From the Laboratory of Biomechanics, Orthopaedic Hospital, Aarhus, Denmark

Stability of the Ankle Joint

Analysis of the Function and Traumatology of the Ankle Ligaments

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Chapter I

Introduction

The ankle joint is traditionally considered a hinge joint permitting a rotatory movement in the sagittal plane – the dorsal and plantar flexion of the foot. However, this is a gross simplification of the extremely complicated function of the ankle. As early as 1890 Tillaux observed that there might also be a certain rotation in the horizontal plane – i.e. internal or external rotation of the talus. Moreover, Bonnin (1944) and Rubin and Witten (1960) established that appreciable rotation in the frontal plane could be induced passively, manifesting itself primarily as adduction of the talus. Barnett and Napier (1952) and Hicks (1953) demonstrated that dorsiplantar flexion occurs on an axis changing in direction during the movement from maximum dorsiflexion to maximum plantar flexion. Calculating instant centres, Sammarco et al. (1973) D'Ambrosia et al. (1976), and Parlasca et al. (1979) showed that this change in the axis is continuous in the course of the dorsiplantar movement. Merely this change in the axis implies that the dorsiplantar movement cannot be restricted to the sagittal plane, but must be associated with a certain rotation in other planes too.

Stability in the ankle joint is secured passively in part by the shape of the bones that make up the joint – i.e. the distal part of the tibia with the medial malleolus, the lateral malleolus and trochlea of the talus – and partly the ligamentous structures which surround the joint laterally, medially and between the distal portions of the tibia and fibula.

Injuries to the ligaments of the ankle joint are common and are often sustained during sports performances (Broström 1965, Solheim and Aasen 1976, Crean 1981, Ştarke et al. 1981). Considering the increase in athletic activities through recent years, ruptures in the ligamentous structures of the ankle joint may be assumed to constitute a growing problem, as they may give rise to chronic instability and posttraumatic osteoarthritis (Kelley and Janes 1956, Niethard 1975, Dejour 1975, Harrington 1979, Althoff et al. 1981, Sukosd 1981, Zingher et al. 1981). The specific role of the individual ligamentous structures in ankle stability still remains uncertain. It has best been elucidated in the case of the lateral collateral ligaments, but even here it is difficult to draw definite conclusions – owing to differences in the technique, methods, and materials of previous authors.

On this background the present study was undertaken, partly to design an apparatus whereby it was possible to clarify the joint-stabilizing function of ligamentous structures in general and partly to employ this apparatus for analysing the ligaments of the ankle joint, the mechanisms at which they may rupture, and the types of instability that may be caused by such ruptures.

The present volume has been preceded by previous publications on the ankle joint ligaments. The material from these previous studies, plus a few supplements, also makes up the material of the present report. However, the total material affords a possibility of drawing further conclusions and permits certain revisions of conclusions published previously.

Previous publications:

I Anterolateral Rotational Instability in the Ankle Joint. An Experimental Study of Anterolateral Rotational Instability, Talar Tilt, and Anterior Drawer Sign in Relation to Injuries to the Lateral Ligaments.
Ove Rasmussen and Ib Tovborg-Jensen.
Acta Orthop. Scand. 52, 99-102, 1981.

II Mobility of the Ankle Joint. Recording of Rotatory Movements in the Talocrural Joint in vitro with and without the Lateral Collateral Ligaments of the Ankle.
Ove Rasmussen and Ib Tovborg-Jensen.
Acta Orthop. Scand. 53, 155-160, 1982.

III An Analysis of the Function of the Posterior Talofibular Ligament.
Ove Rasmussen, Ib Tovborg-Jensen and Jess Hedeboe.
Internat. Orthop. 7, 41-48, 1983.

IV Ligament Function and Joint Stability Elucidated By a New Technique.
Ove Rasmussen and Kurt Andersen.
Engineering in Med. 11, 77-81, 1982.

V Distal Tibiofibular Ligaments. Analysis of Function.
Ove Rasmussen, Ib Tovborg-Jensen and Svend Boe.
Acta Orthop. Scand. 53, 681-686, 1982.

VI Deltoid Ligament. Functional Analysis of the Medial Collateral Ligamentous Apparatus of the Ankle Joint.
Ove Rasmussen, Claus Kromann-Andersen and Svend Boe.
Acta Orthop. Scand. 54, 36-44, 1983.

VII Experimental Ankle Injuries. Analysis of the Traumatology of the Ankle Ligaments.
Ove Rasmussen and Claus Kromann-Andersen.
Acta Orthop. Scand. 54, 356-362, 1983.

Below, these publications will be referred to as I-VII.

Chapter II

2.1 Nomenclature

The terms used in the literature to designate movements in the ankle and subtalar joints are extremely confusing, one movement being called by several names and the same name being taken to mean movements into different directions. In the present study the following nomenclature will be used:

Dorsiflexion:
Tibiotalar movement in the sagittal plane whereby the tip of the foot turns upwards.

Plantar flexion:
Tibiotalar movement in the sagittal plane whereby the tip of the foot turns downwards.

Adduction:
Tibiotalar movement in the frontal plane whereby the hindfoot turns medially, and a laterally open angle forms between the joint surfaces on the tibia and talus. This movement is sometimes designated talar tilt.

Abduction:
Tibiotalar movement in the frontal plane whereby the hindfoot turns laterally, and a medially open angle forms between the tibia and talus. This movement is also designated negative talar tilt.

Internal rotation:
Tibiotalar movement in the horizontal plane whereby the tip of the foot turns medially.

External rotation:
Tibiotalar movement in the horizontal plane whereby the tip of the foot turns laterally.

Supination:
Subtalar movement consisting of internal rotation as well as adduction of the forefoot.

Pronation:
Subtalar movement consisting of external rotation and abduction of the forefoot.

2.2 Abbreviations

ADS:	anterior drawer sign
ATaFL:	anterior talofibular ligament
ATFL:	anterior tibiofibular ligament
ATTL:	anterior tibiotalar ligament, incl. the tibionavicular ligament
CFL:	calcaneofibular ligament
ITTL:	intermediate tibiotalar ligament
PTaFL:	posterior talofibular ligament
PTFL:	posterior tibiofibular ligament
PTTL:	posterior tibiotalar ligament
TCL:	tibiocalcaneal ligament
TT:	talar tilt

2.3 Anatomy

The general anatomy of the ankle joint is of course well-known, but seeing that a more detailed knowledge of the ligament structures is of particular importance in our connection, a brief description thereof will be given below:

The anterior talofibular ligament (ATaFL) is an approx. 5 mm wide band situated in the anterolateral joint capsule. Its attachment is the anterior aspect of the lateral malleolus, close to its apex, and it proceeds, when the ankle joint is in the neutral position, almost horizontally forward and medially to gain insertion on the lateral aspect of the talar neck. As with the other collateral ligaments, the direction of its fibres varies according to the degree of dorsal or plantar flexion in the ankle joint (Fig. 1 a and b).

The calcaneofibular ligament (CFL) is an extracapsular, styloid ligament having a diameter of about 5 mm and a length of 20-25 mm. It extends from the tip of the lateral malleolus to the lateral surface of the calcaneus (Fig. 1 a and b). At a neutral position in the ankle joint, its direction may range from almost vertical downwards to almost horizontal backwards (Ruth 1961).

The posterior talofibular ligament (PTaFL) is a strong, intracapsular ligament originating in the fossa of the lateral malleolus and spreads in the shape of a fan, its anterior, short fibres inserting laterally on the posterior edge of the talus and its posterior, long fibres medially on the lateral tu-

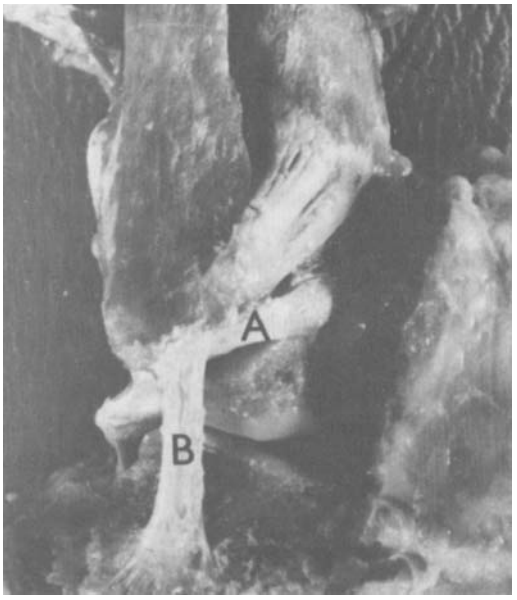


Fig. 1a: Ankle joint in dorsiflexion. ATaFL almost horizontally directed (A) and CFL vertical (B).

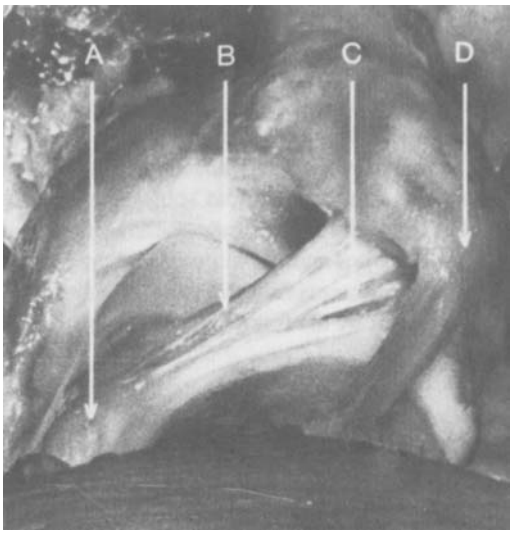


Fig. 2: PTaFL viewed infero-postero-laterally. A: posterior process of talus, B: long fibres of PTaFL, C: short fibres of PTaFL, D: apex of lateral malleolus.

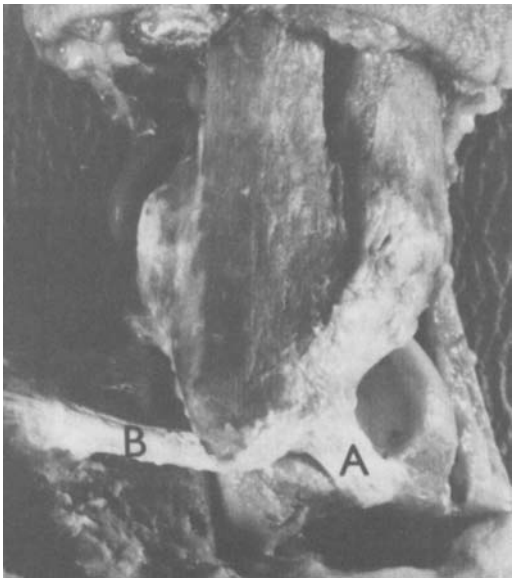


Fig. 1b: Ankle joint in plantar flexion. ATaFL oblique-plantarwards (A) and CFL horizontal (B).

bercle of the posterior process of the talus (Fig. 2) (de Vogel 1970, Prins 1978).

The anterior tibiofibular ligament (ATFL) is a strong ligament, 20 mm wide and 20-30 mm long, originating in the anterior tubercle of the tibia and the anterolateral part of the tibial epiphysis. Its fibres proceed in an oblique distal and lateral direction in order to insert anteromedially on the upper portion of the lateral malleolus.

The syndesmosis between the distal parts of the tibia and fibula is also designated the interosseous ligament. Some authors take the syndesmosis to represent the total ligament complex: The anterior tibiofibular ligament, the interosseous ligament, and the posterior tibiofibular ligament. In the present paper the term syndesmosis will be applied exclusively to the actual, false joint between the tibia and fibula, whereas the ligaments on either side of it will be considered collateral ligaments. The syndesmosis constitutes the distal prolongation of the interosseous membrane. Its fibres originate in the greater part of the fibular notch and insert at the same level on the anterior two-thirds of the medial fibular surface.

The posterior tibiofibular ligament (PTFL) is a strong ligament measuring about 5 mm × 20 mm

× 30 mm which runs from the distal, lateral part of the tibial epiphysis in an oblique course distally and laterally to the insertion on the posterior surface of the lateral malleolus. Deep down there are more transversely proceeding fibres, sometimes designated the transverse tibiofibular ligament. Here they are considered to be part of the posterior tibiofibular ligament.

The deltoid ligament is a large, strong ligament spreading fan-shaped over the medial part of the ankle joint, as it originates in the medial malleolus and inserts on the navicular bone, talus, and calcaneus. There have been wide variations in the descriptions of its details. Bonnin (1950) and Dziob (1956) have stated that it consists of fibres in one layer, while others report that it consists partly of a superficial and partly of a profound layer (Grath 1960, Broström 1964, de Vogel 1970, Gerbert 1975). The most detailed description of its anatomy is by Pankovich and Shivaram (1979) who make a distinction between a superficial part – the tibionavicular ligament, the tibiocalcaneal ligament, and the superficial talotibial ligament – and a profound part consisting of the anterior and posterior tibiotalar ligaments. They admit, however, that the individual structures are not sharply demarcated from each other. In the present paper, in view of the different function of the various structures, the ligament will be divided into four structures (Fig. 3):

The tibiocalcaneal ligament (TCL) constitutes the superficial part of the deltoid ligament and originates a couple of cm up the medial surface of the medial malleolus. Its fibres extend distally-posteriorly to the attachment on the medial surface of the calcaneus.

The anterior tibiotalar ligament (ATTL) originates anteromedially and distally in the tibial epiphysis and proceeds distally and forward to insert on the neck of the talus. The most superficial fibres continue across the talus and insert on the navicular bone. In the present paper these fibres will be included in the anterior tibiotalar ligament. This ligament makes up the anterior portion of the deep layer of the deltoid ligament.

The intermediate tibiotalar ligament (ITTL) has been included by some authors either in the anterior or the posterior tibiotalar ligament. It originates in the tip of the medial malleolus and proceeds distally and posteriorly to its insertion on the talus, deep to the tibiocalcaneal ligament. Its fibres are only a few mm in length, but very strong.

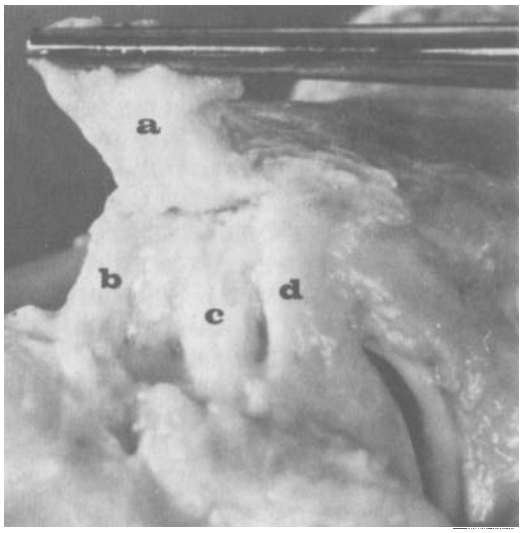


Fig. 3: Deltoid ligament viewed infero-postero-medially. a: TCL, b: ATTL, c: ITTL, d: PTTL.

The posterior tibiotalar ligament (PTTL) is a short, strong ligament extending from the posteromedial aspect of the medial malleolus to the posteromedial part of the talus. It is situated on a level with the anterior and intermediate tibiotalar ligaments and consitutes the posterior, deep portion of the deltoid ligament.

2.4 **Mobility in the Normal Ankle Joint**

A movement may be translatory – i.e. rectilinear – or rotatory, around an axis. In a biomechanical system there are often combinations of the two, although the rotatory movements play by far the greater role in the ankle joint. Movements may be characterized by the direction or the plane in which they take place. In our context, therefore, it is expedient to consider the movements of the ankle joint in the sagittal, frontal, and horizontal planes, taking into consideration all the time that the function of the ankle joint is so complex that one movement is hardly restricted to just the same plane. The named planes may be considered as the planes in a 3-dimensional coordinate system.

2.4.1 **Movement in the sagittal plane** is the most striking movement in the ankle joint, corresponding to dorsal and plantar flexion of the foot. The extent of the normal dorsiplantar movement has been studied, by varying methods, by a number of authors (Glanville and Kreezer 1937, Bonnin 1950, Weseley et al. 1969, Sammarco et al. 1973, Segal 1979, Boone and Azen 1979, Lindsjö 1981). However, their statements vary – in the case of dorsiflexion between 10° and 51° and in the case of plantar flexion from 15° to 56°. Krämer and Gudat (1980), studying 75 ankle joints, found the total mobility in the sagittal plane to amount to 62.2° Apparently, however, some of the named authors have included the subtalar mobility in their statements.

Some translatory movement can also be produced passively in this plane – the so-called anterior drawer sign (ADS). It is often interpreted as a sign of rupture of the ATaFL, but it can also be produced on an intact ankle joint. At intact ligaments the ADS has been reported to range between 1.5 and more than 9 mm (Landeros et al. 1968, Delplace and Castaing 1975, Laurin and Matthieu 1975, Hackenbruch and Noesberger 1976, Larsen 1976, Dannegger 1979, Hackenbruch et al. 1979, Frölich et al. 1980), depending presumably upon the force used to induce the movement, but also varying according to the method by which it has been measured (Fig. 4).

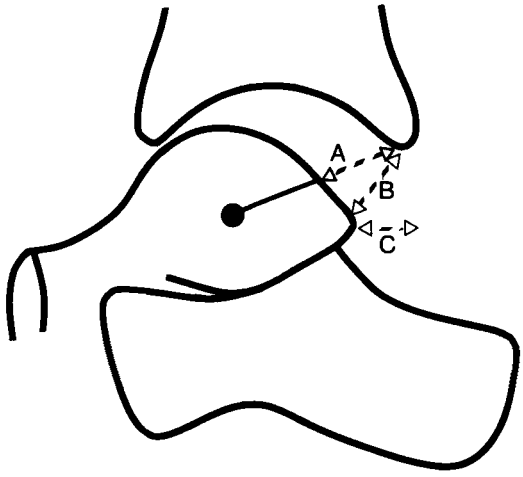


Fig. 4: Various ways of measuring an ADS radiologically.
A: After Dannegger (1979)
B: After Hackenbruch (1979)
C: After Johannsen (1978).

2.4.2 **Movement in the frontal plane** takes place around a sagittal axis and consists in adduction or abduction of the talus in the ankle mortise. It can be measured radiologically, either by the distance between the talus and the tibial joint surface at the lateral or medial edge of the trochlea or – more expediently – by the laterally or medially open angle which can be produced between the joint surfaces of the tibia and the talus.

The extent of movement in this plane has been widely discussed. It is largely agreed that in an intact ankle joint there occurs no abduction, but nevertheless Husfeldt (1939) did find a few degrees of abduction. Reports on adduction are more varied. In the opinion of some authors adduction cannot be induced at all, or at least of no more than 5° (Pennal 1943, Anderson and Lecocq 1954, Lee 1957, Staples 1965, Pascoet et al. 1972, Cox and Hewes 1979), while others say it may amount to 5°-10° (Goldstein 1948, Frölich et al. 1980, Glasgow et al. 1980). Others again have reported 10°-20° (Coltart 1951, Sedlin 1960, Edeiken and Cotler 1978, Brooks et al. 1981), while a few (Bonnin 1950, Rubin and Witten 1960, Laurin et al. 1968, Quellet et al. 1968) have found even higher values. According to Bonnin (1950), however, the adduction is generally below 5°, while higher values occur only in individuals with "hypermobile ankles". Among a material of 404 ankle joints, Cox and Hewes (1979) could not produce any adduction in 90 %, whereas almost 8 % could be adducted 1°-5°. Judging by the literature, then, the extent of movement in this plane is somewhat uncertain.

2.4.3. **Movement in the horizontal plane** takes place around a vertical axis through the talus, the movement being an internal or external rotation of the talus in the ankle mortise. As the talar trochlea is not shaped like a transversely placed cylinder, but rather as the segment of a cone, and as the mean axis on which the dorso-plantar movement occurs is not horizontal, a certain internal and external rotation must necessarily take place

in the ankle joint during movements in the sagittal plane. This is, furthermore, enabled by a certain laxity of the medial collateral ligament (Inman 1976). In the words of Wyller (1963), the talus may be considered a badly mounted wheel.

Several authors have mentioned this horizontal rotation without stating its extent (Kleiger 1956, de Vogel 1970, Shoji et al. 1976, Shoji et al. 1977, Morris 1977, Wirth et al. 1978). Close (1956) has reported that non-forced horizontal rotation during the dorsiplantar movement amounts to 5°-6°. In Larsen's (1976) opinion, however, there may occur an internal rotation of 2°-3° and an external rotation of 5°. Parlasca et al. (1979) have estimated the total rotation in the horizontal plane at about 2°, while Laughman et al. (1980) found internal rotation during walking to be 0°-1° and external rotation 4°-5° – but this included any subtalar rotation.

Only a few authors have reported on the extent of the possible horizontal rotation when exerting a known, transverse force upon the talus or foot. In studies of amputated legs, however, McCullough and Burge (1980) observed that the movement – apparently comprising internal as well as external rotation – amounted to about 24°. Johnson et al. (1981) reported a forced, horizontal rotation of 14°-15°, but do not state which force they used to affect the talus.

2.5 **Abnormally Increased Mobility – Instability**

A normal ankle joint can move within the physiological limits. If they can be surpassed, actively or passively, the stabilizing structures are insufficient. The ankle joint can be moved beyond normal – viz. it is *unstable*.

Theoretically, such an instability might occur in any plane and in almost any direction, depending upon which parts of the stabilizing ligamentous structures are insufficient. If a trauma in a given direction causes a ligament rupture, the joint must become unstable in that direction, unless the rupture heals without lengthening. Reversely, an ankle joint which is unstable in a given direction must house an injury to that structure which was to maintain stability in that very direction. The next chapter reviews the literature on the experimental investigations which have so far formed the basis of interpreting the function and traumatology of the individual ligaments.

Chapter III

Previous Experimental Studies

The function of the ankle ligaments has previously been investigated, either by acting upon the ankle joint in a given direction and registering the resulting ligament tensions, ligament tears, or avulsion fractures or by recording the changes in mobility occurring after transection of the various ligaments. As a matter of course, nearly all these investigations have been performed on osteoligamentous preparations.

3.1 Observations During Forced Movements in Given Directions

3.1.1 Dorsiflexion:

As early as 1835 Dupuytren induced experimental fractures of the malleoli in cadaver legs. He observed that forced dorsiflexion entailed ruptures in the collateral ligaments of the ankle joint. In the same situation, Hönigschmied (1877) observed rupture of the deltoid ligament or fracture of the medial malleolus, associated with rupture of the PTaFL and at times also the CFL. Ashhurst and Bromer (1922) reported tightening of the ATFL, PTFL, and CFL during dorsiflexion and – independently of the position of the ankle joint – a constant tension of the PTaFL. Robichon et al. (1972) found tightening of the CFL and PTaFL, while de Vogel (1970) could experimentally induce isolated rupture of the most plantar fibres of the ATaFL by forced dorsiflexion. Unlike the above authors, Bonnin (1950) considered that dorsiflexion is not restricted by the ligaments, but by a collision between the anterior edge of the tibia and the talar neck. The most detailed study is Wirth et al.'s (1978) who ascertained, by strain gauge measurements, that dorsiflexion tightens the ATaFL, CFL, PTaFL, ATFL, as well as TCL.

Thus, dorsiflexion is probably not controlled by a single structure, but rather by an interaction of most of the ankle ligaments – presumably depending upon whether the foot is in internal or external rotation or in more or less ad- or abduction.

3.1.2 Plantar flexion:

By forced plantar flexion Hönigschmied (1877) first produced rupture of the deltoid ligament and ATaFL, and thereafter also of the CFL and PTaFL. Ashhurst and Bromer (1922), Bonnin (1950), Robichon et al. (1972), and Wirth et al. (1978) found the ATaFL to constitute the primary limitation to plantar flexion. However, Wirth et al. observed in plantar flexion also tension of the CFL and – though only to a slight extent – of the PTaFL, and Bonnin (1950) has also reported that plantar flexion is restricted by a collision between the posterior edge of the tibia and the posterior process of the talus. In their *in vivo* studies on amputation patients, Nevin and Post (1964) observed that forced plantar flexion primarily resulted in a rupture of the bifurcate ligament and anterior part of the deltoid ligament – presumably the ATTL, but possibly also of more posterior structures.

It might be expected perhaps that the ATTL and ATaFL would play the same role in plantar flexion. However, since this movement is associated with a certain amount of internal rotation (Parlasca et al. 1979) the ATaFL tightens first, and indeed this fits in with most authors' statements.

3.1.3 Adduction on dorsiflexed ankle joint:

Only a very few authors have tried to produce injuries by this movement. Makhani (1962) reported that by forcing the movement he obtained tightening of the talocalcaneal ligament, while during dorsiflexion the talus is so firmly locked in the mortise – because of the latter's shape and the relatively greater width of the talus anteriorly – that the collateral ligaments of the ankle joint are not strained. Nevertheless, Robichon et al. (1972) did find this movement to tighten the CFL.

3.1.4 **Adduction on neutrally positioned ankle joint:**

By this movement Maissonneuve (1840) and Lauge-Hansen (1942) sometimes produced fracture of the lateral malleolus, but most often it is said to result in rupture of the lateral collateral ligaments. There have been reports of ruptures of the ATaFL + CFL (Hönigschmied 1977, Hendelberg 1943), isolated rupture or tightening of the CFL (Hönigschmied 1877, Cosentino 1956, Robichon et al. 1972, Dias 1979, Taillard et al. 1981), rupture of the CFL + PTaFL (Lauge-Hansen 1949, Nevin and Post 1964), or rupture of the ATaFL as well as CFL and PTaFL (Hönigschmied 1877, Nevin and Post 1964).

3.1.5 **Adduction on plantar-flexed ankle joint:**

This movement produces primarily tension or rupture of the AtaFL (Cosentino 1956, Makhani 1962, Nevin and Post 1964, Dias 1979). According to Nevin and Post, however, such a rupture is not total, and it is associated with partial rupture of the vertical fibres of the CFL and rupture of the short fibres of the PTaFL. However, it is not quite evident what the vertical fibres are taken to mean, as the course of the CFL fibres in plantar flexion is practically horizontal (Fig. 1 b). Makhani (1962) has claimed that the CFL does not rupture on adduction in plantar flexion, while Dias (1979) stated that this produced an injury starting anteriorly in the ATaFL and spreading backwards to the CFL and possibly also the PTaFL. Incidentally, by further forcing the plantar flexion, he finally succeeded in also tearing the ATTL.

Thus, when the ankle is in the neutral position or dorsiflexed, the CFL appears to be the main factor which restricts adduction, as it is included in all the combinations reported. In plantar flexion, on the other hand, the ATaFL is tense and of a more vertical position (de Vogel 1970) (Fig. 1 b). In this position it forms a large, anteriorly open angle with the distal joint surface of the tibia and with the axis on which the adduction takes place. Thereby its adduction-inhibiting function must be increased. It is likely, therefore, that adduction in plantar flexion is inhibited by the ATaFL, while otherwise it is primarily limited by the CFL.

3.1.6 **Abduction on neutrally positioned ankle joint:**

The first stage of an injury produced by forced abduction seems to be rupture of the deltoid ligament or fracture of the medial malleolus (Maissonneuve 1840, Tillaux 1890, Lauge-Hansen 1942, Hendelberg 1943). If the movement is continued, it produces tears of the ATFL, syndesmosis, and PTFL (Tillaux 1890, Lauge-Hansen 1942), but such injuries were apparently not obtained by Hönigschmied (1877).

There have been no reports of injuries produced by forced abduction on dorsiflexed or plantar-flexed ankles.

Accordingly, there is little doubt that abduction is restricted primarily by the deltoid ligament, but this structure is composed of several elements whose specific function is not apparent from the named investigations. It is also not clear whether the distal, tibiofibular structures can rupture in an abduction trauma.

3.1.7 **Internal rotation on neutrally positioned ankle joint:**

By this movement Maissonneuve (1840) induced avulsion fracture in the lateral malleolus, at the insertion of the lateral collateral ligaments. Hönigschmied (1877) and Hendelberg (1943) also found rupture of the ATaFL and – sometimes – of the CFL. In one case, however, Hönigschmied could produce a spiral fracture of the tibia and in another case a similar fracture of the fibula. Dehne (1934) noted tightening of the ATaFL as well as PTTL in forced internal rotation.

ATaFL seems to be the essential structure in restricting internal rotation. The fracture of the lateral malleolus described by Maissonneuve is presumably interpretable as an avulsion fracture caused by traction from the ATaFL and perhaps the CFL. It still remains unelucidated whether the PTTL plays any role in this movement as claimed by Dehne.

The literature does not seem to have given any descriptions of forced internal rotation on the dorsiflexed or plantar-flexed ankle joint.

3.1.8 **External rotation on dorsiflexed ankle joint:**

Maissonneuve (1840) produced by this movement

either (a) a fracture of the lateral malleolus followed by rupture of the deltoid ligament or fracture of the medial malleolus or (b) rupture of the distal tibiofibular ligaments followed by fracture of the lateral or both malleoli.

3.1.9. **External rotation on neutrally positioned ankle joint:**
Hönigschmied (1877) has described two types of injuries caused by this movement. In part, he observed fracture of the lateral malleolus, sometimes combined with rupture of the deltoid ligament or fracture of the medial malleolus, and in part he produced rupture of the ATFL and syndesmosis plus – in one case – also of the PTFL, usually associated with rupture of the deltoid ligament. Hendelberg (1943) induced a medial malleolar fracture, while Frick (1978) reported that this was the only movement during which the distal tibiofibular structures could be torn without other simultaneous injuries.

3.1.10 **External rotation on plantar-flexed ankle joint:**
Injuries arising during this movement have been produced only by Maissonneuve (1840) who reported a tear of the deltoid ligament.

Apparently, then, external rotatory traumas may result in injuries laterally, tibiofibularly, and medially, but the sequence in which the various structures are hit remains unelucidated.

3.1.11 **Internal rotation on adducted ankle joint:**
Güttner (1941), by this movement, first induced a rupture of the ATaFL, thereafter partial rupture of the PTaFL, then of the CFL and lastly of the remaining part of the PTaFL. Lauge-Hansen (1946) and Dias (1979) also found rupture of the ATaFL to be the first injury, but thereafter Lauge-Hansen observed rupture of the CFL. So did Dias in some cases, while in others he first induced rupture of the short fibres of the PTaFL and not until thereafter of the CFL. Lastly, he also found a rupture of the deep fibres of the deltoid ligament.

In other words, it seems to be agreed that internal rotation on adducted ankle joint is restricted primarily by the ATaFL, while it is less certain whether the CFL or the PTaFL is the stabilizing factor next in order.

3.1.12 **Internal rotation on abducted ankle joint:**
As far as can be seen, Lauge-Hansen (1946) is the only one to have induced injuries by this movement which in his experiments did not cause ligament injuries or avulsion fractures, but instead a spiral fracture through the tibia.

3.1.13 **External rotation on adducted ankle joint:**
This movement corresponds to what Lauge-Hansen (1942) called supination-eversion whereby he produced the following: stage I rupture of the ATFL, stage II an oblique fracture distally in the fibula, stage III fracture of the posterior edge of the tibia, and lastly as stage IV also a fracture of the medial malleolus. According to his investigations, external rotation in this position of the ankle joint seems to be limited primarily by the distal tibiofibular ligaments. However, Lauge-Hansen later (1950) reported that the primary event is tightening of the tibiofibular structures + the CFL and PTaFL.

3.1.14 **External rotation on abducted ankle joint:**
By this movement Lauge-Hansen (1942) induced as stage I rupture of the deltoid ligament or fracture of the medial malleolus. In stage II there occurred moreover rupture of the ATFL and the syndesmosis between the tibia and fibula, and in stage III also oblique fracture of the fibula. Finally – as stage IV – the posterior edge of the tibia became avulsed. Thus, in external rotation of the foot the deltoid ligament, tense in abduction, seems most prone to damage.

3.2 Findings Following Transection of the Lateral Collateral Ligaments

3.2.1 **Transection of the ATaFL** is stated to entail increased mobility in the frontal plane, in the form of increased adduction (Dehne 1934, Quellet et al. 1968, Padovani 1975). Apparently, adduction increases most in plantar flexion (Pennal 1943, Leonard 1949, Cosentino 1956, Duquennoy et al. 1975) at which it increases by 5°-18° (Wirth and Artmann 1977, Johnson et al. 1981). In the neutral position, on the other hand, the last-mentioned

authors did not find any increase in adduction. Indeed, Carothers (1942), Fürmaier (1951), Anderson et al. (1952), and Castaing and Delplace (1972) do not think that isolated transection of the ATaFL increases adduction at all.

The ADS after cutting the ATaFL was first described by Dehne (1934). In a neutrally positioned ankle joint it is stated by Anderson et al. (1952) to be 4 mm as against 2 mm at intact ligaments, while in plantar flexion it is 7-8 mm, still compared with 2 mm at intact ligaments. Castaing and Delplace (1972) have reported an ADS up to about 10 mm, and Johnson et al. (1981) claim, as the only ones, that the greatest increase after transection of the ATaFL (about 4 mm) can be observed in dorsiflexion.

Increased internal rotation after cutting the ATaFL has been described by Dehne (1934), Hendelberg (1943), Leonard (1949), Anderson et al. (1952), and Castaing and Delplace (1972). Johnson et al. (1981) found an increase of the "torsion" – meaning presumably the internal rotation – of about 10° on the plantar-flexed ankle. Moreover, McCullough and Burge (1980), using simulated weightbearing of 15 kg and a transverse action upon the talus by 3 Nm, could measure an increase of up to about 6°.

Thus, it is doubtful to what extent stability in the frontal plane is affected by transection of the ATaFL. Presumably, adduction will be increased mainly in plantar flexion, if it is affected at all. On the other hand, it seems to be agreed that an increased ADS arises. According to Castaing and Delplace (1972), however, this is associated with some internal rotation, so that it partially represents anterolateral rotatory instability. Thus, the movement approaches the internal rotation found in the horizontal plane, and presumably it is not reasonable to make a distinction between these two types of instability.

3.2.2 **Cutting of the ATaFL + CFL** entails a marked increase in adduction (Carothers 1942, Pennal 1943, Leonard 1949, Makhani 1962), an increase which has been quantitated at between 13.5° (Pascoet et al. 1972) and 30° (Anderson et al. 1952). When the ankle joint is in the neutral position the adduction is increased too (Cosentino 1956), allegedly to between 20° and 30° (Duquennoy et al. 1975, Wirth and Artmann 1977).

The ADS also increases further (Padovani 1975, Delplace and Castaing 1975, Wirth and Artmann 1977).

Leonard (1949) and McCullough and Burge (1980) have reported that the horizontal rotation, by which they are presumably referring to internal rotation, is now greater than following isolated transection of the ATaFL. According to the latter authors, cutting of the CFL almost doubled the instability.

There is little doubt that transection of both the ATaFL and CFL increases adduction appreciably, although Padovani (1975) did not find it was greater than after isolated cutting of the ATaFL. Similarly, ADS as well as internal rotation presumably increase when the CFL is included in the transection.

3.2.3 **Cutting of the ATaFL + PTaFL** has been performed only by Castaing and Delplace (1972). Thereafter, they observed increased adduction and an ADS of 15 mm, but did not state at which degree of dorsal or plantar flexion the examination had been carried out.

3.2.4 **Transection of the ATaFL + CFL + PTaFL** does not, according to Fürmaier (1951), entail abnormal adduction as long as the distal tibiofibular ligaments are intact, while all other authors have reported that total transection of the lateral collateral ligaments renders the ankle joint utterly unstable.

3.2.5 **Isolated transection of the CFL** entailed, according to Carothers (1942) and Pennal (1943), only a minimal increase in adduction, while Anderson et al. (1952) found adduction of as much as 15°. Cosentino (1956) and Makhani (1962) state that the adduction increases only when the ankle joint is in the neutral position. In that position Wirth and Artmann (1977) measured an adduction of 10°, while in plantar flexion it amounted to only 5°. However, several authors have found no instability at all after isolated cutting of the CFL (Leonard 1949, Laurin et al. 1968, Quellet et al. 1968, Vidal et al. 1974, Padovani 1975).

The CFL seems to have but a slight influence upon the stability of the ankle joint, provided that other ligaments are intact. Possibly, it limits adduction, when the ankle joint is in the neutral position, while no data are available as to whether mobility in other planes alters after its transection.

3.2.6 **Cutting of the CFL + PTaFL,** according to Leonard (1949) and Cosentino (1956), gives rise to an increase in dorsiflexion, whereas Wirth and Artmann (1977) found adduction to be increased by 10° in plantar flexion and up to 30° in the neutral position. They also observed a posterior drawer sign – i.e. backward gliding of the talus.

It is comprehensible that a possible increase in adduction is greater in the neutral position than in plantar flexion, as an intact ATaFL in plantar flexion is tense and forms a relatively large angle with the axis around which adduction takes place (Fig. 1 b). In plantar flexion, therefore, it can restrict adduction, which is prevented by the CFL and PTaFL in the neutral position and in dorsiflexion (Fig. 1 a).

3.2.7 **Isolated transection of the PTaFL** entails increased dorsiflexion, though to a lesser extent than combined transection of the CFL and PTaFL (Leonard 1949, Cosentino 1956). Wirth and Artmann (1977) believe that a posterior drawer sign arises, while adduction remains unaffected.

3.3 Findings After Cutting the Distal Tibiofibular Ligaments

3.3.1 **Transection of the ATFL** does not, according to Laurin et al. (1968) and Quellet et al. (1968), increase adduction of the talus. On the other hand, there have been reports that the external rotation of the fibula (Hendelberg 1943) and its forward-backward shift (Henkemeyer et al. 1975) increase. Close (1956) observed increased external rotation and internal rotation of the talus, a finding confirmed for external rotation by Padovani (1975) who also observed a posterior drawer sign. The abnormal external rotation described by Close, however, amounted to only 1°-3°, and the internal rotation to even less. According to Ashhurst and Bromer (1922) cutting of the ATFL entailed diastasis in the ankle joint, the width of the mortise anteriorly increasing by about 10 mm, while Grath (1960) could produce an increase of only a couple of mm in width.

In other words, the mobility of the ankle joint does not appear to increase essentially after cutting of the ATFL. On the other hand, the increase in the width of the ankle mortise perhaps entails other complaints because of altered weightbearing conditions (Ramsay and Hamilton 1976, Kimizuka et al. 1980).

3.3.2 **Transection of the ATFL + the syndesmosis** between the tibia and fibula results, according to Close (1956), in a further, slight increase in external rotation, whereas Husfeldt (1938) found a lateral shift of the talus of 5 mm and an increased external rotation of the fibula itself in this situation.

3.3.3 **Transection of the ATFL + the syndesmosis + PTFL** causes a "spontaneous" increase in the width of the ankle mortise, as these structures are normally tense (Schumann 1955). Close (1956) has reported that external rotation of the talus increases by a further few degrees, just like Staples (1960) who observed a very slightly increased external rotation and abduction of the talus.

3.3.4 **The ATFL + PTFL** have been cut by Henkemeyer et al. (1975) and Padovani (1975) who do not expressly state whether they cut also the syndesmosis. Henkemeyer et al. found greater mobility of the fibula forward-backward than after isolated transection of the ATFL and an external rotation and shift into the lateral direction of the fibula, whereas Padovani could induce an increased internal and external rotation of the talus as well as an increased posterior drawer sign. Neither author, however, quantitated the instability.

3.3.5 **Isolated cutting of the PTFL** was done by Ashhurst and Bromer (1922) who found merely a negligible increase in the width of the mortise. There have been no reports on the influence of such a transection upon internal rotation.

3.4 Findings After Cutting the Medial Collateral Ligaments

3.4.1 According to Padovani (1975), **cutting the superficial part of the deltoid ligament** does not increase abduction, but paves the way for an increased ADS, combined with external rotation of the talus – i.e. an anteromedial rotatory instability.

3.4.2 **Transection of the anterior two-thirds of the deltoid ligament** was carried out by McCullough and Burge (1980) who found, on simulated weightbearing of 15 kg and an action upon the talus by 3 Nm, that this increased the horizontal rotation of the talus by about 7°. However, they did not specify the distribution between internal and external rotation.

3.4.3 **Transection of the entire deltoid ligament,** according to Hendelberg (1943), Fürmaier (1951), and Padovani (1975), causes a marked increase in abduction. The last-mentioned author also observed an increased ADS.

3.5 Findings After Combined Transection of the Lateral Collateral and Distal Tibiofibular Ligaments

3.5.1 **Transection of the ATaFL + ATFL,** Fürmaier (1951) says, increases adduction which according to him does not occur on isolated cutting of the ATaFL. Laurin et al. (1968) too found increased adduction of the talus, but did not mention whether it was greater than after isolated cutting of the ATaFL.

3.6 Findings After Combined Transection of the Medial Collateral and Distal Tibiofibular Ligaments

3.6.1 **Cutting of the ATFL + the anterior two-thirds of the deltoid ligament** was performed by McCullough and Burge (1980). On simulated weightbearing of 15 kg and an action upon the talus by 3 Nm, they found horizontal rotation – by which they probably mean external rotation – to increase by about 15° and after *further cutting of the PTFL* (and possibly the syndesmosis?) by another 6°-8°.

3.6.2 **Transection of the ATFL + syndesmosis + PTFL + the superficial part of the deltoid ligament** (Close 1956) resulted in an increased width of the mortise. After *further cutting of the deep fibres in the deltoid ligament,* the width increased even further. Padovani (1975) too performed these transections and found, besides, a distinctly increased external rotation of the talus.

3.7 Findings After Combined Transection of the Lateral and Medial Collateral Ligaments

3.7.1 **After cutting ATaFL + ATTL** Castaing and Delplace (1972) observed a marked ADS, but no instability in the frontal plane – i.e. no increase in ad- or abduction.

3.7.2 **The ATaFL + CFL + ATTL** have also been cut by Castaing and Delplace who stated that it caused increased adduction and an ADS of about 15 mm.

Chapter IV

Status after review of literature

4.1 Mobility of the Ankle Joint at Intact Ligaments

The extent of the most striking movement of the ankle joint – its dorsiplantar movement – as reported by the various authors – has differed appreciably. The reason is presumably that the movements have been measured by different methods, *in vivo* – with or without weightbearing – or on osteoligamentous preparations. Besides, some authors have included in their grading not only the movement in the talocrural joint, but also the subtalar movement – at times without saying so. Dorsiflexion has been reported to range from 10° to 51° and plantar flexion from 15° to 56°. According to Sammarco et al. (1973) the two movements are of approximately the same extent, whereas Boone and Azen (1979) claim that the extent of plantar flexion is 4-5 times that of dorsiflexion.

Also the degree of possible adduction is somewhat uncertain, the descriptions stating from 0° to more than 20°, while aparently there is agreement on no notable abduction in the ankle joint.

Possible internal rotation at intact ligaments has been reported by only a very few authors (McCullough and Burge 1980, Johnson et al. 1981), and their results differ considerably. The former authors used simulated weightbearing, and it must be mentioned that horizontal rotation in the ankle joint decreased appreciably with increasing weightbearing. As to the extent of movement in the joint without weightbearing there have been no definite statements.

External rotation in the talocrural joint has also been deficiently elucidated. According to Close (1956) it amounts to 5°-6°, but this applies only to the spontaneous external rotation of the talus which occurs during walking. No information seems to be available on the extent of the maximum possible external rotation of the talus.

4.2 On the Individual Ligaments

ATaFL seems to limit primarily internal rotation of the talus and also ADS. Possibly, this ligament has some inhibitory effect upon adduction, but it has not been clarified whether this applies only when the ankle joint is in plantar flexion. One author (Fürmaier 1951) even states that the ATaFL plays no role at all in adduction, as long as the ATFL is intact. Moreover, it possibly has a restrictive effect upon plantar flexion, as some authors have been able to rupture it by forced plantar flexion. One investigator (de Vogel 1970) even could produce rupture of its most plantar fibres by forced dorsiflexion, so that possibly it also limits movement in that direction.

CFL – by virtue of its situation and fibre direction – is expected to inhibit adduction, but it is uncertain whether it possesses this function as long as the other ligaments are intact. Some authors have been able to obtain increased adduction after cutting it – others have not. Nevertheless, it must be assumed that in certain positions of the ankle it restricts this movement primarily, as several authors have reported the production of isolated CFL rupture by forced adduction. Its role in dorsiplantar flexion is uncertain. Some authors believe that it tightens in maximum dorsiflexion, while others have found it tense in plantar flexion. Nor has its role in internal rotation after preceding rupture of the ATaFL been clarified, although it has been stated by Leonard (1949) and McCullough and Burge (1980) that this movement increases further when the CFL is included in the transection.

PTaFL presumably limits dorsiflexion, since some writers have been able to tear it during forced dorsiflexion – usually in combination with other ligamentous structures (Hönigschmied 1877). Likewise, cutting of this ligament is said to have entailed increased dorsiflexion (Leonard 1949, Cosentino 1956). It has not been possible to produce isolated injuries to this ligament. However, by forced adduction on the neutrally placed ankle joint Lauge-Hansen (1942) and Ne-

vin and Post (1964) have been able to tear the PTaFL together with the CFL. This indicates that these two structures combined limit adduction in the ankle joint in a neutral position. PTaFL – as also stated by de Vogel (1970) – consists of two anatomically and presumably also functionally different structures, the anterior short and the posterior long fibres. In the literature it has not been possible to find a proper analysis of the function of these two structures or an investigation of which movement can rupture them – together or possibly separately.

ATFL apparently influences rotation of the talus – and of the fibula – in the horizontal plane, as several investigators have been able to produce primary rupture of this ligament by forced external rotation, generally in combination with rupture of the syndesmosis (Maissonneuve 1840, Hönigschmied 1877), and as cutting of the ligament gives rise to a slight increase in external rotation (Close 1956, Padovani 1975). Lauge-Hansen (1942) induced rupture of the ATFL, as the first injury, by "supination-eversion", meaning in the present terminology external rotation of the talus on an adducted ankle joint. Also, Fürmaier (1951) reported that cutting of the ATFL was a presupposition if cutting of the lateral collateral ligaments was to cause increased adduction. However, it has not been possible, either by forced adduction or abduction, to cause primarily rupture of the ATFL. Accordingly, its role in mobility has not yet been elucidated.

The syndesmosis between the tibia and fibula seems to be functionally closely bound up with the ATFL. Experimentally induced, isolated rupture of this structure is not on record, and it cannot be cut without simultaneously cutting the ATFL or PTFL. Apparently, external rotation of the talus increases somewhat more after combined cutting of the syndesmosis and ATFL than after isolated cutting of this ligament (Close 1956), whereas its interaction with the PTFL does not seem to have been investigated.

Little has been published regarding the function of the *PTFL*. After cutting it, Ashhurst and Bromer (1922) found a slight increase in the width of the mortise, but apart from this its role has not been studied experimentally.

From the literature it is difficult to assess the function of the *TCL*, as very few authors have made a distinction between the various components of the deltoid ligament. After cutting the TCL, Padovani (1975) found increased external rotation of the talus, in the form of anteromedial rotatory instability, whereas Wirth et al. (1978) observed tightening of the ligament, both at dorsal and plantar flexion. By forced abduction several authors have induced rupture of the deltoid ligament, but without making any mention of whether TCL was the first structure to be torn. Thus, the investigations on record so far indicate that the superficial part of the deltoid ligament influences external rotation, dorsiflexion, and plantar flexion.

The role of *ATTL* by itself has not been elucidated experimentally, but in combination with the ATaFL it appears to restrict a translatory forward gliding of the talus, viz. ADS (Castaing and Delplace 1972). Presumably, these two structures combined also inhibit plantar flexion, as they could be torn by Hönigschmied (1877) by forcing this very movement. Owing to the direction of the ATTL fibres it would be expected that this ligament would also limit external rotation of the talus. However, investigators who have induced injuries in the deltoid ligament have not made a distinction between its various elements.

Nor has the specific function of the *ITTL* been elucidated. Isolated cutting of this structure is not practicable, but presumably it plays a restricting part in abduction, together with the other components of the deltoid ligament. Dias (1979), by forced internal rotation on the adducted ankle joint – after rupturing the lateral collateral ligaments – found rupture of the deep fibres of the deltoid ligament, of which the ITTL makes up the central part. Apart from this, there have been no studies to elucidate the function of this ligament.

PTTL constitutes the posterior, deep part of the deltoid ligament. Isolated cutting of this ligament has not been reported, but Dehne (1934) observed tightening of the ATaFL and PTTL at the same time on forced internal rotation. Thus, the PTTL possibly prevents internal rotation which would be of the nature of posteromedial rotatory instability. Hönigschmied (1877), by forced dorsiflexion, usually induced fracture of the medial malleolus, at times accompained by rupture of the PTaFL,

but in some cases he observed instead rupture of the deltoid ligament. Considering the situation of the PTTL, there is reason to believe that such a rupture would first affect the PTTL. However, this has not been demonstrated experimentally.

4.3 Comments on Previous Investigations

Even despite the use of complicated technical apparatus and under defined, uniform conditions, the various investigators in measuring the mobility in a joint have usually arrived at divergent results (Boone et al. 1978). The studies reviewed above have been carried out by widely different techniques and by different persons over a period of approx. 140 years. Often, the manner of measuring the various movements is not mentioned, and only a few authors have used a known force to induce the movements (McCullogh and Burge 1980, Johnson et al. 1981). Frequently, it is not stated whether instability was found at dorsiflexed, neutrally positioned, or plantar-flexed ankle joints, and even where this information is given, the degree of dorsi- or plantar flexion is rarely specified. The dissection of the ankle preparations is not always described with equal thoroughness. Indeed, this must also be presumed to render difficult a comparison of the various results.

Even when disregarding the above-mentioned deficiencies, the information obtainable by the aid of the techniques used so far is limited by the fact that although it is endeavoured, during the course of an investigation, to state accurately the mobility in the different planes, the picture obtained of the mobility must be discontinuous, as the findings in fact represent random samples.

A more detailed and nuanced picture of the function of the individual ligaments would be obtained, if the talus were affected by a defined torque, and the mobility – viz. internal-external rotation or adduction-abduction – were depicted graphically in a curve, presenting these movements at any degree of dorsal or plantar flexion. Thereby, it would be possible to elucidate the function of the individual ligaments – either by recording the mobility before and after cutting ligaments in different combinations, or by forcing movements into varying, accurately defined directions and studying the damage induced.

Chapter V

Present investigations – methods and material

All the investigations were carried out on osteoligamentous preparations, and they may be divided into *three phases:*

In *phase 1* the relationship of injuries to the lateral collateral ligaments to internal rotatory instability, talar tilt, and the anterior drawer sign was elucidated by a radiological technique. (I)

In *phase 2* an apparatus was designed by which it was possible to affect the talus by a defined torque and to record the corresponding rotatory movements graphically in the form of mobility patterns. The lateral collateral, the distal tibiofibular, and the medial collateral ligaments were cut successively, separately and in various combinations. In each situation mobility patterns were traced, visualizing the altered mobility – and thereby the instability. (II, III, IV, V, VI)

In *phase 3* forced movements in the ankle joint were done in recorded, accurately defined directions, and the injuries thus produced were described. (VII)

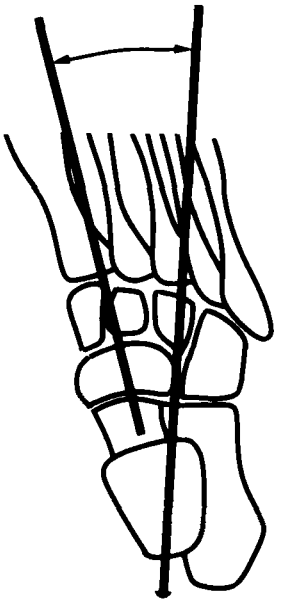


Fig. 5: Internal rotation, determined by the angle between two Steinmann pins.

5.1 **Method Used in Phase 1**

On amputated legs the ankle region was freed of all muscles, tendons, major vessels and nerves passing the joint, thus leaving an osteoligamentous preparation consisting of bones, joint capsule and ligaments. Into such a preparation two Steinmann pins were inserted in the postero-anterior direction – one through the talus and the other one through the distal end of the tibia. The preparation was fixed on a stand. Thereafter, the ankle joint was affected manually, by internal rotation or adduction of the foot, until firm resistance was met – both at intact ligaments and after successive transection of the lateral collateral ligaments in an antero-posterior sequence. Moreover, a 7 kg traction on the foot was applied. By these procedures it was attempted to produce maximum internal rotation as well as TT and ADS. The investigations were performed with the ankle joint in 20° dorsiflexion, in neutral position, and in 20° plantar flexion. At intact ligaments, and at all degrees of ligament injuries, X-ray films were exposed in the sagittal, frontal, and horizontal planes. Internal rotation of the talus was determined by the aid of the angle between the two Steinmann pins (Fig. 5). Adduction – or TT – was defined, according to

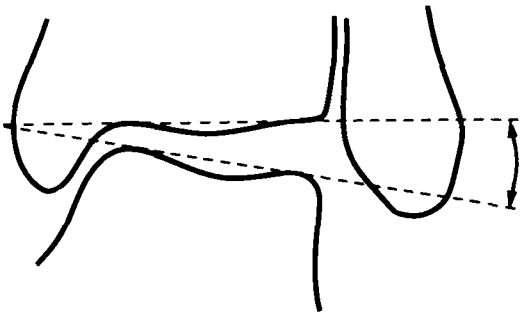


Fig. 6: Talar tilt or adduction, determined by the angle between the joint surfaces of the tibia and the talar trochlea.

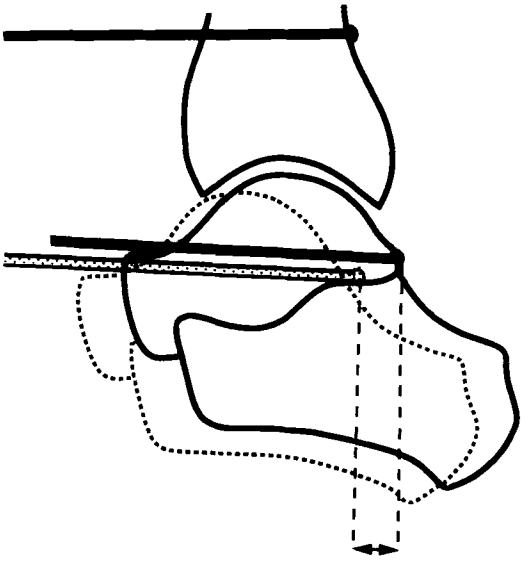


Fig. 7: Measurement of ADS as the horizontal shift of the nail in the talus.

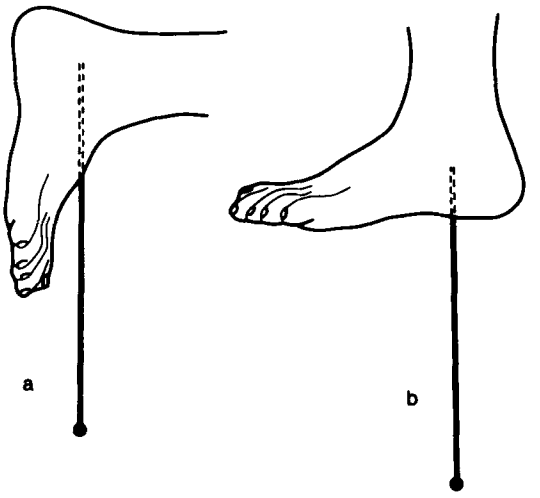


Fig. 9: Placement of lever for investigation in (a) the sagittal and horizontal planes and (b) in the sagittal and frontal planes.

Rubin and Witten (1960), as the laterally open angle between the joint surfaces of the tibia and talus (Fig. 6) and ADS by double exposure of the X-ray film (Johannsen 1978), first on an unloaded ankle joint and then after forward traction of the foot. ADS was then measured as the shift of the head of the distal pin (Fig. 7).

Study I was carried out by this method.

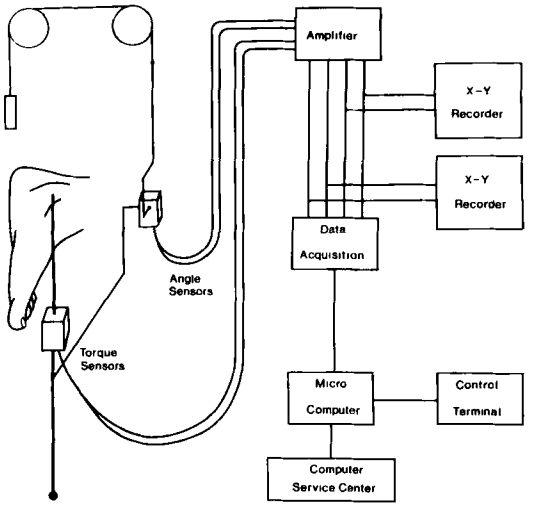


Fig. 8: Schematic experimental design.

5.2 **Method Used in Phase 2**

An apparatus was developed, enabling registration of rotatory movements in the ankle joint in two planes simultaneously when affecting the talus by a defined torque (Fig. 8).

An osteoligamentous preparation is fixed in a stand and a nail inserted into the talus, either in the postero-anterior direction (Fig. 9 a) for measurements in the sagittal and horizontal planes, or from below up through the calcaneus and into the talus (Fig. 9 b) for measurements in the sagittal and frontal planes. On the nail is mounted a lever furnished with strain gauges in two planes at right angles to each other, for measurement of the torque used. Furthermore, two potentiometers for measuring the appurtenant rotatory movements are coupled to the lever. Signals from the strain gauges and potentiometers pass an instrumentation amplifier with off-set adjustment to compensate for any inaccuracies in the fixation of the preparation. Two potentiometer recorders are coupled to the amplifier. One records the size and direction of the torque and the other one the appurtenant rotatory movement. The lever is moved manually, so that the torque recorder traces as accurately as possible the circumference of a predrawn torque square whose sides correspond to

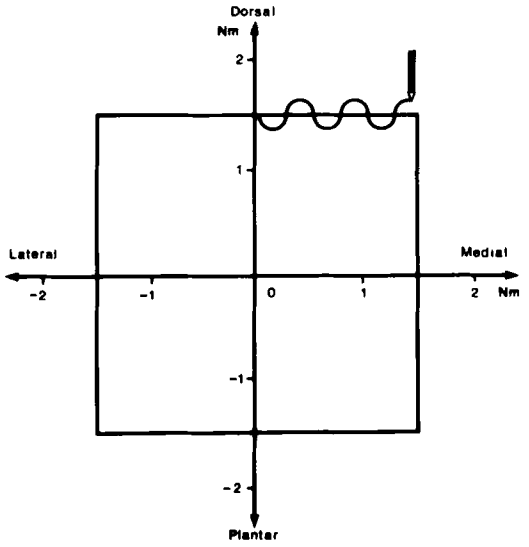


Fig. 10; Torque square corresponding to an action upon the talus by 1.5 Nm into the dorsal, medial, plantar and lateral directions respectively.

twice the torque by which the talus is to be affected into the dorsal, medial, plantar, and lateral directions respectively (Fig. 10). At the same time, the rotation recorder traces the rotations corresponding to the torque, thus giving a mobility pattern which shows the degree of internal and external rotation or of the adduction and abduction at any degree of dorsi- and plantar flexion.
Studies II, III, and VII were performed by the use of this apparatus.

With a view to being able to maintain an absolutely uniform torque and optimal tracing of the mobility patterns, the apparatus was further developed by coupling a data acquisition system to the instrumentation amplifier. Instead of following the torque square as accurately as possible, the writer can now zig-zag over its circumference. Corresponding measuring results for torque and rotation are then collected in a microcomputer which processes the collected data in a way so that only values containing relevant information are stored – i.e. only those data which show a difference between the values for torque, rotation, or both in relation to the values collected immediately before. After the measurement, the measuring results are copied on to a disc, and now the computer can produce a check picture of the mobility pattern on a control terminal. Later, the measuring results are transmitted, via the telephone net, to a computer service centre (RECAU). On the basis of the relevant measuring results the rotation at the desired torque is calculated, and by means of a standard plotting programme (DISSPLA) the mobility is plotted in the form of mobility patterns consisting of a dorsal, medial, plantar, and lateral curve.

Studies IV, V, and VI were carried out by the use of this apparatus.

5.2.1 **Technical specifications of the apparatus**
Strain gauges: 120 ohm bridge coupling. Instrumentation amplifier: AD 521 from Analog Devices, with ± 5 V output for ± 3 Nm on the lever. After amplification the potentiometers have an output of ± 5 V for ± 90° rotation. The data acquisition system converts to 8 bit (-5 V corresponding to 0 and +5 V to 256). Scanning rate about 200 Hz. Microcomputer: RC 702, based on Z 80 with 64 K RAM. Double disc drive for separating programme and data.

5.2.2 **Calibration of apparatus**
When making the measurements in practice, it was assumed that the centre of rotation was midway between the two malleolar apices. Variations in the placement of the nail and in the size of the various preparations caused differences in the length of the lever from one preparation to the other. As the torque is the product of force and arm, the force – and thereby the output from the strain gauges – has to be corrected accordingly by the ratio standard lever/present lever. The torque recorder and the data acquisition system were corrected by this factor. By current control measurements, it was ascertained, moreover, that the standard adjustment was stable.

5.2.3 **Conversion to digital values**
The data acquisition system converts to 8 bit (Fig. 11). This involves that the applicable area for measurement ranges from 0 to 255. At a standard amplification, therefore, the greatest measurable torque in one direction will be 3 Nm, but in the opposite direction only 3 × 127/128 Nm = ca. 2.98 Nm. Correspondingly, the greatest measurable

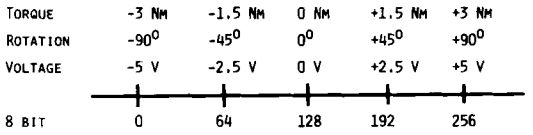


Fig. 11: Conversion of measuring results into digital values.

rotation in one direction is 90°, whereas in the opposite direction it is only 90 × 127/128 = ca. 89.3°.

5.2.4 **Resolution of measuring results**

The conversion system gives a resolution of the measuring results as regards the torque of 3/128 Nm = 0.0234375 Nm. Since in these studies the torque was kept at a constant average size = 1.5 Nm (cf. section on torque size), this corresponds to a resolution of 0.0234375 × 100/1.5 = ca. 1.56 %. The corresponding result for rotation is a resolution of 90/128 = 0.703125°. As rotation – unlike torque – is a variable size, the resolution of the measuring results cannot be stated in per cent, as such a percentage must always be reversely proportional to the extent of rotation.

5.2.5 **Tracing of the curves**

This was done in a regional computer service centre (RECAU). The calculations were carried out in a binary arithmetical system and the results converted to decimal values immediately before plotting the curves. For the sake of simplicity, however, the procedure will be described here in the decimal arithmetical system.

Primarily those measuring results are sorted off in which the torque differs by more than 10 % from the selected torque size – in these studies 1.5 Nm. Thus, the relevant rotation corresponds to a torque of between 1.35 Nm and 1.65 Nm. The four curves which form together a mobility pattern are inspected separately (Fig. 12). The findings at a given rotation – e.g. a dorsiflexion of 19° – are examined. This shows a number of corresponding values for torque and rotation in the medial direction, and the "mean value" for medial rotation is calculated. The same procedure at 18° or 20° dorsiflexion, however, gives an irregular curve owing to inaccuracies in the measurements or a varying

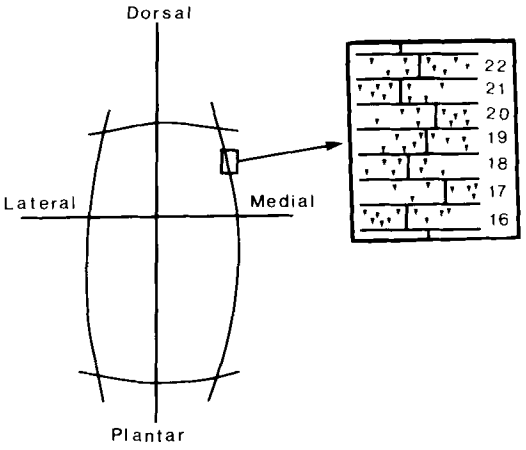


Fig. 12: Schematic mobility pattern. Detail insert showing internal rotation at a given dorsiflexion, measured only as the average value at this very degree of dorsiflexion.

number of measuring results. To smooth the curve, the calculation of the "mean value" at 19° dorsiflexion is performed not only at this degree of dorsiflexion, but on the basis of all data between 16° and 22° dorsiflexion. Similarly, the "mean value" for medial rotation at 18° dorsiflexion is calculated as the mean value of all measuring results from 15° to 21° dorsiflexion. Thus, at a given degree of dorsiflexion, the calculation of medial rotation also includes the medial rotation at the nearest degrees of dorsiflexion. Such "mean values" have been calculated at every degree of dorsiplantar flexion, and through them is plotted the curve indicating the medial rotation. All four curves – dorsal, medial, plantar, and lateral – are traced in this way, thus forming a mobility pattern. In the final tracing of the curve, moreover, smoothing is performed by means of a standard programme.

Calculation of "mean values", e.g. for medial rotation:

Fig. 13 shows, at a given degree of dorsiplantar flexion, the corresponding values for a medially directed torque and rotation (•) and the measuring results from the nearest degrees of dorsiplantar flexion (○). On the basis of all measuring results the best straight line is constructed, intersecting the line at torque = 0 in point X. From X through the individual measuring results at the

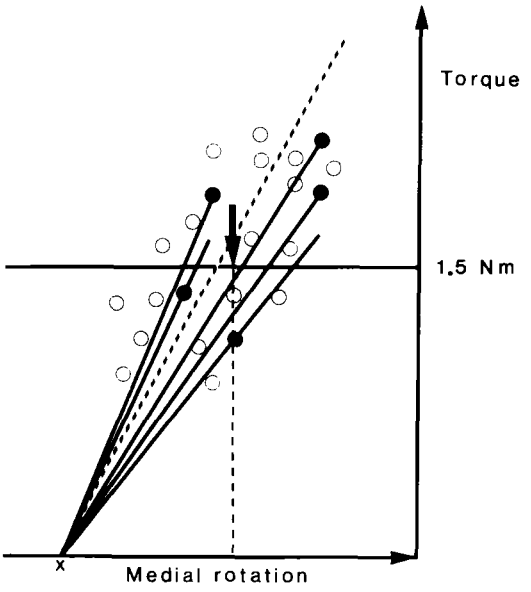


Fig. 13: Calculation of the "mean value" for medial rotation at a given degree of dorsiflexion.

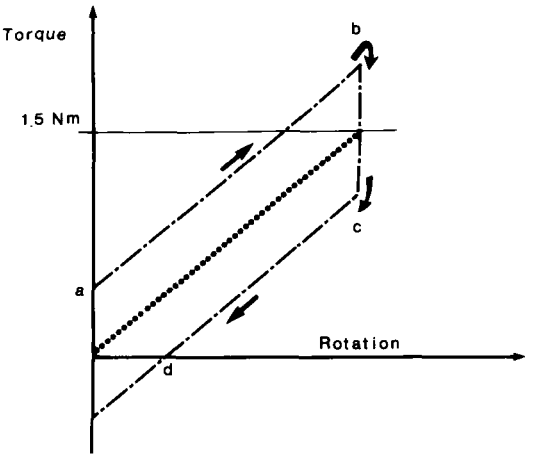


Fig. 14: Hysteresis in the system. The movement starts when the torque has attained a certain magnitude (a). At b the torque decreases, but the movement does not decrease until the torque has been reduced to c. When the torque is = O, there is still some rotation (d) which does not reach its initial value until the torque is negative.
– · – · –: relation between torque magnitude and rotation
● ● ● ●: relation between mean magnitude of torque and rotation

given degree of dorsiplantar flexion, lines are plotted. The mean value of their point of intersection with the line corresponding to 1.5 Nm indicates the calculated medial rotation at this very value of 1.5 Nm at the given degree of dorsiplantar flexion.

5.2.6 **Hysteresis in the experimental design**

When the lever is moved in a way so that the torque recorder zig-zags over the circumference of the torque square (Fig. 10), this is tantamount to the torque changing all the time in size within certain limits. The rotation produced by an increasing torque does not appear until the torque has attained a certain size, and reversely rotation does not decrease until after a certain diminution of the torque (Fig. 14). This implies that a given rotation can be recorded at a major or minor torque, and that the variation in rotation is shifted in time in relation to that of the torque (Fig. 15). By performing numerous zig-zag movements, so that the torque is now below 1.5 and now above, it is permissible to consider the torque practically constant and equal to the mean value.

5.2.7 **Torque size**

With the use of too great a torque there occur irreversible stretchings of ligaments and joint capsule, so that a mobility pattern – at an otherwise unchanged ligamentous condition – is not reproducible. The optimal torque is the maximum which only just does not produce such changes (Fig. 16). It has proved expedient to use a torque of 1.5 Nm, and this was done in plotting all mobility patterns in the present studies.

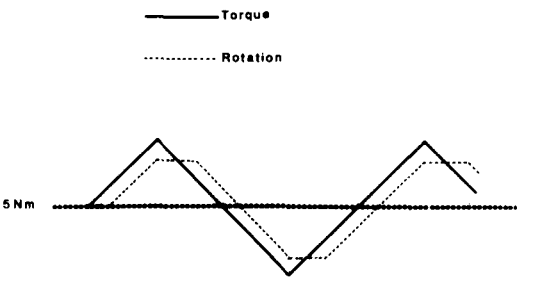


Fig. 15: Rotation altering, delayed in time, as compared with the torque.

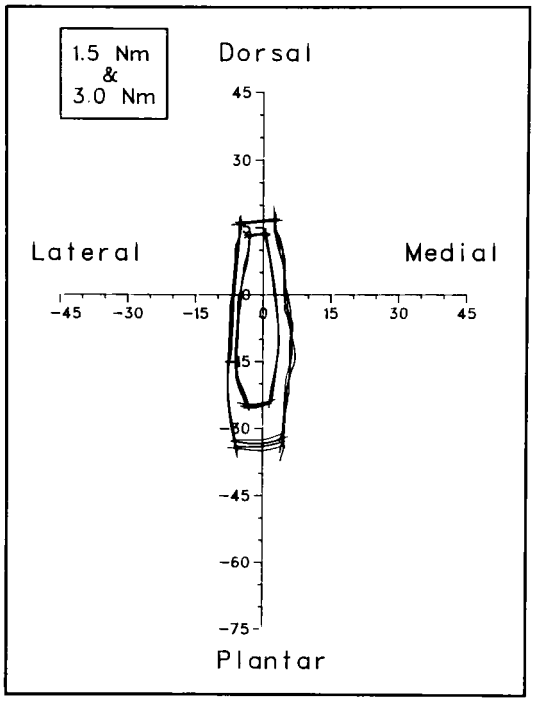


Fig. 16: Repeated plotting of the same situation: Centrally at a torque of 1.5 Nm and peripherally at a torque of 3.0 Nm. In the latter case the pattern is not reproducible in the plantar direction.

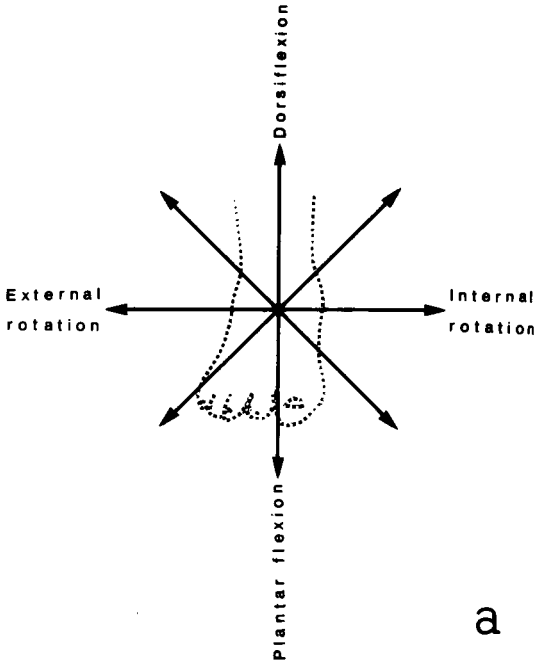
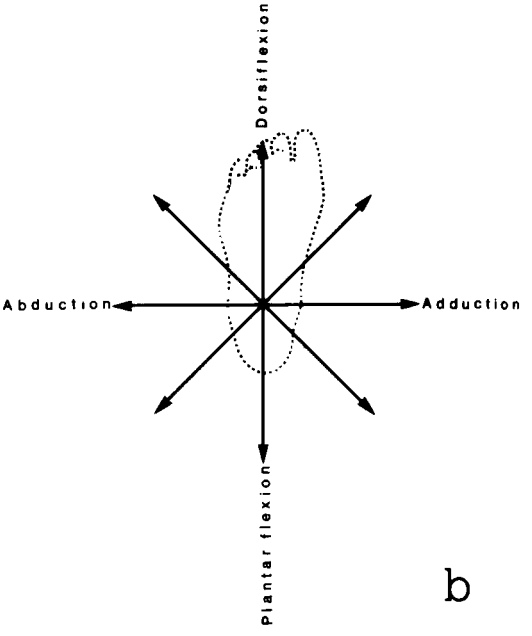


Fig. 17: Directions into which forced movements were performed: (a) in the sagittal and horizontal planes and (b) in the sagittal and frontal planes.

5.3 **Method Used in Phase 3**

The apparatus described above was used in part also in the studies of this phase. The preparations were fastened and the lever mounted as previously. Thereafter the foot – not the lever – was affected manually in a given direction with the guidance of that potentiometer recorder which traces the movements by an analogous technique. Thus, it is ensured that the ankle joint is affected in a definite and defined direction. The movement is forced until an injury is produced. This injury is then described, and thereupon the movement is further forced until a new injury occurs. This is continued until the ankle joint is completely unstable. The directions into which the movements are performed may be seen from Fig. 17 a and b.



5.4 **Material**

The studies included a total of 152 legs – predominantly knee-exarticulated or above-knee amputated. By far the greater part of the preparations were derived from patients having ischaemic diseases, so that the material comprises mainly preparations from elderly persons. Only a few of the amputation preparations were from young or youngish patients with neoplastic diseases, and others – also only a few – were from a post-mortem material. Possibly, this age difference in the material may result in the recorded movements – especially at intact ligaments – being smaller than if the material had been of a more even age distribution, as mobility in the ankle joint decreases and stability increases with age (Quellet et al. 1968, Boone and Azen 1979). However, it was not practically possible to take this into consideration in collecting the material.

Most of the amputated legs were collected from hospitals scattered over most parts of Denmark. As soon as possible after the ablative procedure they were deep frozen at about -16° C, but in some cases it had been possible to cool them only to around freezing point until they were collected. However, the tensile strength of the ligaments does not notably alter after a few days' storage, even at normal room temperature, provided that they are protected by the surrounding soft tissues (Viidik et al. 1965). Therefore, this has not had any essential influence upon the value of the preparations as study objects. On the other hand, the deep freezing has possibly somewhat reduced the ligament strength (Viidik and Lewin 1966) owing to precipitation of crystals in the substance – a factor which could not be avoided.

Immediately after thawing of a preparation, the ankle joint was freed of skin, subcutaneous tissue, passing tendons, vessels and nerves. Immediately thereafter the examination was performed, so that autolysis was restricted as far as at all possible, and dehydration of the preparation dit not occur.

Before and after the examination, the preparation was inspected for necroses in the ankle region, osteoarthritis, previous fractures or ligament injuries, etc., and in the case of pathological findings the preparation was discarded.

Chapter VI

Results

6.1 Phase 1: Radiological Study of the Correlation of Varying Degrees of Injuries to the Lateral Collateral Ankle Ligaments to Internal Rotatory Instability, Talar Tilt, and Anterior Drawer Sign

These studies were performed on 7 osteoligamentous ankle preparations, each investigated for all three forms of instability.

ATaFL, CFL, and PTaFL were successively cut in the named sequence, as it is assumed that ruptures of these ligaments generally start anteriorly and spread posteriorward (Broström 1964, Bouretz 1975, Duquennoy et al. 1975, Judet 1975, Noesberger 1976, Sanders 1977). The movements were produced by manually affecting the foot until meeting firm resistance, and the X-rays were exposed in 20° dorsiflexion, in the neutral position, and in 20° plantar flexion.

There proved to be a correlation between the degree of ligamentous injury and the extent of internal rotation (Fig. 18) in the form of anterolateral rotatory instability, the talus gliding forward and more or less out of the ankle mortise. At every degree of ligamentous injury this anterolateral rotatory instability was most marked with the ankle in plantar flexion, and it was distinctly apparent already after the ATaFL had been cut.

TT also showed agreement between the extent of injury and of instability (Fig. 19). Upon isolated severing of the ATaFL, the TT was most marked in plantar flexion, but after continued cutting of

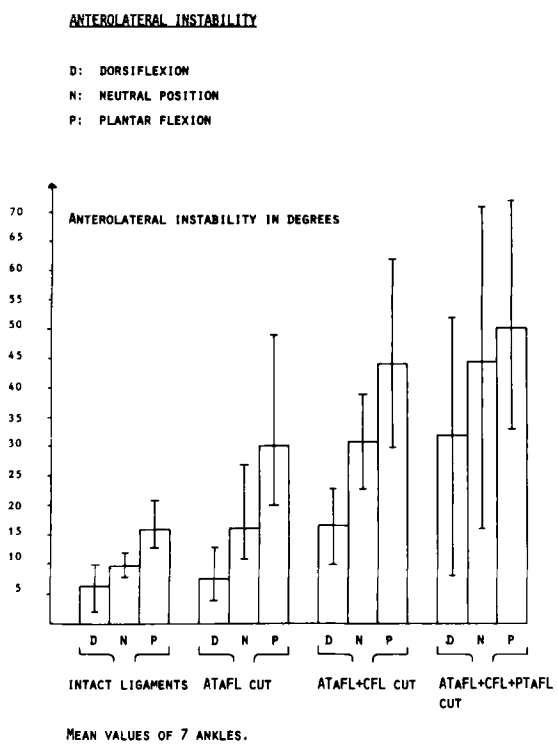


Fig. 18: Internal rotatory instability or anterolateral instability in various positions of the ankle joint and in relation to a varying degree of ligament transection.

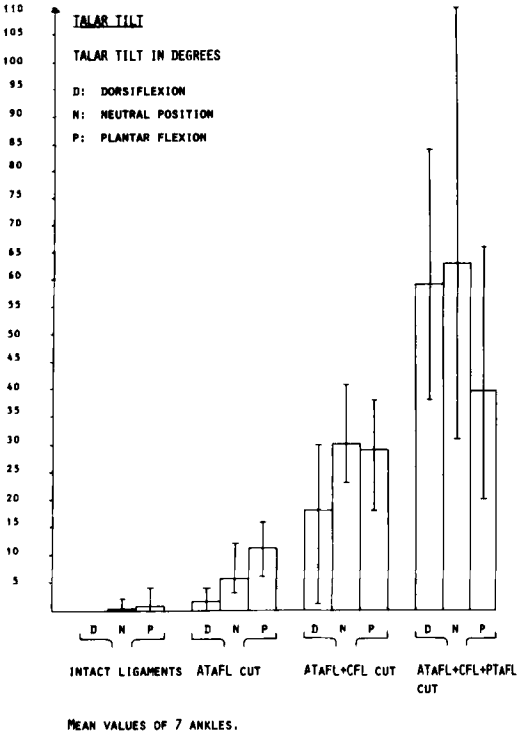


Fig. 19: Talar tilt in various positions of the ankle joint and in relation to varying degrees of ligament transection.

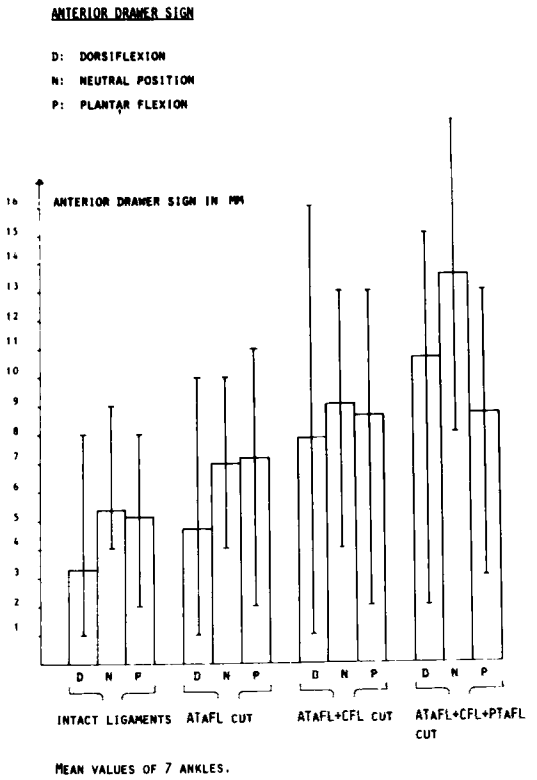


Fig. 20: ADS at various positions in the ankle joint and in relation to varying degrees of ligament transection.

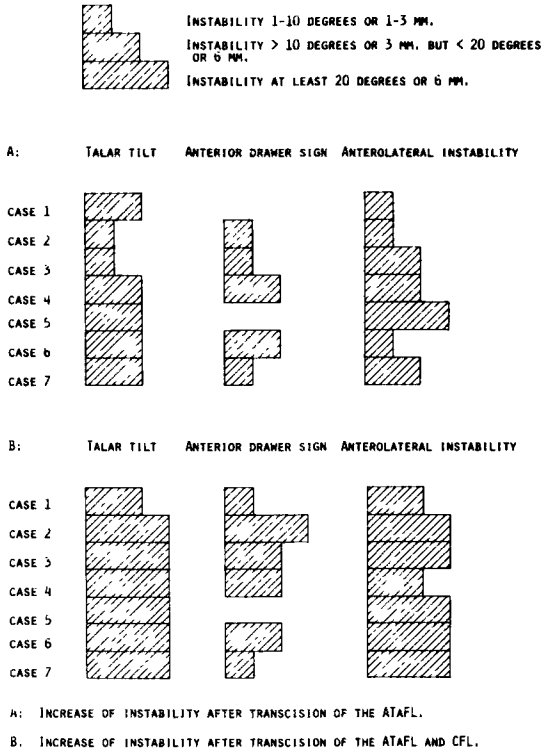


Fig. 21: Correlation between anterolateral rotatory instability TT, and ADS, given in the most favourable position for each instability group.

the ligaments it increased most in neutral position of the ankle. The limited increase of TT in plantar flexion after cutting also the PTaFL was due to tension of the anterior joint capsule.

Even at intact ligaments there was an ADS of 3.5 mm to 5.5 mm, depending upon the position of the foot in the sagittal plane (Fig. 20). At intact ligaments, the ADS was most marked in the neutral position, and after progressing cutting of the ligaments the increase in ADS was least in plantar flexion, alhough the values showed quite a marked dispersion.

In Fig. 21 it is endeavoured to visualize the relationship between the three types of instability. In this connection it must be borne in mind that two of them – anterolateral rotation and TT – are measured in degrees and the third one – ADS – in mm. Anterolateral rotatory instability and TT appear to be invariably increased, both after isolated cutting of the ATaFL and after cutting of this ligament plus the CFL. ADS, on the other hand, is somewhat inconstant, even in the latter situation. After cutting of all three ligaments all three types of instability were indeed extremely marked.

6.2 Phase 2: Kinesiological Study of the Ankle Joint After Cutting the ATaFL, CFL, PTaFL, ATFL, the Tibiofibular Syndesmosis, PTFL, TCL, ATTL, ITTL, and PTTL – Separately and in Various Combinations

These studies were carried out on 113 osteoligamentous ankle preparations. Mobility in the ankle joints was depicted as mobility patterns, showing at the top dorsiflexion and at the bottom plantar flexion. On patterns plotted in the sagittal-frontal plane movement in the medial direction signifies adduction of the talus, and movement in the lat-

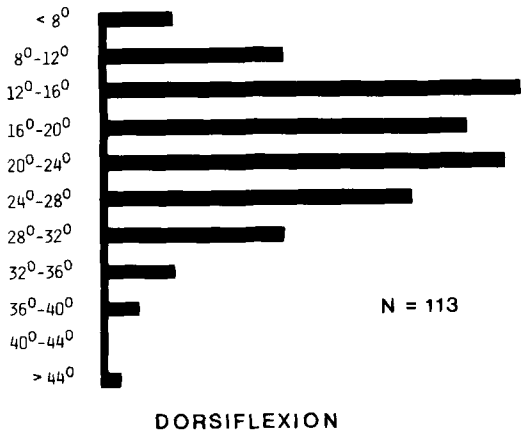


Fig. 22: Distribution of the extent of dorsiflexion at intact ligaments.

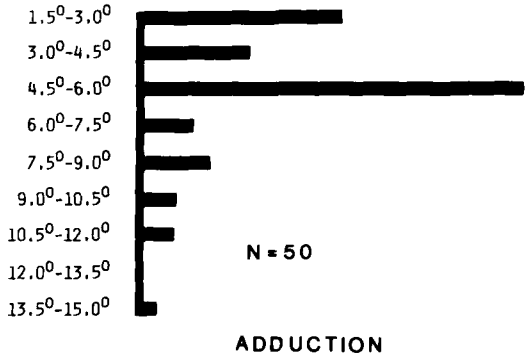


Fig. 24: Distribution of extent of adduction at intact ligaments.

eral direction signifies abduction. Medial and lateral directions on patterns in the sagittal and horizontal planes, on the other hand, correspond to internal and external rotation respectively. Centrally. each figure has a mobility pattern presenting the conditions at intact ligaments around which, more or less concentrically, the increase in mobility at successive ligament cutting is depicted.

The illustrations shown do not represent mean values, but characteristic examples of patterns found in the present material.

6.2.1 Mobility at intact ligaments

The extent of dorsiflexion and plantar flexion (113 preparations), adduction and abduction (50 preparations), and internal and external rotation (63 preparations) was plotted on histograms (Figs. 22-27). From these graphs it is apparent that the extent to which the preparations could be moved into each direction is distributed according to a normal distribution curve. Accordingly, the material is considered a normal material, and the results of measurements in each group are given as

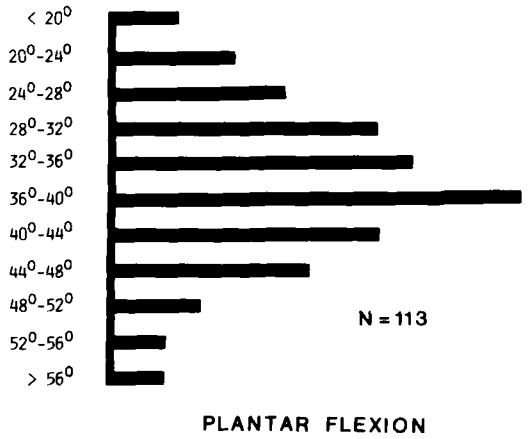


Fig. 23: Distribution of extent of plantar flexion at intact ligaments.

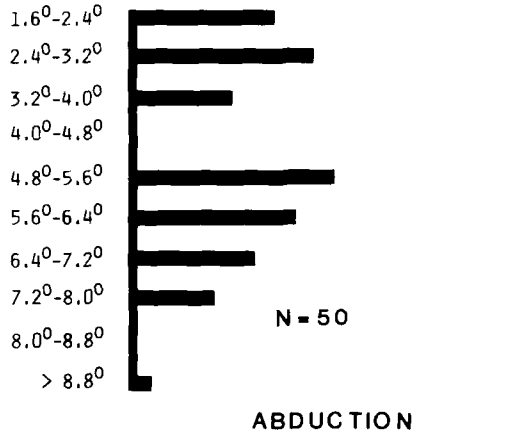


Fig. 25: Distribution of extent of abduction at intact ligaments.

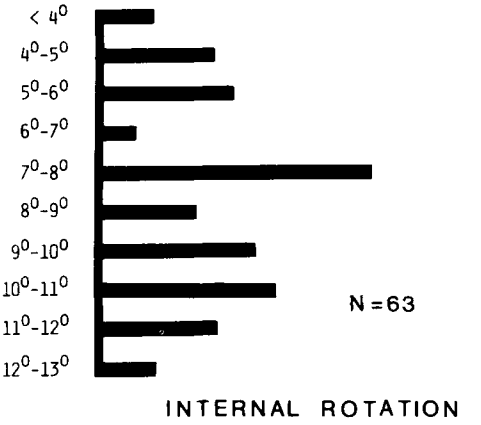


Fig. 26: Distribution of extent of internal rotatin at intact ligaments.

the mean value ± the standard deviation. This is done also in the section on ligament transections, to be consistent also for the very small groups. The mobility indicated by figures represents the maximum values for adduction-abduction as well as internal and external rotations, regardless of the degree of dorsi- or plantar flexion.

Dorsi-plantar flexion, in measurements on the patterns from all 113 preparations was about 58°, dorsiflexion making up 20.87° ± 7.53 and plantar flexion 36.89° ± 8.97.

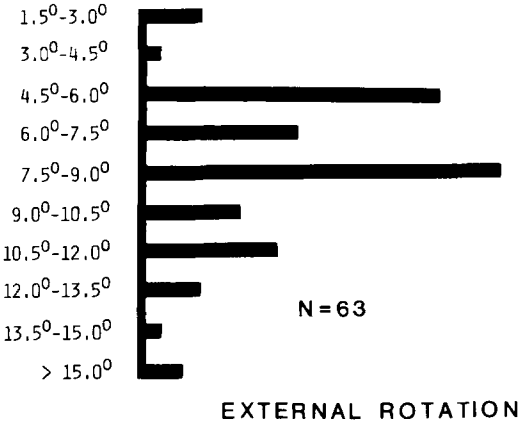


Fig. 27: Distribution of extent of external rotation at intact ligaments.

Adduction-abduction, measured on 50 preparations, amounted to rather more than 10°, distributed on adduction of 5.58° ± 2.68 and abduction of 4.84° ± 1.96.

Internal-external rotation, measured on 63 preparations, was about 17°, internal rotation being 8.60° ± 2.62 and external rotation 8.21° ± 3.02.

Statistical analysis of the material has demonstrated no mutual correlations between the degree of dorsiflexion, plantar flexion, internal rotation, external rotation, or mobility in the frontal plane (cf. for example Fig. 28). On the other hand, there is presumably some relationship between the degree of adduction and abduction (Fig. 29), an ankle joint with little adduction being probably also capable of only little abduction. Apart from this, no deductions can be drawn from a known extent of mobility to the mobility into other directions.

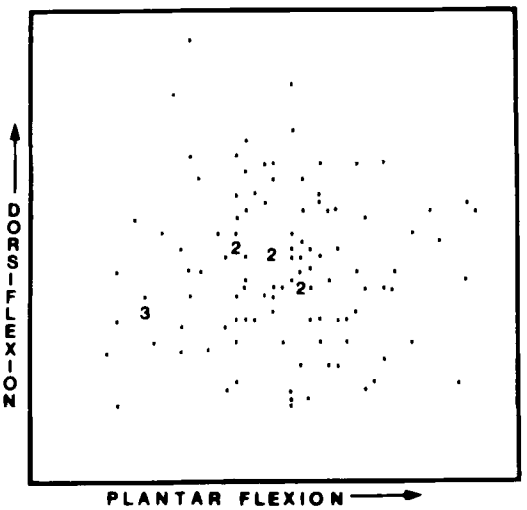


Fig. 28: Relation between extent of dorsi- and plantar flexion at intact ligaments. The figures represent more than one preparation showing the same values for both movements.

6.2.2 Mobility after transection of the lateral collateral ligaments

Cutting of the ATaFL often caused only a very minimal increase in dorsal and plantar flexion, in the range 0.5°-2°.

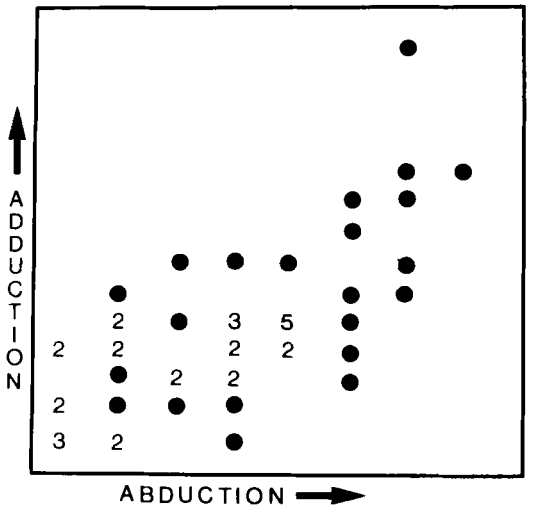


Fig. 29: Relation between extent of ab- and adduction at intact ligaments. The figures represent preparations with the identical excursions of movement into both directions.

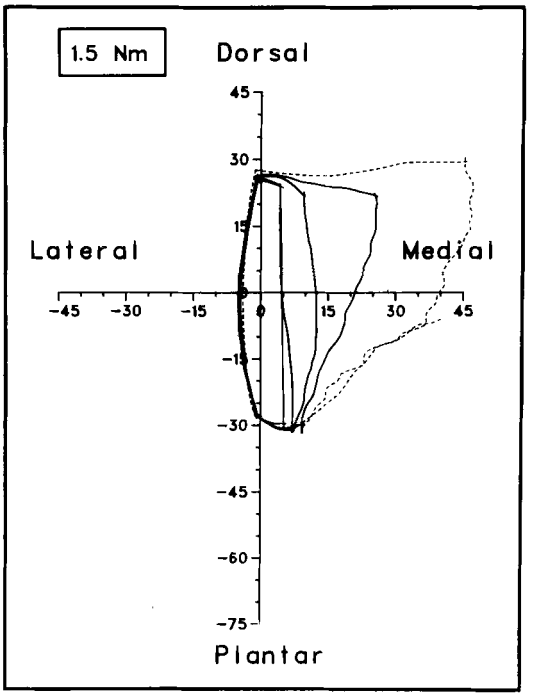


Fig. 30: Mobility patterns into the sagittal and frontal planes. In this as well as the following patterns the unit is degrees. Centrally: Mobility at intact ligaments, then mobility after cutting the ATaFL, after further cutting of the CFL, after cutting also the short fibres of the PTaFL, and lastly, outermost, mobility after cutting all the lateral collateral ligaments.

In the frontal plane this examination was performed on 12 preparations. As might be expected, abduction did not change, whereas adduction increased by 4.83° ± 1.80, mainly – and often only – in plantar flexion (Fig. 30).

In the horizontal plane the mobility was also studied on 12 preparations. While external rotation remained unaffected, internal rotation increased by 10° ± 3.59 (Fig. 31), i.e. an essential increase merely by cutting the ATaFL.

Cutting of the ATaFL + CFL increased adduction quite appreciably – also in dorsiflexion and neutral position (Fig. 30). The increase caused by including the CFL (9 preparations) amounted to 11.44° ± 6.15, while abduction remained unchanged.

Internal rotation increased by 1.90° ± 1.29 and was thus practically unchanged. Nor did external rotation alter after this transection (Fig. 31).

Cutting of the ATaFL + the short fibres of the PTaFL (3 preparations) hardly affected adduction at all (Fig. 32), as it increased by only 1.67° ± 1.15 more than after isolated cutting of the ATaFL. Internal rotation (Fig. 33) was augmented by another 2.50° ± 0.71, but in this instance the figures indicating maximum mobility are misleading, as there occurred an essentially greater, relative increase in the neutral position of the ankle, as is apparent from Fig. 33. This investigation was performed on 2 preparations. When the mobility pattern for a third preparation was being plotted, "spontaneous" rupture of the short fibres of the PTaFL occurred after transection of the ATaFL, while CFL still remained intact.

Further cutting of the long fibres of the PTaFL did not alter either internal rotation, adduction, or abduction, whereas external rotation increased minimally (1°-2°).

Cutting of the ATaFL + CFL + the short fibres of the PTaFL: In the frontal plane (6 preparations), the further inclusion of the short PTaFL fibres increased adduction by another 13.17° ± 4.17, while abduction was unaffected (Fig. 30).

In the horizontal plane transection of the short fibres increased internal rotation by 4° ± 1.58,

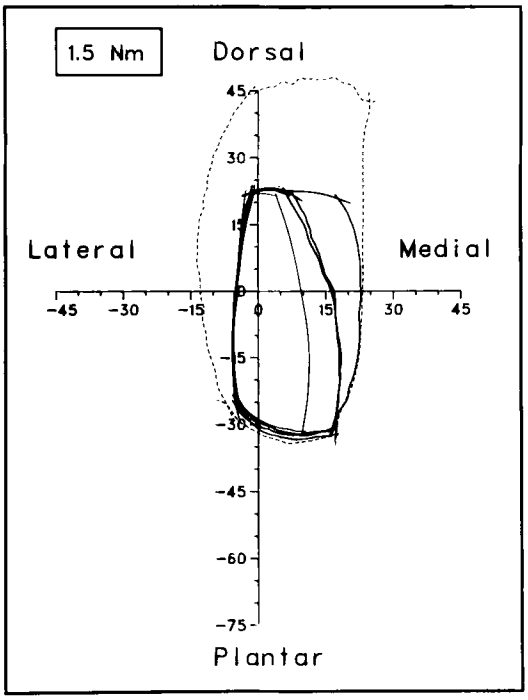


Fig. 31: Sagittal and horizontal planes. Cutting of ligaments as in Fig. 30.

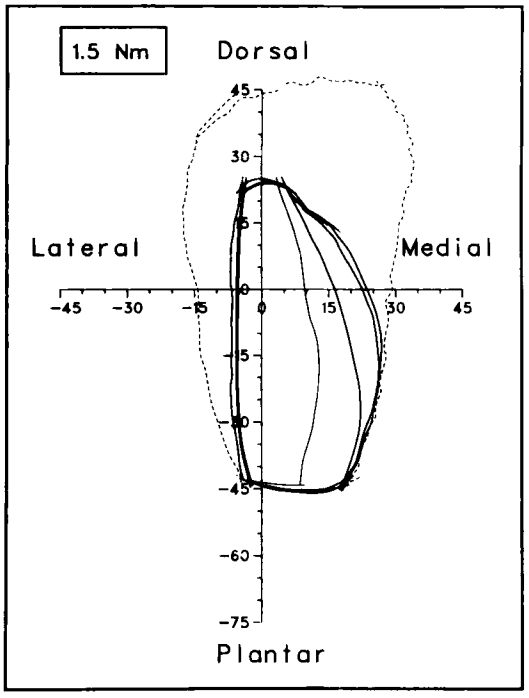
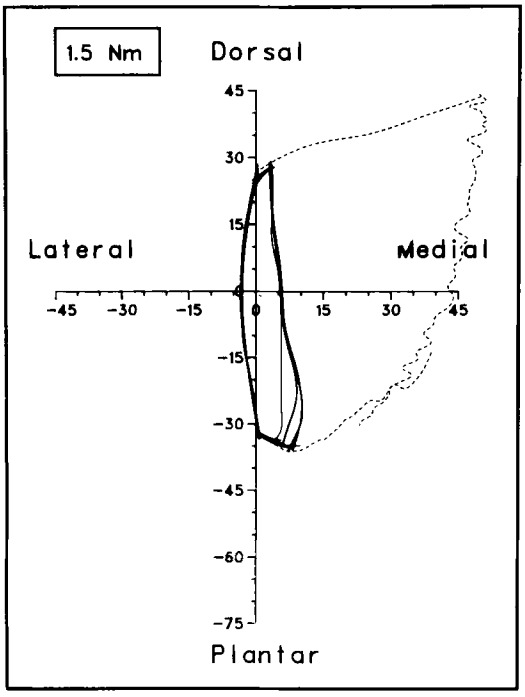


Fig. 33: Sagittal and horizontal planes. Cutting in the same sequence as in Fig. 32.



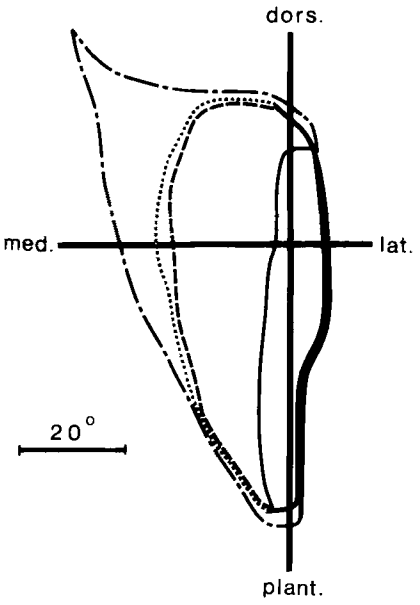
although the relative increase was even greater in dorsiflexion of the ankle (Fig. 31).

Cutting of the ATaFL + CFL + the long PTaFL fibres: Neither in the frontal plane (Fig. 34) nor in the horizontal plane (Fig. 35) did cutting of the long PTaFL fibres alter stability by more than a single degree (3 preparations in each group).

Cutting of the ATaFL + CFL + the entire PTaFL resulted in marked instability of the ankle. Adduction increased so that often it was impossible to plot mobility patterns in the frontal plane at a 1.5 Nm load. When it could be done the total adduction was 45°-60°, most marked in dorsiflexion. Abduction remained unaffected by these transections (24 preparations, Fig. 30).

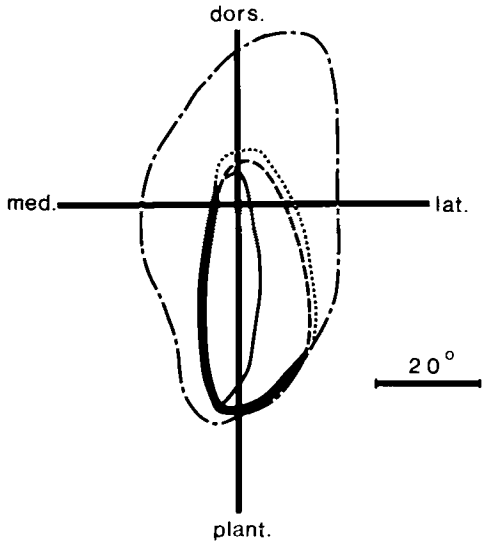
In the horizontal plane (also 24 preparations) internal rotation increased to more than 21°, the

Fig. 32: Sagittal and frontal planes. Centrally the mobility at intact ligaments and then after cutting the ATaFL, the short fibres of the PTaFL, the long fibres of the PTaFL, and finally of the CFL, in the named sequence.



INTACT LIGAMENTS
ATaFL AND CFL CUT
ATaFL , CFL AND LONG FIBRES OF PTaFL CUT
ATaFL , CFL AND WHOLE PTaFL CUT

Fig. 34: Sagittal and frontal planes.



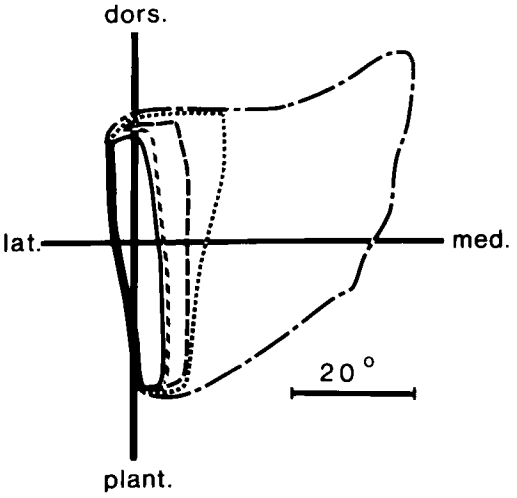
INTACT LIGAMENTS
ATaFL AND CFL CUT
ATaFL , CFL AND LONG FIBRES OF PTaFL CUT
ATaFL , CFL AND WHOLE PTaFL CUT

Fig. 35: Sagittal and horizontal planes.

abnormal part of the movement amounting to 13.92° ± 5.93. External rotation also increased, by 10.67° ± 4.63 (Fig. 31).

In the sagittal plane plantar flexion increased by only a single degree, whereas dorsiflexion increased by 12.58° ± 7.77, to an average of 30.63°. Owing to the laxity in the joint, the talus tended to be violently adducted in dorsiflexion, and indeed dorsiflexion was at a maximum when the talus was adducted. To avoid misleading results, dorsiflexion was measured only on the 24 preparations which were examined in the sagittal and horizontal planes.

Cutting of the CFL entailed, in the frontal plane (6 preparations) an increase in adduction of 1.33° ± 1.37 – i.e. a rather doubtful increment – whereas abduction was unaffected as might also be expected (Figs. 36 and 38). However, the relative increment in adduction was somewhat greater



INTACT LIGAMENTS
CFL CUT
CFL AND SHORT FIBRES OF PTaFL CUT
CFL AND WHOLE PTaFL CUT
ATaFL , CFL AND PTaFL CUT

Fig. 36: Sagittal and frontal planes.

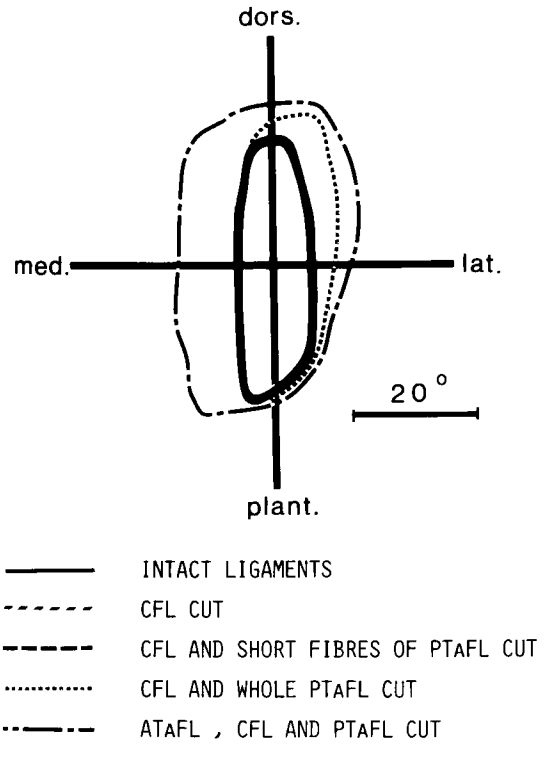


Fig. 37: Sagittal and horizontal planes.

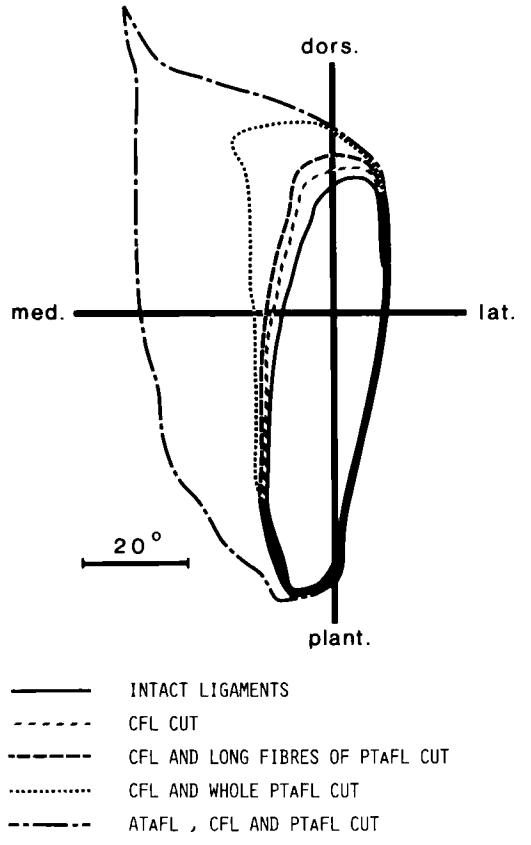


Fig. 38: Sagittal and frontal planes.

in dorsiflexion of the ankle, although the maximum value was found in plantar flexion.

Neither internal nor external rotation was affected by this transection (6 preparations), but in a few cases dorsiflexion increased by a single degree (Figs. 37 and 39).

Cutting of the CFL + the short fibres of the PTaFL was done on 6 preparations, 3 of which were studied in the sagittal and frontal planes and the other 3 in the sagittal and horizontal planes. Cutting of the short PTaFL fibres increased adduction by a further 1.67° ± 0.58, whereas abduction did not alter (Fig. 36).

In the horizontal plane (Fig. 37) mobility was not affected.

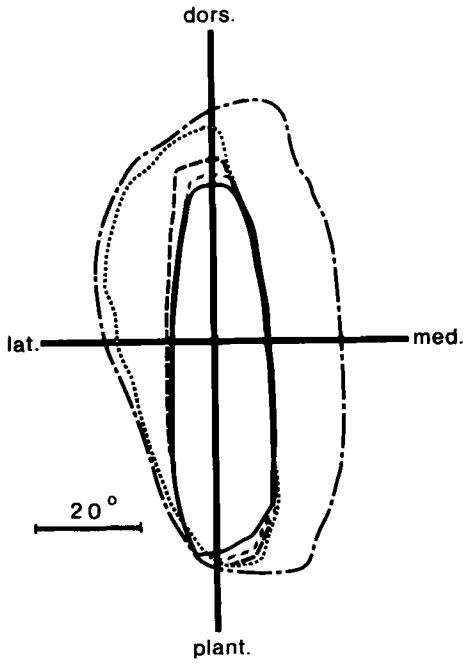
Cutting of the CFL + the long PTaFL fibres was also performed on 6 preparations. Adduction was studied in 3, and transection of the long fibres further increased it by a single degree, whereas abduction did not increase (Fig. 38). However, the further increase in adduction is somewhat greater in dorsiflexion than indicated by this value.

Internal rotation remained unaltered, whereas external rotation increased by 1.33° ± 0.58 (Fig. 39).

Dorsiflexion increased in the 6 preparations by 3.5° ± 2.17.

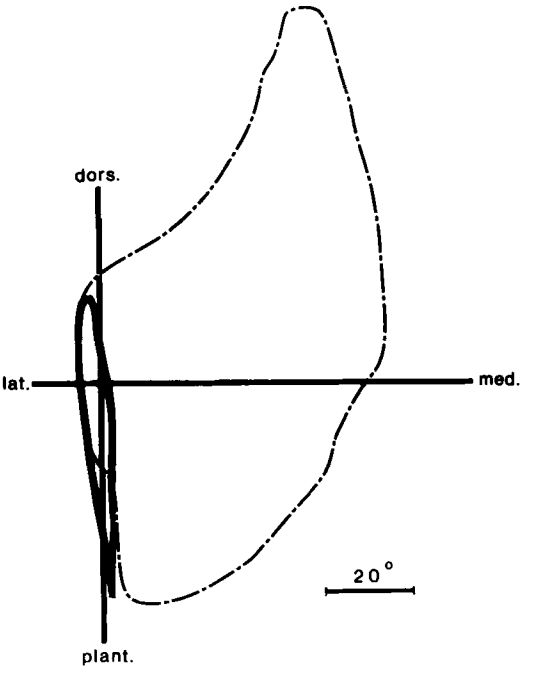
Cutting of the CFL + the entire PTaFL was done on 10 preparations, 4 of which were studied in the sagittal and frontal planes and 6 in the sagittal and horizontal planes. Isolated cutting of the CFL increased adduction only minimally, but now it increased by a further 5.25° ± 2.99, reaching a maximum not in plantar, but in dorsiflexion. Abduction was unaffected (Figs. 36 and 38).

Nor did internal rotation alter, whereas maximum external rotation increased by 6.17° ± 3.49



INTACT LIGAMENTS
CFL CUT
CFL AND LONG FIBRES OF PTaFL CUT
CFL AND WHOLE PTaFL CUT
ATaFL , CFL AND PTaFL CUT

Fig. 39: Sagittal and horizontal planes.



INTACT LIGAMENTS
SHORT OR LONG FIBRES OF PTaFL CUT
WHOLE PTaFL CUT
ATaFL , CFL AND PTaFL CUT

Fig. 40: Sagittal and frontal planes.

(Figs. 37 and 39), and by even more in dorsiflexion of the ankle.

In the sagittal plane dorsiflexion now increased by a total of 8.10° ± 3.90 – i.e. quite appreciably.

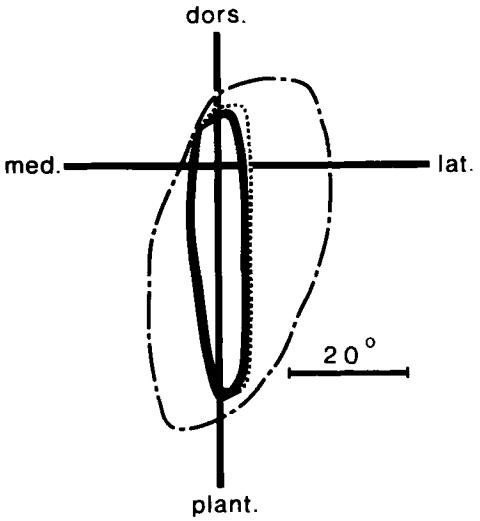
Isolated cutting of the PTaFL did not alter mobility in the frontal plane (3 preparations, Fig. 40).

In the horizontal plane too 3 preparations were studied. Internal rotation proved unchanged, whereas external rotation was mildly increased, by 2.33° ± 2.08 (Fig. 41).

In the sagittal plane dorsiflexion increased by only 1.17° ± 0.75, whereas plantar flexion remained unaffected.

6.2.3 Mobility after cutting the distal tibiofibular ligaments

Cutting of the ATFL did not affect mobility in the frontal plane (3 preparations, Fig. 42). In the hori-



INTACT LIGAMENTS
SHORT OR LONG FIBRES OF PTaFL CUT
WHOLE PTaFL CUT
ATaFL , CFL AND PTaFL CUT

Fig. 41: Sagittal and horizontal planes.

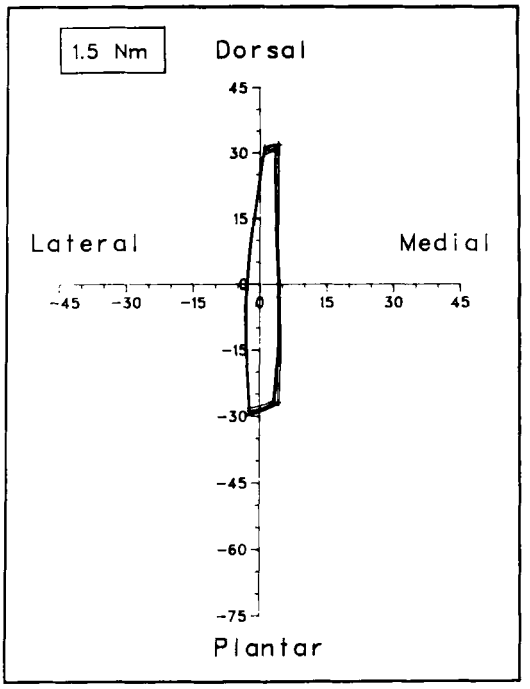


Fig. 42: Sagittal and frontal planes. ATFL, tibiofibular syndesmosis, and PTFL cut in the named sequence.

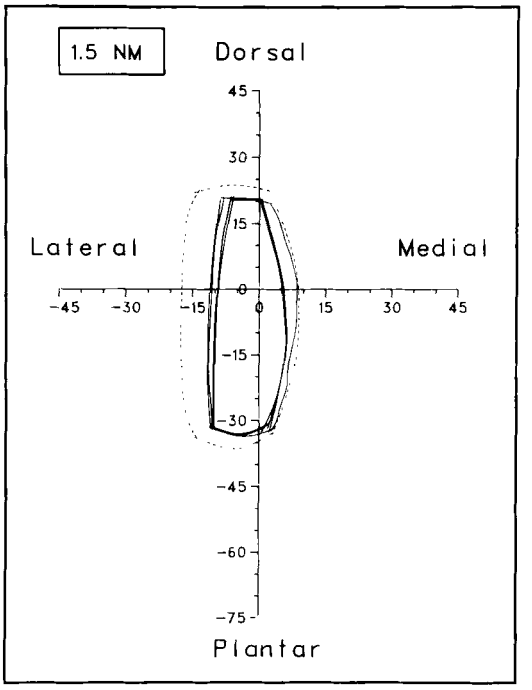


Fig. 43: Sagittal and horizontal planes. Successive cutting as in Fig. 42, and thereafter also of the PTaFL.

zontal plane (5 preparations) too there was no influence upon internal rotation, while external rotation increased negligibly by 1° ± 1.22 (Fig. 43). Dorsiflexion (all 8 preparations) increased by only 1° ± 1.22, i.e. practically not at all.

Cutting of the ATFL + the tibiofibular syndesmosis also had no influence upon ad- or abduction (3 preparations, Fig. 42). In the horizontal plane (5 preparations, Fig. 43) internal rotation still remained unchanged, whereas external rotation showed a further, but minimal increase (1.2° ± 0.45) after inclusion of the syndesmosis in the transection.

Dorsiflexion increased by a further 1° ± 0.92.

Cutting of the ATFL + tibiofibular syndesmosis + the PTFL did not affect mobility in the frontal plane (6 preparations, Fig. 42), except that one preparation exhibited an increase of 2° in adduction.

In the horizontal plane (9 preparations, Fig. 43) there occurred a total increase in normal external rotation of 2.78° ± 1.64 and an increment in normal internal rotation of 2.44° ± 1.67.

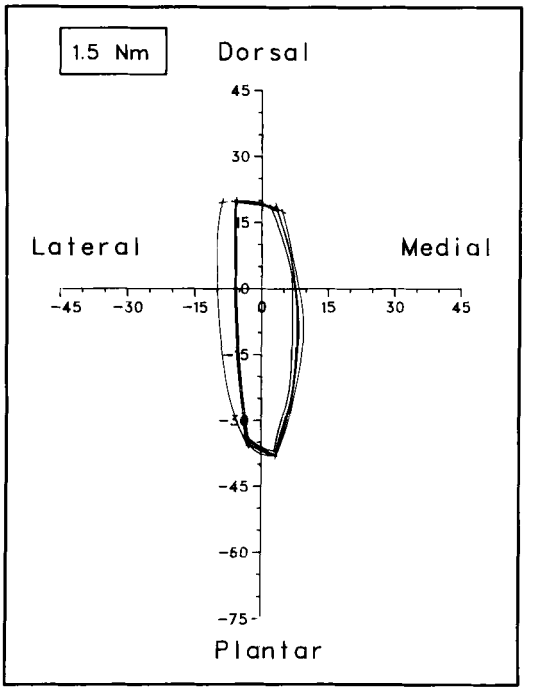


Fig. 44: Sagittal and horizontal planes. PTFL, tibiofibular syndesmosis, and ATFL cut in the named sequence.

The total increase in dorsiflexion (all 15 preparations) amounted to 1.47° ± 1.40 – i.e. overall a very doubtful increase in dorsiflexion, and the effect upon plantar flexion was even lesser.

Cutting of the PTFL entailed in the frontal plane (3 preparations) no change as might also be expected, but in the horizontal plane (4 preparations) internal rotation increased by 0.75° ± 0.5. External rotation was not affected (Fig. 44), and also not dorsiflexion.

Cutting of the PTFL + the tibiofibular syndesmosis did not alter mobility in the frontal plane (3 preparations), but in the horizontal plane the pre-existing, minimal abnormal internal rotation increased further, but very slightly, to 1.25° ± 0.5 (4 preparations, Fig. 44).

6.2.4 Mobility after cutting the medial collateral ligaments

Cutting of the TCL did not, as also not expected, affect adduction, while abduction increased by 2.27° ± 1.83 (15 preparations, Fig. 45). In the horizontal plane (23 preparations, Fig. 46) mobility did not alter, and there was no influence upon dorsi- or plantar flexion.

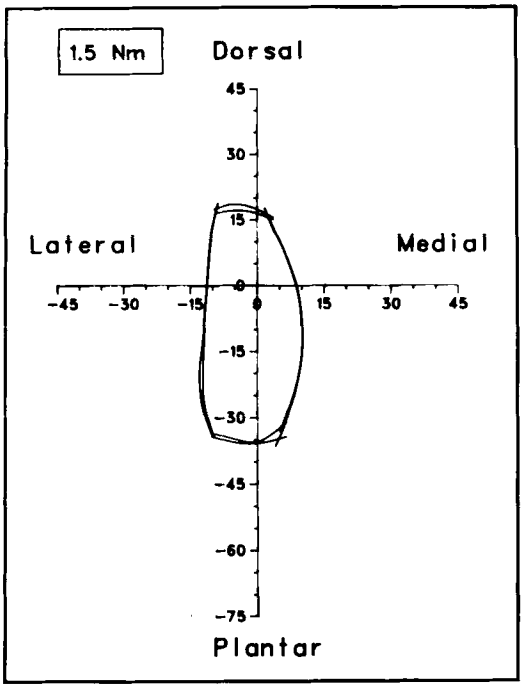


Fig. 46: Sagittal and horizontal planes. Cut TCL.

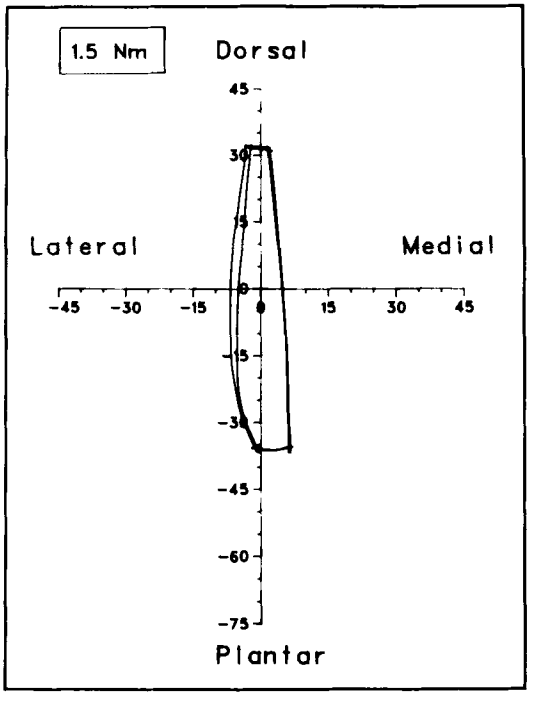


Fig. 45: Sagittal and frontal planes. Cut TCL.

In the subsequent studies, using transection of the components of the deltoid ligament, the TCL was always cut first, as it was not possible to transect the deeper ligaments without simultaneously injuring the TCL.

Cutting of the TCL + ATTL did not definitely increase mobility in the frontal plane (7 preparations, Fig. 47), as adduction increased by only 1.33° ± 1.15 and abduction by 1.00° ± 1.00.

In the horizontal plane (7 preparations, Fig. 48) nothing was altered. Nor do these structures appear to mean much to plantar flexion, as it increased by only 0.86° ± 0.07.

Cutting of the TCL + ATTL + ITTL caused, in the frontal plane (4 preparations, Fig. 47), an increase of normal abduction by not less than 8.75° ± 4.19, most marked in plantar flexion.

In the horizontal plane (5 preparations, Fig. 48) internal rotation increased by 1.00° ± 0.71, while now the abnormal external rotation amounted to 2.40° ± 1.52.

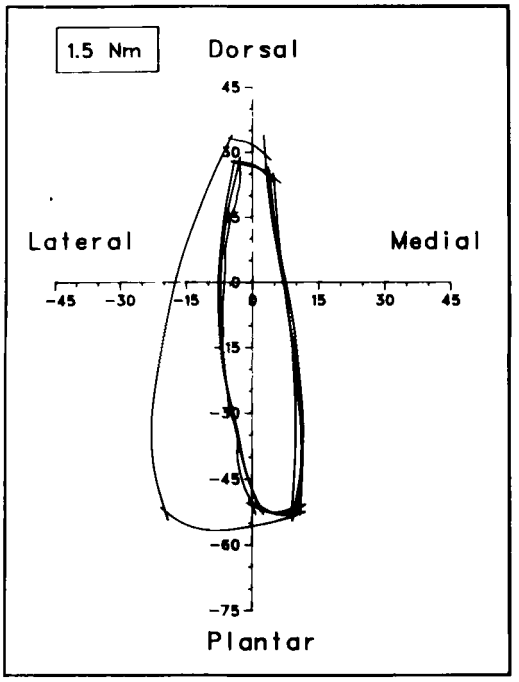


Fig. 47: Sagittal and frontal planes. Cutting of TCL, ATTL, and ITTL in the named sequence.

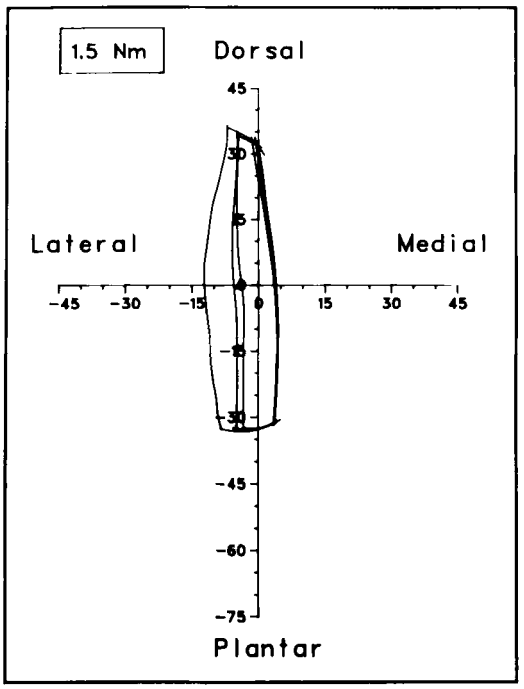


Fig. 49: Sagittal and frontal planes. Cutting of TCL and ITTL in the named sequence.

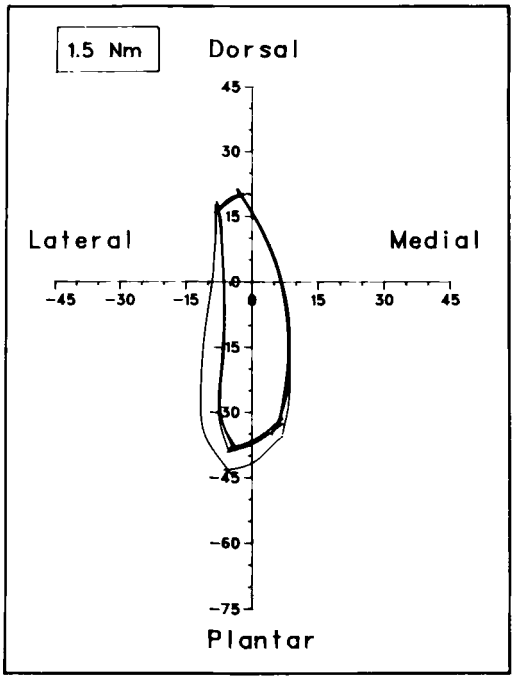


Fig. 48: Sagittal and horizontal planes. Cutting as in Fig. 47.

In the sagittal plane (9 preparations) dorsiflexion increased by 2.22° ± 1.79 and plantar flexion by 3.56° ± 2.46.

Cutting of the TCL + ATTL + ITTL + PTTL entailed such a lax ankle joint that no mobility patterns could be plotted.

Cutting of the TCL + ITTL resulted in a marked increase of abduction, 8.67° ± 2.08, while adduction did not alter (3 preparations, Fig. 49). In the horizontal plane (3 preparations, Fig. 50) mobility was not affected, and dorsi- as well as plantar flexion remained unchanged.

Cutting of the TCL + ITTL + PTTL rendered the joint extremely unstable, the abnormal abduction increasing to 20.20° ± 16.91 (5 preparations, Fig. 51), while in the horizontal plane (5 preparations, Fig. 52) there was a total increase in internal rotation of 7.60° ± 4.16 and in external rotation of 4.60° ± 3.58. In the sagittal plane normal dorsiflexion increased by 16.80° ± 6.65, while plantar flexion remained unchanged.

Cutting of the TCL + PTTL entailed in the frontal plane (3 preparations, Fig. 51) an increase

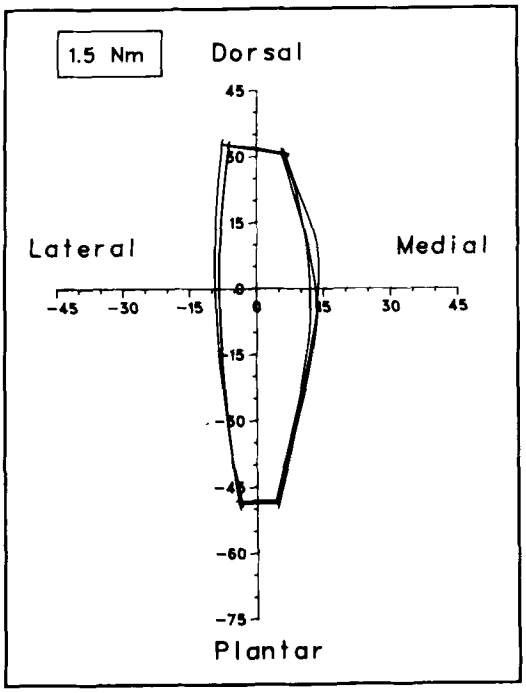


Fig. 50: Sagittal and horizontal planes. Cutting as in Fig. 49.

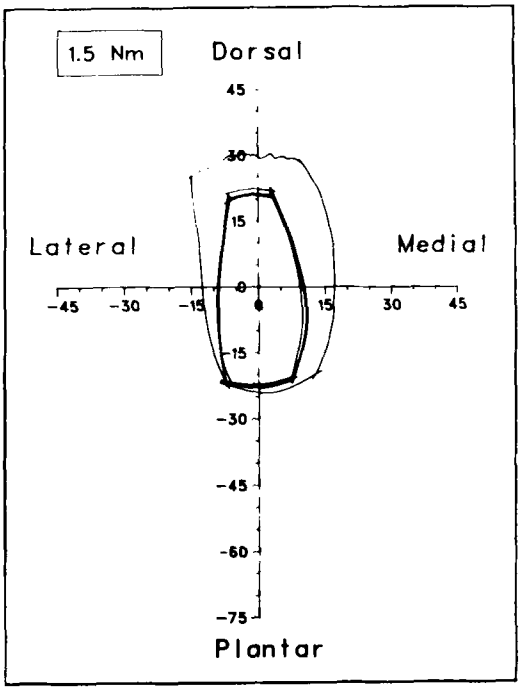


Fig. 52: Sagittal and horizontal planes. Cutting as in Fig. 51.

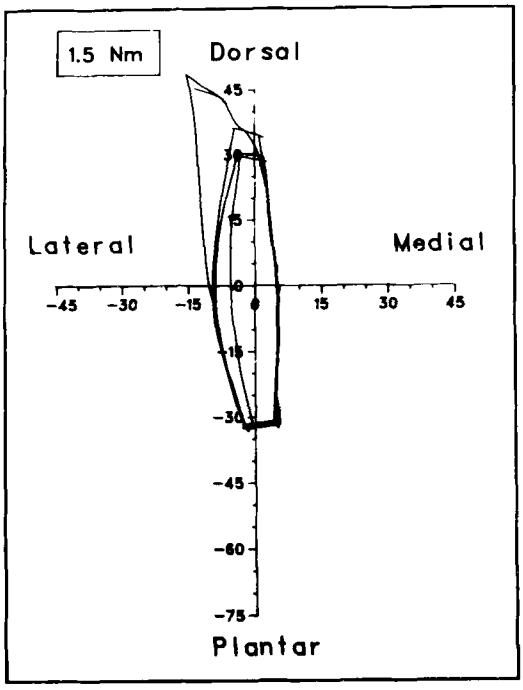


Fig. 51: Sagittal and frontal planes. Cutting of TCL, PTTL, and ITTL in the named sequence.

in abduction of 2.33° ± 1.53 in all – viz. hardly more than after isolated cutting of the TCL. In the horizontal plane (3 preparations, Fig. 52) internal rotation increased by only 1.33° ± 0.58, while in the sagittal plane dorsiflexion increased by 3.33° ± 1.86.

6.2.5 Mobility after combined cutting of the PTaFL and the distal tibiofibular structures

This combination was selected, because it may be assumed that the tibiofibular ligaments will rupture in external rotation traumas and that the PTaFL participates in the inhibition of external rotation. The investigation was carried out on 4 preparations which now exhibited a considerably greater instability than after isolated cutting of the tibiofibular ligaments, as normal external rotation increased by 7.00° ± 1.15 (Fig. 43). At the same time, dorsiflexion increased by 3.00° ± 1.41 and plantar flexion by 2.00° ± 0.82.

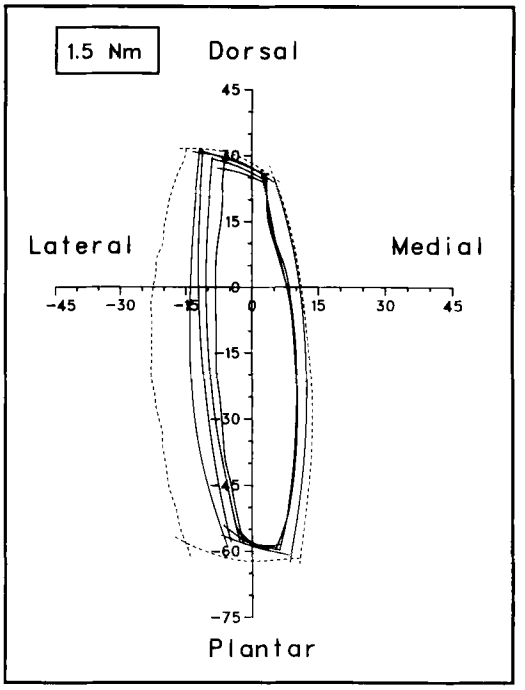


Fig. 53: Sagittal and horizontal planes. Cutting of ATFL, tibiofibular syndesmosis, PTFL, and ATTL in the named sequence.

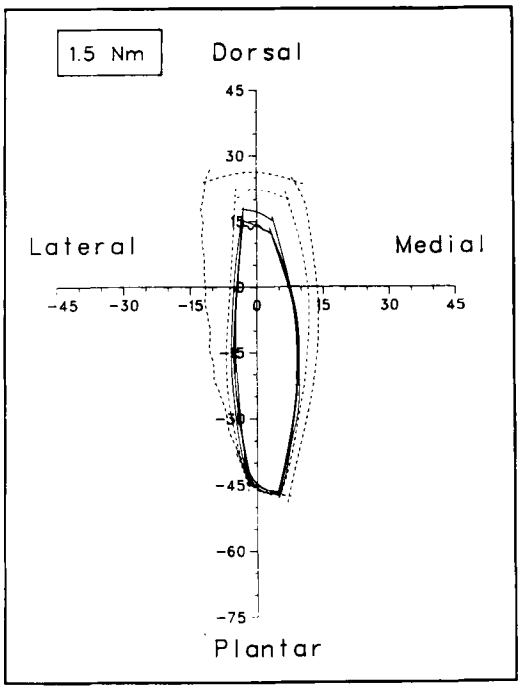


Fig. 54: Sagittal and horizontal planes. Cutting of PTFL, tibiofibular syndesmosis, ATFL, PTTL, and ITTL in the named sequence.

6.2.6 Mobility after combined cutting of the medial collateral and distal tibiofibular ligaments

Cutting of the ATTL + ATFL + tibiofibular syndesmosis + PTFL increased, in the frontal plane (2 preparations), adduction by only a single degree, while in the horizontal plane (also 2 preparations) it resulted in an abnormal internal rotation of 3.50° ± 0.71 and an abnormal external rotation of as much as 10.50° ± 6.36, viz. an instability considerably greater than after cutting of the ATTL and the tibiofibular structures separately (Fig. 53).

Cutting of the TCL + PTTL + ITTL + ATFL + tibiofibular syndesmosis + PTFL was done on only one preparation which was studied in the sagittal and horizontal planes. The tibiofibular structures were cut first and thereafter the PTTL + the posterior portion of the TCL. This caused an increase in internal rotation of about 2°, but the relative increase in dorsiflexion of the ankle was somewhat greater (Fig. 54). Dorsiflexion itself also increased.

Further cutting of the remaining portion of the TCL and of ITTL again increased, as may be seen from Fig. 54, both internal rotation and dorsiflexion, and an increment in external rotation occurred.

6.2.7 Mobility after combined cutting of the medial and lateral collateral ligaments

Cutting of the TCL + ATaFL: Isolated cutting of the TCL did not affect mobility in the horizontal plane (Fig. 46), whereas cutting of the ATaFL increased internal rotation (Fig. 31). After combined cutting of the two structures an abnormal internal rotaton of 9.28° ± 3.45 was observed, presumably due exclusively to the transection of the ATaFL (7 preparations, Fig. 56). In the frontal plane too (4 preparations, Fig. 55) the changes did not exceed what might be expected after transection of each structure in isolation.

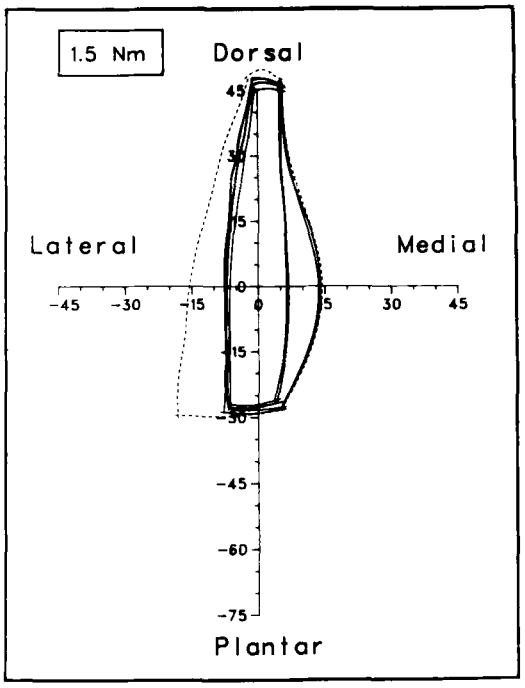


Fig. 55: Sagittal and frontal planes. Cutting of TCL, ATaFL, ATTL, and ITTL in the named sequence.

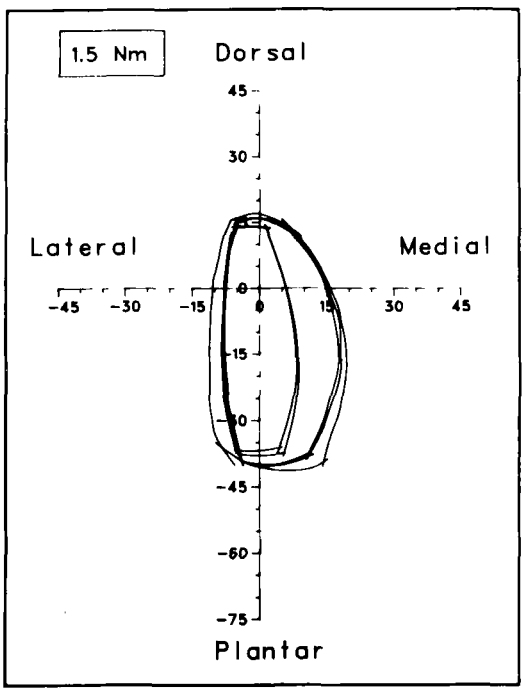


Fig. 56: Sagittal and horizontal planes. Cutting as in Fig. 55.

Dorsiflexion increased (in all 11 preparations) by a total of 1.36° ± 1.02, i.e. hardly more than after isolated cutting of the ATaFL, while plantar flexion increased by 2.18° ± 1.16.

Cutting of the TCL + ATTL + ATaFL did not result in greater changes in the frontal plane than would have isolated cutting of each structure (3 preparations, Fig. 55). In the horizontal plane (4 preparations, Fig. 56) internal rotation increased, after inclusion of the ATTL in the transection, by a further 1.25° ± 0.5 and external rotation by 1.50° ± 2.38. In the sagittal plane normal dorsiflexion increased by a total of 1.71° ± 0.96 and normal plantar flexion by 3.00° ± 1.63.

Cutting of the TCL + ATTL + ITTL + ATaFL: Inclusion of the ITTL in the transection caused, in the frontal plane (3 preparations, Fig. 55), an increase in the abnormal abduction to 15.33° ± 3.79, while in the horizontal plane there was practically no change in internal rotation, and external rotation increased by a further of only 1.75° ± 1.71 (4 preparations, Fig. 56). In the sagittal plane dorsiflexion did not increase, while plantar flexion increased by a further 1.42° ± 1.27.

Cutting of the TCL + ATTL + ITTL + PTTL + ATaFL rendered the joint so unstable that mobility patterns could not be plotted.

Cutting of the TCL + PTTL + ATaFL: Cutting of the TCL and ATaFL entailed increased internal rotation, but not more than might be expected after isolated cutting of the ATaFL. Further inclusion of the PTTL in the transection increased internal rotation by 2.67° ± 1.53, while external rotation remained unaffected (3 preparations, Fig. 57). In the sagittal plane dorsiflexion increased after cutting of the PTTL by a further 6.50° ± 5.92, and in the frontal plane (1 preparation) abduction increased by 3.00° in maximum plantar flexion.

Cutting of the TCL + PTTL + ITTL + ATaFL increased, in the horizontal plane (3 preparations, Fig. 57), internal rotation essentially, so that its abnormal part made up 22.60° ± 16.17. In 2 of the preparations external rotation increased by 1°,

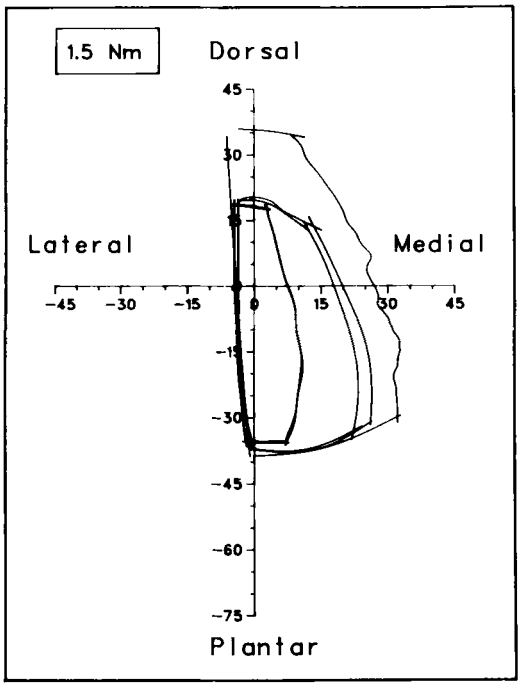


Fig. 57: Sagittal and horizontal planes. Cutting of TCL, ATaFL, PTTL, and ITTL in the named sequence.

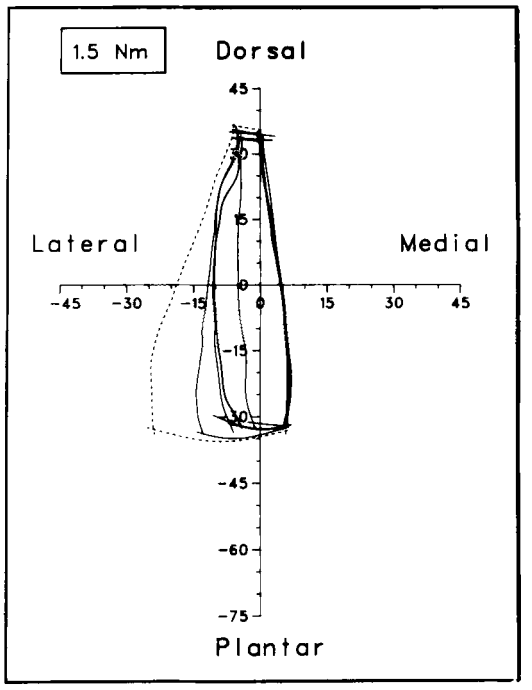


Fig. 58: Sagittal and frontal planes. Cutting of TCL, PTaFL, ATTL, and ITTL in the named sequence.

while in the third preparation it increased by as much as 17° when the ankle was in dorsiflexion. At the same time, normal plantar flexion increased by a total of 4.00° ± 1.00 and dorsiflexion by a total of 18.67° ± 11.15. In the frontal plane plotting of mobility patterns had to be abandoned because of marked instability in the joint.

Cutting of the TCL + PTaFL did not entail more instability in any direction than might be expected after cutting each ligament separately (8 preparations, Figs. 58 and 59), i.e. enhanced abduction because of the transection of TCL and a slight increase in external rotation in dorsiflexion after transection of the PTaFL. Dorsiflexion increased by a total of 2.83° ± 2.64, while plantar flexion remained unchanged.

Cutting of the TCL + ATTL + PTaFL increased, in the frontal plane (1 preparation), abduction a bit in plantar flexion (Fig. 58) in relation to the situation found after cutting the TCL + PTaFL. In the horizontal plane (3 preparations, Fig. 59) the total abnormal internal rotation

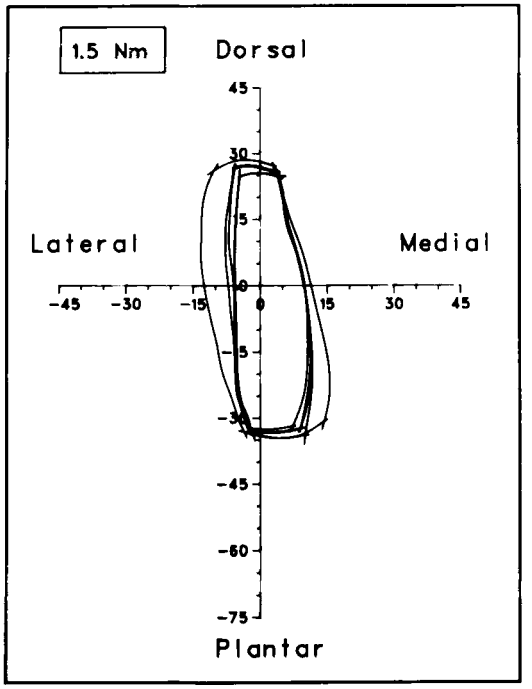


Fig. 59: Sagittal and horizontal planes. Cutting as in Fig. 58.

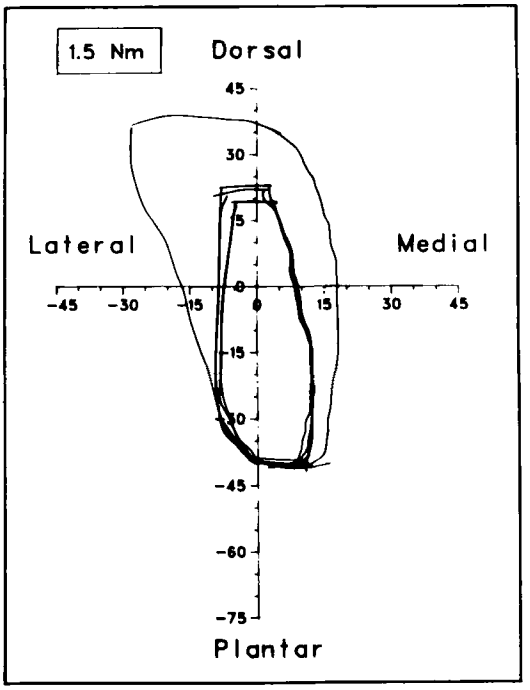


Fig. 60: Sagittal and horizontal planes. Cutting of TCL, PTaFL, PTTL, and ITTL in the named sequence.

amounted to only 0.33° ± 0.58 and the corresponding increase in external rotation to 2.33° ± 0.58.

Cutting of the TCL + ATTL + ITTL + PTaFL: The addition of cutting also the ITTL caused, in the frontal plane (1 preparation, Fig. 58), an increase in abduction during plantar flexion of almost 10°. In the horizontal plane (3 preparations, Fig. 59), there occurred a further internal rotation of 4.00° ± 1.73 when the ankle joint was plantar flexed, while in dorsiflexion external rotation increased by exactly the same. Dorsiflexion and plantar flexion showed practically no change.

Cutting of the TCL + PTTL + PTaFL did not, either in the frontal plane (1 preparation) or in the horizontal plane (3 preparations, Fig. 60) result in greater instability than found after cutting the TCL + PTaFL. In the sagittal plane the abnormal part of mobility in the dorsal direction increased to 7.50° ± 4.73, whereas plantar flexion did not alter.

Cutting of the TCL + PTTL + ITTL + PTaFL resulted in such great laxity that mobility patterns could not be plotted in the frontal plane. In the sagittal and horizontal planes (3 preparations, Fig. 60) dorsiflexion increased, so that now its abnormal part amounted to a total of 22.67° ± 6.43, while the total increase in internal and external rotation amounted to 8.33° ± 4.93 and 14.33° ± 8.96 respectively – most marked in dorsiflexion.

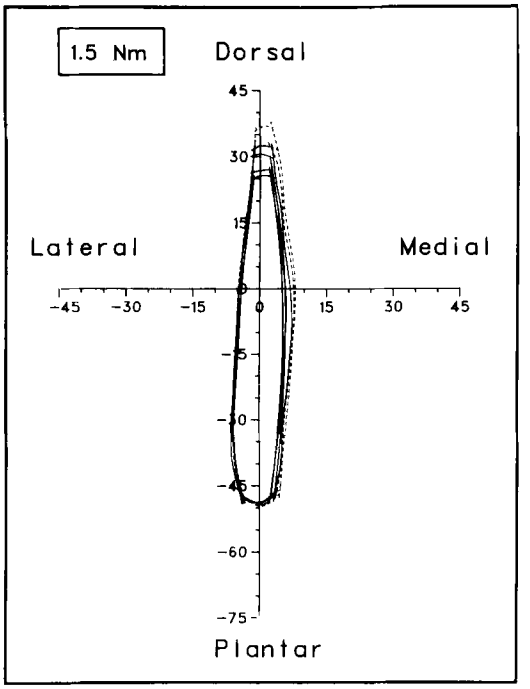


Fig. 61: Sagittal and frontal planes. Cutting of ATTL, ATFL, tibiofibular syndesmosis, PTFL, and PTaFL in the named sequence.

6.2.8 Mobility after combined cutting of the tibiofibular, medial, and lateral ligaments

In this part of the study the *TCL + ATTL + ATFL + tibiofibular syndesmosis + PTFL + PTaFL were cut,* as these structures combined and separately, must be assumed to influence external rotation. In the frontal plane (2 preparations, Fig. 61) even such a great degree of ligamentous injury has but little influence upon stability, abduction increasing by only a few degrees – corresponding to the transection of TCL – and adduction remaining completely unaffected. In the horizontal plane (4 preparations, Fig. 62) normal external rotation increased by a total of 11.60 ± 4.93, while internal

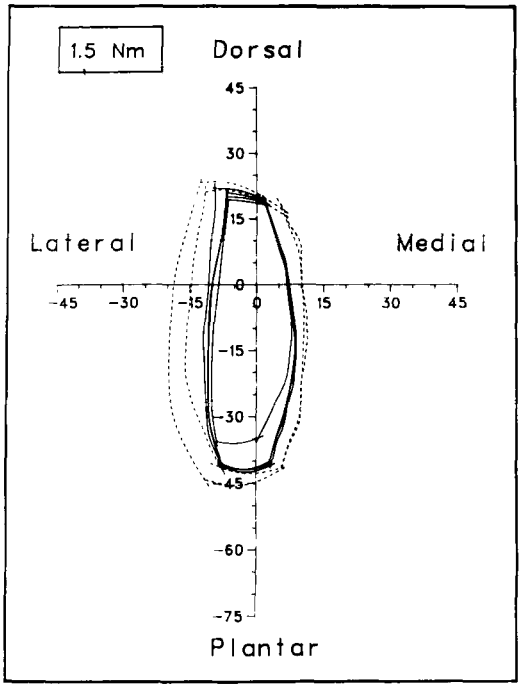


Fig. 62: Sagittal and horizontal planes. Cutting as in Fig. 61.

rotation was not affected more than after cutting of the tibiofibular structures + either the TCL, ATTL, or PTaFL.

6.3 Phase 3: Ligamentous Injuries Produced By Forced Movements into Various Directions

6.3.1 *Forced dorsiflexion* was done on 4 preparations. The injuries produced may be seen from Table 1. In all cases a primary rupture occurred posteriorly in the deep layers of the deltoid ligament.

TABLE 1. LESIONS FROM FORCED DORSIFLEXION

STAGE	CASE 1	CASE 2	CASE 3	CASE 4
1	AVULSION FRACTURE BY TRACTION FROM PTTL	RUPTURE OF PTTL	AVULSION FRACTURE BY TRACTION FROM PTTL	PARTIAL RUPTURE OF PTTL AND TCL
2	AVULSION FRACTURE BY TRACTION FROM TCL + PARTIAL RUPTURE OF CFL	RUPTURE OF ITTL + SHORT FIBRES OF PTAFL	AVULSION FRACTURE BY TRACTION FROM ITTL	RUPTURE OF PTTL + ITTL + TCL + PARTIALLY PTAFL
3	TOTAL RUPTURE OF CFL + PTAFL	TOTAL RUPTURE OF CFL + PTAFL	RUPTURE OF TCL + SHORT FIBRES OF PTAFL	AVULSION FRACTURE BY TRACTION FROM PTAFL AND CFL

6.3.2 *Forced plantar flexion* was also done on 4 preparations, and the results are shown in Table 2. In these cases the primary injury was found in the anterior joint capsule and ATaFL.

TABLE 2. LESIONS FROM FORCED PLANTAR FLEXION

STAGE	CASE 1	CASE 2	CASE 3	CASE 4
1	RUPTURE OF ATaFL + ANTEROLATERAL CAPSULE	RUPTURE OF ANTERIOR CAPSULE	RUPTURE OF ANTERIOR CAPSULE	RUPTURE OF ANTERIOR CAPSULE + PARTIALLY OF ATaFL
2	RUPTURE OF THE WHOLE ANTERIOR CAPSULE	RUPTURE OF ATaFL AND PARTIALLY CFL	RUPTURE OF ATaFL	TOTAL RUPTURE OF ATTL
3	RUPTURE OF ATTL	RUPTURE OF SHORT FIBRES OF PTaFL + SUBTOTALLY OF CFL	RUPTURE OF ATTL + ITTL	RUPTURE OF ATaFL
4	TOTAL DELTOID LIGAMENT RUPTURE	TOTAL DELTOID LIGAMENT RUPTURE	TOTAL DELTOID LIGAMENT RUPTURE + RUPTURE OF SHORT FIBRES OF PTaFL	RUPTURE OF ITTL + SHORT FIBRES OF PTaFL

6.3.3 *Forced adduction-dorsiflexion* – like all the following investigations – was performed on 2 preparations. This movement produced exclusively injuries to the lateral ligaments, and in one of them primary rupture of the CFL (Table 3).

TABLE 3. LESIONS FROM FORCED DORSIFLEXION-ADDUCTION

STAGE	CASE 1	CASE 2
1	RUPTURE OF CFL	RUPTURE OF ATaFL + PARTIALLY CFL
2	RUPTURE OF PTaFL	RUPTURE OF CFL + PTaFL
3	RUPTURE OF ATaFL	

6.3.4 *Forced adduction* also resulted, in one case, first in a rupture of the CFL (Table 4).

TABLE 4. LESIONS FROM FORCED ADDUCTION

STAGE	CASE 1	CASE 2
1	TOTAL RUPTURE OF ATaFL + CFL + PTaFL	AVULSION FRACTURE FROM LAT. MALLEOLUS BY TRACTION FROM CFL
2		FURTHER FRACTURE BY TRACTION FROM ATaFL + CFL + SHORT OF PTaFL
3		RUPTURE OF LONG FIBRES OF PTaFL

6.3.5 *Forced adduction-plantar flexion,* on the other hand, caused primarily rupture of the ATaFL combined with a partial or complete tear of the CFL (Table 5).

TABLE 5. LESIONS FROM ADDUCTION-PLANTARFLEXION

STAGE	CASE 1	CASE 2
1	RUPTURE OF ATaFL + PARTIALLY CFL	PARTIAL RUPTURE OF ATaFL + CFL
2	RUPTURE OF ANTERIOR CAPSULE AND, SUB-TOTALLY, OF CFL	RUPTURE OF ANTERIOR CAPSULE + THE REST OF ATaFL AND, SUB-TOTALLY, CFL
3	RUPTURE OF PTaFL	RUPTURE OF THE REST OF CFL + SHORT FIBRES OF PTaFL
4		RUPTURE OF LONG FIBRES OF PTaFL

6.3.6 *Forced abduction-dorsiflexion* tore in one case the entire deltoid ligament, but in the other case its posterior portion ruptured first (Table 6).

TABLE 6. LESIONS FROM FORCED ABDUCTION-DORSIFLEXION

STAGE	CASE 1	CASE 2
1	TOTAL RUPTURE OF THE WHOLE DELTOID LIGAMENT	AVULSION FRACTURE FROM MED. MALLEOLUS BY TRACTION FROM PTTL, TCL, AND ITTL
2		FRACTURE OF THE MED. MALLEOLUS
3		RUPTURE OF THE ANTERO-MEDIAL CAPSULE

6.3.7 *Forced abduction* primarily caused rupture of the TCL – i.e. the superficial component of the deltoid ligament (Table 7).

TABLE 7. LESIONS FROM FORCED ABDUCTION

STAGE	CASE 1	CASE 2
1	RUPTURE OF TCL + PARTIALLY ITTL	RUPTURE OF TCL
2	RUPTURE OF ATTL + PTTL + THE REST OF ITTL	RUPTURE OF ITTL
3		RUPTURE OF ATTL + PTTL

6.3.8 *Forced abduction-plantar flexion* induced first rupture either of the superficial or of the anterior deep portion of the deltoid ligament (Table 8).

TABLE 8. LESIONS FROM FORCED ABDUCTION-PLANTARFLEXION

STAGE	CASE 1	CASE 2
1	RUPTURE OF TCL	RUPTURE OF ATTL
2	RUPTURE OF ITTL	RUPTURE OF ANTERIOR CAPSULE + TCL + ITTL
3	TOTAL RUPTURE OF DELTOID LIGAMENT	TOTAL RUPTURE OF DELTOID LIGAMENT

6.3.9 *Forced internal rotation-dorsiflexion* made primarily the lateral collateral ligaments burst, and thereupon the posterior deep component of the deltoid ligament ruptured (Table 9).

TABLE 9. LESIONS FROM FORCED DORSIFLEXION-INTERNAL ROTATION

STAGE	CASE 1	CASE 2
1	RUPTURE OF ATaFL + SHORT FIBRES OF PTaFL	RUPTURE OF SHORT FIBRES OF PTaFL
2	RUPTURE OF PTTL + PARTIALLY CFL	RUPTURE OF PTTL + ANTERIOR CAPSULE + PARTIALLY CFL
3	RUPTURE OF CFL + LONG FIBRES OF PTaFL	RUPTURE OF CFL + ITTL
4		RUPTURE OF ATaFL + TCL

6.3.10 *Forced internal rotation* entailed, as the first injury, a rupture of the ATaFL. Thereupon, the anterior, short fibres of the PTaFL were torn off the talus, but in no case did their substance itself rupture (Table 10).

TABLE 10. LESIONS FROM FORCED INTERNAL ROTATION

STAGE	CASE 1	CASE 2
1	RUPTURE OF ATaFL	RUPTURE OF ATaFL
2	RUPTURE OF SHORT FIBRES OF PTaFL	RUPTURE OF SHORT FIBRES OF PTaFL + LATERAL CAPSULE
3	RUPTURE OF CFL + LONG FIBRES OF PTaFL	RUPTURE OF CFL
4		RUPTURE OF LONG FIBRES OF PTaFL

6.3.11 *Forced internal rotation-plantar flexion* resulted in similar injuries, in one case finally followed by rupture of the ATTL, presumably due more to the plantar flexion than to the internal rotation of the talus (Table 11).

TABLE 11. LESIONS FROM FORCED PLANTAR FLEXION-INTERNAL ROTATION

STAGE	CASE 1	CASE 2
1	RUPTURE OF ATaFL + SHORT FIBRES OF PTaFL	RUPTURE OF ATaFL + ANTERIOR CAPSULE
2	RUPTURE OF ANTERIOR CAPSULE	RUPTURE OF SHORT FIBRES OF PTaFL
3	RUPTURE OF ATTL	PARTIAL RUPTURE OF CFL

6.3.12 *Forced external rotation-dorsiflexion* induced injuries medially as well as laterally, but in both cases the primary injury occurred posteriorly. In both preparations, moreover, the PTaFL was involved in the injury at some stage of the movement (Table 12).

TABLE 12. LESIONS FROM FORCED DORSIFLEXION-EXTERNAL ROTATION

STAGE	CASE 1	CASE 2
1	RUPTURE OF PTTL	AVULSION FRACTURE FROM LAT. MALLEOLUS BY TRACTION FROM CFL AND PTaFL
2	AVULSION FRACTURE DISTALLY IN THE MED. MALLEOLUS	FRACTURE OF THE MED. MALLEOLUS + RUPTURE OF ATaFL
3	RUPTURE OF PTaFL	

6.3.13 *Forced external rotation* also induced injuries laterally as well as medially. The primary damage was fairly comprehensive. In one case the deep portion of the deltoid ligament ruptured completely, while the superficial part of the ligament remained intact (Table 13).

TABLE 13. LESIONS FROM FORCED EXTERNAL ROTATION

STAGE	CASE 1	CASE 2
1	AVULSION FRACTURE FROM LAT. MALLEOLUS BY TRACTION FROM PTaFL AND CFL + RUPTURE OF ATTL + ITTL + PTTL	AVULSION FRACTURE FROM MED. MALLEOLUS BY TRACTION FROM ATTL, ITTL, AND TCL
2		AVULSION OF THE REST OF THE MED. MALLEOLUS + RUPTURE OF CFL + PTaFL

6.3.14 *Forced external rotation-plantar flexion* also resulted in one case in rupture of the deep part of the deltoid ligament without injury to the TCL, while in the other case the primary injury was more comprehensive, but still chiefly of a medial location (Table 14).

Let it be emphasized, furthermore, that *it was in no case possible to produce rupture of the distal tibiofibular ligaments!*

TABLE 14. LESIONS FROM FORCED PLANTAR FLEXION-EXTERNAL ROTATION

STAGE	CASE 1	CASE 2
1	RUPTURE OF ITTL + PTTL	RUPTURE OF ATTL + ITTL + PTTL + PARTIALLY OF TCL + MINOR AVULSION FROM THE LATERAL MALLEOLUS
2	AVULSION FRACTURE FROM THE MEDIAL MALLEOLUS BY TRACTION FROM ATTL	RUPTURE OF TCL + FRACTURE OF LAT. MALLEOLUS BY TRACTION FROM CFL AND PTaFL
3	AVULSION FROM MED. MALLEOLUS BY TRACTION FROM TCL	

6.4 Summary of the Functions Observed in the Individual Ligaments

ATaFL limits primarily internal rotation of the talus and also has an adduction-inhibiting function when the ankle joint is plantar flexed. Besides, it restricts, together with the anterior joint capsule, plantar flexion and possibly also exerts some inhibitory effect upon dorsiflexion which did increase sligthly after the ligament was cut. However, it was not ever possible to burst any part of the ATaFL by forced dorsiflexion.

CFL inhibits first and foremost adduction. When the ankle joint is in the neutral position and in dorsiflexion it appears to have at times an independent function, while in plantar flexion it acts in combination with the ATaFL. It plays no major role in internal rotation and also does not exert an independent inhibitory effect upon external rotation. At quite a late point of the trauma it may rupture, both in dorsi- and plantar flexion traumas, but it does not primarily influence these movements.

PTaFL exerts no independent stabilizing function apart from the fact that in dorsiflexion of the ankle both its short and long fibres inhibit external rotation. It also has an inhibitory effect upon dorsiflexion which, however, is primarily restricted by the medial ligaments. The short fibres of the ligament inhibit – after the ATaFL has been torn – internal rotation, whereas its long fibres do no

play any role in this movement. After rupture of the CFL first the short and then the long fibres inhibit adduction when the ankle joint is in dorsiflexion.

ATFL has only a questionable independent effect upon ankle stability, as its transection entailed only a very minimal increase in external rotation and an even slighter increment in dorsiflexion. It was also not possible to rupture this ligament – or other tibiofibular structures – by forced movements in the ankle joint.

The *tibiofibular syndesmosis* could not be cut in isolation owing to its anatomical situation. As also the ATFL, it plays only a very minor role in external rotation and apparently has just as little effect upon dorsiflexion.

PTFL has *per se* only a doubtful restrictive effect upon internal rotation. Cutting of the PTFL + the syndesmosis resulted in a further increase in internal rotation, but still of a slight extent.

Thus, the anterior part of the tibiofibular structures appears to play only a minimal role in external rotation – and their posterior part likewise in internal rotation. None of these structures has any influence upon ad- or abduction in the ankle joint.

TCL primarily limits abduction, as it increased after transection of this ligament which may also rupture isolated on forced abduction.

ATTL also has no independent function apart from some restrictive effect upon plantar flexion which is otherwise controlled primarily by the ATaFL and anterior joint capsule. In particular, cutting of this ligament does not increase external rotation, and also not abduction essentially. Together with the tibiofibular structures, it inhibits external rotation which increases far more after the combined transection of these structures than after isolated transection of each. In combination with the ITTL too, it exerts a limiting function upon this movement.

ITTL – after preceding rupture of the TCL – inhibits abduction which increases considerably after transection of these two ligaments. Both could also be ruptured by forced abduction, before any injury to other ligaments occurred. After rupture of the PTTL, it also restricts dorsiflexion, internal rotation, and external rotation.

PTTL has a restrictive effect upon dorsiflexion which increases after its transection. In all cases in which forced dorsiflexion was done, PTTL was the first structure to be torn. Besides, internal rotation increased – though minimally – after transection of this ligament which indeed was also included in the injuries that could be induced by forced internal rotation – but not until ruptures had occurred in the lateral ligaments.

Chapter VII

Discussion

7.1 Axial Relations of the Mobility Patterns

The mobility patterns were traced on the basis of a pre-defined centre of rotation, situated centrally in the talus on a line connecting the two malleolar apices when the ankle is in the neutral position. The mobility was traced as taking place around two defined – and accordingly constant – axes, i.e. a frontally placed axis around which dorsiplantar movement occurs and a vertically or horizontally placed axis around which internal/external rotation and adduction/abduction respectively occur. The strain gauges which measure the torque and the potentiometers measuring rotation are fixed to the lever which again is fixed to the talus. Therefore, the axes around which the torque is exerted and the movements take place, are constant in relation to the talus, but not in relation to the mortise (Fig. 63).

As already mentioned, the findings can be recorded only in two planes at a time. However, movements in all three planes generally occur during dorsiplantar flexion of the ankle. This is apparent from previously reported changes in the axis that take place in the joint during this movement (Barnett and Napier 1952, Sammarco et al. 1973, D'Ambrosia et al. 1976, Parlasca et al. 1979). Thus, the movement which must occur in a third plane is not recorded, but then it hardly plays any role in analysing the function of the individual ligaments. On the other hand, other unintended movements may occur in the unrecorded plane which may indirectly affect the mobility measured in the other two planes. An example: During the investigation of dorsiplantar flexion and internal-external rotation – i.e. movements in the sagittal and horizontal planes – there may theoretically occur, after transection of the lateral collateral ligaments, "spontaneous" and unintended adduction of the talus due to the weight of the movable parts of the preparation and apparatus. By reason of this unintended movement, not recorded in the mobility patterns, the axes in the talus alter in relation to the ankle mortise, and thus also in relation to the crural attachments of the remaining ligaments. Thereby, the extent of movement in the two recorded planes will be affected: It will increase if the movement in the third plane has entailed slackening of the remaining ligaments concerned, while it will decrease if it has entailed tightening. This error, caused by gravity, was eliminated as far as possible, partly by the position in which the preparations were fastened and partly by accurate equilibrium of the foot and that part of the apparatus which was fixed to the talus.

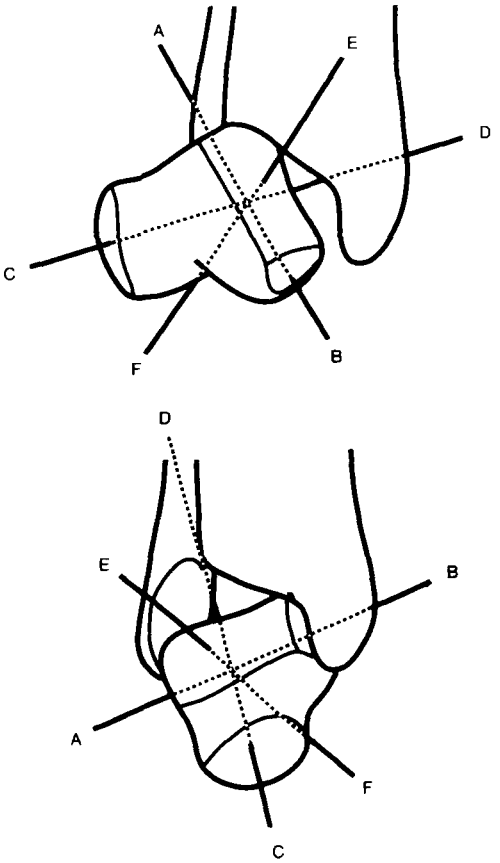


Fig. 63: The defined axes in the talus:
A-B: Dorsi-plantar flexion axis.
C-D: Adduction-abduction axis.
E-F: Internal-external rotation axis.

7.2 Mobility Patterns Versus Numerically Recorded Mobility

The mobility patterns afford a continuous picture of abduction and adduction as well as internal and external rotation respectively at any degree of dorsi- or plantar flexion. This continuity is lost when the mobility is expressed numerically, as also done in the present study, since that states only the maximum extent of the movement, regardless of the position in the ankle joint in other respects. By way of example, transection of a ligament may lead to only a very few degrees' increase in maximum internal rotation, which in dorsiflexion may perhaps increase appreciably more, but without reaching its peak value (Fig. 64). Here, it must be emphasized that it is *the appearance of the mobility patterns which is most representative of ligamentous function, not the numerical peak values.*

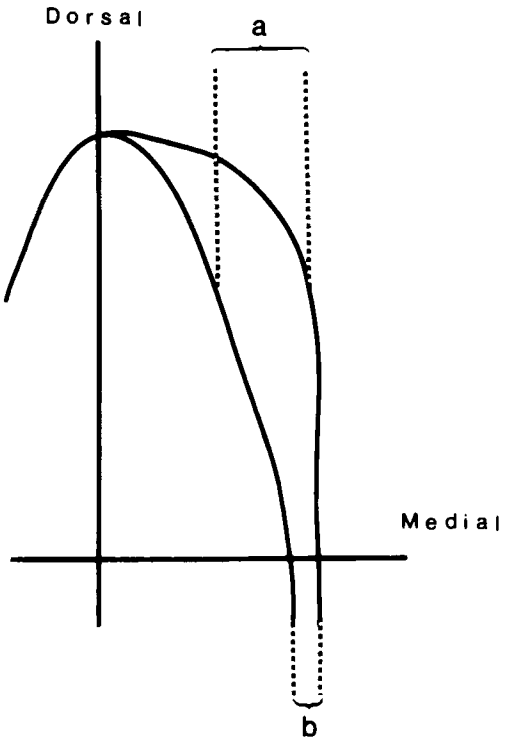


Fig. 64: The increase of, e.g. internal rotation, in dorsiflexion of the ankle may be greater (a) than the increase in maximum internal rotation (b).

7.3 Staging of Manually Induced Ligamentous Injuries

In the phase 3 studies the foot was affected manually into pre-defined directions until some kind of injury was produced. The force used was not recorded, as neither the talus nor the apparatus could tolerate that the force was exerted by the lever. Therefore, the findings do not indicate the absolute strength of the ligaments. The force was difficult to grade in practice. In some preparations – and in some directions – injuries were fairly easy to produce, while in other cases considerable force was required. The greater the force applied, the more difficult to stop the action at the first sign of an injury. Consequently, there was a tendency to the sudden occurrence of extensive injuries at great force. So, a sudden, extensive damage need not necessarily indicate that the injury occurs in fewer stages; the explanation may be lacking control of the induced movement.

Nor can it be taken for granted that injuries occurring *in vivo* proceed in exactly the same stages as in these studies. It must be taken into account that they were carried out on osteoligamentous preparations, without the active stabilization of the joint exerted by the intact muscles and tendons of the leg. Besides – and perhaps most essentially – the experimental situation differs from that of an actual trauma because the ankle preparations were not weight-bearing. This is perhaps the explanation why it was not possible to produce injuries to the distal tibiofibular ligaments.

7.4 Mobility at Intact Ligaments

Irrespective of the degree of dorsi- or plantar flexion, it is possible, even at intact ligamentous apparatus, to produce a certain translatory forward gliding of the talus in the mortise – an ADS. In the present studies this sign was most marked in a neutrally positioned ankle joint and least marked in dorsiflexion (Fig. 20). There was an enormous variation from preparation to preparation. An ADS must apparently attain at least 8-9 mm before it can, with certainty, be designated abnormal. In contrast, Anderson et al. (1952) – also on osteoligamentous preparations – found ADS of up to 2 mm at intact ligaments, regardless of the degree of dorsi- or plantar flexion, viz.

appreciably less than in the present studies. However, these authors dit not state which force they used to produce the ADS, and it is the very magnitude of this force which is decisive for the size of the ADS (Delplace and Castaing 1975). Ultimately, the ADS depends upon the laxity of the ligaments which may vary individually within wide limits (Bonnin 1944).

The total extent of dorsi-plantar movement was found to be about 58° in the present studies. Thereof dorsiflexion made up about 21° and plantar flexion about 38°. Here too, there was a certain dispersion, and analysis of the relationship between dorsiflexion and plantar flexion demonstrated that their extent was not mutually correlated (Fig. 28). Thus, a marked dorsiflexion in a preparation does not indicate anything concerning the size of plantar flexion or *vice versa*. Sammarco et al. (1973) reported that movements were equal, while Boone and Azen (1979) have claimed that plantar flexion is 4-5 times the dorsiflexion. Unlike their investigations, the present study was not performed *in vivo*. It showed that plantar flexion exeeded dorsiflexion in extent by an average of about 80 %.

Adduction at intact ligaments was 5°-6° and abduction almost 5°. Adduction did not in any case show the extremely high values reported by Bonnin (1950), Rubin and Witten (1960), Laurin et al. (1968), and Quellet et al. (1968), although the present study would be expected to show occasionally even higher values, as there was no active stabilization of the ankle joint. Only Husfeldt (1939) has stated that in intact ankle joints there may be some, slight abduction, while the present studies have demonstrated an abduction not much lesser than the adduction. This might indicate that abduction is primarily inhibited actively – i.e. by the tendons which pass the ankle joint medially – mainly the tendon of the posterior tibial muscle. The various movements in the ankle joint were unrelated to each other, except for ad- and abduction (Fig. 29). Abduction of a given extent seems to correspond to adduction of approximately the same extent.

Total mobility in the horizontal plane was found to be about 17° in the present study, distributed on internal rotation of rather more than 8° and external rotation of approximately the same extent. McCullough and Burge (1980) found the total horizontal rotation to be about 24°, produced by a torque of 3 Nm when the ankle joint was simultaneously loaded with 15 kg, and less at greater loading. Johnson et al. (1981) have reported the excursion of the movement to be 14°-15° without giving any clear indication about the torque with which they have affected the joint. Both statements may agree with the present results, but are not directly comparable, as the present experiments were performed with a torque of 1.5 Nm and no loading.

7.5 **Translatory Instability – ADS**

For a long time this type of instability has been utilized in diagnosing ruptures of the lateral collateral ligaments (Anderson et al. 1952, Gschwendt 1958, Broström 1966, Lindstrand 1976, Prins 1978).

The radiological investigations in phase 1 demonstrated, as already mentioned, appreciable ADS, even at intact ligaments. On the other hand, the increment in the ADS at progressing ligament transection of the lateral collateral ligaments into the anteroposterior direction was relatively slight, and there was a great dispersion of the values found (Fig. 20).

Furthermore, the study demonstrated that the greatest diagnostic benefit of the ADS is obtained when the examination is performed with the ankle joint in the neutral position or in dorsiflexion, not in plantar flexion.

Radiographically, the ADS can be measured in various ways (Fig. 4). Matters are complicated also by the fact that ADS is not a pure translatory forward gliding of the talus in the ankle mortise, but includes also a certain lateral shift of the talus, a certain dorsiflexion and – primarily – an internal rotation of the talus, simultaneously with its distal discplacement, the trochlea passing forward and down beneath the anterior edge of the tibia (Lindstrand 1976). The very fact that the ADS occurs in an increasing extent upon rupture of the lateral ligaments may seem strange, as the medial structures are intact. According to Inman (1976), the explanation is that the talus is not tightly bound to the medial malleolus by the deltoid ligament, but at this site the structures permit some forward and backward shift of the talus.

Indeed, the value of the ADS in diagnosing lateral ligamentous ruptures has been a matter of discussion. Güttner (1941) and Freeman (1965) have rarely been able to induce it, while Sanders (1977) admitted that it does occur, but dared not attribute to it any diagnostic importance. On the other hand, Castaing and Delplace (1972), Hackenbruch et al. (1976), Dannegger (1979), Noesberger (1979) state that on the basis of its size it is possible to deduce which ligaments are injured.

As reported by practically all authors, ADS can be interpreted with greater accuracy, if the examinations are performed also on the contralateral ankle joint. In the present study, however, this was not possible, as it was carried out on amputated limbs. However, the marked variations in the instability from one preparation to another do indicate that ADS can hardly be interpreted precisely enough to permit a deduction concerning the degree of a ligamentous injury.

7.6 **Rotatory Instability**

Just as the translatory instability in the ankle joint includes a certain rotatory instability, the types of rotatory instability are not devoid of some translatory element. Ad- as well as abduction entail distal shifting of the centre of rotation centrally in the talus. Moreover, internal and external rotation of the talus entail distal shifting of this bone, as the trochlea – laterally or medially – passes downward and forward beneath the distal anterior edge of the tibia. However, the translatory element is relatively modest in the rotatory movements: dorsi-plantar flexion, ad- and abduction, and internal and external rotation.

7.6.1 **Dorsiflexion instability**

From the *phase 2* investigations it is apparent that isolated cutting of the PTTL as well as PTaFL, ATaFL, PTFL, and ATFL increases dorsiflexion somewhat. Thus, it is a movement which is primarily restricted by these ligaments. This accords with Hönigschmied's finding (1877) that rupture of the deltoid ligament and PTaFL occurs in dorsiflexion traumas and with Ashhurst and Bromer's view (1922) that the movement is restricted by the ATFL and PTFL. It also confirms de Vogel's finding (1970) that the most plantar fibres of the ATaFL tighten during dorsiflexion. On the other hand, Bonnin's (1950) view, viz. that the movement is restricted by osseous collision between the anterior distal tibial edge and the talus, cannot be confirmed, as dorsiflexion increased after cutting of the ligaments.

However, the effect of ATFL and PTFL upon the extent of dorsiflexion is but indirect, as these ligaments do not insert on the talus itself. Transection of these ligaments renders it easier for the anterior, wide part of the trochlea to press the tibia and fibula apart and thus facilitates its entrance into the ankle mortise. In the *phase 3* investigations it was also not possible to induce rupture of the distal, tibiofibular structures – neither by forced dorsiflexion nor incidentally by any other form of forced rotation. Possibly, these strong ligaments rupture only if the ankle joint is weight-bearing.

In all cases in which forced dorsiflexion was exerted (*phase 3*, Table 1) the primary rupture was of the PTTL – i.e. the posterior component of the deltoid ligament. Not until the trauma was more violent did it involve also the lateral ligaments.

Cutting of all lateral collateral ligaments caused only a slight increase in dorsiflexion, unless the talus was simultaneously adducted. In that case dorsiflexion increased quite appreciably. Thus, in order to be able to cause rupture of the lateral ligaments a dorsiflexion trauma must be associated with adduction.

Cedell (1974) has described 4 cases of isolated rupture of the PTTL. In his opinion they were due to what he calls dorsiflexion-pronation traumas. On the basis of the transection experiments in *phase 2* as well as the provocation experiments in *phase 3*, it must be assumed that such an injury can arise also from a pure dorsiflexion trauma.

Accordingly, it appears that the PTTL, PTaFL, ATaFL, and – to a certain extent – the ATFL, syndesmosis, and PTFL contribute to restricting dorsiflexion in the intact ankle joint, while the primary injury in a dorsiflexion trauma affects the posterior, deep component of the deltoid ligament, viz. the PTTL.

7.6.2 **Plantar flexion instability**

Isolated cutting of the ATaFL causes an increase –

though slight – of plantar flexion (Figs. 30-33), while such an increase was questionable after cutting of the ATTL (Figs. 47-48). On the other hand, plantar flexion increased more when both structures were cut (Figs. 55-56). In the provocation experiments in *phase 3* the primary injury was observed in the anterior joint capsule or in the ATaFL which is anatomically situated as a constituent of the anterolateral joint capsule (Table 2). This agrees with what has previously been stated by Ashhurst and Bromer (1922), Robichon et al. (1972), and Wirth et al. (1978), but does not indicate that the movement is normally arrested by osseous collision. In one case of the present material rupture of the ATTL was included in the primary injury, as observed also by Nevin and Post (1964) in one case.

Thus, it seems that the structures which restrict plantar flexion are located mainly anteriorly and laterally, whereas ATTL and the more posterior ligaments are merely of secondary importance to the extent of plantar flexion.

7.6.3 **Adduction instability**

In the present study isolated cutting of the ATaFL increased adduction by an average of 5° – and in most cases only when the ankle joint was plantar flexed (Figs. 30 and 32). Further transection of the CFL again increased adduction by 11°-12°, in plantar flexion as well as in the neutral position (Fig. 30), while isolated cutting of the CFL had practically no effect upon stability (Figs. 36-39). Combined transection of the CFL and the long or short fibres of the PTaFL augmented adduction somewhat, and transection of the CFL plus the entire PTaFL entailed a marked increase of adduction in dorsiflexion of the ankle (Figs. 36 and 38). The last-named situation corresponds to the injury which Lauge-Hansen designated "supination-adduction injury, stage I". Presumably, it is a result of a pure adduction trauma (Frick 1981), but on a dorsal-flexed ankle. Isolated cutting of the PTaFL did not influence adduction (Fig. 40). In other words, the CFL must be considered the "key ligament" in adduction, although its transection in isolation plays only a questionable role in stability. It must be mentioned, however, that Pennal (1943), Leonard (1949), and Coltart (1951) have reported a slight TT after sectioning the CFL and that it has been measured by Wirth (1951) as no less than 10°. However, in conformity with the present findings, most authors (Carothers 1942, Laurin et al. 1968, Quellet et al. 1968, Vidal et al. 1974, Padovani 1975) have found the CFL to be of practically no importance to the stability of the talocrural joint.

Nevertheless, isolated rupture was induced by forced adduction in the provocation experiments of *phase 3* (Tables 3 and 4). Similar injuries have indeed been produced experimentally by Hönigschmied (1877) and Dias (1979). There have also been clinical reports on isolated CFL rupture (Hughes 1942, Berridge and Bonnin 1944, Coltart 1951, Evans 1953, Francillon 1962, Bouretz 1975, Prins 1978, Orlin et al. 1980, Solheim et al. 1980, Buch et al. 1981, Kievernagel 1981, Taillard et al. 1981, Tiedtke and Rahmanzadeh 1981). It may seem surprising that the CFL can rupture in isolation, when considering that such a rupture does not give rise to any notable talocrural instability, but it must be borne in mind that the CFL inserts onto the calcaneus and is accordingly also a subtalar ligament. The explanation is presumably that when it ruptures without simultaneous tears of other lateral collateral ligaments, it is an event associated with subtalar injury. Thus, it does not result in talocrural, but in subtalar instability (Francillon 1962, Laurin et al. 1968, Vidal et al. 1974).

Kaufmann (1922) maintains that an adduction trauma may cause rupture of the ATFL – possibly in conjunction with the ATaFL and CFL, and Murray (1942) as well as Mullins and Sallis (1958) have reported increased adduction after rupture of the ATFL or PTFL. On the other hand, Laurin et al. (1968) state that injury to these ligaments does not entail an increased TT. From the present study it is apparent that transection of the distal, tibiofibular ligaments does not increase adduction (Fig. 42) and that they could also not be damaged by forced adduction. Thus, the ATFL, syndesmosis, and PTFL do not play any role in adduction in the ankle joint.

Adduction is controlled exclusively by the lateral collateral ligaments which are the only structures that rupture in an adduction trauma. Radiographic examination for TT can, if performed in plantar flexion, neutral position as well as dor-

siflexion, contribute to a further specification of which ligaments are ruptured in each case. A finding of slight TT in plantar flexion, but no abnormality in the neutral position (Fig. 30) indicates isolated rupture of the ATaFL. This also accords with Broström's peroperative observations (1964). If major TT is found in the neutral position there is a likelihood of injury to the ATaFL as well as CFL (Fig. 30). If a TT cannot be induced in plantar flexion or in the neutral position but – of a slight extent – in dorsiflexion, this suggests rupture of the CFL plus PTaFL (Figs. 36 and 38). The most marked TT of course arises when all the lateral ligaments have been torn, and it is accompanied by a pronounced increase of dorsiflexion during simultaneous adduction (Figs. 30, 32, 34, 36, and 40). After total transection of these ligaments in the *phase 2* experiments the ankle joint was frequently so loose that it was impossible to plot a mobility pattern.

7.6.4 **Abduction instability**

It is agreed that abduction injuries affect primarily the deltoid ligament (Maissonneuve 1840, Hönigschmied 1877, Tillaux 1890, Husfeldt 1939, Lauge-Hansen 1942, Hendelberg 1943). Purvis (1982) states that these injuries affect first and foremost the anteromedial joint capsule – meaning evidently the ATTL – but otherwise it is still not clear which part of the deltoid ligament is most exposed in this situation.

On isolated cutting of the TCL the present experiments demonstrated an increase of 2°-3° in abduction (Fig. 45 and 49), and forced abduction in the neutral position and plantar flexion usually caused first rupture of this ligament (Tables 7 and 8). Further cutting of the ATTL or PTTL did not notably alter the abduction which, however, increased appreciably when the ITTL was transected (Fig. 49). The provocation experiments in *phase 3* showed that the ITTL rupture constitutes the 2nd stage in an abduction injury (Tables 7 and 8), thereafter followed by rupture of the ATTL or PTTL.

TCL reminds of the CFL in the way that it inserts onto the calcaneus and must accordingly be assumed to influence subtalar stability. Grath (1960) and Lanzetta and Meani (1979) have reported that this ligament does not possess an independent function in talocrural stability, whereas Padovani (1975) found increased external rotation after its transection. Moreover, Wirth et al. (1978) claim that it tightens in dorsiflexion, and thus is supposed to restrict this movement. These data are at variance with the present findings which demonstrated that the TCL is a factor in abduction – and only in that movement. This agrees with Kleiger's description (1956) of isolated TCL tear occurring in an "eversion trauma", meaning in the present terminology an abduction trauma.

When forced abduction was performed simultaneously with plantar flexion, there was one case of primary ATTL rupture (Table 8), while in dorsiflexion the rupture appeared more posteriorly in the deltoid ligament (Table 6), corresponding to the injury which Cedell (1974) described in just this type of trauma.

The mobility patterns did not exhibit increased abduction after sectioning the distal, tibiofibular ligaments (Fig. 42), and it was also not possible to rupture these structures by forced abduction. Consequently, the tibiofibular ligaments are of no importance in the abduction stability of the ankle.

In other words, abduction is limited primarily by the TCL and secondarily by the ITTL. In abduction traumas there may consequently be isolated injury to the superficial portion of the deltoid ligament or an injury to this portion plus the central, deep part of the ligament, while the anterior and posterior components may be intact. Reversely, abduction instability indicates rupture of the TCL and ITTL.

7.6.5 **Internal rotatory instability**

Numerous authors have reported internal rotatory instability following rupture of the ATaFL – often in association with an ADS (Dehne 1934, Hendelberg 1943, Leonard 1949, Anderson et al. 1952, Broström 1964, Cedell 1967, Olerud 1967, Percy et al. 1969, Robichon et al. 1972, Delplace and Castaing 1975, Lindstrand et al. 1978, Hackenbruch et al. 1979, Glasgow et al. 1980). On the other hand, the role of the CFL and PTaFL in internal rotation has not been elucidated.

In the present study there was, at intact ligaments, an internal rotation of about 8°. After cutting of the ATaFL it increased to about 18°, and after further cutting of the anterior, short fibres of

the PTaFL to about 23° (Fig. 33). If the CFL was cut instead of the short PTaFL fibres there was no increase in mobility (Fig. 31). Nor did isolated cutting of the PTaFL result in any internal rotatory instability (Fig. 41). Thus, internal rotation is primarily inhibited by the ATaFL and secondarily by the short fibres of the PTaFL. This agrees completely with the findings in the *phase 3* study in which provocation experiments almost constantly showed just this order of ruptures (Tables 9, 10 and 11). The above-mentioned increase of internal rotation is in the form of anterolateral rotatory instability, the movement occurring around an axis situated posteromedially in the ankle joint. Increased internal rotation also appeared after cutting of the PTTL or – considerably more marked – after cutting the PTTL + ITTL (Fig. 52). In that case, however, it is a question of posteromedial rotatory instability with the axis situated anterolaterally in the joint. By forced internal rotation it was not possible to induce primarily injuries of the medial ligaments – presumably because they are considerably stronger than the ATaFL (Sauer et al. 1978).

McCullough and Burge (1980) observed that horizontal rotation – by which they are presumably referring to internal rotation – increased by about 6° after cutting the ATaFL and by an almost equal amount after further cutting of the CFL. These findings are in contrast to the present ones which showed by far the greater increase in internal rotation already after cutting the ATaFL and that CFL played no essential role in this connection.

Güttner (1941) and Prins (1978) have described rupture of the ATaFL and the short fibres of the PTaFL, but without a simultaneous damage to the CFL. This is ascribed by Prins to the variation in the fibre direction in CFL pointed out by Ruth (1961). According to the present study, however, such an injury would rather be expected in an internal rotation trauma.

Transection of the tibiofibular ligaments increased internal rotation by only a very few degrees, but owing to their strength (Sauer et al. 1978) they are unlikely to rupture instead of the ATaFL.

Thus, internal rotation is primarily limited by the ATaFL and the short fibres of the PTaFL. Not until after these structures have been torn will injuries occur also in the CFL, PTTL, and ITTL.

7.6.6 External rotatory instability

Ruptures of the ATFL, the syndesmosis between the distal parts of the tibia and fibula, and PTFL have generally been attributed to external rotation traumas – possibly combined with simultaneous adduction or abduction in the ankle joint (Hönigschmied 1977, Lauge-Hansen 1942, Outland 1943, Berridge and Bonnin 1944, Bonnin 1950, Coltart 1951, Proctor 1954, Kleiger 1956, Kristensen 1956, Bolin 1961, Golterman 1964, Magnusson 1965, Cedell 1967, Frick 1978, Heim 1981). However, these ruptures are said to occur most often in association with other ligamentous injuries or fractures.

The present findings indicate that external rotation traumas may give rise to even extremely complex injuries. Transection of the ATaFL and CFL did not increase external rotation (Fig. 31), and isolated transection of the PTaFL increased it only very slightly in dorsiflexion (Fig. 41). Combined cutting of the CFL and PTaFL, in contrast, caused an increment in external rotation of about 6° (Figs. 37 and 39), as posterolateral rotatory instability, the movement taking place around an axis situated anteromedially in the ankle joint. Further involvement of the ATaFL in the injury entailed but a slight increase.

Cutting of the medially situated TCL did not alter either internal or external rotation, and further cutting of the ATTL (Fig. 48) also played no noticeable role. When also the ITTL was cut, internal rotation increased by one degree and external rotation by 2°-3°, viz. very slightly. Cutting of the TCL + PTTL had little effet upon external rotation which, however, increased by about 4° when the ITTL was sectioned too (Fig. 52). This increase in external rotation takes place around a posterolateral axis in the ankle joint and is thus an anteromedial rotatory instability. Combined cutting of the ATTL and PTaFL increased external rotation by only a couple of degrees (Fig. 59), as long as the distal tibiofibular structures were intact.

Cutting of the tibiofibular ligaments gave rise to an increase in internal as well as external rotation of 2°-3° (Fig. 43), which is very little when consid-

ering that often these structures are regarded as the site of predilection of external rotation injuries. However, external rotation increased essentially if the ATTL or PTaFL was cut as well (Figs. 43 and 53).

It follows that isolated cutting of each of the external rotation-inhibiting structures gives rise to only very little instability. This indicates that Staples (1960) is right in saying that external rotation traumas result in extremely complex injuries.

In the *phase 3* provocation experiments, forced external rotation gave predominantly injuries in the deep components of the deltoid ligament, and a tear of the TCL did not occur until later (Tables 12, 13, and 14). It was only in one case that it was possible to induce lateral injuries primarily – viz. by external rotation-dorsiflexion which tore the CFL and PTaFL – or more correctly avulsed them from the lateral malleolus at the site of their insertion. Incidentally, the same injury could be produced by forced adduction-dorsiflexion (Table 3). In no case could the distal tibiofibular ligaments be torn, but here it must be borne in mind that the investigations were performed on non-weigth-bearing ankle joints. Possibly, weight-bearing might have altered this condition.

In other words, external rotation is controlled by the ATTL, ITTL, PTTL, ATFL, tibiofibular syndesmosis, PTFL, PTaFL, and CFL, but these structures rarely seem to rupture isolated in an external rotation trauma. Indeed, it has been stated by Kleiger (1954), Dziob (1956), Staples (1960), and Edeiken and Cotler (1978) that such isolated injuries are rare and that the tibiofibular structures usually rupture together with the deltoid ligament. Hönigschmied (1877) could induce rupture of the tibiofibular ligaments in association with the PTaFL, and on forced supination-external rotation Lauge-Hansen (1950) observed tightening of the tibiofibular structures and PTaFL at the same time. This is in conformity with the present finding that cutting of the distal tibiofibular ligaments entailed but little increase in external rotation which, however, increased appreciably when the PTaFL too was cut. This is an injury which is presumably identical with the stage II supination-external rotation fracture described by Lauge-Hansen, the ligament being ruptured instead of producing a fibular fracture by traction.

Thus, an external rotation trauma can induce injuries of several ligamentous combinations. It can cause rupture of the CFL + PTaFL, rupture of the ATTL + ATFL + the syndesmosis between the tibia and fibula + PTFL, and it can rupture the tibiofibular structures + PTaFL or – finally – rupture of the PTTL + ITTL. In the present *phase 3* investigations it was striking to note that the TCL was not involved in the trauma until a late stage. Consequently, an intact superficial part of the deltoid ligament, tantamount to the absence of abduction instability, does not guarantee intact deep components too.

Chapter VIII

Conclusions

On the basis of the results reported in Chapter VI and the discussion in Chapter VII concerning the various types of instability and appurtenant ligaments, the following conclusions can be drawn:

8.1 Conclusions Regarding the Restriction of Movements in the Ankle Joint

Dorsiflexion in the ankle joint is primarily restricted by the posterior tibiotalar ligament which may rupture in isolation in a dorsiflexion trauma. The posterior talofibular ligament contributes in some measure to limiting dorsiflexion. Moreover, the distal tibiofibular ligaments play an indirect role.

Plantar flexion is restricted by the anterior joint capsule and the anterior talofibular ligament which could be ruptured by forced movement in this direction in the phase 3 provocation experiments.

Adduction is restricted in plantar flexion by the anterior talofibular ligament, while in the neutral position it is inhibited by this ligament in association with the calcaneofibular ligament. In dorsiflexion of the ankle adduction is inhibited by the calcaneofibular ligament plus the posterior talofibular ligament.

Abduction is restricted primarily by the tibiocalcaneal ligament which indeed ruptures in isolation on forced movement in this direction. Thereafter, the movement is inhibited by the intermediate tibiotalar ligament. Not until then do the anterior and posterior components of the deltoid ligament appear to enter into their role in abduction.

Internal rotation is primarily restricted by the anterior talofibular ligament and thereafter by the anterior, short fibres of the posterior talofibular ligament. The posterior tibiotalar ligament too inhibits internal rotation, especially in association with the intermediate tibiotalar ligament, but these ligaments are so strong that an internal rotation trauma will instead primarily tear the lateral ligaments.

External rotation is restricted by an interplay of many structures. Forced external rotation can induce rupture of (a) the calcaneofibular ligament + the posterior talofibular ligament, (b) the anterior tibiotalar ligament + the anterior tibiofibular ligament + the tibiofibular syndesmosis + the posterior tibiofibular ligament, (c) the tibiofibular ligaments + the posterior talofibular ligament, or (d) the posterior and intermediate tibiotalar ligaments.

8.2 Conclusions Regarding the Function of Individual Ligaments

The anterior talofibular ligament limits plantar flexion as well as internal rotation of the talus, and with the ankle in plantar flexion it restricts adduction. Besides, it appears to exert an inhibitory function in dorsiflexion which tightens its most plantar fibres.

Rupture of this ligament gives rise to increased internal rotation of the type anterolateral rotatory instability.

The calcaneofibular ligament hardly plays any independent role in the stability of the ankle joint. Together with the anterior talofibular ligament it limits adduction when the ankle is in the neutral position and in plantar flexion, and together with the posterior talofibular ligament it restricts the same movement in dorsiflexion. In addition, these two ligaments combined inhibit external rotation.

Isolated rupture of the calcaneofibular ligament does not give rise to any major talocrural instability. The cases of isolated rupture of this ligament reported in the literature are presumably interpretable as a manifestation of a subtalar trauma rather than a talocrural injury.

The posterior talofibular ligament is composed of two structures, the anterior short and the posterior long fibres. *Per se* this ligament seems to have little influence upon ankle stability, providing an otherwise intact ligament apparatus. It is only in dorsiflexion that it appears to exert a restricting function upon external rotation. The short fibres restrict internal rotation after the anterior talofibular ligament has been torn. In asso-

ciation with the long fibres they limit dorsiflexion to some extent, and the ligament as a whole inhibits, in association with the calcaneofibular ligament, partly adduction in dorsiflexion and partly – more markedly – external rotation. Together with the distal tibiofibular ligaments it also has a definitely limiting effect upon external rotation.

The anterior tibiofibular ligament, the tibiofibular syndesmosis, and the posterior tibiofibular ligament play only a small part in ankle stability, providing an otherwise intact ligamentous apparatus.

Presumably, they rarely rupture without the presence of injuries to other structures. In the course of an external rotation injury they may rupture – either together with the anterior portion of the deltoid ligament or together with the posterior talofibular ligament. Theoretically, they may also rupture in internal rotation traumas in which, however, the far weaker anterior talofibular ligament must be expected to be torn first. They have no influence upon the extent of ad- or abduction.

The tibiocalcaneal ligament restricts abduction and can rupture in isolation in an abduction trauma and thus give rise to abduction instability.

The anterior tibiotalar ligament, including the fibres to the navicular bone, does not play any independent role in ankle stability. In association with the anterior talofibular ligament it exerts some inhibitory effect upon plantar flexion, and in association with the distal, tibiofibular structures or with the intermediate tibiotalar ligament it inhibits external rotation. It seldom ruptures in isolation, but most often in combination with other ligaments.

When the tibiocalcaneal ligament has already been torn, the *intermediate tibliotalar ligament* inhibits abduction. These two structures may rupture in an abduction trauma without any damage to the anterior and posterior components of the deltoid ligament. Together with the anterior or posterior tibiotalar ligament it limits external rotation, and together with the latter also internal rotation and dorsiflexion.

The posterior tibiotalar ligament restricts dorsiflexion and may rupture in isolation in a dorsiflexion trauma. It also limits internal rotation – less so alone than together with the intermediate tibiotalar ligament. However, an internal rotation trauma will primarily cause rupture of the more slender anterior talofibular ligament.

With the reserve needed because the investigations were performed on non-weightbearing osteoligamentous preparations, it may be mentioned that they also demonstrated the following:

The lateral collateral ligaments do not necessarily rupture in an anteroposterior sequence. The anterior talofibular ligament may rupture together with the short fibres of the posterior talofibular ligament in an internal rotation trauma, leaving an intact calcaneofibular ligament which, on the other hand, may rupture in association with the short fibres of the posterior talofibular ligament in an external rotation trauma or an adduction trauma in dorsiflexion of the ankle. Isolated rupture of the calcaneofibular ligament, reported occasionally, is presumably caused by a subtalar, not a talocrural trauma.

Adduction instability may to some extent contribute to a distinction between the various forms of rupture in the lateral ligaments. A slight talar tilt in plantar flexion, but none in the neutral position, indicates rupture of the anterior talofibular ligament.

A more pronounced tilt in the neutral position will be found when both the anterior talofibular and the calcaneofibular ligaments have been torn.

A slight talar tilt in dorsiflexion, but none in the neutral position or in plantar flexion, may be taken to suggest rupture of the calcaneofibular as well as posterior talofibular ligament.

In abduction traumas the superficial components of the deltoid ligament may rupture without any injury to the deep components.

In external rotation traumas the deep portion of the deltoid ligament may rupture, although the superficial component remains intact.

Summary

The purpose of this study was to elucidate the function of the ligamentous structures of the ankle joint, the traumas in which they may rupture, and the types of instability caused by such ruptures.

Most previous experimental investigations on the function of ankle ligaments have been performed on osteoligamentous preparations either by forcing a movement in the joint into more or less well-defined directions and observing the resulting injuries or else by cutting ligaments in various combinations and describing the resulting instability. As a rule, this has been done without inducing the increased mobility by a defined torque and without being able to demonstrate the instability continuously in all degrees of dorsi- or plantar flexion.

On the basis of these previous studies, the anterior talofibular ligament appears to limit internal rotation, while its role in adduction of the talus has not been clarified. The calcaneofibular ligament *per se* does not seem to be a factor in adduction. True, there is not complete agreement in this respect, and a few authors have been able to rupture this ligament in isolation by forced adduction. The posterior talofibular ligament seems to restrict dorsiflexion, and perhaps it plays a role, in conjunction with the calcaneofibular ligament, in adduction when the ankle joint is in dorsiflexion. The anterior tibiofibular ligament, and the distal tibiofibular structures on the whole, are assumed to limit external rotation, but it has not been clarified whether they influence adduction and abduction in the ankle joint. Little has been reported about the individual structures which make up the deltoid ligament, as most authors have not distinguished them from each other. However, in combination with the anterior talofibular ligament, the tibiotalar ligament seems to limit the translatory forward gliding of the talus – the so-called anterior drawer sign – and together they presumably inhibit plantar flexion. It has not been possible to find any description of the function of the intermediate tibiotalar ligament, while the posterior tibiotalar ligament has been reported by one author to inhibit internal rotation. Judging by the literature, the function of the tibiocalcaneal ligament seems comprehensive, since it is reported to limit external rotation, dorsiflexion, as well as plantar flexion.

The present investigations were divided into three phases:

Phase 1 was concerned with elucidating the correlation of injuries to the lateral collateral ligaments to internal rotatory instability, talar tilt, and the anterior drawer sign. Like the subsequent investigations, this phase was carried out on osteoligamentous preparations. Through the distal end of the tibia and through the talus a nail was inserted in the posteroanterior direction. These nails were used as radiopaque indicators in radiographic measurement of the various degrees of instability. The anterior drawer sign was produced by a 7 kg traction on the calcaneus, while internal rotation and adduction were produced by manual action upon the foot. In the course of successive cutting of the ligaments in the anteroposterior direction 7 preparations were studied.

For *phase 2* a measuring apparatus was devised to investigate the mobility of the ankle joint. By the aid of this apparatus it was possible to record rotatory movements in the joint when the talus was affected by a defined torque. A lever, furnished with strain gauges and connected to two potentiometers, was fastened to the talus. This lever was moved manually in a way so that during a "circumduction" of the lever the torque was constant at 1.5 Nm in the dorsal, medial, plantar, or lateral direction. By means of an X-Y writer or – at a later stage of the studies – by a computer, the resulting movement could be plotted in the form of mobility patterns. Patterns were plotted partly at intact ligaments and partly after transection of the lateral and medial collateral ligaments and the distal tibiofibular ligaments, isolated and in various combinations. A total of 113 preparations were investigated in this manner.

The apparatus was also used, partially, in *phase 3*. In this phase injuries in the ankle ligaments were induced. With the guidance of the X-Y writer, which plots the movement in the joint, the talus was forced into given, accurately defined directions. Thereafter, the induced injuries and

their sequence were described. The movements were dorsiflexion, plantar flexion, adduction, abduction, internal rotation, external rotation, and various combinations thereof. A total of 32 preparations were studied in this way.

The radiographic *phase 1* study demonstrated a correlation between the degree of ligament transection laterally and internal rotation which increased in step with progressing ligament transection in the anteroposterior direction. Both internal rotatory instability and talar tilt seem to be reliable diagnostic signs in detecting injuries to the lateral collateral ligaments, while the anterior drawer sign was less constant.

In *phase 2* cutting of the anterior talofibular ligament entailed an increase in internal rotation. There was no notable further increase on cutting the calcaneofibular ligament as well, but it did occur when the short fibres of the posterior talofibular ligaments were cut instead.

Isolated cutting of the anterior talofibular ligament gave rise to increased adduction in plantar flexion, but not in the neutral position of the ankle. Further cutting of the calcaneofibular ligament caused marked adduction instability – viz. talar tilt – at all degrees of dorsi- and plantar flexion.

Cutting of the calcaneofibular ligament and posterior talofibular ligament entailed external rotatory instability as well as adduction instability – but the latter only in dorsiflexion of the ankle.

Isolated cutting of the distal tibiofibular ligaments did little to alter ankle stability, but further cutting of either the posterior talofibular or the anterior tibiotalar ligament appreciably increased the external rotatory instability.

Cutting of the tibiocalcaneal ligament resulted in increased abduction which further increased when the intermediate tibiotalar ligament was cut as well.

Isolated cutting of the anterior tibiotalar ligament did not cause any instability, while isolated cutting of the posterior tibiotalar ligament increased dorsiflexion a little and entailed increased internal rotation which was further augmented by cutting also the intermediate tibiotalar ligament.

In the *phase 3* study it was observed that forced dorsiflexion induced primarily rupture of the posterior tibiotalar ligament.

Forced internal rotation caused first rupture of the anterior talofibular ligament and then of the short fibres of the posterior talofibular ligament, whereas the calcaneofibular ligament remained intact.

Forced external rotation entailed primarily rupture of the deep layers of the deltoid ligament, while the superficial tibiocalcaneal ligament remained intact.

Forced adduction caused, in the neutral position and in dorsiflexion, isolated rupture of the calcaneofibular ligament in a few cases, whereas the anterior talofibular ligament was involved in the primary injury when the ankle was plantar flexed.

Forced abduction first tore the tibiocalcaneal ligament – i.e. the superficial portion of the deltoid ligament – and thereafter the intermediate tibiotalar ligament. Only in plantar flexion did this movement cause, in one case, a primary injury to the anterior tibiotalar ligament.

The anterior drawer sign is considered unreliable by some authors, while others claim that on the basis of its size they can deduce which of the lateral collateral ligaments have ruptured in a given case. The present findings indicate that in rupture of the lateral ligaments – in which it is used chiefly – the sign is not reliable enough to permit such a distinction.

Dorsiflexion is restricted by the ligaments situated posteriorly on the ankle joint – first and foremost the posterior tibiotalar ligament which may rupture in isolation on forced dorsiflexion. The tibiofibular ligaments do not appear to play any direct role in the extent of this movement. One author has been able to induce partial rupture of the anterior talofibular ligament by forced dorsiflexion. This could not be done in the present studies – which could also not confirm the view that the movement is limited by the calcaneofibular ligament or that the limitation could be osseous.

In most authors' view *plantar flexion* is restricted by the anterior joint capsule and the anterior talofibular ligament, and this was confirmed by the present studies. They did not, however, confirm the opinion that this movement is inhibited by the calcaneofibular or posterior talofibular ligament. The anterior tibiotalar ligament appears

to play a less essential role in plantar flexion than would be expected in view of its situation.

Adduction is limited, in plantar flexion, by the anterior talofibular ligament, in the neutral position by this ligament plus the calcaneofibular ligament, and in dorsiflexion by the calcaneofibular ligament plus the posterior talofibular ligament. The cases of isolated rupture of the calcaneofibular ligament on record can hardly be interpreted as a consequence of a talocrural trauma, but rather as the result of a subtalar injury.

Numerous authors have reported that *abduction* is limited by the deltoid ligament, but without stating by which part of it. According to the present studies, the tibiocalcaneal ligament has this specific function, and the superficial portion of the deltoid ligament may rupture in isolation in an abduction trauma. On the other hand, the studies, done on non-weightbearing preparations, could not confirm that abduction can give rise to injuries to the distal tibiofibular ligaments.

According to previous authors, *internal rotation* is limited by the anterior talofibular ligament, and this was confirmed by the present studies. A few authors have stated that after rupture of this ligament, the movement is restricted by the calcaneofibular ligament. This could not be confirmed. On the contrary, this ligament does not appear to exert any special influence upon internal rotation which is restricted, under these circumstances, by the anterior short fibres of the posterior talofibular ligament.

External rotation is reported by most authors to be limited by the distal tibiofibular ligaments, in particular the anterior tibiofibular ligament, alone or together with the deltoid ligament. However, the limitation of external rotation is extremely complex. Cutting of the distal tibiofibular structures entails but little increase in external rotation, and rupture of these structures must be expected to be most often accompanied by rupture of the anterior tibiotalar ligament, the posterior tibiotalar or posterior talofibular ligament. Still, forced external rotation may also cause injuries to the calcaneofibular ligament in combination with the posterior talofibular ligament, without simultaneously injuring the tibiofibular ligaments.

Further investigation of existing *adduction instability* can contribute to some extent to distinguishing between various forms of rupture of the lateral collateral ligaments: A slight increase of adduction in plantar flexion indicates rupture of the anterior talofibular ligament, while a more marked increase in the neutral position indicates rupture of this ligament as well as the calcaneofibular ligament. Minor increase of adduction in dorsiflexion, without a corresponding increase of adduction in plantar flexion, however, indicates rupture of the calcaneofibular and posterior talofibular ligaments.

Previously, a distinction has been made only exceptionally between the various structures of the deltoid ligament. Generally, it has merely been stated that this ligament may be injured in abduction and in external rotation injuries. The present investigations demonstrated that the *superficial portion of the deltoid ligament* may rupture in isolation on forced abduction, whereas its *deep components* may rupture in external rotation traumas without the superficial portion necessarily being involved.

Resumè på dansk

Formålet med dette arbejde har været at belyse funktionen af ankelleddets ligamentære strukturer, de traumer, ved hvilke disse ligamenter kan rumpere, samt de instabilitetsformer, sådanne rupturer kan give anledning til.

Tidligere experimentelle undersøgelser over ankelligamenternes funktion er oftest foretaget på osteoligamentære præparater, ved at man enten har forceret en bevægelse i leddet i mere eller mindre definerede retninger og iagttaget de herved opståede læsioner, eller ved at man har foretaget ligamentoverskæringer i forskellige kombinationer og beskrevet den instabilitet, der herved er opstået, – som oftest uden at frembringe den øgede bevægelighed med et defineret moment, og uden at man har kunnet demonstrere instabiliteten kontinuerligt ved enhver grad af dorsal- eller plantarflektion.

Ud fra disse arbejder synes lig. talofibulare anterius at begrænse indadrotationen, mens dets betydning for adduktionen af talus ikke er klarlagt. Lig. calcaneofibulare synes ikke i sig selv at betyde noget for adduktionen, omend der ikke er fuld enighed herom, og det har været muligt for enkelte forfattere at sprænge dette ligament isoleret ved forceret adduktion. Lig. talofibulare posterius synes at begrænse dorsalflektionen og spiller måske sammen med lig. calcaneofibulare en rolle for adduktionen, når ankelleddet er dorsalflekteret. Lig. tibiofibulare anterius og i det hele taget de distale, tibiofibulære strukturer antages at begrænse udadrotationen, men det er uklart, hvorvidt de har indvirkning på adduktionen og abduktionen i ankelleddet. De enkelte ligamenter, der tilsammen udgør lig. deltoideum, er kun lidt belyst, idet de færreste forfattere har skelnet mellem dem. Lig. tibiotalare anterius synes dog, – sammen med lig. talofibulare anterius – at begrænse den translatoriske fremadgliden af talus – det såkaldte forreste skuffesymptom, og sammen hæmmer de formentlig plantarflektionen. Lig. tibiotalare intermedius' funktion er ikke fundet beskrevet, mens lig. tibiotalare posterius af en enkelt forfatter angives at hæmme indadrotationen. Lig. tibiocalcaneares funktion må ud fra litteraturen forventes at være omfattende, idet ligamentet angives at begrænse både udadrotation, dorsalflektion og plantarfleksion.

I det her foreliggende arbejde er undersøgelserne opdelt i tre faser.

I *fase 1* belyses sammenhængen mellem læsioner af de collaterale, laterale ligamenter og indadrotationsinstabilitet, talar tilt og det forreste skuffesympton. Som også de efterfølgende undersøgelser er denne foretaget på osteoligamentære præparater. Der er igennem den distale del af tibia og gennem talus indbanket søm i retningen bagfra-fortil. Disse er anvendt som røntgenfaste indikatorer ved en radiologisk udmåling af de forskellige instabilitetsgrader. Det forreste skuffesystem frembragtes ved traction med 7 kg på calcaneus, mens indadrotation og adduktion frembragtes ved manuel påvirkning af foden. Under successiv ligamentoverskæring i retningen forfra-bagtil undersøgtes 7 præparater.

Til *fase 2* udvikledes et måleapparatur til undersøgelse af ankelleddets bevægelighed. Dette apparatur muliggjorde registrering af rotatoriske bevægelser i leddet, når talus påvirkedes med et defineret moment. En momentarm, forsynet med strain gauges og forbundet med to potentiometre, fixeredes til talus. Denne arm førtes manuelt således, at momentet under en "circumduction" af armen konstant var 1.5 Nm i enten dorsal, medial, plantar eller lateral retning, og ved hjælp af en X-Y skriver eller – senere i undersøgelsesrækken – en computer, kunne den resulterende bevægelse optegnes i form af bevægemønstre. Der optegnedes mønstre dels ved intakte ligamenter, og dels efter overskæring af de collaterale laterale og mediale ligamenter og de distale, tibiofibulære strukturer, – enkeltvis og i forskellige kombinationer. Ialt 113 præparater undersøgtes på denne måde.

Ovennævnte apparatur anvendtes tildels også i *fase 3*. Her frembragtes læsioner i ankelligamenterne, idet man under vejledning af den X-Y skriver, som optegner bevægelsen i leddet, forcerede talus i givne, således nøje definerede retninger. De herved frembragte læsioner og deres rækkefølge blev derpå beskrevet. Der udførtes forceret dorsalflektion, plantarflektion, adduktion, abduktion, indadrotation, udadrotation samt for-

skellige kombinationer af disse bevægelser. Ialt 32 præparater undersøgtes således.

Den radiologiske *fase 1*-undersøgelse viste, at der er korrelation mellem graden af ligamentoverskæring lateralt og indadrotationen, således at denne øges i takt med fremadskridende ligamentoverskæring i retningen forfra-bagtil. Både indadrotationsinstabiliteten og talar tilt synes at være pålidelige diagnostika, når det drejer sig om læsioner af de collaterale, laterale ligamenter, mens det forreste skuffesymptom var mere inkonstant.

I *fase 2* fandtes, at overskæring af lig. talofibulare anterius medførte en forøgelse af indadrotationen. Denne tiltog ikke mærkbart ved yderligere overskæring af lig. calcaneofibulare, hvilket den derimod gjorde, når i stedet lig. talofibulare posterius' korte fibre blev gennemskåret.

Isoleret overskæring af lig. talofibulare anterius gav øget adduktion i ankelleddets plantarflektionsstilling, men ikke i neutralstilling. Ved yderligere overskæring af lig. calcaneofibulare fandtes udtalt adduktionsinstabilitet – altså talar tilt – ved enhver grad af dorsal- eller plantarflektion.

Overskæring af lig. calcaneofibulare og lig. talofibulare posterius medførte både en udadrotationsinstabilitet og en adduktionsinstabilitet, – det sidste dog kun i ankelleddets dorsalflektionsstilling.

Overskæring af de distale, tibiofibulære ligamenter alene ændrede kun i ringe grad ankelleddets stabilitet, mens ved yderligere overskæring af enten lig. talofibulare posterius eller lig. tibiotalare anterius udadrotationen øgedes betydeligt.

Overskæring af lig. tibiocalcaneare medførte øget abduktion, som igen tiltog, når også lig. tibiotalare intermedius blev gennemskåret.

Isoleret overskæring af lig. tibiotalare anterius gav ingen instabilitet, mens isoleret overskæring af lig. tibiotalare posterius øgede dorsalflektionen lidt og medførte forøget indadrotation, som yderligere tiltog, når også lig. tibiotalare intermedius blev couperet.

Ved *fase 3*-undersøgelsen fandtes, at forceret dorsalflektion primært frembragte ruptur af lig. tibiotalare posterius.

Ved forceret indadrotation opstod der først ruptur af lig. talofibulare anterius og derefter af de korte fibre i lig. talofibulare posterius, mens lig. calcaneofibulare fortsat var intakt.

Ved forceret udadrotation forvoldtes primært ruptur i lig. deltoideums profunde lag, mens det overfladiske lig. tibiocalcaneare fortsat var intakt.

Forceret adduktion medførte i neutralstilling og dorsalflektion i enkelte tilfælde isoleret ruptur af lig. calcaneofibulare, mens lig. talofibulare anterius indgik i den primære læsion, når ankelleddet var plantarflekteret.

Ved forceret abduktion rumperede først lig. tibiocalcaneare – altså den overfladiske del af lig. deltoideum – efterfulgt af lig. tibiotalare intermedius. Kun i plantarflektion fandtes i et enkelt tilfælde primært læsion af lig. tibiotalare anterius.

Det forreste skuffesymptom anses af nogle forfattere for upålideligt, mens andre ud fra dets størrelse mener at kunne slutte sig til, hvilke af de collaterale, laterale ligamenter, der i et givet tilfælde er rumperede. Herværende undersøgelse tyder på, at tegnet ved ruptur af de laterale ligamenter – hvor det først og fremmest anvendes – ikke er så pålideligt, at det muliggør en sådan findiagnostik.

Dorsalflektionen begrænses af de bag ankelleddet liggende ligamenter, – først og fremmest lig. tibiotalare posterius, som kan rumpere isoleret ved forceret dorsalflektion. Derimod synes de tibiofibulære ligamenter ikke direkte at spille nogen rolle for bevægelsens omfang. En enkelt forfatter har ved forceret dorsalflektion kunnet frembringe en partiel ruptur af lig. talofibulare anterius, hvilket ikke lykkedes i disse undersøgelser. Heller ikke den opfattelse, at bevægelsen begrænses af lig. calcaneofibulare eller at begrænsningen skulle være ossøs har kunne bekræftes.

Plantarflektionen begrænses af den forreste ledkapsel og lig. talofibulare anterius i overensstemmelse med de fleste undersøgeres opfattelse. Derimod har undersøgelserne ikke bekræftet den opfattelse, at bevægelsen skulle hæmmes af lig. calcaneofibulare eller lig. talofibulare posterius. Lig. tibiotalare anterius synes at spille en mindre væsentlig rolle for plantarflektionen end man kunne forvente ud fra dets beliggenhed.

Adduktionen begrænses i ankelleddets plantarflektionsstilling af lig. talofibulare anterius, mens den i neutralstilling begrænses af dette ligament sammen med lig. calcaneofibulare og i dorsalflektionsstilling af lig. calcaneofibulare sammen med lig. talofibulare posterius. De i litteraturen be-

skrevne tilfælde af isoleret ruptur af lig. calcaneofibulare er næppe at opfatte som en følge af et talocruralt traume, men må snarere være et led i en subtalær læsion.

Abduktionen begrænses ifølge talrige forfattere af lig. deltoideum, uden de dog har angivet, hvilken del af ligamentet, det drejer sig om. Ifølge disse undersøgelser er det specifikt lig. tibiocalcaneare, der har denne funktion, og denne overfladiske del af lig. deltoideum kan rumpere isoleret ved et abduktionstraume. Derimod har man, ved disse undersøgelser på ikke vægtbærende præparater, ikke kunnet bekræfte, at der ved abduktion kan opstå læsioner i de distale, tibiofibulære ligamenter.

Indadrotationen begrænses ifølge tidligere arbejder af lig. talofibulare anterius, hvilket også bekræftes ved herværende undersøgelser. Enkelte forfattere har angivet, at efter overskæring eller ruptur af dette ligament, så begrænses bevægelsen af lig. calcaneofibulare. Dette har ikke kunnet bekræftes. Tværtimod synes denne struktur ikke at have nogen væsentlig betydning for indadrotationen, som under disse forhold begrænses af de forreste, korte fibre i lig. talofibulare posterius.

Udadrotationen angives af de fleste at begrænses af de distale, tibiofibulære ligamenter, – specielt lig. tibiofibulare anterius, alene eller sammen med lig. deltoideum. Begrænsningen af udadrotationen er imidlertid meget komplex. Overskæring af de distale tibiofibulære strukturer medfører kun en ringe forøgelse af udadrotationen, og ruptur af disse strukturer må forventes oftest at være ledsaget af ruptur af lig. tibiotalare anterius, lig, tibiotalare posterius eller lig. talofibulare posterius. Dog kan forceret udadrotation også bevirke læsioner af lig. calcaneofibulare sammen med lig. talofibulare posterius, uden samtidig læsion af de tibiofibulære ledbånd.

En nærmere undersøgelse af en tilstedeværende *adduktionsinstabilitet* kan i nogen grad bidrage til en skelnen mellem de forskellige former for ruptur af de collaterale, laterale ligamenter, idet en beskeden forøgelse af adduktionen i plantarflektion tyder på ruptur af lig. talofibulare anterius, mens en mere udtalt forøgelse i neutralstilling viser ruptur af både dette ligament og lig. calcaneofibulare. En mindre adduktionsforøgelse i dorsalflektion, uden tilsvarende øget adduktion i plantarflektion tyder derimod på ruptur af lig. calcaneofibulare og lig. talofibulare posterius.

Man har tidligere kun undtagelsesvis skelnet mellem de forskellige strukturer i lig. deltoideum, og har almindeligvis kun omtalt, at ligamentet kan læderes ved abduktionstraumer og udadrotationstraumer. Disse undersøgelser viser, at *den overfladiske del af lig. deltoideum* kan rumpere isoleret ved forceret abduktion, mens *den dybe del af ligamentet* kan rumpere ved udadrotationstraumer, uden at den overfladiske del nødvendigvis også er læderet.

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