

BIOMECHANICS ANALYSIS OF ANKLE SUPINATION SPRAIN INJURY

Daniel Tik-Pui Fong^{1,2}, Youlian Hong³, and Kai-Ming Chan^{1,2}

¹Department of Orthopaedics and Traumatology; ²The Hong Kong Jockey Club Sports Medicine and Health Sciences Centre; ³Department of Sports Science and Physical Education; The Chinese University of Hong Kong, China.

One male athlete accidentally sprained his ankle with a supination mechanism while performing a series of cutting motion trials in a laboratory. The injury was immediately diagnosed as a grade one mild anterior talofibular ligamentous sprain, and was simultaneously recorded by three calibrated cameras and a pressure insole system at 100 Hz. For the injury trial, results showed that the foot and ankle was more internally rotated and greatly inverted at foot strike, followed by a fast plantarflexion and shift of center of pressure to forefoot. The rearfoot was lifted and swung to the lateral aspect, thus increasing the moment arm and the ankle joint torque. The ankle joint finally reached an orientation of 66 degrees dorsiflexion, 48 degrees internal rotation and 103 degrees inversion.

KEY WORDS: sports medicine, inversion sprain, anterior talofibular ligamentous sprain

INTRODUCTION:

The most direct way to study the injury mechanism is to investigate the real injury. However, mimicking injury in laboratory is practically very difficult or impossible. Moreover, recruiting participants for such study is also unethical. In sports, ankle sprain injury is the most common single type of injury (Fong et al., 2007). In studying its mechanism, researchers studied the foot and ankle kinematics during simulated sub-injury or close-to-injury situations, i.e., sudden simulated ankle spraining motion on inversion platforms. In some cases, sports injuries had occurred in video-taped competitions, which provided limited but valuable information for qualitative injury biomechanics analysis (Andersen et al., 2004). However, quantitative biomechanics analysis of sport injury is not easy as it required calibrated multi-view video sequences. This study presented an accidental supination ankle sprain injury occurred in a laboratory under a high-speed video and plantar pressure capturing setting.

METHOD:

Data Collection: One male athlete (age = 23 years, height = 1.75m, body mass = 62.6kg) wore a pair of high-top basketball shoes and performed a series of cutting motion trials in a laboratory. In each trial, the subject ran forward for six meters with his maximum speed, stepped in the capture area with his right foot, and changed his moving directions to the left in the shortest time he could. In the fourth trial, the athlete accidentally sprained his right ankle within the capture volume with a supination mechanism. The injury was immediately diagnosed as a grade one mild anterior talofibular ligamentous (ATFL) sprain by a well-trained orthopaedic biomechanist. The injury motion was simultaneously videotaped by three calibrated cameras at 100 Hz with a shutter speed of 1/250s and an effective capture volume of about 1m³. The plantar pressure and the excursion path of the center of pressure were simultaneously recorded at 100 Hz by a pressure insole system (Novel Pedar, Germany).

Data Analysis: Part of the video sequence from the three cameras is shown in Figure 1 (in every 0.04s). The positions of the fifth metatarsal head, lateral malleolus, heel, lateral femoral epicondyle and tibial tubercle were manually digitized ten times with a motion analysis system (Ariel Performance Analysis System, USA). Calculation of the foot and ankle kinematics was done with a standard method to present the lower extremity biomechanics (Vaughan et al., 1992). A standing trial served as the offset position. The same procedure was performed for the three successful normal trials before the injury trial for comparison.

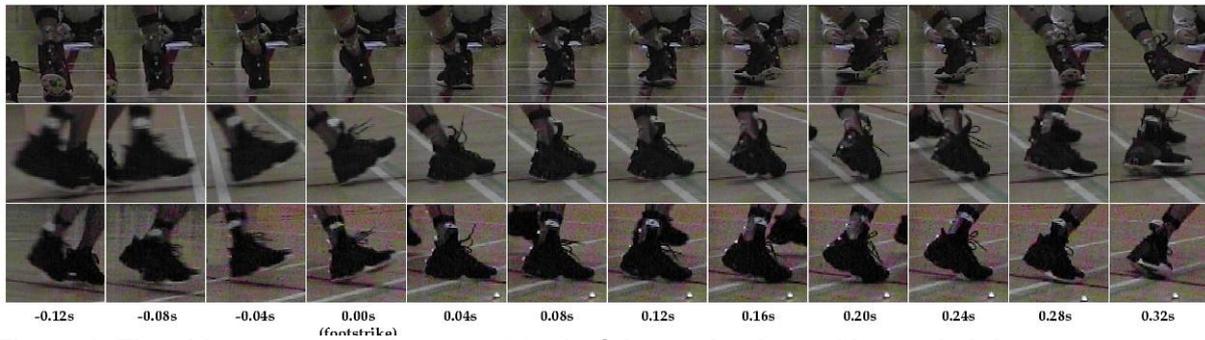


Figure 1: The video sequence (in every 0.04s) of the supination ankle sprain injury

RESULTS:

Figure 2 shows the foot and ankle angles for the successful normal trials and the injury trial. At foot strike, for the injury trial, the ankle was more internally rotated for 7 degrees (less externally rotated from 21 to 14 degrees) and more inverted for 6 degrees (from 9 to 15 degrees). After landing, there was a two-phase change of ankle kinematics, as primarily determined by the profile changes of inversion. Firstly, from 0.06s, the ankle entered a risk-developing phase (Phase I) as the kinematics profile started to deviate from that of normal trials, as shown by a larger inversion. At 0.11s, the deviation halted and the ankle was inverted for 32 degrees, externally rotated for 5 degrees and dorsiflexed for 14 degrees. Secondly, from 0.11s onwards, the ankle entered the injury phase (Phase II), as there was another explosive inversion and internal rotation. The ankle further inverted for 16 degrees and internally rotated for 15 degrees. At 0.20s, the ankle reached its greatest angular displacement from the offset anatomical position. The orientation was at an absolute measure of 48 degrees inversion, 10 degrees internal rotation, and 18 degree dorsiflexion.

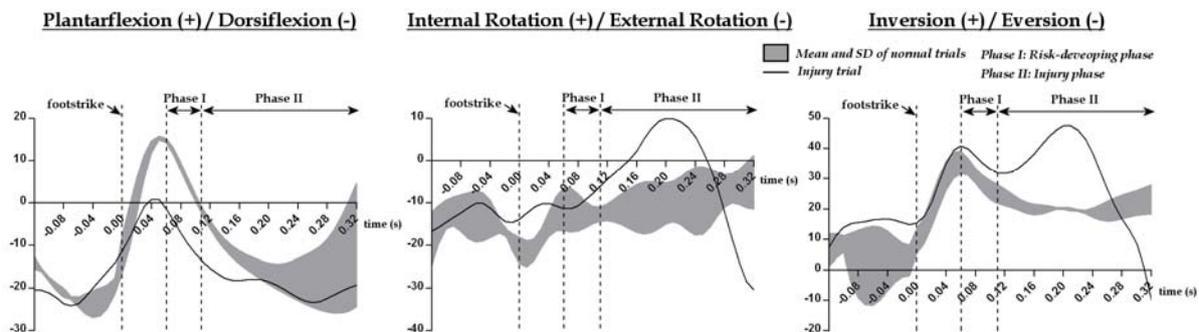


Figure 2: Foot and ankle angle for the normal (3 trials) and the injury trial (1 trial)

Figure 3 shows the plantar pressure distribution of one selected normal trial and the injury trial. The hallux was found to contribute to greater contact with the ground during most of the stance, especially in normal trials. For the injury trial, higher pressure at both heel and forefoot region was found at 0.02s after the foot strike, indicating a firm and forceful foot strike. At 0.06s onwards, the pressure at the heel reduced quickly and shifted to the forefoot region. Such a pattern suggested a lift of the rearfoot and a quick shift of center of pressure to the forefoot after foot strike, from 0.02 to 0.08s, as also shown by a quick move of the center of pressure from heel to mid-foot region in Figure 4 (the injury trial). From 0.08s to 0.20s, a chaotic pattern of the center of pressure excursion at the third and fourth metatarsal region was found, indicating an unstable foot support during this period. After 0.24s, the center of pressure shifted forward to the proximal third metatarsal, and further to the first metatarsal region finally. In normal trials, the excursion path of the center of pressure moved progressively from heel to metatarsal region in a rather stable manner.

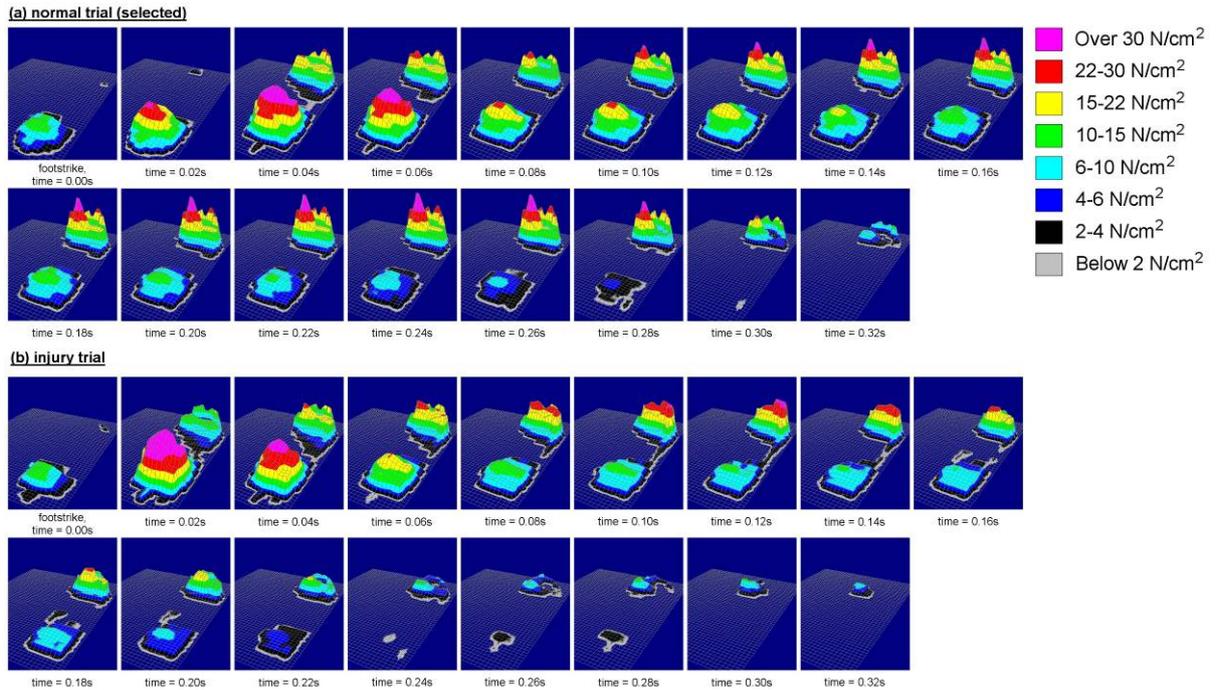


Figure 3: Plantar pressure (in every 0.02s) of (a) one normal trial, and (2) the injury trial

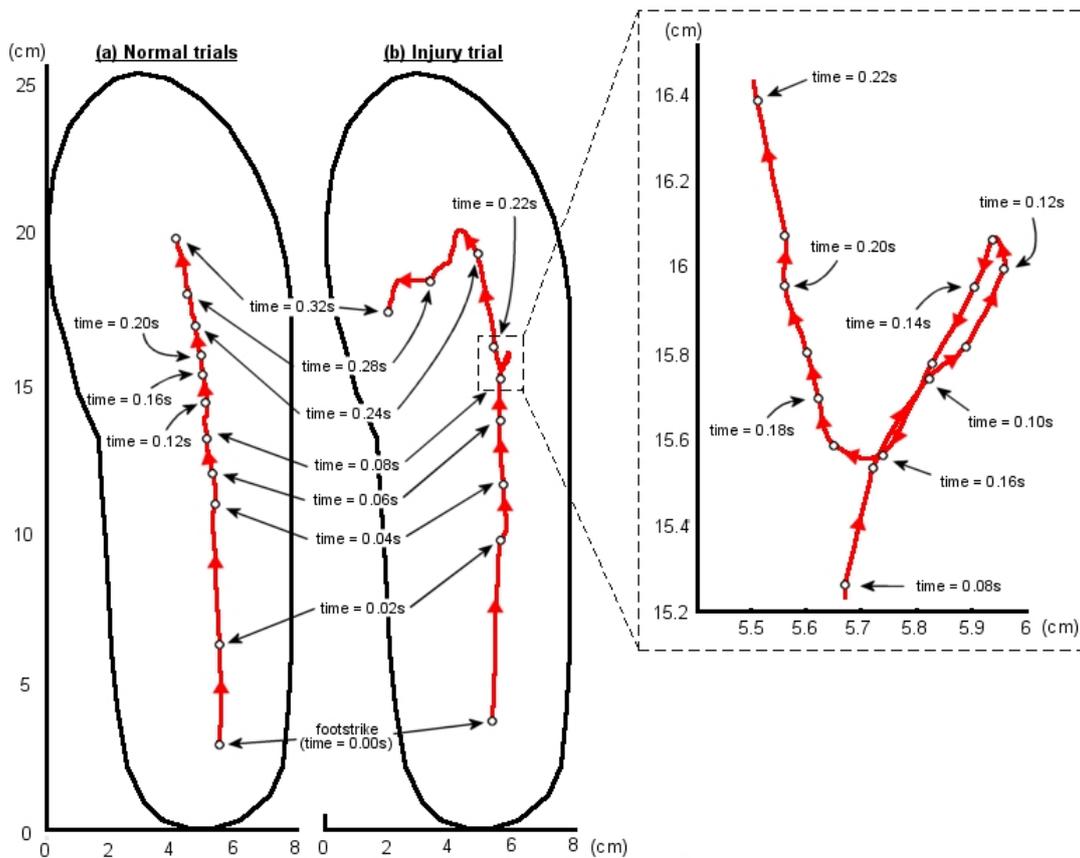


Figure 4: The excursion path of the center of pressure of (a) the normal trials, (2) the injury trial

DISCUSSION:

For the successful normal trials, the foot and ankle was externally rotated and slightly inverted at foot strike. This enhanced a flat foot landing with a maximum foot-ground contact

surface. Moreover, the externally rotated foot allowed the ground reaction force to act opposite to the landing direction for a vigorous braking and a subsequent propulsion to the contralateral side. For the injury case, the foot and ankle was more internally rotated (or less externally rotated) and greatly inverted at foot strike – this was suggested to be a vulnerable orientation for sustaining ankle sprain injury (Andersen et al., 2004). Right after landing, the foot and ankle plantarflexed in 0.04s, shifted the center of pressure to forefoot, lifted and drifted the rearfoot to the lateral side – this was a pivoting internal rotational motion. Such motion swung the ankle joint center to the lateral aspect and deviated it from the application point of the ground reaction force. It was speculated that such foot and ankle orientation resulted in a longer moment arm along the ankle axis and thus the ankle joint torque (Wright et al., 2000). Therefore, the lift and the lateral swing of the rearfoot may contribute to a sudden explosive torque and the subsequent abrupt kinematics changes.

The changes of foot and ankle kinematics were with a two-phase pattern. In the risk-developing phase, the foot and ankle orientation was within the normal foot and ankle motion range (Hertel, 2002). Therefore, it was postulated that the ATFL sprain injury was not induced in this phase. However, after this phase, at 0.11s, the foot and ankle entered an at-risk orientation – an internally rotated and inverted position, which may lead to the second injury phase that induced the ATFL sprain injury. At the lateral aspect of ankle, the peroneal muscles play a role to pronate the foot, which oppose the supination or inversion motion. Previous myoelectric investigation suggested that the reaction time of peroneal muscles in healthy male subjects with stable ankles was 55-80ms (Konradsen and Ravn, 1991). In the current case report, although the muscle reaction time was not collected, we believed that the peroneal muscles were not yet activated before the start of the risk-developing phase, that is, at 0.06s, to protect the ankle joint from going into the second injury phase at 0.11s. During this period, explosive inversion and internal rotation were observed, which reflected how the explosive ankle supination torque introduced the grade one ATFL sprain injury.

CONCLUSION:

This study presented the biomechanics of an accidental supination ankle sprain injury. The findings of this study add knowledge to the current understanding of ankle sprain mechanism and provide valuable information for designing prophylactic device for ankle sprain prevention.

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