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CALCANEAL FRACTURES CAUSE A LATERAL LOAD SHIFT IN CHOPART JOINT CONTACT STRESS AND PLANTAR PRESSURE PATTERN *IN VITRO*

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Abstract—In order to evaluate the effects of the anatomical changes after calcaneal fractures on joint loading characteristics we investigated the effects of simulated calcaneal fractures on intra-articular loading in the Chopart joint and on plantar pressure patterns *in vitro*. Five fresh-frozen lower leg specimens were axially loaded with 500 N in three positions: neutral, 10° plantarflexion and 10° dorsiflexion. The loading characteristics were determined before and after creating a tongue-type fracture by osteotomy. Plantar loading patterns were measured with a capacitive pressure distribution platform. Intra-articular contact areas and pressures were recorded on pressure sensitive film introduced into the talonavicular and calcaneocuboid joint. Increased loading in the calcaneocuboid joint (+ 25%, $p = 0.005$) and decreased loading in the talonavicular joint (− 16%, $p = 0.01$) corresponded to increased loading of the lateral aspects of the plantar surface. These findings were independent of the foot position. The results suggest that the effects of increased lateral foot loading which have been observed in calcaneal fracture patients (Rosenbaum *et al.*, *Clin. Biomech.* **10**, 345–351, 1995) are caused by changes in joint kinematics and do not appear to result from a protective gait pattern established after the injury. Copyright © 1996 Elsevier Science Ltd.

Keywords: Calcaneal fractures; Chopart joint; Joint contact stress; Plantar pressure distribution; Fuji Prescale film.

INTRODUCTION

The importance of the anatomic reconstruction of the hind foot following intra-articular calcaneal fractures has been increasingly emphasized in recent years. In order to attain satisfactory clinical results the restoration of the anatomy to a pre-traumatic state has been suggested by many authors (Colburn *et al.*, 1989; Hansen, 1991; Mutschler *et al.*, 1993; Myerson and Quill, 1993; Paley and Hall, 1993; Zwipp *et al.*, 1993). Intra-articular fractures of the calcaneus usually cause a depression of the subtalar joint and therefore lead to a reduction of Boehler's angle in combination with a shortening and widening of the calcaneus. The altered joint geometry is clinically manifested in a reduced range of subtalar motion, as seen in eversion and inversion measurements (Mittlmeier *et al.*, 1992). Furthermore, it has been suggested that altered joint loading can contribute to the development of arthrosis (Ross and Sowerby, 1985). Pathologically high local pressures have been reported as a secondary deficit in the ankle joint following calcaneal fractures (Carr *et al.*, 1988) and may cause degenerative changes in the joint cartilage (Paar *et al.*, 1991; Stockenhuber *et al.*, 1991). Arthrosis in the calcaneocuboid joint is often observed since this joint is involved in up to 50% of all calcaneal fractures (Nagar, 1995; Zwipp *et al.*, 1989).

Our investigations on plantar pressure measurements after calcaneal fractures revealed changes in the gait patterns of patients even after average healing periods of four years (Rosenbaum *et al.*, 1995). The patients produced significantly increased pressures under the mid-foot and the fifth metatarsal head and a decrease under the first metatarsal head indicating an overall load shift towards lateral aspects of the plantar surface. Similarly, a lateralization of the foot loading pattern has been described by Mittlmeier and coworkers (Mittlmeier *et al.*, 1992). The authors reported a deflection of the center-of-pressure line to the lateral side of the foot and attributed this finding to an impairment of the dynamic eversion ability after calcaneal injury.

However, it has yet to be determined whether the observed changes in foot loading patterns after calcaneal fractures are adaptational changes or are caused by damage to the hind foot joints. In the first case, it is conceivable that the patients have altered their gait pattern more or less consciously in order to avoid painful joint loading in the early healing period and have retained it even after complete fracture healing. In the second case the transfer of the force from the talus through the calcaneus to the hind foot and through the Chopart joint line to the forefoot may have been affected by post-traumatic changes in the shape of the calcaneus or by cartilage damage at the time of injury.

The present study aimed to answer the following two questions: (1) Can malunions of calcaneal fractures alone cause the abnormal plantar pressure patterns seen in post-traumatic patients? (2) Can malunions elevate contact stress in the talonavicular and calcaneocuboid joint? Answers to these questions should help to decide whether differences between pre- and post-traumatic plantar pressure patterns under *in vitro* conditions are comparable to

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