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EXPERIMENTAL ANKLE INJURIES

Analysis of the Traumatology of the Ankle Ligaments

OVE RASMUSSEN and CLAUS KROMANN-ANDERSEN

Laboratory of Biomechanics, Orthopaedic Hospital, Århus, Denmark

On 32 osteoligamentous ankle preparations forced movements were performed in varying, accurately defined directions. The sequence in which this caused rupture of the individual ligamentous structures of the ankle is described. Dorsiflexion traumas predominantly injured the posterior part of the deltoid ligament, while in plantar flexion traumas the injuries primarily involved the anterior capsule and the anterior talofibular ligament. Internal rotation traumas injured the anterior talofibular ligament and the short, anterior fibres of the posterior talofibular ligament before the calcaneofibular ligament was damaged, whereas in adduction traumas the calcaneofibular ligament ruptured first. Forced external rotation primarily caused rupture of the deep structures of the deltoid ligament, while conversely abduction traumas first caused rupture of the superficial part of this ligament.

Key words: ankle instability; ankle joint; deltoid ligament; lateral ankle ligaments; ligament injuries

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The ankle joint is a hinge joint whose stability is secured by the surrounding ligamentous structures. These comprise laterally the anterior talofibular ligament (ATaFL), the calcaneofibular ligament (CFL), and the posterior taofibular ligament (PTaFL). The latter consists functionally of two parts: the short anterior and the long posterior fibres (de Vogel 1970, Rasmussen et al. 1983a). Between the distal parts of the tibia and fibula there are the anterior tibiofibular ligament (ATFL), the tibiofibular syndesmosis, and the posterior tibiofibular ligament (PTFL). Medially, the stability is secured by the deltoid ligament which consists of the superficial tibiocalcaneal ligament (TCL) and the deeper anterior, intermediate, and posterior tibiotalar ligaments (ATTL, ITTL, and PTTL) (Rasmussen et al. 1983b).

The function of the individual structures has previously been elucidated by cutting experiments (Rasmussen & Tovborg-Jensen 1981, 1982, Rasmussen & Andersen 1982, Rasmussen et al. 1982, 1983a, 1983b). However, the fact that cutting of a given structure entails increased mobility in a given direction does not mean that this structure is going to rupture first during a forced movement in that direction. A weaker structure will rupture before a stronger one if both are exposed to the same force, and if two structures are equally strong the one exposed to a

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greater force will rupture first. For example, cutting of the PTTL + ITTL enables internal rotation of the talus (Rasmussen et al. 1983b). However, cutting of the ATaFL also causes increased internal rotation (Johnson et al. 1981, Rasmussen & Tovborg-Jensen 1981). As the ATaFL is the weakest of these structures, this ligament must be expected to rupture first in an internal rotation trauma.

The present study was undertaken to elucidate the sequence in which the individual ligamentous structures rupture when the ankle joint is forced in different, given directions.

MATERIAL AND METHOD

For the present investigations we used an apparatus which renders it possible to force the talus in accurately defined directions, to affect the talus by a defined torque, and to record the movements corresponding to this torque (Rasmussen & Andersen 1982, Rasmussen et al. 1983a).

On an osteoligamentous ankle preparation a nail is inserted into the talus. On this nail is mounted a lever, provided with strain gauges for measuring the torque and connected with two potentiometers for measuring the rotation corresponding to the torque. On X–Y recorders the torque as well as the produced movement are followed, and under the guidance of the recorder it is possible to affect the talus by an increasing torque in varying, accurately defined directions.

The study comprised 32 ankle preparations half of which were investigated with the talus nail inserted in the posteroanterior direction so that the findings in the sagittal + horizontal plane could be elucidated. The other half were investigated with the nail inserted from below, up through the calcaneus and into the talus, to measure in the sagittal + frontal planes. The directions in which the talus was turned are shown in Figure 1a and b. In each of the directions shown the effect of forced movement was studied in two preparations except for pure dorsiflexion and pure plantar flexion, which were studied in four preparations each. In each instance the movement was forced in the given direction until visible injury had been inflicted. The instability was then demonstrated by tracing a mobility pattern at a torque of 1.5 Nm (Rasmussen & Andersen 1982). Thereafter, the forced movement was continued until a new injury had been inflicted, and a new pattern was then recorded. We continued this until the ankle joint was completely unstable. A few of the recorded mobility patterns are shown below.

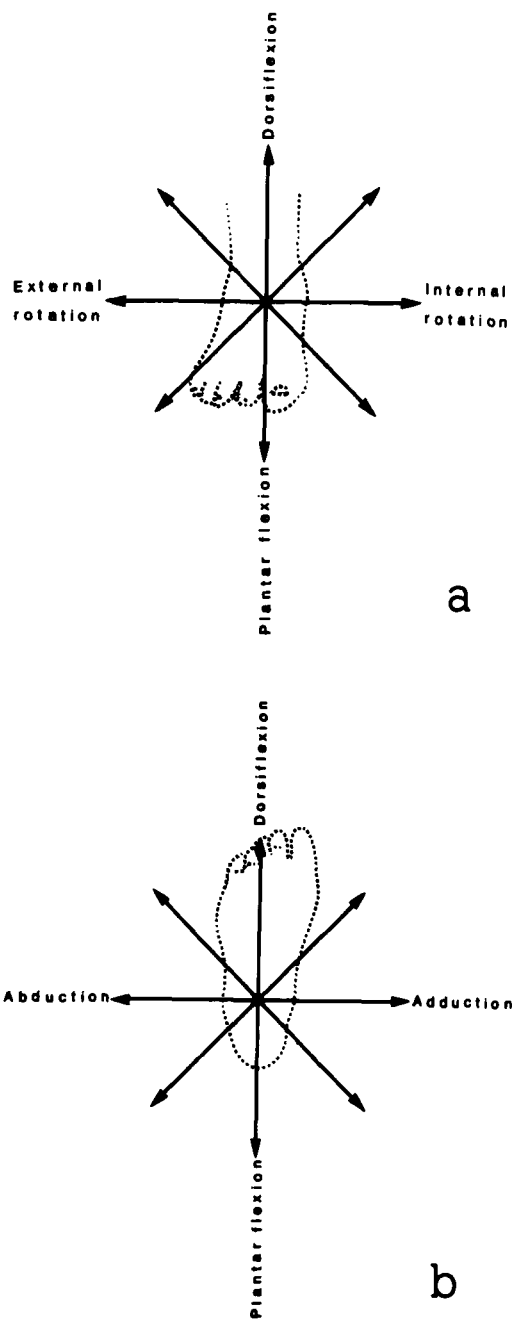

Figure 1. Directions in which forced movements were made: (a) in the sagittal and horizontal planes; (b) in the sagittal and frontal planes.

Table 1. Lesions in dorsiflexion traumas

	Case 1	Case 2	Case 3	Case 4
Stage 1	Avulsion fracture, PTTL	Rupture of PTTL	Avulsion fracture, PTTL	Partial rupture of PTTL and TCL
2	Avulsion fracture, TCL + partial rupture of CFL	Rupture of ITTL + short fibres of PTaFL	Avulsion fracture, 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, PTaFL and CFL

Table 2. Lesions in plantar flexion traumas

	Case 1	Case 2	Case 3	Case 4
Stage 1	Rupture of ATaFL + anterolateral capsule	Rupture of anterior capsule	Rupture of anterior capsule	Rupture of anterior capsule and partially of ATaFL and ATTL
2	Rupture of 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 and 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

RESULTS

Forced dorsiflexion was done in four cases. The sequence of the injuries is shown in Table 1. In all cases the primary injury affected the medial ligamentous apparatus.

Forced dorsiflexion-internal rotation in one case caused firstly rupture of the ATaFL and the short fibres of the PTaFL. Thereafter, the PTTL ruptured, and at the same time a partial rupture of the CFL occurred. Lastly, the remainder of the lateral ligamentous apparatus ruptured. In the other case the short fibres of the PTaFL ruptured first, then the PTTL, the lateral capsule and – partially – the CFL. Lastly, the CFL underwent total rupture together with the ITTL, the TCL remaining intact.

Forced internal rotation in both cases caused rupture firstly of the ATaFL and thereafter of the short fibres of the PTaFL. Thereupon, the CFL as well as the remainder of the PTaFL ruptured.

Forced plantar flexion-internal rotation in one case resulted primarily in rupture of the anterior capsule + the ATaFL. Then the short fibres of the PTaFL ruptured and finally the CFL partially. In the other case the ATaFL + the short fibres of the PTaFL ruptured primarily, thereupon the anterior capsule and at last the ATTL.

Forced plantar flexion was performed on four preparations. It caused the injuries shown in Ta-

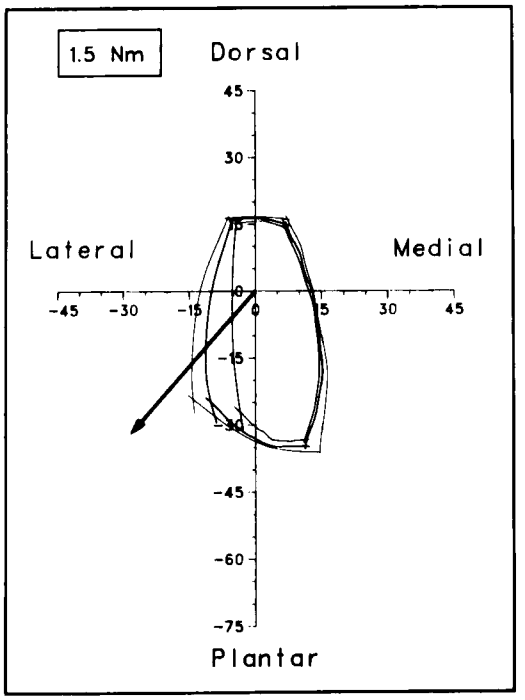

Figure 2. Mobility patterns after forced plantar flexion-external rotation, partly at intact ligaments, after rupture of the ITTL + PTTL, and after rupture of the entire deep part of the deltoid ligament. The arrow indicates the direction of the forced movement.

ble 2, viz. first injuries mainly of the lateral ligamentous apparatus.

Forced plantar flexion-external rotation in one case entailed firstly rupture of the ITTL + PTTTL, then an avulsion fracture from the medial malleolus at the attachment of the ATTL, and finally another avulsion fracture from the medial malleolus at the attachment of the TCL (Figure 2). In the other case the entire deep part of the deltoid ligament ruptured, and an irreversible stretching of the TCL occurred. Moreover, an avulsion fracture of the anterior edge of the lateral malleolus was produced. Then a further fracture of the lateral malleolus occurred at the attachment of the CFL and PTaFL, and the TCL underwent total rupture.

Forced external rotation on ankle joints in a neutral position produced in one case simultaneous avulsion from the lateral malleolus at the attachment of the CFL and PTaFL and rupture of the entire deep part of the deltoid ligament. In the other case there was primarily an avulsion fracture in the medial malleolus at the ATTL + ITTL + TCL. Then the remainder of the medial malleolus fractured and the CFL as well as PTaFL ruptured.

Forced dorsiflexion-external rotation in one case induced firstly rupture of the PTTL, then an avulsion fracture distally in the medial malleolus, and lastly rupture of the PTaFL. In the other case there was first fracture in the lateral malleolus at the attachment of the CFL and PTaFL, then fracture of the medial malleolus and rupture of the ATaFL.

Forced dorsiflexion-adduction in one case primarily caused rupture of the CFL, then of the PTaFL, and lastly rupture also of the ATaFL. In the other case the primary event was rupture of the ATaFL + partially of the CFL, then total rupture of the CFL and PTaFL.

Forced adduction on neutrally positioned ankle

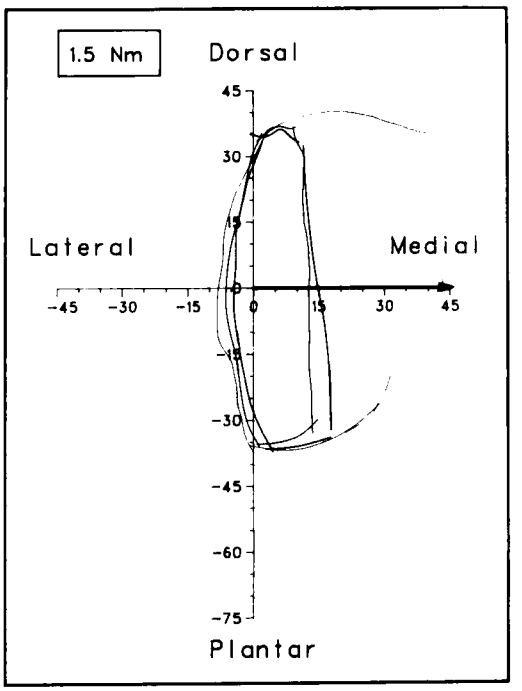

Figure 3. Mobility patterns after forced adduction, partly at intact ligaments, after avulsion of the CFL, and after rupture of the ATaFL, CFL, as well as the short fibres of the PTaFL.

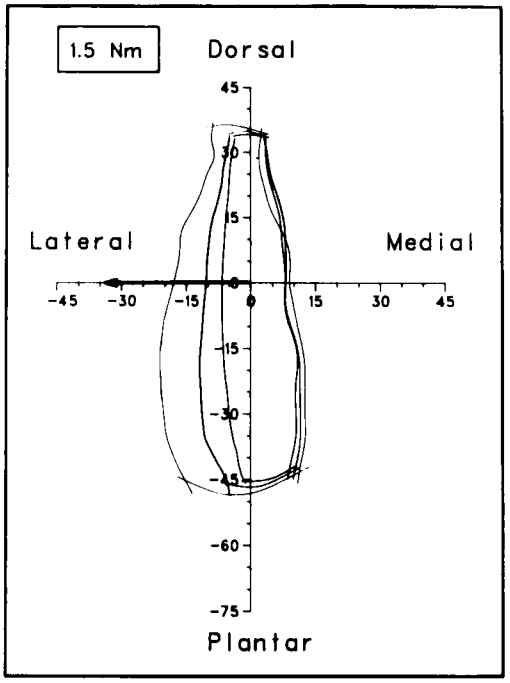

Figure 4. Mobility patterns after forced abduction, partly at intact ligaments, after rupture of the TCL, and after rupture also of the ITTL.

joints produced in one case simultaneous rupture of the ATaFL, CFL, and PTaFL. In the other case it caused first an avulsion fracture in the lateral malleolus at the attachment of the CFL. Thereafter, the malleolus showed a further fracture, now at the site of the ATaFL, CFL, and the short fibres of the PTaFL, and lastly rupture of the long fibres of the PTaFL (Figure 3).

Forced plantar flexion-adduction in one case caused firstly rupture of the ATaFL and, partially, of the CFL. Then the anterior capsule ruptured while further stretching of the CFL occurred, and finally the CFL ruptured completely together with the PTaFL. In the other case there was first partial rupture of the ATaFL and CFL. Then the ATaFL and the anterior capsule ruptured completely while the CFL lengthened further, and at last the CFL ruptured completely together with the PTaFL.

Forced plantar flexion-abduction in one case produced rupture firstly of the TCL, then of the ITTL, and finally of the entire deltoid ligament. In the other case the ATTL ruptured first, then the anterior capsule + TCL + ITTL, and at last the remainder of the deltoid ligament.

Forced abduction in one case caused firstly rupture of the TCL and partial rupture of the ITTL. Then the ATTL + PTTL + the remainder of the ITTL ruptured. In the other case (Figure 4) the TCL ruptured first, then the ITTL, and lastly the ATTL + PTTL.

Forced dorsiflexion – abduction in one case simultaneously produced total rupture of all structures of the deltoid ligament. In the other case there was first an avulsion fracture from the medial malleolus at the TCL, ITTL, and PTTL. Then the entire medial malleolus was avulsed, and finally the anteromedial capsule ruptured.

DISCUSSION

Robichon et al. (1972) reported that the CFL and PTaFL grow tense on dorsiflexion, while de Vogel (1970) could experimentally induce isolated rupture of the most plantar fibres of the ATaFL by forced dorsiflexion. Our investigations indicate that the restriction of dorsiflexion takes place primarily in the medial ligamentous apparatus, as in all cases we observed rupture of the PTTL first.

de Vogel (1970), Robichon et al. (1972), as well as Wirth et al. (1978) found tension or rupture of the ATaFL on forced plantar flexion, and this is largely confirmed by our findings. A probable explanation is that owing to the varying axis of the dorso-plantar movement the plantarflexion is accompanied by some compensatory internal rotation of the talus (Barnett & Napier 1952), making the ATaFL tense.

Experimental injuries following forced internal rotation on a dorsiflexed or plantar flexed ankle joint are not on record, but Hönigschmied (1877) induced rupture, primarily of the ATaFL, by internal rotation of neutrally positioned ankle joints.

Regardless of the degree of dorsiflexion or plantar flexion, internal rotation appears to be restricted by the ATaFL, and thereafter by the short fibres of the PTaFL. Thus, the lateral ligaments do not always, as reported by Broström

(1964), rupture in the antero-posterior direction as under these conditions the CFL does not rupture until after the short fibres of the PTaFL.

According to Lauge-Hansen (1942) forced external rotation entailed rupture of the tibiofibular ligaments, primarily the ATFL. Irrespective of the degree of dorsal or plantar flexion we could not induce such an injury. In two cases we observed primarily a rupture of the CFL and PTaFL, but in the other four cases rupture of the deep structures of the deltoid ligament. This suggests that the tibiofibular structures burst only during simultaneous weight-bearing. The superficial part of the deltoid ligament ruptured very late in the course of forced external rotation, and accordingly an operative finding of an intact superficial structure is no guarantee that the deep part is intact, too.

The injuries arising on forced adduction seem to depend upon the degree of simultaneous dorsal or plantar flexion, the ATaFL rupturing primarily when the ankle joint is in plantar flexion. Indeed, this has been reported also by Nevin & Post (1964) and by Dias (1979). In the neutral position or in dorsiflexion the first injury often affects the CFL, which thus may show the only rupture in adduction traumas.

Forced abduction on neutrally positioned ankle joints was reported by Lauge-Hansen (1942) to cause primarily rupture of the deltoid ligament or fracture of the medial malleolus and thereafter rupture of the distal tibiofibular structures. We found primarily rupture of the TCL and thereafter of the ITTL. Apparently, then, an abduction trauma may entail isolated rupture of the superficial part of the deltoid ligament; moreover, the central fibres of its deep part may rupture without simultaneous damage to the anterior or posterior part of this ligament complex. At an increasing degree of simultaneous dorsal or plantar flexion, however, the PTTL and ATTL respectively will be involved in the injury.

CONCLUSION

In dorsiflexion traumas the injury affects mainly the medial collateral ligaments, and in plantar flexion traumas the lateral collateral ligaments.

In internal rotation traumas rupture of the ATaFL and of the short fibres of the PTaFL may occur even though the CFL remains intact.

In adduction traumas the CFL may rupture first, possibly together with the short fibres of the PTaFL, while the ATaFL may remain intact.

In external rotation traumas the deep part of the deltoid ligament may rupture before its superficial part.

In abduction traumas the superficial part of the deltoid ligament may rupture while its deep part remains uninjured.

REFERENCES

Barnett, C. H. & Napier, J. R. (1952) The axis of rotation at the ankle joint in man. Its influence upon the form of the talus and the mobility of the fibula. *J. Anat.* **86**, 1–9.

Broström, L. (1964) Sprained ankles. I. Anatomic lesions in recent sprains. *Acta Chir. Scand.* **128**, 483–495.

Dias, L. S. (1979) The lateral ankle sprain. An experimental study. *J. Trauma* **19**, 266–269.

Hönigschmied, J. (1877) Leichenexperimente über Zerreissungen der Bänder im Sprunggelenk mit Rücksicht auf die Entstehung der indirechten Knöchelfrakturen. *Deutsch. Zschr. Chir.* **8**, 239–260.

Johnson, E. E., Markolf, K. L., Sakai, D. N. & Dunn, J. P. (1981) Contribution of the anterior talofibular ligament to ankle stability. *Orthop. Trans. J. Bone Joint Surg.* **5**, 214–215.

Lauge-Hansen, N. (1942) Ankelbrud. I. Genetisk diagnose og reposition. Thesis, Munksgaard, Copenhagen.

Nevin, J. E. & Post, R. H. (1964) Lateral ligament ankle sprain. A clinical-pathological correlation based on the production and examination of controlled sprains in preamputated extremities. *J. Trauma* **4**, 292–300.

Rasmussen, O. & Andersen, K. (1982) Ligament function and joint stability elucidated by a new technique. *Engineering Med.* **11**, 77–81.

Rasmussen, O. & Tovborg-Jensen, I. (1981) Anterolateral rotational instability of the ankle joint. An experimental study of anterolateral rotational instability, talar tilt, and anterior drawer sign in relation to the lateral ligaments. *Acta Orthop. Scand.* **52**, 99–102.

Rasmussen, O. & Tovborg-Jensen, I. (1982) 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. *Acta Orthop. Scand.* **53**, 155–160.

Rasmussen, O., Tovborg-Jensen, I. & Boe, S. (1982) Distal tibiofibular ligaments. Analysis of function. *Acta Orthop. Scand.* **53**, 681–686.

Rasmussen, O., Tovborg-Jensen, I. & Hedeboe, J. (1983a) The posterior talofibular ligament. Analysis of its function. *Intern. Orthop.*, in press.

Rasmussen, O., Kromann-Andersen, C. & Boe, S. (1983b) Deltoid ligament. Functional analysis of the medial collateral ligamentous apparatus of the ankle joint. *Acta Orthop. Scand.*, in press.

Robichon, J., Pegington, J., Moonje, V. B., & Desjardins, J. P. (1972) Functional anatomy of the ankle joint and its relationship to ankle injuries. *Canad. J. Surg.* **15**, 145–150.

Vogel, P. L. de (1970) Enige functioneel-anatomische aspecten van het bovenste spronggewricht. Thesis, Leiden.

Wirth, C. J., Küsswetter, W. & Jäger, M. (1978) Biomechanik und Pathomechanik des oberen Sprunggelenkes. *H. Unfallheilkd.* **131**, 10–22.

Correspondence to: Ove Rasmussen, Korshøjen 103, 8240 Risskov, Denmark.