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ORIGINAL ARTICLE

Theory of biomechanical Evolution of the Rheumatoid Foot: a narrative review

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Background: In patients with rheumatoid arthritis (RA), the pathological progression of lower limb biomechanics is established. Although specific aspects of RA gait patterns have been studied and described, we are aware of no studies of gait pattern compensations over the entire disease course. This study aimed to describe a model that could predict the evolution of lower limb pathomechanics in patients with RA.

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Methods: A literature review was conducted of electronic databases (MEDLINE, PEDro, Trip Database, DOAJ, BioMed Central, PLOS clinical trial, ScienceDirect, and CRD York University, AHRQ, NICE, Cochrane Library) to October 3, 2023.

Results: A theory was developed that all people with RA induce or augment gait evolution syndromes following the same biomechanical course. Specifically, we postulate the "rheumatoid equinus syndrome," the "rheumatoid abnormal pronation syndrome" and the "rheumatoid shuffle syndrome," which have never been described before.

Conclusions: A new model of the evolution of gait compensation in RA is proposed. An important challenge of RA is that it increases the risk of ulcerative lesions, falls, pain, fractures, and healthcare costs. The proposed model can be used to reduce morbidity in this patient group by helping to explain and reduce the pain, deformity, and ankylosis of foot RA.

Key words: Lower limb, rheumatoid arthritis, biomechanics, functional diagnosis, equinus, flat foot, prevention, windlass mechanism, gait rehabilitation

1. Introduction

Rheumatoid arthritis (RA) is a chronic, severe, inflammatory autoimmune disease associated with disability, systemic (skeletal) complications, early death, and socioeconomic costs. Moreover the cause remains unknown, with classification based on the clinical phenotype (e.g., polyarthritis with or without autoantibodies) and the prognosis remaining guarded.[1,2,3,4] RA affects approximately 1% of the population.

A hallmark of RA is persistent synovitis that results from the sustained influx of immune cells

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(leukocytes) into the joints and degrades cartilage and bone tissue.[1] Associated bone loss is characterized by three manifestations: local erosions in the inflamed joints, periarticular bone loss of trabecular and cortical bone close to the inflammation, and systemic osteopenia and osteoporosis.[5] RA is thought to follow a stepwise disease process that originates with the generation of autoimmunity against post-translationally modified proteins, finally leading to chronic inflammation.[6]

More mechanistic disease concepts can be envisioned in the future. These may consider that the preclinical and clinical disease phases associated with anti-modified protein antibody formation, particularly anti-citrullinated protein antibodies (ACPAs), are just different phases of a single disease. The effect of ACPAs on bone damage is reflected by three important clinical observations.[6] First, patients with RA typically have low bone mass at the start of their disease, indicating that bone damage already occurs before clinical inflammation. Second, ACPA-positive RA shows more severe bone lesions than ACPA-negative RA. Third, ACPAs have a causal role in bone loss, even when in clinical remission.

The current treatment strategy is to initiate aggressive therapy soon after diagnosis and to escalate therapy guided by disease activity to achieve clinical remission. Severe disease manifestations associated with persistent and uncontrolled inflammation are now rare.[2] Clinically, RA has changed from a debilitating destructive disease with few therapeutic options to a disease that should prompt early intervention, the targeted control of inflammation, and the prevention of joint damage. Patients now have a real chance of achieving drug-free remission that, if sustained, can equal cure.[7]

In this literature review, two independent researchers carried out searches to October 3rd, 2023, using the keywords detailed in Table 1. The objective is to describe a model that predicts

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the evolution of lower limb pathomechanics in patients with RA to better explain foot pain, deformity, and ankylosis.

2. RA foot pathomechanics

Traditional signs/symptoms in the feet of patients with RA range from pain and synovitis to muscle dysfunction, bone erosions, and morning joint stiffness, and can lead to structural deformities. Few patients refractory to pharmacological treatment will actually maintain a state of chronic inflammation.

The forefoot is very often the first anatomical district involved in RA, with a high prevalence of hallux valgus and toe deformities.[8] RA of the foot includes plantar hyperkeratosis, mainly affecting metatarsal heads II and III, but also affective IV and V. These sites are often the site of intensely painful and fistulized subcutaneous bursitis. Foot pain is generally widespread, progressive, spontaneous, present at rest, and increased by load stress, palpation, or active/passive mobilization.

Inflammation shown on magnetic resonance imaging has been shown not to explain fatigue at baseline or during the disease course.[9] This could suggest ceiling effects for the damage and fatigue caused by inflammation, and furthermore, could support the concept that damage and fatigue in RA are at least partly disconnected from the inflammation.[9] We postulate that foot pathomechanics related to abnormal pronation and equinus foot are responsible.

2.1. The RA pronated foot

Foot pronation in RA seems to be characteristic of the rheumatoid gait pattern. Many authors have focused on describing the results of gait analysis. However, there have been no adequate

studies of tissue stress syndrome induced by this pronation, where the foot fails to become a "rigid lever" with pathological propulsion.[10]

In 2006, Van der Leeden[11] was among the first to suggest that forefoot joint pathologies in RA were significantly related to the gait deficit and plantar pressure overloading. Thus, a hypothesis began to arise that joint damage may correlate to, among other factors, peak pressure and integral pressure-time on the metatarsophalangeal joints; that is, "tissue stress syndrome" in abnormal pronation.[11] The physiological gait pattern studied by Bojsen-Møller introduced the abnormal pronation when foot "stiffness" is lost and a "midtarsal break" appears at each step, stating "the foot keeps its pliancy, but becomes less effective as a lever, as there is no locking of the calcaneocuboid joint"[10] (Figure 1). The tissue stress syndrome model proposed by McPoil introduced a way to reduce pathological loading forces on the injured structural components of the foot and lower extremity, helping to optimize overall gait function and prevent other pathologies or symptoms.[12]

Although this review does not consider everything related to pronation, but aspects do warrant mentioning. Some confusion exists about the use of the term pronation, which has been used as a synonym for flat foot. But, of course, this is a mistake. Pronation can certainly lead to a variable descent of the medial arch of the foot, but this is not necessarily either a deformity or a pathology. Flat foot and pronated foot are two distinct entities that may be related. In fact, pronation as a normal motion is part of the dynamic development of the foot, and has clear attributed functions, such as cushioning the contact of the foot with the ground or facilitating adaptation.[13]

Foot pronation is not just a single joint movement. Therefore, pathological pronation has repercussions that go beyond a single area of the foot.[14] More than pronation itself, we must

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also consider the timing, amount, duration, and rate of pronation to relate it to pathology, together with the ability of the foot to return to supination motion when necessary. The definitions of abnormal and hyper-pronation offered by Horwood is very useful and includes segmentation of the foot undergoing complex motions.[13]

Although it is relatively easy to identify pronated feet, pronation can be difficult to measure due to the various moving structures involved in motion, their own (and reciprocal) motions, and individual variability. Figure 1 shows that pathomechanics in gait development do not take place on the long lever, which is more efficient by activating the windlass mechanism. In fact, the foot uses the short lever to lift the body weight at the heel, moving the center of pressure (COP) laterally during the first phase of propulsion (toe-off). When the COP remains between the short lever and the long lever during subsequent phases, pathomechanics and the tissue stress syndrome occur. We postulate that the RA foot tends not to use the long lever and persists in using an intermediate lever. The long lever arm is called “high gear push-off” and provides more efficient propulsion, while the short lever is called “low gear push-off” and is related to abnormal propulsion by not increasing tension on the medial band of the plantar fascia.[9]

Feet can pronate and be considered “normal” if they do not exhibit pathology and use the long lever (Figure 1). The pronated foot in RA could play a different role due to structural pathology in joints, bones, and soft tissues. Under hyper- or abnormal pronation, the RA foot does not appears able to resist the tissue stresses produced by compensatory moments and motions.

2.2. The RA equinus foot

The equinus foot is defined as that with restricted dorsiflexion of the ankle joint (AJ), commonly

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defined as less than 8°, during weight bearing with an extended knee and neutral subtalar joint (STJ). Greater AJ dorsiflexion can be observed in a pronated STJ, while STJ supination also limits ankle dorsiflexion.[20,21,22] Although the prevalence of ankle equinus is unknown in an RA population, it causes the characteristic rheumatoid gait pattern. It develops due to shortening of the posterior muscle chains in RA that induce abnormal pronation, medialization of the subtalar axis, and use of a shorter lever during propulsion.

Several effects are related to this pathological scenario in the RA foot. First ray hypermobility, rearfoot valgus misalignment, and a collapsing medial arch can be related to the RA pronated foot.[8] Ankle equinus and higher joint stiffness complete the mechanical dysfunction.[15] Thus, abnormal foot pronation in RA is related to the rheumatoid equinus foot described by several authors.[16,17,18,19] Rheumatoid ankle equinus could be assimilated to an equinus foot and appears to develop in the sagittal plane as a "compensated equinus foot." These compensations are noted both proximally (knee flexion in acquired ankle equinus [Figure 2] and an apropulsive gait [Figure 3]) and distally (abnormal foot pronation and abducted feet [Figure 4]).

Hallux valgus, also related to abnormal pronation, is common in people with RA.[8] Since 1994 it has been agreed that hallux valgus is a multifactorial clinical problem originating mainly from an abnormal pronation syndrome.[23,24,25,26] This is consistent with considering RA equinus and the RA pronated foot as a syndrome.[27,28,29]

3. RA foot classifications

Several classifications of foot functionality have been presented, with each having its own

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characteristics and uses. These include the Foot Function Index, the Foot Health Status Questionnaire, the International Classification of Functioning, and the Swindon Foot and Ankle Questionnaire. Further development and validation of these tools is needed,[19] to ensure that the classification criteria are designed to determine the functional status of the patient's foot and the prescription of therapeutic foot orthoses and shoes. Pain is the main foot symptom in RA and its importance in predicting disability is already well argued.[16] While many patients with RA have foot disorders, these are often overlooked.[30] The DAS-28 score is the most widely used assessment tool for RA, but this does not include the foot joints and may lead to an underestimation of foot problems and related joint damage, along with a delay in podiatric care.[19]

Furthermore, patients in remission may experience foot pain: the symptoms and signs of forefoot damage are often already present at diagnosis and peak within 5–10 years.[31] Hindfoot symptoms also occur very early, often after the first signs of forefoot damage.[31] These data indicate that forefoot damage precedes hindfoot injury. If patients with RA are treated early and correctly, forefoot/midfoot damage could be avoided and possibly delay or avoid hindfoot damage, preserving function and reducing pain and disability. Despite being used more in research than in clinical practice, the Sharp/Van der Heijde and the Larsen scores focus on forefoot deformities and not midtarsal joint (MTJ) or STJ deformities[31,32] that affect forefoot deformities biomechanically.[20,21,22] To our knowledge, there is no sequence-based classification of anatomical changes in the RA forefoot.[33]

4. Discussion of RA foot compensations

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4.1. Rheumatoid equinus, foot functional deterioration, and rigidity

In patients with RA, screening for AJ dorsiflexion evolution may offer an early clinical indicator of functional foot limitation. Reduced range of motion (ROM) of the AJ is associated with RA and the presence of synovitis.[34,35]

Reductions in walking speed and other spatiotemporal parameters are also observed when describing gait cycle quality in RA, including shorter stride length (reduced joint moments), increased mid-stance time, plantigrade ground contact, apropulsive gait (sagittal plane deficit, also called postural sway; Figure 3), decreased foot elevation angle in toe-off, double stance time increase, and heel stance time increase.[34,35] These are associated, in turn, with a significantly increased risk of falls.[36,37,38,39,40]

Other aspects are not adequately clarified in the literature, despite being central and requiring research attention, as the wider flexion angle of the hip to compensate for reduced ROM in the knee joint and AJ, increased angle of step progression, and STJ axis medialization (Figure 5). Notably, the ROM of all foot joints is reduced in all planes. Forefoot ROM appears to decrease by 31%–53%.[29,39] Hindfoot eversion increases, disadvantaging calcaneal inversion, which instead decreases,[29,39] causing a structured hindfoot valgus. Locke reported in 1984 that patients with RA tend to walk with a knee flexed,[40] which we interpret as allowing an artificial increase in AJ dorsiflexion (Figure 2) due to damage suffered by the Achilles tendon and gastrocnemius muscle (i.e., equinus foot compensation). In addition, we note that the pronated foot may happen to allow a further increase (compensation) of the AJ dorsiflexion range.[41] This concept is supported by the work of Lundberg, who established that the medial column may produce more movement than the AJ itself, being a clear component of foot pronation if restriction to ankle ROM exists.[42]

Woodburn hypothesized that the reduction in walking speed is a protective mechanism related to tissue stress at the Achilles tendon.[43] This highlighted a compensation of equinus foot that would arise from RA pain and would develop with a shortening of the posterior muscle chains as the autoimmune disease evolves. Achilles tendonitis therefore requires further study in patients with RA.[44,45,46] The commonly used motion capture, gait analysis, baropodometric/pedobarographic exams can still not be used as diagnostic tests, because they are limited by inadequate inter-operator and intra-operator reliability that prevents their use to provide answers.[47]

The progressive RA foot pronation and dysfunctional calcaneus ligaments and posterior tibial tendons not only induce instability of the rearfoot joints (STJ and MTJ)[32] and rheumatoid pronated foot[8] but also block hindfoot eversion and the structured hindfoot valgus secondary to RA.[48,49,50] Among post-menopausal women with RA, Aleixo noted the development of a very "controlled" dorsiflexion sub-phase of the AJ during gait evolution, which on average, took longer than in controls.[51] Changes in lower limb biomechanics in people with RA is neither natural nor under the patient's control, but instead, represents necessary compensation for the pain and functional limitations of their disease (Figure 5).

Tenosynovitis of the peroneal, tibialis posterior, and flexor digitorum longus muscles, together with chronic edema of the ankle, can obscure the rearfoot (STJ and MTJ) syndrome,[52] which may reflect a "pronation syndrome." In the forefoot, pain and edema may instead be related to mechanical stress on the metatarsal heads, synovial hypertrophy, tenosynovitis, bursitis, or plantar hyperkeratosis in people with a long history of RA disease.[52]

The worsening joint biomechanics increases the altered kinematic strategy at the hip joint. In this "hip strategy" people with RA "drag" the leg forward in the swing phase instead of

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"pushing" it forward. AJ movement is mainly described in the sagittal plane, with deficits related to functional AJ limitations in ROM that trigger compensation (Figure 5). Thus, the gait cycle also presents oscillations in the front and transverse planes[53] that were described as embryonic in the early 1980s.[40] Movement recorded in other anatomical planes is not as useful for propulsion, which mainly, if not exclusively, takes place in the sagittal plane.[53] This suggests evidence for pathological compensation and exemplifies the waste of non-objective energy. The reduced concentric work of lower limb joints in people with RA alters joint kinetics.[54,55]

Rheumatoid ankle equinus appears to generate a change in the walking strategy and induces abnormal pronation and medialization of the STJ axis.[56,57,58] However, the rheumatoid equinus foot is not comparable to ankle equinus in healthy people: although rheumatoid equinus is caused by pain, it also persists over time, even during remission, justifying the greater permanence on the heel in the characteristic RA gait pattern.[59] Van der Leeden argues that alteration in plantar pressure distribution results when the gait evolves from physiological to shuffling.[59] Rheumatoid ankle equinus does not lead to a premature lifting of the heel, as often happens in the equinus feet of healthy people, but instead, seems to induce all the other gait compensations. Michelson provides some evidence that patients with RA have reduced AJ dorsiflexion, even in the absence of pain, reporting limited ankle movement in half of patients from RA diagnosis,[16,17] with the foot being the main problem in one-third.[18,19] This evolution of foot pathology therefore determines gait compensations and patterns (Figure 5).

Abnormal pronation in RA should be evaluated by diagnostic clinical tests.[47] Palpation tests could detect medialization of the STJ axis (i.e., deteriorated function of the supinator and anti-pronator muscles[56,60,61]) and the insufficiency/hypermobility of the first ray (i.e., deteriorated function of the long peroneal muscle and its pulley[44,45,58,61]).

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4.2. From windlass to dysfunction: the "rheumatoid shuffle"

The windlass mechanism, through hallux dorsiflexion in relaxed standing position, induces raising of the medial longitudinal arch and physiological foot supination, including external rotation of lower limb (Figure 6). This happens due to plantarflexion of the first MTJ and its insertion in the heel due to the tension induced on the plantar fascia. The mechanism, which acts through the plantar fascia, stabilizes the foot arches during walking.[62,63,64,65] Jack's test (Figure 6) is useful to determine the effectiveness and integrity of the windlass mechanism.[47] The windlass mechanism is hindered or absent in abnormal pronation, revealing a condition known as reverse windlass.[66] This will produce a bloked Jack's test where passive hallux dorsiflexion is not viable and stabilization of the medial longitudinal arch does not occur.[67] The windlass mechanism has does not appear to have been evaluated in foot RA; however, deterioration and blockage of the mechanism must be measured in each rheumatoid foot to evaluate functional status and the absence of gait pathology (abnormal pronation).[68,69] This leads to poor adaptation that induces greater gait imbalance, shorter steps, an apropulsive gait (sagittal plane deficits), an unstable posture when standing, a greater risk of falls and microtraumas during walking (due abnormal pronation), and windlass mechanism dysfunction,[67,70,71,72] leading to RA pathomechanics (Figure 5).

The apropulsive gait, sagittal plane deficits, and abnormal pronation compensations will induce functionally limited dorsiflexion of the hallux and cause a rigid hallux. This will come with an aggravation of gait deficits, risk factors, hyperkeratosis beneath the hallux, and metatarsalgia. These gait cycle alterations can be summarized with the term "rheumatoid shuffle," which has been reported in prospective studies as walking characterized by slow and creeping steps with loss of the normal sequence of contact, full support, and propulsion, and by

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an altered gait angle (small step length). The "rheumatoid shuffle" is therefore a walking pattern that is characteristic of RA, not being a real limp, but a non-fluid and non-spontaneous gait. It comprises a conscious breakdown of the step to reduce the pain, a walking rhythm that is much slower than normal, failure to lift the feet too high, and almost crawling on the ground. Each step is deliberate, slow, and usually triples the time taken for the flat path. Confronting stairs induces an even slower reduction because each step must be climbed with both feet before engaging another. Descending the stairs is often even more insecure and dangerous where the hip strategy predominates.

In the RA foot, the trend of the tibio-tarsal flexion-extension angle during the gait cycle is characterized by reduced movement in both plantarflexion and dorsiflexion. Electromyography (EMG) could show reduced anterior tibial muscle contraction and early recruitment of the gastrocnemius and long peroneus muscles in the first contact phase (i.e., heel-to-toe roll-over failure). All the gait stance phase is synthesized in one mid-stance that loses the initial contact, loading response, terminal stance, and pre-wing phases.[34,35] This walking pattern seems compatible with painful RA foot symptoms and/or mechanical deficits, performing an adequate heel-to-toe roll-over process due to the limited foot joint ROM excursion.

In abnormal pronation, we see greater EMG activity in the inverting muscles (e.g., the tibialis posterior) and lower activity in the stabilizing muscles and those that create eversion (e.g., the peroneus longus). The tibialis posterior and anterior muscles are stressed in abnormal pronation, which could explain the synovitis of the associated tendons in patients with RA. The characteristic path pattern in RA induces pathological inactivity of the tibialis anterior with little involvement of its tendon. The peroneus longus, in relation to its stabilizer role, generates a strong physiological plantarflexion action on the first ray axis (the cuboid groove, in which it

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runs, functions as an effective reflection pulley that allows action almost perpendicular to the first ray axis). In pathological pronation, we see ineffective activation due to lifting of the reflection pulley on the cuboid.[50,73,74]

If the cuboid is stable, the peroneus longus can stabilize the first ray on the ground. If the STJ hyper-pronates in the mid-stance phase, the peroneus longus loses most of its first ray plantarflexion force (when the base of the first ray is at the same height as the cuboid in the same transverse plane). In this case, hypermobility of the first ray and abnormal pronation syndrome will be observed.

4.3. External lower limb rotation: pelvic misalignment

Since 1994, many authors have written about the higher incidence of foot abduction in patients with RA.[8,48,50,53,58,75] It appears to be known that adult acquired flatfoot is usually associated with forefoot abduction.[76] To date, pelvic misalignment with arthritic foot abduction seems to be explained by compensation from abnormal rheumatoid pronation. This biomechanical evolution of the walking pattern in patients with RA can be associated with further gait compensation due to the theorized “rheumatoid abnormal pronation syndrome” model (Figure 4).

5. Discussion of compensations due to RA pathomechanics

5.1. Rheumatoid abnormal pronation and rheumatoid equinus syndromes

The rheumatoid equinus foot results from ankle equinus compensation: the pronated foot and medialization of the STJ axis allow further compensatory increase in AJ dorsiflexion range.[40] Many joints are involved in foot pronation. STJ hyper-pronation and MTJ motion/deformity

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(abnormal pronation) facilitate increased dorsiflexion of the foot to complement restricted ankle motion in the same way as the medial column increasing its sagittal plane motion. Abnormal pronation induces foot instability between the hindfoot and forefoot, causing them to share the stress under load. Thus, the pathological gait that results from an equinus foot deformity leads to compensation (abnormal pronation), trying to reduce the negative effect of ankle equinus. This compensation, along with first ray hypermobility and foot abduction, tends to induce the changing "strategy" from ankle to hip. Forefoot plantar overload is a consequence of not only rheumatoid equinus but also of the theorized rheumatoid abnormal pronation syndrome. Moreover, the rheumatoid foot has pathologic structural elements in the joints, bones, and soft tissues that make it unable to resist the tissue stresses produced by compensatory moments and motions. These numerous biomechanical factors and compensatory mechanisms are relevant to the onset of the RA foot syndrome (Figure 5).

The need for an RA foot evolution paradigm is evident. Discrepancies between instrument-based examination and foot symptoms have been observed: instruments are often unable to diagnose the precise extent of joint and soft tissue involvement in the feet of patients with RA.[77] In fact, patients may complain of ankle pain, swelling of the malleoli or back of the feet, and poor localization of signs, making it difficult to locate specific tissue structures or joints. These problems are experienced by too many patients, but the signs of these symptoms occur over time, consistent with our view of evolving biomechanical changes where pain appears first.

Among patients with disease durations <1year, individual foot joints, particularly the fifth metatarsophalangeal joint, have been shown to erode more often than individual hand joints within a year.[77] The prevalence STJ and MTJ damage in RA was reported by Vainio in 1956, with

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STJ, talonavicular, and calcaneocuboid joint involvement in 70% of patients, underestimating the true value, which is close to 100% at 3–5 years after diagnosis.[19,32]

5.2. Therapeutic anti-pronation shoes and exercises for preventing foot RA

At present, the role of footwear is confined to containing the foot deformity and holding the foot. In our opinion, however, footwear should be designed to prevent and rehabilitate biomechanical function to correct the theorized RA pathomechanics in this review. Estimating walking energy expenditure is essential for evaluating the gait in RA, and Kavlak observed that orthotic foot therapy and therapeutic shoes can provide shock absorption, pain relief, comfort, and a more efficient gait with increased stride length and walking speed.[55] It is our opinion that therapeutic shoes should always be geared toward anti-pronation running shoes (A3 or A4 classification) in an effort to stem the rheumatoid abnormal pronation syndrome and its compensations from the diagnosis of RA. This thesis must be evaluated by good quality clinical trials.

Another therapeutic path to be explored is rehabilitation therapy. Kinetics and kinematics exercise appear to improve the gait in people with RA pathology, with training programs able to improve the quality of walking and prevent falls.[51] Numerous clinical trials have reported improvements in muscle strength, physical function, and aerobic capacity with dynamic exercises.[78,79] Some papers have even shown significant reductions in the progression of foot joint radiographic damage after dynamic exercise,[80] while others have also reported a slowing of disease activity and small improvements in hip bone mineral density. No studies have reported worse outcomes for function, disease activity, or aerobic capacity with dynamic exercise. Most patients with RA should be encouraged to undertake dynamic and/or strength training exercises.[81,82,83,84]

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Many lower limb disorders are related to calf muscle tension and reduced ankle dorsiflexion. Treatment typically involves stretching the calf muscles to increase dorsiflexion at the AJ, which has been demonstrated in meta-analysis reviews. Further trials are required to determine how much the calf muscles and hamstrings lengthen, together with how effective these exercises are in reducing pain and increasing walking autonomy in patients with RA. Despite the evidence that is already emerging, entry to this therapeutic path is still late in the RA guidelines, also to stem the risk of falls.[85,86,87] Moreover, good quality clinical trials are needed to determine how much the lengthening of these muscles, when performed regularly (e.g., biweekly) from RA diagnosis, improves the gait pattern, rheumatoid AJ equinus, rheumatoid abnormal pronation, rheumatoid shuffling, plantar overload, and fall risk. It is possible that a regime of stretching the posterior chains by dynamic exercise may stably increase the ROM of the ankle and knee in patients with RA.

Perhaps the most surprisingly overlooked aspect is the effect of dynamic exercise on cardiovascular outcomes (e.g., coronary heart disease and stroke) and important risk factors (e.g., hypertension, dyslipidemia, obesity, and diabetes). Since excess cardiovascular mortality is the primary cause of reduced life expectancy in RA, interventions to reduce cardiovascular disease and risk factors by taking better care of the lower limb could be vital.[88]

6. Conclusions

This review has described a model that predicts the evolution of lower limb pathomechanics in patients with RA, although to date, this paradigm and the proposed therapeutic hypotheses still need to be tested in clinical trials. If rheumatoid equinus syndrome, rheumatoid abnormal

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pronation syndrome, and the rheumatoid shuffle syndrome are tested successfully, they could characterize a rule valid for all rheumatoid patients from the diagnosis. The goal is to reduce morbidity in this complex group of patients by considering the impact of tissue stress on generating lower limb dysfunction. RA is an inflammatory disease that can significantly affect joint structure, deformity, and pain in the foot, with resulting gait patterns that influence posture and biomechanics despite current pharmacological treatment. However, we could go further still: modifying the biomechanical deficits of the foot could reduce the incidence of foot pain and the consequences associated with RA. Joint damage and muscle weakness are directly associated with the severity of inflammatory synovitis, but they do not independently affect foot or ankle kinematics. Although conservative treatment based on a better understanding of foot biomechanics may not alter the inflammatory process, it could reduce the pathological effects. RA not only alters gait biomechanics in RA but also complicates any pre-existing lower limb biomechanical dysfunction that precedes the diagnosis of RA.

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Conflict of interest

The authors declare the absence of any conflicts of interest.

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Table 1. The queries used in the paper project work.

(Equinus OR ankle dorsiflex*) Rheumatoid,
ankle (equin* OR tightness) (gastrocnemius OR ((plantar fascia) OR (plantar aponeurosis))),
Windlass (("flat foot") OR flatfoot OR (pes plano*) OR pesplano* OR pronat*),
Windlass Mechanism,
Reverse Windlass,
medialization axis (STJ OR (subtalar joint)),
(Arthritis OR Rheumatoid OR rheumatic) shuffl*,
stress syndrome (*synovitis OR (bone marrow oedema)),
Arthritis Rheumatoid AND “the following queries”
“biomechanic*”,
“(("flat foot") OR flatfoot OR (pes plano*) OR pesplano* OR pronat*)”,
“(STJ OR (subtalar joint)) NOT(Surger*)”,
“(aponeuros* OR Achilles OR (plantar fascia)) (biomechanic* OR gait OR foot OR (walking* strategy))”,
“(aponeuros* OR Achilles OR (plantar fascia) OR tendon*)”,
“(biomechanic* OR gait OR (walking* strategy))”,
“Fasciitis Plantar”,
“(equin* OR ("limited joint")) foot (aging OR ageing OR aged)”,
“hallux (valgus OR alignment*) (epidemiolog* OR prevalence OR incidence OR etiology* OR (cross sectional) OR Cohort OR Case-control)”,
“(first-ray OR "first ray") (mobility OR instability OR biomechanic*)”,
“(orthosi* OR offload*)”,
“(aponeuros* OR Achilles OR (plantar fascia)) (Strength* OR stretch* OR Exercise)”,
“(heavy OR mass) shoe biomechanic*”,
“biomechanic* (Kinet* OR kinem*) NOT(surger*)”,
“(gait OR biomechanic* OR (walking* strategy)) NOT(surger*)”,
“((Lower limb) OR foot OR feet OR gait OR biomechanic* OR (walking* strategy)) (electromyograph* OR EMG) NOT(surger*)”,
“pain foot”,
“valgus (deformit* OR rearfoot OR (rear foot)) foot”,

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“team (podiatry OR foot)”,
“Multidisciplinary foot”,
“((limb rotation) OR (pelvic misalignment))”,
“(tenosynovitis OR Synovitis) (foot OR feet OR leg)”,
“muscle* (foot OR feet OR leg) (epidemiolog* OR prevalence OR incidence OR (cross sectional) OR Cohort OR Case-control) NOT(surgery)”,
“((rearfoot eversion) OR valgus)”,
“navicular height”,
“tibialis posterior tendon”,
“((peroneus longus tendon) OR (cuboid pulley))”,
“Achilles tendon”,
“Foot function”,
“energy (foot OR feet OR leg OR step)”,
“(Strength* OR stretch* OR Exercise) (gait OR biomechanic* OR (walking* strategy))”,
“(Strength* OR stretch* OR Exercise) muscle* (foot OR feet OR leg)”,
“foot hand (*synoviti* OR (joint deformities) OR (bone marrow edema))”.

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Figure 1
The Bojsen-Moller theory and its two-propulsion axis: transverse (as a high gear) and oblique (as a low gear). Perpendicular bisections, from each axis to heel, show different lengths. The longer one is more able to create great trust for propulsion than shorter one, and more tarsus stability by calcaneocuboid joint close-packed position. We theorize that the RA foot doesn't use the long arm lever but a lever between the short and long lever in the RA pathomechanics gait pattern.

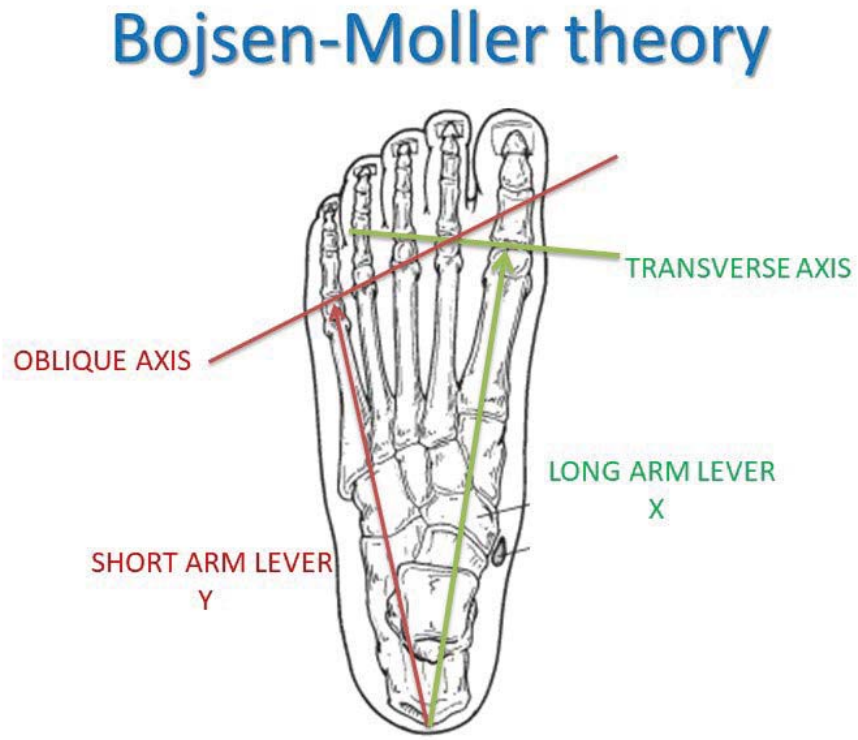

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Figure 2
The Flexed knees are a compensation of the posterior muscle chains shortening: it is a characteristic of equinus foot compensation.

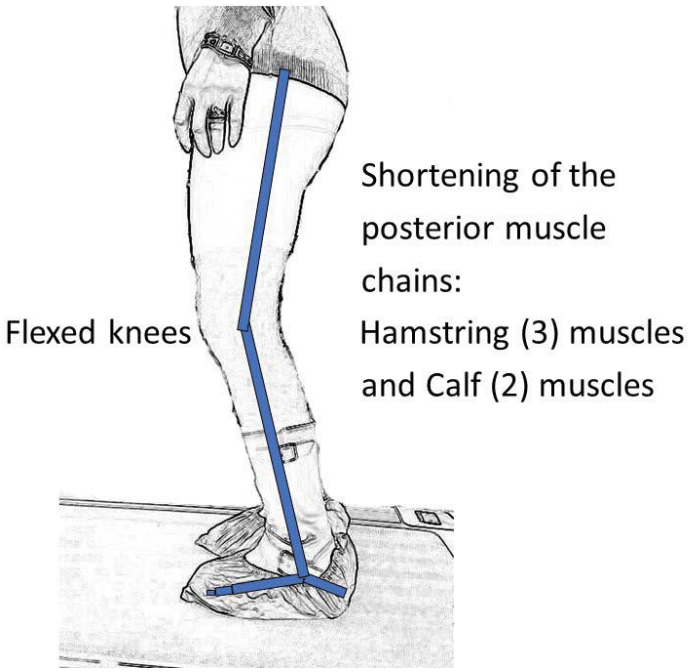

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Figure 3
The Apropulsive gait is named “Sagittal plane deficit”, “Frontal oscillation” and “Swinging gait”: it is a characteristic of pathological pronation compensation.

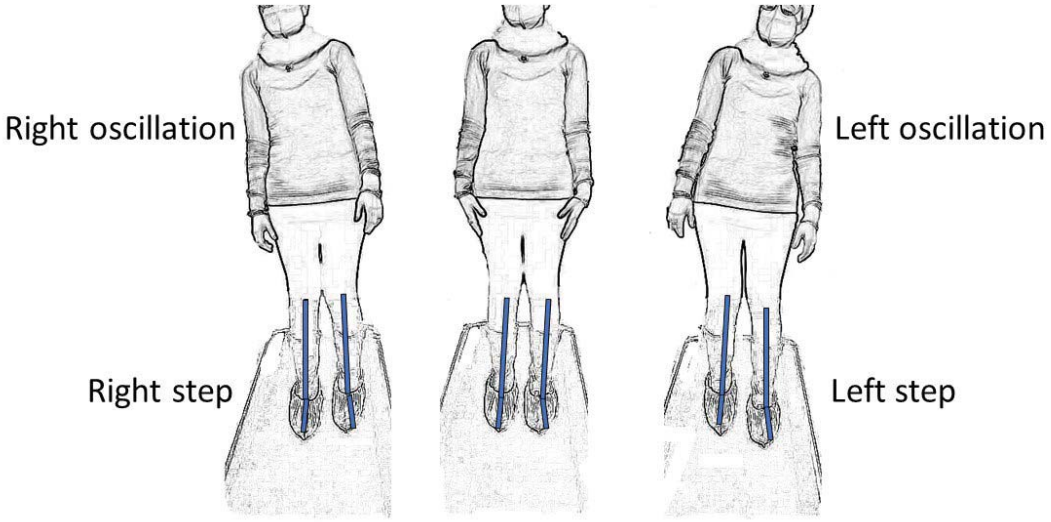

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Figure 4
The external rotation of the lower limbs is called "pelvic misalignment" and "abducted feet": it is a characteristic of pathological pronation compensation.

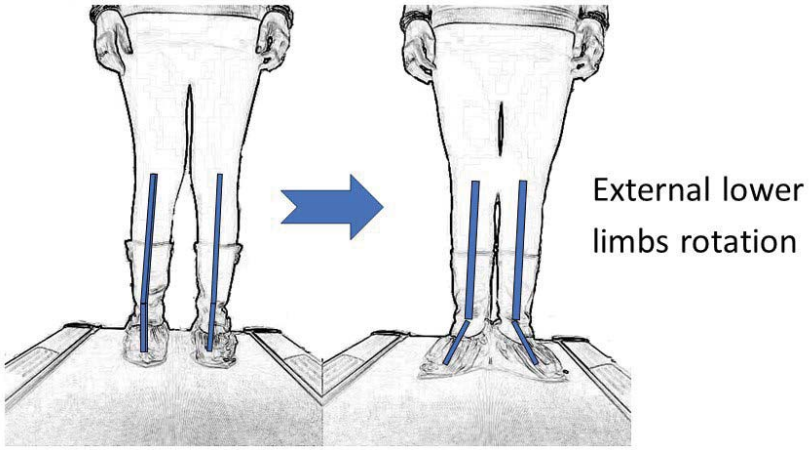

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Figure 5 (next page)
Venn's diagram of the "Rheumatoid Abnormal Pronation Syndrome" theory.

The "RA patients" set contains all the RA foot compensations subsets.

The "RA patients" set contains the following compensations, starting from diagnosys: "a) RA equinus syndrome", "b) RA pronation syndrome", "c) Windlass mechanism deficit".

All the RA foot compensation subsets d), e), f), g), h) are generated from the "RA equinus syndrome".

These RA compensation subsets hypothesis is here represented although these have to be proven. The true prevalence magnitude of the individual subsets is not known yet.

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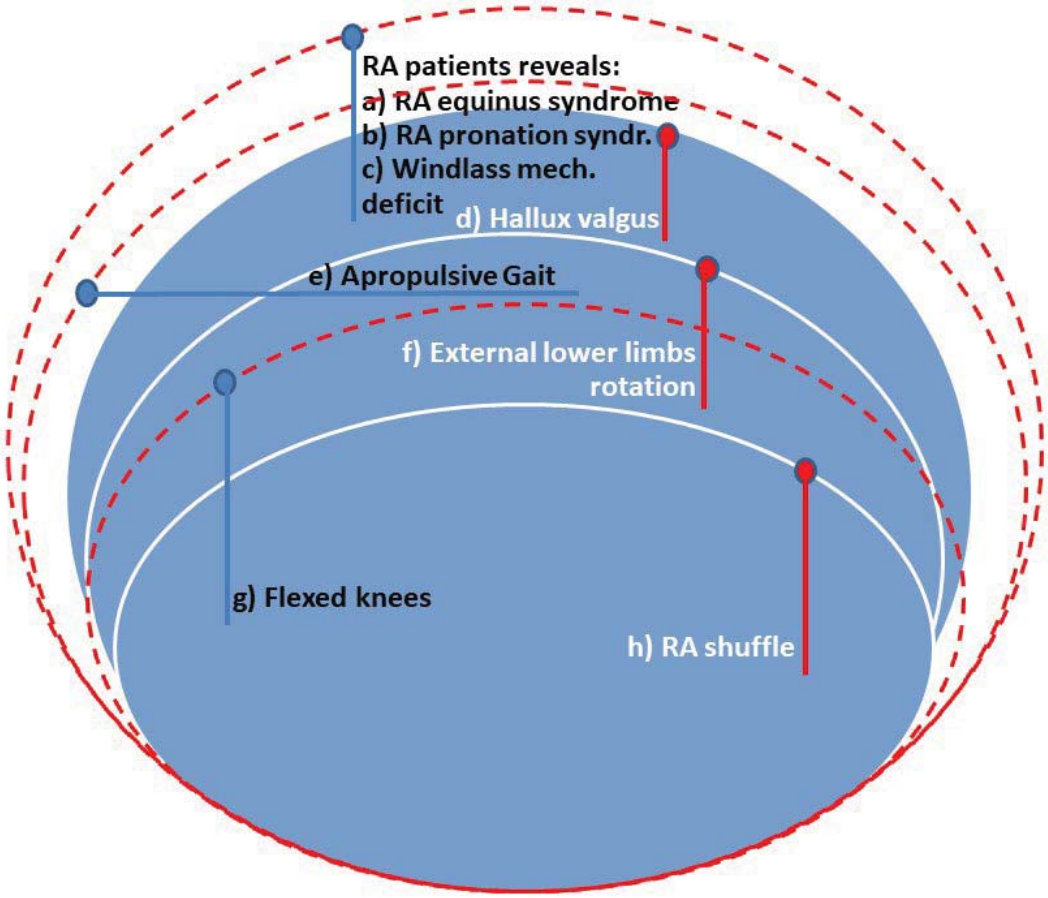

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Figure 6
The Jack's test is a clinical test claimed to be an estimation of the efficacy and integrity of the windlass mechanism.