Biomechanics of the Normal and Arthritic Ankle Joint

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KEYWORDS

• Osteoarthritis • Ankle joint • Biomechanics • Arthroplasty

KEY POINTS

- OA of the ankle can be debilitating and has a significant impact on quality of life.
- When compared with the hip and knee joint where primary OA is more common, the ankle seems less susceptible to onset of primary OA. Onset of OA can be related to altered mechanical loading patterns of the cartilage, and downstream changes in tissue metabolism and joint structure.
- Understanding biomechanics of the normal and arthritic ankle joint can aid in the analysis of an underlying clinical problem and provide a strategic basis for a more optimal management.

INTRODUCTION

Osteoarthritis (OA) of the ankle can be debilitating and has a significant impact on quality of life. When it comes to the decision that treatment should be considered, physicians can choose among conservative and surgical measures. The palette of treatment is manifold and should always be tailored to the needs of patient and the degree of disease. To select the most appropriate treatment for a given patient, it is often helpful to understand the biomechanics (functional anatomy) of the normal ankle joint, how pathology can alter normal function, and the biomechanical consequences that a given surgical intervention will provoke.

When compared with the hip and knee joint where primary OA is more common, the ankle seems less susceptible to onset of primary OA. Onset of OA can be related to altered mechanical loading patterns of the cartilage, and downstream changes in tissue metabolism and joint structure. Altered joint loading patterns have been associated with aging and trauma. In contrast to the knee and hip, a high degree of joint surface congruency and correspondingly constrained kinematics, combined with

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a generally more limited range of motion may make the cartilage of the intact ankle joint less sensitive to age-related changes that can play a role in primary OA. This is supported by the fact that nearly 70% of cases of ankle (OA) are secondary to a traumatic event, most frequently involving ankle fractures and subsequently unaddressed chronic ankle instability. Here disruption or injury to cartilage tissue itself, or injury-related changes in loading of the cartilage surfaces can thus initiate a progressive degeneration of the joint that eventually requires operative intervention.

Understanding biomechanics of the normal and arthritic ankle joint can undoubtedly aid in the analysis of an underlying clinical problem and provide a strategic basis for a more optimal management. The challenge to the clinician and the biomechanist is that the mechanical (and for that matter, physiologic) complexity of the ankle joint still clouds current understanding. This article provides the reader with an overview of current understanding of functional ankle anatomy, how this function can be altered in the degenerated ankle, and how surgical intervention further affects foot and ankle biomechanics. The intention is to provide the reader with insight to the mechanopathophysiology of degenerative cartilage tissue remodeling, how it can affect the kinetics and kinematics of the ankle joint, and the postoperative implications of a tibiotalar surgical intervention. Here the focus is on how altered (often increased) loading of neighboring joints in the midfoot and hindfoot may induce postoperative joint remodeling and can manifest in secondary clinical problems.

FUNCTIONAL ANATOMY OF THE HINDFOOT

To understand the biomechanics of the hindfoot a thorough knowledge of its anatomy is essential. During gait the hindfoot distributes forces from a vertical position into a horizontal one and vice versa This is a complex kinematic process and requires the tibiotalar, subtalar, and distal tibiofibular joints to act as a combined osseous and soft tissue (ligaments, capsule, retinacula) support complex, which provides proprioception, stabilization, and control over the movement of the talus and calcaneus around their axes of motion. A stable ankle results from a perfect interplay of static (bones, ligaments, retinaculum) and dynamic anatomic structures (muscles, tendons).

The ankle joint is a highly constrained articulation, composed of the tibia, talus, and fibula, which provide stability together with the tendons, ligaments, and syndesmoses. Tendons and ligaments attribute to dynamic stabilization of the joint. Motion at the ankle is multiplanar and linked to the tibia. The course of movement within the ankle joint is predominantly from plantarflexion to dorsiflexion, but contains mild degrees of internal and external rotation.

The talus articulates with the tibial plafond superiorly, the tibia medially, and the fibula laterally. The talus has a convex dome and is wider anteriorly than posteriorly, thus the greatest contact between tibiotalar and tibiofibular surfaces is achieved during midstance phase. As such, the ankle in dorsiflexion is in its most stable condition.[1] Despite this the talus exhibits a cone-shape nature with a greater radius of its medial part than the lateral. The strongest part of the tibial plafond is found posteromedially. Resection of the subchondral layer reduces the compressive resistance of the bone by 30% to 50%. When resecting 1 cm of the distal tibia compressive resistance is reduced 70%–90%).[2]

Passive stability of the ankle depends on congruity of its articular surfaces and the integrity of the ligamentous and retinacular complexes.[3–6] Under weightbearing conditions the congruency of bones provides 100% of stability for eversion and inversion but only 30% of rotational stability. The ligamentous complexes predominantly control

rotatory stability and anterioposterior tibiotalar shifting. Under nonweightbearing conditions, stability in the frontal plane is supported by the malleoli and in the sagittal, frontal, and transversal plane by the collateral ligaments together with the musculature.[3] The anterior talofibular ligament (ATFL) originates at the distal anterior fibula and inserts on the body of the talus and blends into the anterior joint capsule, approximately 18 mm above the subtalar joint line.[7–9] It is approximately 20 mm long, 8 mm wide, 2 mm thick, and spans the anterior ankle joint. The angle in relation to the floor averages approximately 75 degrees. The ATFL primarily restricts internal rotation of the talus in the mortise. The ATFL has the highest degree of deformation (ie, greatest strain) but the lowest load to failure when compared with the calcaneofibular ligament (CFL). The CFL originates from the anterior border of the distal lateral malleolus, close to the origin of the ATFL and attaches to a small tubercle posterior and superior to the peroneal tubercle of the calcaneus approximately 13 mm distal and posterior to the subtalar joint line. It is confluent with the peroneal tendon sheath. The CFL is 20 to 30 mm long, almost 5 mm wide, and 3 to 5 mm thick. Together, the ATFL and CFL form an angle of about 105 degrees in the sagittal plane and an angle averaging 90 to 100 degrees in the frontal plane The CFL stabilizes the subtalar joint and inhibits adduction and exerts its greatest effect in the neutral and dorsiflexed position. In dorsiflexion, the CFL approaches a vertical position with respect to the subtalar joint and acts as a true collateral ligament, preventing talar tilting. The PTFL is a strong ligament with broad insertion on the talus and the fibula.[10] It originates at the posteromedial aspect of the fibula and runs in a horizontal direction to insert at the posterolateral aspect of the talus.[9] The PTFL becomes taught during dorsiflexion of the ankle and rarely ruptures.[11–14]

The inferior extensor retinaculum adds mechanical support to the ATFL and CFL. It is divided into three bands (lateral, intermediate, and medial roots) that retain the extensor digitorum longus, extensor digitorum brevis, and peroneus tertius. The inferior extensor retinaculum is attached to the lateral talus and calcaneus. Together with the CFL, the lateral root of the inferior extensor retinaculum constitutes the superficial ligamentous support of the subtalar joint.

On the medial side the deltoid ligament or medial collateral ligament (MCL) provides a strong ligamentous support to the ankle joint. It is divided into two portions: the superficial and deep layers.[10,15,16] According to Milner and Soames's classification the MCL complex consists of six ligaments.[17,18] The superficial layer of the deltoid is made up of the tibiospring; the tibionavicular; the superficial posterior tibiotalar, and tibiocalcaneal ligaments. The differentiation between these structures is difficult. The superficial layer is a broad, fanshaped, and continuous structure arising from the anterior colliculus of the medial malleolus. The tibionavicular component attaches to the navicular bone medially and blends with its fibers into the superomedial component of the spring ligament.[19,20] The tibiospring ligament extends to the superior border of the plantar calcaneonavicular ligament (spring ligament).[21] The tibiocalcaneal ligament runs in a vertical direction and inferiorly to insert onto the sustentaculum tali.[8] The deep layer of the MCL includes the deep posterior tibiotalar and deep anterior tibiotalar ligaments. Both are intra-articular but extrasynovial. Therefore, deep portion and superficial portion of the MCL complex are anatomically separated. The anterior tibiotalar ligament arises from the lateral anterior colliculus and inserts on the medial aspect of the talus just distal to the articular surface. The posterior tibiotalar ligament originates from the intercollicular groove to extend posterolateraly to its attachment on the medial tubercle of the talus.[8] The MCL complex acts equally to resist valgus tilting of the talus and as a secondary restraint against anterior translation. The deep layer of the MCL provides the greatest restraint against lateral translation.[22,23]

Considering the ligamentous support for the subtalar joint there is no consensus found in the literature regarding precise terminology of the ligaments or function. However, some agreement has been reached in classifying the subtalar ligaments into three layers: (1) a superficial layer containing the lateral root of the inferior retinaculum, the lateral talocalcaneal ligament, and the CFL; (2) an intermediate layer containing the intermediate root of the inferior retinaculum and the cervical ligament; and (3) a deep layer containing the medial root of the inferior retinaculum and the interosseous talocalcaneal ligament.[7,21,24] The interosseous talocalcaneal ligament ensures adequate function of the hindfoot and is a strong bond between the calcaneus and talus. It originates at the most medial part of the sinus tarsi (some fibers attach to the deep portion of the deltoid ligament), courses downward and lateral to the sulcus calcanei, where it blends with the most medial fibers of the cervical ligament.[8] The cervical ligament is located within the sinus tarsi and runs in an oblique fashion from the neck of the talus to the superior surface of the calcaneus and separates the anterior from the posterior joint capsule The cervical ligament is a resistive bundle connecting talus and calcaneus. It is thought that the cervical ligament together with the interosseous talocalcaneal ligament might play an important role in subtalar instability.

There are three axes of motion: one in the transversal, one in the frontal, and one in the sagittal plane (**Fig. 1**). Each of these axes generates a rotatory plane perpendicular to that axis. According to Huson,[25] rearfoot motion is often described as motion according to these cardinal planes (**Fig. 2**). However, it is important to understand that rearfoot motion does not happen in these isolated planes only. The ankle and subtalar joint both have oblique axes of motion. With regard to the ankle joint Kelikian[26] stated that "the axis of motion is the imaginary line through which motion occurs. It can be static, dynamic, single or multiplanar." Sarrafian[9] described two concepts: the single axis of motion and multiple axis of motion. According to the work of Inman,[27,28] the empiric axis of the ankle joint runs slightly distal to the tip of the medial malleolus (5 mm $\pm$ 3 mm) and distal (3 mm $\pm$ 2 mm)-anterior (8 mm $\pm$ 5 mm) through the tip of the lateral malleolus. Related to the frontal plane the axis is inclined downward and laterally and in relation to the horizontal plane it is rotated posterolaterally. The angle between axis of the tibial plafond and the midline bisecting the tibial shaft projected to the frontal plane is 82.7 degrees ($\pm$ 3.7 degrees). Projected to the transverse plane the angle of the empiric axis of the ankle joint with the transverse axis of the knee is 20 to 30 degrees.[27,28] Barnett and Napier[29] and Hicks[30] were first to introduce the concept of multiple axes at the hindfoot and recognized that the axis of

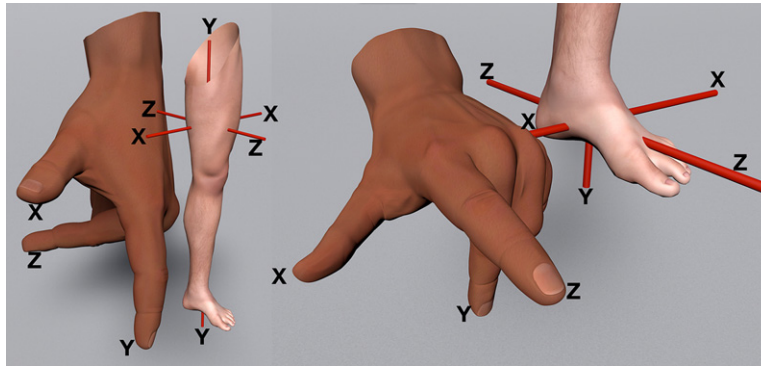

Fig. 1. Schematic representation of the axes of motion.

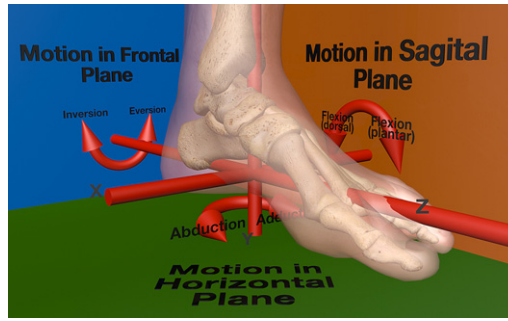

Fig. 2. Rearfoot motion is often described as according the cardinal anatomical planes.

motion at the ankle is inclined downward and laterally at dorsiflexion and downward and medially in plantarflexion. The different curvatures of the talar trochlea have been found to be responsible for this phenomenon. The medial curvature radius of the marginal profile of the trochlea tali is higher posteriorly than ventral. The opposite is found on the lateral marginal profile of the trochlea tali.[31] The lateral profile is almost a true circle, whereupon the medial profile is formed by an arc of two circles with different radii.[29] A virtual line connecting the center of these arcs specified by the curvature radius of the medial and lateral marginal profile of the talar trochlea represents the axis of motion.

The subtalar joint consists of an anterior, medial, and posterior joint, each of them having its own capsule and separated by the sinus tarsi and the tarsal canal. As mentioned, the axis of the subtalar joint is also oblique.[32–35] According to Perry and Schoneberger[36] and Viladot and colleagues,[37] the posterior subtalar joint and the anterior subtalar joint have a different center of rotation. The resulting axis of the subtalar joint is oriented upward, anteriorly and medially. Close[32] and coworkers reported a 42-degree ($\pm$ 9 degrees) upward tilt and 23-degree ($\pm$ 11 degree) medial deviation in the horizontal plane to the perpendicular axis of the foot (**Fig. 3**). Van Langelaan[38] was able to show that subtalar joint motion followed a helical character. The calcaneus rotates around the interosseus ligament, resulting in a screw-like motion associated with translation and rotation. According to Inman,[39] motion at the subtalar joint is triplanar, comprised of inversion (calcaneus turns inward) and eversion (calcaneus turns outward).

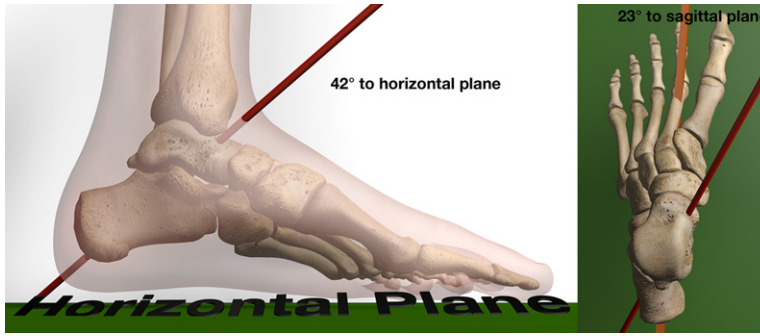

Fig. 3. The tibio-talar and subtalar joints cooperate to yield complex joint kinematics. These are visualized here in the neutral position by an instantaneous axis of rotation.

A BRIEF REVIEW OF THE POTENTIALLY CRUCIAL ROLE OF JOINT BIOMECHANICS IN ONSET OF OA

Details regarding the initiation and mid-stage progression of OA remain largely unclear, at least partly because human studies have focused on late and end stages of the disease. However, it is thought that the initiation and progression of OA is centrally related to the composition and structural organization of articular cartilage, and depends heavily on the molecular mechanisms that regulate metabolic activity (and extracellular matrix synthesis) of chondrocytes. In addition to strong dependence of chondrocyte behavior on their local biochemical environment (eg, oxygen tension), it is clearly established that the anabolic and catabolic activity of chondrocytes depends heavily on the local mechanical environment to which the cells are exposed. Both of these facts have implications to the orthopedic surgeon, who should concern himself or herself with ensuring adequate tissue blood supply and normal mechanical tissue loads.

The onset of OA represents a progression of cartilage calcification (from the subchondral bone toward the articular surface) that is often characterized as a so-called tidemark advance. The tidemark is identifiable in histologic analysis under a microscope as a rough delineation between normal hyaline articular cartilage and calcified cartilage in the deeper layers. The process of advancing calcification could possibly depend on abnormally high proangiogenic cell signaling in the joint tissues. It has also been related to aberrant cell-mediated repair of microdamage. Here, very small cracks form in the extracellular matrix as a result of normal joint loads during daily activity. This damage should initiate a targeted remodeling response to repair the matrix. For largely unknown reasons, cells seem to lose their ability to adequately respond to this microdamage as the body ages. This insufficient response is widely thought to rely on a breakdown on appropriate cell-matrix interplay.

This interplay is largely mechanics driven, with cells synthesizing matrix toward the goal of restoring a normative local mechanical environment. As the advancement of OA corresponds to the thinning of the hyaline cartilage layer, the mechanical stresses in the cartilage matrix typically increase (less tissue is available to support joint loads). Thus begins a spiraling cycle of degeneration, whereby aberrant mechanical feedback drives a chronic and pathologic remodeling process accelerating the progression of OA.

In this sense, normal articular cartilage tissue loads are essential to maintaining a healthy joint. Trauma to the periarticular bone or the constraining ligaments can be sufficient to alter the mechanical loading profile of the cartilage, and this can lead to a similarly mechanics-driven degenerative modeling process. Trauma often also directly involves the articular cartilage itself, creating a much more challenging mechanical/molecular environment for chondrocytes attempting (usually vainly) to repair the tissue. Restoring normal joint kinematics and consequent articular tissue loads may be critical to preventing the onset of secondary OA after trauma, or in slowing the progression of OA after a surgical intervention. To enable the foot and ankle surgeon to better weigh these considerations, the following section reviews the current state of knowledge regarding clinical biomechanics of the arthritic ankle.

JOINT KINEMATICS AND KINETICS IN THE HEALTHY, PATHOLOGIC, AND TREATED ANKLE

The chief obstacle to a thorough biomechanical understanding of foot and ankle function is the huge number of potential kinematic degrees of freedom presented by these structures.[40] More specifically, there exist an infinite number of plausible possibilities for stabilization of these motions by ligaments and active musculature.[41] Thus, despite

enormous technologic improvements in osteokinematic assessment (in vivo biplanar fluoroscopy being the current gold standard[42]), understanding of how soft and hard, active and passive skeletal structures interact to provide foot and ankle function remains limited.

Nonetheless, biomechanical descriptions of foot and ankle function have drastically evolved over the last 30 years. Descriptive models have advanced from extremely rudimentary (and functionally incorrect) modeling of the joint as two rigid bodies revolving around a skewed axis hinge, to more accurate and complex models that include numerous interacting joint articulations.[43–46] Such models have been developed and tested against a large body of experimental studies. The most salient findings of these studies are briefly summarized next. In particular, the focus is on joint function (walking velocity, cadence, stride length), and the underlying biomechanics of these measures (kinematics and kinetics).

Humans adopt a wide range of self-selected walking styles, with similarly ranging velocities.[47,48] In this sense self-selected (preferred) walking speed is a product of the stride length and cadence, and can be viewed as being characteristic of a given style. The painful ankle is generally associated with reduced velocity and an altered style (shorter strides, slower cadence, adoption of an asymmetric stance phase, with less time spent on the painful limb).[49–52] When the painful, degenerated ankle is treated using arthrodesis, walking velocity may be partially restored, although visible alterations in cadence and stride length generally persist.[53–57] Although early efforts using total ankle replacement (TAR) to treat the degenerated ankle generally failed to restore normal gait cadence or stride length,[50,58] results using second-generation TARs have proved more effective, but these metrics still seem to be generally reduced compared with normal controls.[51,59,60]

Similarly, ankle joint kinetics (the forces that result in a joint movement) are altered in patients with an osteoarthritic ankle.[49–51] In asymptomatic patients, kinetic analysis indicates that foot-ground reaction forces perpendicular to the ground reach a characteristic peak of approximately 120% body weight on heel strike, dropping to about 80% during mid-stance (while the opposite leg swings forward), finalized by a second peak of 120% body weight during push-off.[61] In patients with a degenerated ankle, the magnitudes of these peak forces are diminished (corresponding to an off-loading of the joint), but the spatiotemporal shape characteristics of the force-time curve remain generally unchanged.[50,51] Ground reaction forces in the transverse plane (shear forces) have been reported to be unaltered in patients with ankle degeneration.[62]

Although comparisons of ground reaction forces and corresponding joint loads is complicated by a need to control for individual walking style (cadence and stride length),[63] analysis of ground reaction forces in the arthodesed ankle has indicated generally lower forces than normal, with an anteriorly shifted center of pressure.[53,56,57] These altered kinetics have been suggested to potentially contribute to postoperative onset of mid-foot OA.[55] Ankle arthroplasty achieved using various implant designs has been shown to retain normal peak force associated with heel strike, but with diminished ground reaction forces afterward in the gait cycle.[51,59,64] This has been attributed to diminished muscle tone and an altered muscle recruitment strategy designed to protect the painful joint.[52,60,63] Whatever the cause, kinetic analysis indicates that along with diminished ground reaction forces, joint reaction moments (plantar flexion, adduction, and inversion) are similarly reduced.

Such altered gait and loading patterns are also generally reflected in joint kinematics that differ from the normal state. Exactly what constitutes normal is a problematic benchmark; the kinematic movements between bones of the foot and ankle are not only difficult to accurately quantify, but are also highly variable within the

population.[65–67] Nonetheless, some ankle pathologies have been associated with altered kinematic patterns compared with those in the asymptomatic ankle. For instance, several studies have characterized ankle kinematics in patients presenting with joint degeneration, reporting altered sagittal motion with a predisposition for maintaining the foot in a plantar-flexed posture during the swing and stance phases of gait.[50,51] Kinematic analysis of the hindfoot in patients with ankle arthrosis have shown reduced ranges of motion in all functional planes, but with sagittal plane motion reduction being the most dominant characteristic.[49]

More sophisticated kinematic analyses of the foot and ankle have indicated that the degenerated ankle joint is associated with substantially altered subtalar joint motions. Here the normally coordinated kinematic coupling of tibiotalar and subtalar joints is altered, with both joints possibly undergoing an external rotation during the stance phase of gait.[68] Thus, not only the magnitude, but even the directionality of normal subtalar motion seems to be affected by degeneration at the tibiotalar joint. This secondary alteration in subtalar joint loading seems to have implications for a "knock-on" effect that may predispose the eventual onset of degeneration in the subtalar joint. This knock-on effect may similarly follow ankle arthrodesis, by which even larger changes are required at the subtalar joint to compensate for reduced sagittal motions at the tibiotalar joint.

The downstream kinematic implications of tibiotalar fusion are not only limited to the subtalar joint. Compensatory hyperextension of the knee has been observed,[54,55] presumably to extend the duration of the midstance phase of gait. The duration of this phase of gait critically limits walking cadence and stride length. Although evidence of kinematic alterations in the neighboring midfoot joints is inconsistent,[53,54,69,70] rates of degeneration secondary to arthrodesis in these joints are relatively high,[71] and may be attributed to an adopted strategy of kinematically compensatory midfoot hyperextension.

Ankle joint motion preservation by TAR presents a more complex picture, with a wide range of reported postoperative joint kinematics that seem to be heavily dependent on implant design, and that do not always positively correlate to long-term implant survival. More specifically, first-generation TARs were reported to better preserve ankle kinematics compared with arthrodesis, but were simultaneously characterized by unacceptably short implant survival rates.[50,58] Conversely, modern design TARs are only inconsistently linked to improved ranges of sagittal ankle motion, yet are increasingly reported to yield favorable clinical outcomes with encouragingly high rates of implant survival.[51,52,59,60,72] Reduced mid- to long-term complications in modern TAR implants may be caused by many operative and nonoperative factors.[73,74] Whether these factors include a better preservation of hindfoot and midfoot joint biomechanics lacks supporting quantitative kinematic evidence, primarily because of the technical challenge of quantifying small kinematic differences at these joints in vivo.[51,59,60] However, some limited evidence exists to suggest that onset of hindfoot and midfoot OA secondary to TAR can increase after joint arthroplasty.[75]

SUMMARY

The preservation (or restoration) of normal joint kinematics and kinetics is paramount to the long-term success of a therapeutic course of action. Altered joint loads can induce and accelerate the progression of OA, and this must be considered with respect to the most appropriate course of treatment. More specifically, the unstable ankle must be considered with care, because chronic instability can create pathomechanical joint loads leading to the onset of OA.[76] Surgical modification of the tibiotalar

joint leads to altered joint loads in the midfoot and hindfoot that can be similarly problematic. In tailoring a therapy according to individual patient anatomy and presentation, the clinician and patient profit when the clinician incorporates these concerns into his or her decision-making process.

A solid grasp of foot and ankle structure and function is thus imperative to improving clinical treatment at the level of the individual patient and in setting standards of care. Historically, understanding has been hindered by the sheer complexity of this anatomic system, and limits on the ability to quantitatively measure in vivo joint motions. However, recent advances in experimental and analytical techniques have yielded drastic increases in the quality of data one can obtain, and corresponding advances in understanding have followed.

The tried-and-true clinical approach of tibiotalar fusion remains an attractive option for many patients. However, along with an ever increasing understanding of ankle biomechanics, it is not coincidental that viable last-line operative treatment of the painful arthritic ankle has expanded to include TAR. Promising reports of mid- to long-term success of next-generation TAR continue to emerge, as indications and techniques for the use of these devices become better defined and the designs of the implants themselves continue to evolve and improve. Regardless of the chosen treatment strategy, the surgeon must weigh the options that are available and the benefits that a patient is likely to derive from each. To properly weigh this balance the surgeon must consider the biomechanics at play, including downstream changes in neighboring joints that inevitably accompany any surgical intervention.

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