# POSITION DEPENDENCE OF ANKLE JOINT DYNAMICS

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# ABSTRACT

The objective of the work that comprises this thesis was to examine the position-dependent behaviour of the human ankle joint dynamics. A series of experiments based on the use of system identification techniques were carried out; the experimental stimulus consisted of small-amplitude, stochastic perturbations of the ankle. The response to this input was modulated by two, experimentally-controlled parameters: tonically-maintained mean joint angles and tonically-maintained triceps surae or tibialis anterior contractions. This permitted the identification of the dynamic response of the torque (passive and active mechanics) and agonist electromyogram (reflex dynamics) for a functionally significant span of mean ankle angles and levels of tonic muscle contractions.

The major conclusions of this work were that (1) the position-dependent changes in the passive joint mechanics were large and functionally significant; (2) the active joint mechanics depended entirely on the magnitude of the actively-generated torque with position-dependent changes in the active mechanics being relatively small and variable; and (3) the triceps surae stretch reflexes depended upon the position of the ankle while tibialis anterior stretch reflexes did not. The functional implications of these results and, in particular, the relative importance of the active and passive processes in the behaviour of the ankle joint have been considered in light of these findings.

# RÉSUMÉ

L'objectif de cette thèse consiste à étudier la dépendance des propriétés dynamiques de la cheville chez les humains en fonction de sa position. Une série d'expériences basées sur l'utilisation de techniques d'identification des systèmes dynamiques ont été réalisées; le stimulus expérimental consistait en des perturbations stochastiques de faible amplitude de la cheville. La réponse à cette entrée était modulée par deux groupe de paramètres contrôlés expérimentalement: les angles du joint maintenu toniquement et les contractions toniques du muscle jambier antérieur et du triceps sural. Ceci a permis l'identification de la réponse dynamique du torque (des propriétés mécaniques actives et passives) et de l'électromyogramme agoniste (dynamique des réflexes) sur un domaine physiologiquement fonctionnel d'angles moyens de la cheville et de niveaux de contractions musculaires toniques.

Les conclusions majeures de ce travail furent: (1) les changements de la mécanique du joint passif en fonction de sa position sont importants et fonctionnellement significatifs; (2) la mécanique du joint actif depend entièrement de la valeur du torque généré activement et les effets de la position du joint sont relativement mineurs et variables; et (3) le réflexe myotatique du triceps sural dépend de la position de la cheville ceci n'étant pas le cas du réflexe myotatique du muscle jambier antérieur. A la lumière de ces résultats, et en particulier l'importance relative des processus passifs et actifs dans le comportement de la cheville, les conséquences ont été considérés plus profondiment.

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## GLOSSARY OF ABBREVIATIONS

| | |
|---|---|
| **A/D** | Analog-to-Digital |
| **B** | Viscous parameter |
| **D/A** | Digital-to-Analog |
| **EMG** | Electromyogram |
| **I** | Inertial parameter |
| **IRF** | Impulse response function |
| **K** | Elastic parameter |
| **MVC** | Maximum voluntary contraction |
| **NP** | Neutral-position |
| **postMVC** | Post-experiment maximum voluntary contraction |
| **PRBS** | Pseudo-random binary sequence |
| **preMVC** | Pre-experiment maximum voluntary contraction |
| **RIRF** | Reflex impulse response function |
| **ROM** | Range of motion |
| **SD** | Standard deviation |
| **TA** | Tibialis anterior |
| **TAP1** | First TA RIRF peak-to-peak magnitude |
| **TAP2** | Second TA RIRF peak-to-peak magniutde |
| **TS** | Triceps surae |
| **TSP** | TS RIRF peak-to-peak magnitude |
| **VAF** | Variance accounted for |

# CHAPTER 1: INTRODUCTION

The ankle is a remarkably versatile joint that appears to be well-adapted for both stability and mobility. These diverse, and potentially conflicting, capabilities have been achieved by a unique combination of hard and soft tissues which include a distinctive articulation, an extensive network of ligaments and retinaculae, and muscles of diverse structure and function. Thus the ankle is capable of generating the small, sustained torques that are required to minimize joint motion during quiet stance and also of withstanding the large but transient torques at a wide variety of joint angles during movement. Moreover, the ankle joint is able to adapt to the unusual forces or motions necessitated by the performance of elite activities such as ballet or gymnastics or by the deficits resulting from neuromuscular pathology such as pes equinus.

Although such functional abilities are recognized as important, very little quantitative data is available to support hypotheses of the underlying mechanisms. Indeed examinations of ankle function, and in particular its dependence on mean joint position, in intact human subjects are rare although the number of anatomical, biomechanical and physiological studies has increased in recent years. These studies have included an examination of changes in the intra-articular pressure (Eyring & Murray, 1964), in the location of the joint axis of rotation (Sammarco et al., 1973), in the EMG and torque responses to electrically-evoked or voluntary contractions (Marsh et al., 1981;

Sale et al., 1982) and in H-reflex (Mark et al., 1968; Burke et al., 1971) and microneurographic studies (Vallbo, 1974; Hulliger et al., 1982). Moreover, some progress has been made at the development of models that are able to predict the behaviour of certain characteristics such as joint structure (Procter & Paul, 1982) and stiffness (Gottlieb & Agarwal, 1978).

The results of these and other studies indicate that the position of the ankle can modify its behaviour. However, the interpretation of these effects has been hampered by the scope of the techniques and paradigms employed. Four important limitations may be identified:

(1) Inadequate range of motion (ROM).

In spite of measurements showing that many normal and abnormal functional movements entail large ankle joint excursions, most examinations of position-dependent behaviour have been restricted to a relatively small, and often ill-defined, portion of the ROM.

(2) Inadequate range of tonic muscle activity.

Ankle function has generally been studied either while the muscles were at rest or maintaining relatively small isometric contractions. Yet ankle torques of large magnitude are frequently encountered in functional situations.

(3) Separate examination of Tibialis Anterior and Triceps Surae.

Although the number of anatomical and physiological differences between these muscles suggest that their position-dependent responses will not be similar, the two antagonists have rarely been studied concurrently.

(4) Lack of dynamic analyses.

While considerable progress has been made toward delineating the static effects of changes in mean ankle position, very little is known about the dynamic effects of these changes.

## OBJECTIVES

The objective of the work that comprises this thesis was to examine the position dependence of ankle joint dynamic behaviour. This was accomplished by:

(1) delineating the effect of changes in mean ankle position on the passive joint mechanics, active joint mechanics and reflex dynamics;

(2) comparing the relative magnitudes of the position-dependent mechanical and reflex responses to those generated by active torque-dependent responses; and

(3) interpreting the results in the light of the functional requirements of the ankle.

Thus, a series of experiments using system identification techniques was carried out. Experimental stimuli consisted of small-amplitude, stochastic perturbations of the ankle (the dynamic position input). Responses to this input were characterized as functions of two parameters: (1) rotation of the ankle to a randomly selected ankle angle (tonic position parameter) and (2) generation of target levels of tonic muscle contractions (tonic voluntary parameter). This permitted the identification of the dynamic response of joint torque (passive and active mechanics) and agonist EMG (reflex dynamics) as functions of mean ankle position and torque.

This present work represents a significant improvement over previously reported studies in that:

(1) nine or ten angles, spanning more than 85% of the ROM and measured relative to a pre-determined absolute reference angle, were tested;

(2) testing was done in the absence of muscle activity and during tonic contractions of functionally-significant magnitudes;

(3) Tibialis anterior (TA) and triceps surae (TS) were tested by similar techniques so that their behaviour could be directly compared; and

(4) experimental and analytic techniques that permitted identification and subsequent modelling of dynamic responses at the different maintained positions were employed.

## SUMMARY

The literature pertaining to position-dependent changes in the static and dynamic behaviour of the ankle joint is presented in chapter 2. This review includes: a description of the notation used throughout the thesis; an evaluation of the magnitude and intersubject variability of the ankle ROM; and reviews of mechanical and sensory correlates of changes in mean ankle position.

The general experimental methods common to all the studies are presented in chapter 3. These methods include: a summary of the experimental paradigm and details related to the subjects and their attachment to the equipment; descriptions of the stimulus variables and the equipment controlled by these stimuli; an outline of the data acquisition procedures including details about the transducers and equipment used to sample these signals; and a review of the analytic techniques. Specific details pertaining to individual experiments are provided as required in later chapters.

Chapter 4 presents the results of a study designed to examine the effect of changes in mean position on the passive mechanical behaviour of the ankle. The major finding was that the position-dependent changes in the passive joint mechanics were much larger than previously thought; the ankle became considerably stiffer as it was rotated from mid-range positions toward either extreme of the ROM in the absence of voluntary muscle contraction. These changes in stiffness were found to be linearly related to the torque that was generated as the passive joint tissues were elongated. Consequently,

the passive ankle stiffness could be predicted from the passive joint torque. This work has been reported in abstract (Weiss et al., 1984b) and paper form (Weiss et al., 1985a).

Chapter 5 describes the effect of changes in mean position on the active mechanical behaviour of the ankle. The major finding was that the active joint mechanics depended entirely on the magnitude of the actively-generated torque; position-dependent changes in the active mechanics were relatively small and quite variable. Consequently, the total joint stiffness generated by any combination of ankle position and agonist activity can be determined from the total (active plus passive) joint torque. These results have been reported in abstract (Weiss et al., 1984c) and paper (Weiss et al., 1985b) form.

Chapter 6 considers the effect of changes in mean position on the TS and TA stretch reflexes. The major finding was that the magnitude of TS stretch reflexes depended upon the position of the ankle while TA stretch reflexes did not. Thus large increases in the TS stretch reflex magnitude resulted when the ankle was progressively dorsiflexed. In contrast, ankle mean position had only a minor effect on the TA stretch reflex magnitude. This work has been reported in abstract (Weiss et al., 1985c) and paper form (Weiss et al., 1985d).

The functional implications of these results and, in particular, the relative importance of the active and passive processes in the behaviour of the ankle joint are presented in chapter 7. This chapter also includes a statement of original contributions, limitations of the study, and recommendations for future work.

# CHAPTER 2: REVIEW OF THE LITERATURE

## INTRODUCTION

In this chapter the literature pertaining to position-dependent changes in the static and dynamic behaviour of the ankle joint will be examined. A brief description of the notation and terminology used throughout the thesis will be presented in the first section. The second section will include an overview of ankle joint structures. This brief description of the major hard and soft tissues provides an anatomical framework for the remaining sections. Ankle ROM, its extent and variability, will be the topic of the third section. The issues examined in this section are particularly relevant to the formulation of the experimental paradigm. In the next two sections, the mechanical and sensory correlates of changes in mean ankle position in the human subject will be reviewed. The results of these studies are useful to the interpretation of the findings obtained in this investigation.

## NOTATION AND TERMINOLOGY

Notation for the specification of ankle joint kinematics and kinetics has not been standardized; the notation used throughout this thesis is defined in this section. Whenever possible, results from other laboratories will be given in this notation to ensure consistency.

Ankle motion in the sagittal plane is measured with respect to the reference-position shown in Fig. 2.1 (top graph). This position is defined by orienting the plantar surface of the foot at right angles to a line marked between the fibular head and the lateral malleolus. (The reference-position is also known as the 'zero-position'.)

The ankle is dorsiflexed when the dorsum of the foot is rotated from any initial position toward the leg. Maximum or extreme dorsiflexion is reached when the ankle can be rotated no further. Dorsiflexor positions, denoted by positive values, comprise only those angles between the reference-position and maximum dorsiflexion. The ankle is plantarflexed when the dorsal surface of the foot is rotated from any initial position away from the leg. Maximum plantarflexion is reached when the ankle can be rotated no further. Plantarflexor positions, denoted by negative angles, comprise only those angles between the reference-position and maximum plantarflexion. These positions are illustrated in Fig. 2.1 (bottom graph). (All angles are reported in radians.)

Dorsiflexor torques, denoted by positive values, are generated by forces that tend to dorsiflex the foot about the ankle joint. Plantarflexor torques, denoted by negative values, are generated by forces that tend to plantarflex the foot about the ankle joint. (All torques are reported in Newton-meters.)

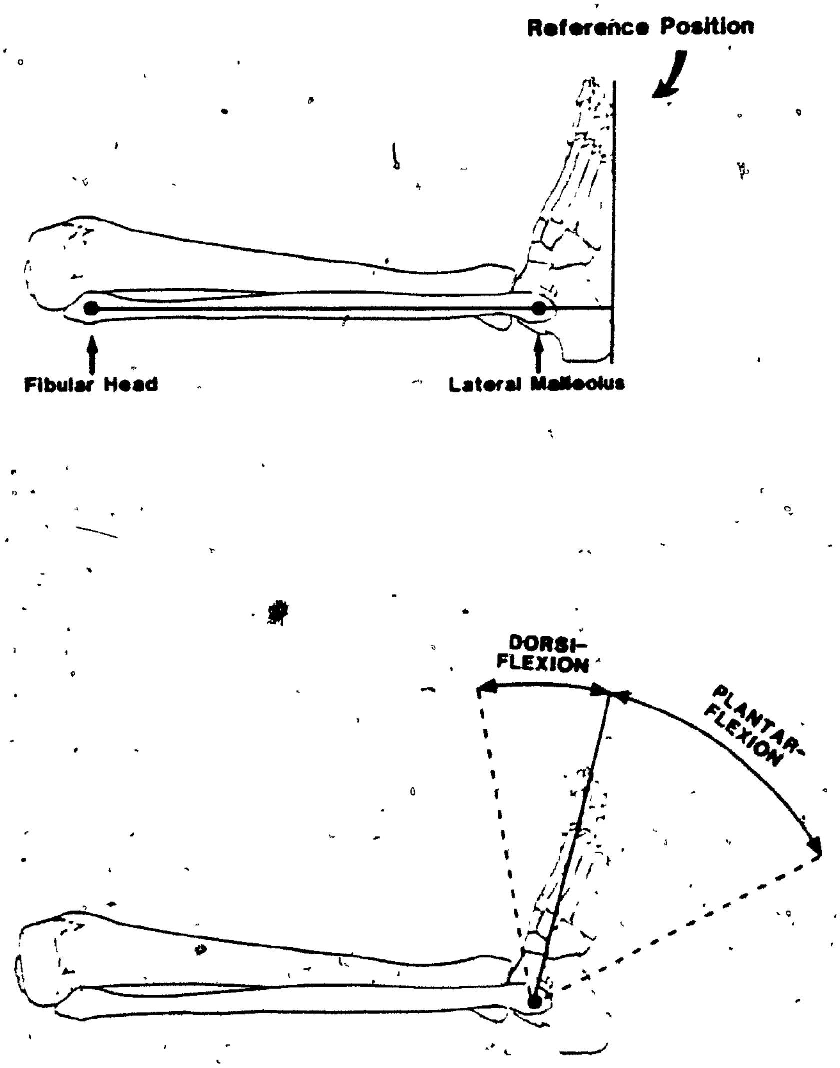


Fig. 2.1: View of the lover leg illustrating the reference-position used for measuring ankle position (top graph) and the sagittal plane motions (bottom graph).

Active dorsiflexor and plantarflexor torques are generated by the contraction of the respective ankle joint muscles. Rotation of the relaxed ankle (i.e. when the ankle muscles are not contracting) results in a change in the passive joint torque. However, there exists one ankle angle, called the neutral-position (NP), at which no passive joint torque is generated. This position is located near the center of the ROM. When the ankle is passively plantarflexed beyond the NP a dorsiflexor torque is generated and when the ankle is passively dorsiflexed beyond the NP a plantarflexor torque is generated. The total joint torque is the sum of the active and passive torques.

## OVERVIEW OF ANKLE JOINT ANATOMY

The objective of this section is to provide an anatomical framework for the studies reviewed in the remainder of the chapter. It includes a brief summary of the bony structures, the articulation, the ligaments and the plantarflexor and dorsiflexor muscles. Some of these structures are shown in Fig. 2.2.

### Bony Structures

The ankle joint, also known as the talocrural joint, is formed by the articulation of three bones: the tibia, fibula and talus. The large, medial tibia is formed from a triangular-shaped shaft that widens proximally into lateral and medial condyles and distally into the medial malleolus and a contoured articular surface known as the

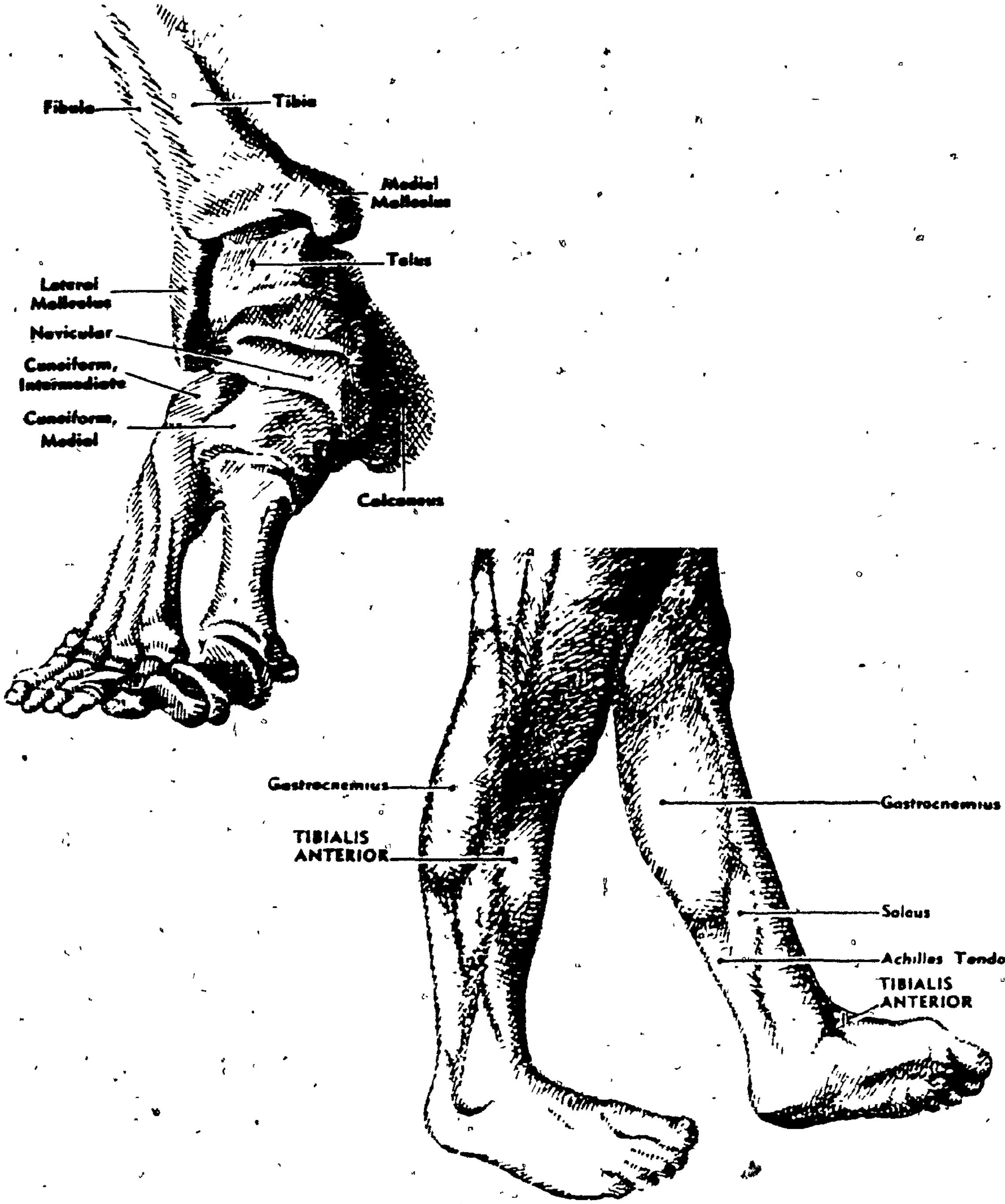


**Fig. 2.2:** Anterior view of the right foot and ankle joint (top drawing) showing the tibia, fibula, medial and lateral malleoli, talus, calcaneus, navicular, and medial and intermediate cuneiform. Lateral view of the lower legs (bottom drawing) showing the lateral gastrocnemius, soleus, Achilles tendon and tibialis anterior.

tibial plafond. The tibial condyles articulate with their femoral counterparts to form the knee joint.

The lateral-lying fibula has a long, slender shaft that expands proximally into a small head and distally into the lateral malleolus. A styloid process is located on the fibular head and it articulates with the lateral condyle of the tibia to form the proximal tibiofibular joint. The distal tibiofibular joint is located between the lower medial surfaces of these two bones. The fibula does not articulate with the femur.

The foot is composed of seven tarsal bones (including the talus, calcaneum, navicular, cuboid, and medial, intermediate and lateral cuneiform bones), five metatarsal bones and 14 phalanges. Of these bones, only the talus is of direct interest with respect to the talocrural articulation.

The talus is a small, saddle-shaped bone consisting of an anterior head and a posterior body that are joined by a narrow neck. The anterior and inferior surfaces of the head articulate with the navicular bone and the calcaneum. A second taleocalcaneal articulation is formed between the inferior surface of the talar body and the calcaneum. The cuboid talar body has a superior surface, called the trochlea, that is convex in the anterior-posterior direction and slightly concave in the lateral direction. Viewed from above, the trochlea is wedged-shaped, being wider anteriorly than posteriorly. The extent of this asymmetry varies from about 5% (less than 2 mm) to about 25% of trochlear width (about 6 mm) (Barnett &

Napier, 1952; Inman, 1976).

## Articulation

The distal juxtaposition of the tibia and fibula is such that the two malleoli and the tibial plafond form a mortise or slot into which the talar body fits. The mortise is a stable structure whose shape does not change as the ankle is rotated through its ROM (Inman, 1976). The distance between the inner edges of the two malleoli is larger anteriorly than posteriorly (Close, 1956) thereby matching the asymmetry of the wedge-shaped talus. The talus is held tightly within the mortise by ligaments and by bony projections from the anterior and posterior margins of the tibial surface (Kapandji, 1970).

Although the extent of the contact area between the plafond and the trochlea may vary with ankle angle (Gordon, 1970; Wynarsky & Greenwald, 1983), its articular cartilage is much thinner than other joints of the lower limb (Simon, 1971; Simon et al., 1973). The ankle is therefore considered to be a particularly congruent joint (Inman, 1976).

## Ligaments

The ankle joint ligaments have been divided into four functional groups: the lateral and medial collateral ligaments and the anterior and posterior ligaments. The lateral collateral ligament is formed from three bands each originating from the lateral malleolus. Two of these, the anterior and posterior talofibular ligaments insert on the talus whereas the calcaneofibular ligament inserts on the lateral surface of the calcaneum. The function of the anterior talofibular ligament is to limit inversion and plantarflexion whereas the posterior talofibular and calcaneofibular ligaments restrict inversion and dorsiflexion. The second functional group, the medial collateral ligament, is comprised of the deeper anterior and posterior talotibial ligaments and the more superficial deltoid ligament. These ligaments limit eversion, plantarflexion and dorsiflexion. Finally, the anterior and posterior ligaments are essentially localised thickenings of the capsule. Although not directly associated with the talocrual joint, the tibiofibular syndesmosis, which links the inferior tibiofibular joint, is important for the stability it provides to the mortise.

Investigations of ligament function have focussed primarily on geometric reconstructions, on mathematical models or on a combination of the two approaches (e.g. Zitzlsperger, 1960; Simkin, 1982; Allard et al., 1985). There is, however, still very little known about the position-dependent changes in length and forces generated by these ligaments.

## Plantarflexor and Dorsiflexor Muscles

The muscles of the posterior and lateral compartments plantarflex the ankle. Included in the posterior compartment are the triceps surae (formed from the soleus and the medial and lateral gastrocnemii), tibialis posterior, flexor hallucis longus, flexor digitorum longus and plantaris. These muscles are innervated by branches of the tibial nerve. Peroneus longus and brevis, the lateral compartment plantarflexors that also evert the foot, are innervated via branches of the superficial peroneal nerve. The anterior compartment muscles, including tibialis anterior, extensor hallucis longus, extensor digitorum longus and peroneus tertius dorsiflex the ankle. These muscles are innervated by branches of the deep peroneal nerve.

Soleus originates from the middle third of the medial border of the tibia, from the posterior surface of the head and the upper third of the shaft of the fibula and from a tendinous sheath arched between the upper portions of the two lower leg bones. Lateral gastrocnemius originates from the lateral aspect of the lateral femoral condyle. Medial gastrocnemius originates from the popliteal surface of the femur above the medial condyle. The gastrocnemii therefore also act as knee flexors. All three muscles are unipennate; the angle of pennation for soleus is about 0.35 rad, for medial gastrocnemius, about 0.3 rad and for lateral gastrocnemius about 0.15 rad (Alexander & Vernon, 1975). The more deeply lying soleus fibers join the Achilles tendon from below whereas the superficial lateral and medial gastrocnemii join it from above; the Achilles tendon then attaches to the posterior border of the calcaneum. Together these three muscles comprise the triceps surae (TS).

Tibialis anterior (TA) is a bipennate muscle with an angle of pennation of about .15 rad. It originates from the upper half of the lateral surface of the tibia and from the interosseous membrane. Its centrally located tendon passes beneath both extensor retinaculi and then attaches to the medial cuneiform bone and the adjoining base of the first metatarsal bone. The location of this distal attachment with respect to the subtalar joint means that TA also acts as an ankle inverter.

Early determinations of muscle cross-sectional area (e.g. Fick, 1910-1911) did not consider the muscles' angle of pennation. This error was corrected by Haxton (1944) who calculated the cross-sectional area by summing the diameters of small strips of each muscle. In an effort to obtain more realistic estimates, Alexander & Vernon (1975) made their measurements on a younger, more robust cadaver and considered the muscle mass and thickness as well as the angle of pennation. The TS cross-sectional area was 102 $cm^2$ whereas that of the only other plantarflexors studied, tibialis posterior and peroneus longus, was 28 $cm^2$ (Alexander & Vernon, 1975). The cross-sectional area of TA was 14 $cm^2$ whereas that of its synergist, extensor digitorum longus, was 8 $cm^2$ (Alexander & Vernon, 1975).

The TS moment arm is approximately 50 mm (Jergensen, 1951; Alexander & Vernon, 1975), about twice as large as the other plantarflexor muscle moment arms (Jergensen, 1951; Procter, 1980). Based on Fick's (1910-1911) estimates of cross-sectional area, Haxton's (1944) value for absolute muscle force and Jergensen's (1951) measured moment arm lengths, Murray et al. (1976) estimated that the TS accounted for about 80% of the total plantarflexor torque (Murray et al., 1976). However, the torque generated by a subject whose TS had been completely excised was almost 40% of that exerted by the intact leg. Although this would appear to contradict the theoretical estimates and indicate that the contributions of the other plantarflexors are relatively important, it should be noted that these latter muscles may have hypertrophied in the four month period between the surgical excision and the experiment.

Considerably less information is available on changes in the magnitude of the moment arm as a function of mean joint position. Procter (1980) and Procter & Paul (1982) report, for example, that the TS line of action shifted significantly over a joint excursion of about 0.8 rad. Changes in TA and peroneus brevis action lines relative to the sole of the foot were less than 0.1 rad over the same ROM (Procter, 1980). In another study, the magnitude of the TS moment arm, determined from an articulated skeleton and from radiographs obtained when the foot was placed at the extremes of range, estimated it to be about 1.7 times larger when the ankle was plantarflexed than when it was dorsiflexed (Sale et al., 1982); the moment arm magnitudes were not, however, provided by these authors.

## Conclusions

The ankle joint is a complex structure that has not yet been fully characterized. Fundamental issues related to the forces generated by the individual soft tissues, shifts in muscle and ligament action lines, and the changing axis of rotation have not been resolved. It is therefore evident that some assumptions will be required in any biomechanical analysis of this joint and that some important questions will continue to be unanswered.

One issue related to the design of the experimental paradigm is resolved in view of the information presented in this section. Due to its relatively large cross-sectional area and moment arm and due to its superficial location, TS has been chosen to represent the ankle plantarflexors. The role of TA as the dominant dorsiflexor is less evident; however, in the light of its superficial location, it has been chosen to represent the ankle dorsiflexors. Therefore, electrodes placed over these muscles can be expected to provide a good representation of the contraction characteristics during tonic activity and to indicate the absence of activity in the passive state.

## ANKLE JOINT RANGE OF MOTION

The extent and variability of the ankle joint ROM is reviewed in this section. This information is used in the formulation of the experimental paradigm.

### Variability and Magnitude

There is considerable variability in the magnitude of ankle ROM reported in the literature. This is due, in part, to different methods of measurement, inconsistent use of anatomical landmarks and the use of widely varying paradigms. For example, goniometry (Roaas & Anderson, 1982), radiography (Weseley et al., 1969), and cineradiography (Sammarco et al., 1973) are three very different methods used to record ROM. Results have been reported for active (Glanville & Kreezer, 1937) and passive (Roaas & Anderson, 1982) non-weight bearing or during weight bearing (Weseley et al., 1969). The ROM has also been shown to change with knee position (Sale et al., 1982).

Further variability in the ROM can be attributed to the anatomical and physiological changes which result from, for example, pathology or aging. Traumatic ankle injuries affecting bony or ligamentous structures can cause either decreased or increased ROM (Sammarco et al., 1973). A small but significant decrease in ROM was recorded for adult subjects (Boone & Azen, 1979).

Intersubject variability persisted even when the above factors were strictly controlled; the ROM of an homogenous group of middle-aged, male subjects varied between 0.5 to 1.5 rad (Segal, 1983; Roaas & Anderson, 1982). Thus, a priori estimates of a subject's ROM are not feasible.

## Axis of Rotation

Although little research had been done to determine the location of the ankle axis of rotation and position-dependent variations in that location, it was generally thought to run horizontally (i.e parallel to the tibial plafond) and to behave as a simple hinge joint (e.g. Cunningham, 1943; Inman, 1976). It was not until 1952 that Barnett & Napier, based on examinations of talar shape and malleolar facet curvature, proposed the existence of an oblique axis whose location depended on whether the joint was dorsiflexed or plantarflexed. This result was supported by Hick's (1953) empirical determination of the ankle axis. Dempster (1955) found that there was an erratic shift in the axis over the upper third of the trochlea and even larger fluctuations have been shown in the cineroentgenograms taken of normal ankles (Sammarco et al., 1973).

There is, however, no consensus as to whether the ankle axis is multiple or even double. The only study to examine a large number of specimens (Isman & Inman, 1969) used optical methods to locate the points of minimum motion. These researchers found that the axis could be represented as a single location that passed just distal to the distal tips of the two malleoli. A rod that marked this location shifted by less than 3 mm when the ankles were rotated through the full ROM, demonstrating that this location provided a good estimate of the true axis.

More specimens have since been examined (Inman, 1976) and these results confirmed the previous conclusion that the ankle axis of rotation was single. A single axis of rotation was found in 80% of the specimens studied and the remaining 20% had axes that shifted by a relatively insignificant amount (Inman, 1976). The ankle axis of rotation is therefore considered, at least functionally, to be single. It is obliquely oriented to the long axis of the tibia and directed laterally and posteriorly when projected on a transverse plane (Inman, 1976).

## Functional Range of Motion

The ROM required for most daily activities is considerably less than the maximum values reported above. For example, locomotion at a comfortable cadence requires no more than 0.5 rad ROM (e.g. Murray et al.,1964; Winter et al., 1974; Stauffer et al., 1977). Ankle ROM decreased as the speed of walking increased (Stauffer et al., 1977) but is considerably larger during running and jogging (Cailliet, 1983). Stair climbing requires about 0.5 rad ROM. In contrast, certain sports and recreational activities require an extremely large ROM. For example, the 'en pointe' position in classical ballet requires that the ankle be plantarflexed to -1.5 rad; this combined with dorsiflexion means a total ROM of more than 1.7 rad (Teitz, 1982).

## Conclusions

Several issues concerning the experimental paradigm are resolved in view of the evidence presented in this section. First, due to the inability to predict the subjects' expected ROM, a standard procedure must be used for the identification of maximum dorsiflexion and plantarflexion. Second, despite the relatively small ROM required for many functional activites, the ROM required for elite function or in the case of pathology is often much larger. Moreover, the ROM used in some movements, such as the maintenance of quiet stance, is small but located near the extreme of the range. The effects of this parameter over the entire range will therefore be studi~~e~~d. Third, in view of the likelihood of large joint excursions, up to ten test positions are required to adequately characterize any position-dependent effects. Fourth, because there appear to be relatively small position-dependent changes in the joint axis of rotation, the latter is assumed to be fixed and located in accordance with Inman's (1976) bony landmarks. This information is used in the fabrication of the foot fixation cast and in the placement of the cast in the ankle actuator.

## MECHANICAL CORRELATES OF HUMAN ANKLE POSITION

In this section the mechanical correlates of changes in mean ankle position are reviewed. This information will aid in the interpretation of the results presented in Chapters 4, 5 and 6.

### Muscle Length

There have been few direct studies of the relation between changes in joint angle and changes in the length of the associated muscles. In very early work, Fick (1892) attached strings between cadaver muscle origins and the cut end of tendons. As the ankle was rotated, a weight suspended from the proximal end of the string moved along a graduated scale, indicating the magnitude of length change that would have occurred in the muscle had it been in situ. Ambagtsheer (1978) replicated this work with similar results for TA and soleus. When the ankle was dorsiflexed by about 0.5 rad over mid-range, TA shortened by about 32 to 34 mm whereas soleus lengthened by 37 to 44 mm. A similar technique was used to estimate changes in gastrocnemius length (Grieve et al., 1978). They found a 12% change in length when the joint was rotated by about 1 rad.

The magnitude of changes in muscle and tendon length in intact subjects has also been studied (Halar et al., 1978). Surgical wire with a radiopaque marker was hooked into the gastrocnemius muscle-tendon junction. Muscle and tendon lengths were then measured from x-rays which had been taken at different angles. Changes in tendon length over the full ankle ROM were less than 5 mm whereas the muscle lengthened by 42 mm, 24 mm from plantarflexion to neutral and 18 mm from neutral to extreme dorsiflexion.

There have been several attempts to calculate theoretically the correspondence between joint angle and soleus muscle length at the ankle in animals (Rack & Westbury, 1969; Houk et al., 1971; Goslow et al., 1973) and in man (Sale et al., 1982). In these calculations the foot and lower leg have been modelled as two arms of a lever. Assumptions related to the muscle origin and insertion and the location of the axis of rotation have been made, the moment arm has been calculated, and the relation between incremental changes in joint angle and muscle length has been determined from a simple geometric equation. In the cat, this relation is linear for angles within the physiological ROM (Houk et al., 1971). Comparable data were not presented in the human study (Sale et al., 1982).

The significance of the above results is questionable in the light of two issues, the required assumptions and the correspondence between muscle length and the length/tension relation. First, the experimental and theoretical approaches are relatively crude in that they involve assumptions that cannot be verified. For example, the simplification of soleus origin and insertion to discrete locations has not been experimentally verified. Second, knowledge of whole muscle length at different angles does not provide an estimate of sarcomere length. Since it is the relation between the actin and myosin fibril which is the true indicator of the muscle's length/tension relation (Gordon et al., 1966), it is the correspondence between joint angle and cross-bridge overlap, rather than total muscle length, that is needed.

## Joint Torque

With the ankle located at the extreme of its plantarflexor range the passive dorsiflexor torque ranged from about 5 to 10 Nm (Hogins, 1969; Gottlieb & Agarwal, 1978; Tardieu et al., 1982). As the ankle was dorsiflexed, the passive torque decreased in a curvilinear fashion and became very small near the middle of the ROM. It then became a progressively larger plantarflexor torque as the ankle was rotated to maximum dorsiflexion. The passive torque ~~generated~~ at maximum dorsiflexion was about twice as large as the torque generated at maximum plantarflexion (Tardieu et al., 1982)

Gottlieb and Agarwal (1978) reported 1.5 to 2.5 fold increases in TS twitch torque when it was evoked in 0.26 rad dorsiflexion as compared to 0.26 rad plantarflexion. Similar increases in twitch torque over the same ROM have been reported (Sale et al., 1982). In this latter study, the magnitude of twitch torque increased still further when the ankle was rotated from about 0.5 rad plantarflexion resulting in a nearly 30 fold increase.. The magnitude of dorsiflexor torque evoked by a single TA twitch also changes with joint angle, decreasing from 4 to 1 Nm when the ankle was dorsiflexed from 0.5 rad plantarflexion to 0.35 rad dorsiflexion (Marsh et al., 1981).

Active isometric plantarflexor torque, generated at levels varying from 12.5 to 100% of the subject's maximum voluntary contraction (MVC) increased monotonically as the ankle was rotated from 0.5 rad plantarflexion to about 0.17 rad dorsiflexion. It then plateaued as the ankle was rotated to extreme dorsiflexion (Herman & Bragin, 1967). When the ankle was in mid-position, the torque developed during an MVC was 50% greater when the knee was extended than when it was flexed (Sale et al., 1982). However, regardless of knee position, TS MVC was greatest when the ankle was near extreme dorsiflexion; the mean maximum torque generated by the 20 male subjects in this study increased from just under 40 Nm at about 0.5 rad plantarflexion to greater than 175 Nm at 0.25 rad dorsiflexion (Sale et al., 1982).

The demonstration of the position-dependent changes in the magnitude of the torque generated in response to single or tetanic stimuli or by voluntary contractions has been used to support the hypothesis that significant changes in muscle length result when the ankle joint angle is altered (Marsh et al., 1981; Sale et al., 1982). However, a number of alternate mechanisms could also account for this result. As indicated above, the tissues responsible for the generation of passive torque have not been identified and may be due to tendon, ligament, joint capsule tissue in addition to the passive component of muscle. Moreover, position-dependent changes in muscle moment arms and reflex activity could account for position sensitive alterations in actively-generated torque. Therefore, this issue must also remain unresolved.

## Dynamic Stiffness

The position dependence of ankle joint dynamic stiffness has been investigated by Gottlieb & Agarwal (1978) over a small range of mean angles when the ankle muscles were relaxed and for one low contraction level in TA and in TS. They demonstrated an increase in both the viscous and elastic parameters when the relaxed ankle was rotated from about 0.2 rad dorsiflexion to about 0.2 rad plantarflexion. The stiffness/ankle angle relation was modelled as linear with a slope that was independent of the level of tonic muscle activity. The inertial parameter did not vary with joint angle.

These results are of limited use, however, for two reasons, both related to the study's scope. First, only a small percentage of ankle angles was studied. While this ROM was reasonable for investigating the effect of mean dorsiflexor positions, it was inadequate for the examination of plantarflexor angles. Second, the level of tonically-maintained muscle activity was extremely low. Therefore, any conclusions regarding either the passive or active position dependence of the joint mechanics are premature.

## Conclusions

The only available kinematic variable is the joint position. It is evident that there is simply too little information to make any definitive conclusions about what is happening to the soft tissues when the ankle is rotated. Thus we cannot know with any certainty what are the changes in the whole muscle length and certainly not what they would be at the sarcomere level. The only available kinetic variable is the net joint torque. This makes it impossible to know the forces generated by the individual hard and soft tissues.

## SENSORY CORRELATES OF HUMAN ANKLE POSITION

When the ankle joint is rotated to and then maintained in a new position, changes will occur in the associated hard and soft tissues: the muscles and ligaments of one compartment will shorten whereas their antagonists will lengthen, the skin will be distorted, and the tissues lining the joint capsule will be either stretched or compressed. In this section the results of experiments designed to examine the afferent responses to such changes in mean ankle position in man are presented. Regrettably, the techniques more commonly used to investigate such phenomena in animal studies, for example single unit recordings from spinal cord, dorsal root ganglia and peripheral nerves, are evidently not amenable to investigations in a human subject. Indeed, investigations in man have relied on only two techniques, H-reflex and microneurography. The evidence provided by each of these techniques and an assessment of their limitations are presented below.

### H-reflex Studies

Position-dependent changes in the H-reflex have been extensively examined. Stimulation of the tibial nerve resulted in responses that were markedly reduced in amplitude during or following passive dorsiflexion of the relaxed ankle (Paillard, 1960; Mark et al., 1968; Robinson et al., 1982). These findings have been corroborated by another, related paradigm in which the threshold for the M and H responses were measured as functions of ankle angle (Davies & Lader, 1983). The ratio of the threshold of H to the threshold of M increased by a small, but statistically significant amount as the ankle was progressively dorsiflexed. In contrast, when the passively-maintained ankle position was coupled with tonically-maintained contraction of the ankle plantarflexors, the stretch-related inhibition of the H-reflex was eliminated and the response was facilitated (Mark et al., 1968; Delwaide & Hugon, 1969).

Identification of which afferents mediate this response has been based on a number of indirect lines of investigation. First, decreases in the H-reflex amplitude were observed in the absence of ankle movement when soleus was stretched by pressure applied to the Achilles tendon (Mark et al., 1968; Burke et al., 1971; Delwaide, 1973; Robinson et al., 1982). It would appear, therefore, that joint receptors were not responsible for the inhibition of the H-reflex. The role of the skin receptors also appeared to be negligible; the application of local cutaneous anesthesia did not alter H-reflex response to position (Robinson et al., 1982) and pressure applied to the skin adjacent to the tendon and on the heel had no effect on the magnitude of the H-reflex when ankle position was not altered (Mark et al., 1972; Robinson et al., 1982).

The position-dependent depression of the H-reflex may have been mediated by tendon organ receptors since ankle dorsiflexion results in a large passive plantarflexor torque (Gottlieb & Agarwal, 1978; Tardieu et al., 1981). Although tendon organs are considerably more sensitive to actively-generated tension, tendon organ inhibition of the α-motoneuron pool via disynaptic or polysynaptic pathways remains a possibility (Eccles et al., 1957; Alnaes, 1967; Stuart et al., 1970; Houk et al., 1971; Burke et al., 1971; Robinson et al., 1982).

Finally, spindle afferents represent another means by which the position-dependent response could be mediated. Diffuse infiltration of TS by procaine anesthesia resulted in a removal of the position-dependent depression of the H-reflex (Burke et al., 1971). In contrast, ischemic nerve block did not alter the original response (Burke et al., 1971). Since anesthesia by the former technique acts primarily on the smaller diameter fibers (Matthews & Rushworth, 1957a; 1957b; 1958) whereas anesthesia by the latter technique acts primarily on the larger diameter fibers (Magladery et al., 1950), it may be that the spindle secondary inhibitory afferent connections (Eccles & Lundberg, 1959) are responsible for the observed H-reflex depression (Burke et al., 1971). Such a possibility is open to argument, however, in the light of findings which indicate spindle secondary excitatory connections (Kirkwood & Sears, 1974). Moreover, other possible mechanisms, including spindle primary pre-synaptic inhibitory connections (Delvaide, 1973; Burke & Ashby, 1972; Robinson et al., 1982) and occlusion of the spindle primary excitatory drive by its static function (Burke et al., 1971), have not yet been adequately explored.

## Microneurography

Microneurography is a technique that was first developed by Vallbo & Hagbarth (1967) to record from single peripheral human nerve fibers. While this technique has been used to examine afferent responses to, for example, changes in load, small-amplitude perturbations of joint position, and vibration (Burke, 1981), their response to tonically-maintained changes in joint position has been investigated in only a few studies (Vallbo, 1974a; Vallbo et al., 1981; Hulliger et al., 1982).

Spindle afferents located in the finger extensor muscles were monitored during and following passive rotation of the metacarpo-phalangeal joint (Vallbo, 1974a). The discharge rate of spindle afferents in muscles near their resting length was very low with less than 10% of the spindle primaries discharging. However, the rate of steady state discharge in both spindle primaries and secondaries increased linearly as the joint was maintained at angles associated with longer muscle lengths (Vallbo, 1974a). This study was restricted to only the functional ROM for the metacarpo-phalangeal joint; angles near either extreme of the ROM were not tested.

Investigations of actively-maintained joint position indicated an absence of spindle afferent response for tasks involving voluntary position holding at different joint positions (Vallbo et al., 1981; Hulliger et al., 1982). Again spindle receptors in the long finger extensors were monitored during changes in metacarpo-phalangeal joint angle. These authors concluded that, in contrast to the evident sensitivity of the spindle receptors to passive muscle stretch (Vallbo, 1974a; Hulliger et al., 1982), neither spindle primary nor secondary endings provided an active position-dependent response (Hulliger et al., 1982), at least for the relatively small amplitude and small load movements studied to date. It was suggested the differences in the spindle responses to passive and active position maintenance may be related to activity-related differences in the extent of fusimotor drive (Vallbo, 1974b; Hulliger et al, 1982).

There have been comparatively few microneurographic recordings from tendon organ afferents, a phenomenon which reflects the procedure followed when searching for an appropriate recording site (Prochazka & Hulliger, 1983) rather than being indicative of a paucity of these afferents which are of a comparable quantity to the number of spindle primary and secondary afferents (Houk et al., 1979). Thus, to date, no conclusions regarding the role of tendon organ receptors can be drawn from microneurography.

## Conclusions

The results of H-reflex studies should be viewed with caution. In this technique, a highly non-physiological stimulus is employed to test for sub-threshold changes in motoneuron excitability (Magladery, 1951; Hugon, 1973). The position-dependent response to electrical stimuli may differ considerably from more functional stimuli in which the muscle is overtly stretched. This means that the relation between the H-reflex and the stretch reflex is uncertain since these two responses differ in the afferent fibers activated by the stimuli, in the pattern of activity in the activated afferent fibers, in the dispersion of the afferent volley, and in the motoneurons participating in the reflex contraction (Burke, 1983b). In the light of such differences, the results of H-reflex studies cannot be used to further our understanding of the position-dependent behaviour of the stretch reflex.

In contrast, microneurography represents a significant breakthrough in the study of the afferents mediating movements in intact man although there remain three difficulties with its use. First, the classification of afferents, and in particular distinguishing between spindle primary, spindle secondary and Golgi tendon organ receptors can be ambiguous (Prochazka & Hulliger, 1983). Second, there are problems associated with the stability of the recordings with the result that the amplitude, speed and force of the contraction are restricted to the lower end of the physiological spectrum ((Hulliger et al., 1981; Hulliger & Vallbo, 1981; Prochazka & Hulliger, 1983). Third, this technique cannot be used with equal facility on all nerves. Thus, for example, the position-dependent afferent responses of receptors in TA have been examined whereas those of TS have not. Despite these limitations, microneurography remains a very promising technique for the investigation of reflex function in intact man.

# CHAPTER 3: METHODS

## INTRODUCTION

This chapter describes the general experimental methods common to all studies presented in this thesis. Specific details pertaining to individual experiments are provided as required in later chapters.

This chapter has been organised into five sections as indicated in Fig. 3.1. These sections include: details related to the experimental paradigm; information concerning the computer control of the experiments and subsequent analyses; descriptions of the stimulus variables and the equipment controlled by these stimuli; an outline of the data acquisition procedures; and a review of the methods used for data analysis.

## EXPERIMENTAL PARADIGM

### Subjects

Seven healthy men and women, aged 24 to 37 years, participated as subjects in these experiments. They included the principal investigators as well as other university personnel who were paid. Subjects were designated by numbers (e.g. S1, S2) which denoted the sequence in which they were tested.

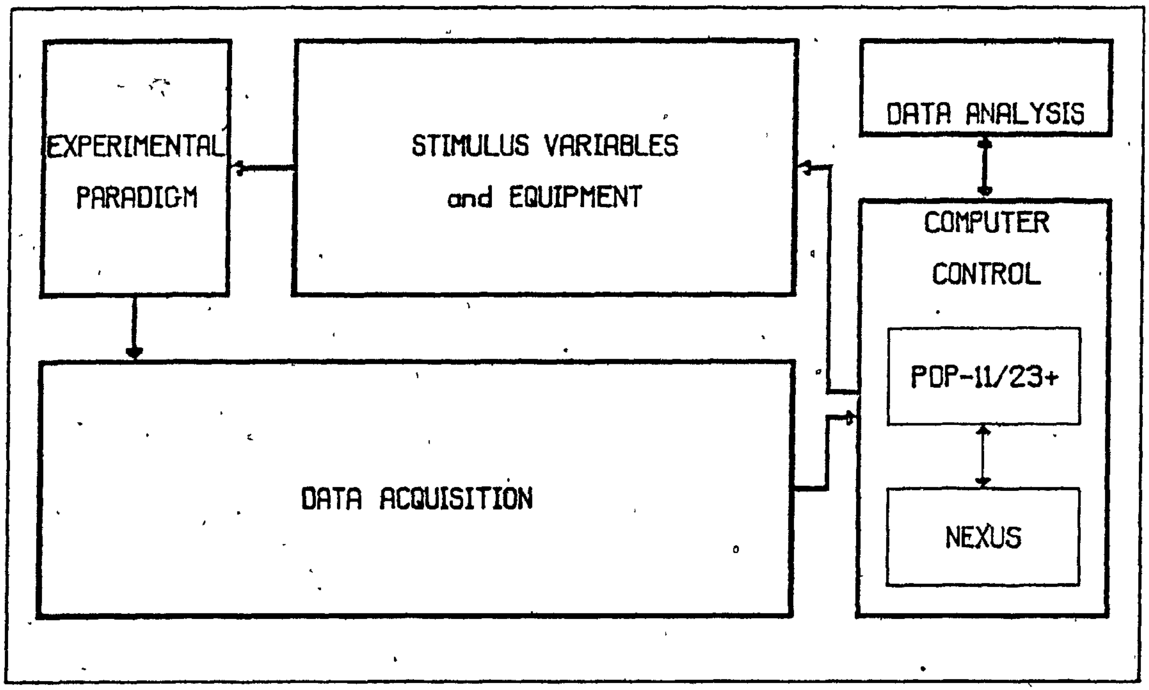


**Fig. 3.1:** Block diagram showing the five sections presented in this chapter.

## Whole Body Fixation

Subjects lay supine on the experimental table as shown in Fig. 3.2. The head was turned to the left facing an oscilloscope display of a tracking stimulus and torque feedback. The arms were extended at the sides of the body, the hips were moderately abducted, and the knees were fully extended. The right foot was supported on a pedal and the left foot was attached to the ankle actuator via a polyurethane foam cast. This posture was maintained by wide straps over the pelvis, thighs, and the right foot. The laboratory lights were dimmed during the experiments.

To ensure the subjects' comfort, the experimental table was covered with 5 mm closed cell sponge neoprene (E.F. Walter, Model 6327). This covering was non-conductive and so insulated the subject from potential current leaks. Pillows were placed beneath the head and under the back at the level of the lumbar lordosis.

## Foot Fixation

The experiments described in this thesis entailed rapid, precise changes of limb position and so required that limb movement relative to the apparatus be minimized. Traditional methods of subject fixation such as straps or clamps were judged to be unacceptable; these could cause local ischemia, pain, and skin injury or could directly perturb the muscles being investigated, hampering the study of their behaviour in response to the applied joint perturbation.

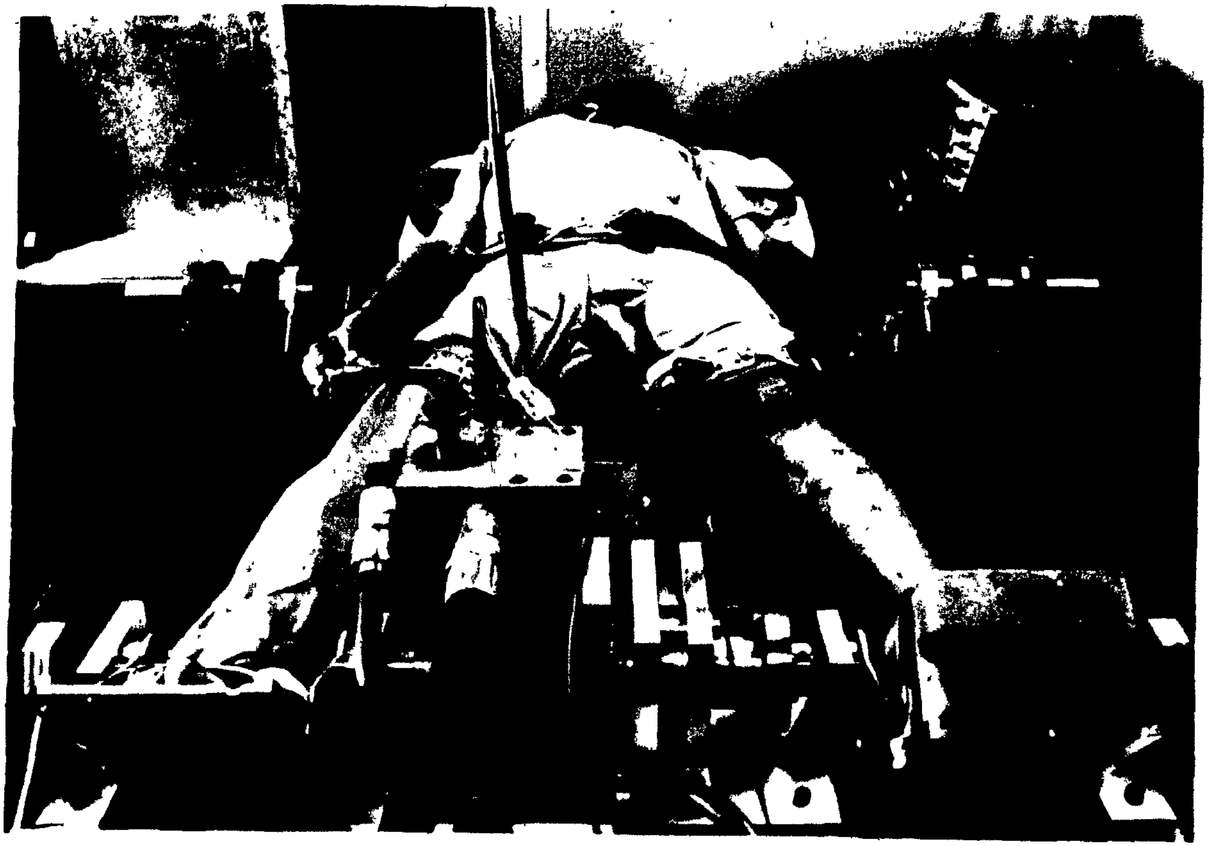


**Fig. 3.2:** Photograph of a subject positioned on the experimental table. The subject's left leg is attached to the ankle actuator via the polyurethane foam cast. His head is turned to face the oscilloscope which is displaying the tracking stimulus.

These problems were avoided in the present experiments by the use of custom-fitted, polyurethane foam, block casts which were strong, rigid, and distributed the forces over a wide skin surface area. The moments of inertia of these casts were of similar magnitude to those of the subjects' feet. They neither restricted the dynamic response of the actuator nor introduced spurious elastic and viscous forces. An overview of the method is presented here; the details have been previously reported (Weiss et al., 1984a).

The casts were made by a simple technique that resulted in accurate negatives of the subject's foot. Once cured, the polyurethane foam was non-toxic to the skin and emitted no hazardous fumes (Burrows, 1983). The fitting procedure required little cooperation from the subject, did not cause discomfort, and could be completed in less than 20 min.

Construction of a cast consisted of three steps: (1) casting a plaster negative of the foot; (2) casting a dental stone positive; and (3) casting the final polyurethane foam foot cast. A detachable aluminum mold was used during all three steps. In the first step, the subject's foot was covered with a thin layer of petroleum jelly and positioned in the mold. The casting material (six parts plaster of paris to one part sand) was combined with four parts water at 37° C. The fluid mixture was poured into the mold and set within 15 min. The sand served to weaken the plaster cast so that it could be split in two for rapid release of the subject's foot. The temperature of the plaster/sand negative was measured in order to ensure that it remained within comfortable limits; it did not exceed 35° C for at least 5 min after it was hard enough to be split.

In the second step, the two pieces of plaster negative were returned to the mold and a fluid mixture of a very hard plaster (dental stone) was poured in. After setting (approximately one hour), the relatively soft plaster negative was destructively removed from the hard plaster positive. The axis of rotation of the ankle was identified on the positive using anatomical landmarks described by Isman & Inman (3 mm distal and 8 mm anterior to the distal tip of the lateral malleolus and 5 mm distal to the distal tip of the medial malleolus; 1969) and a 6 mm hole was drilled through the axis. The dental stone positive was then returned to the mold where it was fixed in the correct position by a 6 mm diameter stainless steel rod driven through the ankle axis of rotation.

Finally, the isocyanate and resin components were mixed and poured into the mold. The resulting polyurethane foam expanded, enveloped the positive, filled the mold, and became very hard within two hours. The rigid polyurethane cast was then removed from the mold and cut in half longitudinally. The positive was removed and the edges and inner surfaces were lightly sanded to remove sharp edges and rough spots.

Cast comfort and fit were checked prior to experimentation; the halves of the cast were tightly secured about the subject's foot with velcro straps and bolted onto the laboratory ankle actuator. If the cast caused undue discomfort or allowed unacceptable movement of the foot it was discarded. Fig. 3.3 shows a typical finished cast and illustrates the good fit obtained.

The typical overall density of the casts resulting from this procedure was 280 kg/m³ with masses ranging from 0.8 to 0.9 kg. The moment of inertia of the laboratory ankle actuator was 0.015 kgm² and that of the combined actuator and foot cast was approximately 0.025 kgm². The polyurethane foot casts thus had moments of inertia in the range of 0.01 kgm², values which are comparable to the moments of inertia of the subjects' feet (Dempster et al., 1955).

## Ankle Joint Range of Motion

The ROM was measured while the foot was attached to the actuator. The NP was determined by locating the ankle angle at which no passive joint torque was generated. The ankle was then rotated from the NP toward maximum dorsiflexion and toward maximum plantarflexion. Displacement in each direction continued until the subject could tolerate no further movement or until motion of the ipsilateral knee or lower leg was discerned. Standardized methods for the determination of the ROM are not available (Roaas & Anderson, 1982), hence this subjective, but repeatable procedure, was used.

The foot was completely encased in the cast, posteriorly as high as the talar head and anteriorly as far as the navicular and cuboid bones (cf. Fig. 3.3). In order to minimize loss of ROM and yet maintain adequate limb fixation, cast height posterior to the malleoli was decreased by 15 mm. (Fig. 3.3 is a photograph of the original cast and does not show the reduced height.) In spite of this

**Fig. 3.3:** Photograph of the halves of a completed polyurethane foam cast. The subject's foot is in the right half of the cast. The empty left side of the cast illustrates that the final cast is smooth, uniform negative that replicates the contours of the subject's foot.

modification, goniometer measurements on the freely-moving foot indicated that the cast reduced ROM by as much as 0.2 rad. A further 0.1 rad restriction on the ROM resulted from the ankle perturbation (see below). Nevertheless, comparisons of the subjects' 'fixed' and 'freely-moving' ROM indicated that at least 85% of the total ROM was tested in these experiments.

For five of the seven subjects, the ROM was divided into ten equally-spaced mean positions. Only nine positions were tested in the other two subjects since their ROM was smaller. Adjacent positions were separated by about 0.1 rad.

<u>General Protocol</u>

The subject was positioned on the table and the left foot rotated to one of the test positions. After a 5 min pause, the ankle was stochastically displaced. The pause was imposed since the passive joint torque took some time to stabilize. When the ankle was rotated to the extremes of its range, the torque decreased by as much as 5 Nm during the first 5 min; at mid-range positions this phenomenon was negligible.

During some trials subjects were requested to exert no voluntary muscle effort while the ankle was being perturbed; otherwise they were instructed to match the torque feedback signal with the displayed tracking stimulus. This procedure was then repeated for the remaining mean joint positions.

## COMPUTER CONTROL

All experimental and analytic procedures were controlled by a PDP-11/23-Plus minicomputer (Digital Equipment Corporation) using Nexus, a language for the analysis of signals and systems (Hunter & Kearney, 1984). The programs written in Nexus (modules) are appended at the end of the thesis. These include the modules used to operate the experiments (Appendix I), generate the stimulus variables (Appendix II), run the data acquisition programs (Appendix III), and analyse the data (Appendix IV). The ankle actuator servo system gains and safety limits were controlled by a LSI-11 microprocessor (Digital Equipment Corporation).

## STIMULUS VARIABLES AND EQUIPMENT

A schematic representation of the equipment used during the experiments is shown in Fig. 3.4. Details related to the stimulus variables and equipment are shown in the block labelled 'Stimulus Variables and Equipment'. The boxes within this block represent the equipment used to display and control the two stimulus variables: the signal used to drive the ankle actuator and the target tracking signal displayed on the oscilloscope.

### Ankle Actuator

The ankle joint was displaced by a rotary electro-hydraulic actuator (Rotec Model RN-32-1V) (Kearney et al., 1983). Actuator flow

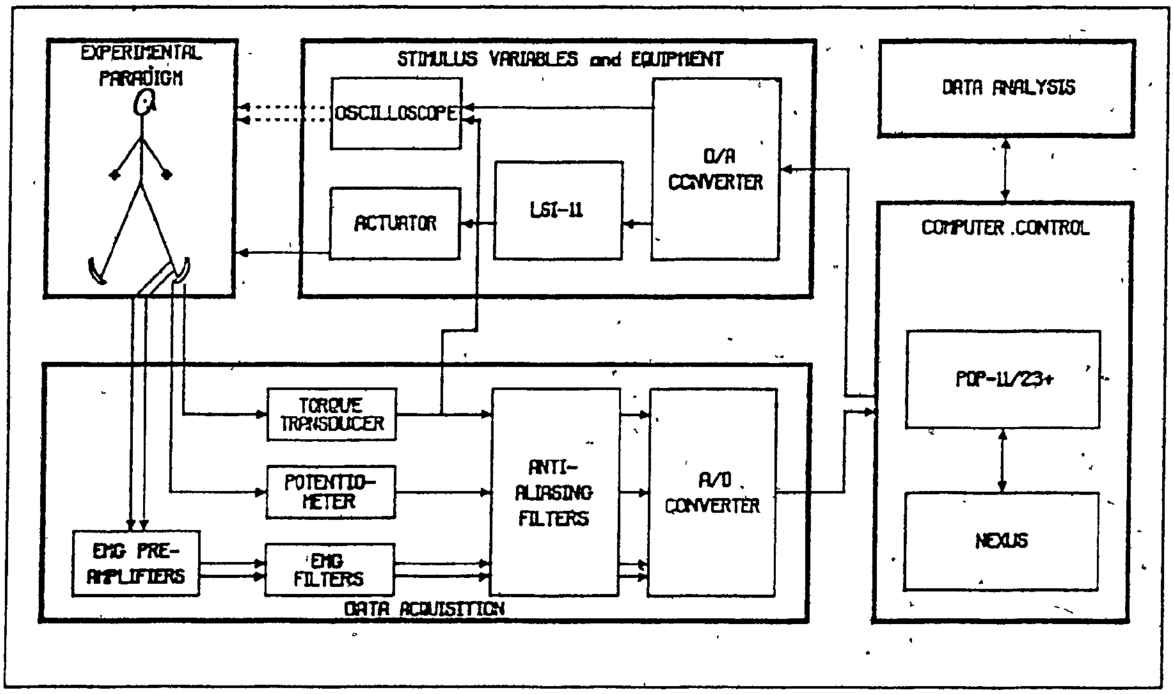


**Fig. 3.4:** A block diagram showing schematically the equipment used during the experiments. See text for details.

was controlled by a servo valve (Moog Model 73-104). The actuator, operated in position-control mode, was able to produce velocities exceeding 26 rad/s.

The dynamic response of the ankle actuator was determined using stochastic system identification techniques. The gain was flat until 25 Hz but then decreased at about 60 dB/decade. Nevertheless, the actuator could apply displacements containing power adequate for system identification at frequencies up to 50 Hz.

The safety of actuator operation was ensured by four independent mechanisms: mechanical stops, a hydraulic stop, a 'panic' button, and the LSI-11 microprocessor. The mechanical stops consisted of two aluminum sleeves bolted to the pedal support at locations corresponding to the extremes of the subject's ROM. The hydraulic stop was also adjustable and removed power from the actuator if the foot pedal was rotated beyond the subject's ROM. The subject could halt actuator operation at will by depressing the 'panic' button that acted by stopping flow to the actuator. Finally, the LSI-11 microprocessor control system limited displacements, velocities, and torques to pre-determined values.

The ankle could be rotated to within 0.001 rad of the desired angle when the leg remained strapped to the table and when the foot remained attached to the actuator. However, alteration of left limb fixation could disturb the location of the NP by as much as 0.1 rad. The leg straps and foot cast were therefore not removed until the end of each experiment.

## Actuator Input

A 1000 point pseudo-random binary sequence (PRBS) was repeatedly displayed at 400 Hz by a 12 bit D/A converter (Digital Equipment Corporation, Model AAV11-A). The duration of each sequence was thus 2.5 s.

The PRBS was applied as input to the servo amplifier thereby driving the actuator to stochastically displace the ankle joint. The frequency content of this displacement was limited to 50 Hz by the low-pass characteristics of the actuator as described above. The gain was adjusted so that the peak-to-peak amplitude of the displacement was 0.09 rad.

Ankle joint dynamics are modified by the amplitude of the joint perturbation (Kearney & Hunter, 1982). It was, therefore, necessary to ensure that the displacement amplitude did not vary consistently with mean position. An examination of the ensemble average (Fig. 3.5, top graph) and standard deviation (SD) (Fig. 3.5, bottom graph) of the ankle displacement records sampled at ten different angles over a subject's ROM demonstrates that the perturbation applied at all mean positions was very similar; the magnitude of the ensemble SD was less than 0.015 rad. Moreover, the dynamic response of the actuator (not shown) was similar at all mean positions. It is therefore concluded that systematic variations of the perturbation amplitude with joint angle were not a problem.

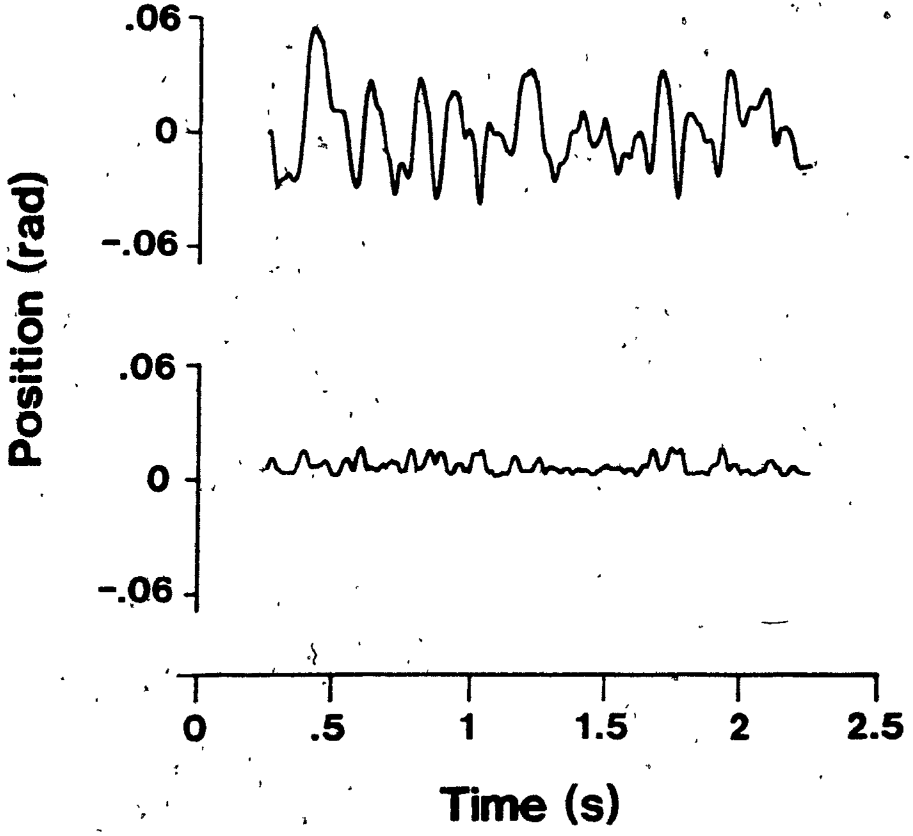


**Fig. 3.5:** The average (top graph) and standard deviation (bottom graph) of position records sampled at ten different mean joint angles throughout the ROM.

### Tracking Stimulus

A second computer-generated stimulus was used when the subject was required to maintain tonic muscle contractions. This signal, scaled to elicit the required level of either dorsiflexor or plantarflexor tonic activity, appeared as a continuous line on the oscilloscope (Tektronix, Model 2215).

## DATA ACQUISITION

The four sampled signals, shown schematically in Fig. 3.4 within the block labelled 'Data Acquisition', included ankle angular position, ankle torque, and TA and TS electromyograms (EMGs). The boxes within this block represent the equipment used to amplify, filter, and sample these signals.

### Angular Position and Torque Transducers

A plastic film rotary position transducer (Beckman helipot, Model 6263-R5K-L.50) was used to measure ankle angular position. Ankle torque was measured with a very stiff (50,000 Nm/rad) torque transducer (Lebow, Model 2110-5k). A torque feedback signal was generated by filtering the torque with a 2-pole lowpass filter having a cut-off frequency of 1 Hz. This signal was displayed to the subject on the oscilloscope.

## Electromyograms

Disposable surface electrodes (Hewlett Packard, Model 14445A), oriented in a bipolar configuration, parallel to muscle fiber direction, were used to detect EMG from TA and TS. The skin was shaved and rubbed briskly with ethanol prior to electrode attachment. TA electrodes were placed just lateral to the tibial crest about 50 mm distal to the tibial tuberosity. TS electrodes were placed on the calf mid-line just distal to the heads of the gastrocnemii. A ground electrode was placed over the patellar head. These electrode locations were chosen to obtain a good representation of the overall activity of the muscle group.

Electrodes were connected to custom built differential preamplifiers incorporating an instrumentation amplifier (Burr-Brown, Model 3620), switchable gains, external offset control, and isolation switches (Ohmic Instruments, Model IS-100). The latter devices were in series with the electrodes and acted to limit current flow to the subject to less than 100 μamp. The amplifier common mode rejection ratio was 100 dB.

The raw EMG signals were then processed by filter amplifiers that were constructed from 2-Pole high-pass Butterworth filters (Frequency Devices, Model 772BT-2) and 4-Pole low-pass Bessel filters (Datel Systems, Model FLT-LP4L5K). The raw EMGs were high-pass filtered (10 Hz), full-wave rectified, and then low-pass filtered (1000 Hz). In order to maintain the polarity conventions (dorsiflexor position and torque positive, plantarflexor position and torque negative), TA EMGs were positively rectified and TS EMGs were negatively rectified.

### Anti-aliasing Filters

All experimental signals were lowpass filtered (8-pole lowpass Bessel filters; Frequency Devices, Model 902LPF) at 50 Hz prior to being sampled at 400 Hz by a 12 bit analog-to-digital (A/D) converter (Digital Equipment Corporation, Model AXV11-C) with a dynamic range of ±5 V.

## DATA ANALYSIS

The methods used to determine the passive and active joint mechanics, statics, and reflex dynamics are outlined in this section.

### Actuator/Cast Dynamics Correction

A portion of the sampled torque signal was due to the dynamics of the ankle actuator and foot cast. It was necessary to identify and remove this torque prior to further analysis. This was accomplished by the following procedure. First, the impulse response function (IRF) between the actuator/cast position and torque was determined from data obtained in a pre-experiment calibration trial. Then each experimental position record was convolved with this IRF. Finally, the resulting estimate of the actuator/cast torque was subtracted from the sampled torque. The signal generated by this procedure will henceforth be referred to as the torque.

## Initial Data Processing

The initial analysis procedure had three phases: (1) the sampled signals were checked for stationarity; (2) the data were ensemble averaged and; (3) the static behaviour of the signals was determined. These procedures are illustrated with reference to the torque records shown in Fig. 3.6; the position and EMG data were treated similarly.

Torques recorded during repetitions of the PRBS stimulus sequence, $X_1$, $X_2$, ... $X_5$ are shown in Fig. 3.6 (top graph). The time average of each record, the interval mean, $\mu_i$, is indicated in each graph. Interval means were examined to monitor stationarity over the five (and in some cases 25) stimulus repetitions. Significant departures from stationarity occurred occasionally, particularly when subjects tracked the higher force levels. Since the ankle joint dynamics change with mean torque (Hunter & Kearney, 1982), data were discarded if the SD of the torque interval means was larger than 1.0 Nm.

The ensemble average, $X$, was then calculated (Fig 3.6, bottom graph). Averaging was not strictly necessary for determination of joint mechanics, but was required for identification of the reflex dynamics (see Chapter 6 for further details). The time average of the ensemble, the mean value, $\mu$, was used as a measure of the static values of each signal.

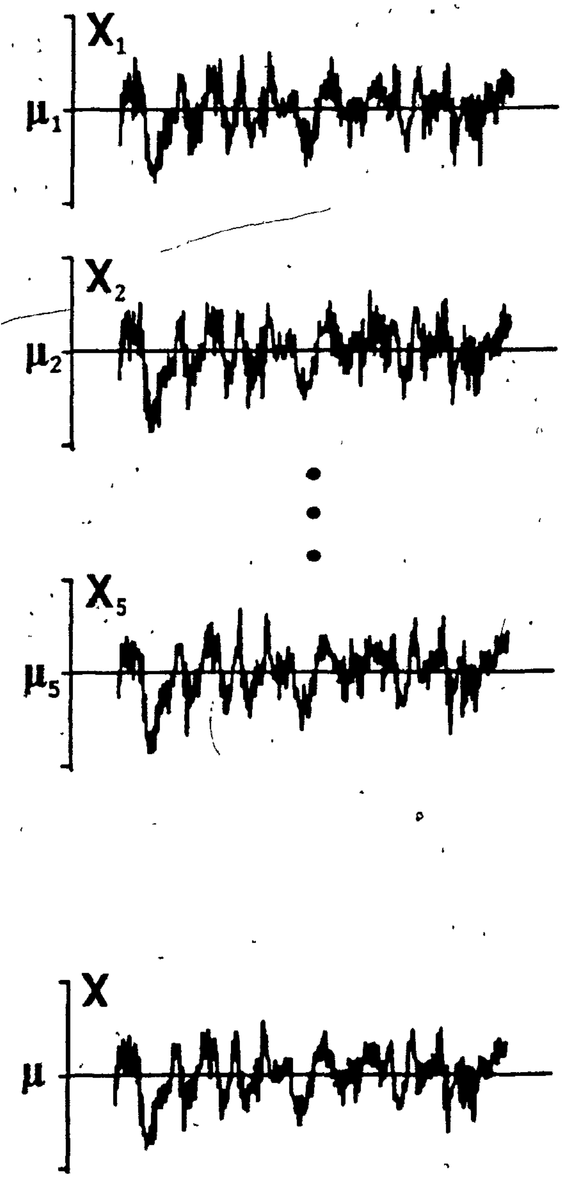


**Fig. 3.6:** Diagram illustrating the procedure used for initial data processing. Each torque record, $X_1$, $X_2$, ... $X_5$, was sampled during *one* repetition of the stimulus sequence. The time average of each record, called the interval mean, was calculated. The five records were ensemble averaged and the time average of the ensemble, called the mean ($\mu$), was calculated.

For trials in which the subject remained at rest, the mean torque was used to identify the change in passive joint torque that resulted when joint position was altered. In these cases, mean EMGs were examined in order to verify that there was no muscle activity. For trials in which the subject maintained a tonic contraction, mean torque and EMGs were used to determine the static relation between these two variables. Finally, mean antagonist EMGs were examined in order to monitor the extent of co-contraction since this is known to modify ankle joint dynamics (Hunter et al., 1983).

## Ankle Joint Mechanics

Ankle joint dynamic stiffness, i.e. the dynamic relation between angular position and torque, was first characterized non-parametrically at each mean joint position. Stiffness transfer function gain, phase, and coherence functions were obtained by calculating the auto- and cross-correlation functions and Fourier transforming them (Box & Jenkins, 1976).

The mechanical behaviour of the ankle joint has been successfully modelled by a second order, underdamped system having inertial, viscous, and elastic terms (e.g. Hunter & Kearney, 1982; Gottlieb & Agarwal, 1978). Furthermore, changes in the parameters of this model have been used to delineate the effect of varying mean torque and displacement amplitude (Hunter & Kearney, 1982; Kearney & Hunter, 1982). Non-linear minimization techniques were used to estimate the parameters of a second-order system which best fit the compliance IRF (the non-parametric representation of ankle mechanics which relates joint torque and position). The model used was:

$$TQ = I\ddot{\theta} + B\dot{\theta} + K\theta$$

where $TQ$ = Ankle Torque

$\theta$ = Ankle Angular Position

$\dot{\theta}$ = Ankle Angular Velocity

$\ddot{\theta}$ = Ankle Angular Acceleration

$I$ = Inertial Parameter

$B$ = Viscous Parameter

$K$ = Elastic Parameter.

This parametric representation of the linear dynamic relation between torque and position provided a concise description of the ankle joint system. These fits accounted for at least 90% and usually more than 95% of the variance of the original compliance functions.

Linearity of the identified system was assessed in two ways. First, stiffness coherence functions, which provided an estimate of how much variance was accounted for at each frequency by the linear dynamic relation between position and torque, were calculated. Values close to one signified that the relation was linear. Values considerably less than one signified that the relation was not linear, that system output was noisy, or that there were other inputs to the system (Bendat & Piersol, 1980). Linearity was also monitored by determining the variance accounted for (VAF) by the compliance IRFs. This value, given as a percent, provided an indication of system linearity at all frequencies.

## Reflex Dynamics

The behaviour of TA and TS reflexes as functions of mean joint position was examined by calculating the linear dynamic relation between ankle angular velocity and rectified, smoothed EMGs. The reflex impulse function between positively rectified ankle angular velocity and TS EMG was used to identfy TS reflexes (Kearney & Hunter, 1983) and the RIRF between ankle angular velocity and TA EMG was used to identify TA reflexes (Kearney & Hunter, 1984). These reflex impulse response functions were calculated with an algorithm that was based on the solution of a matrix equation involving the input auto-correlation function and the input/output cross-correlation function (Hunter & Kearney, 1983b).

# CHAPTER 4: PASSIVE JOINT MECHANICS

## SUMMARY

System identification techniques were used to examine the position dependence of the passive ankle joint mechanics. The relaxed ankle was stochastically perturbed about different angles in the range of motion. The linear dynamic relation between ankle position and torque was identified and modelled as a second-order, underdamped system, having inertial, viscous, and elastic parameters. Mean joint torque changed as the ankle was rotated through the ROM; it was small at mid-range and became much larger toward either extreme. I remained constant but B and K changed as functions of ankle angle. At the extremes of the ROM, K was much larger than previously thought and the relation between stiffness and the passive torque generated when the ankle was placed at different mean positions was linear. These results show that large variations in joint mechanics (1) were possible in the absence of voluntary muscle contraction and (2) appeared to be related to the position-dependent changes in the passive torque.

## INTRODUCTION

Rotation of the relaxed ankle through its ROM results in a characteristic change in the joint torque. Whereas the torque remains small and relatively constant over a large span of middle range angles, it becomes much larger as the ankle is rotated toward either maximum dorsiflexion or maximum plantarflexion (e.g. Hogins, 1969). The position dependence of the passive torque is significant because it represents a means by which the joint torque can be changed with no alteration of the muscle's active state.

This issue has not been adequately explored. In particular, the study of position-dependent changes in the dynamic passive joint stiffness has been restricted to an extremely limited portion of the ROM. The specific objective of the work presented in this chapter was to fully characterize the effect of changes in mean ankle position on the passive joint mechanics in order to determine the magnitude of these effects and to ascertain their potential functional role. These results will then be compared and contrasted to the findings of the next chapter to assess the relative roles of the active and passive processes.

## METHODS

General methods were as described in Chapter 2. For this study, subjects were requested to remain relaxed while the ankle joint was displaced. TA and TS EMGs were monitored to verify that there was no muscular activity. The 2.5 s stochastic sequence was repeated 25 times at each mean joint position. The duration of each experiment (including tests at each mean joint position and the 5 min rest period between successive tests) was 60 min.

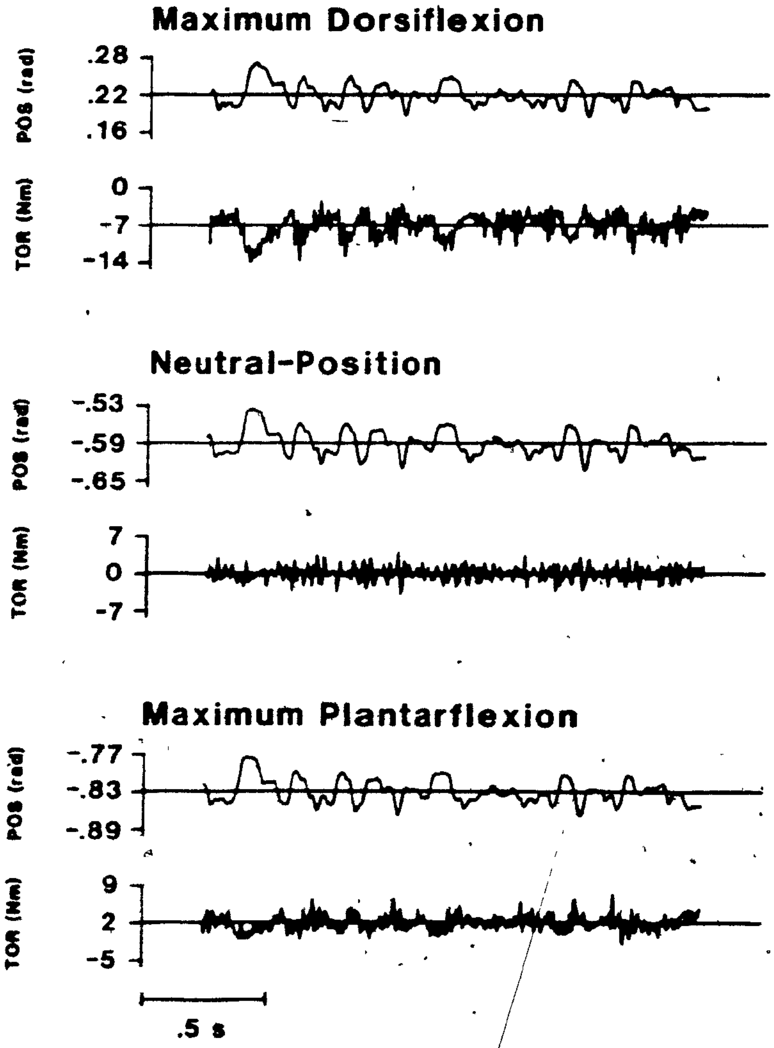
## RESULTS

Fig. 4.1 shows ankle angular position and torque records obtained when the ankle joint was near extreme dorsiflexion (top graph), at the NP (middle graph), and close to maximum plantarflexion (bottom graph). It is evident that the mean torque changed with ankle angle; it was near zero at mid-range and increased as the ankle was dorsiflexed or plantarflexed. Note that the peak-to-peak amplitude of the torque also increased.

### Relation between Mean Torque and Mean Position

The relation between mean joint position and mean torque for all six subjects is shown in Fig. 4.2. All six plots are similar; mean torques were small and relatively constant over a large region of mid-range positions but increased rapidly as the ankle was rotated toward either extreme. The NP occurred at approximately mid-range for each subject.

Mean position and mean torque data obtained when the ankle joint was near extreme plantarflexion, at mid-range and close to maximum dorsiflexion are presented in Table 4.1. Intersubject variability for total ankle ROM (mean ± one SD, .99 ± .12 rad) was consistent with reports in the literature (e.g. Boone & Azen, 1979). Similar variability was also evident in the extent of plantarflexion (-.842 ± .11 rad) and dorsiflexion (.149 ± .072 rad) and in the magnitude of the passive torque generated at the extremes of plantarflexion ([illegible].7 ± 2.9 Nm) and dorsiflexion (-9.3 ± 2.7 Nm) (Boone



Maximum Dorsiflexion

Neutral-Position

Maximum Plantarflexion

Fig. 4.1: Ensemble averages of ankle angular position and torque at maximum dorsiflexion (top graph), the neutral-position (middle graph), and maximum plantarflexion (bottom graph). The time marker refers to all plots. (Subject S1)

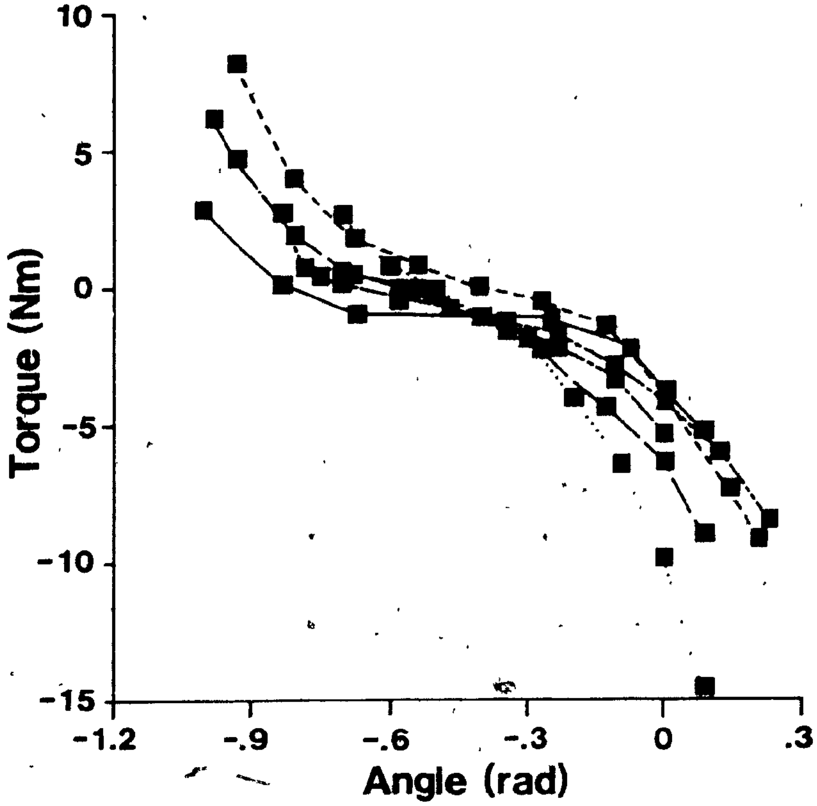


**Fig. 4.2:** The relation between mean position and mean torque. Data from each subject is denoted by a different line type.

& Azen, 1979; Hogins, 1969). There was no apparent relation between the extent of the ROM and the magnitude of the torque generated. All subjects generated more torque at the extreme of dorsiflexion than at maximum plantarflexion.

## Non-Parametric Analysis of Joint Dynamics

It is evident from Fig. 4.1 that the joint dynamic stiffness was also dependent on ankle mean position. Thus the torque records were more deeply modulated by the perturbation when the ankle joint was near the extremes of its range (top and bottom torque traces, Fig. 4.1) than at the NP (middle torque trace, Fig. 4.1). These changes are reflected in the stiffness transfer function gain curves shown in Fig. 4.3. The stiffness gain increased as the ankle was dorsiflexed (top graph) and plantarflexed (bottom graph) from the NP.

A set of compliance impulse response functions, corresponding to the stiffness transfer function gain curves plotted in Fig. 4.3 (top graph), are shown in Fig. 4.4. In this alternate, non-parametric representation of ankle joint dynamics, the behaviour of the system natural frequency and damping as a function of mean position is apparent. The natural frequency, related to the location of the first zero crossing (indicated in Fig. 4.4 by the dashed line), increased as the ankle was dorsiflexed from -.54 to .20 rad. In contrast, the system damping, related to the extent of the oscillation, remained relatively constant.

The VAF by the compliance impulse response functions was always

| VARIABLE | SUBJECTS<br>S1 | S2 | S3 | S4 | S5 | S6 | Mean | SD |
|---|---|---|---|---|---|---|---|---|
| Range of Motion (rad) | 1.04 | .79 | 1.14 | 1.07 | .93 | .98 | .99 | .12 |
| Change in Torque (Nm) | 9.1 | 17.3 | 13.8 | 15.2 | 10.4 | 8.9 | 12.5 | 3.5 |
| **EXTREME PLANTARFLEXION** | | | | | | | | |
| Position (rad) | -.828 | -.705 | -.935 | -.988 | -.840 | -.758 | -.842 | .011 |
| Torque (Nm) | 2.1 | 2.7 | 4.2 | 6.2 | 2.7 | .4 | 3.7 | 2.9 |
| IRF-VAF (%) | 95.5 | 90.6 | 95.9 | 96.0 | 94.6 | 90.0 | 93.8 | 2.7 |
| Natural Frequency (Hz) | 18.2 | 11.6 | 15.3 | 19.9 | 14.7 | 8.7 | 14.7 | 4.1 |
| Damping Parameter | .30 | .27 | .39 | .59 | .33 | .23 | .35 | .13 |
| Model-VAF (%) | 98.2 | 96.3 | 95.4 | 96.8 | 96.4 | 97.8 | 96.8 | 0.9 |
| I ($kgm^2$) | .007 | .011 | .008 | .007 | .008 | .007 | .008 | .002 |
| B (Nms/rad) | .44 | .42 | .57 | 1.07 | .47 | .18 | .52 | .30 |
| K (Nm/rad) | 86 | 57 | 71 | 112 | 66 | 22 | 78 | 38.3 |
| **NEUTRAL-POSITION** | | | | | | | | |
| Position (rad) | -.591 | -.503 | -.473 | -.544 | -.588 | -.590 | -.537 | .070 |
| Torque (Nm) | -.1 | .0 | -.3 | -.7 | .0 | -.4 | -.2 | .3 |
| IRF-VAF (%) | 98.0 | 95.7 | 92.9 | 96.1 | 92.2 | 93.2 | 94.7 | 2.3 |
| Natural Frequency (Hz) | 8.9 | 7.4 | 8.0 | 8.0 | 7.2 | 6.1 | 7.6 | .94 |
| Damping Parameter | .27 | .32 | .38 | .27 | .24 | .20 | .28 | .063 |
| Model-VAF (%) | 97.5 | 96.2 | 95.4 | 98.0 | 97.2 | 97.4 | 97.0 | 0.9 |
| I ($kgm^2$) | .005 | .010 | .007 | .007 | .007 | .007 | .007 | .002 |
| B (Nms/rad) | .16 | .31 | .27 | .20 | .16 | .12 | .20 | .07 |
| K (Nm/rad) | 16 | 22 | 18 | 18 | 15 | 11 | 16 | 3.7 |
| **EXTREME DORSIFLEXION** | | | | | | | | |
| Position (rad) | .215 | .082 | .202 | .084 | .085 | .225 | .149 | .072 |
| Torque (Nm) | -7.0 | -14.6 | -9.6 | -9.0 | -7.7 | -8.5 | -9.3 | 2.7 |
| IRF-VAF (%) | 96.6 | 96.9 | 97.7 | 96.9 | 98.5 | 97.2 | 97.3 | .7 |
| Natural Frequency (Hz) | 19.6 | 20.8 | 19.9 | 17.2 | 22.4 | 19.9 | 20.0 | 1.7 |
| Damping Parameter | .48 | .17 | .28 | .33 | .41 | .26 | .32 | .11 |
| Model-VAF (%) | 96.5 | 95.4 | 97.7 | 97.1 | 95.9 | 98.1 | 96.8 | 1.0 |
| I ($kgm^2$) | .006 | .014 | .009 | .009 | .008 | .008 | .009 | .003 |
| B (Nms/rad) | .76 | .63 | .65 | .63 | .88 | .53 | .68 | .12 |
| K (Nm/rad) | 97 | 236 | 144 | 102 | 152 | 126 | 145 | 50.9 |

**Table 4.1**: Range of motion and the change in passive torque throughout the range of motion for each subject. Static and dynamic data at three ankle angles (maximum plantarflexion, the NP and maximum dorsiflexion) are also presented. The static parameters include mean position and mean passive torque. The dynamic parameters include the natural frequency, the damping parameter, I, B and K. The VAF by the compliance impulse response function (IRF-VAF) and by the fit of a second-order model to these functions (Model-VAF) are also listed. Group mean and standard deviations are provided.

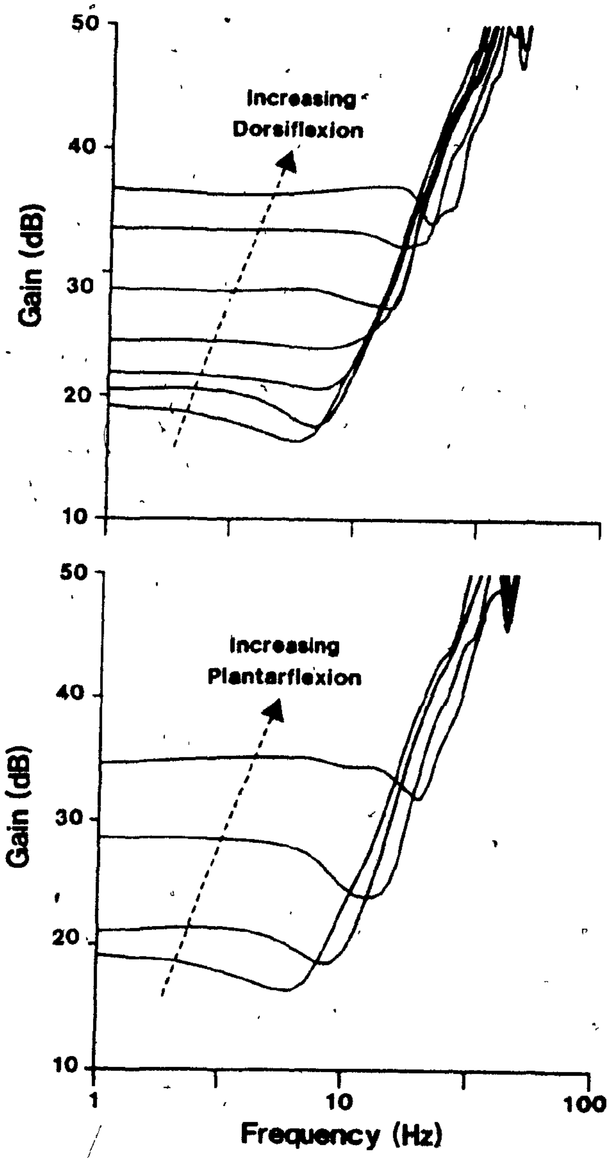


**Fig. 4.3:** Stiffness transfer function gain curves calculated when mean ankle position was progressively dorsiflexed (top graph) and plantarflexed (bottom graph) from the neutral-position (bottom trace in both graphs). (Subject S4)

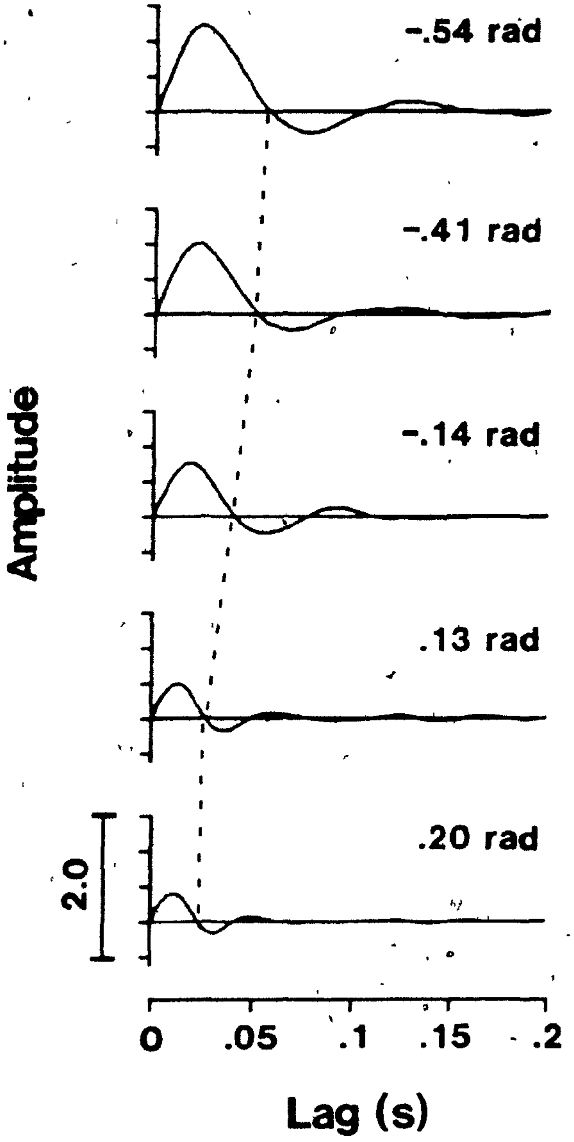


**Fig. 4.4:** Ankle joint compliance curves calculated when the ankle was rotated from the neutral-position (-.54 rad) to maximum dorsiflexion (.20 rad). The dashed line indicates each functions first zero crossing. The amplitude marker and time scale refer to all plots. (Subject S4)

greater than 80% and usually greater than 90%. The VAF was typically about 5% greater when calculated at the more dorsiflexed positions than at the more plantarflexed positions.

## Parametric Analysis of Joint Dynamics

Estimates of the inertial, viscous, and elastic parameters were determined from the compliance impulse response functions. This parametric representation of the dynamic relation between torque and position provided a concise description of the ankle joint system which consistently accounted for more than 90% of the variance of the original compliance function.

I, B, and K are plotted as functions of mean joint position for one subject in Fig. 4.5. The inertial parameter (Fig. 4.5, top trace) was essentially constant throughout the ROM. The viscous parameter (Fig. 4.5, middle trace) was smallest at mid-range and showed a three-fold increase as the ankle was plantarflexed and a five-fold increase when it was dorsiflexed. The behaviour of I and B was similar for all subjects.

The elastic parameter (Fig. 4.5, bottom trace) was small throughout mid-range and increased by a factor of eight as the ankle was either plantarflexed or dorsiflexed. As shown in Fig. 4.6 and Table 4.1, K behaved similarly for the other subjects though the increase at the two extremes of the ROM was often considerably less symmetrical.

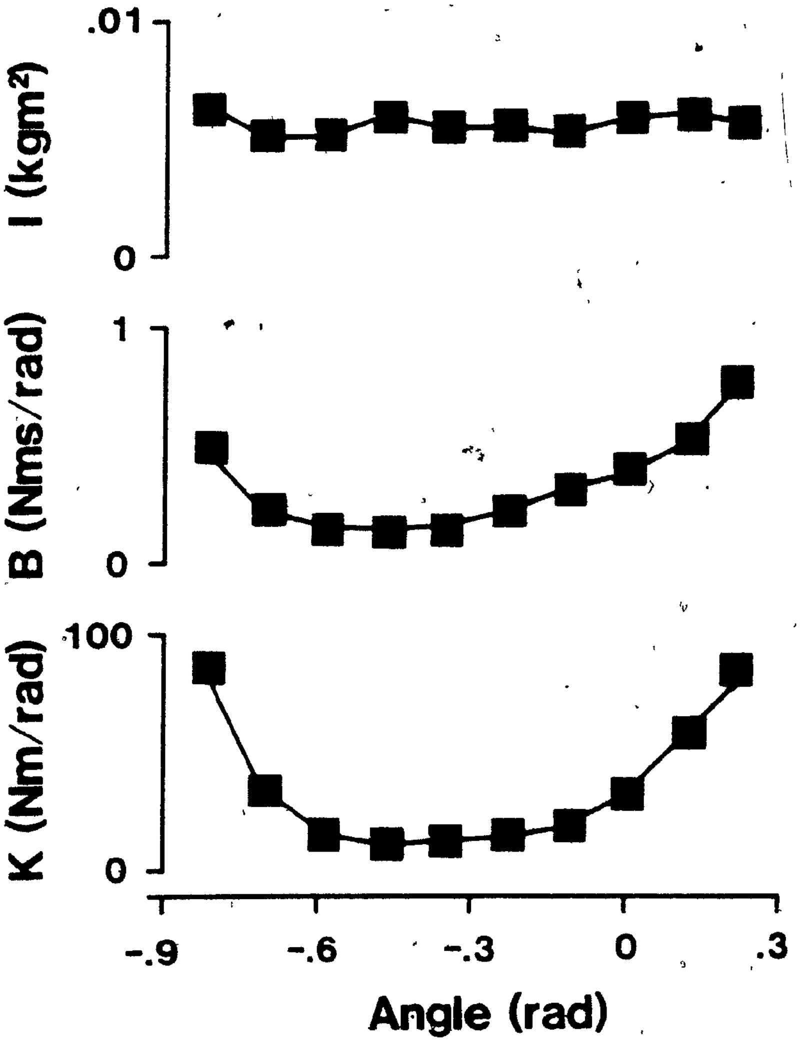


**Fig. 4.5:** The inertial (I), viscous (B) and elastic (K) parameters as a function of ankle angle. (Subject S1)

## Relation between the Elastic Parameter and Mean Torque

Despite obvious differences in polarity (dorsiflexor and plantarflexor torques have opposite polarity whereas K is always positive), there was a striking similarity in the behaviour of the K/angle curves shown in Fig. 4.6 and the mean torque/angle curves plotted in Fig. 4.2. In both cases mid-range positions were associated with relatively small, constant values whereas torque and K became much larger as the ankle was rotated toward the ROM extremes. Furthermore, similar asymmetries were found in both curves. Thus, subjects who generated less torque at extreme plantarflexion than at maximum dorsiflexion also tended to have smaller values of K when the ankle was plantarflexed than when it was dorsiflexed (see Table 4.1 and compare the same line types in Figs. 4.2 and 4.6). This correspondence is evident when K is plotted as a function of mean joint torque, as in Fig. 4.7. There is a clear linear relation between the two parameters.

This relation was quantified for all six subjects by least squares linear regression and the results of these analyses, including the VAF by the model, slopes, and intercepts, are presented in Table 4.2. The VAF was always greater than 80% and usually greater than 90%. The slopes varied from 9.0 to 15.9 Nm/rad/Nm. These slopes are similar to those found previously for the linear relation between K and mean active torque at the mid-range position previously examined (Hunter & Kearney, 1982).

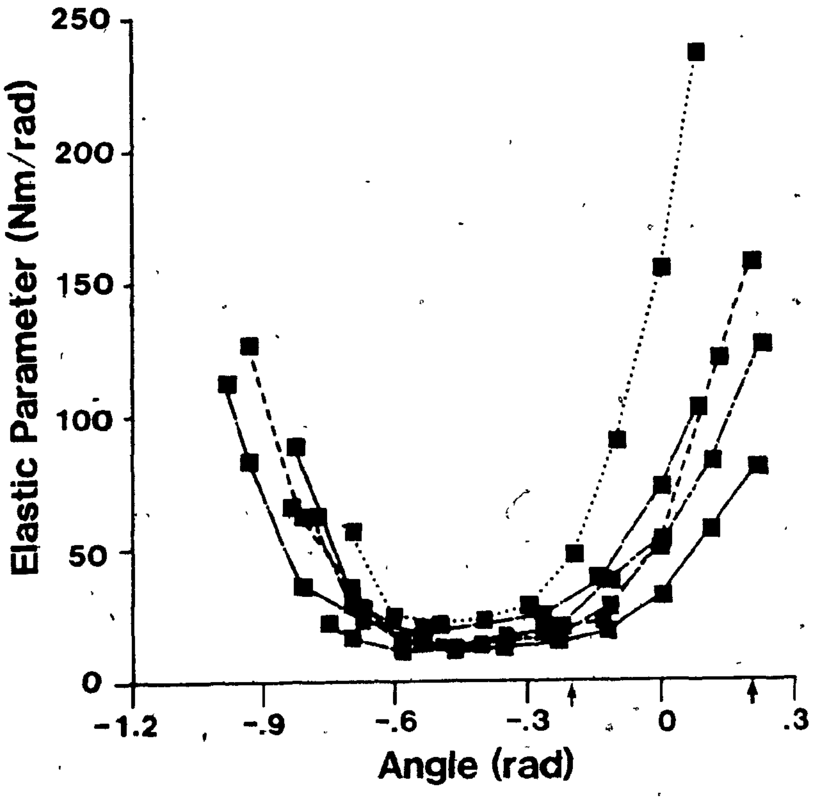


Fig. 4.6: The elastic parameter as a function of ankle angle for all six subjects. The two arrows indicate the range of angles tested by Gottlieb & Agarwal (1978).

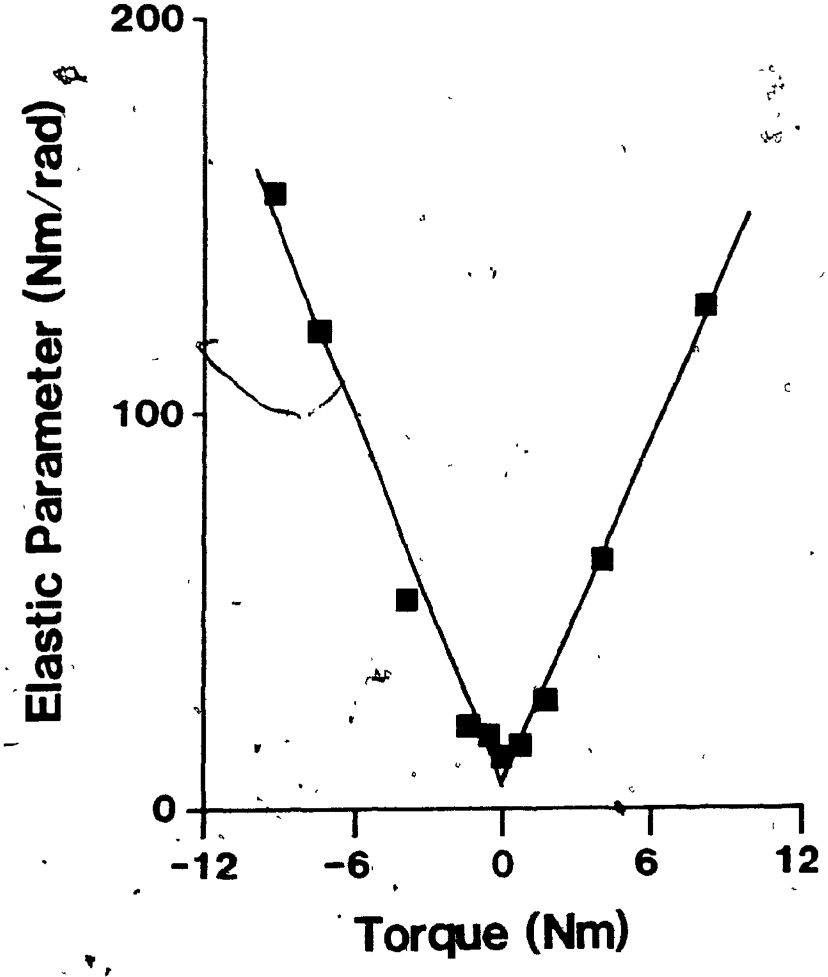


**Fig. 4.7:** The elastic parameter plotted as a function of plantarflexor torque and dorsiflexor torque. The lines were calculated by least squares linear regression. (Subject S4)

| SUBJ | Total N | PLANTARFLEXION N | Slope | Inter. | %VAF | DORSIFLEXION N | Slope | Inter. | %VAF |
|---|---|---|---|---|---|---|---|---|---|
| S1 | 10 | 3 | * | * | * | 8 | 15.4 | 4.2 | 97 |
| S2 | 9 | 3 | * | * | * | 7 | 15.4 | 2.7 | 99 |
| S3 | 10 | 5 | 14.3 | 7.0 | 99 | 6 | 15.6 | 5.7 | 99 |
| S4 | 9 | 4 | 15.9 | 10.8 | 99 | 6 | 9.0 | 13.1 | 97 |
| S5 | 10 | 4 | 15.9 | 26.5 | 87 | 7 | 9.8 | 5.6 | 99 |
| S6 | 10 | 2 | * | * | * | 9 | 13.5 | 1.7 | 98 |

**Table 4.2:** Parameters for the least squares linear regression analysis between K and passive torque. The total N indicates the number of angles tested over the ROM. The number of angles that were associated with plantarflexor torques and and with dorsiflexor torques are also provided. The values obtained when the ankle was at the NP (net joint torque = 0) have been included in both analyses. There were more plantarflexor torques than dorsiflexor torques because the NP was closer to maximum plantarflexion than to maximum dorsiflexion. In some cases there were insufficient plantarflexor torque data points to warrent the linear regression analysis; these cases are indicated by asteriks. The slopes, intercepts and the percent VAF for by a linear relation between K and torque are listed. (The units for the slopes are Nm/rad/Nm and for the intercepts are Nm.)

## DISCUSSION

These experiments have shown that the dynamic stiffness of the ankle joint increased at the extremes of the ROM. Large variations in joint mechanics occurred even in the absence of voluntary muscle contraction. Moreover, it is evident that the position dependent changes in both stiffness and passive torque were very similar; indeed, there was a linear relation between passive joint torque and elastic stiffness. The changes in the passive mechanical properties appeared to be related to the variation in passive joint torque generated when ankle position was altered.

The effect of joint angle on K was much larger than previously shown (Gottlieb & Agarwal, 1978). This discrepancy appears to be related to methodological differences between the two studies. While the ROM tested by these authors was reasonable for measuring the effect of mean dorsiflexor positions, it did not permit the study of position dependency from mid-range through to extreme plantarflexion. Gottlieb & Agarwal (1978) tested angles corresponding to those located between the two arrows in Fig. 4.5. Their observed decrement in K with progressive plantarflexion simply corresponded to the present decrease in K as the ankle was rotated from maximum dorsiflexion to the NP. However, K did not continue to decrease as the joint was plantarflexed beyond mid-range; rather, for five of the six subjects, it increased.

It is not certain how closely the ROM tested in this study approached the subjects' actual ROM since no objective criteria were used to delineate it. Indeed the lack of such criteria is partly responsible for the large variability seen in the ROM literature (e.g. Boone & Azen, 1979). However, in these experiments, the extent of joint excursion was limited only by subject discomfort or evidence of movement at the knee joint which would have resulted in a deceptively larger ankle ROM. Avoidance of the extremes of the ROM on the grounds of a non stationary ankle axis of rotation or changes in foot shape (Gottlieb & Agarwal, 1978) was dismissed since such fluctuations appear to be relatively small (Inman, 1976). Furthermore, the present results demonstrated that the passive mechanical behaviour of the ankle could be identified and modelled over the whole ROM. The VAF by both the compliance impulse response functions and the fit of a second-order, underdamped model to these functions remained high at all mean positions.

The cause of the position dependence of K remains to be determined. It cannot be due to active muscle processes since surface EMG from TA and TS showed that these muscles remained inactive. Neither can it be due to reflex mechanisms since the experimental perturbation and analytic techniques were such that reflex effects on the compliance impulse response functions were insignificant (Hunter et al., 1984b). Rather the magnitude of K appeared to be related to the change in passive joint torque that resulted when mean ankle position was altered.

The passive ankle torque could be altered by position related changes in the joint structure, in the forces generated by the ligaments, tendons, and muscles and in the size of the moment arm associated with these forces. It is not presently possible, however, to identify which of these factors or combination of factors are most significant since there is little quantitative information as to how these hard and soft tissues are affected by changes in ankle position. For example, it has been shown that ankle joint intra-articular pressure increases by up to six fold over the ROM (Eyring & Murray, 1964). The pattern of these pressure changes is very similar to those seen in F; they are minimal at mid-range but then increase rapidly at maximum dorsiflexion and plantarflexion. Such alterations in intra-articular pressure may be related to changes in joint congruency (Wynarsky & Greenwald, 1983) but any subsequent effect on ankle moments is unknown.

Similar difficulties are encountered with regard to the other joint tissues. If either TA or TS were significantly stretched beyond their resting length then large changes in joint torque would be expected. There is, however, no established technique for converting ankle angle to muscle length although such measurements have been attempted experimentally (Fick, 1910; Halar et al., 1978; Grieve et al., 1978) and theoretically (Houk et al., 1971). It is not yet possible to verify how correct these approximations are.

The magnitudes of position related tendon and ligament forces are also uncertain. There is some evidence suggesting that the TS moment arm increases by about two-thirds as the foot is moved from full dorsiflexion to maximum plantarflexion (Sale et al., 1982) which could account for the larger plantarflexor torques generated when the ankle was dorsiflexed. However, the TA tendon is bound by its retinaculae and its moment arm does not appear to vary significantly over the ROM (Procter & Paul, 1982). Thus, the source of the position dependent changes in torque remain uncertain. These issues will be addressed further in Chapter 5.

# CHAPTER 5: ACTIVE JOINT MECHANICS

## SUMMARY

System identification techniques were used to examine the position dependence of active ankle joint mechanics. Subjects were required to maintain tonic contractions in either the tibialis anterior or triceps surae muscles while the ankle was stochastically displaced about different mean positions. The dynamic relation between ankle position and torque was determined for each mean position/tonic torque combination; a non-linear minimization technique was used to estimate the three parameters (inertial, viscous, and elastic) of a second-order, underdamped system. Whereas the inertial parameter remained essentially invariant across all test conditions, the viscous and elastic parameters became larger as the level of tonic activity increased and as the joint was rotated toward the extremes of the range of motion. The relation between K and torque was linear at all ankle angles. The slope of this relation remained constant at all mean positions during plantarflexor contractions; during dorsiflexor contractions the slope increased as the ankle was rotated from maximum plantarflexion to maximum dorsiflexion. These findings are discussed in terms of (1) the physiological correlates of ankle mean position, (2) the relative significance of passive and active joint mechanics and, (3) contrasts in joint behaviour during active dorsiflexor and plantarflexor contractions.

## INTRODUCTION

The relation between ankle joint elastic stiffness and torque has been established for torques generated by predominantly active processes, i.e. due to voluntary muscle contractions (Hunter & Kearney, 1982). K increased linearly with ankle torque with a slope of about 9 Nm/rad/Nm (for dorsiflexor contractions) and of about 12 Nm/rad/Nm (for plantarflexor contractions). Chapter 4 has demonstrated that the relation between K and torque remained linear for torques generated by purely passive processes, i.e. due to changes in the mean joint angle. Moreover, the magnitude of these changes were remarkably similar to the active torque dependent changes in joint mechanics with the K/passive torque slope in the range of 9 to 16 Nm/rad/Nm.

In the first study (Hunter & Kearney, 1982), contributions to the torque by passive tissues were negligible since the ankle was tested about mid-range angles only. In the study described in Chapter 4, contributions to the torque by active processes were negligible since the ankle muscles remained at rest. This separation of active and passive processes is not typical of naturally-occurring ankle movements; many functional activities entail excursions over much of the range of motion (ROM) and require voluntary contractions. For example, very large plantarflexor torques are exerted by ballet dancers to attain and hold the 'en pointe' position and by runners to propel themselves forward (Teitz, 1982).

It is thus of interest to examine the ankle mechanics under experimental conditions that are more representative of the torques and angles encountered in functional situations. In the present study, five levels of active torque up to 50% of the maximum voluntary contraction and mean positions covering more than 85% of the ROM were examined. This greatly extends the only previous study of the position dependence of ankle mechanics (Gottlieb & Agarwal, 1978) in which less than a third of the ankle ROM and only two, very low active torque levels (5 to 10% of the MVC) were examined.

The specific objective of the work presented in this chapter was to characterize the effect of changes in mean ankle position on the active joint mechanics in order to determine the magnitude of these effects and to compare the relative contributions of passive and active joint processes.

## METHODS

The general methods have been described in Chapter 2. In this chapter, details pertinent to experiments designed to investigate active joint mechanics are presented.

### Maximum Voluntary Contractions

TA and TS MVCs were obtained prior to (preMVC) and following (postMVC) the experimental trials. The ankle was rotated to the NP and the subject was required to respond to a step change in the tracking command trace by generating a maximum dorsiflexor torque for period of 5 s. After a 5 min rest this procedure was repeated for a maximum plantarflexor torque. The 5 s maximum contraction period was divided into five 1 s intervals and the mean of each segment was calculated. The largest interval mean torque was designated the maximum torque and the mean of the EMG recorded over the same interval was designated the maximum EMG.

Although muscle fatigue does not alter the passive or active joint mechanics provided that torque remains constant (Hunter & Kearney, 1983a), it does affect the static relation between torque and EMG (e.g. de Vries, 1968). These experiments were therefore designed to avoid the development of muscle fatigue: TA and TS trials were alternated, each tonic level was maintained for only 15 s, and subjects were instructed to immediately discontinue tracking target torques which could not be comfortably maintained. Furthermore, rest periods were provided between successive trials (5 min) and tonic levels (5 s). PostMVCs, recorded in the manner described above, were used to determine whether subjects had become fatigued during the experiment.

## Tracking Stimulus for Tonic Contractions

A tracking stimulus was displayed on the oscilloscope. This stimulus, shown in Fig. 5.1, comprised six levels that ranged from 0 to 50% (in increments of 10%) of the torque generated during the preMVCs. The order of these levels was randomized except that the 50% and 0% levels were always tested last.

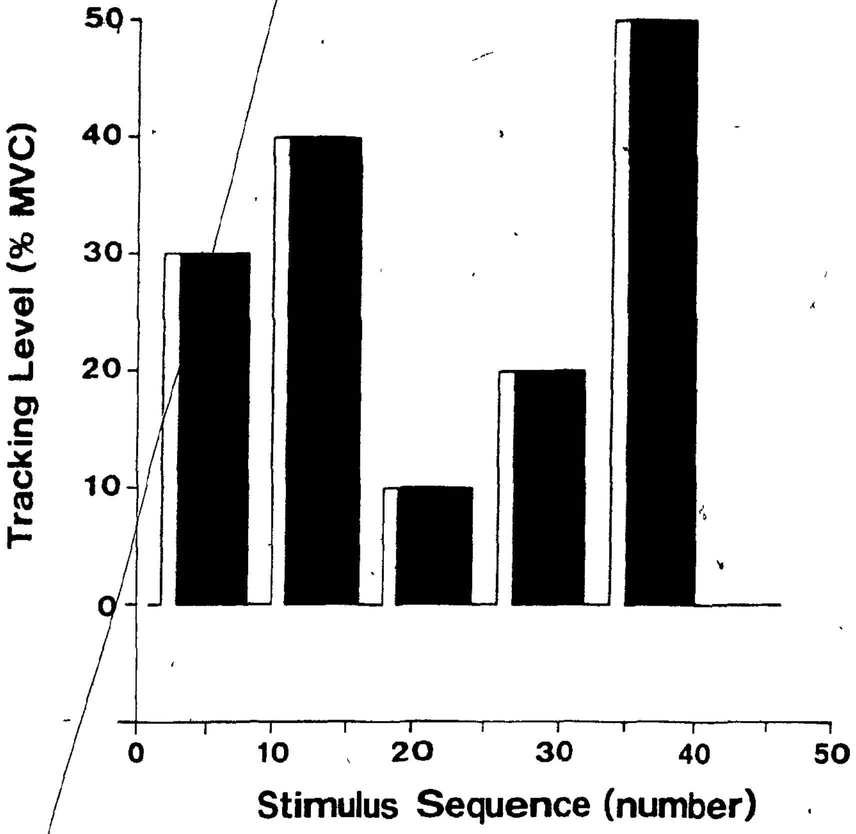


**Fig. 5.1:** A schematic drawing of the tracking stimulus illustrating the randomly-ordered target torque levels. Each of the five active torque levels was held for six repetitions of the stochastic ankle perturbation sequence (i.e. 15 s); only data sampled during the last five repetitions (shaded portion of each bar) were analysed. Tonic levels were separated by a 5 s rest period. The passive condition was tested last.

Torque feedback, torque transducer output filtered at 1 Hz, was also displayed on the oscilloscope. Subjects were required to follow the tracking stimulus with the torque feedback signal. Each tonic level was maintained for 15 s corresponding to six repetitions of the ankle perturbation sequence. The first 2.5 s sequence (the clear portion of each of the histograms shown in Fig. 5.1) was used to enable the subject to achieve the appropriate contraction level and only data from the last five repetitions were analysed.

The passive joint torque, which varied by as much as 16 Nm over the ROM, was subtracted from the feedback signal. This eliminated contributions from passive joint structures so that subjects generated identical levels of active torque at all mean joint positions. Subjects were instructed to relax immediately when a particular contraction level could not be comfortably maintained.

## Experimental Paradigm

The tracking stimulus, adjusted to elicit tonic TS contractions, was displayed to the subject while the ankle was stochastically displaced about a given mean ankle position. This stimulus lasted for 110 s. After a 5 min rest period, the stimulus was adjusted to elicit tonic TA contractions and the ankle was again displaced. The ankle was then rotated to the next mean position and, following a 5 min rest, the experimental sequence was repeated. Ankle mean positions were tested in random order.

Experiments consisted of up to 24 trials and lasted for about

150 min. Subject preparation, including electrode placement, fixation, and amplifier gain and offset adjustments, required an additional 30 min. There was a 15 min rest period at the half-way point; body straps were released at this time and the subject was allowed to sit up. However, in order to avoid disturbing the ankle reference-position, the foot remained firmly encased within its cast and attached to the actuator.

## RESULTS

### Maximum Torques and EMGs

Despite the precautions outlined above, the possibility remained that muscle fatigue did develop during the course of the experiment. An estimate of fatigue was obtained by comparing the pre- and postMVC torques and EMGs. These data are presented in Table 5.1. Also listed in this table are the percent changes between the two values where,

$$\text{Percent Change} = \frac{\text{preMVC Value} - \text{postMVC Value}}{\text{preMVC Value}} * 100$$

The percent change is positive when preMVC > postMVC and is negative when preMVC < postMVC.

The preMVC dorsiflexor torque varied from 30 to 55 Nm whereas the preMVC plantarflexor torque was considerably larger, varying from 54 to 85 Nm. PreMVC TA EMGs varied from 377 to 598 µV whereas preMVC EMGs recorded from TS electrodes varied from -136 to -281 µV. The

| | TA | | | | | TS | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **SUBJECT** | S1 | S2 | S3 | S5 | S7 | S1 | S2 | S3 | S5 | S7 |
| **MAXIMUM TORQUE** | | | | | | | | | | |
| Pre-Experiment | 30.4 | 50.0 | 39.9 | 37.1 | 54.7 | -68.4 | -69.6 | -54.1 | -71.8 | -85.4 |
| Post-Experiment | 28.5 | 48.7 | 40.7 | 38.9 | 48.9 | -66.2 | -71.0 | -52.4 | -69.2 | -75.9 |
| Percent Change | 6.2 | 2.6 | -2.0 | -4.8 | 10.6 | 3.2 | -2.0 | 3.1 | 3.6 | 11.1 |
| **MAXIMUM EMG** | | | | | | | | | | |
| Pre-Experiment | 590 | 598 | 534 | 377 | 479 | -136 | -199 | -144 | -281 | - 277 |
| Post-Experiment | 537 | 528 | 507 | 390 | 360 | -153 | -141 | - 89 | -256 | -293 |
| Percent Change | 9.0 | 11.7 | 5.1 | -3.4 | 24.8 | -12.5 | 29.1 | 38.2 | 8.9 | -5.8 |

**Table 5.1:** Dorsiflexor and plantarflexor torques and EMGs obtained during maximum voluntary contractions immediately prior to and following the experiment. The preMVCs and postMVCs are listed for each of the five subjects. Also listed are the preMVC/postMVC precent changes in the torques and EMG. The units of torque are Nm and the units of EMG are μV.

preMVC torque values were used to obtain the active torque tracking levels.

For four of the five subjects, maximum dorsiflexor torque changed by less than 2 Nm (less than 6% of that generated during the preMVC). For these same subjects, maximum plantarflexor torque changed by less than 3 Nm (less than 4% of the preMVC value). The percent change in maximum torque recorded for Subjects S1, S2, S3, and S5 was similar to that observed during repeated MVC measures at a single ankle angle in this laboratory and elsewhere (e.g. Sale et al., 1982) under experimental conditions that precluded the onset of muscle fatigue.

The results for Subject S7 were less clear-cut. This subject's dorsiflexor maximum torque decreased by 5.8 Nm (10.6% of preMVC) and plantarflexor maximum torque decreased by 9.5 Nm (11.1% of preMVC). Given the magnitude of the percent decrease in torque values (about 10%), the development of muscle fatigue prior to completion of this experiment could not be excluded. However, since the order of presentation of tested positions was randomized, fatigue, if present, would be independent of ankle angle and would therefore contribute to variability in the data rather than cause systematic modifications. Moreover, the general experimental results for this subject did not differ in any substantive way from those of the other subjects. The problem of fatigue was thus not considered further.

The postMVC TA EMG varied from -3.4 to 24.8% of the preMVC value; for maximum TS EMG this variation ranged from -12.5 to 38.2%. The difference between pre- and postMVC EMGs for Subject S7 was comparable to the values recorded for the other subjects. The variation between pre- and postMVC EMGs was generally greater than that seen for maximum torques, a result that is well documented in the literature (e.g. Viitasalo et al., 1980). Again, the variability seen here was no greater than that observed during repeated measures obtained in this laboratory when muscle fatigue was avoided.

## Static Analysis

### Mean Dorsiflexor Torque and TA EMG

In order to demonstrate that subjects were able to maintain the appropriate torque over most of the ROM and to determine the effect of ankle position on the EMG, mean values for the torque, TA EMG, and TS EMG signals were examined. Initially, these values were calculated during intervals corresponding to each repetition of the stochastic perturbation. An example of the interval means obtained during dorsiflexor contractions at 0 rad (the reference-position) are plotted with respect to stimulus sequence number in Fig. 5.2.

Note that the subject attained the required tonic level within one repetition of the ankle perturbation sequence and that the torque dropped to near zero during the 5 s interval between adjacent tracking levels. The agonist EMG dominated the response while the antagonist EMG activity was relatively small and may have been due to either cross-talk from the dorsiflexor muscles or co-contraction. Similar results were obtained for torque and EMG interval means during TS

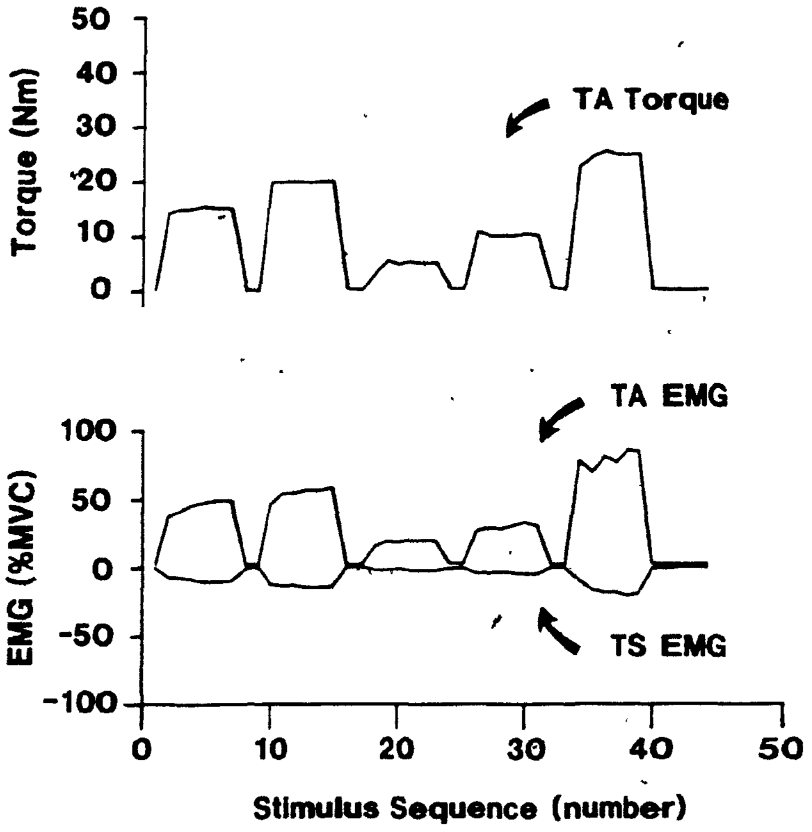


Fig. 5.2: Torque and EMG interval means during tonic dorsiflexor contractions at 0 rad. The agonist (TA) and antagonist (TS) EMGs have been normalized to their respective maximum EMGs obtained with the ankle at the NP. This subject has maintained a series of tonic contractions at 30%, 40%, 10%, 20%, 50% and 0% of the NP TA MVC. Torque and EMG levels returned to 0 following each tonic contraction. The interval mean EMGs have been normalized with respect to the maximum EMG generated during the NP MVCs. (Subject S2)

contractions.

The magnitude of the mean total torque at any given contraction level varied as a function of ankle position since it included both passive and active components. Mean total dorsiflexor torques for one subject are shown in Fig. 5.3. Each square represents the mean torque during each test condition. Mean torques obtained at the same contraction level are joined by solid lines. The passive joint torque, the lowest trace in Fig. 5.3 (circles), shows the characteristic pattern of the torque/angle relation in the relaxed ankle joint presented in Chapter 4.

Active dorsiflexor torques, shown in Fig. 5.4, were obtained by subtracting the passive torque from the total torque. Vertical bars represent the SD of the interval means for each ankle position and contraction level. It is evident that the subject maintained relatively constant levels of active torque over most of the ROM. However, subjects were not able to track all target torques at the extremes of the ROM. For example, this subject was unable to maintain 50% NP TA MVC at the most dorsiflexed position (note the missing point in the top trace of Figs. 5.3 and 5.4). Two other subjects were not able to track the 30% and 40% levels at this extreme angle and, in one case, the 50% level could not be maintained at either of the two most extreme dorsiflexor positions.

Interval mean TA EMGs corresponding to the torque data presented in Fig. 5.4 are plotted in Fig. 5.5. These data were obtained during tonic TA contractions that ranged from 10% (bottom trace) to 50% (top

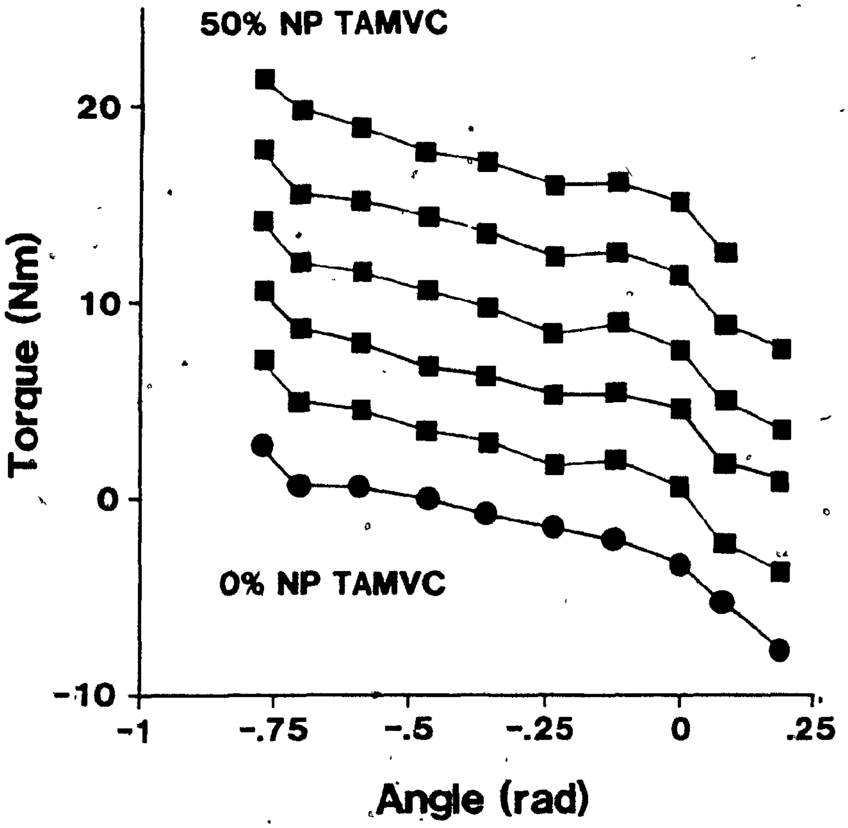


**Fig. 5.3:** Mean total dorsiflexor torque as a function of angle. Each square represents the mean joint torque of five repetitions of the stimulus sequence at a particular active torque level and ankle angle. The passive condition is indicated by circles. Solid lines connecting individual points represent iso-active torque contours over the ROM. (Subject S5)

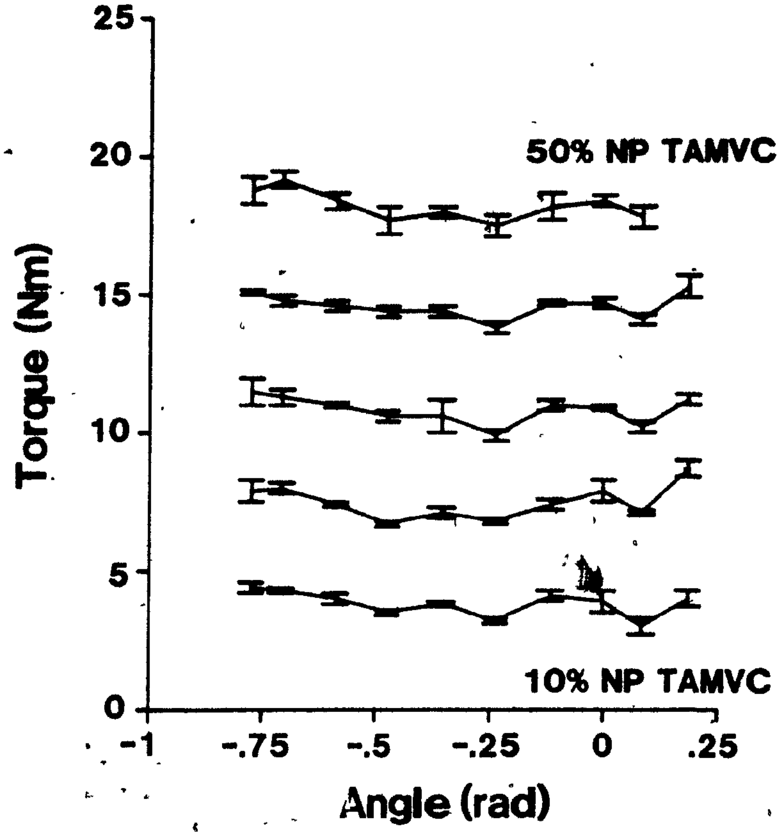


Fig. 5.4: Mean active dorsiflexor torque as a function of angle. The passive torque at each angle shown in Fig. 5.3 has been subtracted from the total torque at the same angle. The vertical bars represent the SD of the mean torque generated during the five repetitions of the stimulus sequence., (Subject S5)

trace) NP MVC. The values have been normalized to the maximum EMG obtained during the NP MVC. The vertical bars represent the SD of the mean over the five stimulus repetitions at each test condition.

A comparison of Figs. 5.4 and 5.5 indicates that mean TA EMG was more variable than mean torque. This variability was comparable to that seen during sub-maximal, isometric contractions of triceps brachii measured with surface electrodes (Yang & Winter, 1983). Despite this variability, TA EMG values at a particular tonic level were usually distinct from values at other levels.

### Mean Plantarflexor Torque and TS EMG

Mean active torques, recorded during plantarflexor contractions, are plotted in Fig. 5.6. As was the case during dorsiflexor contractions, these curves were obtained by subtracting the passive torque from the total plantarflexor torque. The subject held the level of active plantarflexor torque quite constant over the ROM. However, note that this subject was unable to track the highest contraction level when the ankle was placed in extreme plantarflexion.

Mean TS EMGs corresponding to the torque data shown in Fig. 5.6 are plotted in Fig. 5.7. Mean TS EMGs were generally smaller than TA EMGs for comparable contractions. This may reflect differences in electrode location with respect to the underlying muscle fibers. However, TS EMG SDs were larger than those found for TA EMG particularly at the higher contraction levels and in the more plantarflexed positions. Since both soleus and the gastrocnemii would

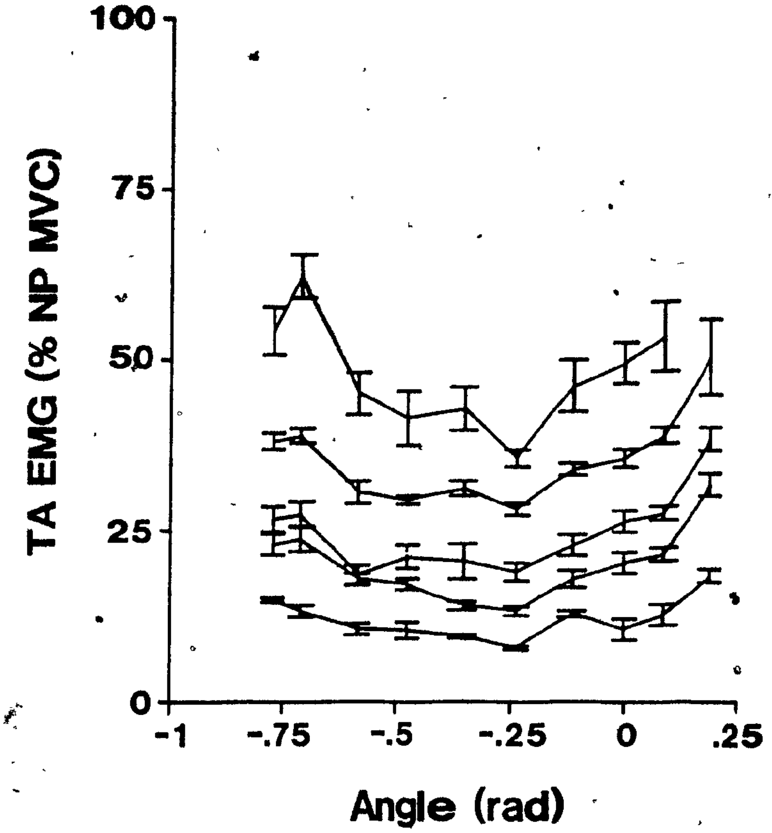


**Fig. 5.5:** Mean TA EMG as a function of angle. The vertical bars represent the SD of the mean EMG generated during each of the five repetitions of the stimulus sequence. The data have been normalized to the maximum EMG generated during the NP TA MVC. (Subject S5)

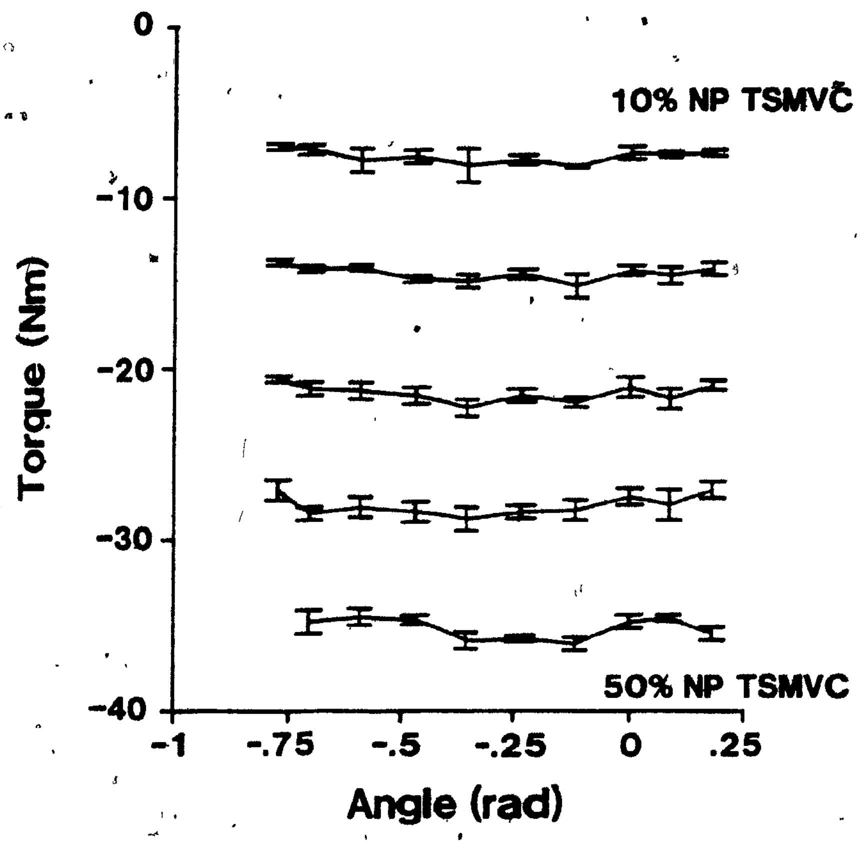


**Fig. 5.6:** Mean active plantarflexor torque as a function of angle. These points have been obtained as outlined for Fig. 5.4 except that in this case the total torque was generated during tonic plantarflexor contractions. Again, the vertical bars represent the SD of the mean torque calculated at each of the five repetitions of the stimulus sequence. (Subject S5)

contribute to the surface EMG, this variability may have been due to alterations in the relative contribution of the three muscles. This has been shown to vary with ankle position (Herman & Bragin, 1969) and level of contraction (Hof & van den Burg, 1977).

### Relation between Torque and EMG

The effect of mean ankle position on EMG was investigated by examining the static relation between mean torque and EMG at all ankle angles. Since active torque was maintained constant over the ROM, changes in the parameters of the torque/EMG relation would reflect position-related changes in EMG. A multiple linear regression model was fitted between active torque and TA and TS EMG. This is referred to as the EMG model. Torques and EMGs generated during both dorsiflexor and plantarflexor contractions were included to provide a more robust data set and to account for possible co-contraction of both agonist and antagonist muscles.

The results of this analysis are presented in Table 5.2 with the VAF by this model, the TA EMG and TS EMG coefficents and the intercepts listed for each subject. The variation in active torque accounted for by TA EMG and TS EMG ranged from 72 to 93%. Thus, moderate to good predictions of ankle torque could be made from the agonist and antagonist EMGs without considering ankle position. TA EMG coefficents varied from .12 to .17 Nm/μV whereas the magnitude of TS EMG coefficients ranged from .21 to .45 Nm/μV. The differences in coefficient size is probably related to electrode placement.

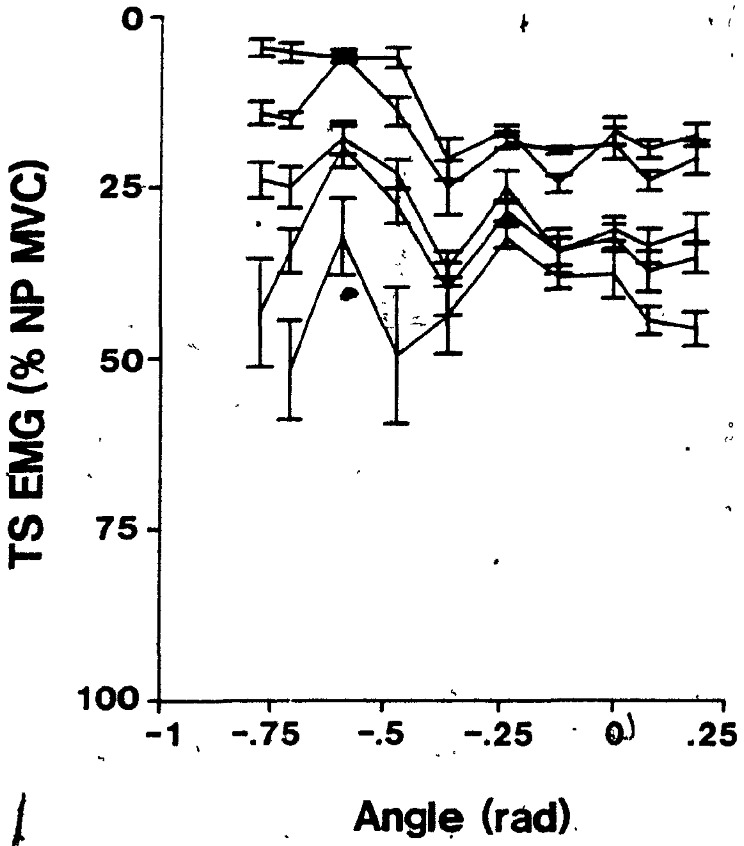


Fig. 5.7: Mean TS EMG as a function of angle. The vertical bars represent the SD of the mean EMG generated during the five repetitions of the stimulus sequence. The data have been normalized to the maximum EMG generated during the NP TS MVC. (Subject S5)

| SUBJECT | INTERCEPT (Nm) | TA EMG Coeff. (Nm/μV) | TS EMG Coeff. (Nm/μV) | EMG MODEL VAF (%) | EMG-ANGLE MODEL VAF (%) |
|---|---|---|---|---|---|
| S1 | -2.3 | 0.17 | 0.45 | 72.2 | 75.5 |
| S2 | -3.1 | 0.13 | 0.24 | 84.2 | 88.7 |
| S3 | -2.0 | 0.14 | 0.21 | 82.1 | 82.1 |
| S5 | -2.2 | 0.16 | 0.31 | 93.0 | 93.0 |
| S7 | 1.5 | 0.12 | 0.33 | 92.2 | 94.1 |

**Table 5.2:** Results of a multiple linear regression analysis between agonist and antagonist EMG and torque. The VAF by the EMG model, the TA EMG and TS EMG coefficients and the intercepts are listed for all subjects. The VAF by the EMG-angle model, which included mean ankle position as a third independent variable, is also listed.

The multiple linear regression analysis was repeated including ankle angle as a third independent variable. If the influence of mean position on the torque/EMG relation was linear then the VAF by this new model, referred to as the EMG-angle model, should be significantly greater than that accounted for by the EMG model. However, as shown in Table 5.2, this was not the case. The EMG-angle model accounted for 75 to 94% of the variation in torque, an increase of less than 5% of the VAF by the EMG model.

The failure of the EMG-angle model to account for more variation in torque than accounted for by the EMG model could result if the effect of mean position was not linear. This possibility was tested by fitting multiple linear regression models between torque and TA EMG and TS EMG at each ankle angle. The results of this analysis are presented in Table 5.3. The VAF by the EMG models, the coefficents and the intercepts are listed for each subject at all angles in an order ranging from maximum plantarflexion to maximum dorsiflexion.

The VAF by the EMG models at each ankle angle was usually greater than 90% and was, in general, larger than the VAF in the previous two analyses. The variation in the magnitude of the VAF was small and non-systematic. In contrast, changes in the magnitudes of the EMG coefficents were more pronounced. TA EMG coefficients varied from .09 to .31 Nm/μV and TS EMG coefficients varied from .10 to 1.23 Nm/μV. The percent change in magnitude for a particular subject varied from 170 to 330% for TA EMG coefficients and from 170 to 560% for TS EMG coefficients. The changes in coefficient magnitude were not, however, systematic. Thus, although it can be concluded that ankle position

| SUBJECT | ANGLE (rad) | INTERCEPT (Nm) | TA EMG (μV/Nm) | TS EMG (μV/Nm) | VAF (%) |
|---|---|---|---|---|---|
| S1 | -0.828 | 1.1 | 0.20 | 0.36 | 86.6 |
| | -0.710 | -1.1 | 0.21 | 0.36 | 87.3 |
| | -0.591 | -0.4 | 0.26 | 0.62 | 93.5 |
| | -0.474 | 2.0 | 0.29 | 0.89 | 95.2 |
| | -0.358 | 1.1 | 0.31 | 1.18 | 96.7 |
| | -0.239 | 3.5 | 0.27 | 1.23 | 96.9 |
| | -0.121 | 1.3 | 0.15 | 0.77 | 93.0 |
| | 0.001 | 3.1 | 0.15 | 0.70 | 93.9 |
| | 0.115 | 2.5 | 0.11 | 0.60 | 89.2 |
| | 0.215 | 1.6 | 0.10 | 0.41 | 86.8 |
| S2 | -0.756 | 1.5 | 0.12 | 0.26 | 95.2 |
| | -0.639 | 1.9 | 0.14 | 0.25 | 96.8 |
| | -0.520 | 0.9 | 0.14 | 0.23 | 95.3 |
| | -0.404 | 1.8 | 0.14 | 0.30 | 98.1 |
| | -0.302 | 3.1 | 0.18 | 0.51 | 94.7 |
| | -0.202 | 4.8 | 0.18 | 0.64 | 93.9 |
| | -0.101 | 4.6 | 0.16 | 0.64 | 92.1 |
| | 0.000 | 6.5 | 0.13 | 0.70 | 92.2 |
| | 0.083 | -1.2 | 0.20 | 0.52 | 88.8 |
| S3 | -0.944 | 0.1 | 0.09 | 0.12 | 99.3 |
| | -0.823 | -0.9 | 0.11 | 0.17 | 99.2 |
| | -0.709 | -1.5 | 0.14 | 0.22 | 99.0 |
| | -0.590 | -2.7 | 0.21 | 0.38 | 98.4 |
| | -0.473 | -2.9 | 0.30 | 0.67 | 96.8 |
| | -0.234 | -3.8 | 0.22 | 0.42 | 80.6 |
| | -0.119 | -2.1 | 0.17 | 0.17 | 76.8 |
| | 0.000 | 1.8 | 0.12 | 0.23 | 92.2 |
| | 0.084 | 1.1 | 0.12 | 0.26 | 87.8 |
| | 0.165 | 1.2 | 0.11 | 0.24 | 91.3 |
| S5 | -0.775 | -4.5 | 0.19 | 0.27 | 98.4 |
| | -0.708 | -4.3 | 0.17 | 0.29 | 99.4 |
| | -0.592 | -4.8 | 0.25 | 0.46 | 97.5 |
| | -0.472 | -3.4 | 0.19 | 0.23 | 98.9 |
| | -0.359 | 1.1 | 0.17 | 0.32 | 97.0 |
| | -0.238 | 0.1 | 0.18 | 0.41 | 95.8 |
| | -0.122 | -0.5 | 0.16 | 0.32 | 93.7 |
| | 0.001 | 0.9 | 0.13 | 0.35 | 96.2 |
| | 0.081 | 0.8 | 0.12 | 0.31 | 97.8 |
| | 0.181 | -1.1 | 0.08 | 0.10 | 99.5 |
| S7 | -0.509 | 0.4 | 0.14 | 0.24 | 98.5 |
| | -0.424 | 0.2 | 0.15 | 0.32 | 99.7 |
| | -0.341 | 1.5 | 0.14 | 0.29 | 98.1 |
| | -0.256 | 0.8 | 0.15 | 0.34 | 94.0 |
| | -0.171 | 1.5 | 0.14 | 0.29 | 96.1 |
| | -0.087 | 1.0 | 0.14 | 0.36 | 96.0 |
| | 0.002 | 2.1 | 0.12 | 0.37 | 94.0 |
| | 0.095 | 1.2 | 0.10 | 0.41 | 96.5 |
| | 0.197 | 2.7 | 0.07 | 0.40 | 95.3 |

**Table 5.3:** Results of a multiple linear regression models between torque and agonist and antagonist EMG data at each ankle angle. The VAF by these models, the TA EMG and TS EMG coefficients and the intercepts are presented for all subjects.

has some effect on the agonist and antagonist EMG this effect cannot be characterized from the present results.

## Dynamic Analysis

### Non-Parametric Analysis of Mechanics

A partial set of stiffness transfer function gain curves obtained during plantarflexor contractions is plotted in Fig. 5.8. Gain curves are shown for the passive case (solid lines) and for 10% (dotted lines) and 50% (dashed lines) tonic contractions. For clarity, only gain curves obtained at ankle angles ranging from the NP (lowermost solid, dotted, and dashed lines) to maximum dorsiflexion (uppermost solid, dotted, and dashed lines) have been plotted.

It is evident that both the low frequency gain and the resonant-dip frequency increased as the ankle was dorsiflexed. This was true at all levels of tonic activity. It is also evident that the low frequency gain and resonant frequency increased as the level of tonic activity increased. This has already been demonstrated for mid-range ankle positions (Hunter & Kearney, 1982) but is now confirmed for other mean ankle positions. The increase in gain that resulted when the passive ankle was rotated from the NP to maximum dorsiflexion (Fig. 5.8, lowest to highest solid lines) was of similar magnitude to that observed when the level of tonic activity in the ankle at the NP changed from 0 to 10% (Fig. 5.8, lowest solid to lowest dotted lines).

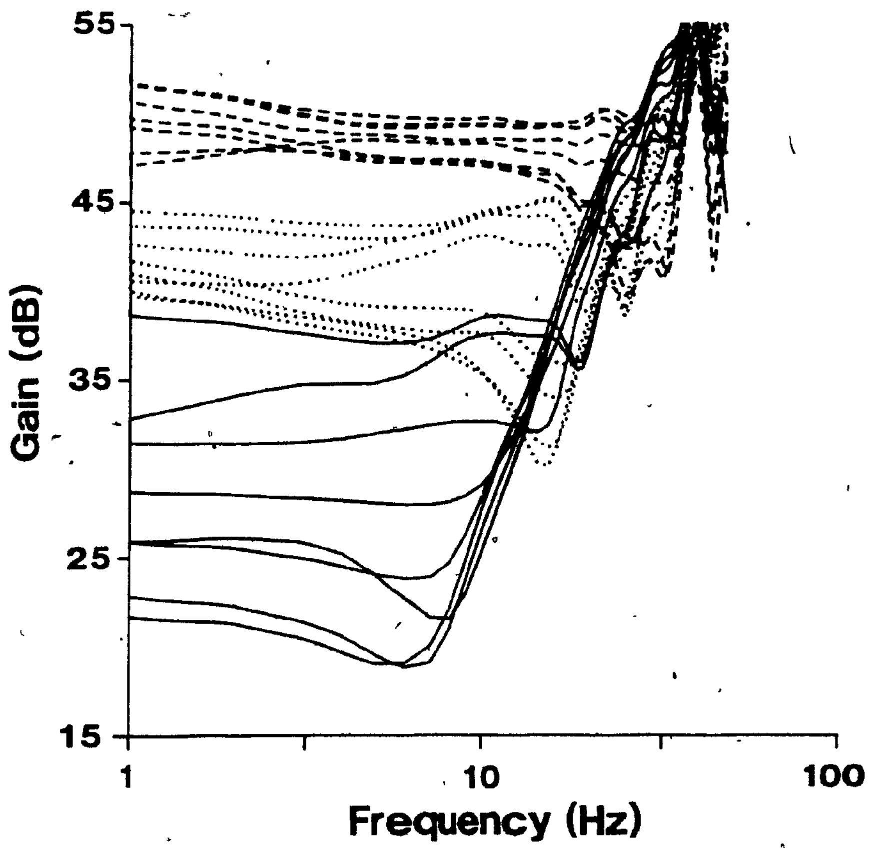


**Fig. 5.8:** Stiffness transfer function gain curves calculated when the ankle joint muscles were at rest (solid lines) or actively contracting at 10% (dotted lines) or 50% (dashed lines) of the NP TA MVC. The data in this figure correspond to ankle angles ranging from the NP (lowest solid, dotted and dashed lines) to maximum dorsiflexion (highest solid, dotted and dashed lines). (Subject S5)

Compliance impulse response functions were also calculated for each test condition. The VAF by these functions, though always larger than 85%, was about 5% lower than those obtained when the ankle muscles were at rest. This decrease was likely due to small fluctuations in the tonically maintained active torque (i.e. minor tracking errors).

## Parametric Analysis of Mechanics

The compliance impulse response functions were modelled as second-order, underdamped systems having inertial, viscous, and elastic terms. The VAF by these models was always larger than 80% and usually greater than 90%. It did not appear to vary systematically with mean position.

These three parameters, obtained for one subject during the various mean torque and mean angle test conditions, are plotted as functions of mean joint position in Fig. 5.9. Each square represents I (Fig. 5.9, top graph), B (Fig. 5.9, middle graph) or K (Fig. 5.9, bottom graph) calculated during tonic dorsiflexor contractions. Circles represent the parameters calculated when the ankle joint muscles were at rest. All points obtained at the same tonic level are joined, thereby generating three sets of six iso-active-torque curves.

The curves in the top graph of Fig. 5.9 are indistinguishable and it is evident that I varied with neither level of contraction nor mean position. In contrast, B and K varied with both mean torque and mean angle. For the subject whose data is presented in Fig. 5.9, B and K

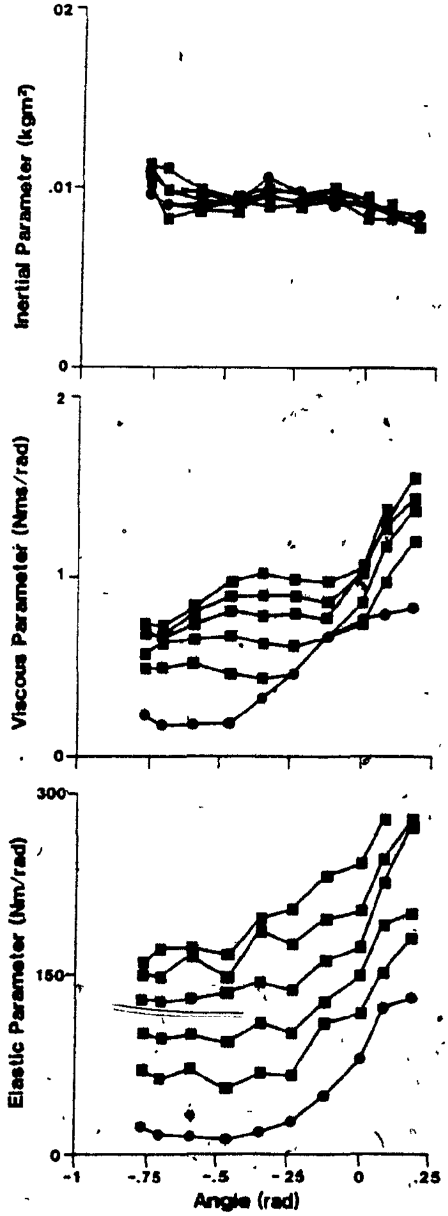


Fig. 5.9: The inertial (I, top graph), viscous (B, middle graph) and elastic (K, bottom graph) parameters as functions of ankle angle for the six active dorsiflexor torque levels. Circles represent values of I, B and K determined while the ankle muscles were at rest. Squares represent values of I, B and K for contractions varying from 10 to 50% of the NP TA MVC. Lines connect points obtained at identical active torque levels. (Subject S2)

increased as the relaxed ankle was progressively dorsiflexed and as the level of tonic activity increased. However, the shape of the B/mean angle and K/mean angle curves remained remarkably similar to their passive counterparts with the result that a set of six parallel iso-active-torque curves were generated for torques varying from 0 to 50% NP TA MVC.

Comparable sets of parallel iso-active-torque curves were obtained for dorsiflexor contractions by other subjects and for plantarflexor contractions by all subjects. However, as was the case for passive joint mechanics, B and K during active contractions usually increased when the ankle was placed at both extremes of the ROM. This particularly asymmetric data set is presented to simplify subsequent presentation of results (see Fig. 5.10 below).

### Relation between the Elastic Parameter and Active Torque

The data in the lowest graph of Fig. 5.9 have been re-plotted in Fig. 5.10 to show the relation between K and active torque as a function of joint position. Each point represents K determined during one of the test conditions; circles indicate an absence of tonic activity and squares indicate tonic contractions. However, now all points obtained at the same mean position are joined, with each iso-angle curve representing the K/active torque relation for angles ranging from maximum plantarflexion (bottom trace) to maximum dorsiflexion (top trace).

Three observations follow from the iso-angle curves presented in

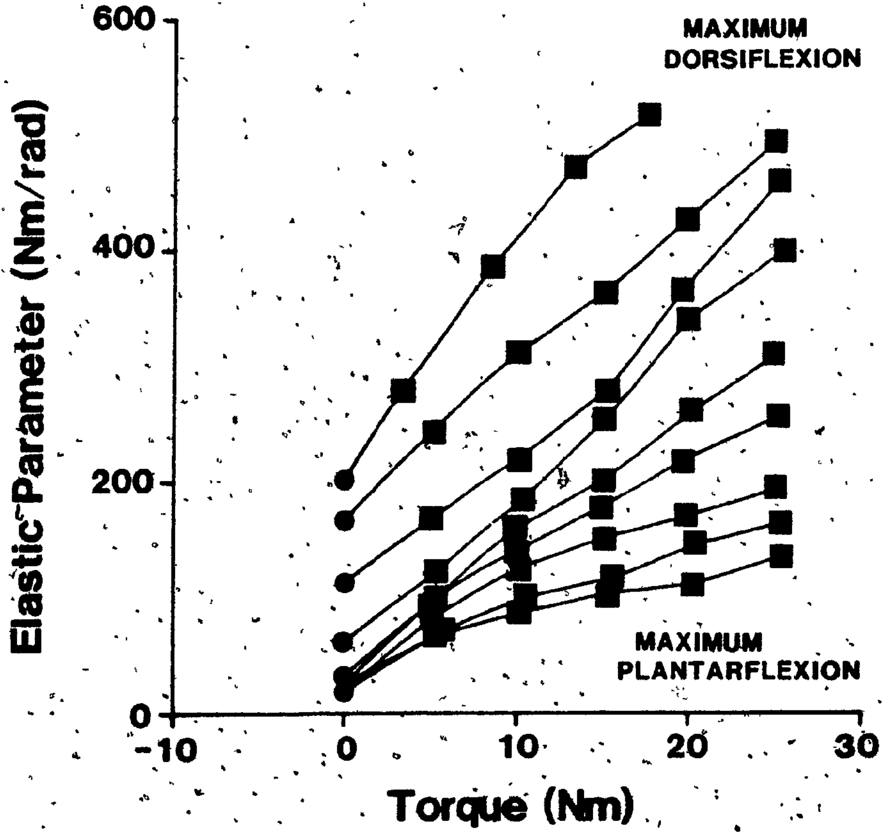


**Fig. 5.10:** K plotted with respect to mean active dorsiflexor torque at ankle positions ranging from maximum plantarflexion (bottom trace) to maximum dorsiflexion (top trace). Individual points, representing active torque levels varying from 0% to 50% of the NP TA MVC, are connected by iso-angle lines. (Subject S2)

Fig. 5.10. First, the relation between K and active dorsiflexor torque appears to be linear at all ankle angles. Second, the iso-angle curves do not appear to be strictly parallel but rather seem to become steeper as the ankle is more dorsiflexed. Third, these curves intersect the K-axis at successively higher values as the ankle is more dorsiflexed. This strictly monotonic increment in iso-angle curve offsets was related to this subject's asymmetrical response at the two ROM extremes.

These observations were quantified by fitting straight lines to the K/active torque data at each angle. The results of a least squares linear regression analysis are shown in Table 5.4 with VAFs, slopes, and intercepts listed for each subject in an order ranging from maximum plantarflexion to maximum dorsiflexion. The VAF was generally greater than 85% and usually greater than 95% and it is evident that the relation between K and active dorsiflexor torque was linear at all mean positions.

The magnitude of the K/active dorsiflexor torque slopes was moderately affected by mean ankle position. Whereas NP slopes varied from 7.2 to 8.9 Nm/rad/Nm, the slope decreased to as little as 2.9 Nm/rad/Nm near maximum plantarflexion (Subject S3 at -.82 rad) and increased to as much as 18.2 Nm/rad/Nm near maximum dorsiflexion (Subject S2 at .08 rad). Individual subjects showed a percent change in slope magnitude that varied from 140 to 460% over the ROM with the slope becoming steeper as the ankle was dorsiflexed. This trend was not, however, systematic. Although two of the five subjects showed a monotonic increase in slope as the ankle was dorsiflexed, the slopes

| SUBJECT | ANGLE (rad) | SLOPE (Nm/rad/Nm) | INTERCEPT (Nm/rad) | VAF (%) |
|---|---|---|---|---|
| S1 | -0.828 | 7.4 | 85.4 | 84.2 |
| | -0.710 | 5.8 | 39.1 | 97.1 |
| | -0.591 | 7.4 | 27.1 | 97.7 |
| | -0.474 | 8.6 | 20.8 | 98.3 |
| | -0.358 | 11.5 | 26.0 | 97.1 |
| | -0.239 | 12.2 | 19.8 | 98.8 |
| | -0.121 | 16.5 | 11.0 | 97.6 |
| | 0.001 | 7.7 | 37.9 | 98.3 |
| | 0.115 | 6.3 | 86.9 | 98.4 |
| | 0.215 | 9.0 | 99.2 | 98.8 |
| S2 | -0.756 | 4.0 | 37.8 | 94.8 |
| | -0.639 | 5.5 | 31.6 | 97.0 |
| | -0.520 | 6.7 | 40.5 | 94.1 |
| | -0.404 | 8.9 | 42.4 | 98.0 |
| | -0.302 | 10.9 | 40.2 | 99.6 |
| | -0.202 | 13.6 | 54.0 | 99.6 |
| | -0.101 | 13.6 | 97.6 | 98.2 |
| | 0.000 | 16.0 | 174.9 | 99.7 |
| | 0.083 | 18.2 | 215.9 | 98.6 |
| S3 | -0.944 | 3.1 | 91.5 | 52.5 |
| | -0.823 | 2.9 | 35.3 | 95.2 |
| | -0.709 | 4.1 | 19.3 | 99.2 |
| | -0.590 | 5.8 | 21.4 | 95.1 |
| | -0.473 | 7.2 | 20.2 | 99.2 |
| | -0.234 | 9.7 | 24.2 | 98.8 |
| | -0.119 | 10.8 | 27.0 | 99.4 |
| | 0.000 | 8.2 | 93.1 | 97.9 |
| | 0.084 | 7.7 | 109.0 | 93.8 |
| | 0.165 | 7.3 | 144.3 | 95.3 |
| S5 | -0.775 | 7.3 | 35.3 | 96.0 |
| | -0.708 | 8.1 | 25.4 | 98.1 |
| | -0.592 | 8.6 | 29.6 | 96.0 |
| | -0.472 | 8.7 | 24.6 | 96.1 |
| | -0.359 | 10.1 | 29.1 | 97.5 |
| | -0.238 | 10.1 | 31.7 | 99.7 |
| | -0.122 | 9.6 | 57.2 | 98.9 |
| | 0.001 | 8.6 | 80.0 | 99.7 |
| | 0.081 | 8.8 | 125.5 | 99.0 |
| | 0.181 | 9.9 | 135.1 | 93.8 |
| S7 | -0.509 | 5.1 | 78.5 | 85.5 |
| | -0.424 | 6.5 | 45.4 | 94.6 |
| | -0.341 | 8.3 | 51.4 | 96.5 |
| | -0.256 | 9.4 | 51.5 | 96.0 |
| | -0.171 | 11.8 | 36.6 | 98.1 |
| | -0.087 | 12.7 | 47.1 | 99.0 |
| | 0.002 | 13.7 | 73.1 | 99.9 |
| | 0.095 | 13.3 | 188.4 | 99.2 |
| | 0.197 | 12.3 | 247.2 | 92.2 |

**Table 5.4:** The slopes, intercepts and VAFs by a linear regression model between K and active dorsiflexor torque at each mean position.

for the remaining three subjects increased initially but then either decreased or remained relatively constant.

The K/active torque intercepts, also shown in Table 5.4, provided an indication of the magnitude of K at each angle when the ankle muscles were at rest (0 active torque). As expected from the characterization of the passive ankle joint mechanics, the intercept generally became larger when the ankle was placed at either end of the ROM. Note, however, that this increase was usually asymmetric with much larger values for K at maximum dorsiflexion.

The values of K obtained during plantarflexor contractions at different mean positions are plotted with respect to active torque in Fig. 5.11. As in Fig. 5.10, a series of iso-angle curves, varying from maximum plantarflexion (bottom trace) to maximum dorsiflexion (top trace), illustrate the relation between K and active plantarflexor torque as a function of ankle angle.

The relation between K and active plantarflexor torque also appears to be linear at all mean positions. There is no apparent position dependent change in slope magnitude but the curves do cross the K-axis at successively higher values. These observations were also quantified by linear regression analysis and the results are presented in Table 5.5.

The VAF by a linear relation between K and active plantarflexor torque was never less than 90%. Again the intercepts reflect the magnitude of K when active torque was 0. These values also correspond

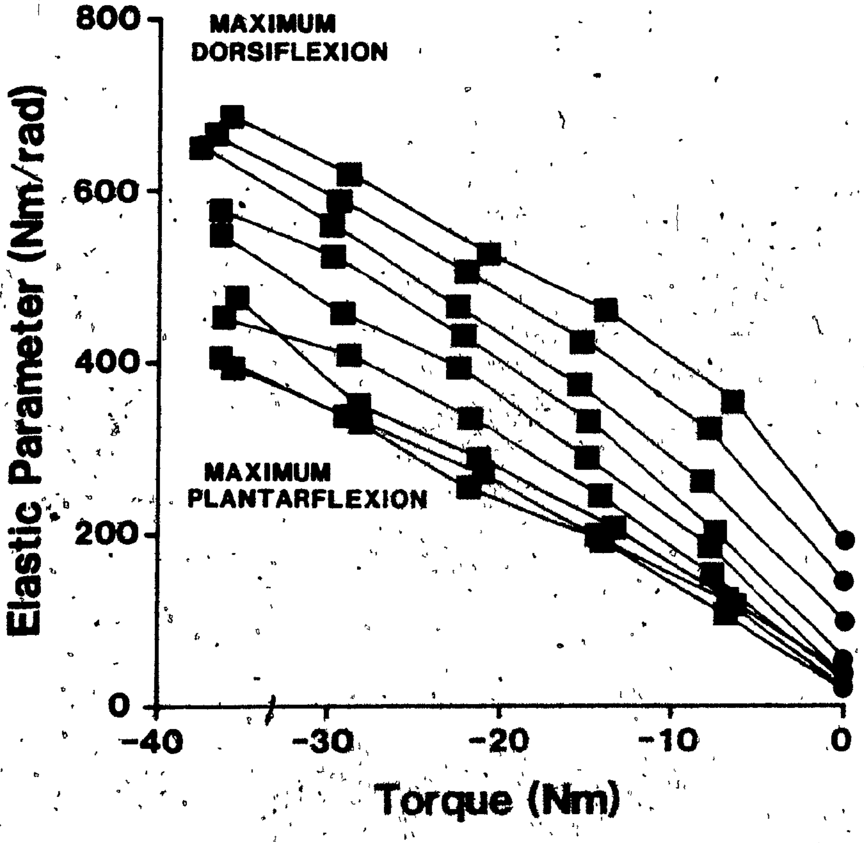


**Fig. 5.11:** K plotted with respect to mean active plantarflexor torque at ankle positions ranging from maximum plantarflexion (bottom trace) to maximum dorsiflexion (top trace). Individual points, representing active torque levels varying from 0% to 50% of the NP TS MVC, are connected by iso-ankle angles lines. (Subject S2)

| SUBJECT | ANGLE (rad) | SLOPE (Nm/rad/Nm) | INTERCEPT (Nm/rad) | VAF (%) |
|---|---|---|---|---|
| S1 | -0.828 | -17.3 | 94.4 | 99.4 |
| | -0.710 | -11.2 | 45.2 | 98.2 |
| | -0.591 | - 7.8 | 37.5 | 96.9 |
| | -0.474 | - 8.5 | 27.2 | 98.6 |
| | -0.358 | -10.6 | 40.6 | 96.8 |
| | -0.239 | - 9.1 | 49.5 | 95.5 |
| | -0.121 | -10.9 | 58.4 | 94.2 |
| | 0.001 | - 9.8 | 72.2 | 92.3 |
| | 0.115 | -10.1 | 84.4 | 95.1 |
| | 0.215 | - 9.9 | 113.5 | 95.1 |
| S2 | -0.756 | -11.8 | 40.9 | 99.2 |
| | -0.639 | -10.5 | 28.6 | 99.6 |
| | -0.520 | -10.3 | 39.5 | 99.0 |
| | -0.404 | -11.8 | 55.9 | 97.8 |
| | -0.302 | -13.9 | 61.8 | 98.7 |
| | -0.202 | -14.4 | 88.3 | 98.1 |
| | -0.101 | -14.5 | 127.5 | 99.0 |
| | 0.000 | -13.7 | 187.4 | 97.6 |
| | 0.083 | -13.3 | 236.6 | 96.0 |
| S3 | -0.944 | -13.1 | 80.9 | 97.1 |
| | -0.823 | - 9.0 | 35.9 | 99.3 |
| | -0.709 | - 9.6 | 41.0 | 95.5 |
| | -0.590 | -10.3 | 32.7 | 98.6 |
| | -0.473 | - 9.7 | 45.0 | 96.5 |
| | -0.234 | - 9.4 | 58.0 | 91.8 |
| | -0.119 | -10.9 | 87.3 | 92.8 |
| | 0.000 | - 9.8 | 103.6 | 93.3 |
| | 0.084 | - 8.8 | 110.6 | 98.5 |
| | 0.165 | - 8.0 | 149.4 | 95.4 |
| S5 | -0.775 | - 9.1 | 34.3 | 99.0 |
| | -0.708 | - 8.7 | 27.1 | 99.0 |
| | -0.592 | - 7.9 | 32.2 | 98.7 |
| | -0.472 | - 7.6 | 44.5 | 95.6 |
| | -0.359 | - 7.8 | 53.6 | 93.9 |
| | -0.238 | - 7.9 | 74.9 | 90.3 |
| | -0.122 | - 8.7 | 85.5 | 94.4 |
| | 0.001 | - 8.1 | 101.9 | 92.8 |
| | 0.081 | - 7.7 | 120.5 | 93.8 |
| S7 | -0.509 | -13.7 | 52.2 | 98.4 |
| | -0.424 | -11.6 | 47.6 | 99.4 |
| | -0.341 | -10.7 | 63.2 | 98.7 |
| | -0.256 | -11.1 | 53.2 | 98.2 |
| | -0.171 | -12.7 | 45.9 | 99.7 |
| | -0.087 | -13.5 | 71.3 | 98.6 |
| | 0.002 | -15.7 | 75.2 | 99.8 |
| | 0.095 | -14.1 | 145.5 | 98.8 |
| | 0.197 | -14.3 | 221.4 | 98.3 |

**Table 5.5:** The slopes, intercepts and VAFs by a linear regression model between K and active plantarflexor torque at each mean position. The parameters are listed in an order ranging from maximum plantarflexion to maximum dorsiflexion.

to the passive condition and are similar to the intercepts listed in Table 5.5.

There was very little change in the magnitude of the K/active plantarflexor slopes as ankle mean position changed. NP slopes varied between 7.8 and 11.8 Nm/rad/Nm; the smallest slope was 7.6 Nm/rad/Nm (Subject S5 at -.47 rad) whereas the largest slope was 17.3 Nm/rad/Nm (Subject S1 at -.83 rad). Individual subjects showed a percent change in slope magnitude that varied from 120 to 220% over the ROM. There was, however, no systematic position-related change in these slopes.

There were several interesting differences between the behaviour of K during active plantarflexor and dorsiflexor contractions. First, the VAF by a linear relation between K and active torque was generally larger during plantarflexor contractions. Second, NP slopes were generally larger than those obtained at the same angle during dorsiflexor contractions. Finally, there was considerably less mean position-related variation in the magnitude of the K/active torque slopes during plantarflexor contractions than during dorsiflexor contractions. The later two points are illustrated in Fig. 5.12 which shows the mean K/active torque slope ±1 SD for each of the five subjects. The K/dorsiflexor slope means, shown in Fig. 5.12 (top graph), are smaller than the K/plantarflexor slope means, shown in Fig. 5.12 (bottom graph). In contrast, the K/dorsiflexor slope SDs are larger than the K/plantarflexor slope SDs.

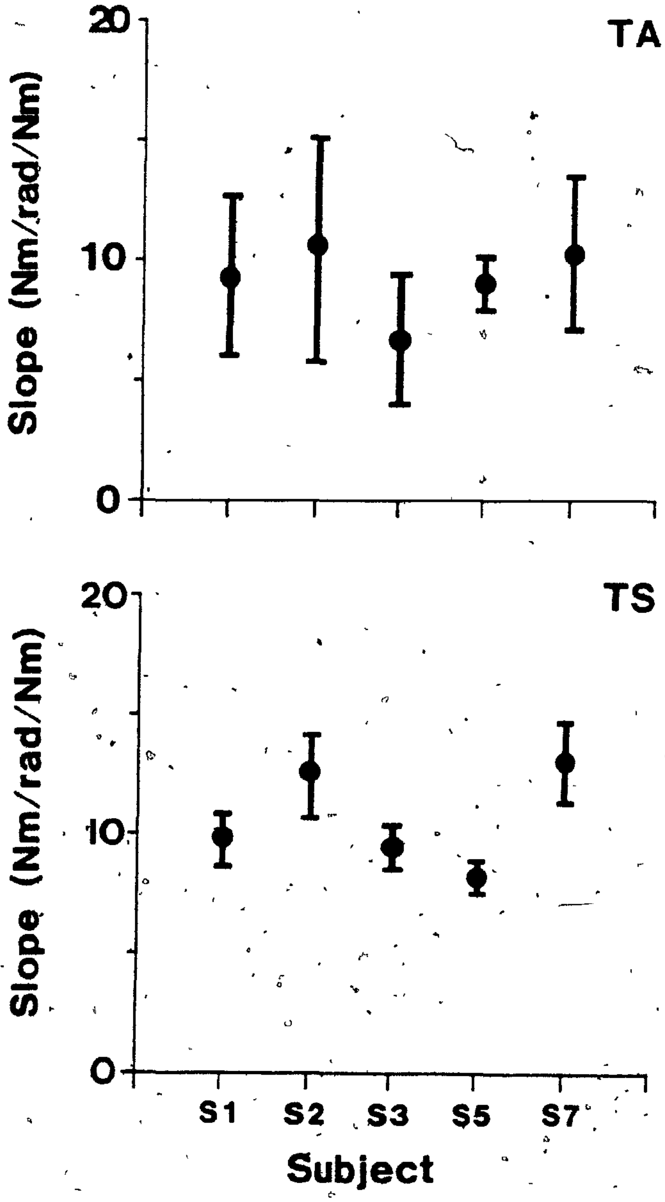


**Fig. 5.12:** The mean ± 1 SD of the K/torque slopes over the tested ankle angles for each of the five subjects. These data are for dorsiflexor (top graph) and plantarflexor (bottom graph) contractions. (Subjects are identified by a number which denoted the sequence in which they were tested; Subjects S4 and S6 did not participate in this study.)

## DISCUSSION

These experiments were designed to examine the static and dynamic behaviour of ankle joint mechanics in the presence of simultaneous changes in mean position and in actively generated torques. The major results relate to the position dependence of the static torque/EMG and K/active torque relations.

It was first demonstrated that the static torque/EMG relation was linear at all ankle angles. Although ankle mean position appeared to alter this relation, the effect was neither large nor systematic. It was then demonstrated that the K/active torque relation was also linear at all ankle angles. There was very little change in the K/active plantarflexor torque gain over the ROM. The position dependent changes in the K/active dorsiflexor torque gain were somewhat larger; the ankle joint tended to be stiffer during dorsiflexor contractions at the more dorsiflexed angles although this effect was not entirely consistent. Thus, contrary to the results from Chapter 4 where it was demonstrated that ankle mean position had a large and predictable effect on the passive mechanics, the present findings indicate that the position dependence of the active mechanics is relatively small and variable.

The first result indicates that good estimates of active joint torque can be obtained if the agonist and antagonist EMGs are known. The second result suggests that very good estimates of K can be obtained if the total (passive plus active) joint torque is known. Thus knowledge of ankle mean position is not essential for the characterization of the static relation between torque and EMG. Nor is it required for the identification of the joint mechanics provided that the total joint torque is known. Finally a number of the key differences which distinguish the behaviour of the ankle joint during dorsiflexor and plantarflexor contractions have been delineated. The functional implications of these results are discussed below.

## The Constant-Torque Paradigm

A constant-torque paradigm was employed in these experiments, i.e. this study was designed to ensure that the subjects maintained the same level of active torque throughout the ROM. Since the magnitude of the TA MVC has been shown to increase (Marsh et al, 1981) and the magnitude of the TS MVC has been shown to decrease (Sale et al., 1982) as the ankle is plantarflexed, the use of this paradigm implied that each torque represented a different percent of the MVC. For example, a given dorsiflexor torque represented a much larger percentage of the TA MVC at extreme dorsiflexion than it did at extreme plantarflexion.

An alternate exerimental paradigm, one in which the subject would have been required to track torques that comprised the same percentage of the MVC at all mean positions, was also considered. This paradigm has the advantage that the subjects' effort would have remained relatively constant over the ROM. This approach was, however, rejected for two reasons. First, repeated determinations of the MVC within the same experimental session would have been required since it was not possible to accurately replicate the ankle reference-position once the subject was released from the foot cast. This would have been a fatiguing, and therefore unacceptable, procedure given the duration of the already lengthy experiment.

Second, although the MVC may be reliably measured on successive occasions if care is taken to follow certain guidelines (Kroemer & Marras, 1980), this variable is neither well-defined nor completely objective. Moreover, there is some indication that complete motor unit activation is not achieved in some muscles (Belanger & McComas, 1981). For these reasons the constant-%MVC paradigm was not used.

## Physiological Correlates of Ankle Mean Position

### Voluntary and Electrically Evoked Torque

The magnitude of the voluntarily generated or electrically evoked ankle torque has been found to change as a function of the joint angle in previous studies. Since a constant-torque paradigm was employed in the present study, position-related changes in the torque have relevance to the interpretation of our results.

TS twitch torques were larger when the ankle was dorsiflexed than when it was plantarflexed (Gottlieb & Agarwal, 1978; Sale et al., 1982) as were torques generated by 10 Hz tetanies and by MVCs (Sale et al., 1982). These increases were not strictly monotonic but appeared to plateau near 0.15 rad. In contrast, the magnitude of TA torques generated by twitches, tetanies of varying frequencies and MVCs increased as the ankle was plantarflexed (Marsh et al., 1981). For MVCs and at the higher stimulating frequencies, the torque appeared to be maximal at about -.1 rad, whereas for the twitches and lower frequency levels, the increase in torque was monotonic (Marsh et al., 1981).

In the present study, the magnitude of the active torque at each of the target levels was maintained constant over the ROM. This resulted in values of K that remained relatively constant at all ankle angles. The question now arises as to whether this result contradicts the above findings which appear to demonstrate that TA and TS contractile properties are modified by ankle mean position. There are two responses to this question.

First, as suggested by Gottlieb & Agarwal (1978), who also employed the constant-torque paradigm, the invariance of K with respect to ankle angle does not denote a lack of change in the contractile properties. Rather, for example, possible decreases in the number of attached cross-bridges at the extremes of the ROM may have been compensated for by concomitant increases in the recruitment or firing rate of the relevant motoneurones.

Second, although the evidence to date suggests that there are some changes in the contractile properties of the ankle dorsi- and plantarflexors, these studies are by no means exhaustive and any conclusions regarding the extent and significance of the presumed sources of these changes (e.g. muscle length) are premature. Our knowledge of other possible sources of the observed changes in torque is rudimentary and theoretical considerations suggest that changes in muscle length are not large enough to account for the experimentally observed changes.

Nevertheless, the present inability to resolve this issue does not detract from the major result of this study. Namely, provided that the total joint torque is known the stiffness of the ankle joint can be reliably and accurately predicted regardless of whether it is generated by passive or active processes.

## EMG

Position-related changes in the EMG are also of interest since the effect of mean ankle angle on the static torque/EMG relation was also examined. There is some indication that the surface EMG changes when a joint is rotated although this issue is complicated by the use of a wide variety of different methodologies. The most direct studies have endeavored to determine whether these changes result from alterations of muscle length. When frog sciatic nerve was supramaximally stimulated with single shocks, the muscle action potential (MAP) amplitude was smaller when the muscle was shorter than rest length than when it is stretched beyond its resting length (Ralsten & Libet, 1953). This effect is seen only when the nerve is stimulated and not when the muscle is stimulated directly. These changes in MAP amplitude appeared to be caused by differences in the extent of muscle fiber recruitment. That is, a supramaximal stimulus via the nerve of an unstretched muscle did not appear to activate all fibers; however all fibers were activated when the muscle was stretched (Ralsten & Libet, 1953). With bipolar electrodes sewn to the surface of a muscle belly, Lewis et al. (1972) recorded an increase in EMG with lengthening for six motor units and a decrease for one unit. In contrast, Stephens et al. (1975) reported a decrease in EMG with lengthening. In both experiments great care was taken to minimize experimental artifact by preventing electrode slippage. Thus the results from isolated muscle experiments provide no consensus.

Previous studies have shown very little change in the surface EMG over muscle lengths that were associated with a 30% change in the force generated by the adductor pollicis (Caferelli & Bigland-Ritchie, 1979) and biceps brachii maximal EMGs showed little systematic change with large alterations of the elbow angle (Komi & Buskirk, 1972).

TA EMG recorded from surface electrodes has been shown to decrease as the muscle was stretched (Inman et al., 1952; Marsh et al., 1981). Both soleus and gastrocnemius EMGs recorded from wire electrodes decreased as the ankle was dorsiflexed though at different rates (Herman & Bragin, 1967). In contrast, the TS EMG recorded during maximal contractions increased as the ankle was dorsiflexed (Sale et al., 1982). However, this result may be explained by the concomitant increase in the M-wave since the EMG:M ratio actually decreased. Presumably, at least some of the EMG changes were related to skin movement (Sale et al., 1982).

The results from the present study indicate that such effects are indeed variable. Since the magnitude of the joint torque remained constant at all ankle angles and the relation between EMG and torque was linear regardless of mean position, changes in the slopes of these curves would indicate alterations in the EMG/joint position relation. The results of the multiple linear regression analysis showed that the effect of joint position was relatively small and quite variable. It is highly likely that these effects were artifactual and related to alterations of electrode properties or location as the ankle joint was rotated over its ROM. Since it is virtually impossible to eliminate such artifacts the issue of the position dependence of surface EMG must remain open.

## Comparison of the Ankle Dorsiflexors and Plantarflexors

There is evidence from this study that TA and TS behave differently. First, the VAF by a linear relation between K and active torque was generally larger during plantarflexor contractions. Second, the NP and other ankle angle K/TS torque slopes were generally larger than those measured at the same angles during dorsiflexor contractions. Finally, there was considerably less mean position-related variation in the magnitude of the K/active torque slopes during plantarflexor contractions than during dorsiflexor contractions. Differences in TA and TS reflex behaviour have also been observed (Kearney & Hunter, 1983; 1984) but these will be discussed in Chapter 6.

Differences in the mechanical and reflex behaviour of the dorsiand plantarflexors are not surprising given the differences in their functional requirements (Herman & Bragin, 1967). The TS are physiological extensors that have a primarily static role in posture; TA is a phasic muscle that is quiet during normal standing (Soames & Atha, 1981). Moreover there are differences in the morphological (Close, 1956), histochemical (Johnson et al., 1973) and contractile (Belanger et al., 1983) properties of these two antagonist muscles. For example, in comparison to the ankle plantarflexors, the dorsiflexor twitches and MVCs were smaller and were more susceptible to fatigue during isometric contractions (Belanger et al., 1983). Although the MVC generated by both muscles changed with joint angle, the change in the TS MVC magnitude was considerably larger than that of the TA MVC (Sale et al., 1982; Marsh et al., 1981).

## The Relative Contribution of Passive and Active Mechanics

Stiffness can be increased by contraction of the agonist muscle (Hunter & Kearney, 1982) and a certain energy cost will be associated with this active muscle process. Still more costly is the increase in stiffness that arises when both the agonist and antagonist muscles contract simultaneously (Hunter et al., 1983). In contrast, the large increases in stiffness which result when the joint angle is passively altered require little energy.

Passively-induced increases in joint stiffness are therefore more efficient. Moreover, they are not insignificant in comparison to the effects of an active contraction. This is demonstrated by an examination of the magnitude of the stiffness that can be achieved by passive and active processes. The results of this comparison are presented in Table 5.6. The total increases in K that were generated when the relaxed ankle was rotated from the NP to extreme dorsiflexion (ΔPK) are listed for each subject. The value of the passive plantarflexor torque (ΔPTOR) that was associated with these changes in joint position are also listed.

The data from active contractions maintained at the NP were then examined to determine comparable increases in active plantarflexor K (ΔAK). These values and the corresponding active plantarflexor (ΔATOR) torques are listed in Table 5.6. Also listed are the percents of the NP MVC which corresponded to these torques.

The torques generated during passive rotation to maximum dorsiflexion varied from -6 to -12.4 Nm. These torques were associated with values of K that varied from 72.5 to 207.3 Nm/rad. Comparable values of K (84.2 to 207 Nm/rad), generated during active plantarflexor contractions were associated with active plantarflexor torques that varied between -5.7 and -14.4 Nm. These torques corresponded to contraction levels that ranged from 10 to 20% of the MVC. Although contractions of this magnitude can be maintained for long periods without apparent fatigue, the generation of comparable levels of joint stiffness by passive processes would result in a moderate energy saving.

The importance of the passive structures is apparent when the behaviour of the ankle joint during postural sway is considered. The

| SUBJECT | PASSIVE | | ACTIVE | | |
|---|---|---|---|---|---|
| | ΔPK | ΔPTOR | ΔAK | ΔATOR | %NP MVC |
| S1 | 72.5 | -6.0 | 84.2 | -5.7 | 10 |
| S2 | 148.5 | -12.4 | 177.7 | -14.4 | 20 |
| S3 | 127.7 | - 9.9 | 151.0 | -11.5 | 20 |
| S5 | 114.8 | -10.1 | 143.0 | -14.1 | 20 |
| S7 | 207.3 | -10.5 | 207.0 | -9.6 | 10 |

**Table 5.6:** A comparison of the passive and active torques generated to achieve similar values of K for the five subjects. ΔPK and ΔPTOR represent the increase in stiffness and torque associated with changes in mean position from the NP to maximum dorsiflexion. ΔAK represents comparable increases in active plantarflexor K and ΔATOR represents the corresponding active plantarflexor torque. The %NP MVC denotes the tonic level corresponding to the active torques.

center of gravity falls about 5 mm anterior to the ankle joint (Hellebrandt, 1938) creating a dorsiflexor torque of approximately 50 Nm (Boussenna et al., 1982) which must be balanced by an equivalent plantarflexor moment. This moment is generated either by contraction of TS, by stretch of the non-contractile joint tissues or by some combination of these active and passive processes (Smith, 1957). Although the ankle oscillates by less than .1 rad during quiet stance this motion occurs about the 0 rad position (Murray et al., 1975). At this angle the joint is quite stiff (as great as 200 Nm/rad) and further anterior sway is retarded. Furthermore, the large passive torque (as high as 12 Nm) serves to plantarflex the limb about the fixed foot with the result that only small, periodic bursts of TS are required to maintain a stable posture.

# CHAPTER 6: STRETCH REFLEX DYNAMICS

## SUMMARY

The purpose of this study was to examine the effect of changes in mean ankle position on the human ankle stretch reflexes during tonically-maintained contractions held over most of the range of motion. The ankle was placed at randomly selected mean positions, target levels of TS or TA tonic contractions were generated and the ankle was displaced by small amplitude, stochastic perturbations. System identification techniques were used to identify the stretch reflex dynamics at each tonic level/ankle angle combination. The TS stretch reflex was characterized as a unidirectional, velocity-sensitive impulse response function whereas the TA stretch reflex was characterized by a linear impulse response function between ankle velocity and TA EMG.

TS stretch reflexes showed a strong dependence on ankle position while TA stretch reflexes did not. Thus, the TS stretch reflex magnitude increased greatly as the ankle was progressively dorsiflexed. In contrast, mean ankle position had only a minor effect on the TA stretch reflex magnitude. The results suggest that the position-dependent facilitation of the TS stretch reflex is not due to changes in the level of skeletal motoneuron excitability. Rather, it may be accounted for by mechanisms that modulate the efficacy of the stochastic ankle perturbation. Such mechanisms could include position-induced: modulation of monosynaptic and polysynaptic afferent input to skeletal motoneurons; alterations in the extent of fusimotor drive; and changes in the transmission of the joint perturbation to spindle receptors. Such mechanisms are discussed in terms of the different behaviour of TS and TA stretch reflexes. Finally, the functional significance of position-dependent reflex responses are considered.

## INTRODUCTION

Although the ankle joint angle varies substantially during functional activities, little is known about the effect this has on stretch reflexes at the ankle. H-reflex and microneurographic techniques have been used extensively to measure reflex function in intact man (e.g. Paillard, 1960; Hagbarth & Vallbo, 1968) and to delineate the effect of changes in the tonically-maintained ankle position on monosynaptic (e.g. Mark et al., 1968) and polysynaptic (e.g. Delwaide, 1973) reflexes and the afferents that mediate these responses (e.g. Vallbo, 1974). However, the H-reflex technique tests for sub-threshold changes in motoneurone excitability using a highly non-physiological stimulus (Hugon, 1973) and its relevance to physiological stimuli remains uncertain (Burke, 1983). On the other hand, microneurography is currently restricted to the study of relatively small contraction levels and joint displacements (Prochazka & Hulliger, 1983).

The range of motion of the ankle can be as large as 1.5 rad

(Boone & Azen, 1979) and the results of Chapter 4 indicate that the behaviour of the joint (e.g. the passive torque and passive mechanics) at mid-range angles is quite different than it is at the extremes of the ROM. Moreover, changes in ankle position are often accompanied by tonic contractions and such contractions appear to significantly modify the position-dependent reflex effects (Mark et al., 1968; Delwaide & Hugon, 1969). Thus it seems important to study stretch reflex responses to physiological stimuli over a functionally significant range of positions and torques.

The specific objective of the work presented in this chapter was to use quantitative techniques to characterize triceps surae and tibialis anterior stretch reflexes as functions of joint angle. This was accomplished by determining the dynamic modulation of TS and TA EMGs in response to small stochastic perturbations of the ankle about different mean ankle angles and during different levels of tonic activity. This extends the results of previous investigations in which the effects of tonic activity and stretch amplitude were determined (Kearney & Hunter, 1983; 1984) and is the first time that system identification methods have been used to examine the position dependence of the human stretch reflex function.

## METHODS

The general methods and specific procedure related to the second series of experiments have been described in Chapters 2 and 5 respectively. Details pertinent to the analysis of the stretch reflex dynamics are provided below.

## Identification of Reflex Dynamics

In accordance with previous results from this laboratory (Kearney & Hunter, 1983; 1984), the TS and TA stretch reflexes were determined by identifying the linear dynamic relation between ankle angular velocity and rectified, smoothed EMGs. These reflex impulse response functions (RIRFs) were calculated with an algorithm that was based on the solution of a matrix equation involving the input auto-correlation function and the input/output cross-correlation function (Hunter & Kearney, 1983b).

TS stretch reflexes were characterized by the RIRF determined between positively rectified ankle angular velocity and TS EMG. A typical TS RIRF is shown in Fig. 6.1 (top graph). It had a characteristic biphasic pattern in which a large negative (excitatory) peak was followed by a smaller positive (inhibitory) peak (Kearney & Hunter, 1983). The negative peak, which due to the sign conventions, corresponded to an increase in EMG activity in response to muscle stretch, occurred at a latency of about 40 ms. The peak-to-peak response is labelled in Fig. 6.1 (top graph) as TSP.

TA stretch reflexes were represented by the RIRF identified between ankle angular velocity and TA EMG. As shown in Fig. 6.1 (bottom graph), this function had a characteristic triphasic response with two negative (excitatory) peaks separated by a positive (inhibitory) peak (Kearney & Hunter, 1984). As above, a negative peak

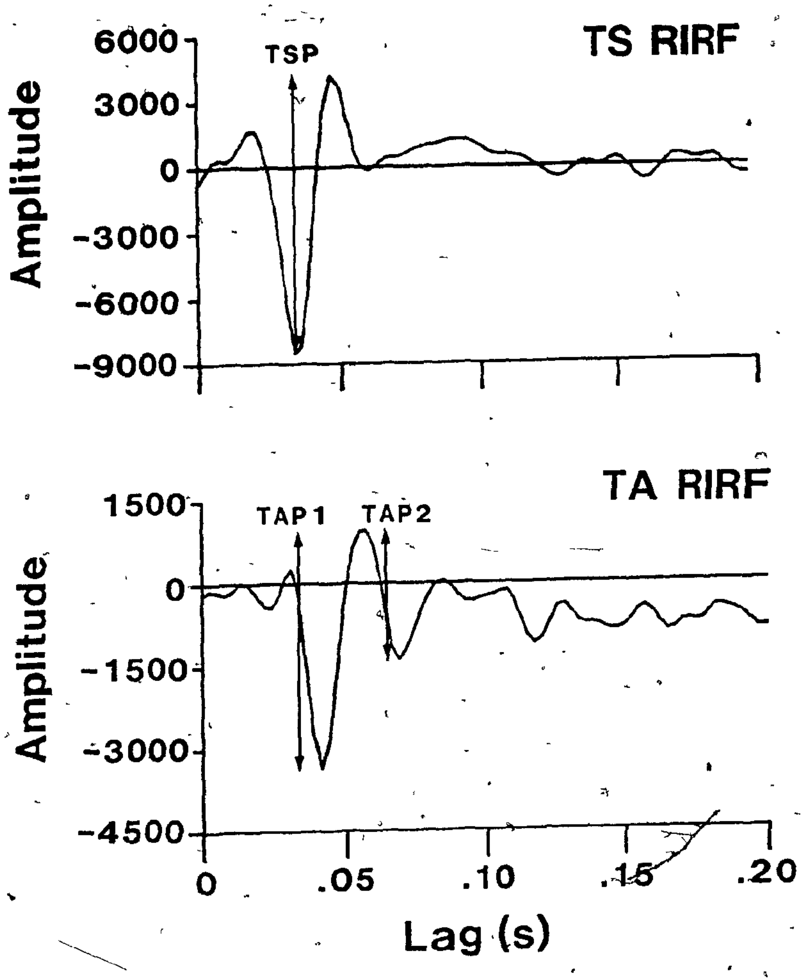


**Fig. 6.1:** Reflex impulse response functions (RIRFs) identified between positively rectified angular velocity and TS EMG (top graph) and between angular velocity and TA EMG (bottom graph). Each RIRF was determined during a 30%MVC tonic contraction with the ankle at the NP (-.47 rad). The peak-to-peak amplitudes used to describe the RIRF magnitudes are labelled, TSP, TAP1 and TAP2. (Subject S5)

in the TA RIRF corresponded to an increase in EMG activity in response to muscle stretch. The first negative peak had a latency of about 40 ms and the second negative peak at about 70 ms. The two peak-to-peak responses are labelled in Fig. 6.1 (bottom graph) as TAP1 and TAP2.

## RESULTS

### Reflex Impulse Response Functions

The VAF by TA RIRFs and TS RIRFs ranged from 40 to 50% and did not vary with ankle position. These VAFs were 10 to 20% lower than those obtained in previous studies (Kearney & Hunter, 1983; 1984). This is undoubtedly due to the fact that in the present experiments five stimulus sequences were averaged whereas previously 25 repetitions were used; the magnitude of the VAF is dependent upon the number of responses in the ensemble average (cf. Kearney & Hunter, 1984, Fig. 3b). The present five-fold reduction in the number of stimulus repetitions was required in order to avoid subject fatigue; the use of 25 stimulus sequences at each test condition would have lengthened the experiment beyond the subjects' endurance. Consequently, the TA and TS RIRFs identified in the present study were somewhat noisier than those obtained previously. Note, however, that the maximum likelihood estimation technique provided good estimates of the RIRF shape even when the signal to noise ratio was low (cf. Kearney & Hunter, 1984, Fig. 4). In the present study, the characteristic triphasic and biphasic patterns were clearly identifiable during tonic dorsiflexor or plantarflexor contractions. This was not the case during the passive condition. The RIRF amplitude is proportional to the level of tonic activity and hence was very small when the muscles are at rest (Kearney & Hunter, 1983; 1984). It was therefore not possible to characterize the peak-to-peak responses of the RIRFs in the absence of muscle activity.

## Relation between Ankle Angle and TS Reflexes

A sample set of TS RIRFs, determined with the ankle at different mean positions but while the same level of active torque was maintained (30% NP TS MVC), is shown in Fig. 6.2. The effect of mean ankle position on the TS RIRF is evident. The reflex magnitude became progressively larger as the ankle was dorsiflexed from -.36 rad (near the NP, bottom trace) to .21 rad (near maximum dorsiflexion, top trace). In contrast, the TS RIRF shape and latency remained the same.

The effect of mean ankle position on the TS RIRF magnitude for all tonic levels is shown in Fig. 6.3. Each square represents the TSP measured at a particular ankle angle and contraction level; TSPs obtained at the same active torque level have been joined to form iso-active-torque contours. It is apparent that the position-dependent change in reflex magnitude was similar for all contraction levels. When the ankle was plantarflexed, the TSP remained relatively constant. However, it became progressively larger as the ankle was dorsiflexed beyond the NP reaching a maximum at the extreme of dorsiflexion. For the data shown in Fig. 6.3, the TSP at maximum dorsiflexion during the 30% NP TSMVC torque level was 17 times

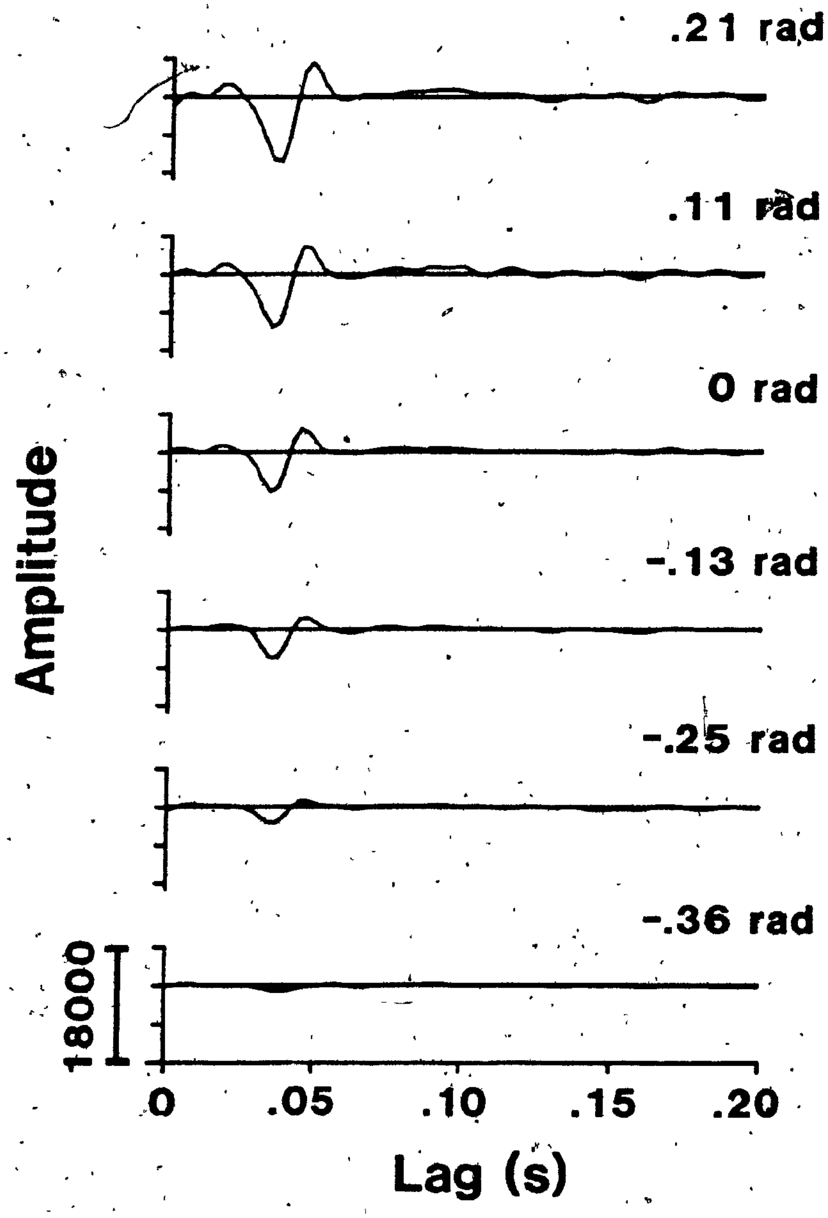


Fig. 6.2: TS-RIRFs determined during a 30% NP TS MVC at ankle angles ranging from -.36 rad (bottom trace) to .21 rad (top trace). The amplitude scale is the same for all six traces. (Subject S1)

larger than its NP value. For other subjects the increase varied from a factor of 4 to a factor of 23. It is also apparent that TSP increased with increasing EMG although this effect is evidently smaller than that due to ankle angle.

Although the shapes of the iso-active-torque curves shown in Fig. 6.3 were similar, their offsets increased with mean active torque. This indicated that the TSP was also modified by the level of tonic activity. The combined effects of mean ankle position and level of contraction on the TS RIRF magnitude were therefore investigated. The TSP data were fitted to a multiple linear regression model having TS EMG and ankle angle as independent variables (denoted as the EMG-angle model). Since the TSPs were minimally modified by ankle plantarflexion, only values obtained at mean positions beyond the NP threshold angle were fitted to the model.

The results of this analysis, listed in Table 6.1 for the five subjects, demonstrated that between 62 and 94% of the variance in the TS reflex magnitude was accounted for by these two parameters. The results of a linear regression analysis between the pooled TSP and TS EMG data are also listed in Table 6.1. The VAF by this model (denoted as the EMG-model) ranged from 1 to 36% which was considerably less than the VAF by the EMG-angle model.

This does not, however, indicate that the effect of TS EMG on the TS reflex magnitude was unimportant. As demonstrated in previous experiments, the relation between TSP and TS EMG was highly linear when the mean ankle position was fixed near the NP (Kearney & Hunter,

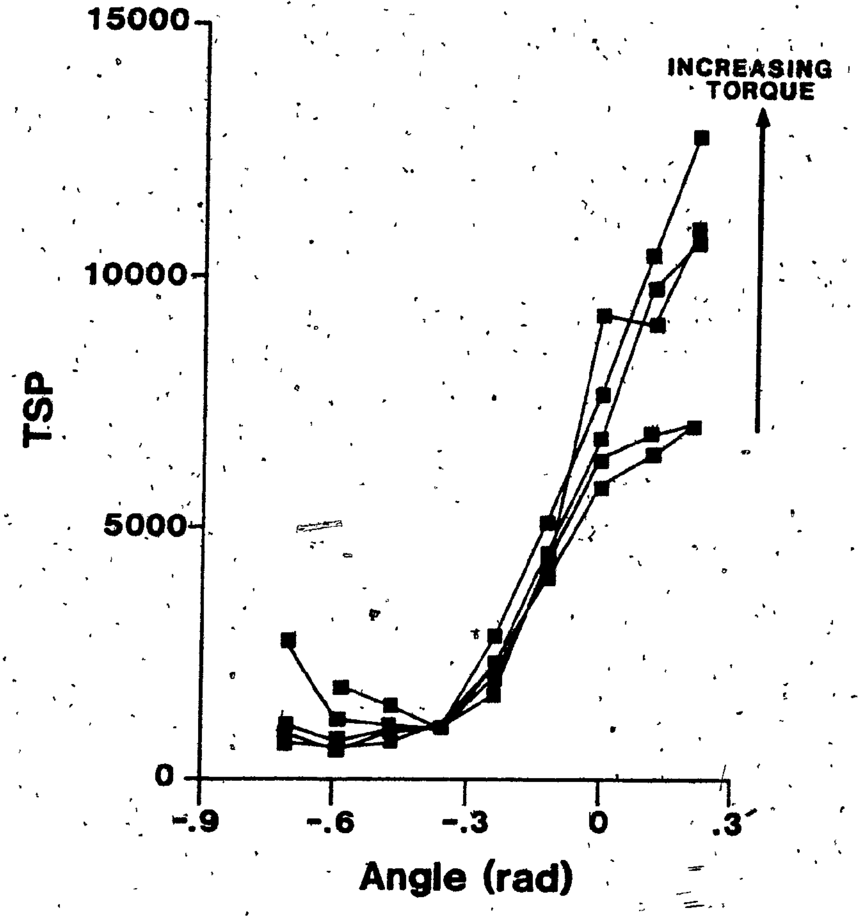


Fig. 6.3: TSP plotted as a function of ankle angle. Each square represents the TSP determined at a particular tonic level and ankle angle. TSPs determined at the same tonic level are joined. (Subject S7)

| SUBJECT | EMG-ANGLE MODEL | | | EMG MODEL |
|---|---|---|---|---|
| | TS EMG Coeff. | ANGLE Coeff. | VAF (%) | VAF (%) |
| S1 | 92.8 | 5486 | 92.3 | 24.4 |
| S2 | 28.4 | 5122 | 62.3 | 1.0 |
| S3 | 78.5 | 1231 | 93.9 | 36.4 |
| S5 | 78.3 | 21139 | 89.2 | 22.8 |
| S7 | 61.9 | 10902 | 85.1 | 21.4 |

**Table 6.1:** Results of a multiple linear regression analysis between TS EMG, ankle angle and TSP (the EMG-angle model). The TS EMG coefficients, the angle coefficients, the intercepts and the VAFs by this model are listed for all subjects. The VAF by a linear regression analysis between TS EMG and TSP (the EMG model) is also listed.

1983). A series of linear regression analyses between the TSP and TS EMG data were therefore carried out to determine whether this relation persisted at all ankle angles. As demonstrated by the VAFs listed in Table 6.2, the relation between TSP and TS EMG was linear at most mean positions. The variance in TSP accounted for by the TS EMG was generally greater than 80% although it did occasionally decline to much lower values.

The results of these analyses revealed a second important finding. Namely, the gain of the TSP/TS EMG relation was dependent upon ankle mean position; the TSP/TS EMG slopes became larger as the ankle was dorsiflexed. Values ranging from 15 to 42 μVs/rad/μV were generated at the NP. These small slopes contrast to values ranging from 137 to 255 μVs/rad/μV at extreme dorsiflexion.

These effects are illustrated in Fig. 6.4. The data plotted in Fig. 6.3 have been redrawn to show the relation between TSP and TS EMG for mean positions ranging from maximum plantarflexion to maximum dorsiflexion. For clarity, data from only seven of the nine ankle angles are shown and the regression lines have been included in the figure. It is evident that the relation between TSP and TS EMG was linear and that the slope of this relation became steeper as the ankle was dorsiflexed.

To summarize, the TS stretch reflex magnitude was altered by both ankle angle and agonist EMG. Whereas the position-dependent effects were independent of the level of tonic activity, the EMG-dependent effects were modulated by mean ankle position.

| SUBJECT | ANGLE (rad) | SLOPE (μVs/rad/μV) | VAF (%) |
|---|---|---|---|
| S1 | -0.710 | 23.4 | 94.7 |
| | -0.591 | 21.8 | 96.9 |
| | -0.474 | 15.1 | 76.3 |
| | -0.358 | 3.4 | 34.4 |
| | -0.239 | 24.9 | 19.0 |
| | -0.121 | 45.9 | 20.1 |
| | 0.001 | 152.1 | 96.3 |
| | 0.115 | 207.6 | 99.9 |
| | 0.215 | 172.1 | 97.4 |
| S2 | -0.756 | 25.0 | 89.5 |
| | -0.639 | 20.3 | 79.8 |
| | -0.520 | 10.8 | 20.9 |
| | -0.404 | 50.6 | 81.7 |
| | -0.302 | 85.1 | 98.5 |
| | -0.202 | 91.1 | 89.0 |
| | -0.101 | 71.4 | 83.0 |
| | 0.000 | 112.0 | 90.2 |
| | 0.083 | 144.3 | 90.1 |
| S3 | -0.823 | 7.5 | 95.0 |
| | -0.709 | 7.9 | 91.3 |
| | -0.590 | 7.6 | 50.5 |
| | -0.473 | 16.2 | 91.2 |
| | -0.234 | 223.4 | 56.6 |
| | -0.119 | 306.2 | 98.4 |
| | 0.000 | 270.7 | 89.4 |
| | 0.084 | 384.1 | 79.8 |
| | 0.165 | 254.9 | 97.6 |
| S5 | -0.775 | 42.7 | 96.1 |
| | -0.708 | 45.8 | 98.4 |
| | -0.592 | 25.6 | 96.9 |
| | -0.472 | 72.7 | 88.1 |
| | -0.359 | 40.9 | 68.3 |
| | -0.238 | 54.3 | 2.5 |
| | -0.122 | 65.8 | 29.7 |
| | 0.001 | 93.4 | 73.6 |
| | 0.081 | 137.2 | 96.3 |
| S7 | -0.509 | 34.6 | 99.3 |
| | -0.424 | 46.3 | 98.0 |
| | -0.341 | 42.2 | 93.5 |
| | -0.256 | 62.1 | 72.9 |
| | -0.171 | 105.7 | 90.1 |
| | -0.087 | 127.7 | 91.8 |
| | 0.002 | 128.9 | 92.7 |
| | 0.095 | 178.6 | 97.6 |
| | 0.197 | 220.8 | 99.0 |

**Table 6.2:** Linear regression analysis of the relation between TSP and TS EMG for all subjects. The parameters are ordered ranging from maximum plantarflexion to maximum dorsiflexion.

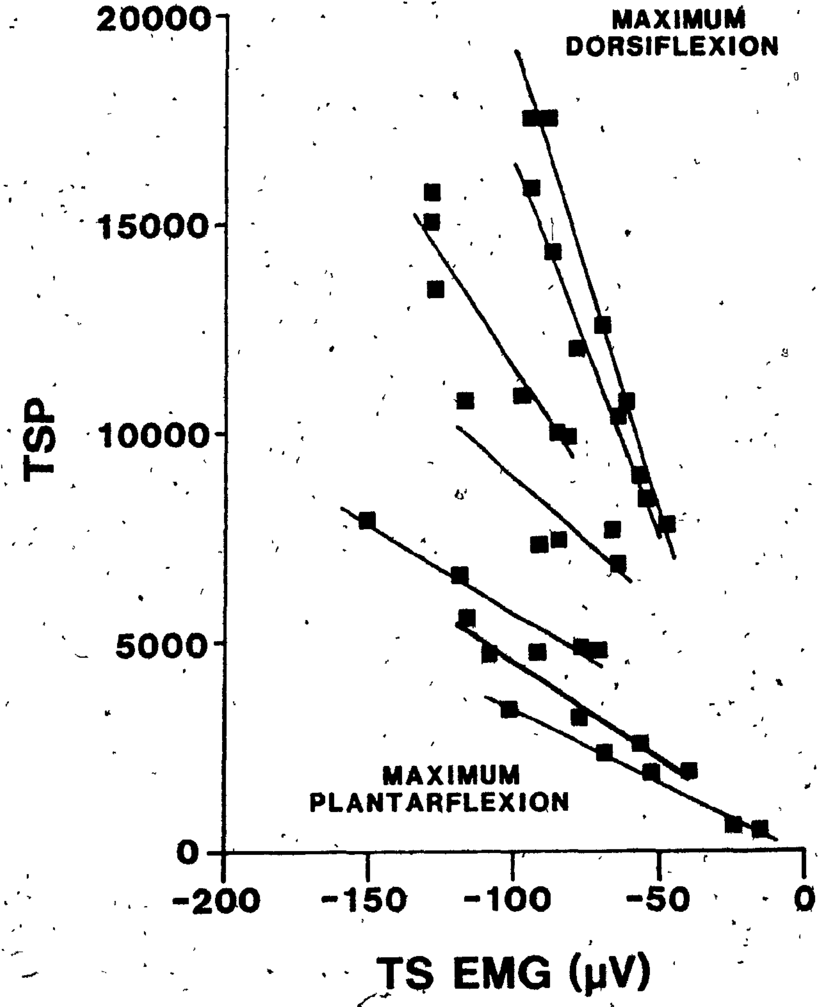


**Fig. 6.4:** Data from Fig. 6.3 replotted to illustrate the relation between TSP and TS EMG. Lines represent the best least squares fit between TSP and TS EMG at each angle ranging from maximum plantarflexion (bottom trace) to maximum dorsiflexion (top trace). (Subject S7)

## Relation between Ankle Angle and TA Reflexes

The effect of mean ankle position and level of tonic contraction on the TA stretch reflex magnitude were also investigated. A sample set of reflex responses, determined at one active torque level (30% NP TA MVC), are shown in Fig. 6.5. These TA RIRFs were obtained at six different ankle angles ranging from .21 rad (near maximum dorsiflexion, top trace) to -.36 rad (near the NP, bottom trace). The shape and latency of these functions did not change with ankle angle. Moreover, in contrast to TS, changes in the TA reflex magnitude were relatively small and variable.

The comparative uniformity of the TA RIRF magnitude with respect to ankle angle is illustrated in Fig. 6.6. Each square represents the TAP2 measured at one angle and tonic level; the TAP2s obtained at the same active torque level have been joined to form five iso-active-torque contours. Two important observations follow from these curves. First, although there appeared to be a small increase in TAP2 as the ankle was dorsiflexed, this trend was small relative to the general variability. Second, the TAP2 contours became more distinct as the level of tonic activity increased.

These observations were considered further by examining the combined effects of mean ankle position and level of contraction on the TA RIRF magnitude. The TAP2 data were fitted to a multiple linear regression model having TA EMG and ankle angle as independent variables (denoted as the EMG-angle model). As there did not appear to be a threshold angle (compare Figs. 6.2 and 6.5) the entire data

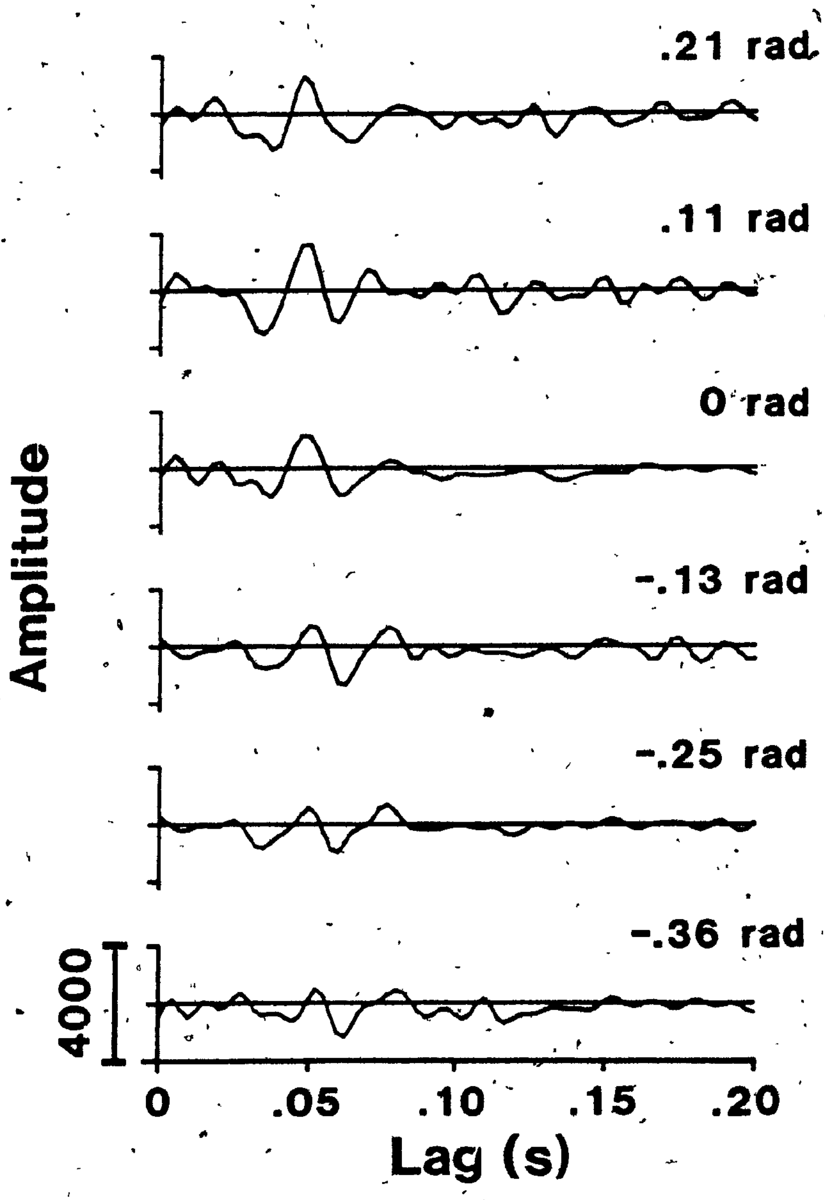


Fig. 6.5: TA RIRFs determined during a 30% NP TA MVC at ankle angles ranging from -.36 rad (bottom trace) to .21 rad (top trace). The amplitude scale refers to all six traces. (Subject S1)

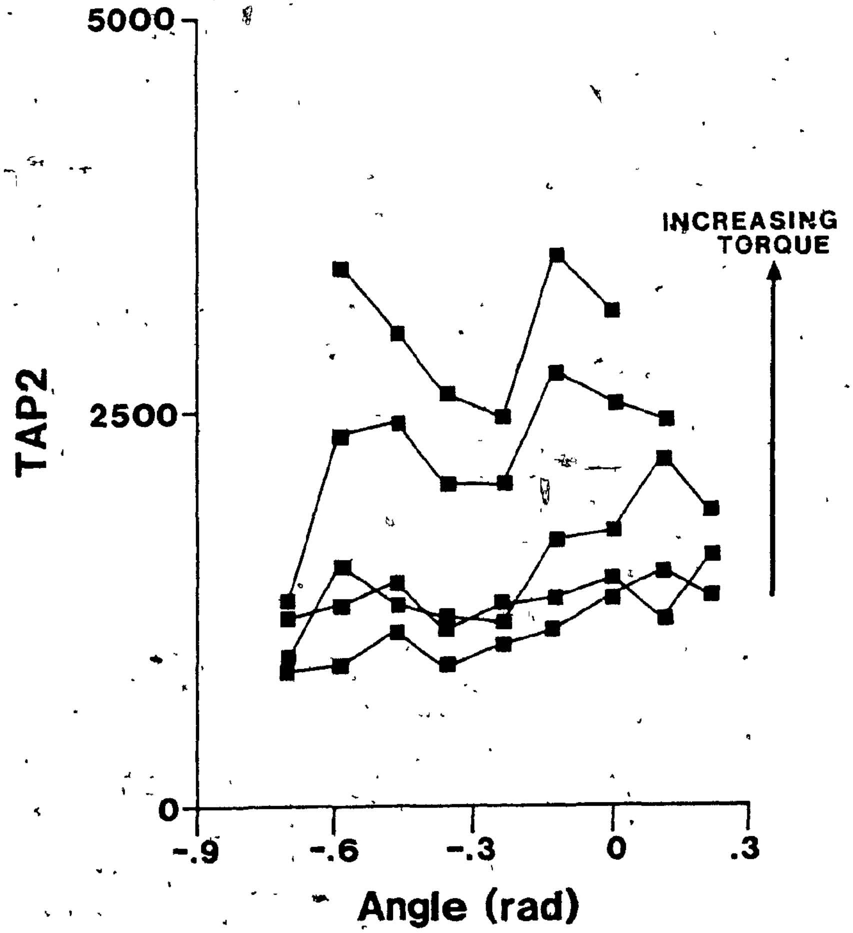


**Fig. 6.6:** TAP2 plotted as a function of ankle-angle. Each square represents the TAP2 determined at one of the tonic level/ankle angle combinations. The TAP2s determined at the same tonic level are joined. (Subject S7)

set was included in the analysis.

The results of this analysis, listed in Table 6.3 for the five subjects, demonstrated that most of the variance in the TA reflex magnitude was accounted for by these two parameters; the VAF by the EMG-angle model ranged from 79 to 86%. However, in contrast to TS, the VAF by a linear regression model that included only TA EMG (the EMG model) decreased by less than 2.5% from that accounted for by the EMG-angle model (see Table 6.3).

These findings suggested that the effect of mean position on TA stretch reflexes was considerably less important than that found for TS. Moreover, as demonstrated by the results of a series of linear regression analyses, the relation between TA reflex magnitude and tonic activity was relatively independent of ankle angle. Indeed the ratio between the largest and smallest slopes for a given subject did not exceed 2.4 and no systematic pattern to these changes in slope could be discerned. As shown by the VAFs presented in Table 6.4, the TAP2/TA EMG relations were highly linear at all mean positions.

The results of the TAP2/TA EMG linear regression analyses for one subject are shown in Fig. 6.7. The data from only six of the nine ankle angles are shown in this figure and the regression lines have been included. It is abundantly evident that the relation between TA reflex magnitude and TA EMG has not been significantly affected by mean ankle position.

The presentation of results for the TA stretch reflex has been

| SUBJECT | EMG-ANGLE MODEL | | | EMG MODEL |
|---|---|---|---|---|
| | TA EMG Coeff. | ANGLE Coeff. | VAF (%) | VAF (%) |
| S1 | 11.2 | 143 | 79.9 | 79.3 |
| S2 | 18.7 | 891 | 86.5 | 83.8 |
| S3 | 20.1 | 430 | 86.7 | 85.1 |
| S5 | 19.0 | 626 | 79.7 | 77.0 |
| S7 | 11.5 | 45 | 79.5 | 79.1 |

**Table 6.3:** The slopes, intercepts and VAFs by a linear regression model between TAP1 and TA EMG and between TAP2 and TA EMG. Note that TAP1 was not identified for Subject S2.

| SUBJECT | ANGLE | TAP1 | | TAP2 | |
|---|---|---|---|---|---|
| | | SLOPE | VAF | SLOPE | VAF |
| S1 | -0.828 | 13.9 | 99.8 | 8.3 | 98.4 |
| | -0.710 | 12.9 | 92.0 | 7.3 | 87.7 |
| | -0.591 | 17.5 | 95.8 | 17.5 | 99.0 |
| | -0.474 | 15.7 | 97.9 | 13.3 | 92.0 |
| | -0.358 | 6.3 | 76.2 | 15.2 | 96.5 |
| | -0.239 | 7.4 | 67.2 | 13.0 | 96.9 |
| | -0.121 | 5.8 | 54.5 | 17.1 | 98.2 |
| | 0.001 | 17.5 | 97.9 | 15.5 | 98.8 |
| | 0.115 | 14.4 | 88.6 | 10.5 | 79.6 |
| | 0.215 | 5.9 | 84.3 | 7.7 | 88.1 |
| S2 | -0.756 | - | - | 21.0 | 97.7 |
| | -0.639 | - | - | 20.1 | 99.0 |
| | -0.520 | - | - | 24.5 | 96.6 |
| | -0.404 | - | - | 23.8 | 95.2 |
| | -0.302 | - | - | 22.0 | 99.3 |
| | -0.202 | - | - | 22.3 | 97.1 |
| | -0.101 | - | - | 20.5 | 95.7 |
| | 0.000 | - | - | 16.5 | 89.9 |
| | 0.083 | - | - | 19.9 | 89.4 |
| S3 | -0.944 | 6.6 | 24.9 | 23.0 | 92.3 |
| | -0.823 | 10.9 | 59.4 | 26.7 | 97.2 |
| | -0.709 | 9.2 | 66.0 | 26.1 | 96.5 |
| | -0.590 | 14.4 | 86.3 | 31.5 | 96.0 |
| | -0.473 | 21.0 | 94.6 | 29.3 | 97.9 |
| | -0.234 | 26.3 | 93.2 | 34.2 | 93.7 |
| | -0.119 | 20.8 | 85.7 | 33.8 | 99.0 |
| | 0.000 | 16.2 | 71.6 | 29.9 | 94.7 |
| | 0.084 | 13.8 | 72.8 | 26.3 | 94.3 |
| | 0.165 | 10.6 | 82.2 | 24.9 | 83.8 |
| S5 | -0.775 | 32.7 | 98.6 | 45.4 | 95.4 |
| | -0.708 | 25.3 | 99.6 | 38.8 | 95.0 |
| | -0.592 | 17.0 | 91.6 | 24.9 | 96.7 |
| | -0.472 | 25.2 | 91.3 | 34.3 | 92.8 |
| | -0.359 | 17.2 | 95.8 | 23.2 | 94.6 |
| | -0.238 | 19.1 | 91.8 | 19.8 | 90.2 |
| | -0.122 | 14.5 | 81.9 | 24.4 | 95.8 |
| | 0.001 | 19.8 | 94.8 | 18.7 | 84.1 |
| | 0.081 | 17.9 | 82.3 | 23.9 | 96.2 |
| | 0.181 | 18.2 | 85.3 | 22.4 | 84.2 |
| S7 | -0.509 | 9.2 | 89.1 | 12.5 | 91.2 |
| | -0.424 | 18.9 | 96.7 | 16.5 | 98.4 |
| | -0.341 | 23.2 | 93.8 | 18.2 | 93.6 |
| | -0.256 | 19.2 | 93.7 | 12.1 | 81.7 |
| | -0.171 | 17.4 | 97.5 | 17.6 | 95.4 |
| | -0.087 | 23.3 | 98.8 | 17.4 | 99.4 |
| | 0.002 | 14.0 | 97.3 | 13.4 | 93.4 |
| | 0.095 | 14.8 | 86.6 | 20.2 | 94.7 |
| | 0.197 | 10.7 | 42.2 | 14.3 | 96.2 |

**Table 6.4:** Results of a multiple linear regression analysis between TA EMG, ankle angle and TAP2 (the EMG-angle model). The TA EMG coefficients, the angle coefficients, the intercepts and the VAFs by this model are listed for all subjects. The VAF by a linear regression analysis between TA EMG and TAP2 (the EMG model) is also listed. (Units are as indicated in Table 2.)

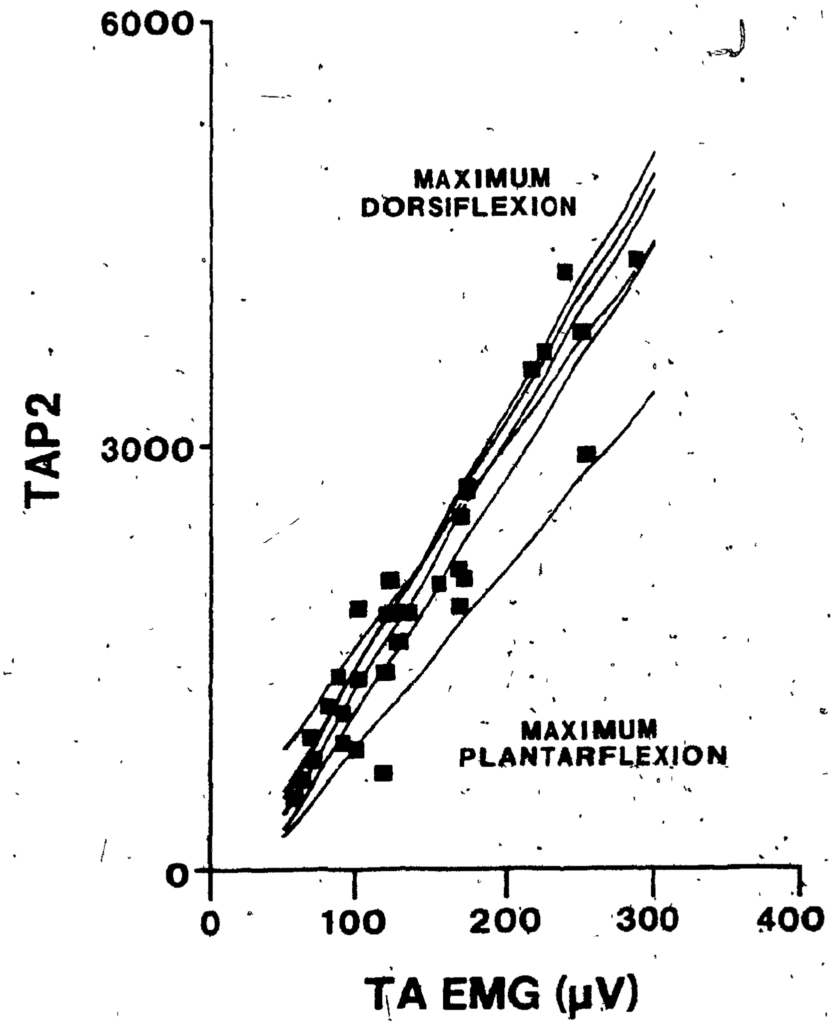


**Fig. 6.7:** Data from Fig. 6.6 replotted to illustrate the relation between TAP2 and TA EMG. Lines represent the best least squares fit between TAP2 and TA EMG at each angle ranging from maximum plantarflexion (bottom trace) to maximum dorsiflexion (top trace). (Subject S7)

restricted to TAP2 since no further information related to the effect of mean position and mean EMG on TA reflexes was revealed by similar analyses of TAP1. Although these two dependent variables did not necessarily covary (the VAF by a linear relation between TAP1 and TAP2 ranged from 56 to 87%), TAP1 and TAP2 behaved similarly as functions of ankle angle and TA EMG. This similarity is demonstrated in Table 6.4 which shows the results of linear regression analyses between TAP1 and TA EMG for four of the five subjects. The VAF was generally high, the slopes were of similar magnitude to the TAP2/TA EMG slopes and also showed little change with ankle angle. Note that TAP1 could not be reliably identified for Subject S2.

To summarize, the effect of ankle angle on the TA stretch reflex magnitude was relatively small and non-systematic. In contrast, it was substantially altered by the level of tonic activity. Thus it appears that the TA reflex magnitude can be determined from the TA EMG without a need to consider the mean position of the ankle.

## DISCUSSION

This study represents the first attempt to examine the effect of changes in mean ankle position on the human ankle stretch reflexes during tonically-maintained contractions held over more than 85% of the ROM. The principal finding was that TS stretch reflexes depended strongly upon the position of the ankle while TA stretch reflexes did not. Thus very large increases in the TS stretch reflex magnitude resulted when the ankle was maintained at progressively more dorsiflexed angles. In contrast, mean ankle position had a minor effect on the TA stretch reflex magnitude which exhibited relatively small and non-systematic variations over the ROM. Such disparate behaviour is consistent with previous results where differences in the shape, linearity, and gain of the TS and TA RIRFs were demonstrated (Kearney & Hunter, 1983; 1984). Moreover, it is not unexpected given the number of anatomical, physiological, and biomechanical differences between these two antagonists (e.g. Belanger et al., 1983; Kearney & Chan, 1982).

The experimental stimuli consisted of small-amplitude, stochastic perturbations of the ankle. The response to this input was modulated by two parameters. The ankle was first rotated to a randomly-selected ankle angle and then TS or TA tonic contractions were generated. In previous experiments, reflex impulse response functions were determined between the first derivative of the dynamic position input and the measured output (agonist EMG) for different levels of the tonic voluntary activity but while the mean joint position was fixed at one angle (near the NP). These velocity-sensitive responses had a uniform latency but a magnitude that increased linearly with the level of agonist contraction (Kearney & Hunter, 1983; 1984). On the basis of these findings, the impulse response functions were considered to represent stretch reflexes mediated primarily via spindle afferent pathways with a gain that was dependent upon the extent of supraspinal drive to skeletal motoneurons (Matthews & Stein, 1969; Gottlieb & Agarwal, 1979; Kearney & Hunter, 1983).

The present addition of the tonic position parameter, and subsequent demonstration, that the stretch reflex dynamics could be satisfactorily identified at all tested ankle angles and tonic levels, permitted an examination of these responses for a functionally significant span of ankle action. Furthermore, the fact that the stretch reflex responses retained their characteristic shape and latency throughout the range of tested conditions enabled the effects of ankle angle and agonist EMG to be characterized simply in terms of changes in the reflex magnitude.

### Origin of the Position Dependent Effect

The present experiments were not designed to identify which afferent pathway(s) convey the static position of the ankle to the central structures. Indeed, attempts to do so in intact human subjects over a range of functionally significant torques and angles are fraught with technical difficulties (e.g. Prochazka & Hulliger, 1983). Nevertheless, some discussion of the possible mechanisms is warranted both in terms of how the position-dependent TS stretch reflex response is mediated as well as possible reasons why the TA stretch reflex was not affected.

First, it is certain that the position-dependent responses were not due to rotation of the ankle to the new mean position since the five minute pause prior to the onset of the dynamic position input was sufficently long to preclude any enduring central effect from this source. Rather, the observed changes in the TS stretch reflex magnitude were the result of the maintenance of this position and the ensuing central input along pathways that may have included the muscle spindle, tendon organ, articular, and cutaneous afferents.

One possibility is that the maintenance of different ankle positions was associated with changes in TS α-motoneuron pool excitability mediated by excitatory post-synaptic potentials (EPSPs). It is, however, unlikely that the observed position-related changes in the reflex response were the result of fluctuations in α-motoneuron pool excitability since significant variations in the mean TS EMG were not evident; for a given level of tonic activity, changes in mean agonist EMG were small and not systematically related to either ankle angle (Chapter 5) or the magnitude of the TS reflex response.

This conclusion would appear to contradict the view that position-dependent changes in the H-reflex were mediated via autogenetic post-synaptic effects from spindle secondaries acting to decrease the TS α-motoneuron excitability (Burke et al., 1971). However, there is little similarity between the H-reflex and present paradigms. First, the two inputs give rise to dissimilar patterns of afferent excitation (cf. Burke, 1983b). Second, the stretch-induced H-reflex inhibition was only observed in the relaxed TS; the inhibition was reversed when the tonically-contracting TS was dorsiflexed (Mark et al., 1968). Moreover, a comparison of the position-dependent responses of the H-reflex and a polysynaptic reflex (elicited by tibial nerve stimulation near the medial malleolus) suggests that the stretch-induced H-reflex inhibition was not mediated via changes in TS α-motorneuron gain. Although the H-reflex was depressed when the ankle was dorsiflexed (e.g. Paillard, 1960; Delwaide & Hugon, 1969), the polysynaptic reflex was facilitated at the same angles (Delwaide, 1973). The polysynaptic reflex would have been similarly reduced had the H-reflex inhibition been the result of decreased TS α-motoneuron excitability (Delwaide, 1973).

## Efficacy of Dynamic Position Perturbations

A more tenable explanation is that changes in the static position parameter acted by modulating the efficacy of the dynamic position input. This could be accomplished in several ways. First, transmission of the afferents mediating the stretch reflex could be altered by position-induced discharge of spinal interneurons. Second, the sensitivity of the muscle spindle endings to the dynamic perturbation may have been alterated by changes in the extent of fusimotor drive. Third, transmission of the dynamic position perturbations to the spindle, and hence the magnitude of the spindle receptor discharge, could be altered by changes in the mechanical properties of either the extrafusal or intrafusal muscle fibers. Each of these possibilities is considered below.

### Modulation of the Monosynaptic and Polysynaptic Primary Afferent Input

Presynaptic inhibition, a diminution of the monosynaptic transmission between the spindle primary afferent and the skeletal motoneuron as a result of primary afferent depolarization (Eccles, 1964) represents one mechanism for the position-dependent modification of the effectiveness of the dynamic position. Presynaptic inhibition resulting from the static ankle position has been demonstrated in the acutely-spinalized cat; stretch of the ankle flexors generated a depression of ankle extensor monosynaptic EPSPs which was demonstrated to have been the result of presynaptic inhibition (Devanandan et al., 1965). Although this finding is compatible with the results of the present study in that the TS RIRF was facilitated upon ankle dorsiflexion and the concomitant decrease in TA stretch, the evidence that this mechanism plays an important role in the mediation of maintained human joint position is indirect only. For example, pre-synaptic inhibition, via homonymous Ia innervation of inhibitory interneurons, has been suggested as a possible explanation of the stretch-induced inhibition of the H-reflex in the relaxed TS (Delvaide, 1973). The importance of presynaptic inhibition in the position dependence of the stretch reflexes cannot yet be determined.

It is noteworthy that there does not appear to be much difference in the nature of presysnaptic inhibition to flexor and extensor spindle primary afferents (Eccles et al., 1962) although the data are far from complete (Baldissera et al., 1981). Thus, on the basis of the limited evidence available, this mechanism does not appear to account for the disparate responses of the TS and TA stretch reflexes.

Finally, in view of the demonstration of polysynaptic spindle primary pathways to homonymous skeletal motoneurons in cat (e.g. Watt et al., 1976; Jankowska et al., 1981) and the more recent inferences regarding the existence of polysynaptic pathways for the H-reflex and tendon jerk in man (Burke et al., 1984), it is certainly possible that the position-dependent changes in TS reflex magnitude were mediated via a network of interneuronal connections. However, the feasibility of this mechanism must await more direct measures of the extent of the polysynaptic contribution to the stretch reflex.

## Altered Fusimotor Drive

Although there appears to be little fusimotor drive to non-contracting human muscles (Burke et al., 1979), this drive increases as the level of skeletal motor activity increases (Vallbo, 1974; Hulliger & Vallbo, 1979). Any position-dependent alterations in the extent of the fusimotor drive represent a mechanism by which the spindle receptor responsiveness and hence the efficacy of the dynamic position input could be altered. The question is whether this mechanism is operational during voluntary, isometric contractions similar to those maintained during the present experiments.

Recent studies have revealed that cat static and dynamic fusimotor neurons are innervated by a wide range of group II and III muscle, joint, and cutaneous afferents as well as homonymous spindle secondary afferents (Appelberg et al., 1981; 1983). Such multisensorial input is certainly compatible with the soft tissue changes produced by the tonically-maintained positions. It is, therefore, possible that any afferent information from these tissues feeding back to the fusimotor neurons could have affected the dynamic spindle response by altering the magnitude of the fusimotor drive and the relative proportion of static and dynamic drive.

However, such pathways have not been demonstrated in man and there does not appear to be a significant position-dependent change in fusimotor drive, at least not for the muscles (TA, Burke et al., 1980 and finger extensor muscles, Vallbo et al., 1981; Hulliger et al., 1982) studied to date. Although these findings suggest that this mechanism is not responsible for the position-dependent changes in the stretch reflex, there are two reasons why the question as to whether maintained changes in ankle position affect the responsiveness of TS muscle spindle to dynamic perturbations must remain unanswered. First, for technical reasons, the position-dependent response of TS afferents have not been examined; the lack of a demonstrated change in the discharge characteristics of TA afferents as a function of joint angle simply confirms the present result for the TA stretch reflex. Second, the levels of tonic activity and range of positions tested in the above studies were extremely limited and may have been inadequate to elicit a position-dependent response.

In view of the above discussion it is of interest to consider the possibility of fusimotor activation as a result of an artifactual, ankle-perturbation-induced soft tissue vibration. Burke et al. (1980) have demonstrated that TA spindle endings were activated at lower force levels when the dorsum of the foot was vibrated. In contrast, higher force levels were required to activate the same spindle ending when the plantar surface was vibrated. Assuming that such changes in spindle ending threshold reflect alterations in the extent of fusimotor drive (Burke et al., 1980), the observed position-dependent changes in reflex magnitude could have been mediated, in part, by unintended but concomitant vibration-induced alterations in cutaneous mechanoreceptor activation. Although this possibility cannot be entirely discounted, it would appear to be unlikely given the polyurethane foam cast fixation and the resulting uniform contact between it and the skin surface of the foot at all mean positions. Moreover, vibration related artifacts would be expected to have a similar effect on the TA reflex response, yet none were observed.

## Peripheral Transmission of the Dynamic Position Input

Position-dependent changes in the extrafusal (Burke, 1981; Rack et al., 1983) or intrafusal (Poppele & Quick, 1982) muscle mechanical properties could alter the transmission of the ankle perturbation and hence the responsiveness of muscle spindle afferents to the dynamic position input. Based on the relative magnitude of the spindle afferent response to sudden joint displacements in relaxed and contracting muscle (Burke et al., 1978), Burke (1981) has suggested that contraction of the extrafusal fibers may "dampen" the transmission of a dynamic joint perturbation with the result that the spindles are exposed to stimuli of smaller intensity. Rack et al. (1983, 1984) have suggested that such spindle stimuli may vary as a function of the level of tonic contraction with the extent of the ankle joint displacement transmitted to the spindle becoming progressively smaller as the stiffness of the muscle relative to the tendon increases. As indicated in Chapter 5, the mean EMG and the mean active torque generated by the subject in the present study did not vary with ankle angle nor did the active joint mechanics change. In the light of such invariance the present findings cannot be interpreted to result from active stiffness-induced changes in joint perturbation transmission.

However, as demonstrated in Chapter 4, there were significant position-dependent changes in the passive joint mechanics. This is relevant to the present study in view of the finding that the magnitude of both the TS stretch reflex and the passive joint stiffness became substantially larger as the ankle was dorsiflexed. Indeed the behaviour of these variables for angles between the NP and maximum dorsiflexion was very similar (cf. Fig. 4.6 and Fig. 6.3). Although it was not possible to ascertain the source of the position-dependent passive joint stiffness, such increases in either TS tendon or muscle could enhance transmission of the joint perturbation.

The contrasting behaviour of the TS and TA stretch reflexes is now considered in view of the observation that the passive torque and stiffness were generally asymmetrical; both were smaller when the ankle was plantarflexed than when it was dorsiflexed. It would appear that plantarflexion is associated with fewer changes in TA muscle and/or tendon properties with the result that transmission of the joint perturbation to the TA spindle receptors may remain quite uniform.

## Effect of the Changing MVC

As indicated in Chapter 5, plantarflexor and dorsiflexor MVCs magnitudes are altered by the position of the ankle joint with the result that varying percentages of the motoneuron pool may have been recruited for identical active torques levels generated at different ankle angles. The observed position-dependent changes in reflex magnitude may, therefore, have been a consequence of the constant-torque paradigm and the resulting changes in motoneuron pool excitability. This possibility was considered for both TS and TA reflexes.

The TS MVC increases as the ankle is dorsiflexed (Sale et al., 1982). For example, a plantarflexor torque that corresponded to 30% of the NP MVC would comprise a larger percentage of the MVC when the ankle was near maximum plantarflexion. However, the present results showed that the TS reflex response increased when the ankle was dorsiflexed, a position associated with torques that comprised a relatively small percentage of the MVC. Rather than accounting for the changes in TS reflex magnitude, the use of the constant-torque paradigm may have resulted in the underestimation of the effect of ankle angle on the the TS reflex magnitude.

In contrast, TA MVC decreases as the ankle is dorsiflexed (Marsh et al., 1981). Thus similar torques generated at the more dorsiflexed angles corresponded to a greater percentage of the MVC than those exerted when the ankle was more plantarflexed. Indeed, this effect may have accounted for the small increase in the TA reflex magnitude that was observed when the ankle was rotated from plantarflexion to dorsiflexion.

## Functional Implications

A question to be addressed is whether the position-dependent modulation of the TS stretch reflex is of any functional significance to man. First, is the position-induced facilitation of significant magnitude relative to that generated by other manoeuvers? Second, does it occur at angles where a facilitated response would be appropriate? Regrettably, the ultimate question, concerning the role of the stretch reflex in the functional regulation of movement remains to be clarified.

The first question is answered by comparing the relative changes in the magnitude of the TS stretch reflex as functions of ankle position and tonic activity. The results of this comparison are presented in Table 6.5 which lists the TSP magnitudes obtained at two mean positions (NP and maximum dorsiflexion) and at two tonic levels (10 and 50% NP MVC). The two EMG-dependent changes in reflex magnitude are listed in the first (ratio of the 50% and 10% values at the NP) and second (ratio of the 50% and 10% values at maximum dorsiflexion) columns. In contrast, the two position-dependent changes in reflex magnitude are listed in the third (ratio of the 10% maximum dorsiflexion and NP values) and fourth (ratio of the 50% maximum dorsiflexion and NP values) columns.

It is evident that the position-dependent increases in TS reflex magnitude are as large as or larger than those obtained by increases in tonic activity. Whereas the TSP was 1.2 to 3.3 times larger for a 50% tonic contraction than for the 10% tonic contraction at the same angle, it increased by up to 25.2 times when the ankle was dorsiflexed from the NP at the same tonic level. In contrast to TS, changes in ankle angle had a considerably smaller effect on the TAP2 than did changes in joint torque (also shown in Table 6.5). Thus it can be

| | | NEUTRAL POSITION | | MAX DORSIFLEX | | TORQUE DEPENDENT | | POSITION DEPENDENT | |
|---|---|---|---|---|---|---|---|---|---|
| | SUBJ | 10% | 50% | 10% | 50% | | | | |
| | | <---1---><--------4--------> | | | | RATIO | | RATIO | |
| | | <--------3--------><---2---> | | | | 1 | 2 | 3 | 4 |
| TSP | S1 | 590 | 1840 | 7070 | 10650 | 3.1 | 1.5 | 12.0 | 5.8 |
| | S2 | 1335 | 2870 | 4940 | 6090 | 2.1 | 1.2 | 3.7 | 2.1 |
| | S3 | 860 | 1580 | 21715 | 26385 | 1.8 | 1.2 | 25.2 | 16.7 |
| | S5 | 2020 | 3680 | 15850 | 23340 | 1.8 | 1.5 | 7.8 | 6.3 |
| | S7 | 1820 | 5510 | 10840 | 17500 | 3.3 | 1.6 | 6.0 | 3.2 |
| TAP2 | S1 | 890 | 3420 | 1320 | 2440 | 3.8 | 1.8 | 1.5 | 0.7 |
| | S2 | 1390 | 6070 | 1920 | 6420 | 4.4 | 3.3 | 1.4 | 1.1 |
| | S3 | 1520 | 5850 | 2145 | 5630 | 3.8 | 2.6 | 1.4 | 1.0 |
| | S5 | 1010 | 4180 | 2490 | 5350 | 4.1 | 2.1 | 2.5 | 1.3 |
| | S7 | 648 | 4220 | 1850 | 8070 | 6.5 | 4.4 | 2.8 | 1.9 |

Table 5: A comparison of the relative effect of changes in the level of tonic activity and changes in mean position on the magnitude of the TS and TA reflex response. Values for TSP and TAP2 are listed for all subjects during four test conditions (10 and 50% NP and 10 and 50% maximum dorsiflexion). The increase in reflex magnitude that is associated with changes in torque level at a given angle (columns 1 and 2) is compared to the that which is associated with changes in ankle angle at a given torque level (columns 3 and 4).

concluded that the effect of mean ankle position on the TS stretch reflex was significant relative to both the EMG-dependent facilitation of TSP and the position-dependent facilitation of TAP2.

Is the TS stretch reflex facilitated when a larger response would be functionally advantageous? The response to changes in ankle position are often consistent with the requirements of a movement as, for example, in the regulation of upright stance. Thus as the individual sways forward, the center of gravity shifts anteriorly and a larger plantarflexor moment is required to prevent the body from falling. It was argued in Chapter 5 that the passive plantarflexor torque and resulting stiffness would augment the periodic TS contractions to prevent such occurences. These bursts of TS activity occur when the center of gravity is most anterior (Smith, 1957) i.e. when the ankle is most dorsiflexed and would be reinforced by position-dependent facilitation of the TS stretch reflex. In contrast, the TA is quiet throughout quiet stance; position-dependent facilitation of the TA stretch reflex would, therefore, serve no purpose.

# CHAPTER 7: CONCLUSION

The objective of the work that comprises this thesis was to characterize the position-dependent dynamics of the human ankle joint. Experiments, using systems identification techniques, determined ankle mechanics (position-to-torque dynamics) and stretch reflex dynamics (position to EMG dynamics) over most of the range of ankle movement and over a functionally significant range of tonic contraction levels.

The experimental stimuli consisted of small-amplitude, stochastic perturbations imposed by an actuator on the ankle joint. Responses to these stimuli were modulated by two, experimentally-controlled parameters: mean joint position (controlled by the actuator) and tonically-maintained TS or TA contractions (achieved by voluntary tracking of displayed target levels). Ankle joint torque and agonist EMG were the output variables measured.

Two systems were identified in these studies: the joint mechanics and the stretch reflex dynamics. Although there are two components to the joint mechanics, passive and active, the paradigm was arranged so that these could be investigated independently. Thus the passive component was identified with the ankle muscles at rest and the active component was determined for identical levels of tonic contraction at all mean positions after the passive contribution was removed. The TS and TA stretch reflex dynamics were identified by determining the dynamic relation between ankle angular velocity and agonist EMG.

## SUMMARY OF MAJOR ORIGINAL FINDINGS

These studies were designed to provide a quantitative and functionally relevant examination of the position dependence of the human ankle joint dynamics. The principal original contributions are:

1. The position-dependent changes in the passive joint mechanics were large and functionally significant.

2. The active joint mechanics were not modified by mean ankle position but depended primarily on the magnitude of the actively-generated torque.

3. The TS stretch reflexes depended strongly upon the position of the ankle.

4. The TA stretch reflexes did not vary systematically with ankle mean position.

Each of these results, as well as their significance with respect to normal and abnormal human motor function, will be discussed in more detail below.

### Experimental and Analytical Techniques

Techniques based on the methods of system identification were employed. These techniques are particularly useful because they are rapid, requiring less than a minute of data collection to get good impulse response estimates; non-invasive; and amenable to modelling. These methods have been used previously both in this laboratory (e.g. Hunter & Kearney, 1982; Kearney & Hunter, 1983) and elsewhere (e.g. Agarwal & Gottlieb, 1977). However, this is the first time that quantitative methods have been used to model the effect of changes in mean joint position over most of the ankle ROM, over a functionally-significant span of tonic activity, and in both the ankle dorsiflexors and plantarflexors.

This study greatly extends the results of Gottlieb & Agarwal (1978) who studied the effect of mean ankle position on the ankle joint mechanics over a very small range of ankle angles and tonic contraction levels and represents, therefore, the most comprehensive examination of the position dependence of human ankle joint functions to date. The fact that the mechanical and reflex behaviour of the ankle could be identified and modelled over the whole ROM without a significant reduction in the VAF by either the non-parametric or parametric representations of the systems demonstrated that the particular system identification techniques used were appropriate. Furthermore, the fact that the identified parameters were sensitive to changes in mean position indicated that these techniques were appropriate for the present problem.

## Importance of the Passive Mechanics

The first study provided the opportunity to examine the position dependence of the joint mechanics when the ankle muscles were at rest. The results of this investigation demonstrated that the passive elastic stiffness attained much larger magnitudes than previously assumed. Whereas mid-range positions were associated with relatively small, constant values, the elastic stiffness became considerably larger as the ankle was rotated towards the extremes of the ROM.

## Linear Relation between Passive Stiffness and Passive Torque

Both the passive joint torque and the stiffness were shown to behave comparably with respect to mean position; similarly rapid increases and asymmetries were found in both curves. It was not the objective of the present study to investigate the origin of the passive, position-dependent changes in stiffness. However, based on the demonstration of the linear relation between these two variables it was proposed that the position-dependent changes in the passive joint mechanics were related to the change in passive joint torque that resulted when mean ankle position was altered.

## Linear Relation between Active Stiffness and Active Torque

The active stiffness was linearly dependent upon the active torque at all ankle angles. This had already been demonstrated for mid-range ankle positions (Hunter & Kearney, 1982) but was confirmed for all ankle angles for both dorsiflexor and plantarflexor tonic contractions.

## Invariance of Active Joint Mechanics with Joint Position

Contrary to the passive mechanics, the position-dependent changes in the active mechanics were relatively small and variable. Thus, provided the level of active torque remained constant at all mean joint positions (the constant-torque paradigm), there was no additional effect of changes in joint positions on the joint mechanics. Thus knowledge of ankle mean position is not essential for the characterization of the joint mechanics provided that the total joint torque is known.

## Independence of Active and Passive Processes

Position-dependent changes in the passive tissues were not modified by the presence of tonically-maintained muscle contractions. Moreover, there was little systematic change in the gain of the stiffness/active torque relation as the ankle was rotated from maximum plantarflexion to maximum dorsiflexion. It was therefore proposed that the active and passive processes were independent.

## Relative Contribution of Active and Passive Processes to Joint Stiffness

A comparison of the relative contributions of torque-dependent and position-dependent effects on the total joint mechanics has revealed that the position-dependent effect on the passive joint mechanics can be used to more efficiently achieve joint stability. Indeed, the stiffness generated by purely passive processes was demonstrated to be comparable to that produced by a 10 to 20%MVC contraction. It was therefore proposed that there was a real possibility of achieving the required joint stability by a process that would represent a considerable energy saving.

## Position Dependence of TS Stretch Reflexes

The TS stretch reflexes were strongly dependent on changes in ankle position, increasing substantially as the ankle was progressively dorsiflexed. It was proposed that the position-dependent facilitation was a result of mechanisms that modulated the efficacy of the stochastic ankle perturbation. Suggested mechanisms included position-induced changes in (i) the interneuronal transmission of the afferents mediating the stretch reflex, (ii) the extent of fusimotor drive, and (iii) the transmission of the dynamic position perturbations to the spindle.

## Invariance of TA Stretch Reflexes with Joint Position

In contrast, mean ankle position had only a minor effect on the TA stretch reflex magnitude. It appeared that the remarkably different behaviour of these two antagonists was related to their disparate anatomical, mechanical and functional attributes.

## Functional Relevance of Position-Dependent Effects

Both the passive stiffness and the TS stretch reflex magnitude were augmented at joint angles where a facilitated response was functionally useful. This was demonstrated with respect to the example of the maintenance of equilibrium during upright stance.

## LIMITATIONS OF THE STUDY AND RECOMMENDATIONS FOR FUTURE WORK

There were a number of limitations associated with this study. These are outlined below along with a series of recommendations for further study.

### Variables that cannot be measured

One of the major limitations of experimental work on intact human subjects is related to the fact that changes in the soft tissue properties cannot be directly measured. Technical limitations and ethical considerations preclude direct measurement of many of the anatomical and physiological changes which may be associated with variations of ankle joint position in man. Furthermore, results from animal studies have only limited relevance since non-human ankle structure and function differ considerably from their human counterpart (Alexander, 1973). Thus it was not possible to ascertain changes in the length of muscle and the other soft tissues from the observable kinematic variable, joint angle. Nor was it possible to determine the forces generated by these tissues from the observable kinetic variable, measured joint torque. Finally, the sensory pathways could not be identified directly from the estimate of neural motor activity, the measured EMG.

The inability to translate from the experimentally measured inputs and outputs to the function of the individual tissues was a serious limitation and one that makes the interpretation of the data more obscure. For example, the determination of the relative contributions of the soft tissues to the passive joint torque is one important question that must remain unanswered.

It is therefore necessary to use and develop techniques for measuring variables that are more directly related to function of the tissues of interest. For example, microneurography represents a way to monitor afferent function in intact humans (Vallbo & Hagbarth, 1967). However, considerable improvements are required before this technique would be feasible in the present paradigm because stochastic perturbation could result in very unstable recordings. X-rays or other imaging techniques (e.g. nuclear magnetic resonance) may provide ways to measure 'on-line' static and dynamic changes in muscle length during ankle rotation. Finally, clip transducers have been used in animal studies to provide a more direct estimate of the force generated by the muscle (Gregor et al., 1983). A modified version of this transducer may be feasible for human experiments.

Another related issue involves the fact the joint mechanics were identified between ankle angular position and torque, variables that can be measured directly. Angular position was modified by the limb kinematics and the torque reflected the forces generated by the passive and active tissues after these latter had been modified by the mechanical linkage at the joint (for example, the moment arm). It was therefore assumed that the system dynamics were not altered by any of these transformations. The fact that this assumption has not been verified in human subjects represents a limitation of the study.

### Functional requirements during testing

The importance of assessing the effect of functionally significant spans of ankle positions and tonic levels has been stressed throughout this thesis. However, another potentially important element related to functional requirements has not been addressed, namely the subjects' posture. The support arrangement used in the present study (subjects supine) enabled the study to be performed in a rigorously controlled environment. The head and limbs remained stationary and the cutaneous input was constant throughout the experiment.

The work of, for example, Nashner (1976, 1977) on standing subjects and Rossignol et al. (1982, 1983) on treadmill-walking cats suggests that the nature of the response to a particular stimulus may be dependent on whether that response would be functionally appropriate. Although the occurrence of the position-dependent effects of passive stiffness and TS stretch reflexes appeared to be appropriate for functional activities such as the maintenance of a balanced standing posture, it is not known whether these are the responses that would have been measured during this activity.

There are, however, considerable problems associated with adequately securing a human subject during standing or walking. Experimental artifact from, for example vestibular input, would be unavoidable unless subject fixation was so extensive as to render insignificant the functional aspect of the paradigm.

An apparatus is needed that would permit both functional movements and the required experimental control. One possibility is the bicycle ergometer. The seating arrangement is such that head and upper body immobility could be readily achieved. The pedals could be modified to enable powerful and well-controlled inputs to the ankle. Finally, the subject could be required to pedal, a bilateral functionally-relevant task that would alleviate this limitation.

## Applications of techniques to pathology

In addition to the present study of motor control in normal human subjects, the potential use of system identification techniques for the objective assessment of abnormal function is of great consequence. In most clinical settings disorders of motor performance are diagnosed and evaluated qualitatively. For example, hypertonicity is assessed by observation of a patient's response to tendon taps and the sensation of a limb as it is rotated through its range of motion. In the case of trauma, x-rays, arthroscopy, and positive contrast arthograms are used to identify mechanical obstructions. Such methods, however, generally provide information regarding the static anatomical and physiological function rather than providing a quantitative and dynamic assessment of the problem.

Disorders of many different sources lead to alterations in the ability to use the joints of the body. The ankle joint is particularly susceptible to injury and pathology since it is subjected to very large forces over a very large range of positions. The most common injuries are of two types, reduced mobility and hypermobility. In the first case, there is a significant decrease in the ROM often to the point of a fixation at one extreme or the other; the joint is relatively stable but mobility is limited. Causes of this problem include osteoarthritis, impingement syndromes and hypertonicity. In the second case, there is a general increase in the ROM where mobility is enhanced but stability is greatly reduced. Causes of such dysfunction include ligament tears, tendon ruptures and hypotonicity.

Such problems can arise from a number of different disorders ranging from pathology of the peripheral and central nervous system to orthopedic conditions. An additional source of problems is related to the functional adaptations that result from the training for elite performance such as ballet and gymnastics. These adaptations generally involve the need to achieve both increased mobility and strength as in the case of the ballet dancer who maintains the 'en pointe' position for extended periods. However, the body's ability ot adapt is limited and the participants in such activities often develop capsulitis, impingement syndromes, osteophyte formation, stress fractures and tendinitis (Brodelius, 1961; Parkes, 1980).

To date all assessments of the ankle's position-dependent behaviour have been performed on subjects with no evidence of neuromuscular pathology. It is anticipated that the techniques employed to make these measurements will be useful in the evaluation of patients with orthopedic and neurologic disorders. Preliminary results from the study of a deafferented subject indicated that patients can tolerate the experimental environment and techniques (Weiss et al., 1984d). These methods can undoubtedly provide quantitative data that can be used for evaluative and diagnostic purposes but the general clinical feasibility of these techniques remains to be demonstrated.

Although the scope of this thesis did not include the investigation of ankle ROM and position-dependent effects in such pathologies, it is evident that the issues resulting from the results of normal subjects have potential clinical relevance. The present results demonstrate the importance of joint position on the passive mechanical and reflex behaviour of the healthy ankle. If one considers the extent of anatomical, biochemical and physiological alterations that appear secondary to the original trauma or pathology of joint mobility, (e.g. (Tarbary et al., 1972; Williams & Goldspink, 1971; Goldspink, 1977; Odeen, 1981), it is clear that severe deficits in functional disability. It is anticipated that the present paradigm will prove to be sufficiently sensitive to evaluate these functional deficits.

## Multi-joint position dependence

Changes in the orientation of other joints will also affect these tissues. For example, knee position will affect the two-joint gastrocnemii (Herman & Bragin, 1967) whereas TA will be altered by the relative position of the subtalar, tarsal and metatarsal joints. Moreover, in many functional situations, the ankle and subtalar joints work together as a unit (Inman, 1976). The present results represent, therefore, the first step toward identifying the effect that changes in joint position have on the ankle and its associated soft tissues. Future studies should include manipulation of both the knee and subtalar joints.

## The effect of mean ankle position on other variables

Finally, only certain aspects of the position-dependent dynamics were investigated. A more complete characterization of the effect of mean position on dynamic ankle function would include, for example, the dynamic relation between torque and EMG.

# APPENDIX I

## NEXUS MODULES FOR EXPERIMENTAL CONTROL

| | |
|---|---|
| Module CONTROL1: | To run the first experiment (passive mechanics) |
| Module CONTROL2: | To run the second experiment (active mechanics and reflex dynamics). |

## Module CONTROL1

```
#   Module to run the first experiment (passive mechanics).

#   In these experiments, subjects remain relaxed while the actuator
#   perturbs the ankle about different, randomly-selected mean ankle
#   positions.

#   Generate stimulus variables for the actuator (STIM1) and for the
#   oscilloscope (STIM2).  (See Module STIMGEN1 in Appendix II.)

    RUN STIMGEN1

#   Define general experimental details.

    SUBJECT = 'Subject_Name'
    NEWPOS = Initial_Ankle_Position
    NP = Subject_Neutral_Position
    PS = Potentiometer_Calibration
    TS = Torque_Transducer_Calibration
    TAS = TA_EMG_Calibration
    TSS = TS_EMG_Calibration
    CAL = (3.14159/180) * 10
    EXECUTE 'AVGFILE = "PAS"_SUBJECT_".AVG"'

#   Randomize test ankle positions.

    POS = SCR(ANG1_ANG2_ANG3_ANG4_ANG5_ANG6_ANG7_ANG8_ANG9_ANG10; 10000)

#   Run experiment until each angle tested once.

    ANGLENUM = 0
    [NEXTPOS]
         ANGLENUM = ANGLENUM + 1

#        Rotate ankle to next angle.  (See Module ROTATE in Appendix III.)
#        OLDPOS and NEWPOS are the current and new actuator positions,
#        respectively.

         OLDPOS = NEWPOS
         NEWPOS = POS{ANGLENUM}
         RUN ROTATE

#        Sample data.  Average and store data if experiment was
#        successful.  (See Module EXPT1 in Appendix III.)

         RUN EXPT1

#        Loop through for next trial.

         IF ANGLENUM < LEN(POS) THEN GOTO [NEXTPOS]

#   End of Module CONTROL1.
```

## Module CONTROL2

```
#  Module to run the second experiment (active mechanics, statics
#  and reflex dynamics).

#  In these experiments, subjects maintain tonic contractions while
#  the actuator perturbs the ankle about different, randomly-selected
#  mean ankle positions.

#  Generate stimulus variables for the actuator (STIM1) and for the
#  oscilloscope (STIM2).  (See Module STIMGEN2 in Appendix II.)

   RUN STIMGEN2

#  Define general experimental details.

   SUBJECT = 'Subject_Name'
   NEWPOS = Initial_Ankle_Position
   NP = Subject_Neutral_Position
   PS = Potentiometer_Calibration
   TS = Torque_Transducer_Calibration
   TAS = TA_EMG_Calibration
   TSS = TS_EMG_Calibration
   CAL = (3.14159/180) * 10
   TONIC = 1 -1
   EXECUTE 'AVGFILE = "PAS"_SUBJECT_".AVG"'

#  Rotate ankle to Neutral-Position and obtain Pre-experiment TA and
#  TS MVCs.  (See Modules ROTATE and MVC, Appendix III.)
#  OLDPOS and NEWPOS are the current and new actuator positions,
#  respectively.

   OLDPOS = NEWPOS
   NEWPOS = NP
   RUN ROTATE
   RUN MVC

#  Randomize test ankle positions.

   POS = SCR(ANG1_ANG2_ANG3_ANG4_ANG5_ANG6_ANG7_ANG8_ANG9_ANG10; 10000)

#  Run experiment until each angle tested once.

   ANGLENUM = 0
   [NEXTPOS]
        ANGLENUM = ANGLENUM + 1

#       Rotate ankle to next angle.  (See Module ROTATE in Appendix III.)

        OLDPOS = NEWPOS
        NEWPOS = POS{ANGLENUM}
        RUN ROTATE
```

```
#       Run both TA and TS trials at the same angle.

        TONICNUM = 0
        [NEXTTOR]
                TONICNUM = TONICNUM + 1

#               Sample data.  Average and store data if experiment was
#               successful.  (See Module EXPT2 in Appendix III.)

                RUN EXPT2

#               Loop through for next tonic type.

                IF TONICNUM < LEN(TONIC) THEN GOTO [NEXTTOR]

#       Loop through for next trial.

        IF ANGLENUM < LEN(POS) THEN GOTO [NEXTPOS]

#    Rotate ankle to Neutral-Position and obtain Post-experiment TA and
#    TS MVCs.

     OLDPOS = NEWPOS
     NEWPOS = NP
     RUN ROTATE
     RUN MVC

#    End of Module CONTROL2.
```

# APPENDIX II

## NEXUS MODULES TO GENERATE STIMULUS VARIABLES

**Module STIMGEN1:** To generate the stimulus variables required for the first experiment.

**Module STIMGEN2:** To generate the stimulus variables required for the second experiment.

**Module STIMGEN1**

```
#  Module to generate stimulus variables required for first
#  experiment.

#  Actuator Stimulus

#  A 1000 length PRBS is generated.  It contains 499 values at 0.8 V, 499
#  values at -0.8 V and and the first and last points are 0 V.
#  The D/A and actuator calibrations are such that ±0.8 V generates an
#  ankle perturbation of 0.09 rad peak-to-peak.

   PRBS = 0_(1.6 * EXT(PRBS(; 10); 1, 998) - .8)_0

#  Tracking Stimulus

#  Since subjects are not required to maintain during these experiments
#  the magnitude of this stimulus is set to zero.  The length of the
#  stimulus (25) determines the number of repetitions of the PRBS sequence.

   STIM2 = 0 * RAMP(;25)

   RETURN

#  End of Module STIMGEN1
```

## Module STIMGEN2

```
#  Module to generate stimulus variables required for second
#  experiment.

#  Actuator Stimulus

#  A 1000 length PRBS is generated.  It contains 499 values at 0.8 V, 499
#  values at -0.8 V and and the first and last points are 0 V.
#  The D/A and actuator calibrations are such that ±0.8 V generates an
#  ankle perturbation of 0.09 rad peak-to-peak.

   PRBS = 0_(1.6 * EXT(PRBS(; 10); 1,998) - .8)_0

#  Tracking Stimulus

#  This stimulus contains six points at each of the five tonic levels.  In
#  addition, there are a total of 16 zero values.  The order of the four
#  lower tonic levels was randomized but the highest tonic level was always
#  last.  The length of this stimulus (46) determines the number of
#  repetitions of the PRBS sequence.

   T1 = .1_.1_.1_.1_.1_.1
   T2 = .2_.2_.2_.2_.2_.2
   T3 = .3_.3_.3_.3_.3_.3
   T4 = .4_.4_.4_.4_.4_.4
   T5 = .5_.5_.5_.5_.5_.5

   TRACK = 0_0_T3_0_0_T4_0_0_T1_0_0_T2_0_0_T5_0_0_0_0_0_0

   RETURN

#  End of Module STIMGEN2
```

# APPENDIX III

## NEXUS MODULES FOR DATA ACQUISITION

Module ROTATE: To rotate the ankle to the desired mean position.

Module MVC: To sample maximum voluntary contractions.

Module EXPT1: To sample data during the first experiment.

Module EXPT2: To sample data during the second experiment.

**Module ROTATE**

```
# Module to rotate ankle to desired mean position.

# Generate a ramp (Channel R) for input to actuator and adjust it
# to reflect the current and desired angles and the neutral-position.

  R = RAMP(;100)/100
  STIM1 = (-2 * NP) + ( (OLDPOS - ((OLDPOS - NEWPOS) * R)) * -1 * CAL )

# Set sampling rate and rotate actuator.

  SAMRATE = 20
  SET ERROR OFF
  SD2(STIM1,0;'TEMP.SAM',COMMENT,0,8,SAMRATE,0,,)
  SET ERROR ON
  .SD2
  .DELETE TEMP.SAM;*

  RETURN

# End of Module ROTATE
```

## Module MVC

```
# Module to sample maximum voluntary contractions.

# Generate MVC stimulus.  TA MVC is 0 volts for 5 s then 1 volt for 5 s
# during which time the subject should contract TA maximally.  The
# stimulus then returns to 0 volts for 5 s.  TS MVC is the negative
# of the stimulus for TA MVC.

  STIMMVC = 0_0_0_0_0_1_1_1_1_1_0_0_0_0_0

# A dummy stimulus is generated for display to the actuator while
# the MVCs are being sampled.  This is a 100 length channel with
# all values set to 0.  It is adjusted so that actuator will remain
# at the Neutral-Position during the MVC.

   STIM1 = ( 0 * RAMP(;100) ) + (-2 * NP)

# Define local experimental details for TA MVC

  STIM2 = STIMMVC
  SAMRATE = 100
  COMMENT ='TA MVC; '_SUBJECT_'; Neutral-Position; '_TIME(;"S")

# Run experiment

  SET ERROR OFF
  SD2(STIM1,STIM2;'MVC.SAM',COMMENT,0,4,SAMRATE,0,?)
  SET ERROR ON
  .SD2

# Type CONTINUE if TA MVC trial successful.  Data will then be
# sorted and stored.

  PAUSE
  SRT(;AVGFILE,'A','MVC.SAM', , ,'POS','TOR','TA','TS', ,)
  .DELETE MVC.SAM;-1

# Torque interval means calculated and printed on screen.
# Largest value selected as MVC.

  TOR = RCL(;AVGFILE,1,'Y',2,1,1500,.00244)
  TORMEANS = DSU(TOR;'M',100)
  TYP (TORMEANS)
  MVCTA = MAX(TORMEANS) - MIN(TORMEANS)

# Define local experimental details for TS MVC.

  STIM2 = -STIMMVC
  SAMRATE = 100
  COMMENT ='TS MVC; '_SUBJECT_'; Neutral-Position; '_TIME(;"S")
```

```
#  Run experiment

   SET ERROR OFF
   SD2(STIM1,STIM2;'MVC.SAM',COMMENT,0,4,SAMRATE,0,?)
   SET ERROR ON
   .SD2

#  Type CONTINUE if TA MVC trial successful. Data will then be
#  sorted and stored.

   PAUSE
   SRT(;AVGFILE,'A','MVC.SAM', , ,'POS','TOR','TA','TS', ,)
   .DELETE MVC.SAM;-1

#  Torque interval means calculated and printed on screen.
#  Largest value selected as MVC.

   TOR = RCL(;AVGFILE,2,'Y',2,1,1500,.00244)
   TORMEANS = DSU(TOR;'M',100)
   TYP (TORMEANS)
   MVCTS = MIN(TORMEANS) - MAX(TORMEANS)

   RETURN

#  End of Module MVC.
```

### **Module EXPT1**

```
#  Module to sample data during first experiment.

#  Define local experimental details.

   COMMENT = 'Expt1; '_SUBJECT_'; Ankle Angle = STR(NEWPOS)_' Deg.; '
   COMMENT = COMMENT'_TIME(;"S")
   SAMRATE = 400

#  Generate STIM1 by adjusting PRBS to reflect the desired ankle position
#  (Channel NEWPOS) and the subject's neutral position (Channel NP).
#  Calibrate NEWPOS (degrees to volts).

   STIM1 = PRBS + ( (-2 * NP) + (-NEWPOS * CAL) )

#  Run trial.

   SET ERROR OFF
   SD2(STIM1,STIM2; 'PAS.SAM',COMMENT,0,4,SAMRATE,0,?)
   SET ERROR ON
   .SD2

#  Type  CONTINUE  if data successfully collected; data will then be
#  averaged and stored.

   PAUSE
   SRA(;AVGFILE,'A',S,1,1000,25,,'POS',PS,'TOR',TS,'TA',TAS,'TS',TSS,,)
   .DEL PAS.SAM;*

   RETURN

#  End of Module EXPT1.
```

## Module EXPT2

```
#  Module to sample data during second experiment.

#  Adjust STIM1 for appropriate tonic contraction.

   IF TONIC[TONICNUM] == 1 THEN TYPE = 'TA'
      ELSE TYPE = 'TS'
   IF TONIC[TONICNUM] == 1 THEN MVC = TAMVC
      ELSE MVC = TSMVC

#  Define local experimental details.

   COMMENT = 'Expt2; '_SUBJECT_'; Ankle Angle = STR(NEWPOS)_' Deg.; '
   COMMENT = COMMENT_'Tonic '_TYPE_' ; '_TIME(;"S")
   SAMRATE = 400

#  Adjust STIM1 by altering PRBS (generated in Module STIMGENII) to
#  reflect the desired ankle position (Channel NEWPOS) and the subject's
#  neutral position (Channel NP).  Calibrate NEWPOS (degrees to volts).

   STIM1 = PRBS + ( (-2 * NP) + (-NEWPOS * CAL) )

#  Generate STIM2 by adjusting the tracking stimulus (Channel TRACK)
#  to the appropriate direction and magnitude (Channel MVC).

   STIM2 = TRACK * MVC

#  Run trial.

   SET ERROR OFF
   SD2(STIM1,STIM2; 'ACT.SAM',COMMENT,0,4,SAMRATE,0,?)
   SET ERROR ON
   .SD2

#  Type  CONTINUE  if data successfully collected; data will then be
#  averaged and stored.

   PAUSE
   S = 'ACT.SAM'
   SRA(;AVGFILE,'A',S, 3001,1000,5,,'POS',PS,'TOR',TS,'TA',TAS,'TS',TSS,,)
   SRA(;AVGFILE,'A',S,11001,1000,5,,'POS',PS,'TOR',TS,'TA',TAS,'TS',TSS,,)
   SRA(;AVFFILE,'A',S,19001,1000,5,,'POS',PS,'TOR',TS,'TA',TAS,'TS',TSS,,)
   SRA(;AVGFILE,'A',S,27001,1000,5,,'POS',PS,'TOR',TS,'TA',TAS,'TS',TSS,,)
   SRA(;AVGFILE,'A',S,35001,1000,5,,'POS',PS,'TOR',TS,'TA',TAS,'TS',TSS,,)
   SRA(;AVGFILE,'A',S,41001,1000,5,,'POS',PS,'TOR',TS,'TA',TAS,'TS',TSS,,)
   .DEL ACT.SAM;*

   RETURN

#  End of Module EXPT2.
```

# APPENDIX IV

## NEXUS MODULES FOR DATA ANALYSIS

**Module NONPARAM:** To calculate the non-parametric passive and active mechanics.

**Module PARAM:** To calculate the stiffness parameters from the compliance inpulse response functions.

**Module REFLEX:** To identify the TA and TS stretch reflex dynamics.

## Module NONPARAM

```
# Module to calculate non-parametric passive or active mechanics.

# Define analysis details.

  POSRANGE = 'Pos rad'
  TORRANGE = 'Tor Nm'
  TARANGE = 'TA EMG uV'
  TSRANGE = 'TS EMG uV'
  START = 1
  LEN = 1000
  AVGFILE = 'Subject_File_Name'
  INDEX = Data_Storage_Cases

# Initialize analysis variables.

  MEANPOS = ''
  MEANTOR = ''
  MEANTA = ''
  MEANTS = ''
  POSRAN = ''
  VAFTP = ''
  POSOFF = Position_Offset
  TOROFF = Torque_Offset
  TAOFF = TA_EMG_Offset
  TSOFF = TS_EMG_Offset
  CALCASE = Data_Storage_Case_for_Actuator/Cast_Calibration

# Identify actuator/cast dynamics

  POS = RCL(; AVGFILE,CALCASE,'Y',1,START,LEN,1)
  TOR = RCL(; AVGFILE,CALCASE,'Y',2,START,LEN,1)
  POS = POS - MEAN(POS)
  TOR = TOR - MEAN(TOR)
  CALIMP = FIL(POS,TOR;200,"Y")
  .DEL POS.NXC;*
  .DEL TOR.NXC;*

# Analyze next case.  Each case contains data for one tonic level
# at one ankle angle for one subject.

  COUNT = 0
  [NEXTCASE]
       COUNT = COUNT + 1
       CASE = INDEX {COUNT}

#      Recall data and determine ensemble means.

       POS = RCL(; AVGFILE,CASE,'Y',1,START,LEN,1)
       POS = POS - POSOFF
       ST1(POS;'Y')
```

```
        MEANPOS = MEANPOS_MEAN
        POSRAN = POSRAN_(MAX - MIN)
        POS = POS - MEAN (POS)
        POS = CDET(POS;'R',POSRANGE,)

        TOR = RCL(; AVGFILE,CASE,'Y',2,START,LEN,1)
        TOR = TOR - TOROFF
        MEANTOR = MEANTOR_MEAN (TOR)
        TOR = TOR - MEAN(TOR)
        TOR = CDET(TOR;'R',TORRANGE,)

        TA = RCL(; AVGFILE,CASE,'Y',3,START,LEN,1)
        TA = TA - TAOFF
        MEANTA = MEANTA_MEAN(TA)

        TS = RCL(; AVGFILE,CASE,'Y',4,START,LEN,1)
        TS = TS - TSOFF
        MEANTS = MEANTS_MEAN(TS)

#       Remove actuator/cast dynamics

        PREDTQ = EXT(FLT(CALIMP,POS;"Y");START+100,800)
        TOR = EXT(TOR;START+100,800) - PREDTQ
        EXECUTE 'TOR'_STR(CASE)_' = TOR'
        POS = EXT(POS; START+100,800)

#       Calculate compliance impulse response function.

        EXECUTE 'ITP'_STR(CASE)' = FIL(TOR,POS;201,"Y",)'
        VAFTP = VAFTP_VAF

#       Calculate stiffness transfer function gain, phase and coherence.

        EXECUTE 'GAIN'_STR(CASE)_'= TS2(POS,TOR; "G", 200, 200)'
        EXECUTE 'PHASE'_STR(CASE)_'= TS2(POS,TOR; "P", 200, 200)'
        EXECUTE 'COH'_STR(CASE)_'= TS2(POS,TOR; "C", 200, 200)'

#       Delete Redundant files

        .DEL POS.NXC;*
        .DEL TOR.NXC;*
        .DEL PREDTQ.NXC;*
        .DEL TA.NXC;*
        .DEL TS.NXC;*
        .DEL NEX*.NXC;*

#       Loop through for next case.

        IF COUNT < LEN(INDEX) THEN GOTO [NEXTCASE]

# End of Module NONPARAM.
```

### Module PARAM

```
#  Module to calculate stiffness parameters (I, B and K) from
#  compliance impulse response function.

#  Define analysis details.

   INDEX = Data_Storage_Cases

#  Initialize analysis variables.

   I = ''
   B = ''
   K = ''
   VAFFIT = ''

#  Analyze next case.  Each case contains data for one tonic level
#  at one ankle angle for one subject.

   COUNT = 0
   [NEXTCASE]
        COUNT = COUNT + 1
        CASE = INDEX {COUNT}

#       Initialize parameters (P1, P2, P3) and tolerance values
#       (B1, B2, B3).

        P1 = .01
        P2 = 100
        P3 = .5
        B1 = .01
        B2 = 100
        B3 = .3

#       Smooth compliance impulse response function identified in Module
#       NONPARAM and determine I, B and K by fitting this function to
#       second-order equation where P1 = gain, P2 = natural frequency and
#       P3 = damping factor for P3<1.

        H = -EXT(SMO(ITP'_STR(CASE)_';2);100,100)
        FIT = NLF(H; ,P1,B1,P2,B2,P3,B3,'N', ,40, , ,.00001,)
        VAFFIT = VAFFIT_VAF
        I = I_(1 / (P1 * P22))
        B = B_(2*P3 / (P1 * P2))
        K = K_(1 / P1)
```

```
#       Delete redundant channels

        .DEL VAF.NXC;*
        .DEL P1.NXC;*
        .DEL P2.NXC;*
        .DEL P3.NXC;*
        .DEL B1.NXC;*
        .DEL B2.NXC;*
        .DEL B3.NXC;*
        .DEL H.NXC;*
        .DEL FIT.NXC;*
        .DEL NEX*.NXC;*

#       Loop through to next case

         IF COUNT < LEN(INDEX) THEN GOTO [NEXTCASE]

#   End of Module PARAM
```

**Module REFLEX**

```
#  Module to identify TA and TS stretch reflex dynamics

#  Define analysis details.

   VELRANGE = 'Vel  rad/s'
   TARANGE = 'TA EMG uV'
   TSRANGE = 'TS EMG uV'
   START = 1
   LEN = 1000
   AVGFILE = 'Subject_File_Name'
   INDEX = Data_Storage_Cases
   TONICTYP = 1  # (if TA contraction, otherwise = -1)
   STA1 = Start_Index_TA_Reflex_First_Negative_Peak
   LTA1 = Length_TA_Reflex_First_Negative_Peak
   STA2 = Start_Index_TA_Reflex_Positive_Peak
   LTA2 = Length_TA_Reflex_Positive_Peak
   STA3 = Start_Index_TA_Reflex_Second_Negative_Peak
   LTA3 = Length_TA_Reflex_Second_Negative_Peak
   STS1 = Start_Index_TS_Reflex_Negative_Peak
   LTS1 = Length_TS_Reflex_Negative_Peak
   STS2 = Start_Index_TS_Reflex_Positive_Peak
   LTS2 = Length_TS_Reflex_Positive_Peak

#  Initialize analysis variables.

   VAFVTA = ''
   VAFVTS = ''
   PEAKTA1 = ''
   PEAKTA2 = ''
   PEAKTS = ''

#  Analyze next case.  Each case contains data for one tonic level
#  at one ankle angle for one subject.

   COUNT = 0
   [NEXTCASE]
        COUNT = COUNT + 1
        CASE = INDEX (COUNT)

#       Analysis differs for TA or TS reflexes.

         IF TONICTYP == 1 THEN GOTO [TA]
            ELSE GOTO [TS]

        [TA]
#       Recall data, differentiate position and remove means.

        POS = RCL(; AVGFILE,CASE,'Y',1,START,LEN,1)
        VEL = DIF(POS)
        VEL = VEL - MEAN (VEL)
        VEL = CDET(VEL;'R',VELRANGE,)
```

```
          TA = RCL(; AVGFILE,CASE,'Y',3,START,LEN,1)
          TA = TA - MEAN (TA)

#         Identify TA reflexes.

          EXECUTE 'IVTA'_STR(CASE)_' = FIL(VEL,TA;100,"N",)'
          VAFVTA = VAFVTA_VAF

#         Determine Peak-to-Peak Magnitudes.

          EXECUTE IMP = SMO(IVTA'_STR(CASE)_';3)'
          PEAK = -MIN(EXT(IMP;STA1,LTA1)) + MAX(EXT(IMP;STA2,LTA2))
          PEAKTA1 = PEAKTA1_PEAK
          PEAK = -MIN(EXT(IMP;STA3,LTA3)) + MAX(EXT(IMP;STA2,LTA2))
          PEAKTA2 = PEAKTA2_PEAK

#         Delete Redundant Files

          .DEL POS.NXC;*
          .DEL VEL.NXC;*
          .DEL TA.NXC;*
          .DEL PEAK.NXC;*

          GOTO [CASEEND]

          [TS]

#         Recall data, rectify velocity and remove means.

          PVEL = THR (VEL;!)
          PVEL = PVEL - MEAN(PVEL)
          TS = RCL(; AVGFILE,CASE,'Y',4,START,LEN,1)
          TS = TS - MEAN(TS)

#         Identify TS reflexes.

          EXECUTE 'IVTS'_STR(CASE)_' = FIL(PVEL,TS;100,"N",)'
          VAFVTS = VAFVTS_VAF

#         Determine Peak-to-Peak Magnitudes.

          EXECUTE IMP = SMO(IVTS'_STR(CASE)_';3)'
          PEAK = -MIN(EXT(IMP;STS1,LTS1)) + MAX(EXT(IMP;STS2,LTS2))
          PEAKTS = PEAKTS_PEAK

#         Delete Redundant Files

          .DEL POS.NXC;*
          .DEL PVEL.NXC;*
          .DEL TS.NXC;*
          .DEL PEAK.NXC;*

          [CASEEND]
          IF COUNT < LEN(INDEX) THEN GOTO [NEXTCASE]

#    End of Module REFLEX
```

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