A cavovarus foot deformity was simulated in cadaver specimens by inserting metallic wedges of 15° and 30° dorsally into the first tarsometatarsal joint. Sensors in the ankle joint recorded static tibiotalar pressure distribution at physiological load.

The peak pressure increased significantly from neutral alignment to the 30° cavus deformity, and the centre of force migrated medially. The anterior migration of the centre of force was significant for both the 15° (repeated measures analysis of variance (ANOVA), \( p = 0.021 \)) and the 30° (repeated measures ANOVA, \( p = 0.007 \)) cavus deformity. Differences in ligament laxity did not influence the peak pressure.

These findings support the hypothesis that the cavovarus foot deformity causes an increase in anteromedial ankle joint pressure leading to anteromedial arthrosis in the long term, even in the absence of lateral hindfoot instability.

Cavovarus is a complex deformity thought to occur from muscular imbalance of idiopathic or neurological origin. Further causes are residual clubfoot, tarsal coalition and the sequelae of trauma and inflammatory disease. There may be a genetic pre-disposition or an undetected associated neurological disorder. The pathology of the deformity consists of a plantarflexed medial forefoot which accentuates or contributes to hindfoot varus and dorsiflexion of the talus. Consequently, the biomechanics of the ankle are affected and may lead to lateral hindfoot instability.

Biomechanical studies of the ankle joint indicate a shift of pressure in certain foot deformities and in distal tibial malalignment without ligamentous instability. It is suggested that longstanding ankle incongruence of the cavovarus deformity alone increases the contact stresses in the ankle joint enough to lead to anteromedial ankle arthrosis. However, no clinical or scientific evidence is available to explain this.

There is an increased prevalence of cavovarus in patients with chronic lateral instability of the ankle. The association between this instability, idiopathic cavovarus deformity and medial ankle arthrosis has been described by Fortin et al and Valderrabano et al. However, we have observed ambulatory patients with neurogenic pes cavovarus and patients with idiopathic pes cavovarus who presented with ankle arthrosis but without lateral instability.

The purpose of this study was to investigate the biomechanical characteristics of the ankle joint in cavovarus deformity. We postulated that cavovarus causes changes of the pressure distribution in the ankle. An increase of joint pressure, anteromedial shift of the centre of force, and a consequent reduction of the contact area with concentration of the load was expected. These findings would support the hypothesis that a longstanding cavovarus deformity, even without chronic lateral ankle instability, may result in anteromedial ankle arthrosis.

Materials and Methods
Ten fresh-frozen cadaver lower legs (five left, approximately two thirds of the original length) without deformities, prior trauma or arthrosis were used. Bony malalignment was excluded radiologically. A normal range of movement in the ankle joint was verified, the skin and subcutaneous tissue were removed, and ligaments and capsules preserved. In each specimen, the three medial tarsometatarsal joints were opened dorsally, enabling plantar flexion of the medial forefoot. The talonavicular capsule was incised dorsally to allow the hindfoot to spontaneously assume a varus position. A dorsal opening of the first tarsometatarsal joint of 15°, and afterwards 30° was obtained by inserting custom-made aluminium wedges (Fig. 1). The resulting deformity was stabilised by fixing a 3.5 mm
one-third tubular plate dorsally to the first cuneiform and first metatarsal. For each deformity, an individual arch support was moulded from polymethylmethacrylate (PMMA; Beracryl, W. Troller Kunststoffe AG, Jengenstorf, Switzerland) to prevent collapse under axial load. While moulding the medial arch into the cavus position, a spontaneous lateral deviation of the tibia was observed in every specimen. A lateral deviation of approximately 5° for the 15° cavus and 10° for the 30° cavus occurred regularly. This did not happen when all the lateral ankle and subtalar ligaments were released. This was performed in one specimen (not included in the series), which was subsequently tested for changes in ankle joint pressure in the cavus deformity. No changes of pressure were recorded in the different positions. These ligaments were therefore left intact in the test specimens, as this phenomenon may represent a possible mechanism of anteromedial joint overload in pes cavovarus.

Every specimen was fixed to its individual PMMA form with screws through the os calcis and through the first and fifth metatarsal heads. Digital anteroposterior (AP) and mediolateral radiographs documented the deformity. The talo-first metatarsal angle was measured using digital tools on a 3- to 4-fold magnification of the radiographs.

The proximal 30 mm of the tibia and fibula were fixed in a MTS Bionix-858 testing apparatus (MTS-Systems Inc.) by seating the PMMA-form of the foot horizontally on the machine’s load cell. Load was introduced via a metal sphere 25 mm in diameter. In order to simulate physiological conditions, 90% of the load was apportioned to the tibia and 10% to the fibular.

Vertical alignment of the tibial axis was adjusted visually between 70% and 90%. Complete cover of the anteromedial region of interest was always assured.

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Simulation of cavovarus deformity produced by a dorsal opening of the first tarsometatarsal joint and insertion of custom-made aluminium wedges.

Fig. 1

In order to assess an eventual influence of differences in lateral ankle ligament laxity on the joint pressure, 2.5 mm Steinman pins were inserted through the capsule into the lateral ankle joint space directly anterior to the fibula and into the sinus tarsi at neutral alignment. Their position was checked radiologically and found to be consistent in every specimen and stable during measurement. The pins were mounted in the MTS machine, and the displacement recorded statically at 50 N and 80 N distraction force (Fig. 2b).

Pilot testing revealed that the sensors gradually degraded. Only four measurements plus calibration proved reliable with one sensor because of the high pressures (approximately body-weight).

The peak pressure $P_{\text{max}}$ defined as pressure at the highest loaded area $(2 \times 2$ sensels), the force $F$ transferred through the sensor and the total loaded sensor area $A$ (sensel force $< 0$) were calculated from the pressure measurements. Also, the location of the centre of force was determined.
nates were laid out in a mediolateral and AP direction. All migrations of the centre of force were computed in relation to its initial centre of force (0° measurement). For all parameters, the mean of the last ten recorded frames at 700 N static load was computed. Data evaluation used the Software package I-Scan (TekScan Inc.). Subsequent data processing used a custom-made Matlab routine (Mathworks Inc., Natick, Massachusetts).

Statistical analysis. Repeated measures analysis of variance (ANOVA) was performed for the talo-metatarsal angle and for all other parameters to assess differences between groups. The measurement series was considered as the fixed factor. A Bonferroni post hoc test was used for pairwise comparisons. A Spearman rank test was performed to identify potential correlations between talo-metatarsal angle and measured parameters as well as between ligament laxity and joint pressure. The level of statistical significance was p = 0.05. The software package SPSS (SPSS Inc., Chicago, Illinois) was used for all statistical evaluations.

Results
The lateral talo-first metatarsal angle obtained from mediolateral radiographs was a mean of 12.6° (7° to 26°) for the 0° group, 24.4° (19° to 32°) for the 15° and 33.3° (25° to 40°) for the 30° group (Fig. 3). All groups differed significantly from each other (all, repeated measures ANOVA, p < 0.001).

No significant differences between the two measurement series were found for any of the parameters (all, repeated measures ANOVA, p > 0.05).

The mean Pmax was 3.05 MPa (1.55 to 4.22) for the 0° group, 3.90 MPa (2.3 to 7.33) for the 15° group and 4.88 MPa (2.93 to 7.6) for the 30° group. The control measurement on return to 0° was a mean of 2.62 MPa (1.39 to 3.88) (Fig. 4). In calculating the pressure increases, we found 0.85 MPa (-0.85 to 3.79) (0° vs 15° group) and 1.83 MPa (0.5 to 4.06) (0° vs 30° group). The difference between the last and the first measurement at 0° was a mean of -0.43 MPa (-1.05 to 0.78) (repeated measures ANOVA, p = 0.171).

The mean Pmax was significantly different between the 0° and the 30° group (repeated measures ANOVA, p = 0.009). The 15° group was not different to the 0° group (repeated measures ANOVA, p = 0.51); a trend towards a difference was observed between 15° and 30° groups (repeated measures ANOVA, p = 0.098).

The centre of force migrated at a mean of 2.53 mm (0.29 to 11.03) medially for the 15° measurement with respect to the initial position at 0° but this displacement was not sig-
significant (repeated measures ANOVA, \( p = 0.21 \)). A signifi-
cant migration was found for the 30° group compared with
the 0° group with a mean migration of 4.39 mm (0.11 to
11.98; repeated measures ANOVA, \( p = 0.019 \)). The 30° and
the 15° group were not significantly different (repeated
measures ANOVA, \( p = 0.104 \)). In the mean measurement
on return to 0° group, migration was 0.17 mm (-0.43 to
0.64) which was not significantly different from the initial
position at 0° (repeated measures ANOVA, \( p = 0.875 \))
(Fig. 5).

The centre of force migration in the AP direction was
a mean of 1.50 mm (0.53 to 4.31) anteriorly for the
15° group (repeated measures ANOVA, \( p = 0.021 \)) 2.26 mm
(-0.6 to 5) for the 30° group (repeated measures ANOVA,
\( p = 0.007 \)) compared with the basic value of the 0° group.
The 15° and 30° groups were not significantly different
\( p = 0.314 \)). The mean control measurement migration on
return to 0° was 0.32 mm (-0.19 to 0.89) compared with
the initial measurement at 0° (repeated measures ANOVA, \( p = 0.066 \)) (Fig. 6).

The mean transferred force decreased continuously from
465 N (338 to 535) for the 0° group to 352 N (257 to 453) for
the return to 0° control measurement at the end of the test.
Compared with the initial measurement in the neutral
position, the final measurements of this position displayed
a significant loss of force (repeated measures, \( p = 0.001 \);
Fig. 6).

The loaded sensor area was greatest at 0° (with a mean of
745 mm\(^2\) (655 to 826)) and smallest at 30° (with a mean of
627 mm\(^2\) (217 to 832)), but no significant differences
between the groups were observed (all repeated measures
ANOVA, \( p > 0.05 \); Fig. 7).
Despite the thickness of only 0.1 mm, this may lead to slightly lower results for pressure and force. A further problem of intra-articular sensors is their thickness and stiffness. Fuji film inserts (Sensor Products, East Hanover, New Jersey) which are similar to the Tekscan sensors (Tekscan Inc.) were found to have falsely elevated stresses by 10% to 26% because of the stiffness and thickness of the sensors. Despite the thickness of only 0.1 mm, this may account for a slight increase of the results for pressure and force.

For purposes of precision and simplicity, only the lateral talo-metatarsal angle, that is, the extent of the plantar flexion of the medial forefoot, was actively altered to simulate the deformity. The other components of the cavovarus deformity were not changed actively but left to follow the forefoot deformity. This angle was chosen after screening the radiographs of 43 patients with 59 cavovarus feet for ankle arthrosis. Only the extent of the lateral talo-metatarsal angle was found to have a moderately strong correlation \((r = 0.65)\) to the severity of the ankle arthrosis. The other radiological angles defining the cavovarus deformity (calcaneal pitch, AP talo-metatarsal angle and AP talo-calcaneal overlap) showed only minor correlation with the severity of the ankle arthrosis.

Possible mechanisms for the increase in medial ankle joint pressure and subsequent degeneration include the medialisation of the weight-bearing axis because of the medialisation of the ground contact point of the varus heel.

Discussion

Our results demonstrated a trend towards an increase of anteromedial pressure and medial shift of the centre of force in the ankle joint for the lesser deformities and a significant increase and shift for the greater deformities. The anterior shift was significant for both deformities. The relationship between the extent of the deformity and increase of pressure was linear. These results support the hypothesis of an increase and shift of pressure in the anteromedial ankle joint with increasing cavovarus deformity. However, the contact area was not significantly reduced (Fig. 7), probably because the lateral ligaments had been left intact.

Pilot testing revealed a significant loss of the recorded force \((p = 0.001, \text{Fig. } 6)\) indicating a degradation of load recovery of the sensors which has been described by McKinley et al\(^17\) and Brown, Rudert and Grosland.\(^18\) This may lead to slightly lower results for pressure and force. A further problem of intra-articular sensors is their thickness and stiffness. Fuji film inserts (Sensor Products, East Hanover, New Jersey) which are similar to the Tekscan sensors (Tekscan Inc.) were found to have falsely elevated stresses by 10% to 26% because of the stiffness and thickness of the sensors.\(^19\) Despite the thickness of only 0.1 mm, this may account for a slight increase of the results for pressure and force.

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Possible mechanisms for the increase in medial ankle joint pressure and subsequent degeneration include the medialisation of the weight-bearing axis because of the medialisation of the ground contact point of the varus heel.

We think this mechanism is not relevant since we found unchanged joint pressures in cavus deformity after lateral ligament release. Another possible mechanism is a medial hyperpressure by forefoot-driven supination: the plantarflected medial forefoot causes a valgus-pronation posture of the forefoot, which drives the hindfoot into varus during the stance phase of the gait cycle.\(^20\) This supination force on the whole foot will compress the medial side of the hindfoot, since the lateral ankle and subtalar ligaments keep these joints from opening up laterally. The lateral deviation of the tibia in the test set-up makes us believe that this mechanism is the most probable explanation for the pressure increases medially. Lastly, the dorsiflexed talus of the cavus foot could cause or accentuate an anteromedial ankle impingement. A shearing injury of the cartilage could occur by a cam-mechanism of the anteromedial shoulder of the talar dome against the anterior tibial lip, similar to that described for the hip joint.\(^21\) However, this would imply a dynamic phenomenon, which we have not investigated.

The association between lateral ankle instability, cavovarus deformity and ankle arthrosis has been discussed in the literature.\(^3,13,22,24\) In a review of 30 patients with end-stage ankle arthrosis, Valderrabano et al\(^11\) found that ankle sprain or chronic lateral ankle instability were correlated to a varus-malaligned ankle joint in two thirds of the patients. Fortin et al\(^15\) showed ten patients with 13 idiopathic cavovarus feet, chronic lateral ankle instability, and varus tilt of the talus and ankle arthritis ranging from mild to severe. They stated that these findings may represent the natural history of longstanding, untreated ankle instability and cavovarus foot deformity. In these patients, medial ankle arthrosis may be caused by repetitive compressive and shearing injury of the cartilage during the inversion sprains, or by the medial shift of pressure when the chronically-incompetent lateral ligaments allow lateral gaping of the joint.\(^25\)
However, there are no reports about the association of pes cavovarus and anteromedial ankle arthrosis without lateral ankle instability. Our results support the hypothesis that a longstanding cavovarus foot deformity without chronic lateral ligamentous ankle instability may result in anteromedial ankle arthrosis. We believe that the cavovarus deformity itself and not predominantly the lateral ligamentous ankle instability has a major influence on the development of the arthrosis.

The literature on ankle pressure characteristics in distal tibial and foot malalignment is scanty. In a cadaver flatfoot model established by ligament release, Friedman et al found a posterolateral shift of pressure and a decrease of the joint contact area as possibly being responsible for long-term degenerative changes. Tarr et al showed a reduction of the contact area in the ankle joint of six cadaver specimens with varus deformities of the distal tibia. The mean decrease in contact area was 2.3% for a 5° varus deformity, 4.2% for a 10° deformity and 6.5% for a 15° deformity. No pressures were recorded.

The strengths of our study are the direct intra-articular measurement of the biomechanical effects of the cavovarus deformity on the ankle joint using a high-resolution device. The limitations are the lack of dynamic measurements and the lack of representation of the contributing muscle forces across the ankle joint. Also, there are concerns about the validity of the model used, since an instantly simulated cavovarus deformity in a previously normal foot may not lead to the same biomechanical abnormalities as a naturally occurring cavovarus foot. However, ankle joint congruity was maintained, as judged by the absence of any statistically significant changes in the mean contact area between 0° and 30°, and a possible influence of differences in ligament tightness was ruled out, since ligamentous laxity measured for every specimen neither had an influence on the increase of pressure nor on the anteromedial shift of the centre of force. We therefore believe that the simulated deformity was as close to a naturally occurring cavovarus foot as possible.

In conclusion, a cavovarus foot deformity leads to a significant increase of pressure and an anteromedial shift of the centre of force in the ankle joint. This is believed to be a result of the forefoot-driven supinating force on the hindfoot, thereby increasing anteromedial joint pressure in the presence of competent lateral ankle and subtalar ligaments. A longstanding cavovarus foot deformity alone may therefore be an aetiological factor for anteromedial ankle arthrosis. Therefore, patients with cavovarus feet should be alerted to the possible consequences of this deformity on the ankle joint. In patients with symptomatic anteromedial ankle arthrosis surgical correction of the cavovarus would be expected to unload the degenerate area and relieve symptoms.