and,

T is the total time interval

The rms value has not been widely used in the past, but due to the introduction of computer analysis and analogue chips that will perform this operation it has become a much easier form of analysis than previously. It also has advantages over the other types of analysis, rectified integrated (RI) and smoothed rectified, taking into account firing rate, firing frequency, synchronicity and chance superimposition with more completeness than other methods. These other techniques have been widely used without much thought for their methods and the significance of the results. For these reasons they have often been used inappropriately and much significance drawn erroneously from the results. In preliminary studies, both the

root mean square and the rectified integrated values were calculated. It was found that there was no significant difference ($p>0.05$) between the values and the types of relationships seen with force, and so it was decided to use rms values.

The root mean square of the data represents the power of the total signal within the data being analysed.

Frequency Analysis of the EMG and AMG signals.

Complex signals such as EMG and AMG can be thought of as being composed of sine waves of different frequencies, superimposed. The signal can be decomposed into its constituent sine waves and the frequencies of each calculated. Depending on the type of signal the relative importance of each of the sine waves can be determined. Obviously if a signal is simply one frequency this is analysed as having this frequency as its dominant component. As other waves are added the relative weighting will change to accommodate the effect of these other frequencies. The relevant technique is called Fourier analysis, and most commonly the Fast Fourier Transform (FFT) is used for data. This is just a simplified version which allows data to be manipulated much more quickly, and more easily than with the wide array of more complicated Fourier transforms (Bergland, 1969).

Typically EMG has a wide frequency band, ranging from 0 to 400Hz depending on the type of muscle being investigated (Basmajian and De Luca, 1985). AMG has a much narrower band, between 0 and 30 Hz (Rouse and Baxendale, 1991;Barry 1990). Another parameter used to describe the frequency plot is the median frequency. This is defined according to the equation below:

$$\int_0^{fmed} s_m(f)\,df = \int_{fmed} s_m(f)\,df$$

Where f_{med} = median frequency

∞ = infinity

S_m = area under the curve

Thus, the median frequency is the frequency at which the area from zero to the median frequency equals the area from the median frequency to infinity. In practice, it is more often calculated in between limits when the frequency plot is not well defined at the upper frequencies.

The EMG signal is the activity from all the active muscle fibres at any point. A decrease in the firing rate of the motor units will result in a shift of the power density spectrum to lower frequencies. A change in the shape of the motor unit action potentials will also result in a change in the FFT of the wave shifting the emphasis to different frequencies as different sine waves are fitted to the signal and will therefore result in a change in the power spectrum. A waveform with a longer duration will be recorded as having a lower frequency than one with a shorter time duration. As the conduction velocity decreases, as would be expected during a sustained contraction, the time duration of the waveform will increase due to the increased transit time across the electrodes. Thus during a sustained contraction the low frequency components would be expected to become more significant and there would be a simultaneous decrease in the high frequency components.

Within the calculation of FFT, there is a factor taking into account the probability of repeated firing of the same motor unit, synchronisation. This reduces the effect that this would have on the power spectrum, so if there is an increase in the synchronisation of motor units or an increase in the regularity of firing of any one motor unit, then this will increase the power spectrum at the frequency of the waveform. An increase in this type of activity will result in an increase in the power at the lower frequencies.

Figure 1.1a : Simultaneous recordings of EMG and AMG from the orbicularis oculi muscle. EMG is the upper trace for both sets of traces and AMG is the lower trace. The upper panel (i) shows records from opening and closing of the eyelid and the lower panel (ii) shows records with only slight movement. (Gordon and Holbourn, 1948).

Figure 1.1b : Graph showing microphone output during isometric contractions of biceps brachii while supporting weights at a 90° angle between upper and lower arm. (Oster and Jaffe, 1980).

Figure 1.1a

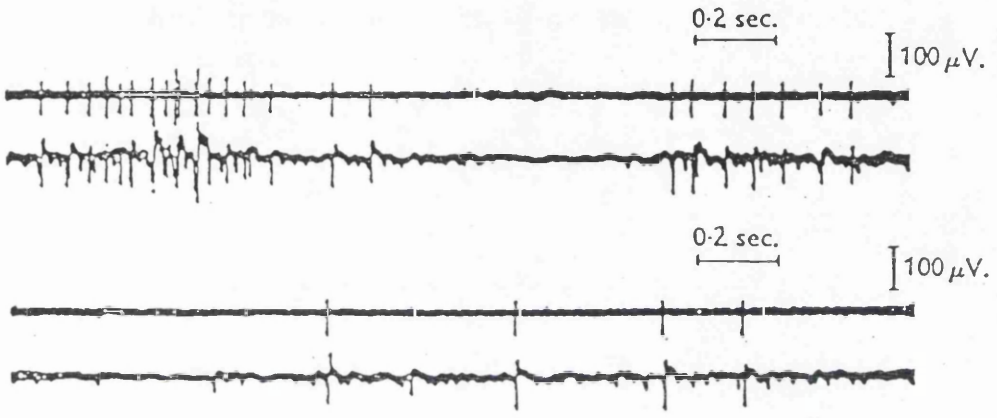


Figure 1.1b

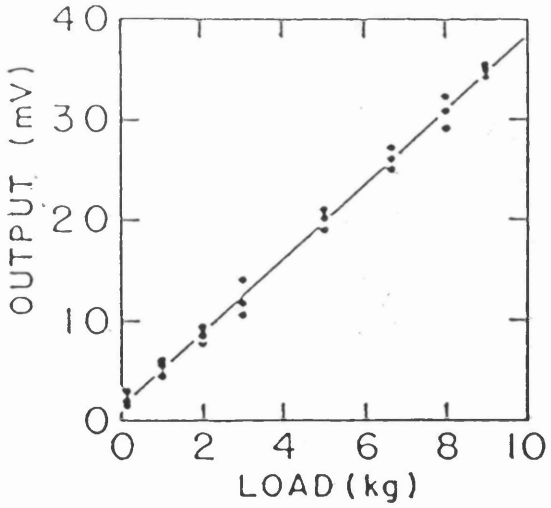


Figure 1.2 : AMG records from anterior and posterior surfaces of the lower arm during wrist extension and flexion. (Barry et al, 1986)

Figure 1.2

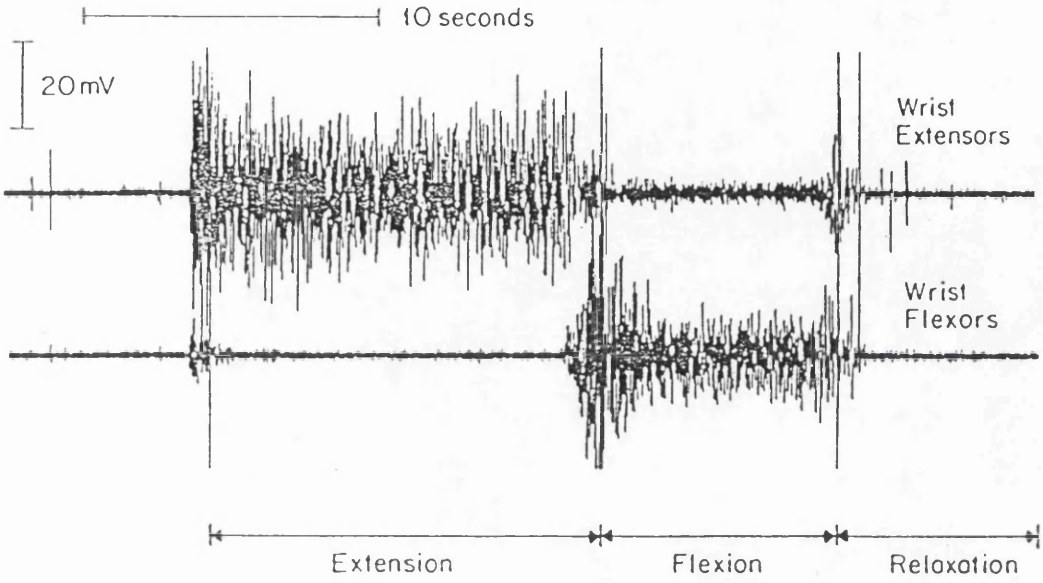

10 seconds
20 mV
Wrist Extensors
Wrist Flexors
Extension
Flexion
Reloxation

Figure 1.3 : Schematic diagram of the arrangement of myosin and actin fibres within one sarcomere of skeletal muscle. (Vander, Sherman and Luciano, 4th Edition Human Physiology.)

Figure 1.3

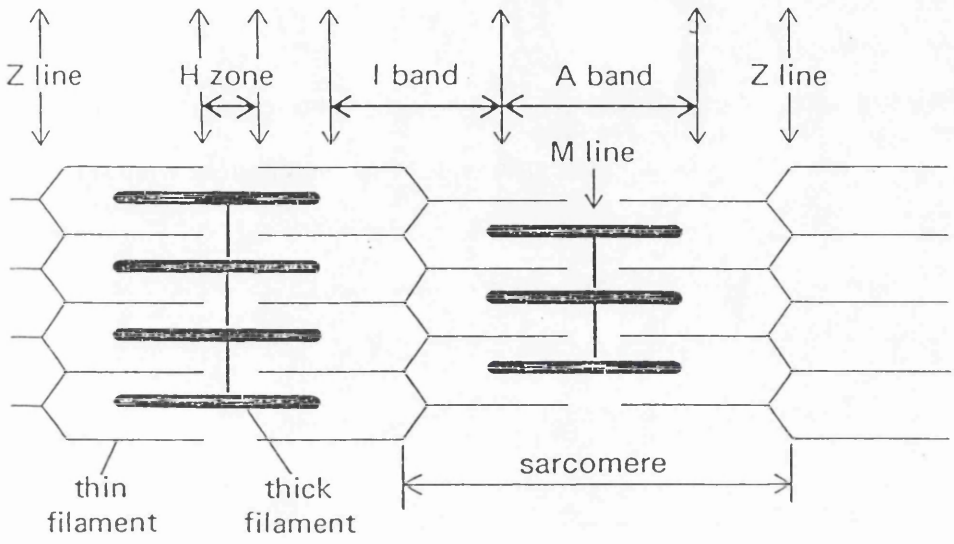
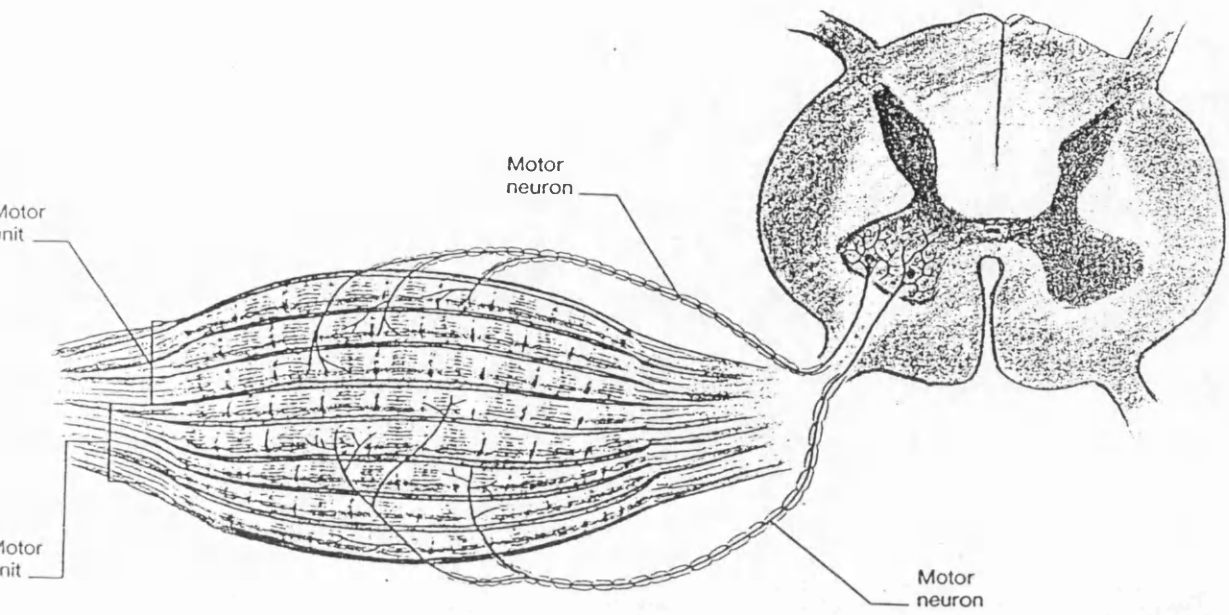

Z line
H zone
I band
A band
Z line
M line
thin filament
thick filament
sarcomere

Figure 1.4 : Schematic diagram of motoneurone innervation of skeletal muscle, and the arrangement of motor units within a muscle. (Vander, Sherman and Luciano, 4th Edition Human Physiology.)

Figure 1.4



MATERIALS AND METHODS

Materials and Methods

Subjects

All subjects were young (20-45 years) males and females, healthy, with no known history of neurological disorder. They were all fully informed of the procedures involved and were told that they could withdraw from the experiments at any time if they felt discomfort, pain or did not want to continue. The exact subject data for each experimental protocol is included at the start of each section.

Ethical Committee

Ethical committee permission for all the experiments was sought and obtained.

Equipment

During the experiments three variables were measured. These were force, EMG and AMG. Within some of the experiments, limb angles were also measured to monitor the position of the subject throughout the procedures.

Force

Force was measured with a load cell (RS 645-811). This was fixed securely to a metal post, which was in turn clamped to the bench. The whole force system was extremely rigid and was unlikely to move during the experiments. Recording from biceps and triceps, the transducer was adjusted to the height of the subject's wrist so that the subject pulled directly in line with the load cell. Recording from first dorsal interosseus and adductor pollicis, the transducer was placed so that either the finger or the thumb pulled on the transducer. During the experiments force was

measured in terms of percentages of the maximal contraction made, the absolute force was subsequently calibrated for by suspending weights from the load cell.
The quadriceps experiments were performed using an isokinetic dynamometer (Merac, Universal Ltd.)(Figure 2.4). The force exerted was measured as Newtons. During the calibration procedure of the machine, before each new subject, the weight of the limb was calculated and the effect of gravity on the force exerted was calculated, and accounted for within the force measurements recorded.

EMG

Surface EMG was measured using standard electrodes. These were small pincer like electrodes that sit in the upper skin layers (Figure 2.1a). The long axis of the muscle was identified and the electrode positions were marked a suitable distance apart along this axis, in line with the orientation of the muscle fibres between the innervation zone and the distal tendon. The skin was cleaned with alcohol, rubbed vigorously to abrade the skin slightly and then small amounts of electrode gel were applied over the electrode positions. Three electrodes were used, two recording electrodes and one earth electrode. The typical recording electrode separations are shown in table 2.1. The EMG signal was either pre-amplified (x1000) and passed through an opto-isolator or passed through a EMG isolator (Digitimer Ltd., NL) and an AC pre-amplifier (Digitimer Ltd.,NL104). The signal was then filtered (>10Hz, <500Hz) (Digitimer Ltd, NL12).

Table 2.1

Muscle	Electrode Separation
Biceps Brachii	5cm
Triceps Brachii	5cm
First Dorsal Interosseus	1cm
Adductor Pollicis	2cm
Rectus Femoris	10cm

AMG

The acoustic myogram was recorded using an accelerometer (Entran 125-100G) sensitive over a range of frequencies, 0-2kHz (Figure 2.1). The calibration curve for the accelerometer is included in the appendix. An assurance of linearity down to below 10Hz was given by the manufacturers although this is not shown on the graph. The accelerometer was used to measure the relative movements of the skin and therefore the vibrations of the muscle underneath. The accelerometer was fixed to the skin over the muscle using double sided sticky tape.

The positioning of the accelerometer was decided after comparison with other experimenters' methods, and some initial observations of my own. The accelerometer were placed over the midpoint of the muscle belly. This was a convenient position, allowing the EMG electrodes to be placed comfortably around it, the muscle curvature was less extreme for most of the muscles investigated and it was therefore easier to obtain good contact with the skin. This was also the position most favoured by other workers and therefore, to enable direct comparison of results this appeared to be the best position. It was also found, in some preliminary tests (Rouse, M.E.,1988) that recording had no one best position between all subjects, although within individual subjects, there did appear to be 'loudest' points at which recording seemed to be best. For these reasons, an arbitrary position, the

centre of the muscle over the belly, was thought to be the best place for the recording equipment.

The data was filtered (>5Hz, <300Hz) (Digitimer Ltd., NL125) and amplified (AC/DC amplifier, Digitimer Ltd, NL 106).

Data Retrieval

To allow analysis of the data at a later date the force, EMG and AMG data was recorded and saved on disk. The data was digitised and sampled at the appropriate rate (EMG: 1.2kHz, AMG: 700Hz) within a commercial data analysis package (Spike 2, Cambridge Electronic Design), operated using a personal computer (NEL) and the data stored on disk.

Protocol for experiments

The protocol was, in essence, identical for all the experiments and will be described in detail in this section. All variations will be fully set out for ease of reference, However in each results section, a brief summary of the exact experimental format will be described.

Isometric Contractions

This protocol applies to all the experiments from muscles **Biceps Brachii, Triceps Brachii, First Dorsal Interosseus and Adductor Pollicis.**

Force, EMG and AMG from the muscle investigated was recorded. The subject was asked to get into position for the experiment. For biceps and triceps brachii this was with a 90^{o} angle between upper and lower arm. The arm was maitained by the subject in this position, and the band from the load cell (RS 645-811) placed around the wrist. The load cell for biceps recording (figure 2.2a) was placed in

front of the subject beyond their arm level with the wrist so that the subject could pull, flexing the elbow. This differed for triceps recording (figure 2.2b), in that the load cell was placed directly in front of them so that they could push on the band, away from them. To record from First Dorsal Interosseus the hand was placed palm down with the load cell on the side of the little finger (figure 2.3a) so that pulling away from the load cell used this muscle and for adductor pollicis (figure 2.3b) the hand was placed palm upwards with the transducer at the side of the thumb, so that pulling with the thumb produced a force record.

The subject was asked to contract the muscle in question and the midpoint of the muscle was identified and marked. The contact sensor or accelerometer was placed over the muscle and secured. The EMG electrodes were placed along the long axis of the muscle.

A series of contractions were then initiated after a period of familiarisation with the equipment. Three maximal contractions were made by the subject holding for 5 seconds for each, with a rest period of at least 1 minute in between each one. If no substantial difference was found between them, the largest contraction was taken as the maximum and percentage levels (20,40,60 and 80% of maximal) calculated from it. If the 3 maximal contractions were not within 2% of each other, further contractions were made until a stable maximum contraction was obtained.

Further contractions were made in an ascending (20,40,60,80 and 100%) and a descending (100, 80,60,40,20%) series. Each contraction was held for 5 seconds with a rest period of 30 seconds between each, with visual feedback of the force level required. This sequence was repeated 3 times.

The subject was then asked to perform a slow ramp contraction. The contraction was performed over approximately 50 seconds, allowing 25 seconds to reach the maximum force and 25 seconds to return to zero force. The subjects were asked to perform this as smoothly as possible and no visual feedback of force was given. This sequence was repeated twice.

The final contraction was at 75% of the maximal force. The subject was asked to maintain this force for as long as possible in an attempt to fatigue the muscle. The contraction was stopped either voluntarily by the subject or when the force dropped below 70%MVC.

Isometric contractions of *Rectus Femoris*

The contraction of the rectus femoris, extending the lower leg at the knee joint, was measured using the isokinetic dynamometer (Merac, Universal Ltd.) set in isometric mode (figure 2.4a). All the force data were recorded on line via the integral computer system and software. EMG and AMG data were recorded as previously described with the arrangement shown in figure 2.4b. Before the subject was positioned in the chair, the machine was calibrated internally for the force measurements. The subject was then positioned in the chair and the seat and the length of the rotating arm adjusted until the bony processes of the knee were in line with the axis of rotation. With the subject sitting in the chair, both the fully extended and fully flexed positions were calibrated. The weight of the limb in the fully extended position was also calibrated, and included in the calculations of force performed by the dynamometer. The fully extended position was taken as 0°, and angles of flexion were measured relative to this. It should be noted that this is not the standard system for angle measurement which is usually taken as 0° at full extension moving to 90° at flexion.

Isometric contractions at different leg angles towards full extension.

The program controlling the dynamometer was set so that in a series of contractions, four different leg angles were experienced. These were 90°,70°, 50° and 30° towards full extension. The standard posture adopted by the subjects was a relaxed one, with the arms folded across their chests. No restraints, other than the immobilisation of the leg under investigation, were used as this was thought to

encourage the subject to push against them, recruiting other muscle groups and obtaining false force readings. The subjects employed submaximal contractions to familiarise themselves with the recording procedures. Once the subjects were warmed up and familiar with the protocol recording started. During recording the subjects were asked to give a maximal contraction at each position. In one run of the experiment, the subject started with leg fully flexed, then at the audible signal given by the computer, moved to the first position and contracted maximally. They relaxed at the next audible signal and moved back into a fully flexed position. At another signal they moved to the next position again contracting maximally. Generally, 1 minutes rest was given in between each contraction. This pattern continued until the four contractions had been performed. The subject then relaxed fully, although still seated in the chair, for at least five minutes before being asked to repeat the process. The subject was asked to perform this series of contractions 3 times.

Contractions at percentages of the maximal contraction.

The second part of the experiment was isometric contractions at one leg angle. At an angle of 70°, the subjects were asked to perform contractions at 25, 50, 75 and 100% of the maximal contraction recorded previously at this angle. The subject was given visual feedback from the force record. At the audible signal the subject moved into position and increased the force exerted until it corresponded to the 25% level. They maintained this for 5 seconds after which time another audible signal was given and they relaxed. After 30 seconds rest, they moved to the position again and exerted 50%, held for 5 seconds and then relaxed. This continued for the remaining two force levels, the subject was given 5 minutes rest and then this sequence was repeated.

Ramp contractions at an angle of 70°

The final set of experiments were to investigate ramp contractions. Again the angle was set to 70°, and the subject moved to the angle at the audible tone, exerted minimum force which was increased steadily up to the maximal force that could be exerted. Due to the limitations of the system, the decrease in force from maximal to zero could not be recorded. This ascending ramp contraction sequence was repeated twice to end the series of contractions.

Notation

The units of force in all the experiments was Newtons (N). The EMG signal was recorded as Volts (V) and then reduced to the raw data level taking into account the amplification involved and this reduced the signal to millivolts (mV). The AMG signal was also recorded in Volts and similarly reduced to millivolts taking into account the amplification.

This differs from some of the published work from other groups using the accelerometer to record the vibrations. In this work the accelerometer was calibrated to certain movements and speeds of movement, with the AMG signal measured in $m.s^{-2}$. It was decided not to adopt this procedure as the accelerometer output was in millivolts originally. The larger and faster the movement resulted in a greater output from the accelerometer and it was decided that this measure was enough without further calibration.

Statistics

Results were presented as raw data, or as the mean and standard error of the mean. The Student's paired t-test was used to compare data. Differences with a probability level of $p < 0.05$ were designated significant. Regression equations were calculated and are shown in the legend corresponding to the appropriate graph.

Figure 2.1a : Photograph of the accelerometer and small clip electrodes used to record AMG and EMG, respectively.

Figure 2.1a

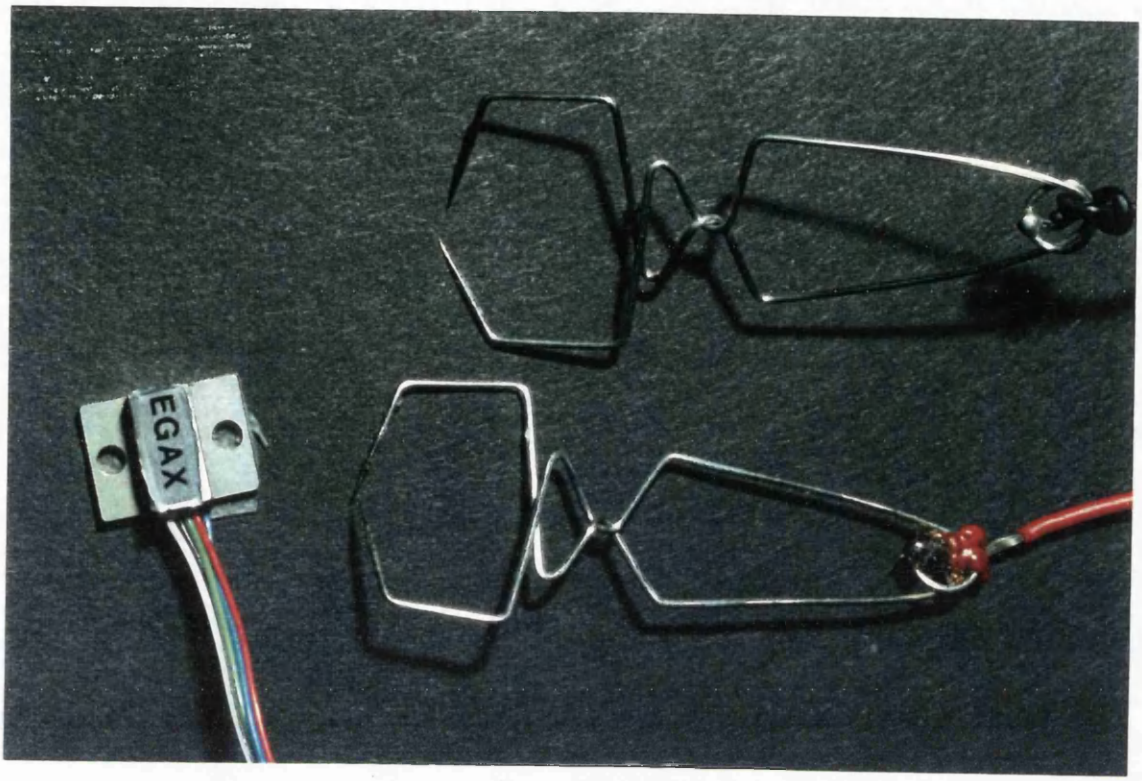


Figure 2.2a : Photograph showing the recording arrangement during contractions of biceps brachii.

Figure 2.2b : Photograph showing the recording arrangement during contractions of triceps brachii.

Figure 2.2a

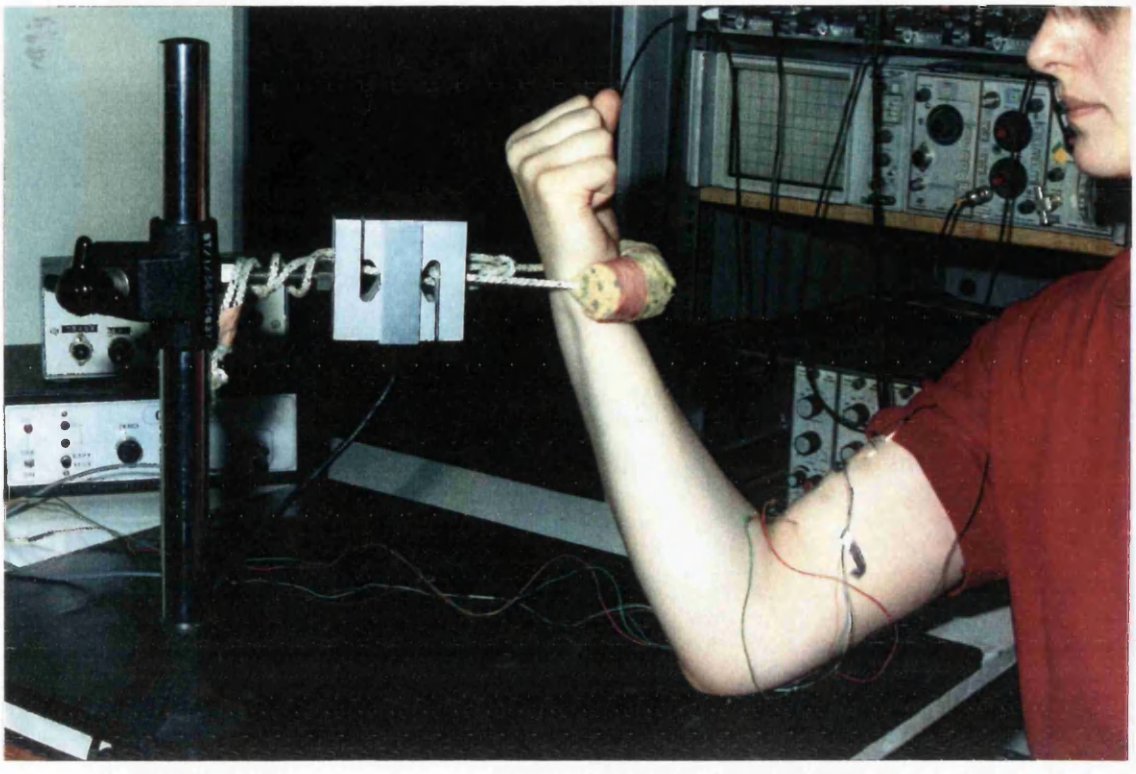
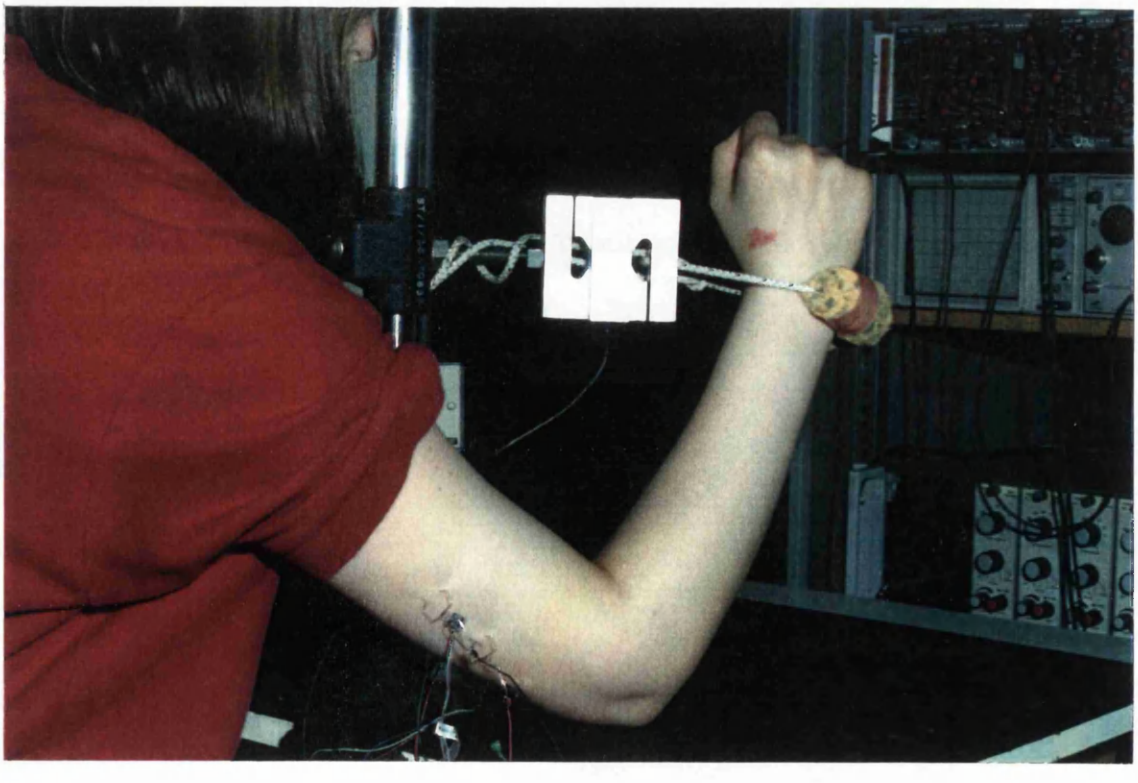


Figure 2.2b



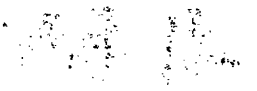


Figure 2.3a : Photograph showing the recording arrangement during contractions of first dorsal interosseus.

Figure 2.3b : Photograph showing the recording arrangement during the contractions of adductor pollicis.

Figure 2.3a

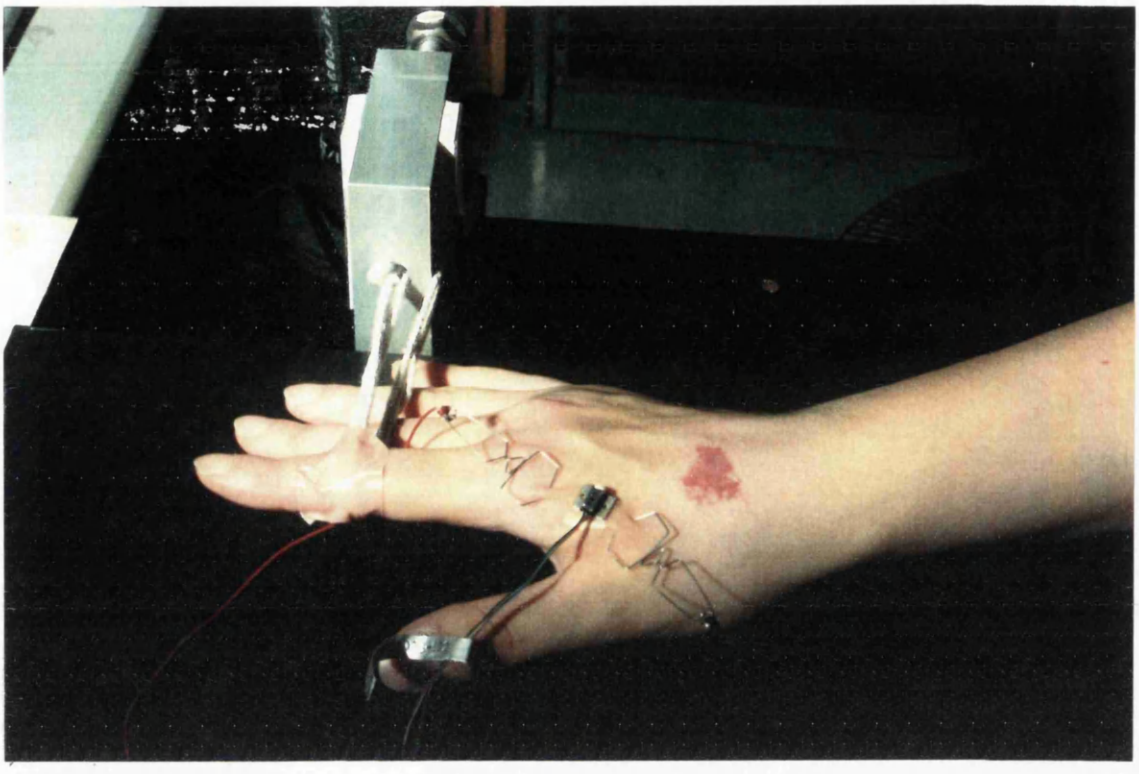


Figure 2.3b

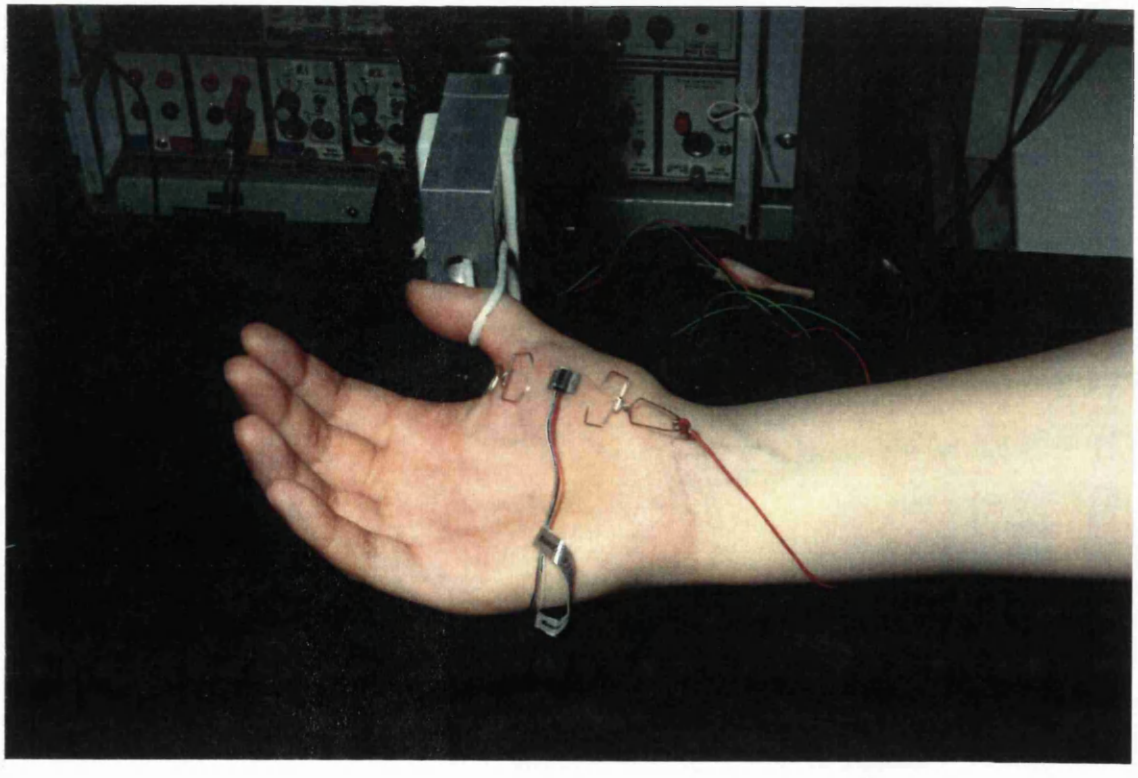


Figure 2.4a : Photograph showing the isokinetic dynamometer used during the recordings from rectus femoris.

Figure 2.4b : Photograph of the electrodes and accelerometer in position over the rectus femoris muscle.

Figure 2.4a

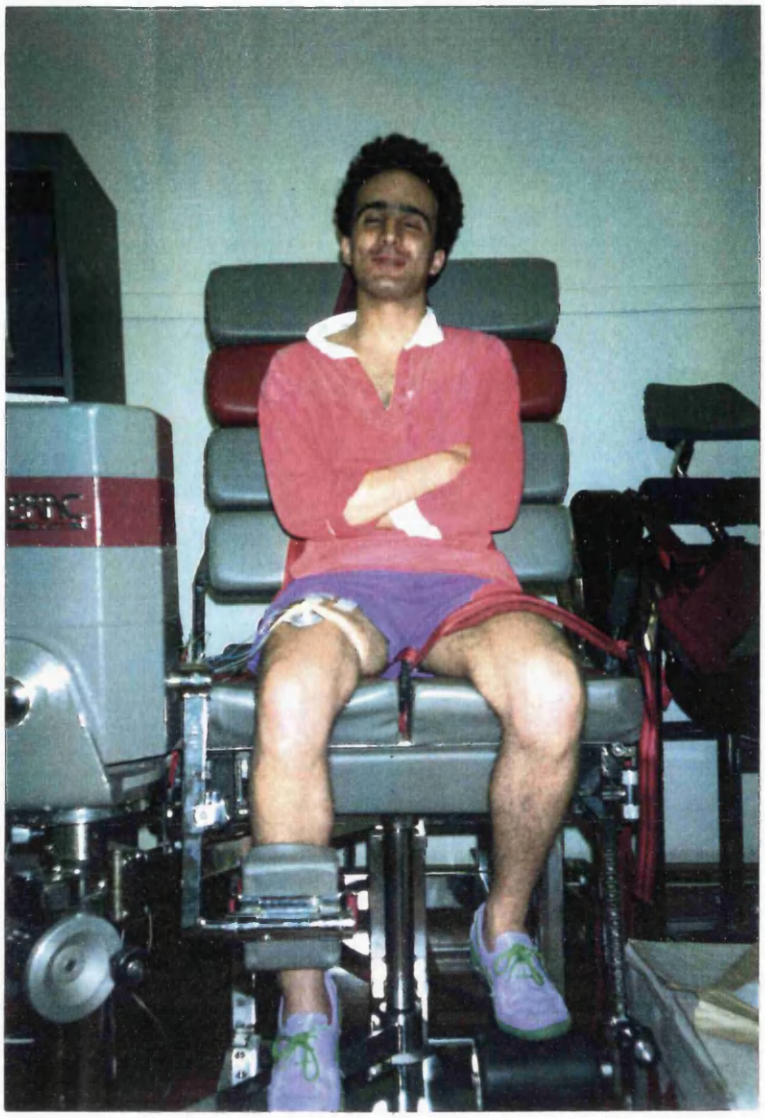
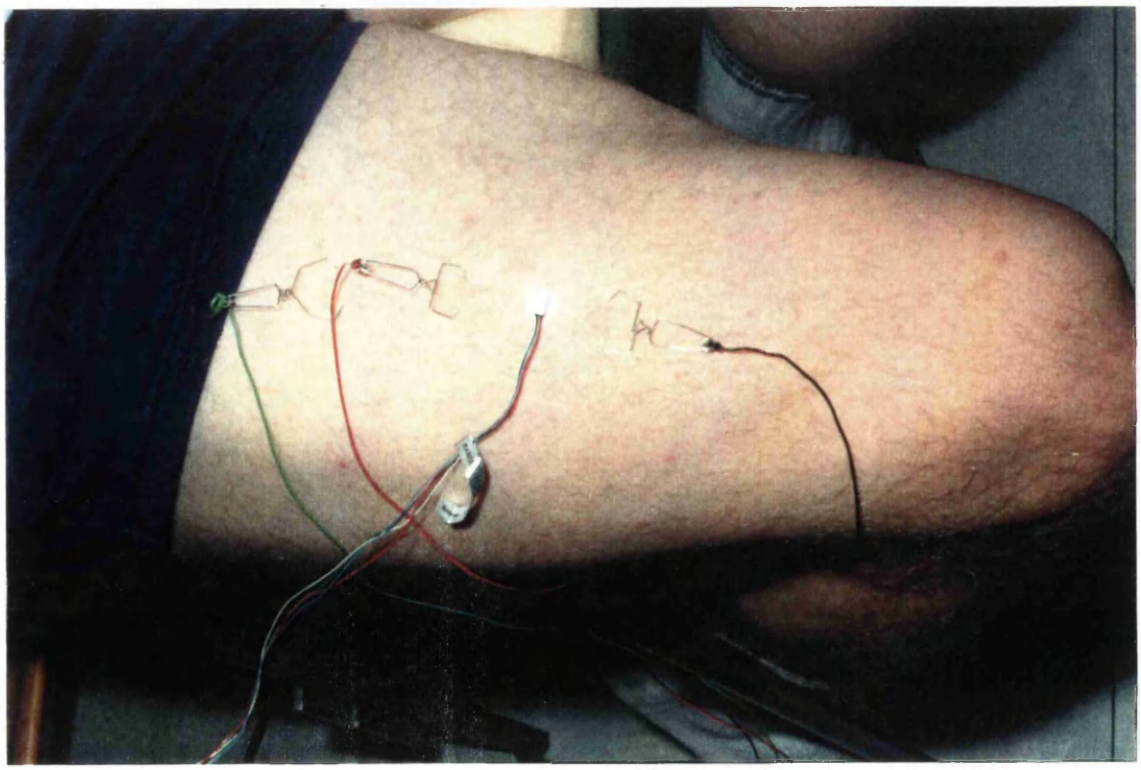


Figure 2.4b



RESULTS

Isometric contractions of Biceps and Triceps Brachii

Subjects

The subjects (n=8) for this series of experiments were taken from the general population within the department. There were both male and female subjects with the age range 23-40. They were all fully informed of the procedures involved and agreed to participate with the understanding that they could withdraw at any point. There was no evidence or history of any neuromuscular disorders within the subject group.

Methods

During biceps and triceps contractions the equipment was arranged as described in the materials and methods chapter. The series of contractions was performed as detailed in the materials and methods chapter with no alterations or additions.

a. Contractions of *Biceps Brachii and Triceps Brachii* at percentages of the maximal contraction.

The traces shown in figure 3.1 are from contractions at percentages of the maximal voluntary contraction. Figure 3.1a shows results from biceps and figure 3.1b shows results from triceps, with force shown as the bottom trace, EMG the middle trace and AMG the top trace for both figures.

The upper panel shows the series of contractions at 25, 50, 75, 100, 100, 75, 50 and 25% MVC. There is approximately 1 contraction per minute, with each

contraction lasting for around 10 seconds and a rest period of 50 seconds. The maximum force was 96N and this level is shown on the plot. The EMG record also shows increases in the amplitudes of the signals and clearly, the higher the force the larger the signal recorded. There is quite a lot of background noise on the EMG record and this results in the signal at the lowest force being quite hard to distinguish from the base line noise. The increases in amplitude with the increase in force are also seen within the AMG signal. However there is a lot of noise between contractions, and some of the contractions are surrounded by large amplitude peaks. The noise in between contractions results from movement by the subject and touching the muscle in the area of the accelerometer. The peaks around the contraction are movement artifacts, resulting from the subject contracting rapidly to the force required and then relaxing sharply.

The contraction of the triceps brachii muscle showed similar results to those already discussed from biceps brachii. Again the force trace is the bottom trace, EMG is the middle trace and AMG is the top trace. Force shows increases according to the predetermined maximum force level, which for this trace is around 72N. The EMG trace shows increases in peak to peak amplitude with the increase in force, showing maximal levels at the maximal force, and there is very little noise on the EMG baseline. AMG has a similar relationship with force and it can be seen that as the force level rises there is an increase in the peak to peak amplitude of the AMG record. Between contractions there is a lot of noise due to the subject moving and repositioning their arm. At the beginning and end of some of the contractions there is a large amplitude spike which is a result of the subject contracting or relaxing sharply.

By taking the rms values of the data the relationship between force, AMG and EMG can be quantified. The collective data from all the subjects from the biceps brachii is shown in figure 3.2a. The subjects were not always able to maintain the given force, and so the most steady value achieved was taken as the force level, resulting

in some scatter around the 25, 50 and 75% levels. There is more spread of the EMG and AMG values at the force levels indicated, with variations of up to 60% for the 75% MVC level. Plotting the residuals showed no non-linear trends. The general trend for the data is for both the EMG and AMG to rise with the increase in force. This can be summarised by taking the mean and standard error of the mean for EMG and AMG within each of the 4 specified force levels. This is illustrated in figure 3.3a and clearly there is a monotonically increasing relationship between both force and EMG, and force and AMG. At zero force there is some basal activity with the amount in the AMG signal being greater than the EMG level. This agrees strongly with the observations made when figure 3.1a was described earlier.

The relationship between force, EMG and AMG for triceps brachii can be summarised similarly. This is shown in figure 3.2b. Clearly, the results for the four force levels are grouped but there is a large amount of scatter both in the force level attained and the rms value of EMG and AMG. There is a clear trend for the EMG and AMG values to rise with the increase in force. A plot of the residuals showed no non-linear trends within the data. By taking the mean and the standard error of the mean for force, EMG and AMG a clearer picture can be obtained and this is shown in figure 3.3b. This graph shows that both EMG and AMG have numerically close, linear relationships with force, however, the AMG y axis intercept is greater than the EMG intercept indicating that there is more activity at zero force within the AMG signal than there is within the EMG signal . It is also interesting to note that the most variation in force level was seen during the low force contractions.

Median frequency

The median frequencies of EMG and AMG were measured from data recorded during the contractions of biceps brachii and an example trace from a maximal contraction is shown in figure 3.4a. The EMG frequencies are shown in the left

hand trace and AMG frequencies in the right. The range of frequencies of EMG is clearly much greater than that for the AMG. EMG ranges over 0-150 Hz, with the main frequencies being between 50 and 100Hz and the median frequency being calculated as 65Hz. AMG has a much narrower band being 0-50 Hz, and the median frequency being 17Hz. This is a typical trace for all of the contractions with differences being seen in the range of frequencies and in the median frequencies, for the different levels of contraction. The summary data for biceps can be seen in figure 3.4b. The data is shown as the mean frequency and the standard error of the mean. The EMG frequency from biceps seems to rise slightly with the increase in force and the frequency at 25%MVC is significantly lower than the frequencies at all other levels ($p=0.0001$), and there is a significant difference ($p=0.001$) between the frequencies at 50% and 100% MVC, although no significant difference can be found between 75% and 100%. The frequencies contained within the AMG signals appear to remain at a constant value throughout the series of contractions and there is no significant difference between the values.

Typical frequency data from triceps brachii is shown in figure 3.5a. As before, the EMG frequency plot is shown on the left and the AMG frequency plot on the right. Frequency, measured in Hertz, is shown along the x axis and it must be noted that the two scales are not identical. The y axis is power. EMG has a frequency range of 0-150 Hz and the median frequency is calculated as 65Hz. AMG has a much lower and narrower frequency range being 0-30 Hz, with the median frequency being 14 Hz. Taking the mean and the standard error of the mean, of the median frequencies at each of the force levels, the plot shown in figure 3.5b can be constructed. The EMG frequencies show a trend of increasing frequency with increase in force, although there is no significant difference between the frequencies at each level. The AMG frequencies show remain at a fairly constant level throughout the series of contractions, although there appears to be a slight increase in frequency between the 25% and 50%, and between the 50% and 75% levels, with a decrease at the 100%

level. This is not supported by statistical analysis, as there is no significant difference between the frequencies at each contraction level ($p>0.1$).

Figure 3.1a : Copy of trace from contractions of biceps brachii at percentages (25, 50, 75, 100, 100, 75, 50, 25) of the maximal contraction.

Figure 3.1b : Copy of trace from contractions of triceps brachii at percentages (25, 50, 75, 100, 100, 75, 50, 25) of the maximal contraction.

Figure 3.1a

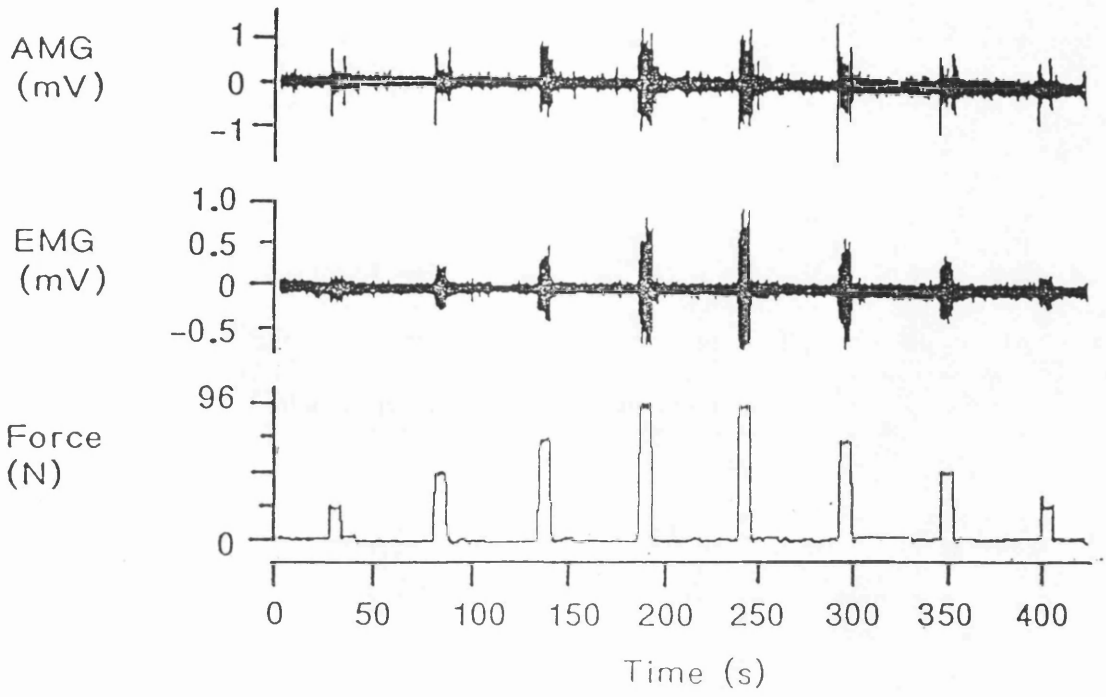


Figure 3.1b

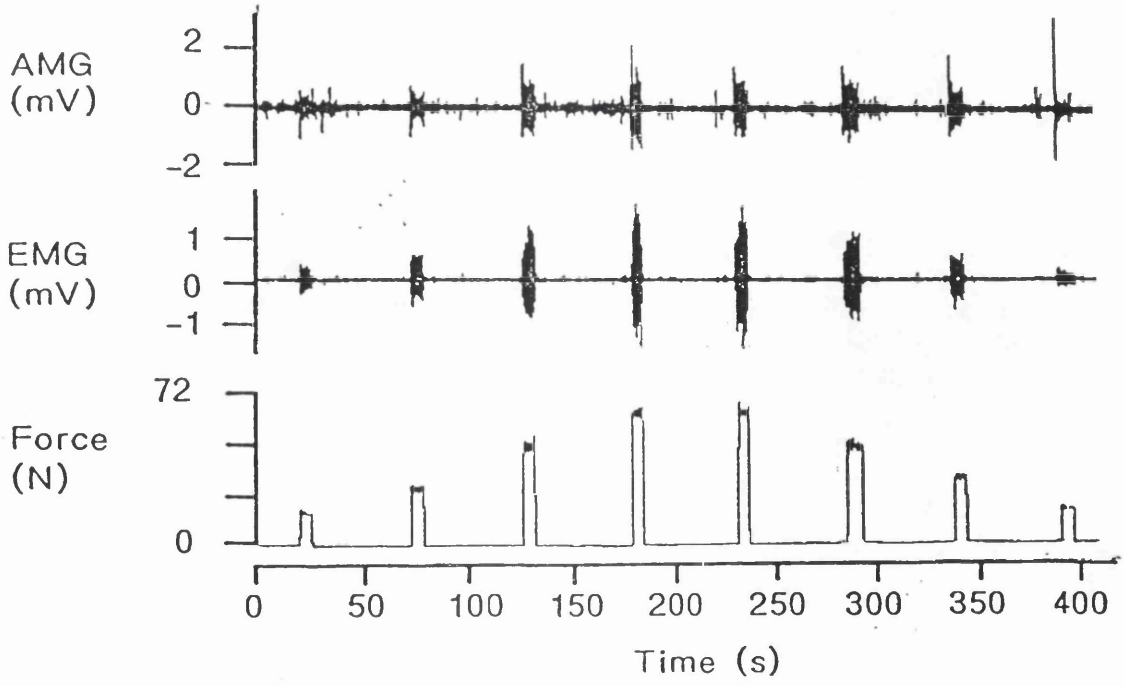


Figure 3.2a : Graph showing root mean square EMG and AMG data from all of the subjects (n=8) plotted against the percentage of the maximal voluntary force from biceps brachii.

Figure 3.2b : Graph showing root mean square EMG and AMG data from all of the subjects (n=8), plotted against the percentage of the maximal voluntary force from triceps brachii.

Figure 3.2a

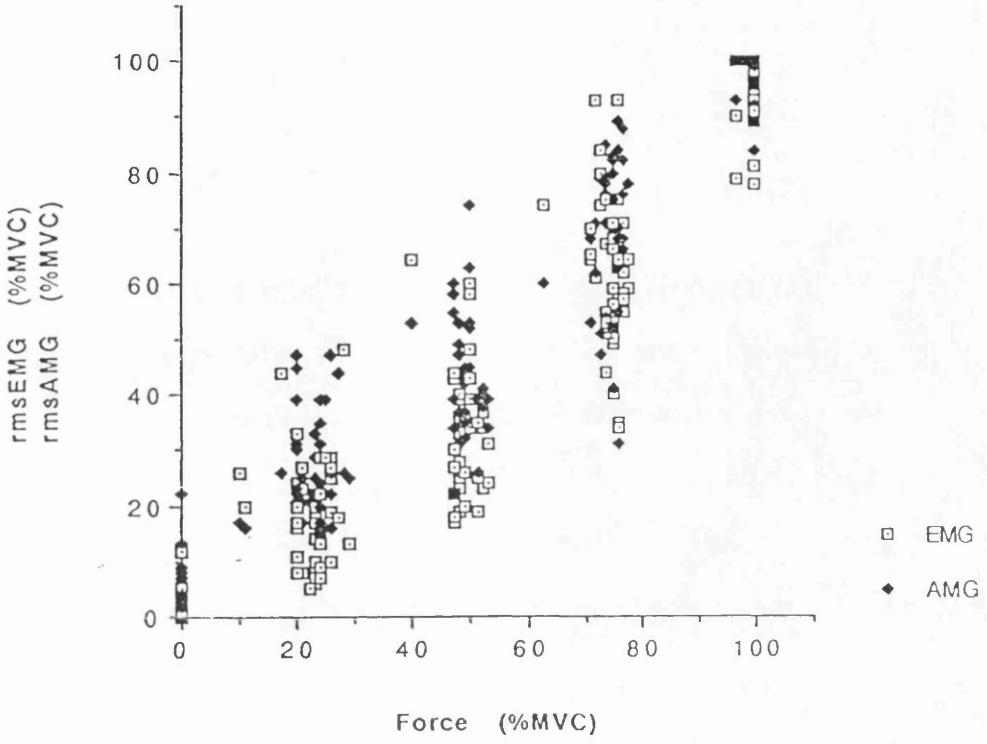


Figure 3.2b

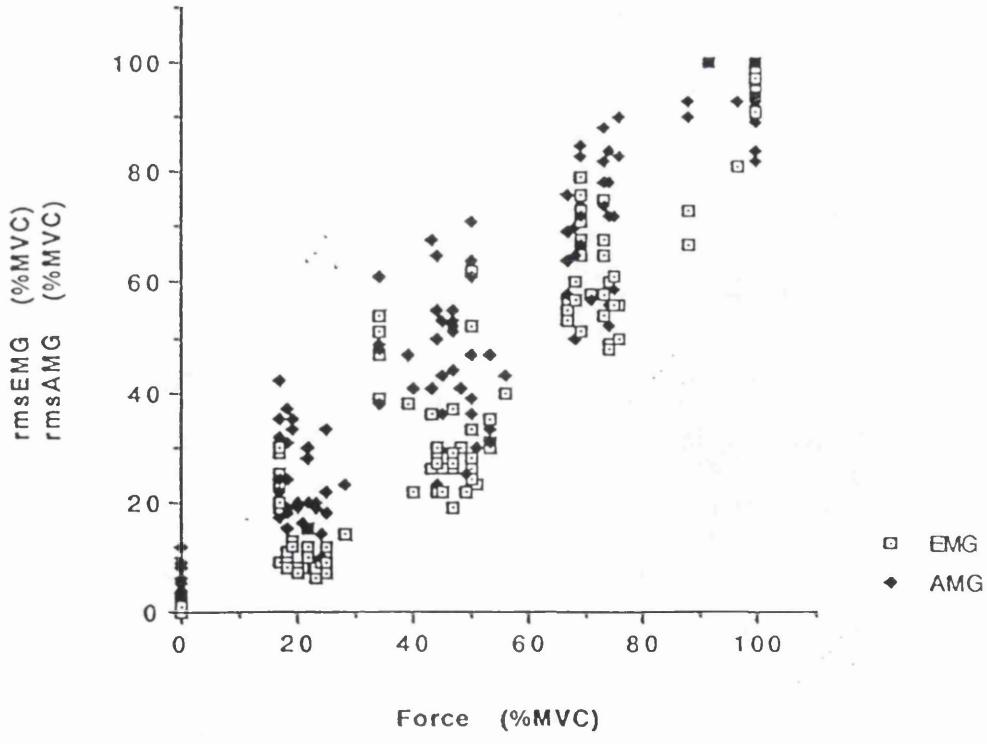


Figure 3.3a : Graph showing data from figure 3.2a, rms EMG and AMG against force, plotted as the mean and standard error of the mean, from contractions of biceps brachii. Regression equations: EMG vs Force $y = -3 + x$, $r^2 = 0.97$; AMG vs Force $y = 5 + 0.9x$, $r^2 = 0.99$.

Figure 3.3b : Graph showing data from figure 3.2b, rms EMG and AMG against force, plotted as the mean and standard error of the mean, from contractions of triceps brachii. Regression equations : EMG vs Force $y = -4 + x$, $r^2 = 0.97$; AMG vs Force $y = 7 + 0.9x$, $r^2 = 1.0$.

Figure 3.3a

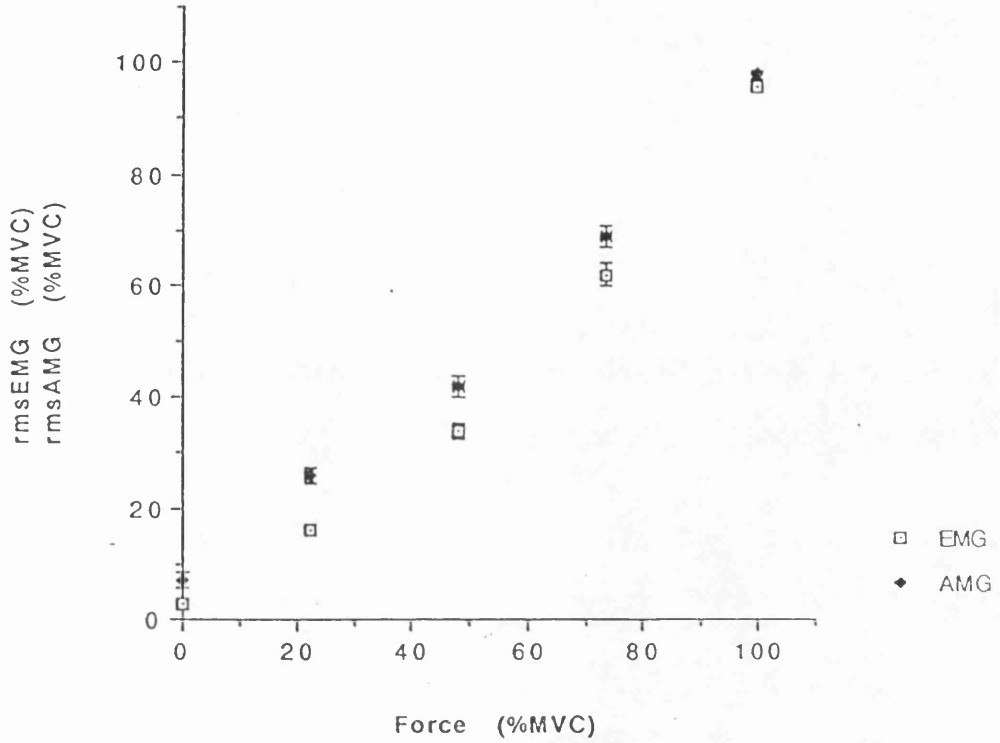


Figure 3.3b

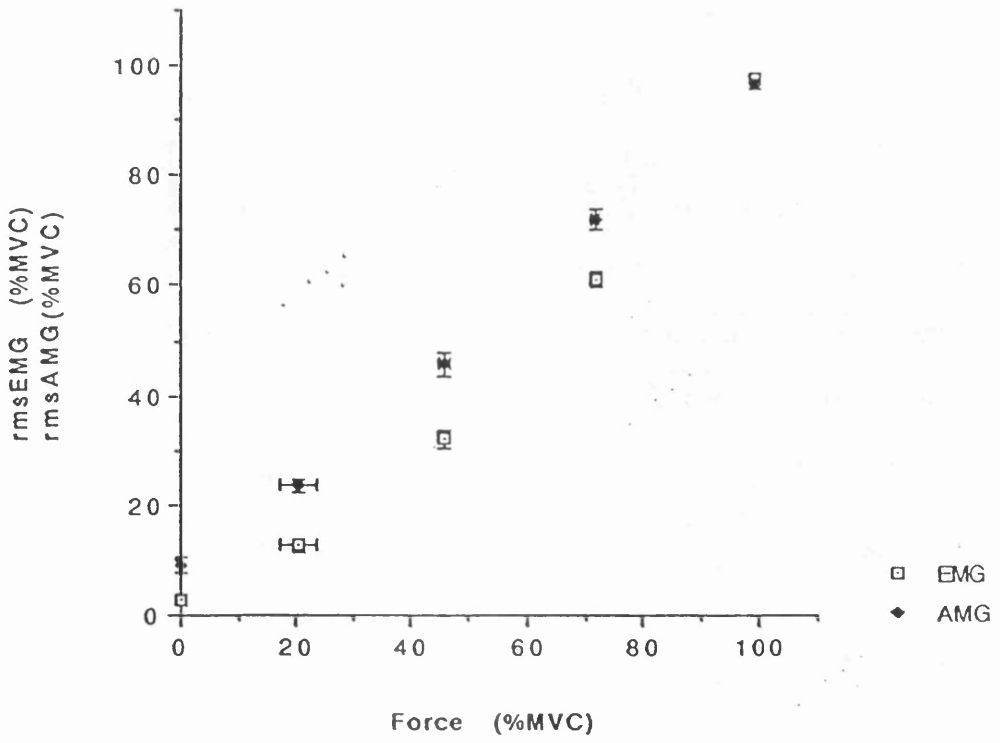


Figure 3.4a : Frequency plots of EMG and AMG from a maximal contraction of biceps brachii.

Figure 3.4b : Bar chart showing the mean and standard error of the mean of the median frequencies of EMG and AMG, from the series of contractions at percentages of the maximal contraction of biceps brachii from all subjects (n=8).

Figure 3.4a

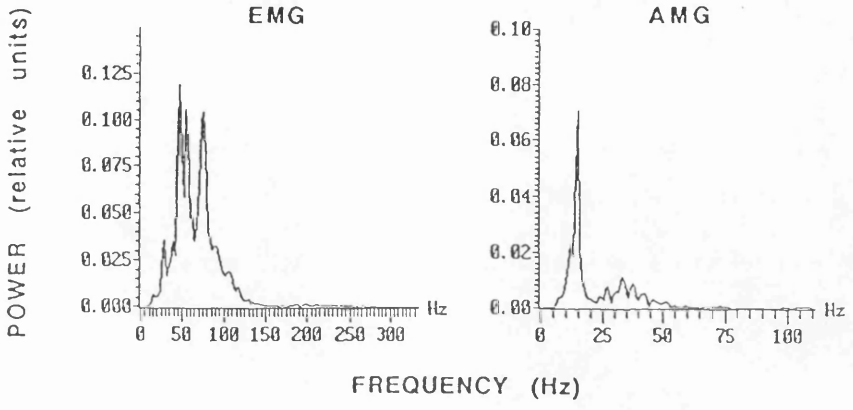


Figure 3.4b

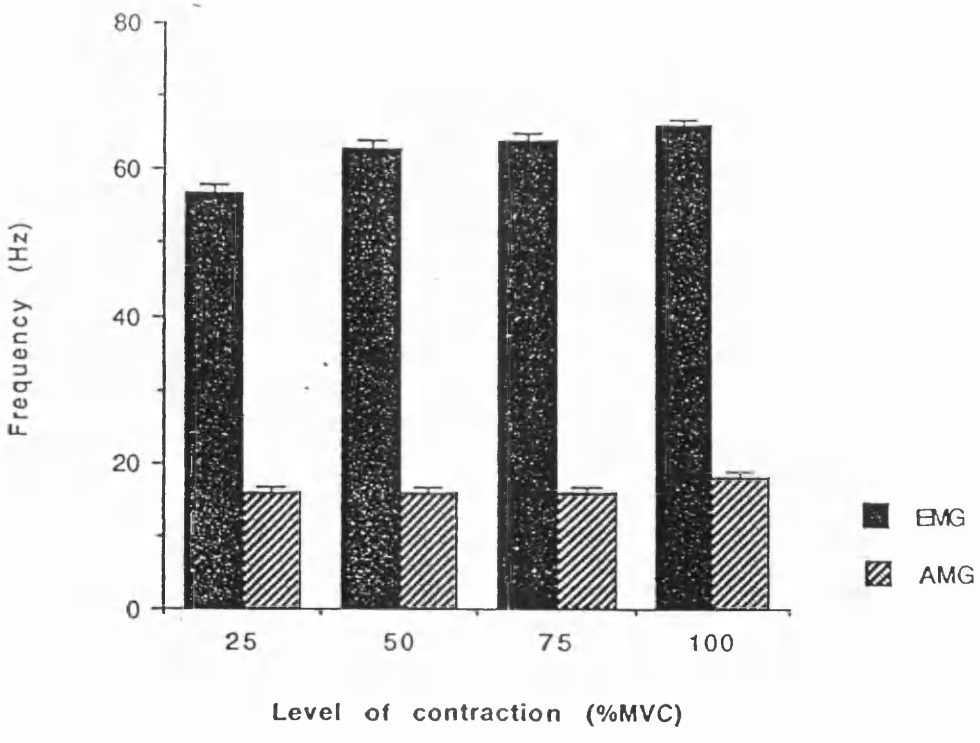


Figure 3.5a : Frequency plots of EMG and AMG from a maximal contraction of triceps brachii.

Figure 3.5b : Bar chart showing the mean and standard error of the mean of the median frequencies of EMG and AMG, from the series of contractions at percentages of the maximal contraction of triceps brachii from all subjects (n=8).

Figure 3.5a

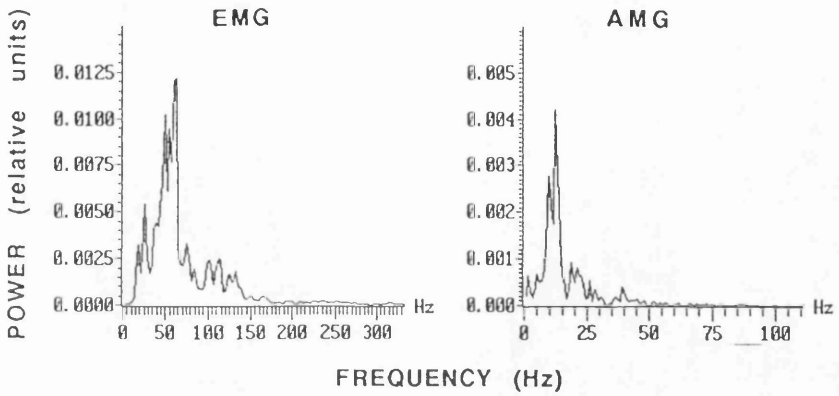
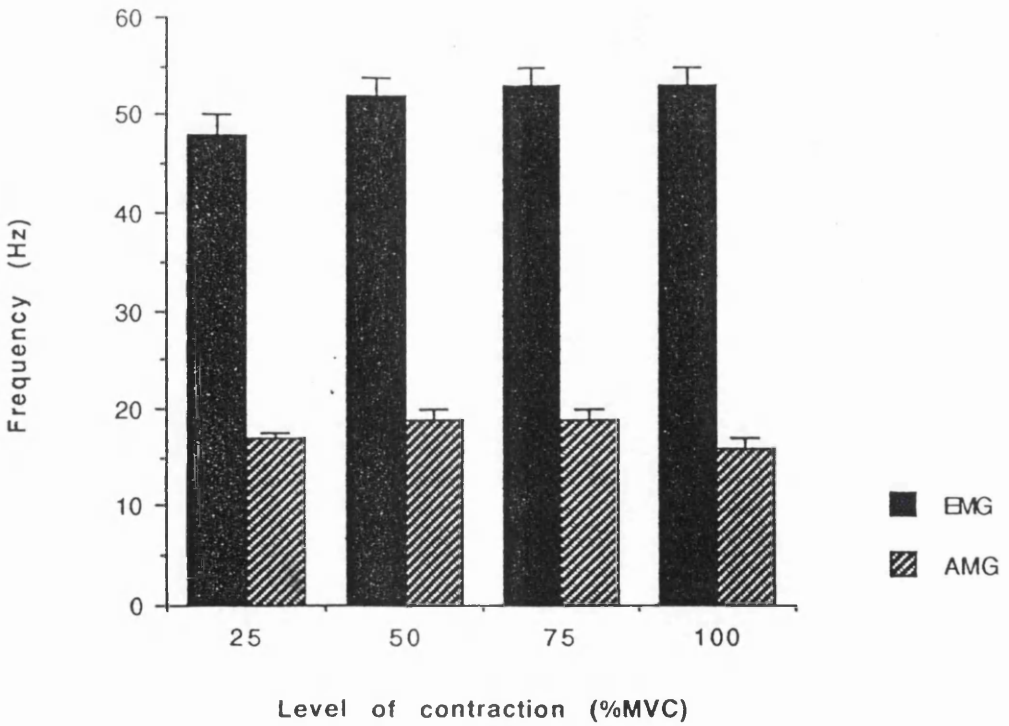


Figure 3.5b



b. Ramp Contractions of *Biceps and Triceps Brachii.*

The subject was asked to increase the force exerted up to the maximum force possible and then to relax until no force was being produced. A contraction from biceps brachii is shown in figure 3.6a and a contraction from triceps brachii is shown in figure 3.6b. Force is shown as the bottom trace, EMG as the middle trace and AMG as the top trace for both plots. In figure 3.6a, two contractions are shown and for each, the force increases steadily up to the maximal force, which is around 96N for both, and then there is a steady decrease in force until the subject is fully relaxed. Both contractions last for around 50 seconds. The first contraction shows increases in the peak to peak amplitude of the EMG signal from the start of the contraction until the maximal force is reached. The amplitude then decreases until the end of the contraction. The second contraction has a more regular appearance and from the beginning of the contraction there is an increase in the peak to peak amplitude of the signal until the maximal force is reached 25 seconds later. There is a motor unit spike evident on the trace that is much larger than any of the other activity occuring at the maximal force. The amplitude then decreases until the end of the contraction. There is very little noise in the EMG trace between contractions and this differs from the AMG trace above which has a high level of noise as can be seen from the baseline in the rest periods between contractions. However, the peak to peak amplitude of the AMG signal increases with the increase in force in a similar manner to that of EMG. During the first contraction the signal rises until the maximal force is reached. It then decreases to around half the amplitude seen during the maximal force, although this is higher than the base line level. It remains at this level until the end of the contraction where a movement artifact can be seen and the amplitude drops back to pre-contraction levels. The AMG signal during the second contraction is a lot easier to describe. There is a movement artifact at the beginning of the contraction and then the amplitude increases slightly, until around 75% of the maximal force is obtained and then there is a sharp rise in the AMG amplitude to

around twice the previous level. There is then a steady increase to the maximal level. There is a decrease in peak to peak amplitude until the force has dropped to around 75%MVC, when the peak to peak amplitude decreases by half and there is a slow decline in amplitude until the end of the contraction.

Two ramp contractions from triceps brachii are shown in figure 3.6b. They are very similar in appearance to those from biceps brachii. Force is shown on the bottom, EMG in the middle and AMG at the top of the figure. The x axis is time measured in seconds (s), with the y axis for force being measured in Newtons (N), and the y axes for EMG and AMG being measured in millivolts (mV). Each contraction lasts for around 50 seconds, with a rest period of 35 seconds in between contractions. Both contractions show increases in the peak to peak amplitude of the EMG signal from the start of the contractions. The signal increases to a maximum level, which is the same for both contractions, and then decreases to the end of the contraction. AMG has a very similar profile to that of EMG. There is an immediate increase in amplitude at the start of the contraction, it then increases steadily until the maximum force is reached. The amplitude then decreases until there is no force exerted and the AMG signal then returns to the base line level. Neither the EMG nor the AMG trace has much noise between the contractions. This was due to the subject remaining still during this record. However, between the two contractions there is a small burst of AMG which is accompanied by some EMG possibly indicating that there was a small muscular contraction.

As the force levels and times of contraction vary for each subject, it is impossible to show the pooled data and be able to extract anything meaningful from it. Therefore, data from individual contractions are illustrated. Figure 3.7a shows data from the second contraction illustrated in figure 3.6a, with force, rmsEMG and rmsAMG, plotted against time. Clearly as the force increases both the AMG and EMG signals increase in the manner already described. The maximal values do not occur at exactly the time, and there is some difference in the signal levels for the increasing

and decreasing sides of the contraction. Force appears to lead the increase in rmsEMG and rmsAMG, and EMG leads the decrease in force and AMG. This is partly a consequence of taking the rms values over a specific time period and perhaps more importantly the the relationship shown in figure 3.3a is not linear. It must also be remembered that this description is true only for this contraction and cannot be applied to all ramp contractions from the biceps brachii muscle. Therefore, there is strong evidence of force leading the AMG and EMG signals. This may be a consequence of the mistiming of data collection.

Figure 3.7b shows the data from the first contraction of triceps illustrated in figure 3.6b. Force, rmsEMG and rmsAMG are all shown normalised to the maximum value and plotted against time. The maximal values of this contraction occur simultaneously, although there is some evidence of the force leading on the increasing side of the contraction, and some variation in the signal levels seen on the descending side of the contraction. The conditions mentioned above namely non-linearity and mistiming of data collection apply here. However, it would be sensible to conclude that in ramp contractions of triceps brachii to the maximal force there is an association between the force, EMG and AMG signals recorded.

In order to show that the same relationship is true for all the data obtained from the ramp contractions, the rmsEMG and rmsAMG levels calculated during both the ascending and descending phases of the contraction are plotted against the percentage of the maximal force. The plot from the contractions of biceps brachii is shown in figure 3.8a. This shows that as force increases there is an increase in both the AMG and the EMG signals. It is not a linear relationship, but can be said to be a monotonically increasing one. There is some AMG and EMG recorded at zero force indicating movement being recorded with the accelerometer and some background EMG activity. During the lower forces, the AMG points appear fairly tightly grouped with a spreading out of values from 80%MVC to the maximal force. EMG has a fairly wide spread throughout the plot. Regressing both AMG and EMG onto

force showed that they had very similar regression equations and investigation of the residuals showed no non-linear trends for the data. It can be said from this that both the rmsEMG and rmsAMG signals show increasing and decreasing amplitudes with an increase and decrease in force as seen during a ramp contraction.

The summary data from the ramp contractions of triceps brachii are shown in figure 3.8b. Plotting rmsEMG and rmsAMG against force shows that as the force increases or decreases, as both sets of points are shown on this graph, there is an accompanying increase or decrease in EMG and AMG. It is certainly not a linear relationship but it is a monotonically increasing one. The amount of scatter of both sets of data particularly from 40%MVC onwards indicates that there is a wide variation in the level of the signals seen at these forces, but there is no real difference in the relationships of each with force. Between 0 and 40% MVC, it would appear as if the AMG signals start at a higher percentage of their maximal level than the EMG signals. This is probably due to the movement at the beginning and end of the contraction and looking at the raw data traces it is possible to see the AMG signal level increasing initially before any force is seen. Regressing AMG and EMG onto force showed that they had very similar regression equations and plotting the residuals revealed no non-linear trends within the data.

In summary, in ramp contractions of biceps and triceps brachii, there are increases in both EMG and AMG with an increase in force and when force is decreased this is accompanied by a decrease in EMG and AMG.

Median Frequency

Frequency plots from the beginning, middle and end of the ramp contraction are illustrated in figure 3.9a. It should be noted that the power (y) axis for the middle plot is shown on a different scale to that of the beginning and end plots. The frequency axes remain constant throughout. The EMG plots are on the left side and the AMG plots are shown on the right side of the figure. The power in the EMG signal is very low at the beginning and the frequency range is 0 - 150 Hz, and the median frequency is 49Hz. Considerable changes in power are seen in the plot from the middle of the contraction, with an increase by a factor of 100. The range does not change remaining at 0 - 150 Hz, and the median frequency increases slightly to 61Hz. The power within the signal falls again at the end of the contraction, returning to a level only slightly higher than at the beginning of the contraction. The range remains unchanged and the median frequency decreases to 48Hz. The AMG frequencies exhibit very similar trends. The range of frequencies at the beginning is 0 - 50Hz, with the median frequency being 15 Hz. The middle section reveals an increase in the power by a factor of 5, within the same frequency range. There is an increase in the median frequency to 22Hz, due to there being an increase in the power associated with the frequencies between 25 and 50Hz. The plot from the end section of the contraction shows no change in the range of frequencies, although the power associated with them decreased back to levels lower than those seen at the beginning of the contraction, decreasing by a factor of 20 from levels at the middle section. Although this is data from one subject, all of the contractions showed these general trends.

Calculation of the mean and the standard error of the mean of the median frequencies for beginning, middle and end sections of all of the contractions from all of the subjects yields figure 3.9b . The median frequency of the middle EMG signal appears to be greater than the frequency at the beginning and end of the contraction. This is supported by statistical analysis showing that there is a

significant difference between the beginning and middle frequencies (p=0.0002) and the end and middle frequencies (p=0.0001), although there is no significant difference between the beginning and end frequencies. The AMG frequencies show no definite trends although there does appear to be a slight tendency for the frequency at the middle section to be slightly higher than both the beginning and end sections. There is some statistical support for this although the statistical difference between the beginning and middle section is low (p=0.01) and there is no significant difference between any of the other sections of the contraction (p>0.5).

The frequency plots from triceps brachii are shown in figure 3.10a. Frequency is the x axis measured in Hz. for all of the plots, and power is shown as the y axes and is shown as arbitrary units. As before the middle plot has a different power scale from the other two plots in order to display the data clearly. The EMG frequencies from the beginning of the contraction span the range 0 - 150 Hz with the median frequency being 56Hz. The power within the signal is less than 0.001 (relative units) at these frequencies. The middle plot shows no change in range of frequencies and the median frequency remains 56Hz. The power within the frequency range increases by up to 20 times from the beginning of the contraction. The plot from the end of the contraction again shows a decrease in power back to levels comparable with the start of the contraction, the range remains the same although the median frequency decreases slightly to 48Hz. AMG frequencies from the beginning of the contraction have quite a wide range from 0 - 50Hz, very little power within this range and the median frequency is 25Hz. The middle plot shows a much more distinct range, still between 0 and 50 Hz, but with most of the power being between 0 and 25Hz. The median frequency is lower, 17Hz, and there is much more power within the signal at these frequencies having increased by a factor of 20. The end plot shows no change in the range although the main power remains at frequencies within the range 0-25Hz. The median frequency has

decreased slightly to 12 Hz and the power within the range has decreased by a factor of 5, from the middle power level. Again it must be remembered that this description was for one contraction from one subject, and it is more informative to study the summary data from this group of experiments.

Shown in figure 3.10b is the summary data from triceps brachii. There is virtually no difference in EMG frequency between the beginning and the middle stages of the contraction, although the frequency at the end of the contraction is lower than either of them. This is supported statistically, although with a low significance, comparing beginning and end there is a statistical difference of $p=0.05$, and comparing middle and end, $p=0.003$. Thus there is a trend for the EMG frequency to fall towards the end of the ramp contraction. The AMG frequency does not appear to change during the ramp contraction and appears to remain at a constant level around 15Hz. This observation is supported by statistical analysis which shows that there is no difference between the frequencies at any stage of the contraction ($p>0.5$).

Figure 3.6a : Copy of a trace showing two isometric ramp contractions of biceps brachii to maximal force.

Figure 3.6b : Copy of a trace showing two isometric ramp contractions of triceps brachii to maximal force.

Figure 3.6a

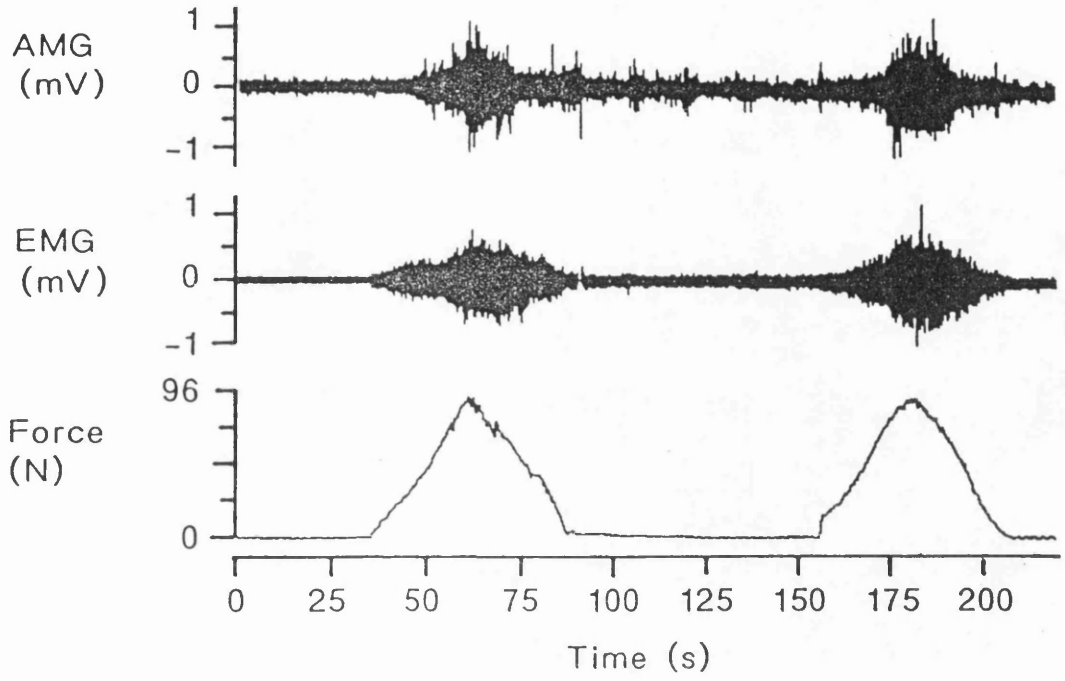


Figure 3.6b

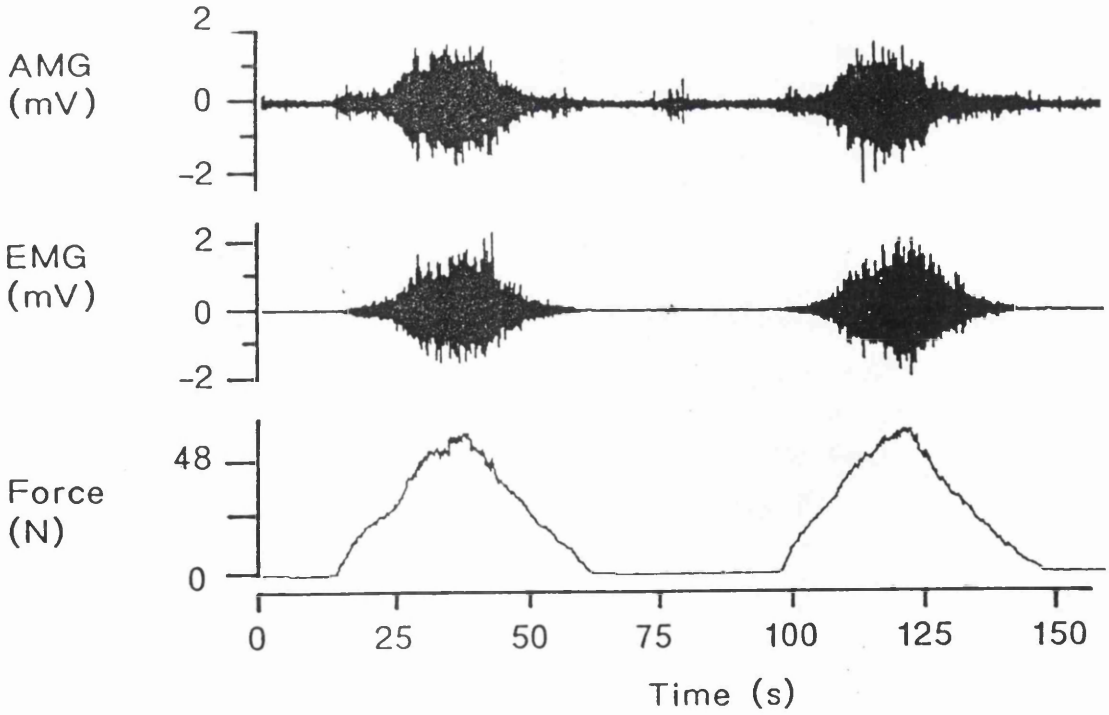


Figure 3.7a : Plot showing the force, rms EMG and rms AMG data (from the second contraction shown in figure 3.6a) against time from a ramp contraction of biceps brachii.

Figure 3.7b : Plot showing the force, rms EMG and rms AMG data (from the first contraction shown in figure 3.6a) against time from a ramp contraction of triceps brachii.

Figure 3.7a

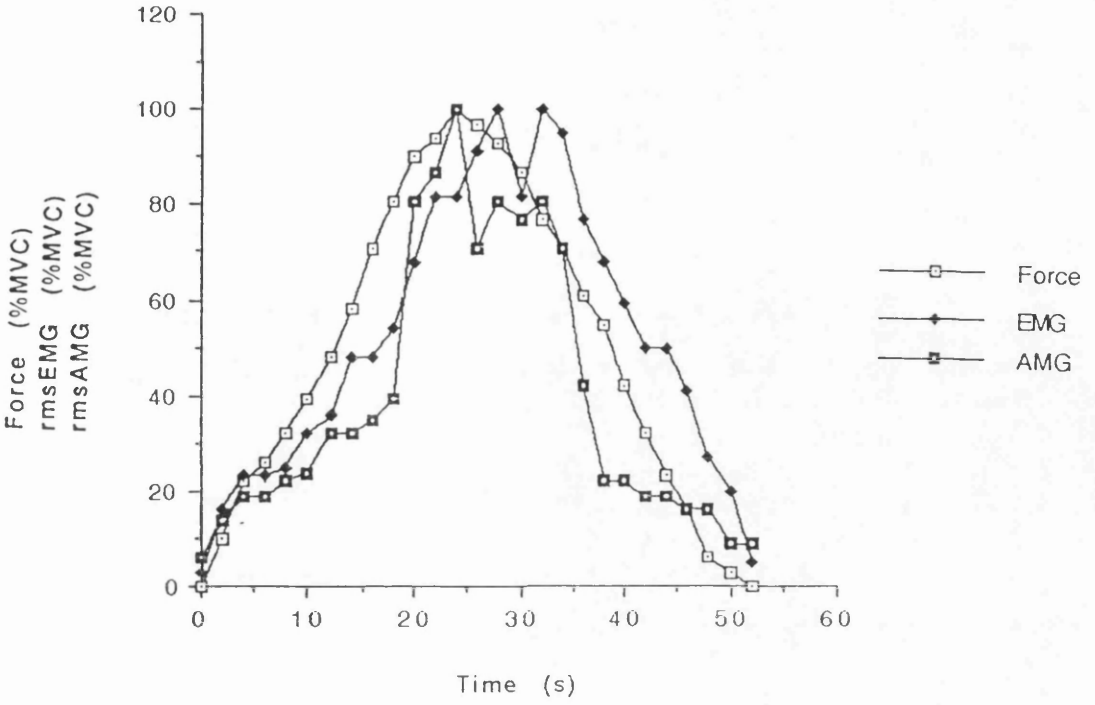


Figure 3.7b

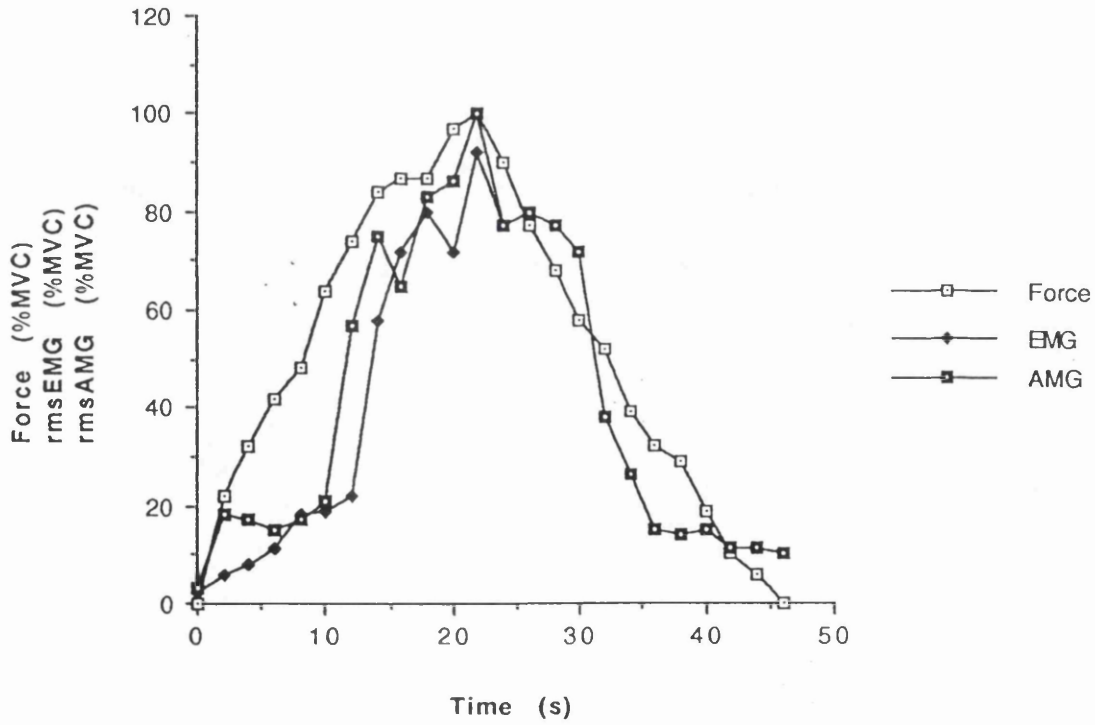


Figure 3.8a : Plot of rms EMG and AMG data from all subjects (n=8) against force from ramp contractions of biceps brachii.

Figure 3.8b : Plot of rms EMG and AMG data from all subjects (n=8) against force from ramp contractions of triceps brachii.

Figure 3.8a

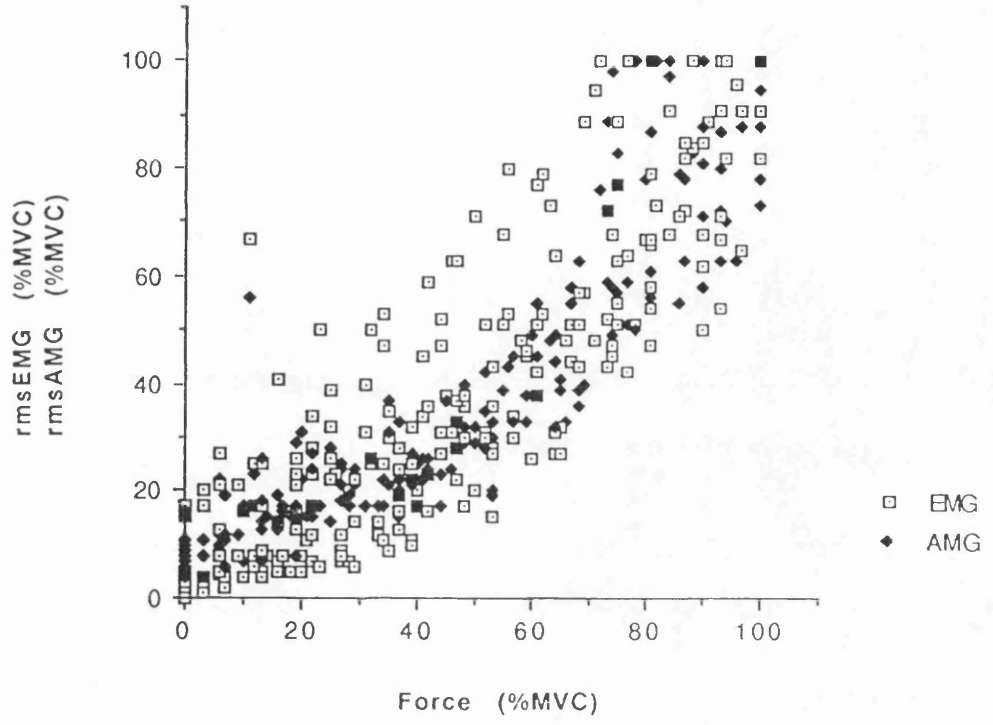


Figure 3.8b

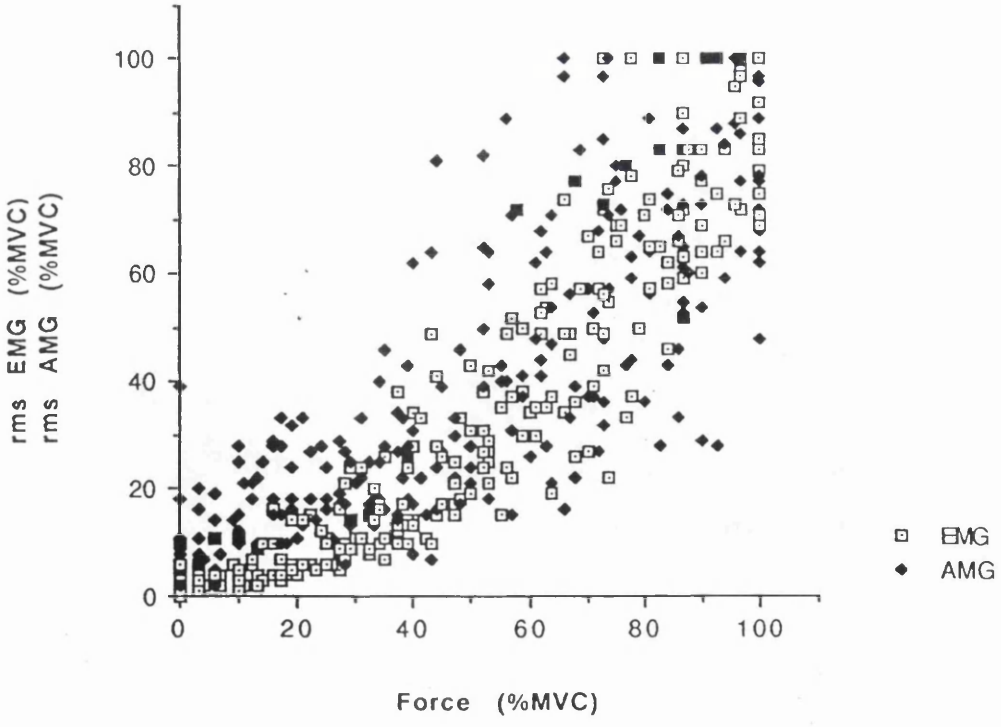


Figure 3.9a : Frequency plots from the beginning, middle and end of one ramp contraction of biceps brachii.

Figure 3.9b : Plot showing the mean and standard error of the mean of the median frequencies from the beginning (B), middle (M) and end (E) of the ramp contractions of biceps brachii from all subjects (n=8).

Figure 3.9a

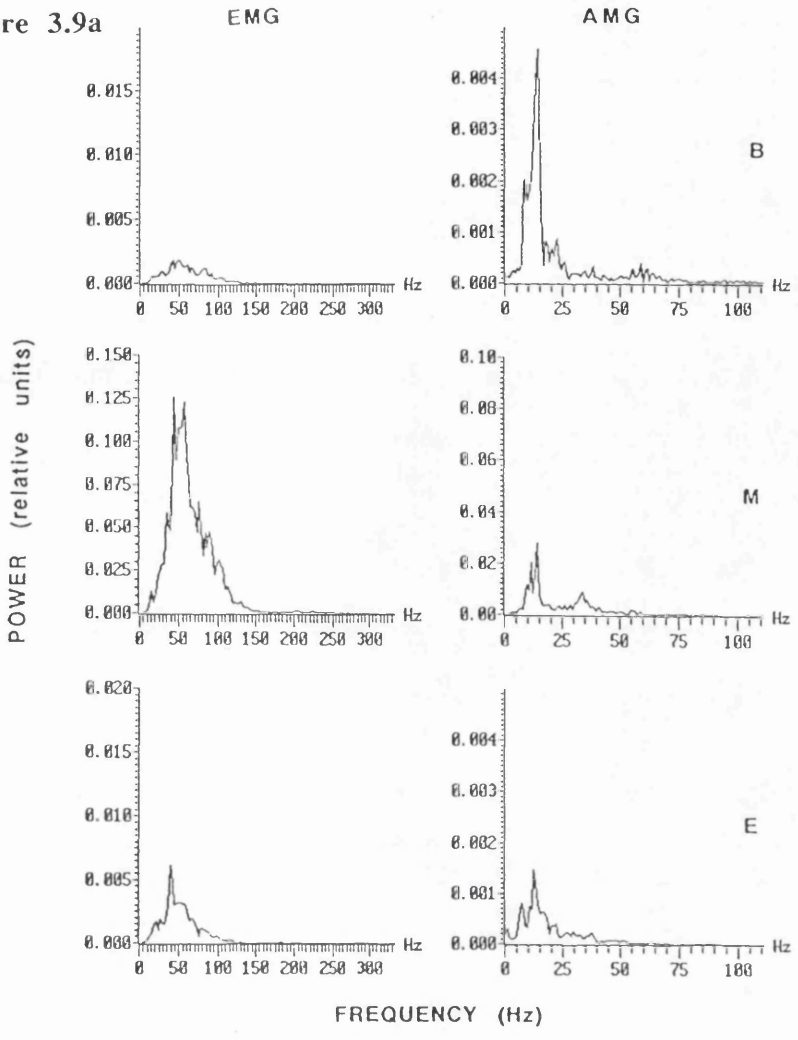


Figure 3.9b

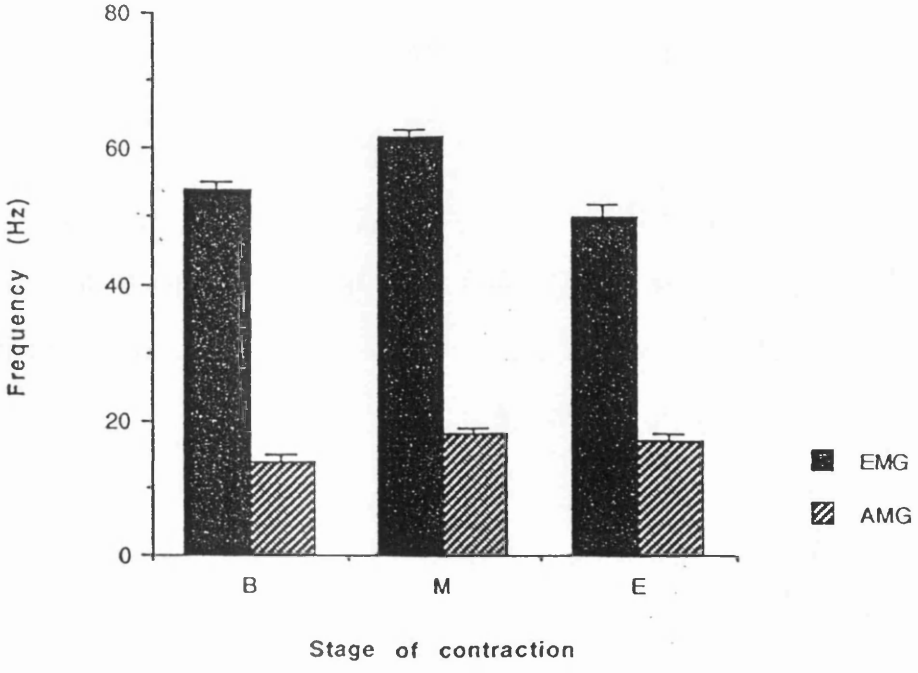


Figure 3.10a : Frequency plots from the beginning, middle and end of one ramp contraction of triceps brachii.

Figure 3.10b : Plot showing the mean and standard error of the mean of the median frequencies from the beginning (B), middle (M) and end (E) of the ramp contractions of triceps brachii from all subjects (n=8).

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Figure 3.10a

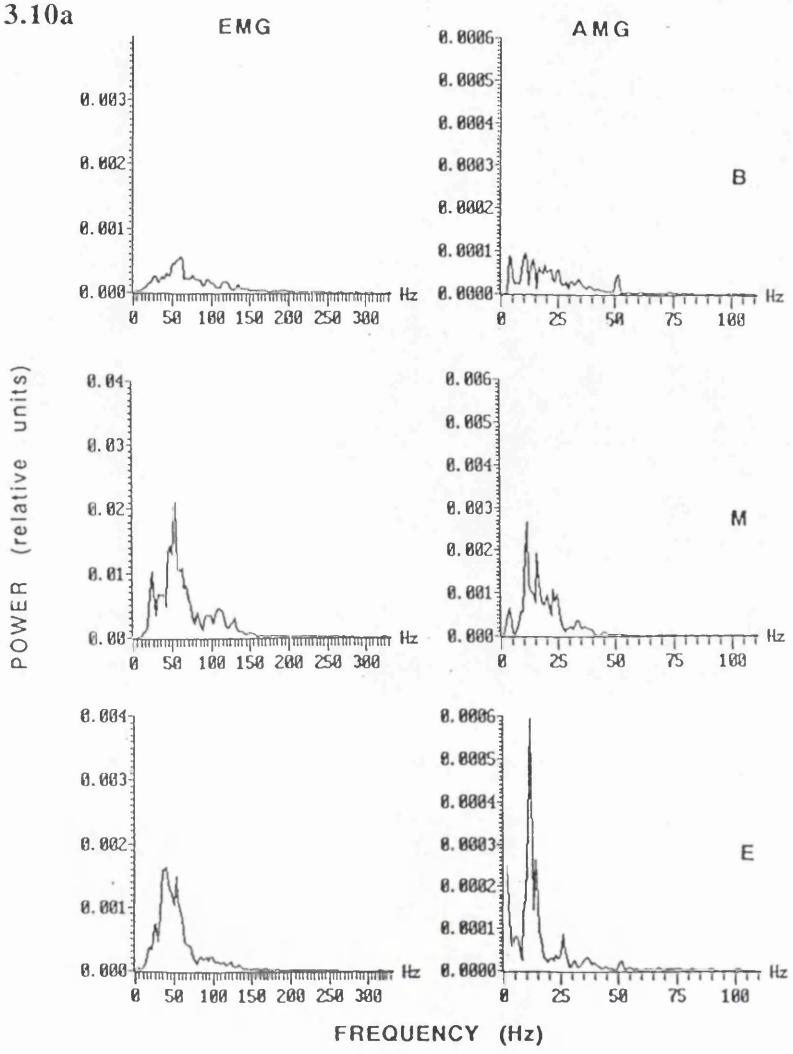
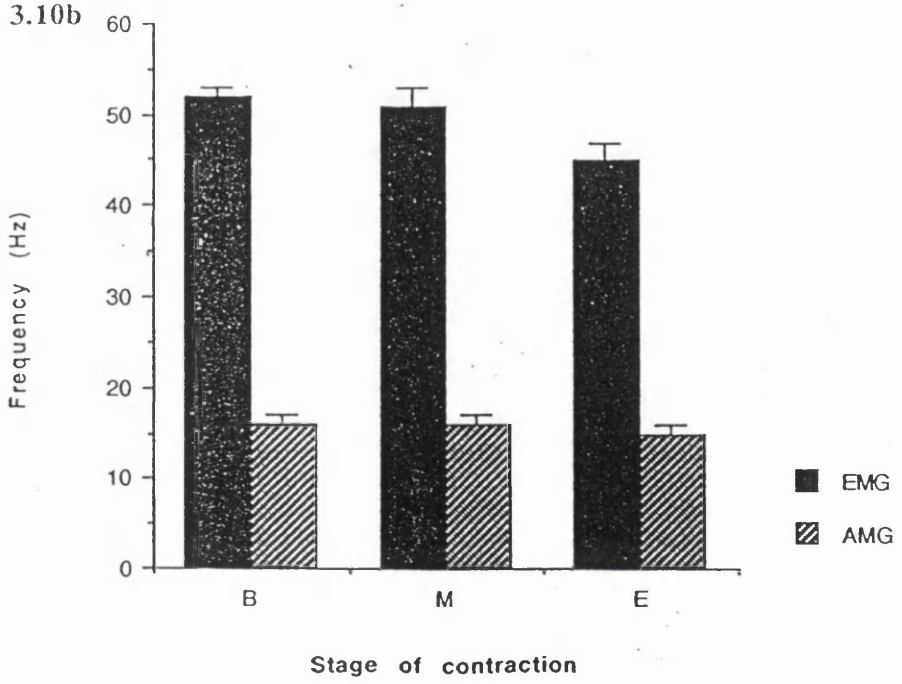


Figure 3.10b



c. Fatiguing isometric contractions of *Biceps and Triceps Brachii* at 75% MVC.

By asking the subject to contract at 75% of the maximal contraction it was hoped that they would be able to maintain the contraction for at least a minute during which time they would be fatiguing and any changes in both signal levels and frequency would be apparent.

An example of a contraction from biceps is shown in figure 3.11a with force as the bottom trace, EMG as the middle and AMG as the top trace. The contraction lasts for 90 seconds and the force level remains very steady throughout at 56N. Before the contraction EMG is at extremely low levels indicating that the subject was fully relaxed. At the start of the contraction the EMG increases until the force level is reached and then starts to increase. The peak to peak amplitude of the signal rises steadily until the end of the contraction when the signal is around 3 times the starting level. The signal level drops back to the initial pre-contraction level. AMG is also at low levels before the start of the contraction. When the contraction begins there is a sharp rise in amplitude and then the signal returns to the contraction level which is approximately twice the signal level before the contraction. The peak to peak amplitude of the signal increases until 50 seconds from the start of the contraction. There is then a transient dip in the signal, with the signal recovering although not to the previous level. The amplitude increases again to reach the maximal level around 60 seconds from the start of the contraction. From here there is a slight decrease in the signal amplitude, another dip after which the signal recovers slightly, and then decreases in amplitude to the end of the contraction. There is some activity in the AMG signal after the force has returned to zero, due to the subject moving and then the signal returns to the pre-contraction levels.

Although this is data from one subject, this description of the trace is quite typical with force being held steady for 60-90 seconds, during this time the EMG signal

increases throughout the contraction and the AMG signal rises initially and then either reaches a steady level or decreases slightly until the end of the contraction.

Analysis of the trace by taking the force and rms levels at 5 second intervals yields figure 3.11b. The contraction starts at the 0 seconds point on the graph. As already described but now reinforced, force rises to the the maximal level and remains constant throughout the contraction. EMG rises until the end and AMG rises to a maximal level until 10 seconds before the end of the contraction when the signal level starts to decrease slightly, both signals then drop to their pre-contraction values.

The triceps contraction exhibits similar trends to that of the biceps contraction and an example is shown in figure 3.12a. The contraction lasts for 48 seconds during which time the force level remains at a fairly steady level around 29N. Before the contraction starts EMG is at extremely low levels, and when the contraction begins the EMG amplitude rises immediately. It continues to rise until 30 seconds from the start of the contraction there is a transient dip. The amplitude is recovered and the signal continues to rise until the end of the contraction when the amplitude is around 3 times the initial level. The amplitude decreases sharply as the force decreases, and it returns to the base line value. There is a large artifact at the beginning of the AMG trace which signifies that the subject moved into position abruptly. The amplitude increases and then remains steady until around 30 seconds into the contraction. There are two bursts of activity, which occur around the same time that the force record varies, and then the amplitude returns to the steady level There is no further change in the amplitude until 5 seconds from the end of the contraction when there is a doubling in value of the amplitude. The signal then decreases again until the end of the contraction.

The EMG signal is easier to describe than the AMG which has an indistinct relationship with force, with small bursts of activity over a fairly steady signal.

Analysis of the trace shown in figure 3.12a gives the plot that is shown in figure 3.12b. The rms EMG and rms AMG signal levels, were taken at 5 second intervals throughout the contraction along with the force level at these intervals. The contraction begins at the 0 seconds point and the force rises immediately and within 4 seconds has reached the maximal level. It remains at this level until the end of the contraction. EMG starts to increase from the start of the contraction, until 20 seconds from the start of the contraction when 80% of the maximal signal is reached. There is a dip in the signal at this point although this level is recovered with 7 seconds and the EMG continues to rise, reaching a maximal value at the end of the contraction, and then decreasing to zero. AMG has a very shallow rise in signal level initially and remains at a steady level for the middle part of the contraction. It is only at the very end of the contraction that there is a rise in the AMG to the maximal level. As seen in the raw data, the AMG then falls until there is very little signal by the end of the contraction.

Median frequency.

The plots shown in figure 3.13a from biceps, show the EMG frequencies on the left side and the AMG frequencies on the right side. At the beginning of the contraction, the power within the EMG is at fairly low levels within the range 0-150 Hz., the median frequency was calculated to be 63 Hz. The plot from the middle of the contraction shows no change in the range, although there is more power associated with the frequencies. The median frequency showed a slight decrease being 55Hz. The final plot shows no change in the overall range of the frequencies although the median frequency decreased to 49Hz and there was more power within the EMG signal over the frequency range. The AMG frequencies shows no distinct trends in either median frequency, the range of frequencies or the power within the range.The median frequency for all of the plots is 15 Hz, the range of frequencies is

0-30Hz, and the power within the range is difficult to determine due to the appearance of one main frequency spike and very low levels at the other frequencies.

The median frequencies from the beginning, middle and end of the contractions are shown in figure 3.13b and are displayed as the mean and the standard error of the mean. Within the biceps brachii EMG signal there is a clear trend for the frequency to decrease from beginning to end of the contraction and this is supported statistically. The difference between the beginning and middle is significant at the level $p=0.002$, and between beginning and end there is a significant difference $p=0.002$. There is less significance in the difference in frequencies between middle and end, $p=0.02$. The AMG frequencies do not show any trend and would seem to remain at a constant level around 16Hz and this is supported by there being no significant difference ($p>0.05$) between the frequencies at any stage during the contraction.

The frequency plots from one contraction of triceps brachii are shown in figure 3.14a. As with biceps brachii, the EMG signal appears to gain more power over the range of frequencies as the contraction progresses. The range remains the same, 0-150Hz, from beginning to end. The median frequency shows only very slight changes, being 50Hz, 50Hz, and 48Hz for the beginning, middle and end plots respectively. AMG does not show many changes either with the range remaining 0-30Hz, the median frequency being 15Hz, 15 Hz and 12Hz for the beginning, middle and end plots respectively. The power of the frequencies over the frequency range is very low for both the beginning and middle plots, although the end plot shows a large increase in the power at 12Hz, increasing by a factor of 4. The frequencies from the fatiguing contraction of triceps brachii are shown in figure 3.14b. The EMG frequencies seem to fall from the beginning to the end of the contraction, but there is no significant difference ($p>0.05$) between the frequencies at any of the stages of the contraction. The AMG frequencies show similar trends

with the frequency falling slightly from the beginning to the end of the contraction. However, there is no significant difference ($p>0.05$) between the frequencies at any stage during the contraction.

Figure 3.11a : Copy of trace showing a contraction of biceps brachii at 75% of the maximal voluntary contraction held to fatigue.

Figure 3.11b : Plot showing force, rms EMG and rms AMG against time from the contraction of biceps brachii shown in figure 3.11a.

Figure 3.11a

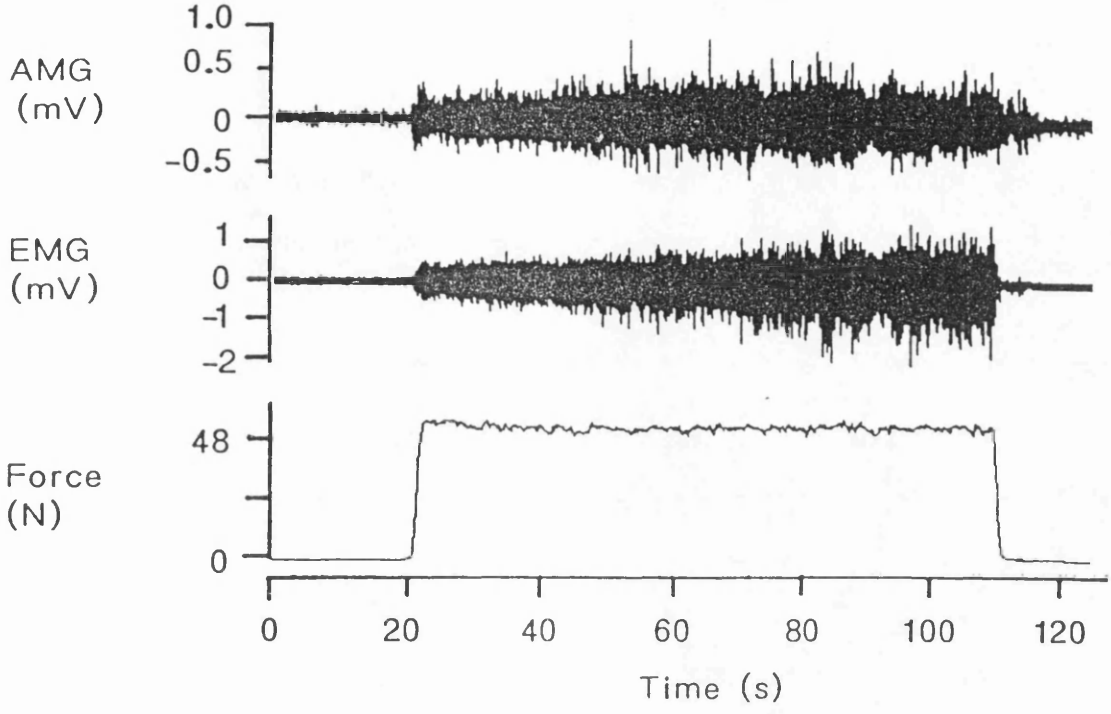


Figure 3.11b

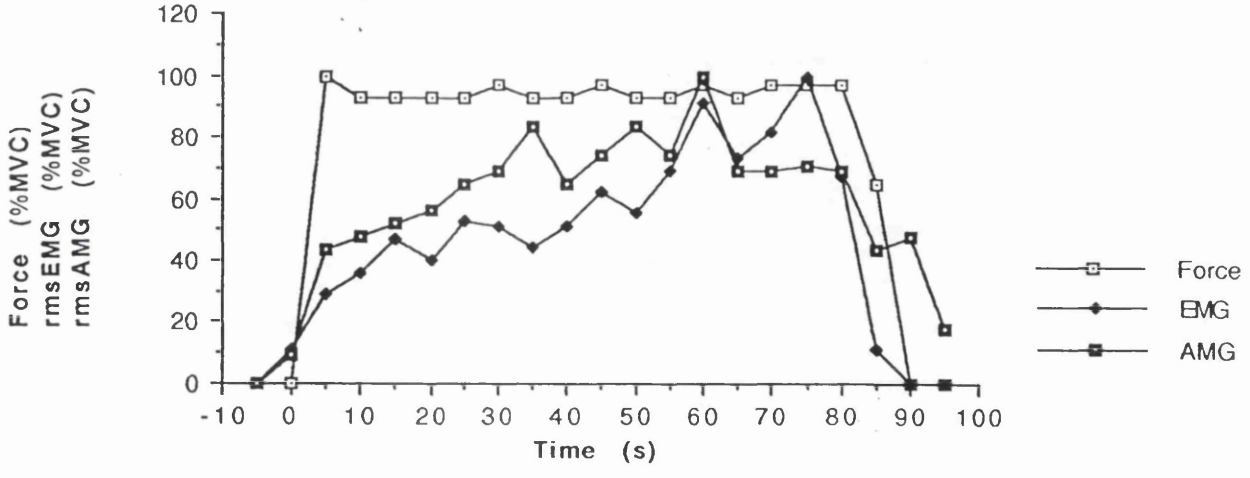


Figure 3.12a : Copy of trace showing a contraction of triceps brachii at 75% of the maximal voluntary contraction held to fatigue.

Figure 3.12b : Plot showing force, rms EMG and rms AMG against time from the contraction of biceps brachii shown in figure 3.12a.

Figure 3.12a

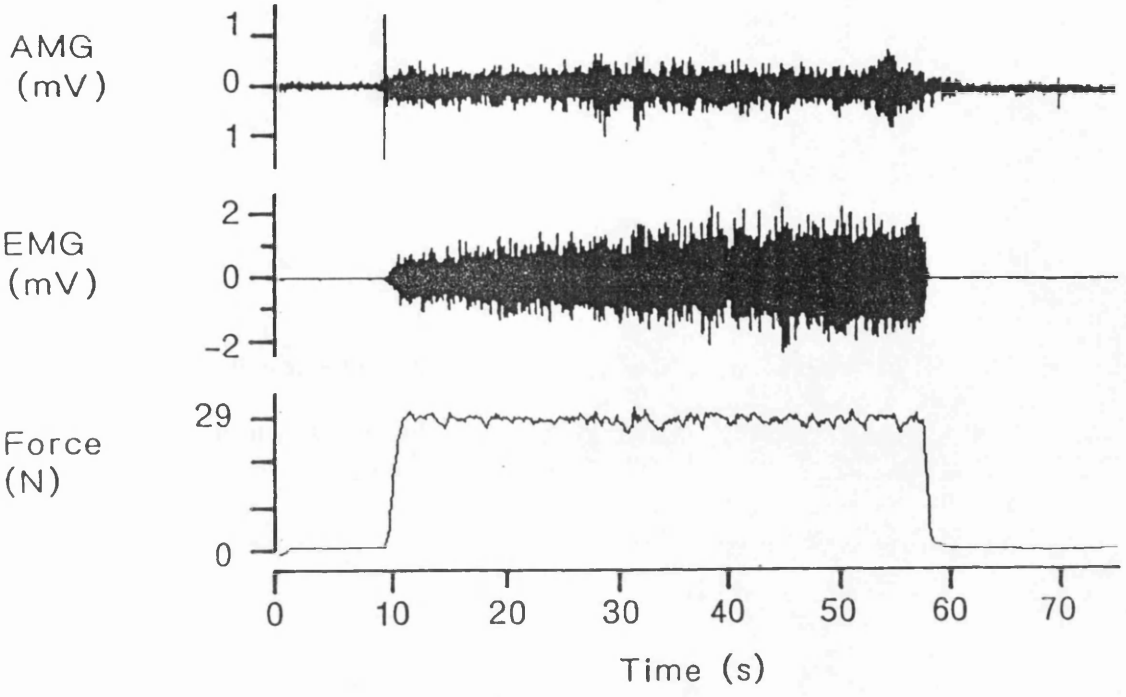


Figure 3.12b

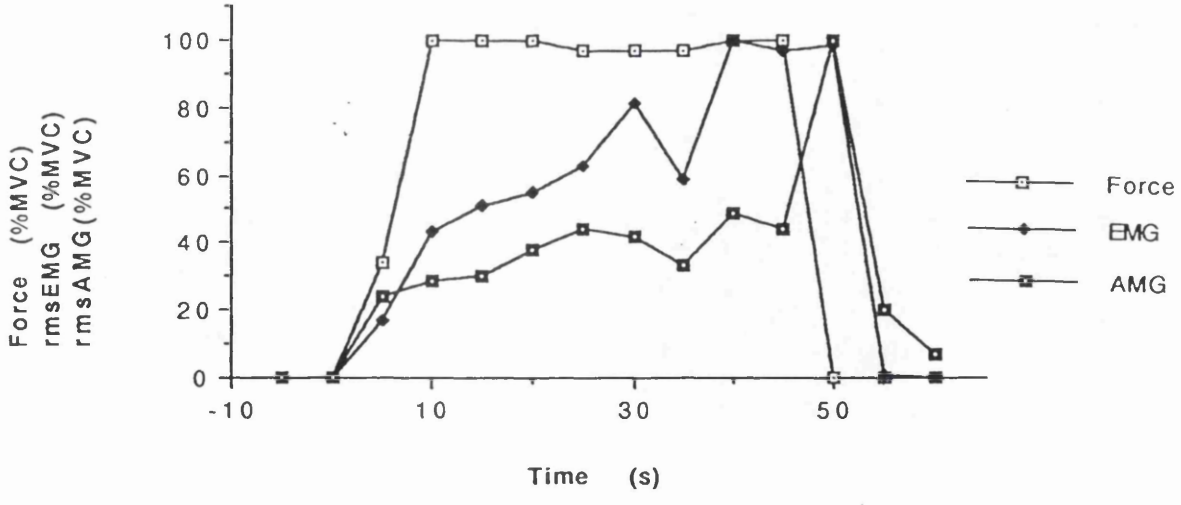


Figure 3.13a : Frequency plots from the beginning (B), middle (M) and end (E) of the contraction of biceps brachii shown in figure 3.11a.

Figure 3.13b : Plot showing the mean and standard error of the mean of the median frequencies from the beginning, middle and end of the contractions of biceps brachii at 75%MVC, from all subjects (n=8).

Figure 3.13a

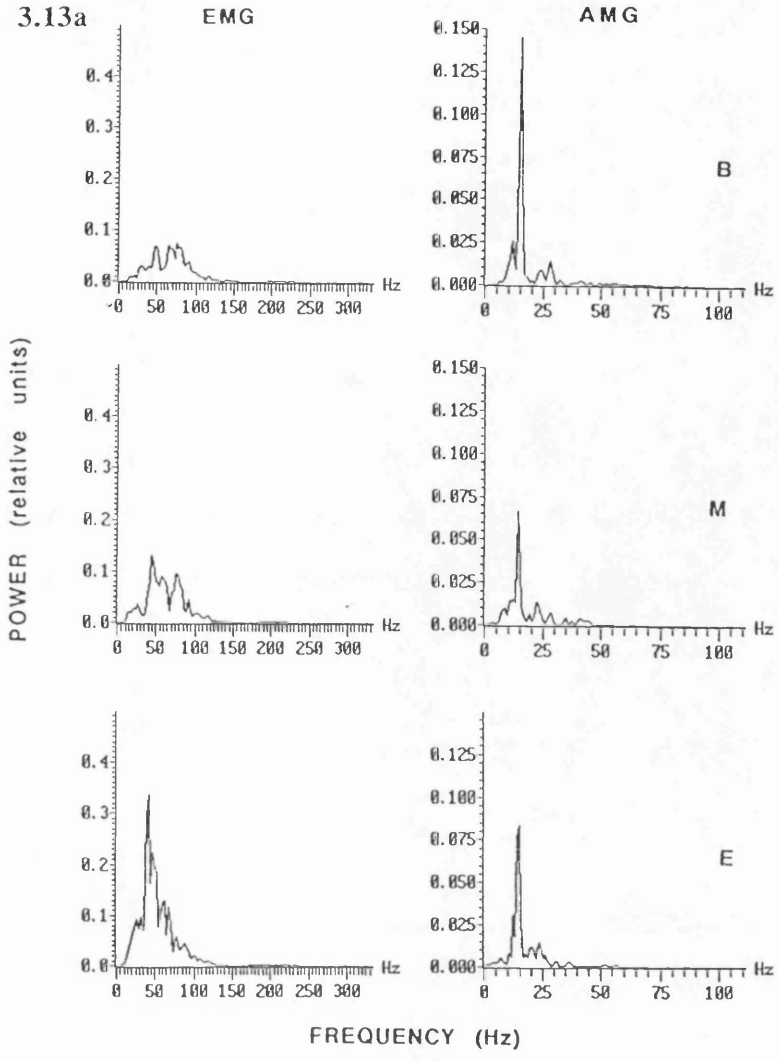


Figure 3.13b

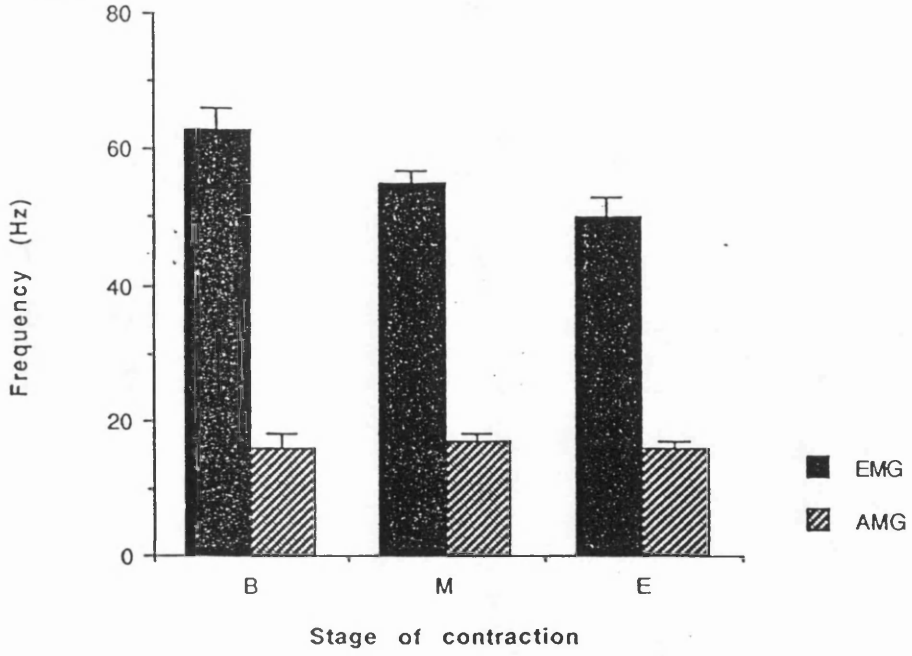


Figure 3.14a : Frequency plots from the beginning (B), middle (M) and end (E) of the contraction of triceps brachii shown in figure 3.12a.

Figure 3.14b : Plot showing the mean and standard error of the mean of the median frequencies from the beginning, middle and end of the contractions of triceps brachii at 75%MVC, from all subjects (n=8).

Figure 3.14a

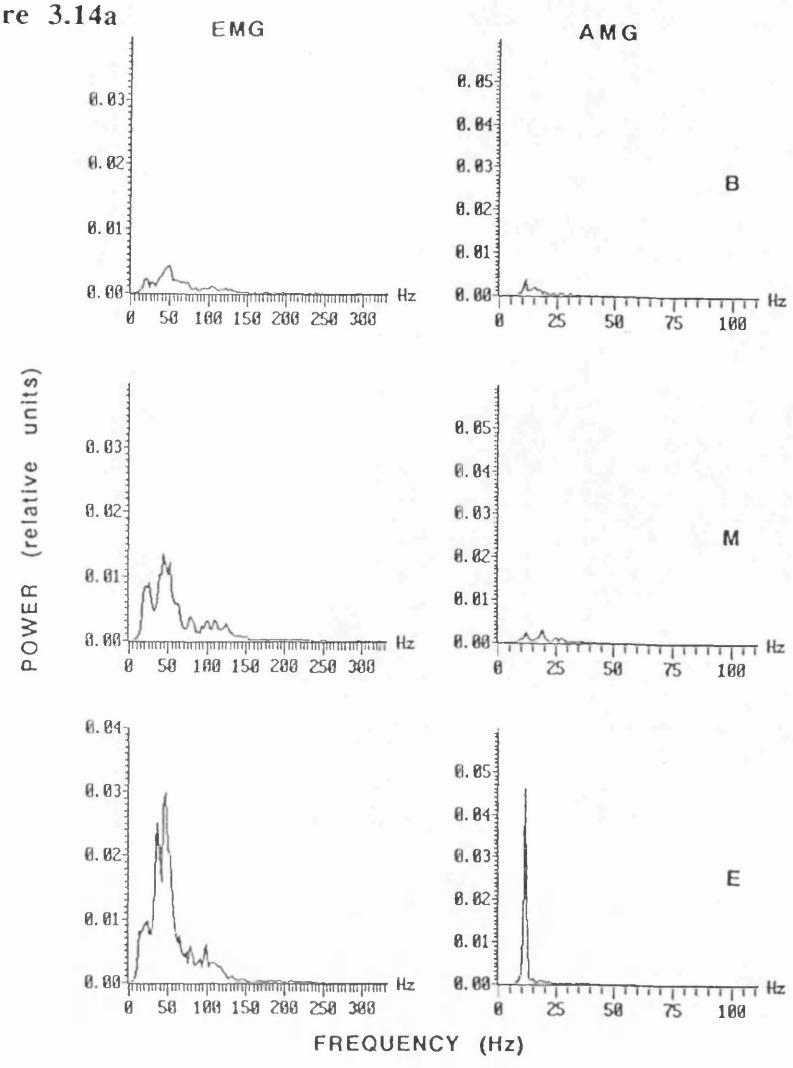
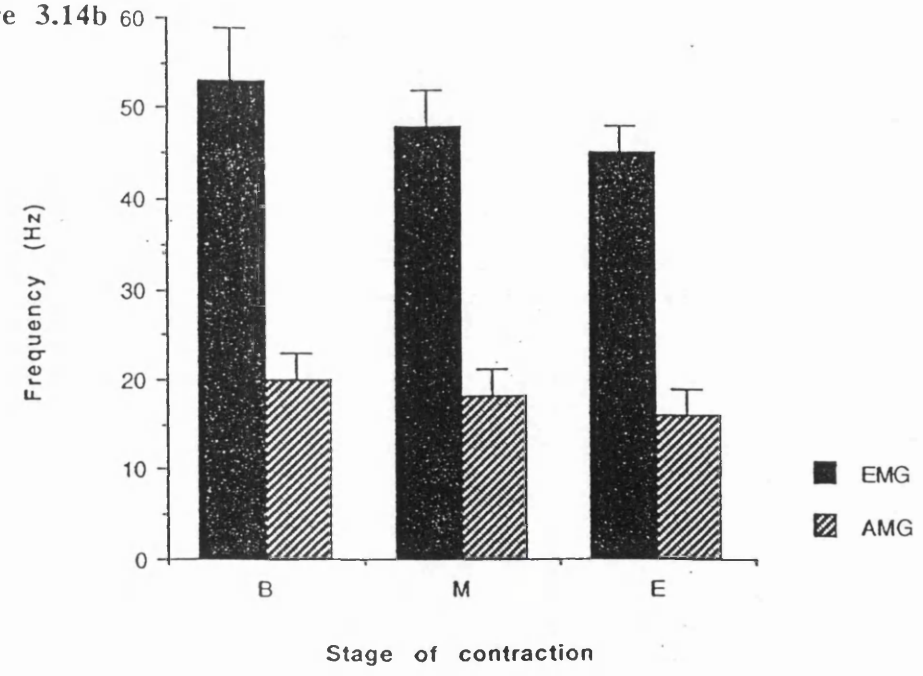


Figure 3.14b



Isometric contractions of First Dorsal Interosseus and Adductor Pollicis

Subjects

The subjects (n=8) were all normal without any history of neuromuscular disorder. Both male and female subjects participated with an age range of 24-33.

Results

The illustrative figures and the summary plots are shown at the end of the section associated with the data being discussed.

a. Contractions at percentages of the maximal isometric contraction.

As before, the subject was asked to make a maximal contraction from which the percentages were calculated. The series of contractions was repeated 3 times and the signal levels were analysed.

One series of contractions from first dorsal interosseus is shown in figure 4.1a. The AMG signal is the top trace, EMG is the middle trace and force is shown as the bottom trace. The forces shown are 25, 50, 75 and 100% and the reverse of this series, 100, 75, 50 and 25%. It must also be noted that the force trace does not originate from the zero force level. This does not indicate that the subject was already producing force, but rather that the force signal had not been balanced at the start. The linearity of the transducer was tested from this starting point and was found to be unchanged from the balanced signal. The maximal contraction from this subject is 4.5N. Both the EMG and the AMG amplitudes rise with the increase in force. The EMG signal increases in regular steps until the 100% contractions, when

there does not seem to be much change in amplitude from 75% to 100% in the data from this particular subject. This is probably due to the first dorsal interosseus contracting close to the maximal level before the middle 2 contractions and the extra force being produced by other muscles, principally the forearm muscles. The implications of this will be discussed later. The AMG signal is at much lower levels, but the amplitude increases with the increase in force. It appears to be slightly more variable than EMG, with some noise in the periods between contractions which may have been due to the subject moving or rubbing the finger close to the muscle.

The adductor pollicis muscle was used with contractions being performed at percentages of the maximal force and an example of a trace is displayed in figure 4.1b. As before, from top to bottom the signals are AMG, EMG and force. The maximal contraction for this subject was 25N. EMG and AMG both display increases in amplitude as the force increases. There is very little noise between contractions on the EMG trace and this is in contrast to the AMG trace. There is quite significant noise, seen as spikes and bursts of activity between contractions on the AMG trace. Also evident are the large amplitude spikes at the beginning and end of contractions due to movement.

Performing rms analysis on the signals during the isometric contractions of first dorsal interosseus yields the data which is shown in figure 4.2a. The data is clustered in four groups but there is variation in the force levels achieved and the percentage levels of EMG and AMG, resulting in some spread of the data. However, a general trend emerges with both the EMG and AMG increasing with the increase in force, with slightly more scatter within the AMG than the EMG at each force level. The mean and the standard error of the mean were calculated for each of the four force groups and this is displayed in figure 4.3a. It is very clear that with the increase in force EMG and AMG rise, with the rms signals having

higher percentage values than the force for the 25 and 50% contractions, and lower percentages for the 75 and 100% contractions.

Figure 4.2b shows the rms data from contractions of adductor pollicis. The data shows that an increase in force is accompanied by an increase in both the rmsEMG and rmsAMG. There is some grouping of the data into the four force levels, but there is a certain amount of variation in the force achieved by the subjects. It can also be seen that there is variation in the rmsEMG and rmsAMG percentages. However, there is virtually no difference in the amount of scatter for the EMG and AMG signals with the two populations overlapping. Thus, EMG and AMG would appear to have very similar relationships with force.

Figure 4.3b displays the summarised data showing the mean and standard error of the mean of the data for the four force levels. Clearly, there is a close relationship between force and the AMG and EMG signals, implying a real trend for EMG and AMG to increase with increasing force.

Median frequency

A frequency plot from a maximal contraction of first dorsal interosseus is shown in figure 4.4a. Frequency (in Hertz), is shown as the x axis for both the plots and the power is the y axis which does not have units and is, therefore, a relative value. The EMG has a wide range of frequencies associated with it, between 0 and 200Hz. The frequencies with the most power seem to be in the range, 30-150 Hz. The median frequency was calculated to be 72Hz. AMG has a much narrower range, 0-25Hz, and the median frequency is 13Hz. The plots from all the contractions were examined and the range did not change for EMG or AMG.

Figure 4.4b shows the mean and standard error of the mean of the median frequencies from the contractions. The median frequencies of EMG are significantly higher than AMG ($p<0.0001$). The EMG frequency tends to slightly decrease with

the increase in force although there is no significant difference between the frequencies. AMG shows similar trends, with the frequency at 25% being significantly higher ($p=0.05$) than the frequency at 100%. The frequency at 75% is also significantly higher ($p=0.04$) than the frequency at 100%. There is no difference ($p>0.2$) between any of the other frequencies.

The frequency plots from a maximal contraction of adductor pollicis are shown in figure 4.5a. EMG is on the left and AMG on the right, frequency is shown as the x axis for both plots and the power of the signal as the y axis. The EMG has a wide range, 0 - 300Hz, although, the frequencies with the most power are in the range 0-130Hz. The median frequency is 83Hz. AMG has a much narrower range 0-50 Hz and the median frequency is 17Hz. This is shown as an example of the frequency plots calculated. Overall, the contractions there was no change in the range of frequencies of either the EMG or the AMG signals and so the median frequency was chosen as the variable to study.

Figure 4.5b shows the mean and standard error of the mean of the median frequencies from each contraction level of adductor pollicis. The frequency of the EMG signal seems to rise for the 50% contraction and then return to the previous frequency for the other contractions. This observation is supported by there being a significant difference between the frequencies at 50% and 25% ($p=0.004$), 50% and 75% ($p=0.06$) and 50% and 100% ($p=0.009$). There does not seem to be any change in the frequencies of the AMG signals, and there is no significant difference between them ($p>0.1$).

Figure 4.1a : Copy of trace from contractions of 1st dorsal interosseus at percentages (25, 50, 75, 100, 100, 75, 50, 25) of the maximal contraction.

Figure 4.1b : Copy of trace from contractions of adductor pollicis at percentages (25, 50, 75, 100, 100, 75, 50, 25) of the maximal contraction.

Figure 4.1a

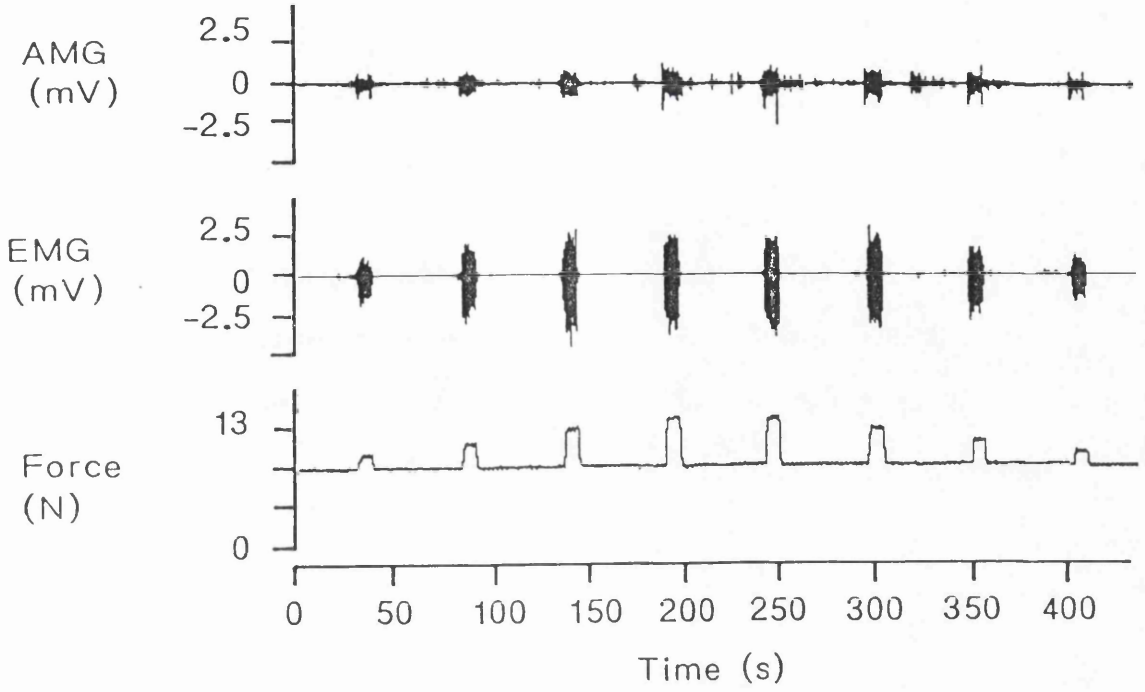


Figure 4.1b

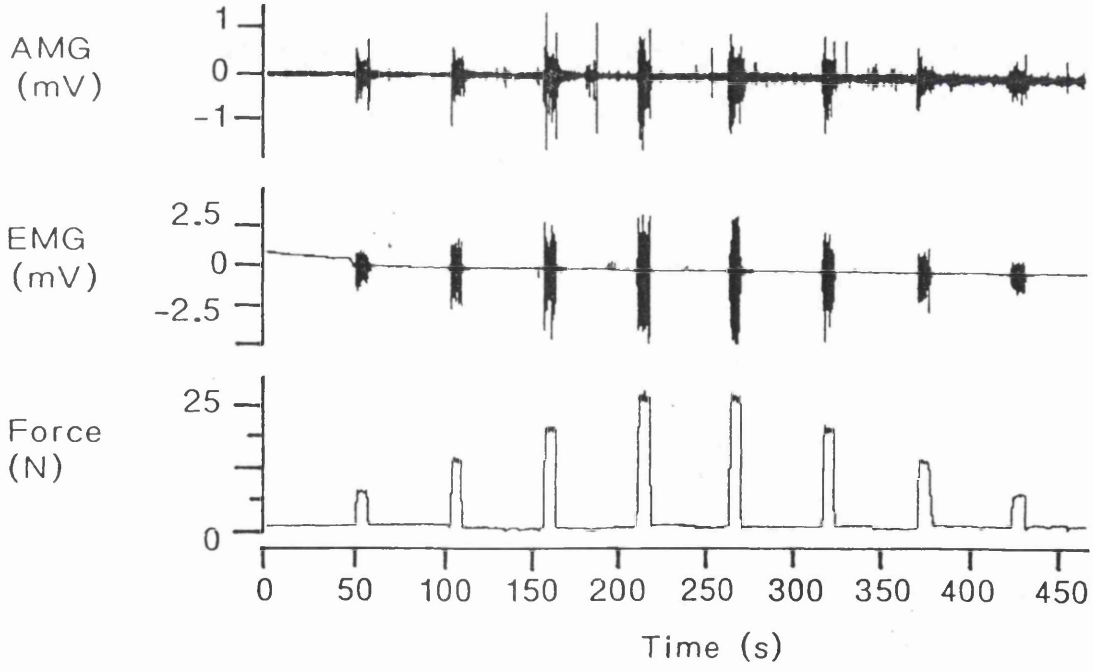


Figure 4.2a : Graph showing root mean square EMG and AMG data from all of the subjects (n=8) plotted against the percentage of the maximal voluntary force from 1st dorsal interosseus.

Figure 4.2b : Graph showing root mean square EMG and AMG data from all of the subjects (n=8), plotted against the percentage of the maximal voluntary force from adductor pollicis.

Figure 4.2a

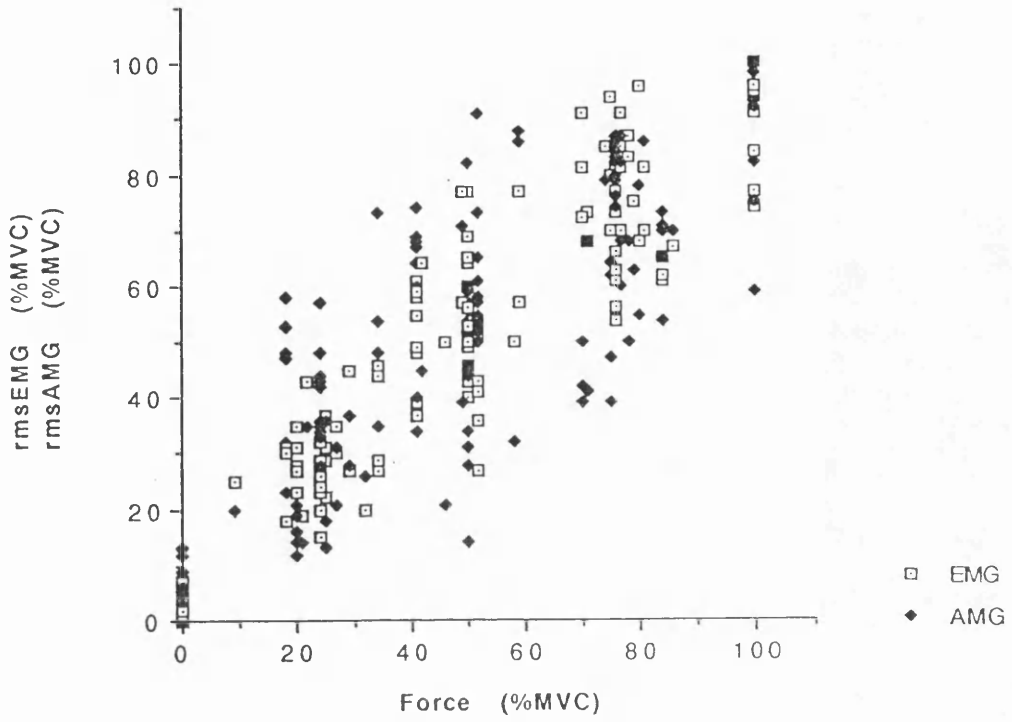


Figure 4.2b

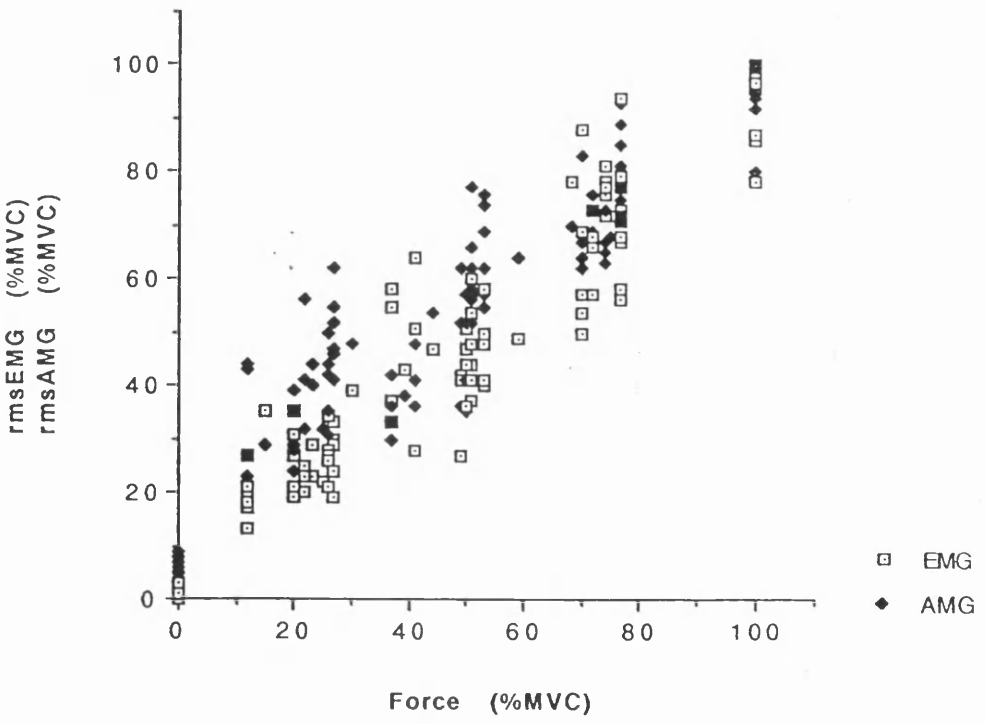


Figure 4.3a : Graph showing data from figure 3.2a, rms EMG and AMG against force, plotted as the mean and standard error of the mean, from contractions of 1st dorsal interosseus. Regression equations: EMG vs Force $y = 6 + 0.9x$, $r^2 = 1.0$; AMG vs Force $y = 10 + 0.8x$, $r^2 = 0.99$.

Figure 4.3b : Graph showing data from figure 3.2b, rms EMG and AMG against force, plotted as the mean and standard error of the mean, from contractions of adductor pollicis. Regression equations: EMG vs Force $y = 4 + 0.9x$, $r^2 = 1.0$; AMG vs Force $y = 13 + 0.8x$, $r^2 = 0.99$.

Figure 4.3a

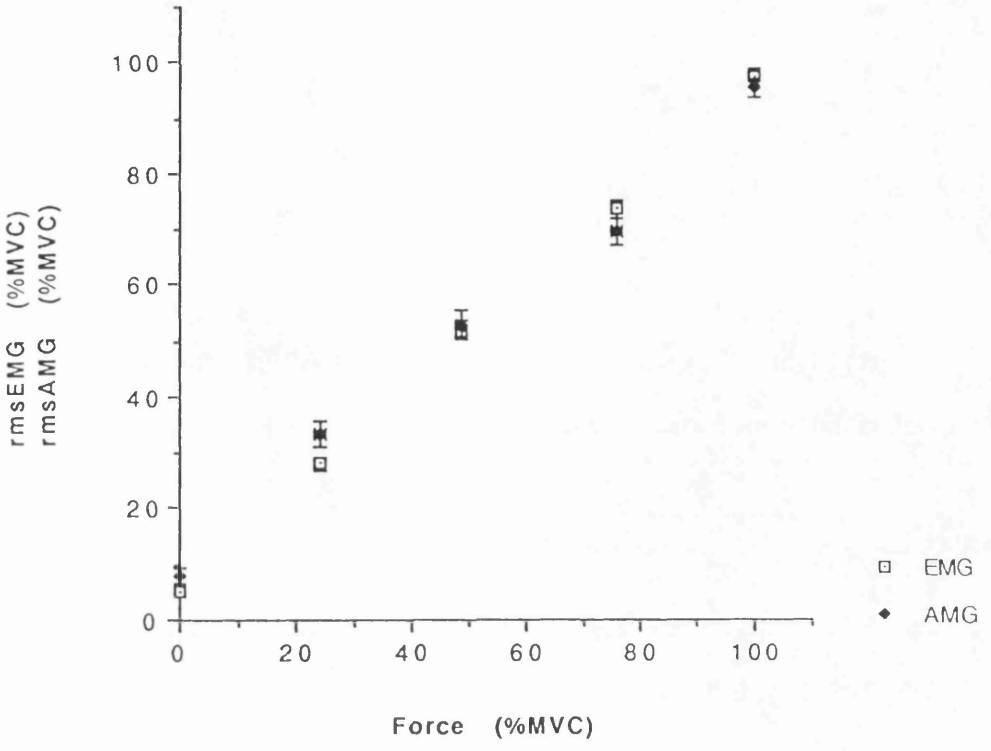


Figure 4.3b

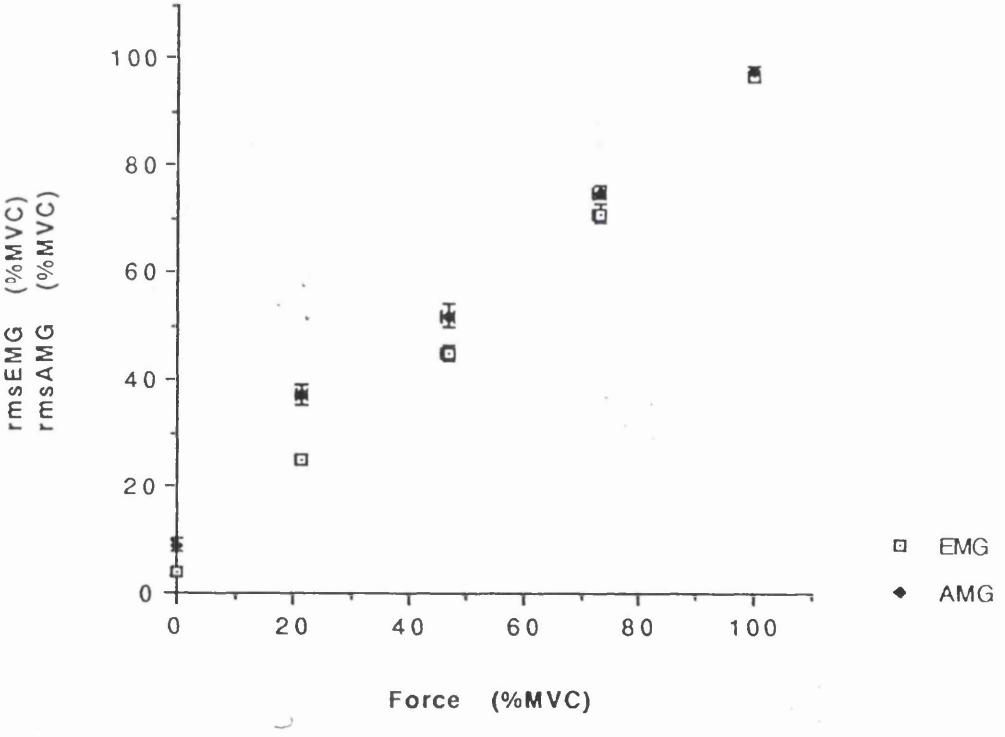


Figure 4.4a : Frequency plots of EMG and AMG from a maximal contraction of 1st dorsal interosseus.

Figure 4.4b : Bar chart showing the mean and standard error of the mean of the median frequencies of EMG and AMG, from the series of contractions at percentages of the maximal contraction of 1st dorsal interosseus, from all subjects (n=8).

Figure 4.4a

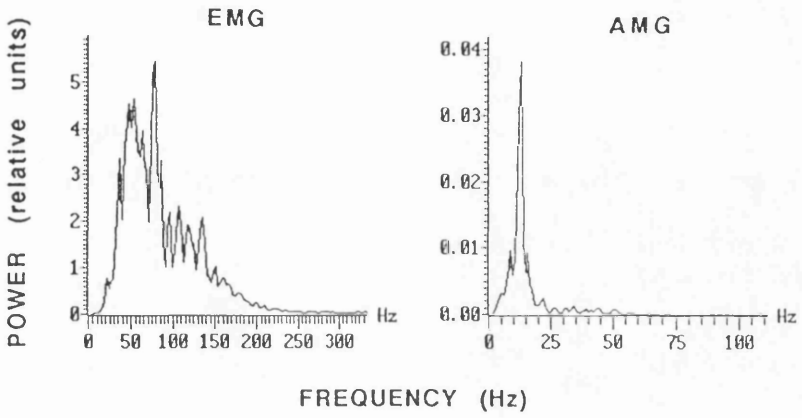


Figure 4.4b

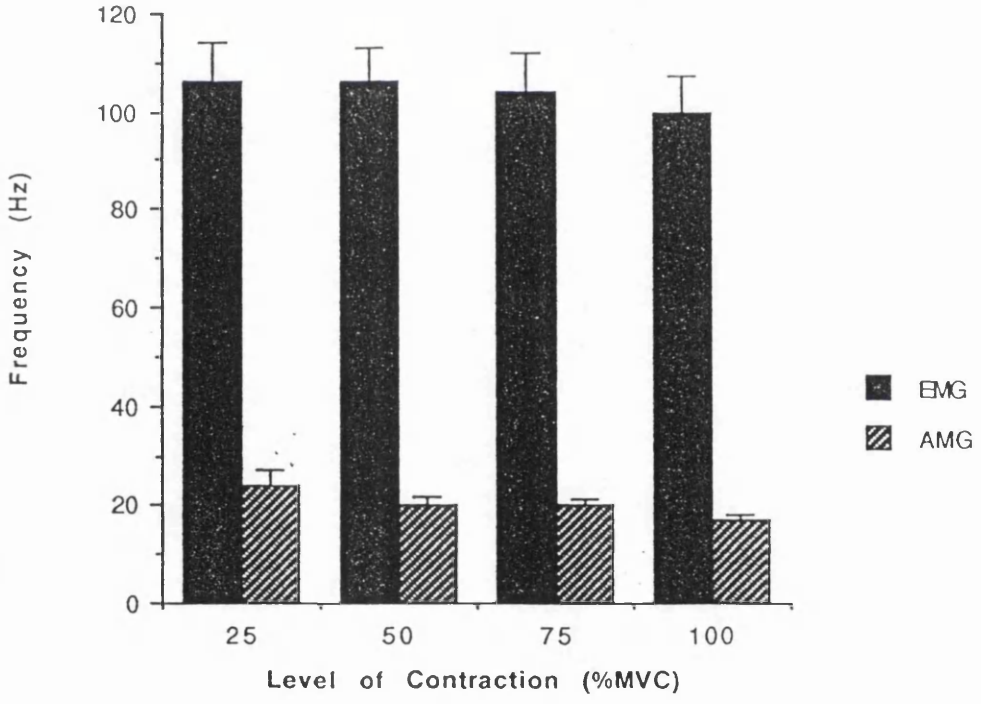


Figure 4.5a : Frequency plots of EMG and AMG from a maximal contraction of adductor pollicis.

Figure 4.5b : Bar chart showing the mean and standard error of the mean of the median frequencies of EMG and AMG, from the series of contractions at percentages of the maximal contraction of adductor pollicis, from all subjects (n=8).

Figure 4.5a

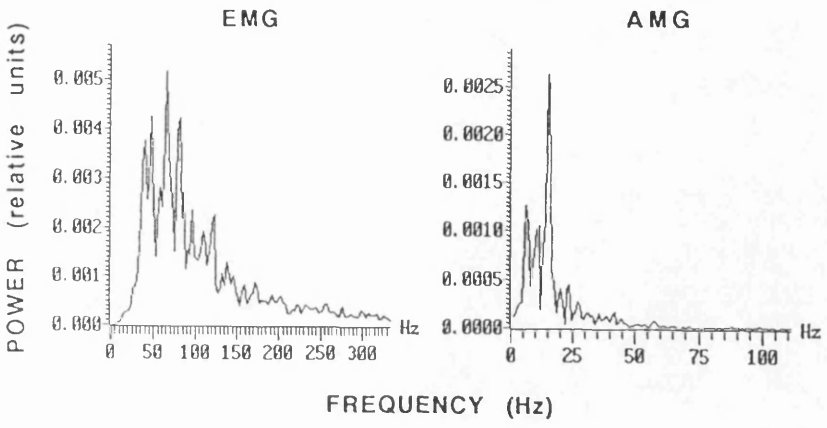
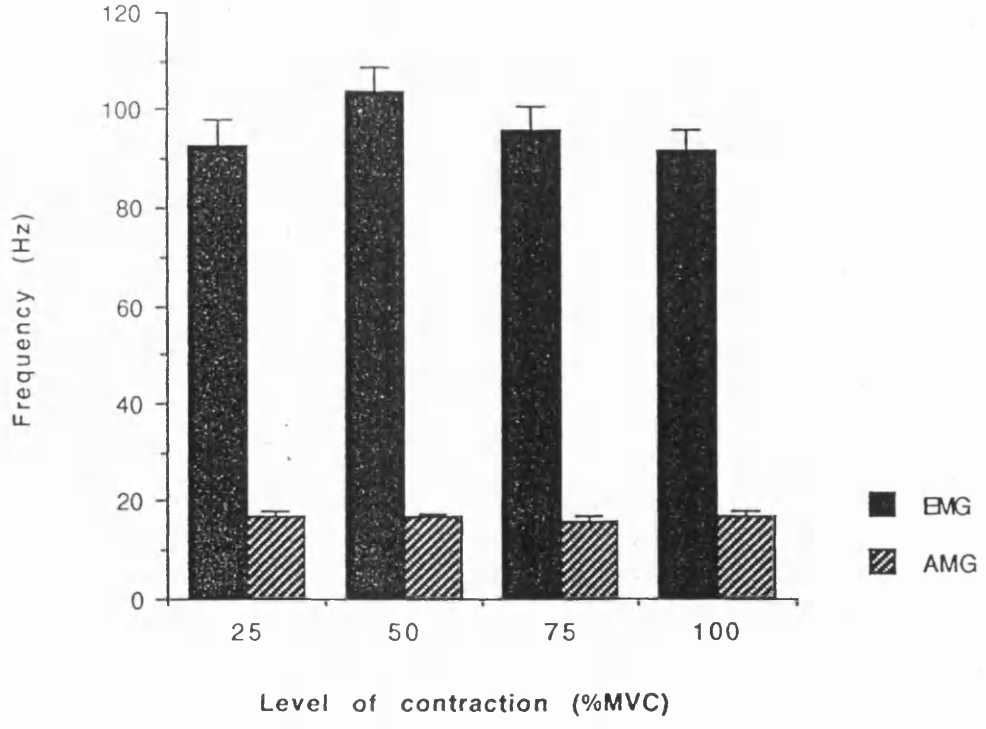


Figure 4.5b



b. Ramp contractions of 1st dorsal interosseus and adductor pollicis.

Figure 4.6a shows a trace of two ramp contraction of 1st dorsal interosseus. As can be clearly seen force, the bottom trace, rises to a maximum level of 4.5N over a period of 20 seconds and then the subject relaxes gradually and the force decreases back to 0N over 20 seconds in the first contraction and similarly in the second contraction with the maximal force being 4.5N. It should be noted that the force trace does not start from a zero position due to the force transducer not being balanced at the start of this series of contractions. The transducer was tested for linearity from this starting position and was found to be unchanged from the balanced signal. The middle trace, the EMG record, shows a steady increase in amplitude in the first contraction from beginning to the middle at the maximal force position, and then a steady decrease in amplitude to the end of the contraction. The amplitude at the middle of the contraction is approximately 15 times the amplitude at the beginning of the contraction. The EMG record for the second contraction shows very similar results although the amplitude at the maximum force level is not as large as the first contraction. The amplitude here is approximately 10 times the initial amplitude. AMG shows a similar relationship although there is more variation in this signal than in EMG. At the beginning of the contraction there is a large movement artifact as the subject moves into position. After this the signal levels off and then increases in amplitude from that point. At the maximal force level the amplitude is around 3 times the initial level. At the height of the contraction there are some large 'spikes' visible, after which the amplitude of the signal decreases as the force decreases, until the end of the contraction. There is still some AMG signal recorded after the contraction is over and the force level has returned to zero, although this quickly diminishes and the signal returns to the level seen before the contraction. In between the contractions there is some low level noise recorded, although there is very little accompanying EMG signal. Most of the AMG will be due to the subject moving or touching the accelerometer. However, on closer

inspection, there is some very low level EMG, and bearing in mind that the two traces are shown on different scales with the AMG being amplified less than EMG, it is quite possible that there is more EMG than is evident from this trace. The second contraction does not show a definite movement artifact at the start of the contraction although there is some noise before the contraction begins. The amplitude increases during the contraction although it is less well defined than the first contraction and it shows a more gradual increase. At the height of the contraction the amplitude is 1.5 to 2 times the amplitude seen at the beginning of the contraction. The amplitude then decreases until there is no force being produced. At the end of the contraction there is a movement artifact, after which the amplitude returns to the levels that were seen before the contraction.

The ramp contractions from adductor pollicis shown figure 4.6b, show very similar relationships between force, EMG and AMG as the contractions from first dorsal interosseus. As before, force is the bottom trace, EMG the middle trace and AMG the top trace. Both contractions last for 35 seconds in total, and the maximal force reached is 19N for both. The EMG trace has a low noise level before and after the contractions, and when the contraction begins the amplitude increases. There is a steady increase until the maximal force is reached and then the amplitude decreases to pre-contraction levels. The second contraction is very similar with there being a steady rise in amplitude to the maximal level which occurs at the maximal force level and then a steady decrease to the end of the contraction. It must be noted that the AMG scale is much lower than the EMG scale, and so it appears as if there are high noise levels throughout the rest periods of the contraction. This is perhaps more correctly interpreted as the signal during the contraction being at low levels. During the first contraction, the amplitude increases during the contraction to a maximum level and then decreases again to the end of the contraction. The second contraction is very similar with the same profile of increasing and decreasing AMG.

Analysis of the rms values yields the plots for first dorsal interosseus and adductor pollicis seen in figures 4.7a and 4.7b, respectively. Figure 4.7a shows a very tight relationship between force, rmsEMG and rmsAMG with all three increasing to the maximum levels, and then decreasing by similar amounts during the contraction. The AMG signal does not reach the zero level at any point, neither at the beginning nor the end of the contraction, reflecting the high noise level seen on the trace in figure4.6a. Figure 4.7b shows that EMG and AMG increase with the increase in force, although there seems to be some evidence of both the EMG and AMG levels lagging behind the force increase, and leading the force decrease. This description is true for this contraction but was not always the case, and examples where there was no difference in the timing of the increases and decreases, were also seen from the data.

As the contractions were all of slightly different length, this makes it difficult to show summarised data. The plot shown in figure 4.8a shows the summary data from first dorsal interosseus plotted against force and figure 4.8b shows the corresponding data for adductor pollicis. Both graphs show a considerable amount of scatter in both the EMG and AMG values, and this agrees with the data already discussed in the previous section from the contractions at 25, 50, 75 and 100% MVC. The upper plot shows more scatter in the AMG values than the EMG, although there is a definite trend for both the EMG and AMG signal amplitude to rise with the increase in force. The lower trace shows even more scatter of results although, the trend for increasing EMG and AMG with the increase in force is definitely evident. This is supported by regressing both EMG and AMG against force and the similarity in equations, and the calculation of the residuals showing that there were no non-linear trends within the data.

Median frequencies

Figure 4.9a shows frequency plots from the beginning, middle and end of a ramp contraction from first dorsal interosseus. Frequency is shown as the x-axis and the power within the signal is shown as the y-axis. The power is a relative value and therefore has no units. The plots from the beginning and end of the contraction show that both signals are at very low levels. There was no difference between the beginning and end range or median frequency, with the range for EMG being 0 - 200Hz with the median frequency being 100Hz, and for AMG the range 0 - 50Hz and the median frequency 22Hz. The middle plot shows considerable changes in the power within the signal, although the range was unchanged. There were no significant differences in the range of frequencies seen in the frequency plots from all of the subjects, and so the median frequency data was examined. Figure 4.9b shows the summarised data as the mean and standard error of the mean of the median frequencies from the beginning, middle and ends of the contractions. There is a clear progressive decrease in EMG frequency through the 3 stages of the contraction, although this is not supported statistically ($p>0.2$). There appears to be no such trend in the AMG median frequencies and they remain at a fairly constant level around 17Hz. This is supported by there being no significant difference between the frequencies ($p>0.2$).

Figure 4.10a shows the frequency plots from the beginning, middle and end of the ramp contractions of adductor pollicis. The x axis is frequency for both the left and right hand plots and the y axis is power. The range of frequencies for the EMG signal remains unchanged for all the plots at 0 - 200 Hz, and the AMG range of frequencies is 0 - 50Hz. The signals within these ranges are very low at the beginning and end of the contraction. The middle plot shows considerable increases in the power within the EMG and AMG frequency ranges, increasing by a factor of between 4 and 6. There is a decrease in median frequency of the EMG signal from beginning to end of the contraction, being 90Hz, 73Hz and 54Hz for the three plots

respectively. The AMG median frequency also shows a decreasing trend being 20Hz, 22Hz and 15Hz for the three plots.

These observations are supported by the summary data in figure 4.10b. The median frequencies are displayed as the mean and standard error of the mean. There is a decrease in median frequency from the beginning to the end of the contraction. After t-test, there is no difference between the frequencies at the beginning and middle, p=0.2, although there is a statistical difference between the frequencies at the beginning and end, p=0.001, and the middle and end, p=0.03. The AMG frequencies are obviously much lower than the EMG frequencies, around 15Hz. There appears to be a decrease in frequency although all values are very close. Statistically, after t-test, there is no difference in the frequencies at the beginning and middle, p=0.5. However, there is a statistical difference between the beginning and end, p=0.03, and the middle and end, p=0.07.

Figure 4.6a : Copy of a trace showing two isometric ramp contractions of 1st dorsal interosseus to maximal force.

Figure 4.6b : Copy of a trace showing two isometric ramp contractions of adductor pollicis to maximal force.

Figure 4.6a

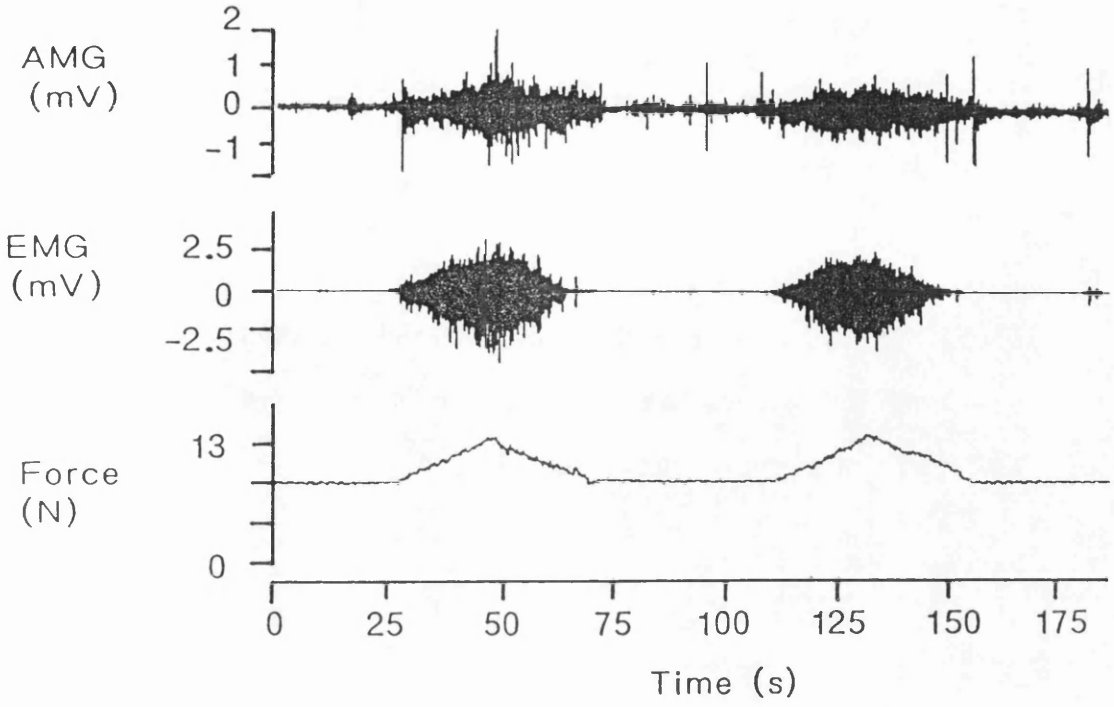


Figure 4.6b

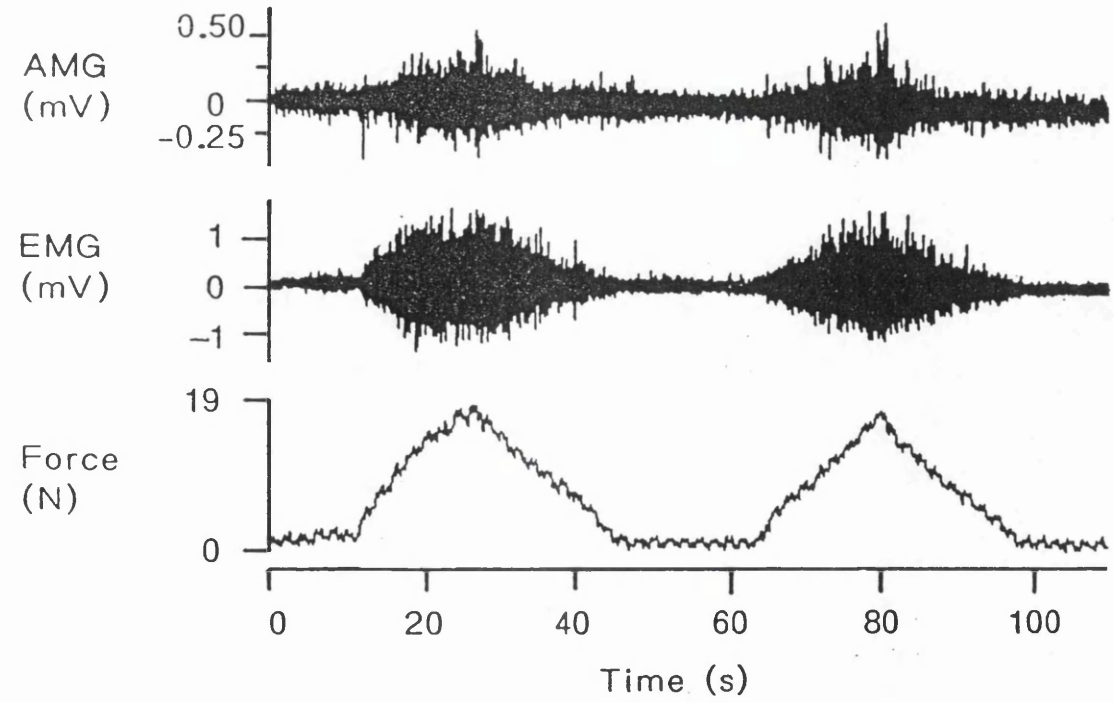


Figure 4.7a : Plot showing the force, rms EMG and rms AMG data (from the first contraction shown in figure 4.6a) against time from a ramp contraction of 1st dorsal interosseus.

Figure 4.7b : Plot showing the force, rms EMG and rms AMG data (from the second contraction shown in figure 4.6a) against time from a ramp contraction of adductor pollicis.

Figure 4.7a

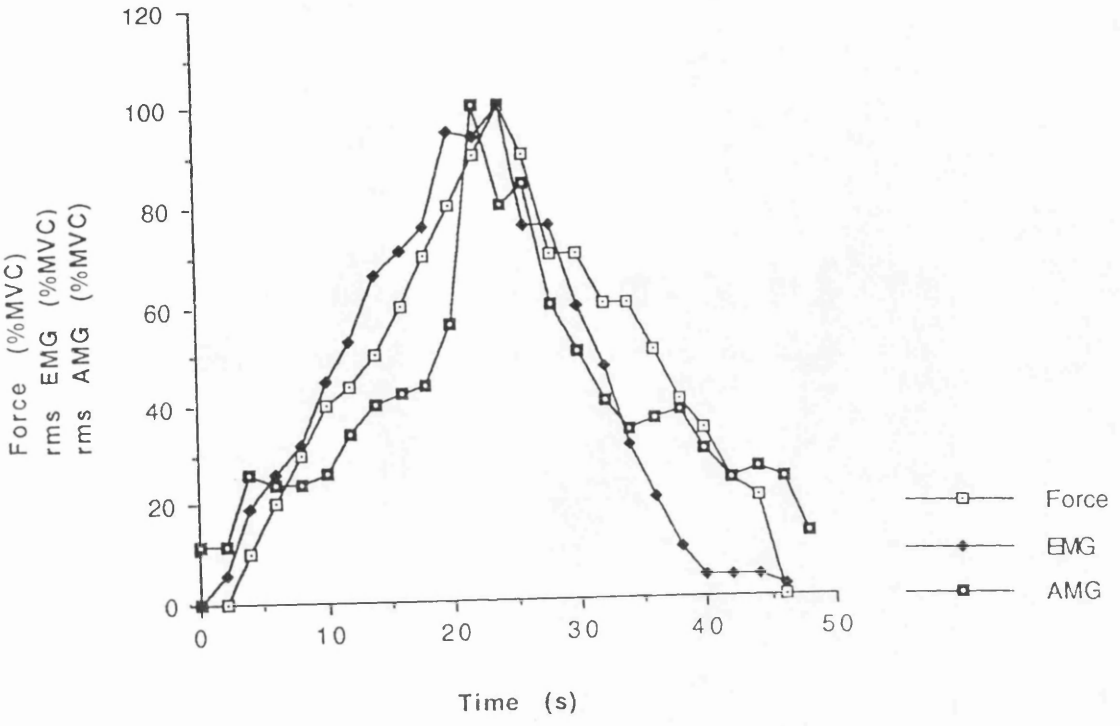


Figure 4.7b

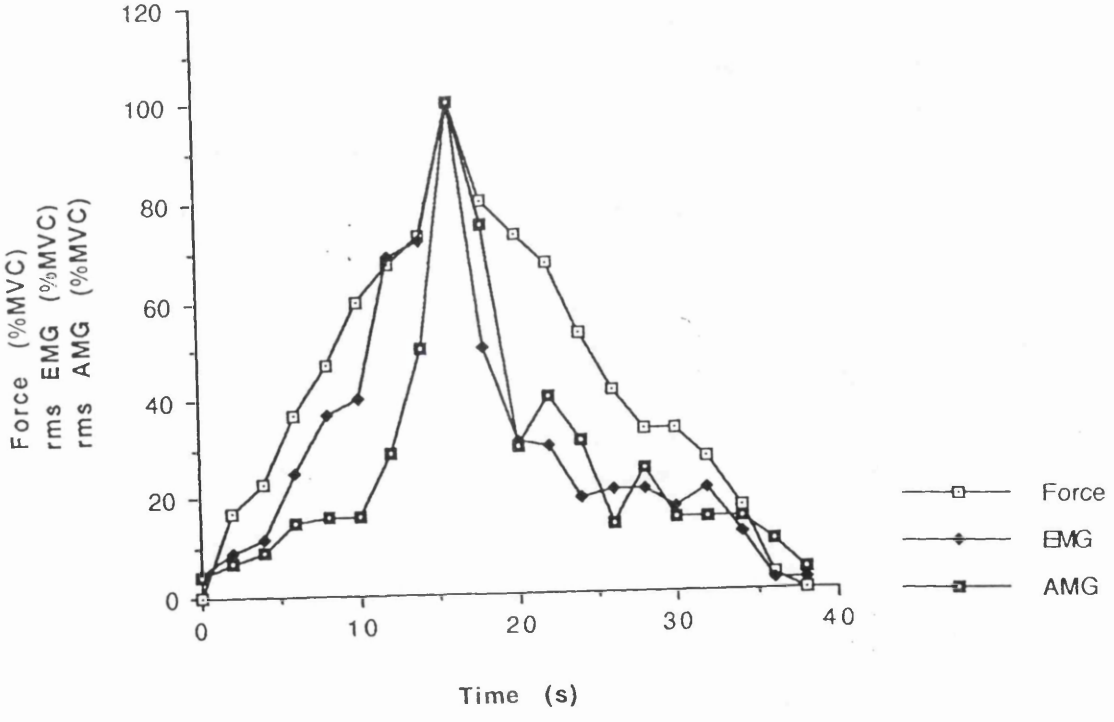


Figure 4.8a : Plot of rms EMG and AMG data against force for all of the subjects (n=8), from ramp contractions of 1st dorsal interosseus.

Figure 4.8b : Plot of rms EMG and AMG data against force for all of the subjects (n=8), from ramp contractions of adductor pollicis.

Figure 4.8a

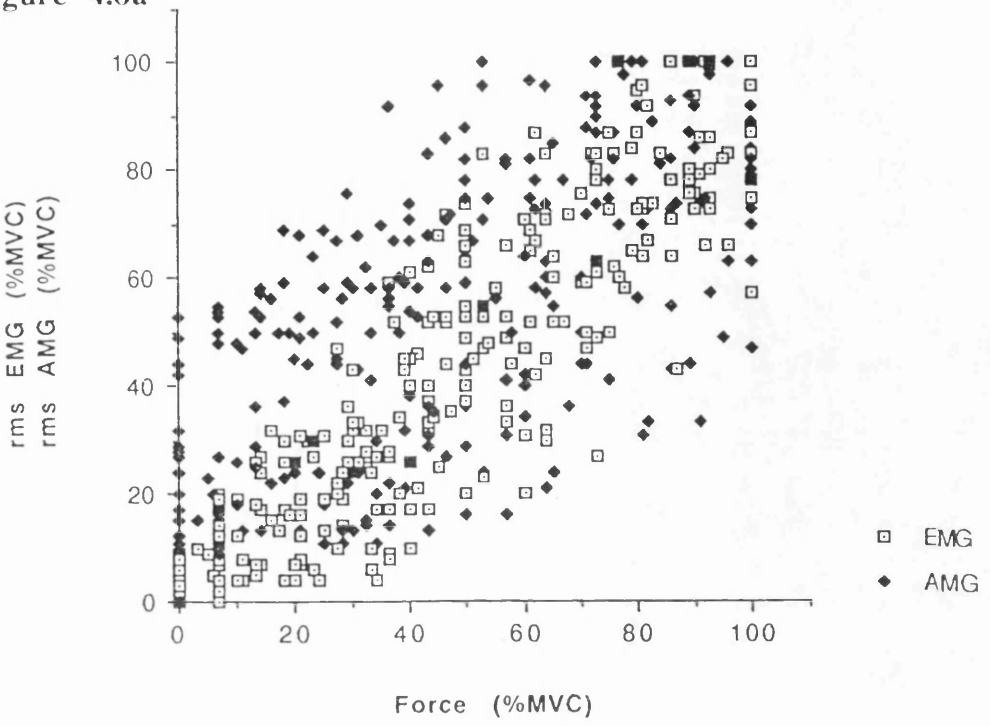


Figure 4.8b

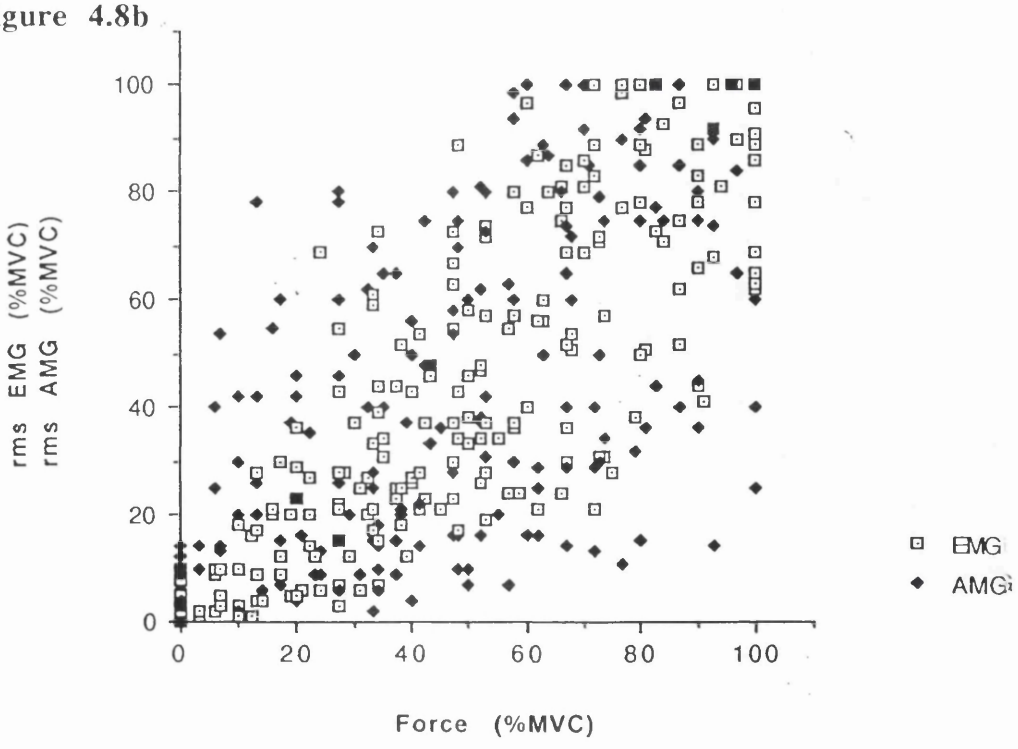


Figure 4.9a : Frequency plots from the beginning (B), middle (M) and end (E) of one ramp contraction of 1st dorsal interosseus.

Figure 4.9b : Plot showing the mean and standard error of the mean of the median frequencies from the beginning (B), middle (M) and end (E) of the ramp contractions of 1st dorsal interosseus, from all subjects (n=8).

Figure 4.9a

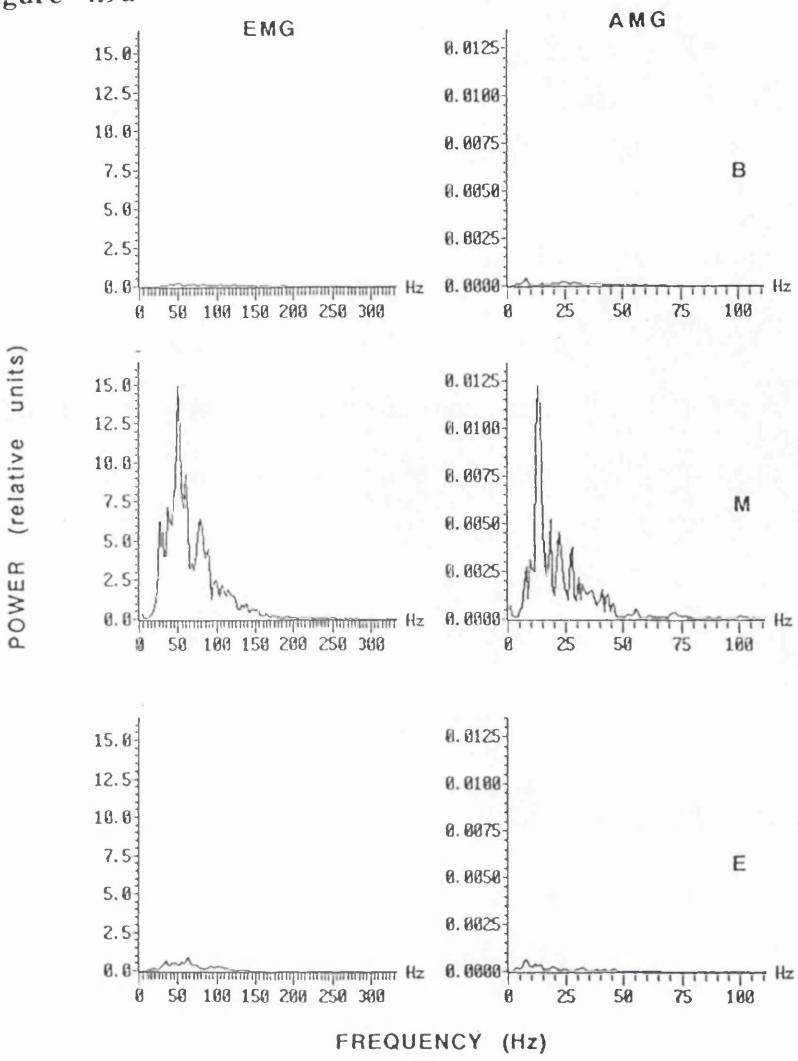


Figure 4.9b

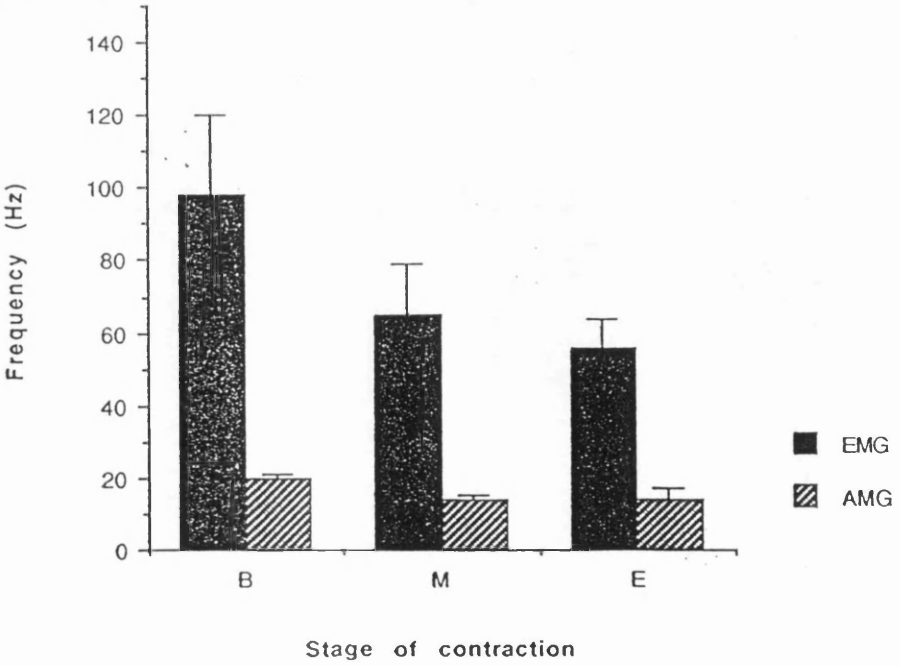


Figure 4.10a : Frequency plots from the beginning, middle and end of one ramp contraction of adductor pollicis.

Figure 4.10b : Plot showing the mean and standard error of the mean of the median frequencies from the beginning (B), middle (M) and end (E) of the ramp contractions of adductor pollicis, from all subjects (n=8).

Figure 4.10a

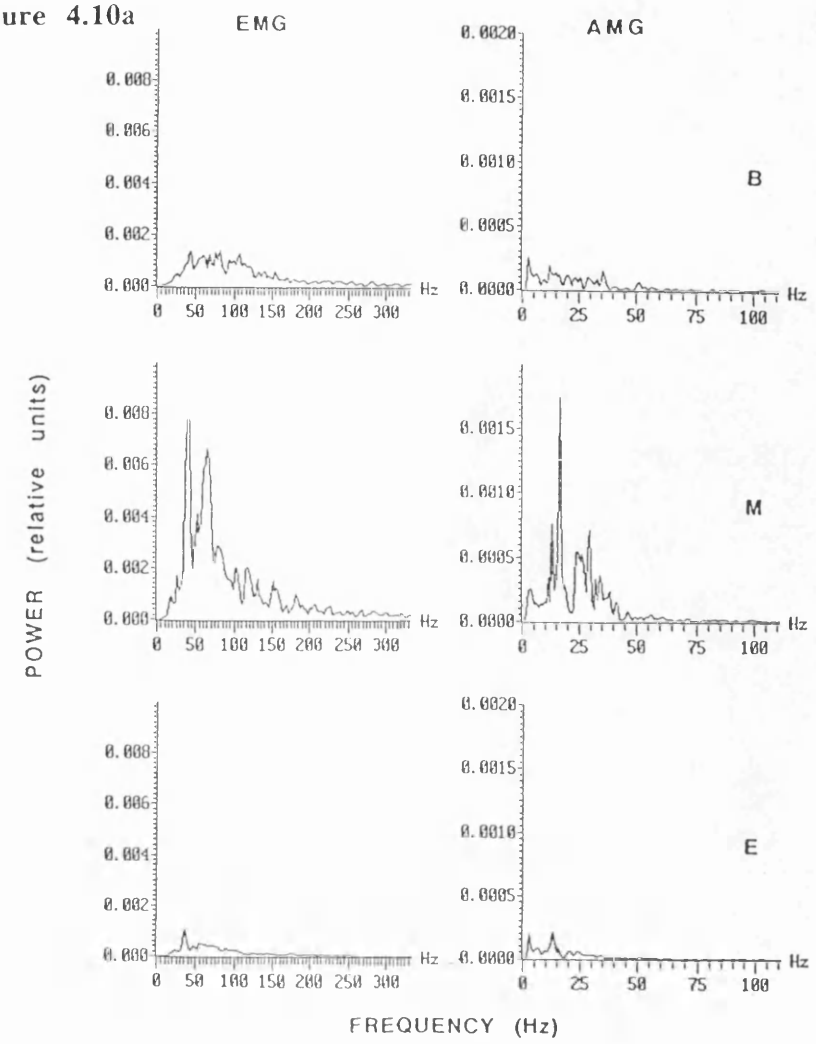
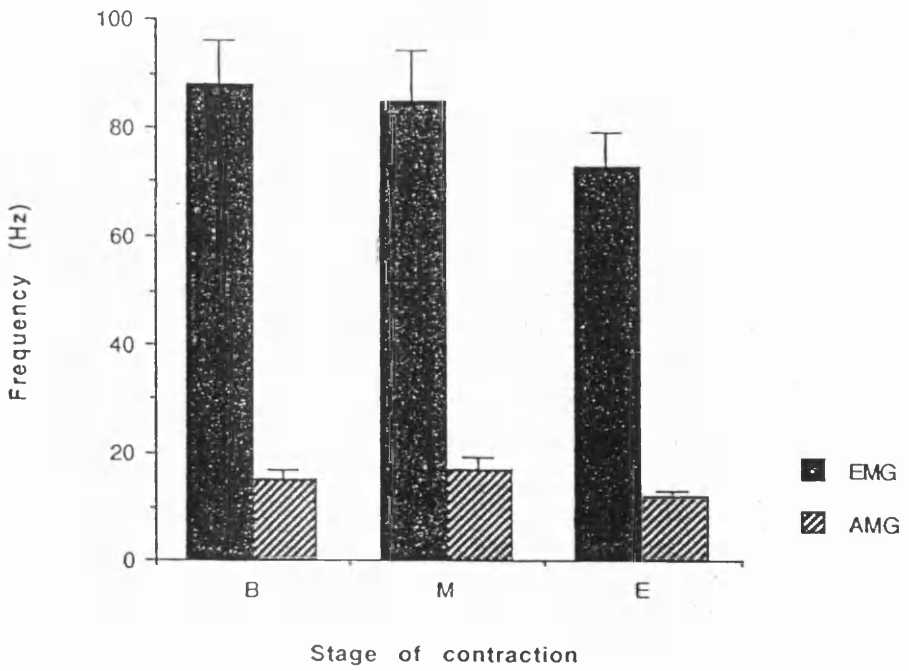


Figure 4.10b



c. Fatiguing isometric contraction at 75% of the maximal force.

The subject was asked to maintain a contraction at 75% of the maximal force determined previously.

Figure 4.11a shows the contraction from first dorsal interosseus showing force as the bottom trace, EMG as the middle trace and AMG as the upper trace. At the start of the contraction the force rises sharply to the 11N level and remains constant for 25 seconds after which the force level drops slightly to around 70%MVC and the contraction was ended with the force returning to zero. The EMG trace is silent before the contraction and the amplitude increases at the start of the contraction. There is a slight rise in the amplitude to around half way through the contraction. The amplitude remains constant for around 5 seconds until the force trace starts to waver after which the EMG decreases and then increases again before decreasing to low levels. When the contraction ends the EMG signal returns to the pre-contraction value. The AMG trace is also silent before the contraction, however the start of the contraction is indicated by a large movement artifact. The AMG amplitude increases from the start of the contraction until about half way through after which there is no further increase. When the force starts to decrease there is an accompanying decrease in AMG amplitude, and when the contraction ends there is another movement artifact and the signal returns to pre-contraction levels.

By taking the force values and rms values of the EMG and AMG signals from the data shown in figure 4.11a, the plot shown in figure 4.11b can be constructed. The contraction begins at time 0 seconds, and all three variables rise, with force reaching a maximum value within 10 seconds. Force does not appear to remain constant although within 20% of the required target force. This is due to the sampling interval, as the force can be seen to remain fairly constant in the above figure. The EMG and AMG values follow each other very closely taking slightly longer to reach their maximum values, 10 seconds after the start of the contraction. There is some fluctuation in the rms values for both EMG and AMG, until they start

to decrease 20 seconds from the start of the contraction. Both the EMG and AMG fall until the end of the contraction, when they return to their pre-contraction levels.

The contraction of adductor pollicis shows the same general trends described above. The force record, the bottom trace, shows no force exerted initially and then a rapid increase to a level of 19N. The subject maintains this force until they can sustain it no longer and they relax rapidly. The whole contraction lasts for around 34 seconds. There is very little noise on the EMG trace before the contraction begins. When force increases there is an increase in EMG amplitude. There appears to be a steady decrease in EMG amplitude to the end of the contraction. The AMG trace shows very little activity before the contraction and when the contraction begins there is a small increase in amplitude. This is followed by a very gradual increase in amplitude with some apparent bursts in the signal level. Around two thirds of the way through the contraction there is a large increase in the AMG amplitude to around 10 times the previous level. At the end of the contraction the signal returns to pre contraction amplitudes. It is interesting to note that there are no obvious movement artifacts on this AMG trace.

The force and rms levels were taken at 5 second intervals and plotted against time to give figure 4.12b. The contraction starts at time 0 seconds, and the force increases sharply to reach the maximum value after 5 seconds. It remains at this level until the end of the contraction. EMG rises initially to reach a maximum level after 15 seconds. It then decreases steadily until the end of the contraction and returns to pre contraction levels. The rmsAMG value increases very slightly for the first 20 seconds after which it fluctuates quite dramatically to reach a maximum value at the end of the contraction. It then decreases to the pre-contraction level within 5 seconds.

Median frequencies

The median frequencies of both the EMG and AMG signals were calculated during the contraction. Examples of the frequency plots for the beginning, middle and end sections of the contraction of first dorsal interosseus are illustrated in figure 4.13a. EMG frequencies are shown on the left and AMG on the right, with frequency as the ordinate and power as the abscissa. Frequency is measured in Hertz and the power axis is in relative units. These records come from the beginning, middle and end portions of the contraction and is to illustrate the range of frequencies that are obtained from both signals. Comparing the EMG frequencies it must be noted that the power scale is not the same for all of the plots. At the beginning of the contraction the range of frequencies is between 10 and 200 Hz, and the median frequency is calculated to be 70Hz. By the middle of the contraction the range has been reduced to 10 - 150 Hz and the median frequency has shifted to 53 Hz. The last plot from the last 10 seconds of the contraction show a slightly narrower range from 10 - 100 Hz, and the median frequency has shifted to around 48 Hz. The power over the range of frequencies seems to increase throughout the contraction.

The AMG frequencies are much lower as can be seen from the plots on the right hand side of figure 4.13a. The power scale for AMG remains the same throughout, and it can be seen that there is more power within the frequencies at the beginning of the contraction than there are at the end. In fact the frequency content of the AMG signal is not well defined and this was a feature of the recorded signal from first dorsal interosseus. The range of frequencies at the beginning of the contraction is difficult to define but the main frequencies are in the band from 0 - 25 Hz and the median frequency being 22 Hz. The plot from the middle of the contraction shows a drop in the power, and there seems to have been a shift to lower frequencies with the range still being 0 - 25 Hz, but the median frequency being 10 Hz. The last plot shows that the AMG is again at very low levels with the main frequency range being the same as the middle plot, 0 - 25Hz. The median frequency is 12Hz for the end section. Thus, for this contraction, the general trend is for the range to narrow

and the median frequency of the EMG to shift to a lower frequency while for the AMG there is no distinct trend although the range seems to become narrower the further through the contraction. There is no trend for the median frequency.

The data from first dorsal interosseus is summarised in figure 4.13b. The median frequencies were calculated for the beginning, middle and end portions of the contraction. The mean and the standard error of the mean are shown plotted against their position from the contraction. There appears to be a decrease in the EMG median frequency from the beginning to the end of the contraction. However, there is very little statistical significance in the differences between the frequencies, $p>0.1$. There is also a slight decrease in the AMG frequencies although, the only statistical significance is seen in the difference between the frequencies at the beginning and middle of the contraction ($p=0.02$). There is no further statistical difference between the frequencies ($p>0.1$).

Figure 4.14a shows the frequency plots from the contraction of adductor pollicis illustrated in figure 4.12a. The EMG frequency range, 0-250Hz, does not change from the beginning to the end of the contraction, although there is a reduction in the median frequency from 62Hz for the beginning of the contraction to 49Hz at the end. The AMG frequencies show a wide range, 0-75Hz, although the main power is below 50Hz. There appears to be a slight decreasing trend for the median frequency from 15Hz at the beginning of the contraction to 11Hz at the end. Figure 4.14b shows the summary data, the mean and standard error of the mean of the median frequencies. There appears to be a trend for decreasing EMG and AMG from the beginning to the end of the contraction. There is slight statistical significance ($p=0.1$)for the difference between the EMG frequencies at the beginning, middle and end of the contraction. There is no statistical difference between the frequencies of the AMG signal ($p>0.5$).

Figure 4.11a : Copy of trace showing a contraction of 1st dorsal interosseus at 75% of the maximal voluntary contraction held to fatigue.

Figure 4.11b : Plot showing force, rms EMG and rms AMG against time from the contraction of 1st dorsal interosseus shown in figure 3.11a.

Figure 4.11a

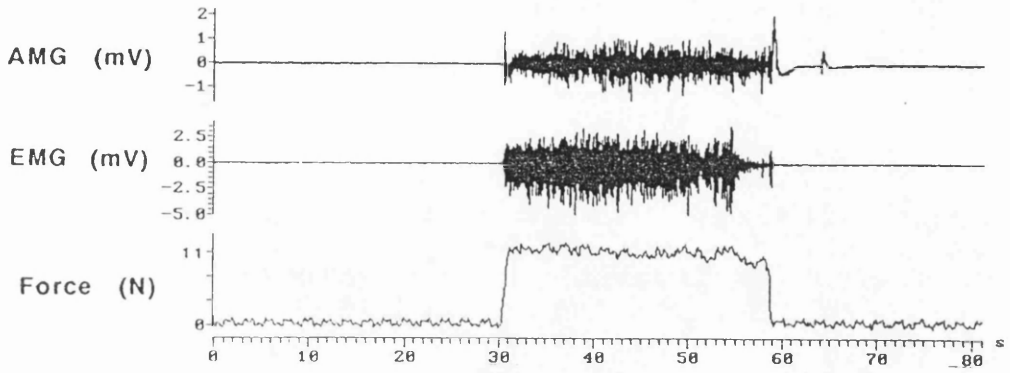


Figure 4.11b

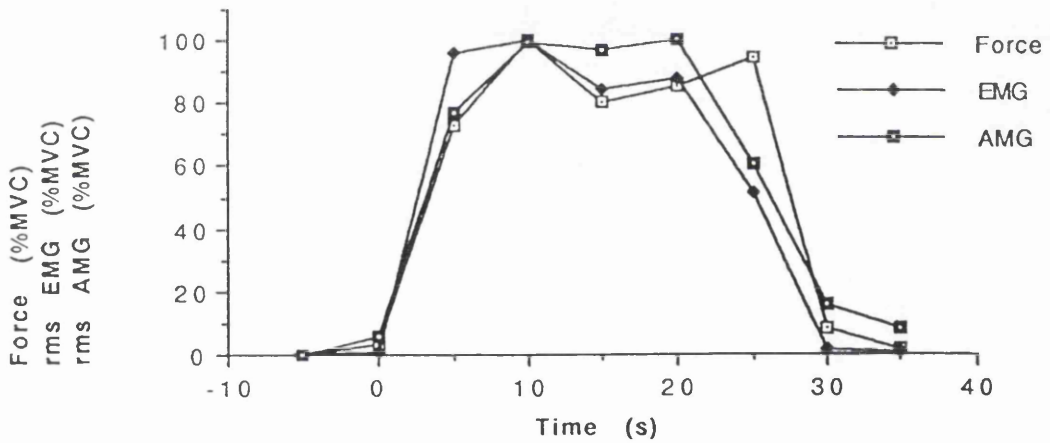


Figure 4.12a : Copy of trace showing a contraction of adductor pollicis at 75% of the maximal voluntary contraction held to fatigue.

Figure 4.12b : Plot showing force, rms EMG and rms AMG against time from the contraction of adductor pollicis shown in figure 3.12a.

Figure 4.12a

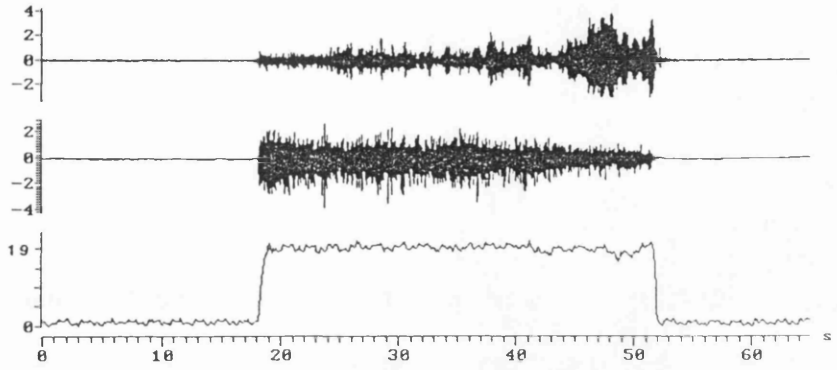


Figure 4.12b

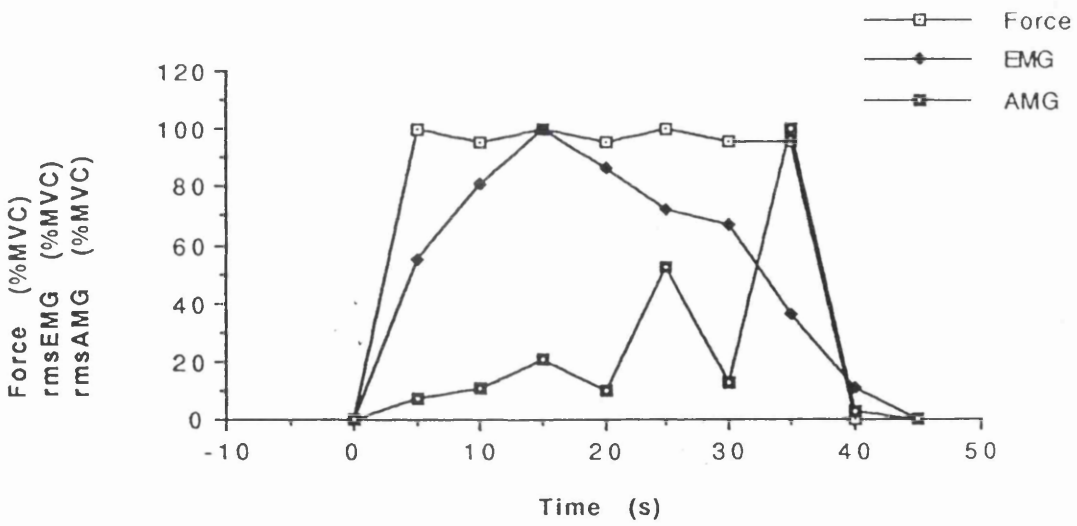


Figure 4.13a : Frequency plots from the beginning (B), middle (M) and end (E) of the contraction of 1st dorsal interosseus shown in figure 4.11a.

Figure 4.13b : Plot showing the mean and standard error of the mean of the median frequencies from the beginning, middle and end of the contractions of 1st dorsal interosseus at 75%MVC, from all subjects (n=8).

Figure 4.13a

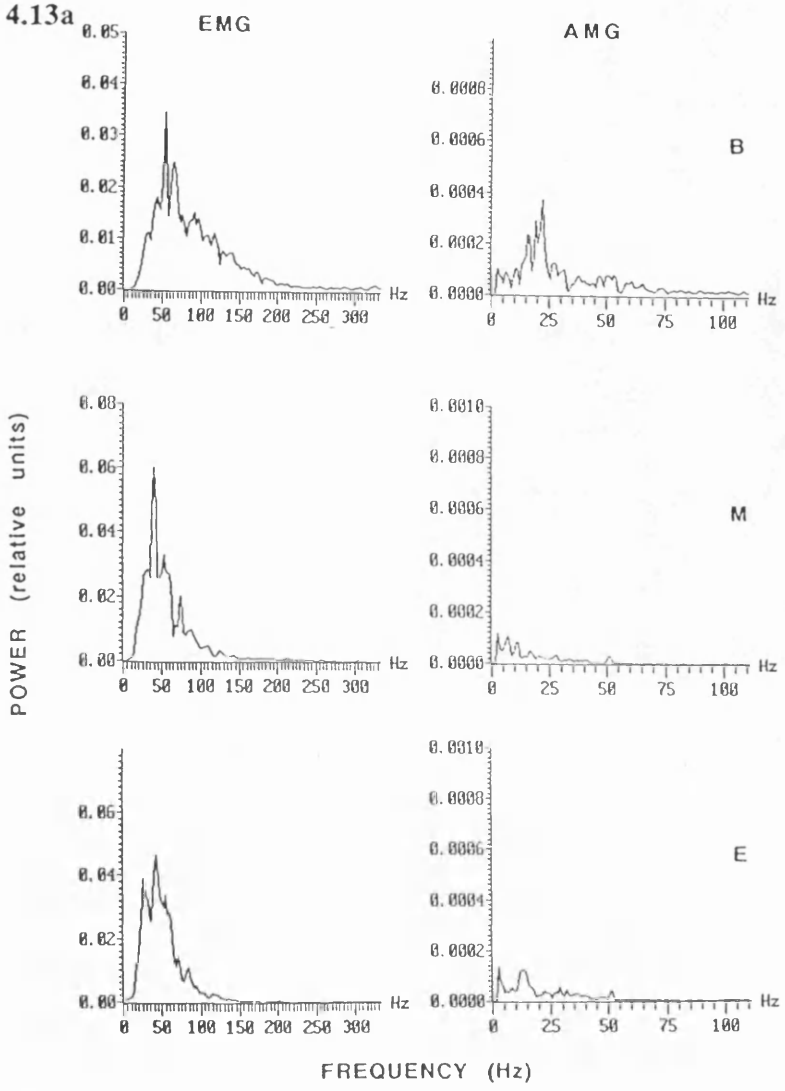


Figure 4.13b

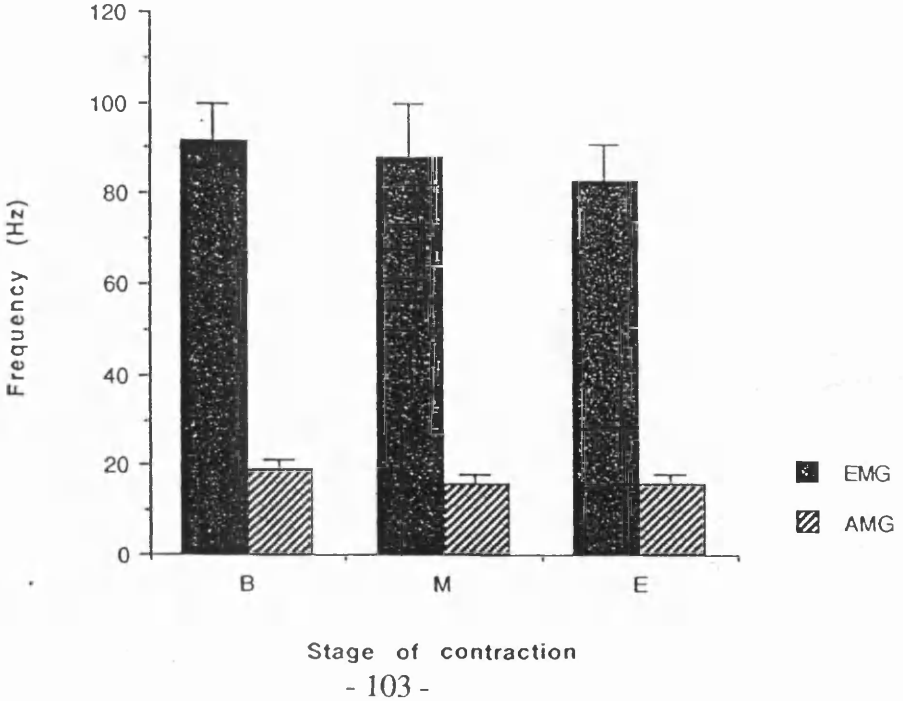


Figure 4.14a : Frequency plots from the beginning (B), middle (M) and end (E) of the contraction of adductor pollicis shown in figure 4.12a.

Figure 4.14b : Plot showing the mean and standard error of the mean of the median frequencies from the beginning, middle and end of the contractions of adductor pollicis at 75%MVC, from all subjects (n=8).

Figure 4.14a

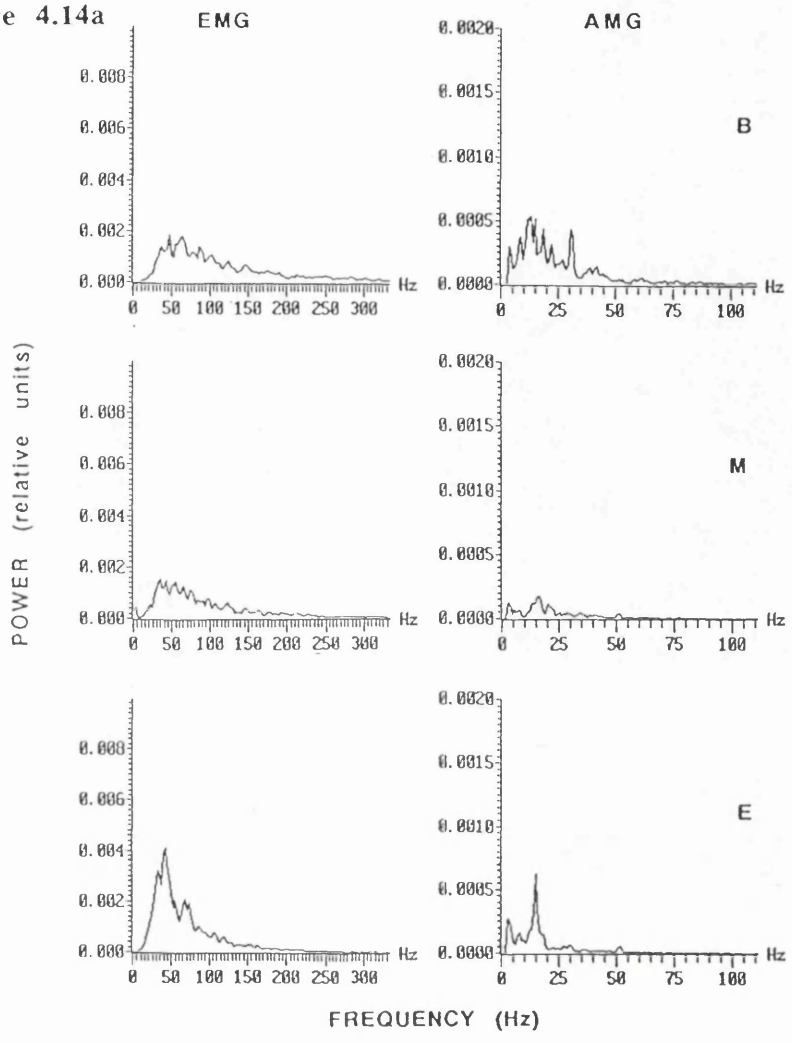
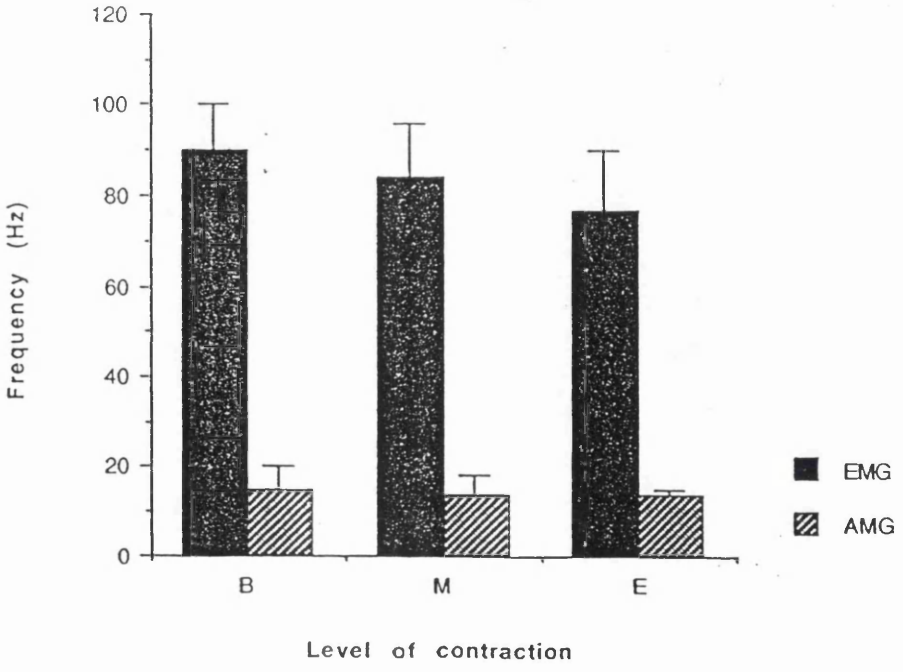


Figure 4.14b



Isometric Contractions of Rectus Femoris

Subjects

The subjects (n=5) were drawn from the staff and postgraduate students of the Institute of Physiology, University of Glasgow and The Queen's College, Glasgow. All subjects were male with an age range of 23-35. All subjects were fully informed of the procedures involved in the tests and understood that they could withdraw from the experiment at any time. None of the subjects had any history of any muscular or neurological disorders.

Methods

The methods are described fully in the methods chapter.

Results

Illustrative results are shown, as before, at the end of the relevant section, in the order to which they are referred.

1. Maximal contractions at knee angles of 90°, 70°, 50° and 30° towards full extension.

The subject was asked to produce maximal contractions at each knee angle and a typical trace is shown in figure 5.1a. Force is shown as the top trace, EMG as the middle trace and AMG as the bottom trace. It was necessary to record force separately from EMG and AMG. As can be seen from this record, force was only recorded during a contraction and so the timing of the contractions is difficult to

determine. As is evident from this trace the maximal forces exerted at each of the 4 angles varies with the extension of the knee. At the first three angles there are only slight differences between the force levels, being around 100N, and the highest force appears to occur at the 70° angle. At the most extended knee angle of 30°, the force produced is lower, being 40N. These are low values differing almost by a factor of 5 from the expected values (Edwards et al, 1977). As no direct force data could be compared this must be borne in mind when examining the data. The EMG signal has very low noise in between contractions, although there is some variation in the base line, especially between the last two contractions, due to movement by the subject. When the contraction starts, the amplitude rises and seems to remain constant for most of the contraction. The EMG amplitude is constant for the first three contractions, with a slight increase in amplitude at the final angle of 30°. The peak to peak amplitude of AMG increases at the start of the contraction and remains at this level for the whole of the contraction. The first three contractions have similar signal amplitudes, although for the last contraction, 30° towards full extension, there is a slight decrease in peak to peak amplitude.

Although this is one result from one subject, these general observations seem to be confirmed by the summed data from all the subjects contractions. Figure 5.1b illustrates the summary data from this part of the experiment. Force, rmsEMG and rmsAMG are shown as the mean and standard error of the mean of the raw data. Evidently, as the knee angle becomes more extended, the force produced is reduced with a drop of 50N from the most flexed angle (90°) to the most extended angle (30°), this represents a drop in force of 40%. There are significant differences between the forces exerted at each angle, $p<0.003$. EMG is constant for the first 3 angles and then there is a rise in the rms signal level at the most extended angle. There is no significant difference between the values at the first three angles, although the value at 30° is significantly different ($p<0.001$) from the other values. AMG is constant during the first three angles and then falls at the most extreme

angle. There is a significant difference ($p<0.01$) between this value and the other values, although there is no significant difference ($p>0.5$) between any of the other values.

Median frequency

Example plots of the frequency data from the 4 contractions are shown in figure 5.2. The EMG frequencies are shown on the right and AMG frequencies on the left. Frequency for both plots is shown as the x axis, and the y axis is power. The EMG has a much wider frequency range than AMG, being 0-300Hz compared with 0-20Hz. The range of frequencies does not change between contractions, although the power associated with the frequencies does seem to change. There appears to be more power in the higher frequencies with the increase in angle. This is not easy to quantify and so the median frequency of the data is calculated. The median frequencies show an increase with the increase in angle, being 56, 69, 69 and 74Hz from flexed to extended respectively. The range of AMG frequencies is very low, being 8-20Hz for all of the plots. There is virtually no change in the power within the range and there is no change in median frequency being 11Hz for all of the plots.

Figure 5.3 shows the mean and the standard error of the mean, of the median frequencies of the EMG and AMG signals. Obviously, the EMG frequencies are much higher than the AMG frequencies being between 60 and 80 Hz. The median frequency of the EMG signal rises with the increase in knee angle, thus the more extended the leg the higher the EMG frequency. Comparing the differences shows that all are significantly different at a level $p=0.0001$, apart from the difference between 90° and 70° ($p=0.002$) and 50° and 30° ($p=0.02$). Conversely, the AMG frequencies are lower than the EMG frequencies, being between 20 and 15 Hz. There is a slight trend for the frequency to decrease with the increase in knee angle.

However, the differences in AMG frequency are very small and have a low level of significance. The most significance is between the values at 90° and 50° ($p=0.004$) and the values at 50° and 30° ($p=0.005$). All other comparisons yielded significance levels of $p>0.01$.

Figure 5.1a : Copies of traces from a series of isometric contractions of rectus femoris at 4 knee angles (90°, 70°, 50° and 30°). Upper trace shows force, middle trace shows EMG and lower trace shows AMG.

Figure 5.1b : Plot of force, rms EMG and rms AMG against the knee angle from isometric contractions of rectus femoris from all subjects (n=5). Data is shown as the mean and standard error of the mean.

Figure 5.1a

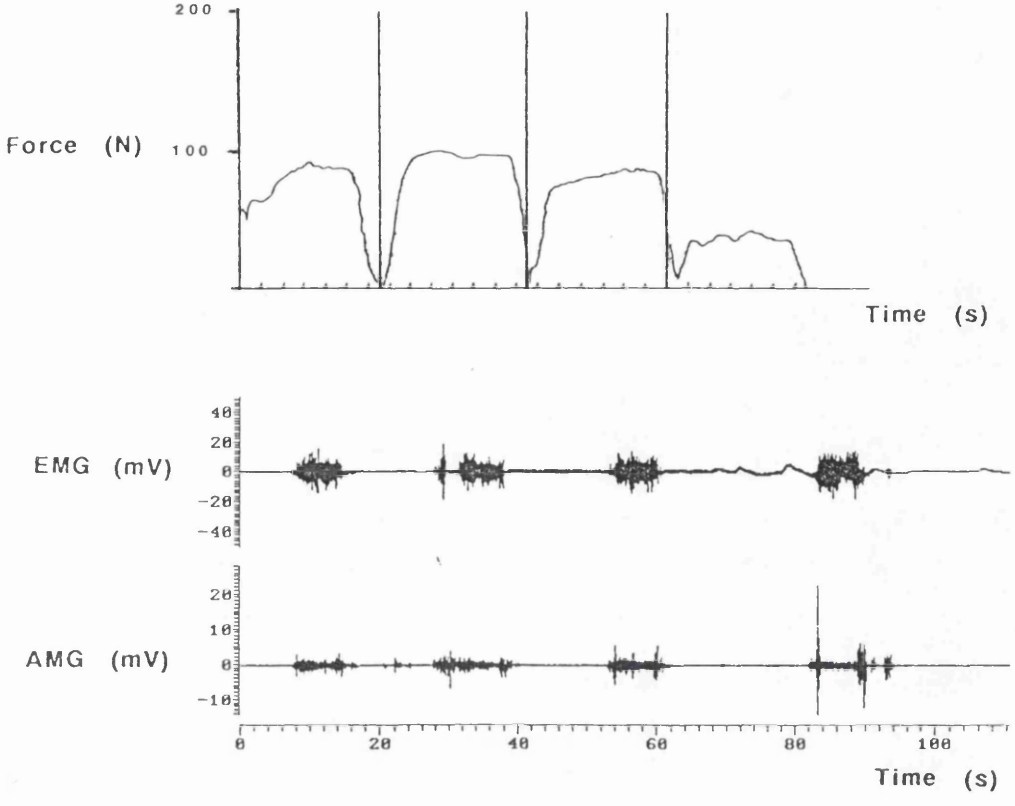


Figure 5.1b

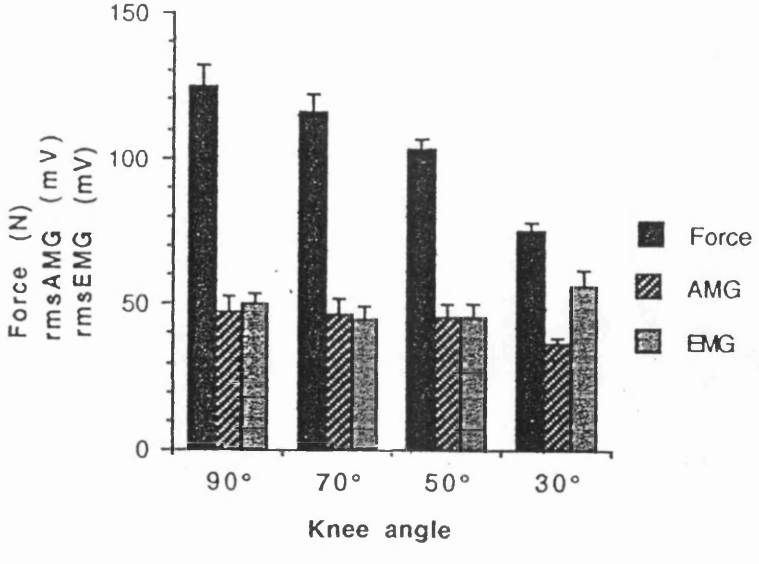


Figure 5.2 : Frequency plots showing EMG and AMG, from contractions of rectus femoris shown in figure 5.1a, at the 4 knee angles, 90°, 70°, 50° and 30°.

Figure 5.2

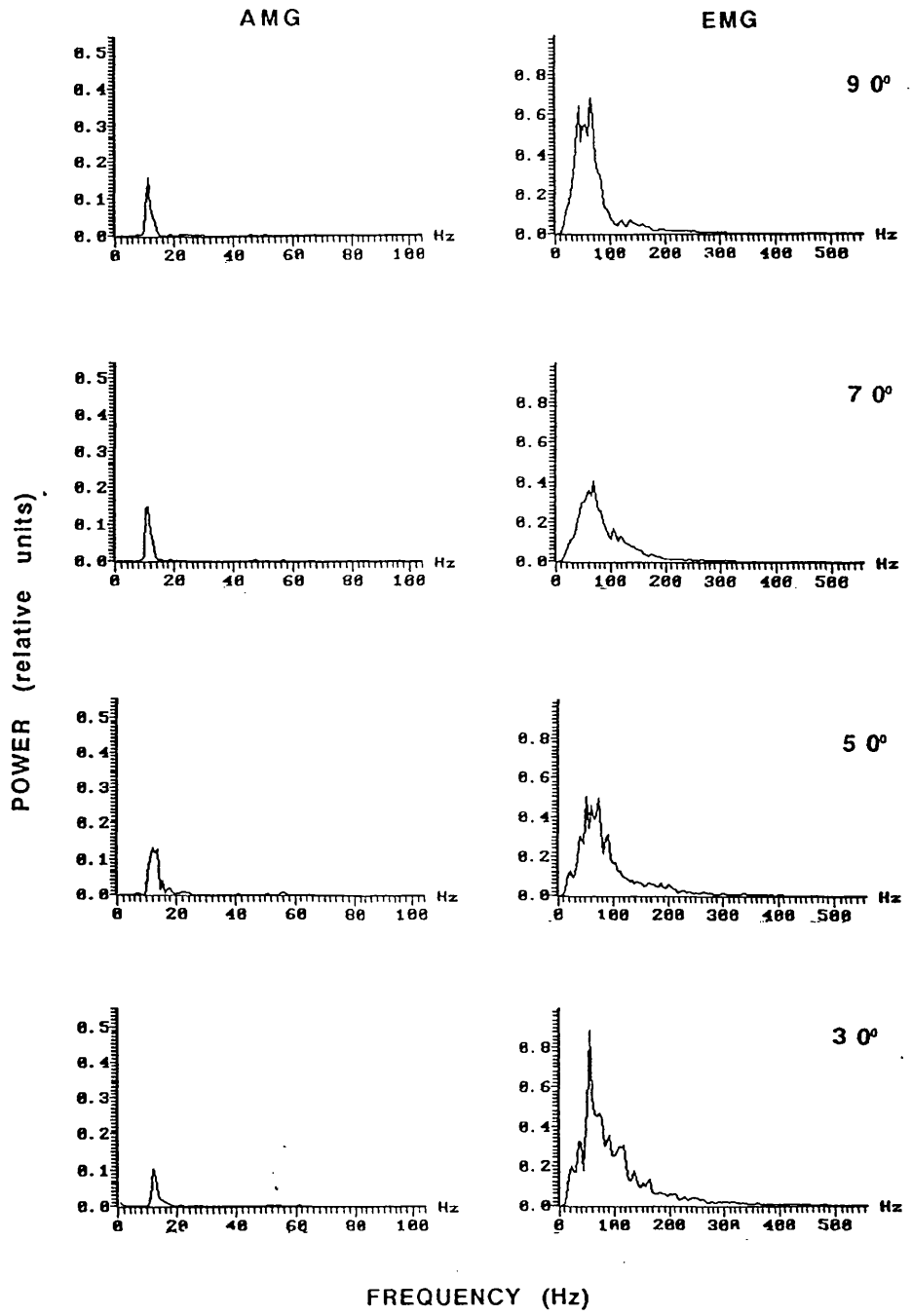
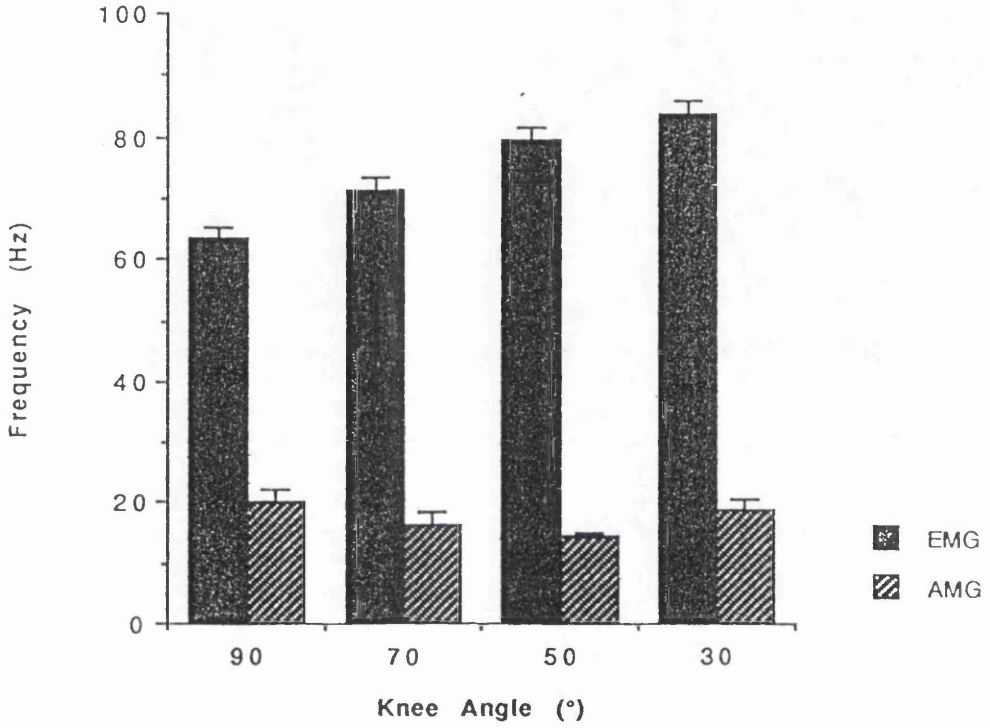


Figure 5.3 : Plot showing the mean and standard error of the mean of the EMG and AMG median frequencies, from the isometric contractions of rectus femoris at the 4 knee angles, from all subjects (n=5).

Figure 5.3



2. Isometric contractions at percentages of the maximal force at 70° knee angle.

Figure 5.4a shows an example of the traces obtained from this set of experiments. Force is the top trace, EMG the middle trace and AMG the bottom trace. The force levels used were 25, 50, 75 and 100% of the previously determined maximal force at a leg angle of 70°. It can be seen quite clearly that as the force exerted increases the peak to peak amplitude of the EMG signal increases. The increase in amplitude occurs in quite regular steps and this is evident from the figure shown. AMG has a similar relationship with an increase in the peak to peak amplitude of the signal. This appears to be less regular than with the EMG. There is also a high level of noise between the contractions.

By normalizing all the data from all the subjects and plotting the rms EMG and AMG signal levels against force, figure 5.4b was obtained. There is a lot of scatter within the data and this is most clearly seen by the lack of distinct groups of data around the 4 force levels 25, 50, 75 and 100% of the maximal force. However, there is a distinct trend for the EMG and the AMG to rise with the increase in force.

Median frequency

The four frequency plots from the four levels of contraction are shown in figure 5.5. EMG is on the right and AMG on the left, with the x axis for both plots being frequency (Hertz) and the y axis power. The EMG frequency range does not change with the increase in force, remaining at 0-200Hz. The power associated with these frequencies increases throughout the series of contractions, particularly in the central band between 40Hz and 90Hz. There is no trend in the median frequencies from these plots being 65Hz for the first three contractions and 61Hz for the last contraction. AMG shows no change in the range of frequencies with the increase in force, remaining at 6-20Hz. There is an increase in the power with the increase in force, particularly around the range 10-16Hz. There is no distinct trend

in median frequency being 11Hz for the first two contractions, 10Hz for the next plot and 12Hz for the last contraction.

The summary frequency data is shown is figure 5.6. Data is displayed as the mean and the standard error of the mean, and is displayed against the target force level. As already discussed in the previous section the forces were not in distinct percentages of the maximal and this must be taken into account when comparing the data. It would appear as if there is a slight tendency for the EMG frequency to rise with the increase in force although the differences in frequency are not significant ($p>0.1$) apart from the comparisons between the 25% level and the rest of the force levels ($p=0.01$). The median frequency of the AMG signal appears to fall from initial to final contraction with the increase in force production. The differences between all frequencies are of no statistical significance with $p>0.1$, apart from between 25% and 75% ($p=0.02$) which is significant, between 25% and 100% ($p=0.06$) and between 50% and 75% ($p=0.07$) which are both deemed non significant.

Figure 5.4a : Copies of traces from a series of isometric contractions (25, 50, 75 and 100%MVC) of rectus femoris at a knee angle of 70°. Upper plot is force, middle plot is EMG and lower plot is AMG.

Figure 5.4b : Plot showing rms EMG and rms AMG data from all the subjects (n=5) plotted against force. Contractions were made at a knee angle of 70°. Regression equations: EMG vs Force $y = -6 + 0.9x$, $r^2 = 0.91$; AMG vs Force $y = -7 + x$, $r^2 = 0.91$.

Figure 5.4a

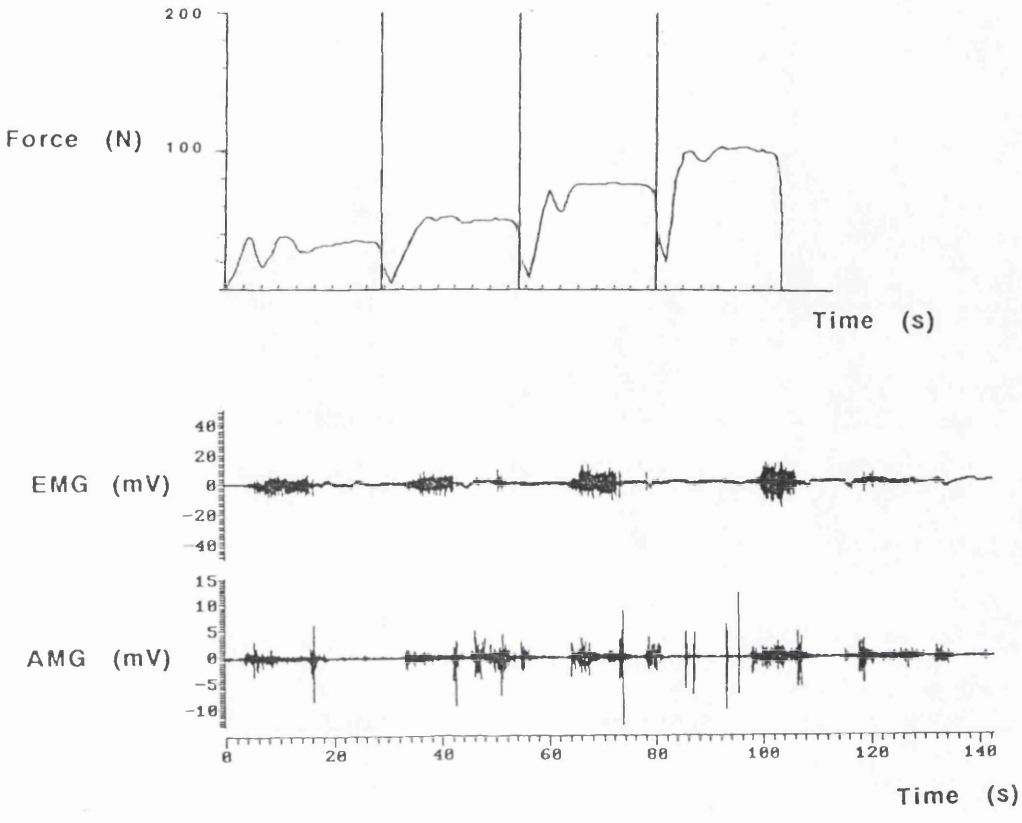


Figure 5.4b

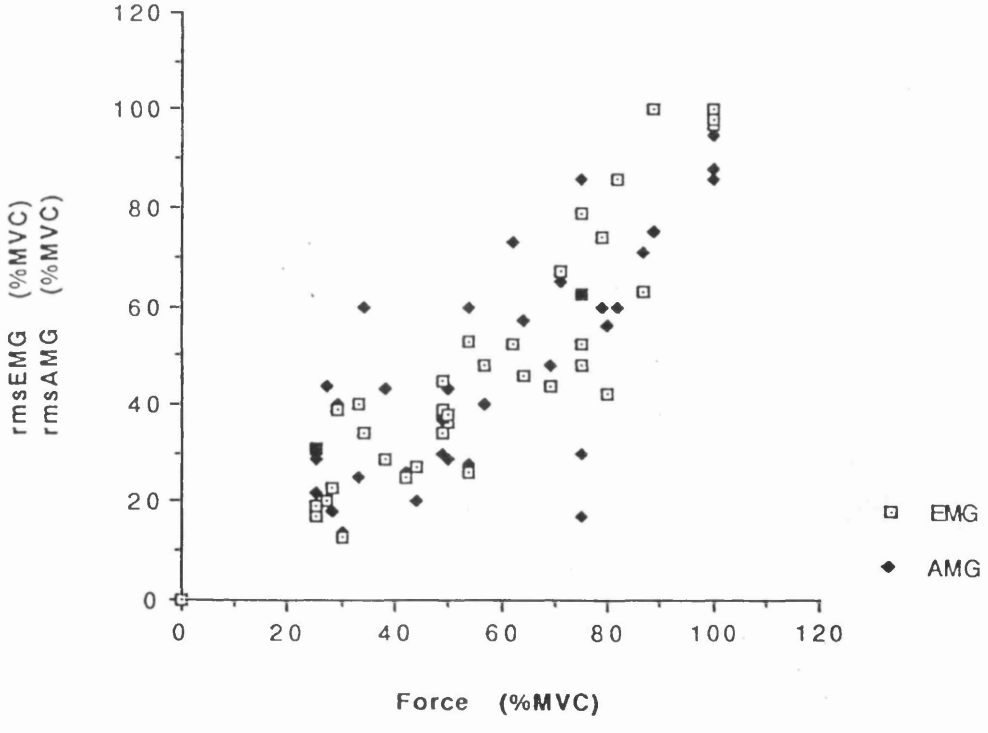


Figure 5.5 : Frequency plots of EMG and AMG data from the series of 4 contractions shown in figure 5.4a, at percentages of the maximal contraction, from all subjects (n=5).

Figure 5.5

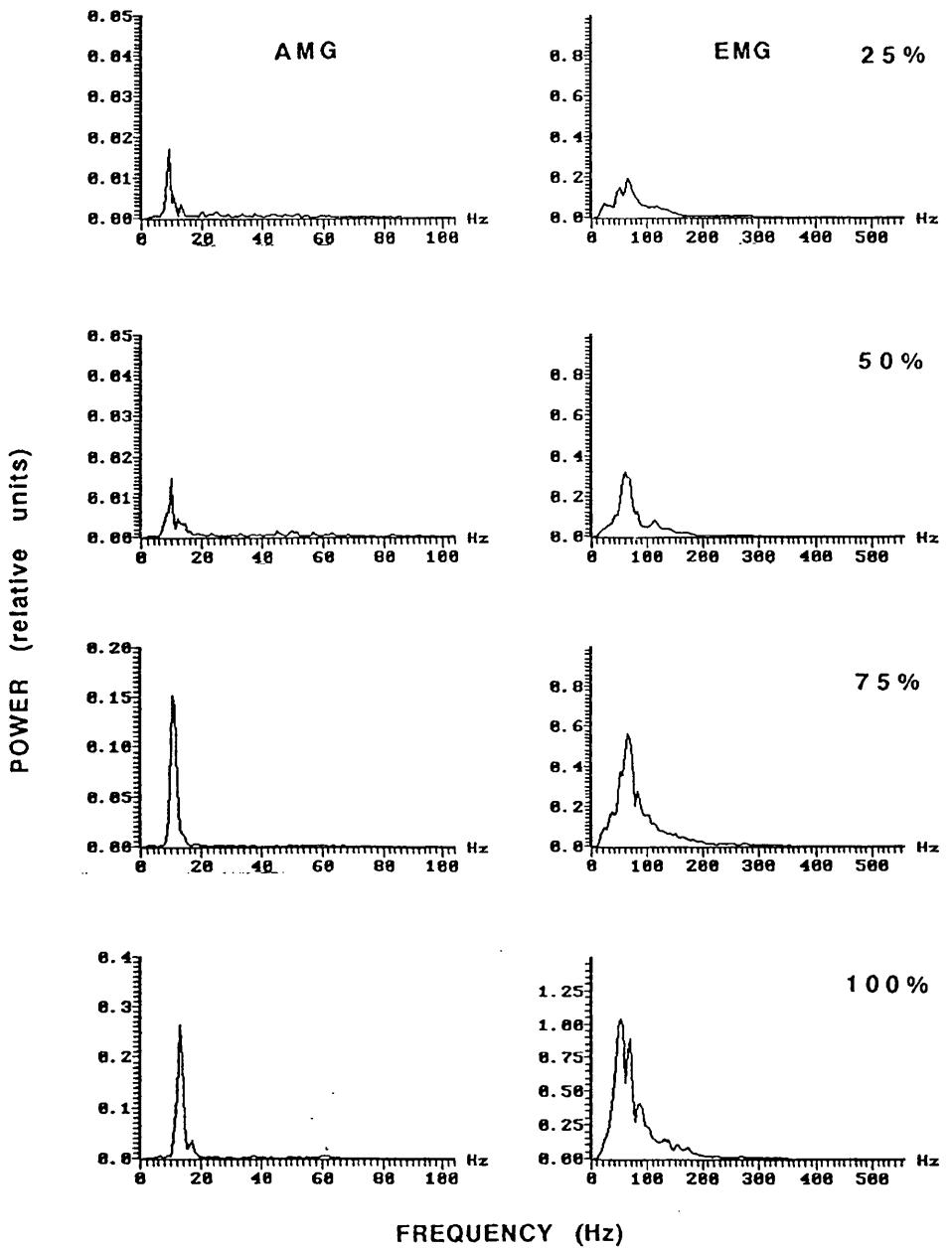
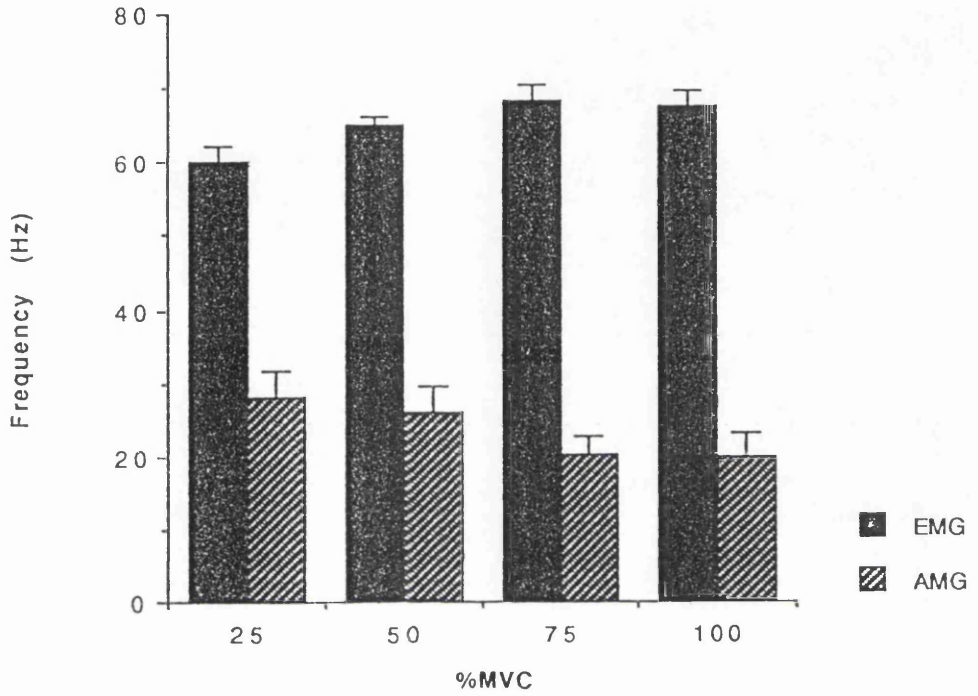


Figure 5.6 : Plot showing the mean and standard error of the mean of the EMG and AMG median frequencies from the isometric contractions of rectus femoris at 25, 50, 75 and 100% MVC, from all subjects (n=5).

Figure 5.6



3. Ramp contractions up to the maximal force at a knee angle of 70°.

An example of a record obtained during this series of contractions is displayed in figure 5.7a. As previously, force is the upper trace, EMG is the middle trace and AMG is the lower trace. Clearly, as the force increases the peak to peak amplitude of the EMG signal increases until it reaches a maximal level. The force is maintained at the maximal level for a few seconds and EMG also appears to remain at this constant level until the subject relaxes with a sharp decrease in both force and the amplitude of the EMG signal. Although the AMG signal is noisy throughout the whole of the contraction, it can be seen that the AMG amplitude increases at the start of the contraction, and continues to increase gradually until the maximum force is reached. The AMG trace also shows high noise levels after the contraction due to the subject moving and easing the discomfort felt after the contraction.

Summing all the data from the subjects and plotting the rmsEMG and rms AMG against the percentage increase in force yields figure 5.7b. There is clearly not a linear relationship between either rmsEMG, or rmsAMG, and force. However, it can be concluded that there is a monotonic relationship, with an increase in force producing an increase in both EMG and AMG. It is also obvious that there is a large amount of scatter within the data, with perhaps more AMG outlying points than EMG. It should also be noted that at zero force there is sometimes as much as 80% of the maximal AMG signal seen.

Median frequency

The frequency plots from the beginning, middle and end of the contraction illustrated in figure 5.7a, are shown in figure 5.8. As before, EMG is on the right and AMG is on the left, for all three plots. The range of EMG frequencies is 0-200Hz, and this does not change for the whole of the contraction. It is easy to see that the power within this range does increase as the contraction progresses. The median frequency shows no trends being 56Hz for the three plots shown. The first

plot from the AMG frequencies is difficult to interpret, showing a wide range of frequencies at very low power. However, the plots from the middle and end portions of the contraction show a very narrow range, 0-20Hz, with increasing power over this range as the contraction progresses. The median frequency for the first plot is high, 35Hz, although there is a decrease in value for the median frequency of the other two plots. There does not appear to be any change in the AMG median frequency, being 12Hz for the middle and end plots.

The summary data is shown in figure 5.9. There appears to be rise in the median frequency of EMG during the ramp contraction, from around 58Hz at the beginning to 73Hz at the end. There is a statistical difference between the frequencies at the beginning and middle ($p=0.006$), and the beginning and end ($p=0.004$), although there is no statistical difference between the middle and end ($p>0.1$). In contrast, there is a decrease in the AMG median frequency from 27Hz to 16Hz through the contraction. There is no statistical difference between the frequencies at the beginning and middle ($p=0.3$), although there is between the beginning and end ($p=0.003$) and the middle and end ($p=0.05$).

Figure 5.7a : Copy of the trace from an isometric ramp contraction of rectus femoris to maximal force.

Figure 5.7b : Plot showing the rms EMG and rms AMG data against force for the isometric ramp contractions to maximal force for all subjects (n=5).

Figure 5.7a

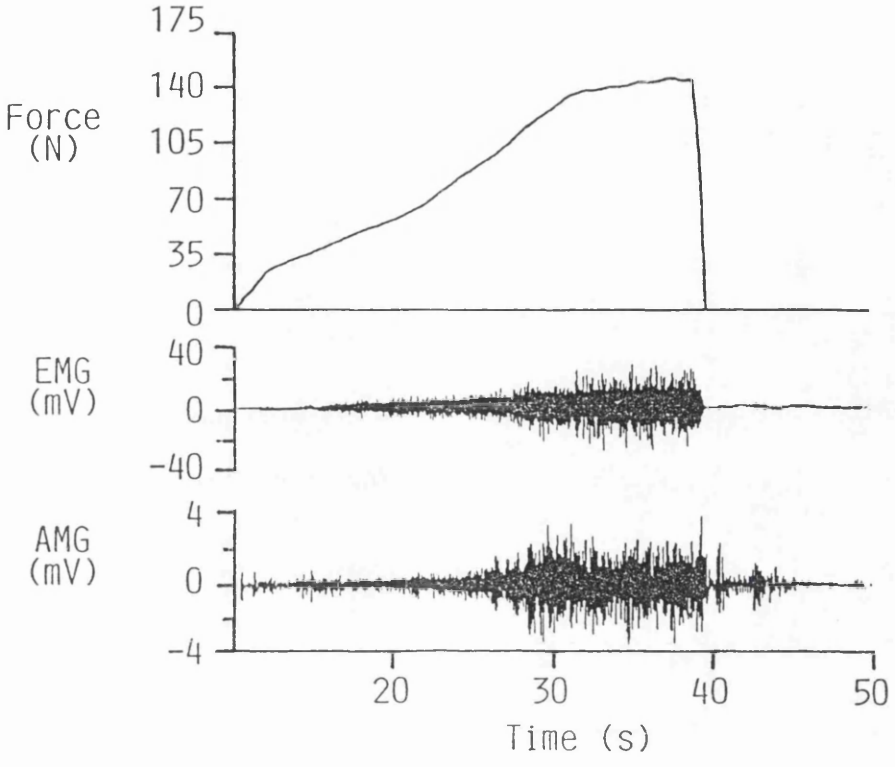


Figure 5.7b

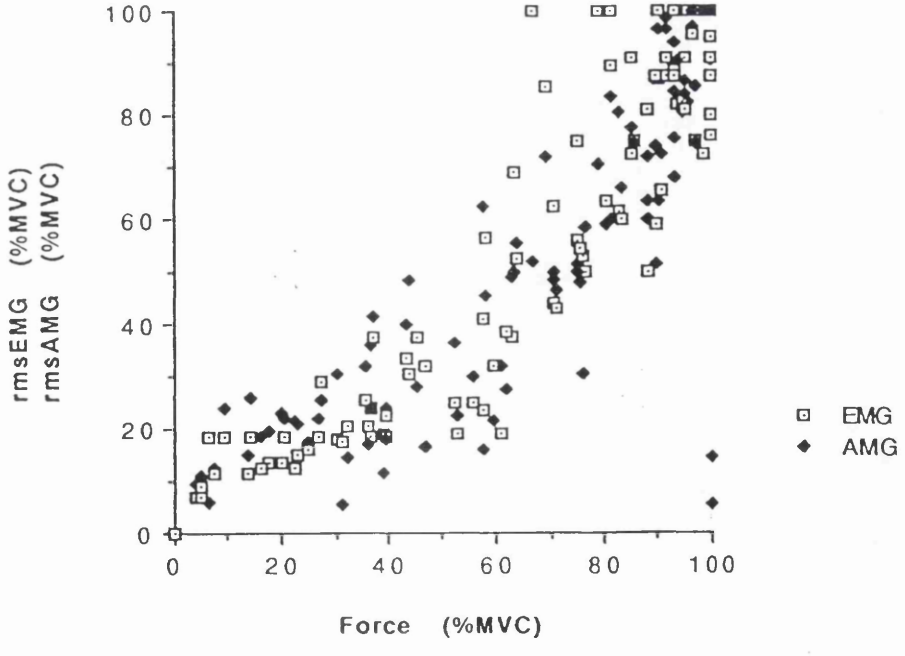


Figure 5.8 : Frequency plots of EMG and AMG from the beginning (B), middle (M) and end (E) of the contraction shown in figure 5.7a.

Figure 5.8

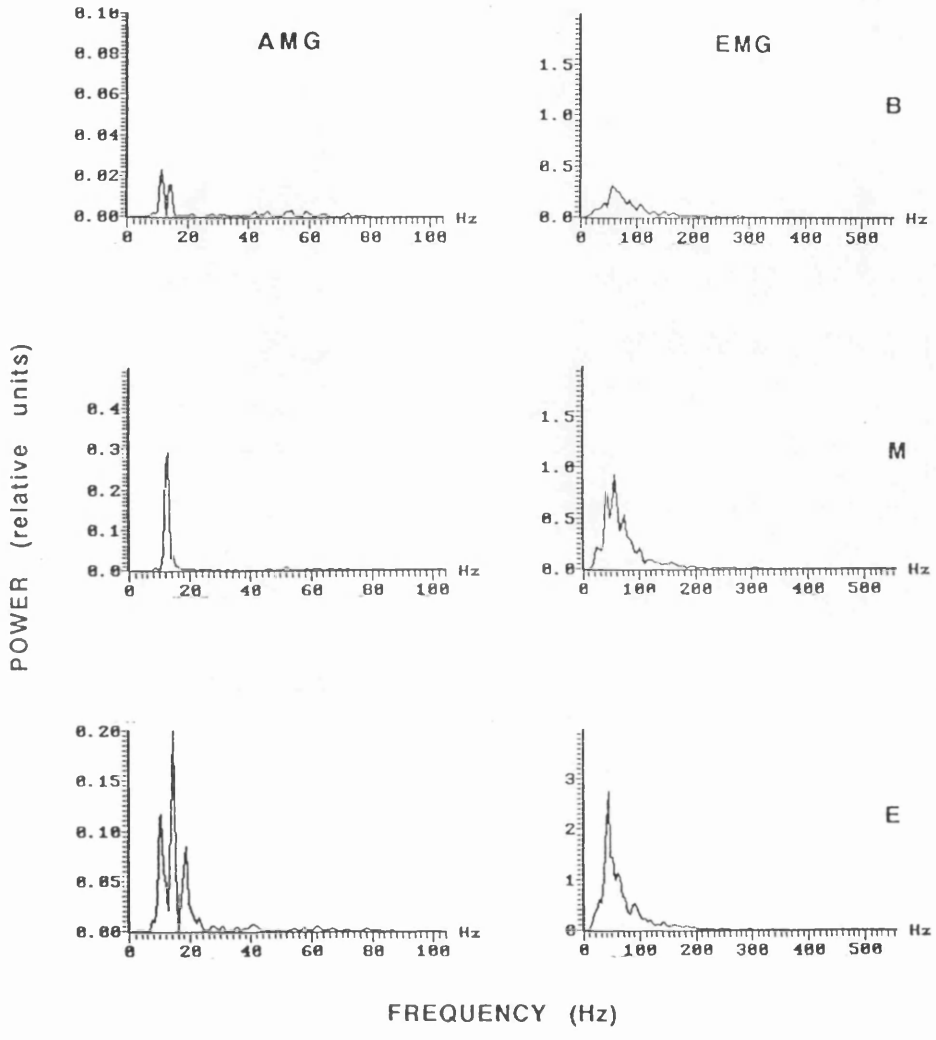
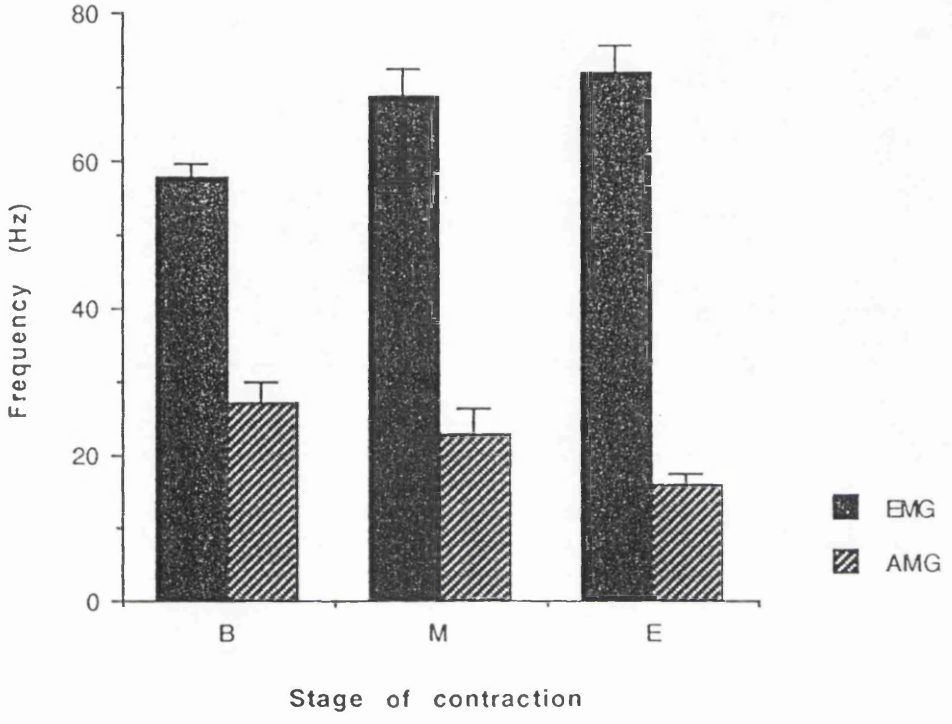


Figure 5.9 : Plot showing the mean and standard error of the mean of the median frequency from the beginning (B), middle (M) and end (E) of the isometric ramp contractions from all subjects (n=5).

Figure 5.9



DISCUSSION

Discussion

It was the aim of this project to study the relationships between force, EMG and AMG recorded from isometric contractions, using a number of muscles and employing isometric contractions under a number of different conditions. As will be discussed in the text, the lack of systematic investigation by other workers using AMG, revealed a large number of unanswered questions, for example, is there a difference in large and small muscles, is there a difference in the relationship between force and AMG during different types of isometric contraction and does the frequency of the AMG signal change with changing conditions? It is hoped to go some way to clarifying the AMG work and to answer some of those questions. With this in mind, the work has fallen largely into two areas of discussion, namely amplitude changes of AMG and EMG with force, and changes in frequency of the AMG and EMG signals during contractions and the discussion will be arranged in this way.

Isometric contractions with changes in force.

A linear relationship between force and the rectified integrated EMG and AMG signals was found when contractions of the quadriceps group were recorded (Stokes and Dalton, 1991). Isometric force was recorded together with the EMG and AMG signals from rectus femoris, in a very similar manner to that used to record the data reported from the contractions of rectus femoris, although the acoustic data was recorded using an electronic condenser microphone, rather than the accelerometer described in the materials and methods chapter. Contractions were made by the subject at percentages of the maximal contraction force, and there was a marked increase in AMG amplitude up to, and including the maximal force. In another set of experiments recording from erectores spinae muscle, which when

contracted produces arching of the back in the lumbar region, it was also found that the AMG signal increased in amplitude up to the maximal contraction (Stokes et al, 1988). In contrast, data from biceps brachii showed decreases in the AMG amplitude at contractions above 80% MVC, although there was no decrease in EMG amplitude (Orizio et al, 1989a). These data raises a number of points that must be remembered when considering the results. There is very little data from voluntary, isometric, non-fatiguing contractions, and every result must be considered independently due to the inconsistent recording methods. From the three reported experiments, three different methods were used for AMG and so a direct comparison of data is very difficult to make, and if done so, should be made with caution. Very little work has been done on the subject of AMG as a whole, and most of it has either been carried out under fatiguing conditions or when the muscle was being stimulated to contract via the supplying nerve. The recordings were made from a group of muscles, quadriceps, or a muscle which does not usually contract in isolation, erectores spinae, which must result in more force being recorded than is actually being produced by the muscle under investigation. Quadriceps is a group of 4 major muscles, vastus medialis, vastus lateralis, vastus intermedius and rectus femoris, that together extend the knee and, additionally, rectus femoris flexes the hip. In passive knee extension, over the range from 90° to 180°, rectus femoris is active for the largest part of the range, from 90° to 140°. However, above 140° there is a decrease in the EMG from these 4 muscles and therefore other muscles, perhaps sartorius, will have a significant role in the latter stages of this movement (Basmajian and De Luca, 1985; Wheatley and Jahnke, 1951). Most work reports on the activity of quadriceps as if it is one muscle (Newham et al, 1991) or records force from the whole muscle but EMG from one of the group of four (Thorstensson et al, 1976; Hertzog and ter Keurs, 1988). It is therefore difficult to ascertain the contributions from all of the muscles throughout the contraction. Therefore, although recording from rectus femoris may not be entirely representative of the contraction as a whole, it would seem to be an accepted method of studying the

quadriceps group. Similarly, the muscle erectores spinae, produces extension of the lumbar region although there are other muscles, both in the lumbar region and further up the back, that are agonistic to this movement. Again it is unlikely that the EMG and AMG signals recorded from this muscle are representative of the whole movement and the force recorded.

The experiments and results described in the sections related to contractions of biceps and triceps brachii, first dorsal interosseus and adductor pollicis, are an attempt to clarify the situation during voluntary isometric contractions up to the maximal force. The muscles under investigation in these chapters had one action, with the exception of biceps brachii which has two actions. However, within the protocol any movement other than flexion at the elbow was eliminated. It was clearly demonstrated during both the contractions at percentages of the maximal contraction, and slow ramp contractions, from zero to maximal and back to zero force, that the AMG and EMG amplitudes increased with the increase in force, over the whole range of force measurements. Thus it would be sensible to conclude that muscles with a simple action, exhibit increases in EMG and AMG amplitude as the force produced by the muscle increases.

From the data shown within this thesis, the relationship between force and AMG appears to be very similar to that between force and EMG. Data from the contractions at percentages of the maximal force exhibits linear trends up to and including the maximal force although there is often a tendency for non linearity between zero force and the first measurement of AMG. This is due to the sometimes large AMG signal at the start of the contraction. A proportion of this is due to movement either by the subject, or close to the accelerometer. There is also a contribution to the signal from tremor, possibly fatigue tremor induced by the exercise protocol. Taking this into account the relationship between force and AMG is linear, which differs from some of the results reported previously. Recording from biceps brachii a curvilinear relationship was seen (Maton et al, 1990) with

both EMG and AMG against force. The data from the ramp contractions shown in this thesis shows curvilinear tendencies which may reflect the recruitment of motor units and changes in motor unit firing rate during the contraction. In all contractions up to the maximal force there was an accompanying increase in AMG amplitude, which is significantly different to the data which shows decreases in signal level after 80%MVC (Orizio, 1989a). This decrease may be due to the increase in relaxation time seen during fatigue (Bigland-Ritchie and Woods, 1984) which would reduce the movement of the muscle. An increase in muscle stiffness as seen during fatigue (Sadamoto et al, 1983) would also reduce movements of the muscle and reduce the sound wave. Thus, the greater the force the quicker the rate of onset of fatigue and the quicker the sound amplitude decreases as was reported.

Changes in the EMG signal.

The increase in EMG seen during contractions of this type are well documented and may be clearly explained. EMG is the summation of all the action potentials from the muscle fibres contracting within the muscle. As described in the introduction, the muscle is composed of motor units, the number of which depends on the size and type of muscle and the number of muscle fibres per motor unit depends on how finely the muscle is controlled. The motor units are controlled by the central and peripheral nervous systems, causing them to fire and then altering their firing rate in order to produce the required contraction.

Peripheral Control

This is achieved by specialized receptors within the muscle, and within associated structures such as tendon and connective tissue. Together they signal the conditions surrounding the muscle and relay this back to the central nervous system (CNS). The CNS receives input not only from the muscle in question but also from the other agonistic and antagonistic muscles and higher centres, integrates the information and then makes an appropriate response.

The muscle spindle is the sensory receptor that is present within the muscle itself. It is fusiform in shape, and lies in parallel with the extrafusal muscle fibres. Within the muscle spindle there are fibres that are also able to contract and these are called intrafusal muscle fibres. The intrafusal muscle fibres can be further subdivided into bag1, bag2, and chain fibres, and the nuclei of the chain fibre are arranged in parallel with the fibres whereas the bag fibre nuclei are clustered in the centre of the spindle. Afferent nerve fibres are wrapped around these structures so that when there is a change in the length of the muscle these nerve endings are affected. The nerves are from two sensory groups, the Ia and II fibres. The Ia fibres have a monosynaptic input to the α motoneurone pool for the muscle and a disynaptic inhibitory input to the antagonistic muscle or group of muscles. The group II afferent nerves also have an excitatory input to the motoneurone pool, although with disynaptic connections. Both of these groups modify their firing rate with the length of the muscle. If the muscle is stretched, the Ia ending will be stretched rather like a spring and the II ending will become more spread out along the fibre. The endings may also be stretched if the intrafusal fibres contract and this is achieved by the γ motoneurone innervation to the ends of the intrafusal fibres. In both of these cases the discharge rate of the receptors would increase. If the muscle is shortened, for example during a contraction, the muscle spindle is relaxed, or unloaded, and the discharge rate from both the Ia and II sensory endings would be decreased. Additionally, it is thought that the Ia sensory endings respond mainly to changes in velocity (Rack, 1981), whereas, the II sensory endings respond only to a change in length. Therefore, when the muscle is stretched both the Ia and II endings will fire during the moving phase, but when the muscle reaches it's new length it will be the II sensory endings that continue to fire.

When a muscle contracts, both the α and γ motoneurones are activated so that both extra and intra fusal muscle fibres contract. Thus, it seems as if the length of the muscle spindle is reset to accommodate the change in muscle length, and their

sensitivity will be maintained over a wide range of lengths. This information leads to the fact that any changes in length and velocity produced by the extrafusal fibres in the vicinity of the muscle spindle will alter the discharge in the afferent fibres.

The muscle spindle has often been visualised to work rather like a servomotor controlling and compensating for loads applied to tendons. However, more recently the effect of individual motor units on the muscle spindle output has been investigated (McKeon and Burke, 1983). It was found that the muscle spindle was most sensitive to the contraction of motor units within its area and was relatively unaffected by motor units more remotely located. Thus it would appear that the muscle is divided into compartments with respect to the sensory information from the muscle spindles, although this may not be true for all muscles.

The sensory input, the Ia and II afferent nerves, synapse directly with the α motoneurones and cause them to fire causing the muscle to contract. The motoneurones innervate groups of muscle fibres and this functional unit is called a motor unit as has been discussed previously. The motor units can be composed of any number of muscle fibres from 2 to 100's, and this is the basis of the grading of force that the motor unit produces. During muscle contraction in order to increase the force produced by the muscle, a combination of two processes can occur. There can be recruitment of more motor units or there can be a increase in the rate at which any one motor unit fires, or both of these patterns may occur simultaneously within the muscle.

Recruitment

As the muscle contracts there is a progressive recruitment of the motor units, starting with the smallest units and progressing to larger units as dictated by the strength of the contraction. This has been termed the 'Size Principle' (Henneman et

al, 1965). Additionally, it was found that the motor units with the lowest conduction velocity were also the most excitable (Kernell and Monster, 1981) and that the fatiguing properties of the muscle were correlated with the recruitment of motor units (Fleshman et al, 1981) with the most fatigue resistant being recruited first. The twitch tension was recorded from motor units in first dorsal interosseus and it was found that the lowest force units were recruited first followed by the higher force units (Milner-Brown et al, 1973a). These observations lead to the general overview that the first motor units to be recruited during a contraction are low threshold, have few muscle fibres associated with them and have a small force output, thus allowing for finer control at the beginning of a movement. These units are also fatigue resistant, which would be a requirement if they remain active for the whole contraction. In contrast the last motor units to be recruited are obviously of much higher recruitment threshold, have a large number of fibres associated with them and have a high force output. They are also fast fatiguing fibres which allows for them to fire for a short time near the end of a contraction. It was found that during rapid contractions of increasing force and ballistic contractions, this pattern of recruitment is still adhered to (Desmedt and Godaux, 1981) although the motor units appeared to fire earlier in the contraction with the increased speed of the contraction. This was explained by the observation that the motor units fired at around the same muscle tension, which was just being achieved more quickly (Budingen and Freund, 1976).

When the force of a contraction is decreased, the motor units stop firing in the reverse order to that in which they were recruited (De Luca et al, 1982a,b; Burke, 1980). This was found to be true for both deltoid and first dorsal interosseus, and did not alter with the speed of reduction of force. There is some indication that motor units cease firing at slightly higher forces than the force at which they were recruited (Milner-Brown et al, 1973a) indicating that there is some lag between firing of the motor unit and the force developed, and this is seen in both directions.

Firing Rate

The firing rate of a motor unit is defined as the number of times it fires per second and increasing the firing rate of a motor unit will increase the contribution of that motor unit to the force measured from the muscle. It was found that the maximum firing rate for a motor unit was 50Hz. (Adrian and Bronk, 1928; Lindsley, 1935). It was also found that the firing rate had a strong dependence on the force during an isometric contraction. In biceps brachii the motor units recruited at the lowest force had firing rates of between 7 and 12 Hz, whereas at the end of the contraction the firing rate had increased to 20Hz (Clamann, 1970). However, this was complicated by the fact that at low percentages of the maximal contraction the firing rate was maximal, being 20Hz, there was no further increase in firing rate even up to the maximal force, and there was no further recruitment after 75%MVC. Contractions of rectus femoris showed that the early firing motor units had a firing frequency of 5 to 11Hz and there was an increase in firing rate of these units to 18 to 21Hz at 45%MVC (Person and Kudina, 1971). It was also found during this investigation that the units recruited later in the contraction, that is with a higher recruitment threshold, showed less increase in the firing rate with the increase in force. This was also found in abductor digiti minimi, with the lower threshold motor units showing much more marked increases in rate than those recruited later in the contraction (Tanji and Kato, 1973a,b). The firing rate also appears to vary with the muscle under investigation. In smaller muscles it was found that the motor units reached relatively higher values than those in larger muscles. In first dorsal interosseus it was found that the motor units started to fire around 6 to 8 Hz and reached a maximum rate of around 30 to 35Hz at 80%MVC, whereas with deltoid the firing rates were higher initially, 12 to 15Hz, but only rose to around 25Hz at the same percentage of the maximal contraction (De Luca et al, 1982a,b).

To summarise, in order to increase the force output from a muscle two methods can be employed, namely recruitment and an increase in firing rate. In general, it

appears as if both processes occur within the same contraction, although at low forces recruitment seems to be the main mechanism for increasing force whereas at higher forces the firing rate of motor units is more important (Milner-Brown et al, 1973b). However, it has also been shown that recruitment occurs up to the maximal contraction in some muscles (Kukulka and Clamann, 1981). Different muscles seem to show different ranges of firing rate and motor units recruited at the beginning of the contraction showing more change in rate than the units recruited later.

Changes in the AMG signal.

During the isometric contractions with increases in force the peak to peak amplitude of the AMG signal also showed increases. Simply, this must be an increase in the amplitude of the vibration detected at the skin surface. However, what causes these changes is much less well documented, or understood, than EMG. The contraction of the muscle must have a major role in altering the vibrations from the muscle and therefore it would seem reasonable to suggest that recruitment and changes in firing rate would be influential on the AMG signal. It was reported that even during a maximal contraction when all motor units are assumed to be active, there is still some movement across the muscle surface as motor units fire (Deleze,1961). In single muscle fibres, there is not a steady shortening of the fibre when it is stimulated, but rather the fibre shortens in steps and even when it reaches the final length at which it will contract, there is some readjustment of the length (Edman and Regiani, 1984). It would be reasonable to suggest that these adjustments in length during a contraction could cause some transmission to the muscle surface.

Root mean square analysis.

Discussion of the EMG and AMG signals leads to the analysis of the EMG and the AMG signal. As both recruitment and firing rate are important in determining the force of the contraction, the analysis of the EMG signal must be able to take this into account when calculating the signal level recorded. As was detailed in the materials and methods section the root mean square analysis takes these variables into account along with the shapes of the motor unit action potentials. This calculation is not affected by chance superimpositions of action potentials which would tend to cancel out activity recorded at the surface. However, it is affected by the synchronisation of motor unit firing and this is considered within the calculation and accounted for. The rms analysis has been shown to be sensitive to firing rate, recruitment and synchronisation of the motor unit action potentials measured using surface EMG electrodes, in sustained contractions at constant force (Stulen and De Luca, 1978). In contrast, the rectified integrated value, which is a more commonly used analysis of the EMG signal, has no means by which synchronisation can be included, and is affected by the superimposition and partial cancellation of two motor unit action potentials (Basmajian and De Luca, 1985). AMG is a very similar type of signal to EMG, crossing the base line positively and negatively, although the frequency of this process is obviously much slower than for the EMG. Therefore, it was decided to use identical analysis techniques for both EMG and AMG, simplifying the procedures and allowing simultaneous analysis.

Contribution of physiological tremor to AMG.

Physiological tremor is the vibrations, or oscillations, that can be seen when a subject maintains a muscular contraction. Tremor has been studied systematically but its origin has never been fully understood. The following comment incorporates some of the relevant experimental data in order to illustrate its complexity. Tremor was first thought to originate from a servo control mechanism of muscle length,

mediated by the muscle spindles and the central nervous system. It was proposed that as the muscle lengthened, the muscle spindle would also be stretched and there would be a reflex shortening of the muscle and this would continue as the length of the muscle changed thus producing an oscillation around the required length. It was found that these oscillations occured with a frequency of around 8-10Hz. The origin of these oscillations was investigated using tremor in an outstretched finger (Lippold, 1969). The finger could be seen to oscillate, at the frequency of physiological tremor, when the finger was outstretched and when the finger was tapped during a contraction, a damped oscillation was elicited at the same frequency as the physiological tremor. The EMG signal from the finger showed bursts of activity in phase with the movement (Lippold et al, 1970.). It was proposed that both this oscillation and the physiological tremor were of the same origin, as both were depressed after a few minutes of ischaemia. It seemed that peripheral receptors, principally the muscle spindles, were being used to keep the position of the outstretched finger constant. The oscillatory behaviour after tapping the limb can be explained by the initial increase in firing frequency of the muscle spindle caused by stretching the muscle. This would cause an increase in α motoneurone firing and cause the muscle to contract, increasing the force. There would be a 'silent period' in the EMG signal recorded from the muscle due to the unloading of the muscle spindle, decreasing the firing rate and the synaptic activity to the α motoneurones. There would also be an increase in the Renshaw inhibition of the α motoneurones. This would cause a decrease in the force from the muscle. After this brief period the EMG increases again, and if the increase is large enough this could initiate this sequence again, thus inducing contraction and relaxation of the muscle, producing oscillations.

It was found that the bursts of EMG and the oscillations were synchronous and occured at a frequency of 9Hz (Lippold et al, 1957). Cooling the muscle reduced the frequency of oscillation, suggesting that the rhythm was due to the decreased

conduction velocity, increasing the servo loop delay. It was also found that if the muscle was stretched during a contraction the oscillations and EMG grouping increased, implying strong influences on the oscillations by the muscle spindles.

Analysis of the tremor frequencies showed 2 main peaks, 4-6Hz and 8-12Hz, when the middle finger was extended for periods of up to 1 minute (Gottlieb and Lippold, 1983). It was shown that the low frequency tremor was a separate entity from the 8-12 Hz tremor, and it was proposed that they both had the same underlying mechanism although the 4-6Hz tremor involved a longer neuronal delay, perhaps involving relay to the cerebral cortex. It should be noted that harmonics of the 4-6Hz tremor could result in the second tremor frequency and thus be determined as a second signal.

The 8-12Hz peak in the frequencies measured during tremor was found to be present when the hand was raised (Burne et al, 1984). When the contraction was isotonic there was a prominent peak in the spectrum at these frequencies, however, when the hand was held against a support and the contraction was isometric, there was no peak seen in the frequency spectrum. The isometric contractions prevented excessive activation of the muscle spindles and tendon organs by keeping the stretch and muscle length constant, and under this condition tremor was abolished. It was proposed that peripheral factors such as inertia of the limb and viscoelastic properties of the muscles and tendons determine the amplitude and frequency of the tremor. It was concluded that the tremor was due to activation of muscle proprioceptors rather than any central control mechanism.

Tremor in Parkinson's disease patients has been found to be generated centrally with no feedback loop from muscle (Walsh, 1969; Walsh, 1970a,b). This tremor continued even when the limb was oscillated by external sources. It has also been proposed that the tremor was due to the pulse of blood through the arterial system, but this was found to be important only where there was no accompanying muscle

contraction (Brumlick,1962). The mechanical resonance in the limb due to inertia and stiffness of the tissue has also been thought to have a role to play in muscle tremor (Rietz and Stiles, 1974).

In summary, tremor in muscles is not well understood although the servo-loop control of the muscle is still thought to pay a major role in its development. The contribution of tremor to the AMG signal must not be discounted, especially at the high force levels where any oscillation in force is bound to be of larger amplitude and therefore more easily detected by the accelerometer. However, all the contractions recorded in this thesis were isometric and tremor has been found to be of low amplitude during these contractions (Burne et al, 1984). The choice of AMG detector is a difficult one and the results reported in this thesis used an accelerometer. It must be remembered that accelerometers are often used to detect tremor and therefore must be sensitive to this type of movement. It is possible that a certain amount of tremor was detected although this was not the aim of the project. This may be part of the cause of the large amount of movement detected at the start of some of the contractions. Positioning of the limb coupled with a long experimental protocol may have resulted in this undesirable effect.

Definition of AMG

One of the main problems is the confusion over what can be termed AMG. The recorded signals from isometric contractions and from stimulated contractions are very different both in appearance and origin. An isometric contraction reveals a vibration continuously throughout, as has been seen in the results described in the previous chapters. In some of the experiments the AMG was detected using a contact sensor rather than the accelerometer used in these experiments. The contact sensor is placed over the muscle and strapped in place. The contact sensor houses a piezoelectric crystal which when distorted, produces an electric signal that can be

related to the amount of displacement. With this type of arrangement, the movements measured are subject to the tightening of the strap over the muscle and around the arm. The contact sensor has an optimum attachment pressure, this is generally achieved in the relaxed position. However, when the subject starts to contract the pressure alters, and this may increase the pressure beyond the ideal level, so the crystal is no longer producing the same response. The initial trials for the experiments presented within this thesis were performed using this type of contact sensor and a number of other problems were found to be associated with the attachment of the contact sensor to the muscle. Due to the fairly rigid strap, the contact sensor tended to remain in one position on the skin, while the muscle tended to move underneath. This obviously altered the contact pressure and the area of contact with the muscle. It is possible that the reported decrease in amplitude of the AMG signal at the higher force contractions, represents the limitations of the recording equipment rather than a real effect of the AMG signal.

The other reports recording AMG during contractions tend to use nerve stimulation to cause muscular contraction either in human (Barry et al, 1992) or frog (Barry and Cole, 1990, 1988a, b; Barry, 1987) muscle. The main difficulty with this is that the twitch itself causes a movement of the muscle, becoming shorter and fatter, which tends to push the skin overlying the muscle outwards. Obviously anything lying over the skin will move in a similar manner. The biphasic signal recorded during this type of contraction is therefore a form of movement artifact, with the accelerometer being moved away from the initial position and then as the muscle relaxes, moving back to the initial position. It has been shown (Frangioni et al, 1987) that in this type of contraction, when multiple stimuli are given to produce an unfused tetanus, the biphasic signals recorded at successive stimuli decrease in amplitude. When a fused tetanus is produced it appears as if there is only one signal at the beginning of the contraction, resembling a damped oscillation. In an unfused tetanus, the muscle relaxes slightly in between stimuli and there is a small amount

of movement with each stimulus and the accompanying oscillation would be transmitted to the accelerometer. During a fused tetanus the stimuli are close enough together to maintain the contraction and allow no relaxation in between stimuli, thus there is no movement apart from at the beginning of the contraction, when a damped oscillation can be recorded with the accelerometer.

Blood flow within the arm is pulsatile flow which must contribute to the overall pattern of movement and vibration within the arm. It is probably at least partly responsible for the signal seen in between contractions when blood flow will be unimpeded. However, during an isometric contraction, especially at the higher force levels, the blood flow would be almost completely stopped due to the contracting fibres pushing on the blood vessels and at least partially occluding them.

The difference in the range is almost certainly accounted for by the fact that during the isometric contraction, the muscle is constricted in movement thus reducing the low frequency content. Probably the most interesting observation from this work comes from the analysis of the recorded wave. Taking the double time integral which corresponds to the displacement of the accelerometer, showed a similarity to the force record. This is fundamental to the whole series of recordings from stimulated twitches. When the muscle is twitched the muscle contracts, pushing the accelerometer out with it, and when the muscle relaxes the accelerometer moves back to the resting position. The difference in latencies can be explained by the electrical activity travelling quickly through the saline medium to the surface and appearing first. The AMG follows closely as the muscle stiffens and starts to contract if it is not an isometric contraction, and this is closely followed by the force record which takes time to develop due to the stretching of the series and parallel components.

Muscle Fatigue

As has been discussed previously, fatigue has a number of causes, but can be characterised by a decrease in force and a slowing of contractile speed (Hill, 1913). The contractions were arranged so that a target force was maintained until the level fell by 10% and the contraction was terminated after this. As has been discussed, EMG amplitude rises during the contraction due to the increase in the number of active motor units, and the rate of firing as the muscle fatigues. This was seen in all of the muscles and most of the subjects. The main problem was the recruitment of other muscles to maintain force, which would result in changes in the force produced from the measured muscle. The behaviour of the AMG signal was more difficult to interpret. In the majority of the contractions, the AMG amplitude increased slightly at the beginning of the contraction, remained at a relatively steady level for most of the contraction and then increased quite dramatically towards the end of the contraction. Thus, it might be sensible to suggest that the AMG amplitude remained unchanged for most of the contraction, perhaps reflecting the level of the contraction and the lack of movement of the arm. Towards the end of the contraction when the subject was experiencing some discomfort, there was a tendency to increase movement and often the subject tried to change position to alleviate any pain, although this was stopped in most of the contractions. At the end of the contractions there was also a certain amount of tremor and this would have been transmitted to the accelerometer, which may explain the increase in signal amplitude, seen in some contractions towards the end of the contraction. Thus, it would be difficult to draw any definite conclusions about the AMG signal during the fatiguing contractions. This is in contrast to work done using sustained contractions of small hand muscles (Goldenberg et al, 1991). It was found that during contractions at 75%MVC AMG amplitude remained constant, whereas at 50%MVC there was a decrease from beginning to end, and for 15 and 25%MVC there was a rise in AMG amplitude. It was concluded that these changes were

reflecting the recruitment strategies of the muscle at different percentages of the maximal force. This is may be true if within the muscle, below 50%MVC, not all motor units are active. There would then be recruitment of larger motor units to maintain force which would result in EMG amplitude rising and the possibility of AMG rising as well. At 50%MVC, no further motor units could be recruited but a change in the rate of the active motor units firing would be expected. However, this was not shown as an increase in the AMG amplitude. It was suggested that rather than reflecting firing properties, AMG reflected lateral expansion of the muscle which would change during recruitment of larger motor units but not with changes in firing rate of units already active. The constant AMG amplitude at 75%MVC was considered to be due to the contraction being too short to see any AMG changes with motor unit firing. This work goes some way to explaining the relationship of AMG and fatigue but there is still no clear picture. Recording from biceps brachii yet another series of relationships was found (Orizio et al, 1989b). It was found that at 20%MVC AMG amplitude was constant for a short period and then increased, at 40%MVC AMG amplitude fluctuated, and above this the AMG amplitude decreased exponentially. This was very similar to the previously described work. The data described in this thesis from contractions at 75%MVC showed no such trends. There were often increases in amplitude towards the end of the contraction or signal levels fluctuated to such an extent that no relationship was clear. This suggests that the signal may have been contaminated by tremor from the muscles involved which increased as the muscle fatigued.

FREQUENCY

The frequency of biological signals can be used in a variety of ways to give information about the changes that are being seen both in the tissue and in the recording condition.

The main observations when comparing the two signals are firstly the difference in median frequency and secondly the difference in the range of frequencies. The AMG median frequency was significantly lower than the EMG frequency, being between 15 and 30 Hz for most of the contractions and changed less during the stages of the experiment. The EMG frequencies showed a much wider range and showed significant shifts in the median frequency depending on the type of isometric contraction. The AMG frequencies agreed with the current literature and showed very similar results to those reported from isometric contractions (Michielli and Oster,1989; Figini and Diemont, 1989). However, there has been very little systematic study of frequency, and therefore this study of AMG frequency during the different types of isometric contraction is unique. The AMG median frequency was slightly higher when recorded from rectus femoris than for any of the other muscles ($p=0.01$). Overall, AMG changed very little during the contractions at the percentages of the maximal contraction, decreasing slightly, although not significantly, for contractions of first dorsal interosseus. EMG showed a tendency for the median frequency to rise with the increase in force, although there was no change in the range of frequencies. Contractions of first dorsal interosseus showed a decrease in EMG median frequency and adductor pollicis showed no change in median frequency. During the ramp contractions, triceps and biceps brachii showed an increase, or a tendency to increase, in both the EMG and the AMG median frequency. However, first dorsal interosseus and adductor pollicis showed a decrease in EMG, and virtually no change in the AMG median frequency. Rectus femoris showed a significant rise in EMG median frequency and no change in AMG median frequency. Therefore summarising, there appears to be a tendency for the EMG median frequency to rise with the increase in force both in contractions to percentages of maximal and during a slow ramp contraction, although there is a dependence on muscle size and location influencing the changes in EMG frequency. The larger muscles would have larger motor units compared with smaller muscles and frequencies would tend to be lower. There does not appear to be much evidence

for the AMG median frequency to change. The change in EMG frequency is due to the changing action potential waveforms, and an increase in frequency must be due to a decrease in the action potential duration. This would be due to an increase in the conduction velocity of the fibres. AMG frequency does not appear to be affected by a change in the force produced by the muscle during isometric contractions.

Changes in EMG frequency are often studied during fatiguing contractions and a frequency shift to lower frequencies is reported (Bigland-Ritchie et al, 1983). This shift in median frequency was seen during the contractions at 75%MVC from biceps, triceps and adductor pollicis, although no trend was seen for first dorsal interosseus. This shift in frequency is due to a decrease in the firing rate of the motor units and a decrease in the conduction velocity in the muscle fibres increasing the action potential duration. Accumulation of hydrogen ions during the contraction appears to decrease the membrane potential possibly either by affecting trans membrane proteins and by altering the electric field (Bass and Moore, 1973; Jennische, 1982). Thus the membrane excitability is reduced and the conduction velocity decreases, and so there is a frequency shift to lower frequencies. AMG showed no systematic change in frequency with both increases and decreases being seen. This may be due to the contribution of tremor to the signal and masking any changes in AMG frequency. This is obviously an important consideration and one which should be carefully considered before any conclusions are drawn about AMG frequencies. It may be true that the recording equipment was not suited to these type of investigations and may explain why no conclusive data was recorded.

Observations made during recording.

Recording vibrations from the surface of the skin brings with it a number of artifacts that must also be taken into consideration. The accelerometer is very sensitive and therefore any interference will be detected as well as the vibrations

from the muscle. Sources of interference are numerous, and includes the subject touching the accelerometer directly which would result in an extremely large amplitude signal. This would be easy to distinguish from any other signal due to this large amplitude, and generally occurs in between contractions if the subject rubs the muscle. Touching the surrounding skin or moving the limb also results in large artifacts by changing the orientation of the accelerometer with respect to the skin surface, however these occur most commonly between contractions and are therefore easy to identify and do not interfere with the recording. More difficult to separate from the contraction are the 'spikes' which often occur at the beginning and end of the contractions. When the subject starts to contract, especially when they are contracting to a target force, there is often a sharp amplitude 'spike' as the subject moves abruptly to the force level. This is often accompanied by a similar 'spike' at the end of the contraction as the subject relaxes rapidly. These artifacts although part of the contraction are quite easily distinguished from the rest of the record. The most difficult situation is when the subject moves or touches the accelerometer during a contraction, this rendered the signal useless and the contraction must be repeated.

The transmission of vibratory signals from an actively contracting muscle to other tissues was investigated by stimulating the biceps muscle and recording the signal both over the muscle and from the underside of the arm over the triceps muscle (Wee and Ashley, 1991). The amplitudes of the signals from both recording sites were compared, and the ratio of biceps to triceps was calculated. It was found that the signals recorded at the distant site were around half the size of those from the stimulation site. This would suggest that there may be a significant spreading of the signal with the potential to contaminate the signal from any one individual muscle. It is important to remember that these signals were recorded using a piezoelectric transducer taped to the skin. This in itself is large enough to distort the skin lying underneath which must affect the recorded signal. When the muscle is stimulated

there is a large contraction 'bulging' into the transducer producing the wave of activity, this must also affect the skin overlying the triceps muscle and the attached transducer. In effect, what is recorded is a large 'wobble' and this is recorded both over the muscle and at the other side of the arm over the triceps. Perhaps more informative would have been the study of the signal during a voluntary contraction and looking at spreading of the signal to sites other than on the upper arm. This does introduce an important area for examination, the possibility of the recorded vibrations being composed of the true vibratory signal from the muscle and a component from other sources not necessarily from other muscles. It is impossible to eliminate this, although it can be easily investigated.

Other sources of error are that if the subject fails to produce a maximal contraction this invalidates all the other contractions at the percentages of the maximal force. In an attempt to eliminate this, the subject was asked to perform 3 trial maximal contractions before the maximum force was obtained, allowing the subject to become familiar with the procedure. This also leads to the question of which muscles are producing the force. Contracting one muscle independently is a very difficult manoeuvre and inevitably other agonistic muscles are also contracting. One of the problems found with the contraction of adductor pollicis was that in the recording position, the subjects seemed to rotate the lower portion of the arm, thus facilitating the contraction. Contraction of triceps brachii lead to a similar problem with the subject 'leaning' into the contraction, pushing against the transducer again, aiding the contraction. The subject was constantly reminded to avoid this condition, and any contractions that were obviously augmenting force production were eliminated and repeated. However, this obviously would increase the force overall, although the muscle under investigation may be contracting maximally or sub-maximally, and it is difficult to determine to what extent the other muscles are contracting during the series of contractions, therefore this should be remembered when examining the results. Some people favour strapping the subject's limb into a

rigid frame during investigation, so that if, for example, first dorsal interosseus was being examined, the forearm, and perhaps the other fingers, would be immobilised. This does not stop the use of other muscles, and may even aid their use as there is something to push against. It was therefore decided to avoid the use of retaining frames and encourage the subject to contract the muscle in question, and relax all the other muscles as much as possible.

The recording of the EMG signal is not without problems as well. Placement of the electrodes over the muscle obviously dictates the portion of the muscle being recorded, surface recording results in filtering of the signal as it passes to the surface (Lynn et al, 1978), changes in the orientation of the electrodes to the muscle surface will result in a change in the detection of the signal (Vigreux, 1979), electrical 'noise' from the equipment used to detect EMG can distort the signal although this can be significantly reduced by having a filtering system incorporated into the recording equipment, movement of the leads during the contraction will result in an induced current and this can be seen clearly in some of the contractions from rectus femoris as the change in the base line as the subject moves. The electrode contact is also crucial in the detection of the EMG signal and the electrodes must be cleaned, the skin cleared of any grease or dirt and abrased slightly to reduce the resistance, and an electrolyte gel applied to reduce this resistance further. Different types of electrodes and recording equipment have different sensitivities, and therefore it is important keep the recording equipment constant throughout a series of experiments to eliminate changes in detection. Placement of the electrodes should not be changed during an experiment if the results are to be directly compared, as this would change the recording area and the population of motor units below the electrodes. During the experiments the placement of electrodes, the preparation of the skin, and the recording equipment was kept constant in an attempt to reduce these sources of error.

SUMMARY

Summary

The data presented within this thesis has attempted to clarify the recording of AMG from human skeletal muscle during voluntary isometric contractions. During this pursuit some important points have emerged. Firstly, during isometric contractions at percentages of the maximal force, a vibratory signal can be recorded from the skin surface overlying the muscle and this accompanies the electrical activity from the muscle. The amplitude of this signal appears to alter with the force of the contraction in a very similar manner to EMG, increasing when the force increases and decreasing when force decreases. In contractions to percentages of the maximal force it appeared as if AMG and EMG had linear relationships with force, although during the ramp contractions to maximal force both EMG and AMG displayed a curvilinear relationship with force. These results appear to reflect the type of contraction and the behaviour of the motor units. The ramp contractions show all the points in between the imposed force levels and show recruitment and changes in firing rate, whereas, the contractions at predetermined force levels show a small part of the range of force and possibly reflect the more 'static' population of motor units firing.

However, during the fatiguing contractions, held at 75%MVC for as long as possible, the two signals seem to diverge with EMG tending to show increases in amplitude with time and AMG showing no distinct trends. This is in contrast to other reported work which showed a tendency for increases in AMG at lower force levels (<50%MVC), decreases in AMG at higher force levels (>50%) and fluctuations in level in the middle. This work has been reported to reflect movement of the muscle due to changes in firing properties of the motor units rather than the activity of the motor units directly.

Thus, from the work described in this thesis, during short contractions, AMG was as equally effective as EMG in predicting the force being produced by the muscle,

although during prolonged contractions AMG was no more useful than EMG in predicting the force and contraction state of the muscle. This probably reflects the additional problems of recording AMG from the surface of the muscle, which have already been discussed. During fatiguing contractions the tremor recorded from the muscle will increase and will inevitably add to the signal already being detected by the accelerometer.

Secondly, while quite distinct changes in EMG frequency with force and fatigue have been described for a variety of muscles, and have been discussed within this thesis, there appear to be no such trends for AMG. All the data has shown that the AMG frequency was much lower than the EMG frequencies, being below 30Hz in most cases while EMG had a wider range and higher mean values, between 60 and 120Hz. While the median frequency of EMG showed increases during the contractions with increasing force, and tended to fall during the fatiguing contractions, the AMG median frequency showed no trends and remained around the same value during most of the contractions. Again this is most probably due to contamination of the AMG signal by tremor, gross movement of the muscle and the equipment being more suited to tremor than AMG.

Thus, it would appear as if there are no distinguishing features of the AMG signal that could be usefully employed to monitor either force production from the muscle or the fatigue processes within the muscle. However, the study of AMG is at a relatively early stage and if continued, it is possible, if not probable, that a clearer understanding of AMG, its sources and its properties, will enable its use as a diagnostic and investigative tool in muscle physiology.

Conclusions

1. In all the muscles investigated when the voluntary force of the contraction was increased, both the EMG and AMG signals increased. Both exhibited similar relationships to force, which leads to the belief that AMG has its origin within the muscle and the contraction of the muscle fibres.

2. During the fatiguing contractions at 75%MVC, the force remained constant while the amplitude of the EMG signal rose throughout the contraction for all the muscles investigated. The AMG amplitude was seen to rise in the contractions of biceps and triceps brachii, and rectus femoris. The AMG amplitude during contractions of adductor pollicis and first dorsal interosseus increased, but with a very different relationship to that shown in the other muscles. This contradicts the data reported previously, which showed the AMG amplitude to remain constant, following the force level more closely than the EMG signal amplitude.

3. The median frequency of the EMG signal was seen to alter with the force and the type of contraction. The EMG median frequency rose with the increase in force during the contractions at percentages of the maximal contraction and during the ramp contractions. During the fatiguing contractions, the EMG median frequency decreased as the contraction progressed. The AMG median frequency showed no distinct trends with the changes in force or the type of contraction. This would appear to suggest that the AMG and EMG signals have separate sources or reflect different properties of the muscle.

Suggestions for future work.

It is essential that some form of standardisation for recording AMG is derived as it is very difficult to compare work from other groups with any degree of confidence. This would involve a decision on the type of equipment to use, units of AMG signal and some determination of what AMG is and how best to measure it.

In order to investigate the sources of sound the next stage of investigation must be done using animal work to provide an in vivo situation, and a point from which detailed analysis can be made. The relationship between AMG and single motor unit activity could be evaluated, expanding to the effect of changes in temperature, single twitch stimulation, tetanic stimulation and the changes during fatiguing contractions and recovery could be examined. This would lead towards the source of the AMG being evaluated which would be useful in the continuing experimentation using human voluntary and stimulated contractions.

In the meantime, it must be remembered that AMG is at a relatively early stage in our understanding and interpretation of results. Therefore, it should not be used, and this applies particularly in the clinical applications, without due consideration to more recognised forms of diagnosis. It is therefore an important area in experimental work but has not reached its full potential in clinical work.

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APPENDIX

per Entran Instruction Manuals unless otherwise indicated.

Property of: PO:

10Hz 2 3 4 5 6 7 8 9 100Hz 2 3 4 5 6 7 8 9 1KHz 2 3 4 5 6 7 8 9 10KHz

±(X) 1dB( )

PLEASE READ OPERATING INSTRUCTIONS BEFORE POWERING UNIT

dB

dB

dB

Entran ( ) Fairfield, N.J., USA ( ) Les Claves-S/Bois, France (X) Garston, Watford, Herts., UK

SPECIFICATIONS

Model : EGAX-F-100 S/N: 90D90D03-E05

Range : ±100g Limit: ±10000g Axis:

Temperature Range, Compensated: 70 to 170°F Operating: -40 to 250°F

CALIBRATION DATA

Subject to 10,000g Shock in: (X) Sensitive Axis Only ( ) All Axes

[1,2] Non-Linearity : ±1%	[1] Hysteresis:	[2] X-axis :	
[1] Zero :	[1] Therm. Z : ±1%FS/100°F	[2] TSS : +1/-4%/100°F	
R Cal :	with :	across :	
Output : 2.02 m/s	with In : 15.0 VDC	Max. In : 18.0 VDC	
Ref. Temp : 75°F	Ref. Hz : 100	[4] Approx. In.:	
Tested with:		S/N:	
Output :	with In :	Max. In :	
[3] Input Ω : 10.21	[3] Output Ω : 480	See Over : ( )	

ACCELEROMETER WIRING

To Module: Total:

+In: (X) Red ( ) +Out: (X) Grn ( )

( ) Grn & Blue

-In: (X) Blk ( ) -Out: (X) Wht ( )

Connector: Mate:

ELECTRONICS

Signal In Conn :	Mate :
+Sensor Power :	+Signal In :
-Sensor Power :	-Signal In :
Amp Out Conn :	Mate :
+Supply :	+Signal Out :
-Supply :	-Signal Out :
(For ±15V Input, Do not use unipolar 30V power supply)	Common :

FORM NO. 2CAL-A-UNIV REV A
SEMI-LOGARITHMIC

[1] If calibrated data equals or exceeds data sheet specifications, value shown is the data sheet value unless purchase order specifies actual calibrated value. [2] Value given is by manufacturing design and not calibrated unless purchase order specifies actual calibrated value. TSS is Thermal Sensitivity Shift. [3] Input/Output impedance is recorded for transducer alone. If [illegible]