Osteochondral talar lesions and ankle biomechanics

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Chapter 1

General introduction
Chapter 1

Broad perspective

The ankle joint is the most congruent joint of the human body. It is also one of the most common joints to be injured.

As humans started to walk upright and assumed a bipedal instead of a quadrupedal locomotion, the kinematics, kinetics and energetics changed. Advantages are that bipedalism is less energetically expensive than quadrupedal walking [80, 91], and that it serves the purpose of carrying things, as it leaves the hands free. A disadvantage may be that bipedal gait has made us more prone to injury and falling. To stay upright, bipedal gait requires a more complex body balance than quadrupedal gait. In case balance is lost, bodyweight is forcefully transmitted through only one extremity, and the result may be that of ankle joint injury.

Many ankle joint injuries occur during sports. Over the past few decades, there has been an ongoing - (r)evolution in sports participation: it has been rising significantly [76]. First, men were the frontrunners in this revolution, but today, women have caught up with men [76]. Sports like long distance running and soccer have gained immense popularity among men and women. This development is gladly seen, because the human body is made to move. Physical activity reduces the risk for several diseases and may influence the course of several chronic diseases in a positive way [77]. Although participating in sports in general is healthy, the risk of sports injuries is a disadvantage of intensive physical activity.

In the Netherlands approximately 3.7 million sports injuries occur yearly. Of these, almost 40% is treated medically [106]. In case of medically treated sports injuries it concerns acute sports injuries in most cases, in other cases the injury developed gradually. Ankle injuries comprise a large part of these sports injuries. In the Netherlands, in 2012, 770.000 sports injuries of the ankle were sustained. This comprised 17% of all sports injuries in that year [106]. After knee injuries, ankle injuries are the most common sports injuries. For almost 4 in 10 ankle injuries, medical treatment is necessary [106].

Injury of the ankle joint may lead to ankle instability and intra-articular pathology. It is associated with loose bodies, fractures, osteoarthritis, and osteochondral defects of the talus. Talar osteochondral lesions are relatively common in patients with a history of ankle trauma [6, 56, 61, 70, 93, 94]. Because of the individual suffering and the burden on society they cause, these injuries require optimal treatment.

Different terms are in use to describe bone-cartilage lesions, among these are: osteochondral lesion (OCL), osteochondral defect (OCD), osteochondral lesion of the talus (OLT), transchondral fracture and osteochondritis dissecans (OD). In this thesis the terms osteochondral lesion as well as osteochondral defect are used.

Ankle anatomy

The specific anatomy of the ankle, with its high congruency, plays a large role in the development of osteochondral defects of the talus. When the stabilizing structures are damaged, ankle biomechanics are altered and damage to the cartilage may occur.
Introduction

The proximal articulation of the ankle is the mortise, which is made up of the lower end of the tibia, its medial malleolus, the lateral malleolus of the fibula, and the transverse tibiofibular ligament. These are the deep fibers of the posterior tibiofibular ligament. Its stability depends on the integrity of the syndesmosis of the inferior tibiofibular joint. All articular surfaces are covered with hyaline cartilage.

The lower articular surface of the tibia, wider in front than posteriorly, is concave antero-posteriorly and is slightly convex from side to side. It articulates with the dorsal aspect or trochlea of the body of the talus. The lateral malleolus is at the distal end of the fibula, and projects more inferiorly and lies more posteriorly than the medial malleolus. There is a triangular articular facet on the lateral aspect of the body of the talus. The malleolar fossa lies posterior to the articular surface and gives attachment to the posterior tibiofibular and the posterior talofibular ligaments.

The talus consists of a rounded head anteriorly, a neck, and a body. The body forms the lower articular surface, is cuboidal in shape, and has a trochlear articular surface. The trochlear surface is wider anteriorly than posteriorly. It is convex from anterior to posterior and slightly concave from side to side. The medial surface has a comma-shaped articular facet for the medial malleolus and the lateral a triangular facet for the lateral malleolus [47].

When the ankle is dorsal flexed, the wider portion lies between the malleoli. This is the closepack, or stable position, of the ankle. During plantar flexion, the narrow posterior area is in the mortise, permitting some side-to-side movement. This is the least pack, or unstable position of the joint. The talus has no muscles attached to it and has a very extensive articular surface. As a result, fractures of the talus may result in avascular necrosis of either the body or the head. The relative avascularity of the talus causes the bone more to be prone to the development of subchondral cysts when the overlying cartilage is damaged. This process is not yet fully understood, though it is thought that a crack in the cartilage may act as a pressure valve. Synovial fluid can enter the bone, but not easily escape. Since fluid pressure is known to cause osteolysis [99], this mechanism could be responsible for cyst formation in talar OCDs.

Biomechanics

The ankle joint complex, mainly formed by the ankle (tibiotalar) and subtalar joints, plays a fundamental role in the human locomotor system, being involved in virtually every locomotion activity [48]. Motion at the ankle and subtalar joints is guided by the osteoarticular and ligamentous structures and induced by the forces and moments of the extrinsic muscles, in addition to the external forces. The talus does not have tendon attachments, and is constrained by ligament and contact forces [48]. When the limits of normal ankle biomechanics are violated, injury to stabilizing structures, and subsequent osteochondral lesions may occur.

The complex morphology of the ankle joint brings about a complex axis of rotation of the ankle [107]. Since the 1950s, it has been recognized that the ankle joint axis during dorsal flexion is different than that of plantar flexion [5, 33]. Barnett and Napier measured the trochlear surfaces of 152 human tali to discover that the medial and lateral curvatures of the talus are different,
indicating that the axis of rotation of the ankle changes its position during the arc of motion [5, 107]. Hicks defined these different axes as the “dorsiflexion axis” and “plantar flexion axis”, stating that movement cannot occur about these two axes simultaneously [33, 107]. In spite of these findings, for a long period of time, combined motion at these two articulations was considered to be a rotation about a single or a double fixed axis [15, 36, 37, 48, 90]. Patterns of joint motion were investigated thoroughly, but basically with this same assumption [48, 55, 89, 107]. More recent studies have reported that the instantaneous axis of rotation translates and rotates during passive dorsal to plantar flexion [49, 50], suggesting that the hinge joint concept is an oversimplification [48].

In 1994, it was stated by Van den Bogert et al., that is was extremely difficult to determine the ankle joint axes, around which the actual rotational movements occur [98]. And consequently, that it was difficult to describe the movement of the ankle joint complex using functional axes. In 2002, it was still concluded by Nigg and Hintermann, that is was practically impossible to estimate the location of the talus during locomotion [66]. A lot of technical progress has been made since however. With new techniques of imaging software and hardware it is possible to accurately determine talar location during motion [23]. Furthermore, research of movement has gone from invasive (insertion of tantalum bone markers), to minimally invasive (by generating CT scans of the ankle and hindfoot).

Fundamental information about the biomechanical characteristics of the ankle joint may contribute to a better understanding of the normal function of the ankle, the pathomechanics of injury and resulting ankle instability, and subsequent intra-articular damage.

History
An osteochondral defect is a lesion involving articular cartilage and subchondral bone. The first report on OCDs of the talus was by Monro in 1856 [62]. He discussed the presence of cartilaginous bodies in the ankle joint. König, in 1888, suspected inflammation to be an etiologic factor. He suggested that the loose bodies were caused by vascular occlusion, leading to spontaneous osteonecrosis. He referred to the condition as osteochondritis dissecans. Inflammation has never been demonstrated to be part of the pathology, however.

In the early 1950s, the cause of talar osteochondral lesions was unknown. In 1953, Rödén et al. reported their series of osteochondral lesions of the talus. They were the first to conclude that almost all lateral lesions were secondary to trauma. In their series, for medial lesions this relationship was not found [79]. In 1959, Berndt and Harty laid a solid foundation, in their elegant clinical and anatomic studies, supporting trauma as a primary cause, particularly of lesions of the lateral talar dome [6]. In their study, 57% was located on the medial shoulder, mostly the posterior third, and 43% of lesions were located on the lateral shoulder of the talus, mostly in the middle third (Fig. 1). They also presented an enduring four-stage radiographic classification scheme. Following their report, several authors reported of talar OCDs as a sequel to ankle sprain [9, 12, 22, 65].
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The lesion can be caused by a single traumatic event, or by multiple, less intense microtraumata. Although the origin of most lesions is traumatic, not all patients report a history of ankle trauma. Atraumatic causes, including endocrine or metabolic factors [71], degenerative joint disease [94], joint mal-alignment [28], genetic predisposition [63], and ischemia and necrosis [84] have been proposed. The occurrence of OCDs in identical twins and siblings [1, 109], and the fact that the defect is bilateral in 4 to 7% of patients [6, 9], further supports the hypothesis that some lesions are of genetic origin.

Today, ankle trauma is accepted as the most frequent etiologic factor for OCDs of the talus [34, 82, 83]. The lesion can be caused by a single traumatic event, or by multiple, less intense microtraumata. Although the origin of most lesions is traumatic, not all patients report a history of ankle trauma. Atraumatic causes, including endocrine or metabolic factors [71], degenerative joint disease [94], joint mal-alignment [28], genetic predisposition [63], and ischemia and necrosis [84] have been proposed. The occurrence of OCDs in identical twins and siblings [1, 109], and the fact that the defect is bilateral in 4 to 7% of patients [6, 9], further supports the hypothesis that some lesions are of genetic origin.

During an ankle sprain or fracture, the talus twists inside the ankle mortise, and the cartilage of the distal tibia and talar dome are forcefully pushed upon each other (Fig. 2). Ankle sprains usually occur when the foot is forcefully inverted when the ankle is plantar flexed [24, 44, 46, 59]; in this position the bony structure allows only minimal stability, the leverage is maximal, and the anterior talofibular ligament (ATFL), which is the weakest component of the lateral ligament complex of the ankle [3, 88], is taut and exposed to injury.

The degree of injury depends on the force exerted and on the range of abnormal motion that is enforced on the ankle. This force could be modified by timely muscle activation, external protection, and reasonable attention to the course or playing ground.
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When defects occur, they can be found on the medial and lateral sides of the talar dome. Occasionally they are located centrally [25]. Approximately two-thirds of OCDs are located medially, and one-third is located laterally [18]. Lateral lesions occur mostly on the anterolateral, and medial lesions on the posteromedial shoulder of the talar dome [73]. Lateral lesions are usually shallow and cup-shaped, and caused by torsional impaction during axial loading of the ankle [6].

Figure 2 During an ankle sprain, the talus twists inside its box-like housing, and the cartilage of the talar dome is pushed forcefully upon the distal tibia, creating an osteochondral lesion (*).

According to Yao and Weis, medial lesions are most likely caused by an inversion of the foot and a plantar flexed ankle. Lateral lesions are caused by eversion of the foot with the ankle in dorsal flexion and the tibia internally rotated on the talus. These lesions are almost always secondary to trauma [110].

The impact on the talus may lead to subchondral bruising and subsequent softening of the cartilage, to a crack and subsequent cyst formation [38, 74, 101], or the shearing off of a chondral or osteochondral fragment [6]. The formation of subchondral cysts is thought to be due to intrusion of fluid through a bony defect, and may hamper effective treatment [75, 78, 87, 101].

To evaluate (osteochondral) damage to the talar dome and tibial plafond after ankle injury, Van Dijk performed ankle arthroscopies 4 to 7 days after trauma. He documented the arthroscopic findings and treatment, especially lavage and, if necessary, ligament repair. During a 1.5 year period he treated and recorded the progress of 30 patients, subsequently documenting his findings. In the series of 30 consecutive patients with rupture of the lateral ankle ligaments after acute supination trauma, 1 medial, 1 lateral and 4 tibial plafond osteochondral lesions were
found. Apart from these lesions in the weight-bearing area, in 16 patients he also found a kissing cartilage lesion between the medial talar facet and the anterior cartilage of the medial malleolus [105].

**Epidemiology**

Osteochondral defects can occur in any joint. The most common location is the knee. It is assumed that OCDs of the ankle account for approximately 4% of the total number of OCDs [13]. OCDs of the ankle used to occur most frequently in 20- to 30-year-old men [61], but with the rising sports and work participation of women, the male-female ratio is now approximately 3:2 [96]. It is estimated that one ankle injury per 10,000 people per day occurs [41]. With this extremely high occurrence, ankle sprains comprise 16 to 45% of sports-related injuries [19, 24, 54, 58, 64]. They represent 25% of volleyball [24] and track and field injuries [57], 20 to 31% of soccer injuries [17, 24, 81], and 45% of basketball injuries [24]. In military training, the rates are even higher, and reported to be 30 to 60% [4, 40].

The exact incidence of talar OCDs in the adult population is unknown. Because they mainly occur after ankle injury, the incidence after ankle sprain and fracture has been determined by several authors. Besides Van Dijk [105], two other investigators who routinely inspected the lateral talar dome during surgery for lateral ankle ligament rupture, determined the rate of lateral OCDs after this event. They reported it to be 5% [7] and 9% [53], respectively. The percentage of medial dome lesions is unknown but estimated to be as high as lateral talar dome lesions [105]. Higher numbers of talar OCDs after ankle injury were published more recently by Takao et al. In a group of 86 patients visiting the hospital for lateral ankle instability, osteochondral lesions were found in 41% of patients. In case of patients with a distal fibular fracture requiring osteosynthesis, the incidence was 71%. Diagnosis was performed by MRI and arthroscopy and included OCDs of the tibial plafond as well [93]. Leontaritis et al. found chondral injuries in 73% of patients (61/84) with an ankle fracture, confirmed during arthroscopy [51].

The incidence of OCDs of the ankle in children was investigated by Kessler et al. [42]. In their population based cohort study they found an incidence of 4.6 per 100,000 in children aged 6 to 19 years. Patients aged 12 to 19 years represented the vast majority of those with OCD, with an incidence of 6.8 per 100,000, compared to 1.1 per 100,000 in those 6 to 11 years of age. Teenagers therefore had nearly 7 times the risk for ankle OCD compared with children 6 to 11 years of age.

**Clinical presentation**

Patients with an OCD of the talus are typically young, active adults with deep ankle pain during or after activity [21]. Other symptoms may include stiffness, swelling, and sometimes a locking sensation of the ankle. Locking and catching are symptoms of a displaced fragment. In most patients who have a non-displaced lesion after supination trauma, the symptoms in the acute situation cannot be distinguished from the soft tissue damage [102].
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When an ankle sprain has occurred, a differentiation has to be made between the acute and the chronic situation. In the acute situation, symptoms of OCDs compare with those of acute ankle injuries. These are swelling, diffuse or localized ankle pain, and pain on weight-bearing. When it concerns isolated ligament injury, pain and swelling usually resolve after a couple of weeks of reduced loading of the ankle, sometimes in combination with tape or softcast. When symptoms persist after this period, a talar OCD may be present. These patients typically present with deep ankle pain on weight-bearing, and often a limited range of motion. Lateral lesions in general cause more symptoms than medial ones [102].

The natural course of degenerative joint disease following an OCD has not been well defined. However, it is known that subchondral cysts [101] may form and that when the lesion is large and the affected piece is dissected, the joint mechanics are altered, which may lead to osteoarthritis.

**Diagnosis**

When after careful history taking and physical examination of the ankle an OCD is suspected, weight-bearing anteroposterior, mortise, and lateral views are taken. A bone fragment may be present in the joint space, and sometimes radiolucency of the talar dome is seen. Often there are no abnormalities found at all during primary routine radiography. In a later stage, the defect sometimes becomes visible when bone loss develops with subsequent radiolucency (Fig. 3). A posteromedial or posterolateral defect may be revealed by a heelrise view with the ankle in plantar flexed position [108]. When multiple injuries are present in the ankle, a bone scan can differentiate between a symptomatic and an asymptomatic lesion [100].
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Computed tomography (CT) and magnetic resonance imaging (MRI) allow a 3-dimensional evaluation of a lesion (Figs. 4 and 5, respectively). The sensitivity and specificity of CT to detect an OCD are 0.81 and 0.99, respectively. Those of MRI are 0.96 and 0.96 [108]. MRI shows the surrounding bony edema, if present. The exact size and location of the lesion are better defined by CT, therefore CT is more valuable for preoperative planning [108].

Treatment
Several surgical techniques are available for the treatment of symptomatic OCDs of the ankle. Non-surgical techniques, like immobilization in a cast, are almost out of use because of their low overall success rates. When conservative therapy is still applied, it usually concerns a younger patient with an acute partially detached lesion, or an acute lesion with a completely detached fragment that is still in place [8].
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The type of surgical treatment needed, and the location of the lesion both determine the surgical approach. Most primary lesions can be treated by means of arthroscopy, but lesions in the posterior half of the talar dome may be hard to address arthroscopically. For secondary lesions, often a different, more invasive treatment is chosen, requiring open surgery, with or without osteotomy [32].

Surgery for primary lesions is mostly done by debridement and bone marrow stimulation. First it was performed during an open surgical procedure; nowadays it is performed almost exclusively arthroscopically. When possible, the OCD is approached by anterior ankle arthroscopy with the ankle in full plantar flexion for adequate exposure of the defect [104]. When needed, noninvasive intermittent distraction is used to improve exposure [103].

After debridement, the subchondral bone is penetrated by drilling or microfracturing. This may be done by a chondral pick or K-wire. The sclerotic zone that is often present is partially destroyed and multiple openings are created into the subchondral bone. This causes disruption of intraosseous blood vessels and subsequent bleeding. Growth factors are released and a fibrin clot forms inside the defect. Neovascularization causes marrow cells to be introduced into the defect and fibrocartilaginous tissue is formed. In the case of large defects, a cancellous bone graft can be applied.

In 1986, Parisien [67] described the arthroscopic procedure to accomplish this formation of fibrocartilaginous tissue. Good results were reported and have been confirmed by others [86, 95, 97]. Also more recent reports continue to show satisfactory results after arthroscopic debridement and microfracturing [11, 52, 68].

Arthroscopic debridement and microfracturing, also known as bone marrow stimulation, remains a popular option, because of the low-cost, minimally invasive character of the procedure. Ankle arthroscopy is regarded a safe procedure, but complication rates need to be diminished [20]. Reduction of complications will contribute to making it an even more attractive procedure for young active patients with ankle pathology.

Rehabilitation after debridement and bone marrow stimulation may take up to 1 year postoperatively. In most cases, surgery is followed by a period of non-weight-bearing, of 4 to 6 weeks. Rationale for this is to give the fibrocartilage an optimal chance to develop and to become secured to the surrounding bone and cartilage. The necessity of this non-weight-bearing period has been debated lately by Li et al. [52], who reported successful outcomes after early weight-bearing. Follow-up however was limited.

Securing the lesion to the talar dome is possible in case of a large loose fragment. One can choose to secure it to the underlying bone using either a screw, a pin, a rod or fibrin glue [111]. In case of a large cyst grafting may be necessary, using either autologous cancellous bone or demineralized bone matrix (DBM) from donors, as an alternative to autologous bone grafting. DBM has osteoconductive, osteoinductive, and osteogenic potential [14, 16, 72]. Its value in the treatment of cystic osteochondral lesions has yet to be proven however.
Introduction

Retrograde drilling is done for primary OCDs when there is more or less intact cartilage with a large subchondral cyst, or when the defect is hard to reach via the usual anterolateral and anteromedial portals [27].

For stimulation of the development of hyaline cartilage, several options are available: allogenous and autogenous osteochondral transplantation, bone grafting, and ACI. They have all been developed in the past 2 decades [2, 10, 25, 26, 29-31, 35, 39, 43, 45, 60, 69, 85, 92]. These techniques have specific indications according to the stage, location, and extent of damaged area. The age and the lifestyle of the patient also influence the specific procedure that is recommended.

ACI with periosteal flapping of osteochondral lesions in the ankle joint is gaining more popularity, since the increasing number of promising clinical reports [30, 69]. In contrast to the good clinical outcome, the length of the rehabilitation, high laboratory costs, and technical problems appear to be disadvantageous elements of this technique.

Treatment results vary widely in literature. It is unknown which strategy is most effective in the treatment of talar OCDs, therefore further study on this topic is imperative. Most surgery for talar OCDs is performed arthroscopically, but not without complications. To improve arthroscopic treatment, increasing knowledge of the number and type of complications is mandatory. Finally, none of the current treatment methods is completely successful, and secondary treatment options have their limitations. This necessitates the search for better treatment methods, in order to deliver optimal care for the individual patient.

Aims and outline of the thesis

This thesis aims at defining the appropriate surgical treatment for osteochondral talar lesions, and how to improve this treatment by investigating ankle biomechanics, evaluating existing techniques and developing new techniques. To accomplish this aim, treatment results, complications in ankle arthroscopy, normal motion patterns of the ankle and arthroscopic access to the talar dome were studied. Furthermore, a new treatment method for talar OCDs was developed.

Part I describes the current concepts in the treatment of OCDs. Part II reviews the current treatment options; it summarizes all eligible studies to compare the effectiveness of treatment strategies for osteochondral lesions of the talus. It also aims at improving the technique of ankle arthroscopy, which is still the most used technique in treatment of talar OCDs. It elucidates the types of complications that may occur during ankle arthroscopy, and describes how the use of the dorsiflexion method may prevent a significant number of complications. Part III presents a chapter describing a computed tomography-based stress-test to determine the three-dimensional position and orientation of the tibial, calcaneal and talar bones in eight extreme foot positions. Talocrural and subtalar range of motion for healthy individuals was determined. Data of this study were used in the next chapter to aim to define arthroscopic access to the talar dome. Part IV describes two studies that led to the development of a new treatment strategy for osteochondral lesions of the talus. Talar geometry and size and location of lesions was measured. With these
Pau Golanó and Jordi Vega commented that the anatomy of structures susceptible to injury during ankle arthroscopy. In our comment we confirm knowledge transverse section of the ankle at the level of the tibiofibular syndesmosis, showing important prevent damage during the procedure. Furthermore, they provide a clarifying picture of anatomical landmarks of the ankle, to facilitate orientation and the joint may not have been sufficiently highlighted in the study. They underline the importance of anatomy as the basis for every surgical technique and affirm the importance of staying up to date with the extensive literature on this subject.
Part III – Ankle biomechanics and arthroscopic access

Up until recently, measuring ankle biomechanics wasn’t possible by using non-invasive techniques. Technical progress however has made it possible to determine the range of motion of a joint by using computed-tomography (CT). CT, together with specifically designed software, can determine the three-dimensional (3D) orientation of bones. By scanning a joint in different extreme positions, the orientations of the bones can be determined. From these, ranges of motion are calculated. These are described as finite helical axis orientation, and rotations around and translations along these axes.

The goal of chapter 6 was to establish a quantitative database of the normal ranges of motion of the talocrural and subtalar joints by using a 3D computed tomography stress-test. A clinical case on suspected subtalar instability demonstrates the relevance of the proposed method.

The optimal arthroscopic approach (i.e. anterior or posterior) can be unclear preoperatively. During anterior arthroscopy for an OCD, the ankle is held in full plantar flexion to optimize exposure of the talar dome, increasing the chance of reaching the lesion. When a lesion is located in a part of the talar dome that remains covered by the tibial plafond in any position, osteotomy might be necessary. Chapter 7 uses the data of the 3D computed tomography stress-test of the ankle to determine and visualize arthroscopic access to the talus. Coverage of the talus by the distal tibia is determined for the neutral position and eight extreme foot positions, among which are maximum plantar flexion and maximum dorsiflexion. The area of the talar surface that may be hard to reach is visualized and described.

Part IV- Future treatment

The disadvantages associated with secondary treatment options, like donor-site morbidity and high costs, leads to the search for new strategies. To treat larger cystic and secondary lesions, current techniques aim to replace the defected cartilage with harvested cartilage or in-vitro cultured cartilage. It was hypothesized that defect can also be covered by a metal device, as is done before in the knee. To develop a metallic resurfacing implant, knowledge of talar geometry, lesion size and location is necessary. Chapter 8 describes in detail the measurements of the sizes of medial and lateral talar OCDs. Furthermore, it describes the measurements of talar radius and angle of the talar shoulder at the location where the osteochondral lesion occurs. These data served as the basis for the development of the talar Hemicap, a prosthetic device consisting of a screw and a metallic cap, which is able to restore the continuity of the talar articular surface, and cover a subchondral lesion. Fifteen offset sizes were developed and it was hypothesized that there would be a matching offset size for each talus, that the prosthetic device can be implanted slightly recessed to the cartilage level, and that excessive contact pressures on the opposite tibial cartilage can be avoided. Chapter 9 aims to test these hypotheses by implanting the prosthetic device on the medial talar surface of 11 intact fresh-frozen human cadaver ankles, aiming its surface 0.5 mm below cartilage level.
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Part V – General discussion
Chapter 10 provides a general discussion and conclusions. In chapter 11 provides a summary of the thesis.

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