INTRODUCTION

Gait analysis is the study of human walking. The walking pattern of patients with rheumatoid arthritis (RA) has been described as one that is slow and modified to lessen pain, and features changes to the pattern and range of joint motion, altered muscle activity and stress distribution to the plantar region. As clinicians, we get the opportunity to observe the patient walking as they enter the consulting room, but too often this is brief and unrewarding as well as impractical as the lower limb and foot is often obscured by clothing and footwear. A formal qualitative approach is recommended as part of the GALS locomotor screening system (see Chapter 4) and this is helpful, but this chapter covers quantitative gait analysis using instrumentation in a laboratory setting. In Leeds, over the past few years, we have been developing a gait analysis ‘toolkit’ to permit analysis of the lower limb and specifically the foot. Our laboratory, shown in Figure 2.1, comprises a short flat 10 m walkway and our ‘toolkit’ contains an instrumented walkway to measure the basic spatial and temporal parameters of gait; force and pressure plates are embedded into the floor to measure those quantities and an arrangement of six cameras record motion.

Gait analysis is carried out following referral from the multidisciplinary team that may include:

- The podiatrist requesting plantar pressure measurement to assist the design of a custom off-loading foot orthosis for an RA patient with forefoot ulceration.
- The physicians may request gait analysis to help explain foot mechanics for a patient with persistent unresolved tibialis posterior tenosynovitis after recognizing the foot was pronated.
The orthopaedic surgeon, planning a total ankle replacement, may request an analysis of the ankle 3D kinematics and kinetics as a baseline to evaluate prospectively any restoration of ankle function.

Finally, the orthotist may request simple spatial and temporal measurements to check the gait symmetry when prescribing orthopaedic footwear and orthoses.

Our laboratory strategy uses gait analysis to understand more fully the relationship between inflammatory joint disease, impairment to foot structure and function and the compensatory gait strategies patients adopt to overcome painful and disabling deformity. Gait analysis is, therefore, aided when we have prior knowledge of the nature and severity of foot/lower limb impairments, and the general and localized inflammatory status. It adds to our patient history, extends our clinical examination by quantifying joint function during activities of daily living and complements other investigations, particularly imaging that aims to localize and quantify joint pathology and impairment. Indeed, there are only a handful of gait-related publications in RA and the most useful are those that explore these relationships, albeit in small study sizes (Siegel et al. 1995, O’Connell 1998).

Applying gait analysis techniques to the study of foot function is challenging. These challenges include elements of the following dilemmas:

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Figure 2.1  Gait Analysis Laboratory, Academic Unit of Musculoskeletal Disease, University of Leeds. Situated in the Rheumatology Outpatient Department the laboratory comprises (1) six-camera motion capture system; (2) plantar pressure distribution analysis system; (3) force plate; (4) computer workstations. Not shown are optical timing device, in-shoe pressure analysis system and instrumented walkway.
Past studies have modelled the foot as a single rigid body that tells us little about the small, interdependent and functionally important small joints of the foot.

The disease itself presents problems: in our kina- matic foot model, anatomical landmarks, where tracking motion markers are placed, can often be obscured by localized joint or soft-tissue swelling introducing error to our motion calculations.

Most techniques require barefoot walking, something patients with RA and painful feet rarely do, so it is difficult to verify the true walking pattern in some cases.

These problems are not insurmountable and we have already introduced multi-segment foot models to study functional groups of small joints, employed ultrasound to measure localized swelling to improve our marker site placement error and measure, where possible, patients shod and barefoot.

Critics of gait analysis often cite the length of time to capture and process data and the challenge of deciphering multiple variables at multiple joint sites in 2- or 3D. Our laboratory employs techniques that mostly automate these processes so we are now in line, in terms of time, with, for example, magnetic resonance imaging (MRI) as a special investigation. As you will see throughout the chapter and from the CD, software visualization of motion from rendered models enables better gait interpretation and clinical reports can be easily standardized. Future challenges include establishing normal values for the common gait parameters measured in relationship to the gender and age distribution of the patients we see with RA, and to introduce prospective studies that enable predictive and prognostic gait variables to be identified with regard to their relationship with underlying disease processes and foot impairment. Finally, continued development of these techniques as potential outcome measures for clinical trial is required (Fransen et al. 1997, Woodburn et al. 2003).

Approximately 90% of our gait laboratory workload is clinical research and we are aware that gait analysis is not commonly undertaken in rheumatology centres. For that reason, this chapter will aim to briefly describe the gait analysis techniques used in Leeds, outline the application to foot disease in RA, assimilate the research evidence and present appropriate case histories to highlight selected areas. Throughout the chapter, abnormal gait features will be related to underlying pathological processes, primarily inflammation and foot anatomy and biomechanics.

KEY POINTS

- Gait analysis is the systematic study of human walking
- Useful parameters to measure in RA include spatial temporal features, 3D joint kinematics and kinetics and plantar pressure distribution
- Given the complex anatomy, the application of gait analysis techniques in the foot is challenging
- Gait analysis can be used alongside the clinical history, examination and other special investigations to gain a better understanding of the relationship between inflammatory joint disease, impairment and compensatory gait mechanisms.

GAIT ANALYSIS TECHNIQUES AND THEIR APPLICATION IN RHEUMATOID ARTHRITIS

There are a number of gait analysis techniques that have been previously employed to study foot function in RA. Over time, these techniques have evolved from 2- to 3D analyses, with data integrated to provide simultaneous measurement of joint angles, electromyographic muscle activity signals and foot pressure distribution, for example. Foot models have increased in their complexity (Woodburn et al. 2004). It is beyond the scope of this book to describe all of these techniques and we focus on those currently employed in our own laboratory. Suggestions for further reading are provided at the end of the chapter.

Observational gait analysis

Observational gait analysis is a qualitative visual description of human walking. It forms part of the GALS system described earlier in the book. Our approach is to observe the patient on the walkway over several trials noting major functional deficits in a systematic way from the head to foot. Initial impressions include walking speed, symmetry, balance, and protective mechanisms for painful joints. We note the posture and movement for the head, neck and shoulders and arm swing through the walking sequence. We observe spine, hip, knee and ankle motion. For the foot we comment on the movement between the rearfoot relative to the leg and the forefoot relative to the rearfoot and the rise and fall of the medial longitudinal arch during the stance phase. We note the position of the hallux and
the weight-bearing capacity of the lesser toes. We classify each defect by predominant direction of motion, e.g. flexed/extended or varus/valgus, and quality of the range of motion on a nominal scale – hypermobile, within-normal-limits, stiff, rigid. These observations are made in the sagittal and frontal planes with the patient barefoot and in their current shoes, including any orthoses, or using normal assistive gait devices. Care should be taken to avoid a prolonged session as comorbidities such as cardiovascular disease may impair valid observations.

In our patients, we regularly observe slow antalgic gait patterns which are asymmetrical and feature motion defects at a number of lower limb joints and the feet, in a variety of patient specific patterns. We regularly note wide arm swing to aid balance, and a forward-tilted head where the patient, keen to avoid painful obstacles, checks the path immediately ahead. The ‘rheumatoid shuffling gait’ has been well-described and is fairly easy to characterize as will be seen later.

As an aid to observational gait analysis, many groups record walking on video media and use slow motion playback to facilitate the qualitative analysis. This can be time consuming and lacks precision and reliability. Our approach is to record video during our instrumented gait analysis because our motion analysis software allows us to synchronize the two formats so we can combine at once our gait metrics with the direct observations. Discordance is common so it is ensured that the quantitative 3D data drives our interpretation of the observed 2D video images. Foot motion appears too complex and occurs too quickly about small ranges of motion to rely on video alone.

Spatial and temporal gait parameters

The overall gait style can first be considered by its basic spatial (distance) and temporal (timing) parameters. Historically, a single temporal parameter – the 50-foot walking time – featured as an outcome measure in over one-quarter of therapeutic trials of anti-rheumatic drugs in the mid 1970–80s (Grace et al. 1988). However, the test lacked responsiveness; only 41% of studies detected statistically significant differences, with a mean difference of only 2 s improvement, mostly of interventions tested for 6 weeks or less. Despite respectable reliability, this simple temporal measure lost favour as disease driven outcomes were established and the HAQ became the gold standard functional outcome. Nevertheless, Fransen and Edmonds (1999) revived interest in these gait parameters as outcomes for therapeutic trials, this time for orthopaedic footwear in rheumatoid arthritis, implementing electronic timing and footswitches to record walking speed, cadence and stride length (Fransen & Edmonds 1999). When shortened to three averaged trials of 8 m, these variables were highly reliable and responsive. These shorter walking distances are much simpler to measure and have the benefit of reducing the co-morbid effects of cardiovascular and pulmonary disease and minimizing fatigue.

Tethered electric footswitch systems, video analysis and pencil and paper exercises where sequential chalked or inked footprints are measured by hand, are amongst the techniques used to measure spatial and temporal gait parameters. Some are manually timed, cumbersome and time-consuming, whilst others involve tethered devices and body-placed sensors. Indeed, the latter require patients to adapt to the techniques and Fransen further concluded that for therapeutic trials two assessment sessions were necessary for a superior baseline (Fransen & Edmonds 1999).

In the modern gait laboratory, there is now a preference for instrumented walkways; solid or portable mat walkways with grids of embedded pressure sensors that record each footfall and automatically calculate and display spatial and temporal parameters via dedicated computer software. Algorithms permit analysis of standard parameters, such as walking speed, cadence, cycle-time, stride length and double-support, and other features such as the timing of stance and swing as a percentage of the gait cycle, toe-in/out angles (angle of gait) and heel-to-heel distance (base of gait). Walkway systems have the advantage of portability and can measure patients

KEY POINTS

- Observational gait analysis is a qualitative visual description of human walking
- Initial impressions in our RA patients can be gained for walking speed, symmetry, balance, and compensatory antalgic patterns including limp and the shuffling gait
- In RA patients we systematically observe alignment and motion in the upper and lower limbs and spine in the frontal and sagittal planes
- Video recording facilitates review and can be integrated with quantitative techniques.
barefoot and shod and using assistive walking devices. Independent evaluation shows they are valid and reliable tools for clinical gait analysis (McDonough et al. 2001, Bilney et al. 2003).

Looking at the footfall pattern in the CD and depicted diagrammatically in Figure 2.2, we can delineate the sequence by periods when the foot is in ground contact (stance phase), when the foot is airborne (swing phase) and when both feet are on the ground (double-support time). These variables can be represented in absolute units (s) or as a percentage of the time taken for one complete gait cycle. The number of steps taken over the measurement period can be counted and cadence established and if the time and distance measurements are known, average walking speed can be calculated. Five key variables, routinely reported in studies of people with rheumatoid arthritis, are defined in Table 2.1 and summary data presented for a cross-sectional sample of patients and a cohort of able-bodied subjects. Our data support that of others showing that patients with rheumatoid arthritis typically walk at a slower speed with a lower step rate and a longer cycle time. Stride length is shorter and the double-support period lengthened. These changes are associated with a number of disease related factors including impairments in the lower limb and foot, primarily pain, stiffness and deformity. Consider the following case.

---

![Figure 2.2](image)

**Table 2.1** Definition of five spatial and temporal gait parameters used in rheumatoid arthritis (RA) gait analysis and mean (SD) data for able-bodied subjects and RA patients

<table>
<thead>
<tr>
<th>Variable (unit of measurement)</th>
<th>Definition</th>
<th>Typical findings in RA</th>
<th>Able-bodied[^1] (n=45)</th>
<th>RA[^2] (n=40)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walking speed (m/s)</td>
<td>Distance per unit of time</td>
<td>Slow</td>
<td>1.26 (0.15)</td>
<td>0.88 (0.23)</td>
</tr>
<tr>
<td>Cadence (number of steps per min)</td>
<td>Step rate per min</td>
<td>Reduced</td>
<td>115 (9)</td>
<td>103 (10)</td>
</tr>
<tr>
<td>Cycle time (s)</td>
<td>The elapsed time between the first contact of two consecutive footfalls of the same foot</td>
<td>Longer</td>
<td>1.05 (0.08)</td>
<td>1.17 (0.12)</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>The distance between the sequential points of initial contact by the same foot</td>
<td>Shorter</td>
<td>1.33 (0.13)</td>
<td>1.03 (0.22)</td>
</tr>
<tr>
<td>Double-support time (% of gait cycle)</td>
<td>Part of the gait cycle characterized by both feet on the ground simultaneously. There are two double-support periods during one gait cycle</td>
<td>Longer</td>
<td>16.5 (2.9)</td>
<td>21.4 (5.4)</td>
</tr>
</tbody>
</table>

[^1]: Forty-five able-bodied control subjects (20 males/25 females) with a mean age of 54.9 years (SD 11.9) and[^2]: forty RA patients (9 male and 31 female), with a mean age of 56.2 years (SD 12.7) and a mean disease duration of 13.7 years (SD 10.9) (unpublished data from the University of Leeds).
**Case study**

This 55-year-old female patient with 18 years' disease duration underwent gait analysis in 2001. She presented with marked stiffness and 15° of fixed flexion deformity in the right knee and extensive impairment in the forefoot including pain and stiffness from severely eroded MTP joints. Observing the overall footfall pattern (Figure 2.3) we can see that the gait pattern is asymmetrical with stride-to-stride variability and lateral drift from the forward line of progression. Her spatial-temporal parameters are abnormal (Table 2.2, column A). Three years later her gait parameters remain unchanged against a background history of quiescent disease and stable drug management (Table 2.2, column B). She underwent an intensive footcare programme including orthopaedic footwear, customized foot orthoses and physical therapy. One month later (Table 2.2, column C) we note good improvement in her walking parameters, which were associated with self-reported improvements in pain and stiffness.

The case reported above highlights a number of important relationships between gait parameters, disease activity and impairment of structure and function in the lower limb and foot. Pain is perhaps the most significant factor related to altered gait function and so we may expect changes not only in well-established disease as highlighted above, but in early disease as well. The relationship is complex but interesting to study in individual cases and five are presented in Table 2.3 to illustrate this point:

![Figure 2.3](image)

*Figure 2.3* Overall footfall pattern from one pass on the instrumented walkway.

### Table 2.2 Spatial and temporal gait parameters for a rheumatoid arthritis patient measured on three occasions over a 36-month period.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Walking speed (m/s)</td>
<td>0.78</td>
<td>0.77</td>
<td>0.93</td>
<td>21% faster walking speed</td>
</tr>
<tr>
<td>Cadence (steps/min)</td>
<td>101</td>
<td>100</td>
<td>108</td>
<td>8% increased step rate</td>
</tr>
<tr>
<td>Cycle time (s)</td>
<td>1.18</td>
<td>1.20</td>
<td>1.11</td>
<td>8% shorter cycle time</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>0.92</td>
<td>0.92</td>
<td>1.06</td>
<td>15% longer stride length</td>
</tr>
<tr>
<td>Double-support (% gait cycle)</td>
<td>18.9</td>
<td>17.5</td>
<td>15.1</td>
<td>14% reduction in double-support time</td>
</tr>
</tbody>
</table>

### Table 2.3 Summary demographic, disease, foot impairment and gait parameters from five selected rheumatoid arthritis cases of different disease duration.

<table>
<thead>
<tr>
<th>Patient/sex/age/ Disease duration (years)</th>
<th>DAS (2–10)</th>
<th>LFIS (0–51)</th>
<th>Foot deformity (0–19)</th>
<th>SJC (0–14)</th>
<th>TJC (0–14)</th>
<th>Speed (m/s)</th>
<th>Cadence (steps/min)</th>
<th>Cycle time (s)</th>
<th>Stride length (m)</th>
<th>Double-support time (% gait cycle)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/43</td>
<td>&lt;1</td>
<td>7.29</td>
<td>33</td>
<td>0</td>
<td>14</td>
<td>14</td>
<td>0.86</td>
<td>95</td>
<td>1.27</td>
<td>1.09</td>
</tr>
<tr>
<td>2/F/29</td>
<td>&lt;1</td>
<td>4.13</td>
<td>19</td>
<td>0</td>
<td>1</td>
<td>5</td>
<td>1.30</td>
<td>122</td>
<td>0.98</td>
<td>1.29</td>
</tr>
<tr>
<td>3/M/72</td>
<td>5</td>
<td>5.73</td>
<td>35</td>
<td>11</td>
<td>12</td>
<td>12</td>
<td>0.54</td>
<td>96</td>
<td>1.26</td>
<td>0.68</td>
</tr>
<tr>
<td>4/F/60</td>
<td>7</td>
<td>3.49</td>
<td>13</td>
<td>11</td>
<td>12</td>
<td>6</td>
<td>0.63</td>
<td>85</td>
<td>1.42</td>
<td>0.90</td>
</tr>
<tr>
<td>5/F/53</td>
<td>14</td>
<td>5.48</td>
<td>47</td>
<td>13</td>
<td>8</td>
<td>8</td>
<td>0.90</td>
<td>115</td>
<td>1.05</td>
<td>0.94</td>
</tr>
</tbody>
</table>

DAS: Disease Activity Score; LFIS: Leeds Foot Impact Scale; SJC: Swollen Joint Count for the foot; TJC: Tender Joint Count for the foot.
Pathomechanics and the application of gait analysis in rheumatoid arthritis

Patient 1 is a newly diagnosed case who has just started DMARD therapy. Her disease activity is high (elevated DAS) and locally very active in the feet (high number of swollen and tender foot joints) with marked foot impairment (high Leeds Foot Impact Scale [LFIS] score), but no deformity noted. Her inflammatory status and pain drive the functional changes characterized by slow walking speed, reduced cadence and stride length, and longer cycle time and double-support phase. By contrast, patient 2, a younger female patient who has started her DMARD therapy, has less disease activity, less foot impairment, fewer tender or swollen foot joints and also no foot deformity. Her gait parameters are well within normal limits. Patient 3 is an older male RA patient entering disease flare, determined by a high DAS score (5.73) and a high number of swollen and tender joints, with active disease in the feet. He also has significant foot impairments as identified by his high LFIS score and his forefeet are markedly deformed. These combinations of factors give rise to the classic shuffling gait of RA characterized by very slow walking speed, the very short stride length and the very long double-support phase. By contrast, patient 4 is a 60-year-old female patient with severe and painful forefoot deformity. Her disease was relatively quiescent and only her MTP joints were tender on palpation, but not swollen. She reported a ‘guarded’ gait to avoid walking on her MTP joints hence the long double support time, short stride and slow walking speed. So, although she was tender on examination, her foot impact scores were low and we conclude that this may be the result of her compensatory antalgic gait. Finally, patient 5 represents a typical female RA patient with long-standing disease who has both active disease, marked rigid foot deformity at which a high number of joints are swollen and tender and high self-reported foot-related impairment and disability. Her gait parameters are abnormal yet not as marked as some of the other patients. She has slowed her walking down, shortened her stride length with a subsequent increase in contact time but still maintains a normal step rate.

These cases serve to illustrate the complex relationship that exists between disease related factors, impairment and the basic spatial and temporal parameters of gait. Disease duration alone is not predictive of change as we can see from Figure 2.4, a trend towards decreased walking speed and stride length with increased disease duration. However, the relationship is not strong with a correlation coefficient of around 0.4. When grouped by the LFIS scores, no differentiating clusters emerged so foot impairment may have a limited effect. Intuitively, change in basic gait parameters are probably influenced by factors such as disease activity, age, impairment, co-morbid disease and proximal limb joint involvement amongst others.

Across a range of studies where patient cohorts have differed by disease duration, impairment and disability, spatial and temporal gait parameters in RA are consistently reported as abnormal. For example, Isacson and Brostrom (1988) studied 17 female RA patients less than 50 years of age, with average disease duration of 17 years and found the mean velocity to be

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**Figure 2.4** The relationship between disease duration and walking speed (A) and stride length (B) for 71 rheumatoid arthritis cases. Individual cases are identified by severity of foot impact as determined by the Leeds Foot Impact Scale where 1=mild impact, 2=moderate impact and 3=severe impact (University of Leeds, unpublished data).
0.6 m/s, stride length 0.9 m and gait cycle duration 1.4 s. These findings are typical and as a general rule these gait parameters fall between 50 and 90% of normal values when adjusted for age and sex (Locke et al. 1984, Isacson & Brostrom 1988, Kennan et al. 1991, Platto et al. 1991, O’Connell et al. 1998, Fransen & Edmonds 1999).

The association between specific foot impairments and gait parameters has also been investigated. In the rearfoot, Locke and colleagues (1984) demonstrated that five patients with isolated and untreated ankle and subtalar joint pain had a walking speed of 0.69 m/s and a single limb support time at 72% of normal (Locke et al. 1984). Hindfoot pain and deformity are important in rheumatoid arthritis and Platto et al. (1991) showed stronger correlations between these impairments and gait parameters, including speed, stride length, double-support and cadence, than those for the forefoot. Keenan et al. (1991) supported these observations in a study of 10 patients with pes planovalgus confirmed by standard radiographic measurements. They found walking speed and cadence to be 51% and 84% of normal, respectively, whilst the stride length was 0.86 m. These cases had long-standing disease averaging 25 years, but matched to a similar group (with rheumatoid arthritis) by age and sex without flatfoot deformity, changes to gait parameters were markedly worse. We have also studied 23 patients with earlier disease averaging 7 years who had acquired pes planovalgus and found gait cycle times longer on average by 0.15 s, stride length shorter by 0.31 m, double-support times increased by 8.1%, speed slower by 0.39 m/s and cadence reduced by 17 steps/min in comparison with normal (Turner et al. 2003).

Impairments associated with forefoot disease in RA are well documented and O’Connell’s group measured the gait of 10 patients with symptomatic disease (O’Connell et al. 1998). Using the Sickness Impact Profile subsection for ambulation they showed a strong correlation between increasing disability and reduced walking speed ($r = -0.74$) and stride length ($r = -0.72$). In this middle-aged group with average disease duration of 12 years, the average walking speed (0.97 m/s) was 71% of normal; stride length (1.06 m) 77% of normal and cadence (112 steps/min) 96% of normal.

Spatial and temporal gait parameters have featured as the main gait outcome in around eight intervention studies. The trend is towards short-term improvements across all the gait variables and this is summarized for walking speed in Figure 2.5. In the majority of these studies, relief of pain and foot-related disability accompanied improved walking parameters. However, no generalizable conclusions can be drawn because of the variation in the nature and conduct of each study. Nevertheless, the overall trend is encouraging since these interventions are often adjunct to systemic treatment as well as other ongoing therapy and educational care. These small-to-medium effects are well recognized in rehabilitation therapy (Ottenbacher 1989).
Joint kinematics

Joint kinematics describes the relative motion between two adjacent bones, but ignores the causes of that motion. Kinematic measurement allows us to quantify the range and pattern of motion during gait. In RA, since we know repeated episodes of synovitis weaken and eventually destroy joints, changes in motion parameters should be expected and these changes may be associated with laxity in early disease and stiffness and deformity later. Furthermore, we know patients develop antalgic gait patterns and may position and hold joints in a certain pose to lessen symptoms and this compensatory strategy may also be characterized by joint kinematics. However, it is widely acknowledged that measuring joint kinematics in the foot is challenging: a combination of the complex anatomy and the technical limitation of measurement systems.

To briefly explain, in Figure 2.6, a severely deformed foot is presented (Fig. 2.6A) and we must consider how to define the joints of interest. The measurement system uses passive retroreflective markers whose positions are tracked by cameras as the patient walks. Three markers are required to track each segment to allow 3D measurement and others markers are required to define specific landmarks to enable the geometry of the segment to be defined. It is clear from the size of the markers, the geometry and tight packing of the foot bones that studying, for example, the small inter-tarsal joint would be extremely difficult (Fig. 2.6B). Furthermore, bones such as the talus are locked in the ankle mortise and have inaccessible surface landmarks to place skin sensors or markers. To overcome this, we combine groups of bones into larger and more accessible segments with relevant functional meaning. In our case (Fig. 2.6C), we have created segments for the shank, rearfoot, forefoot and hallux.

Because of these limitations, only a small handful of studies have reported kinematic parameters from the foot in RA (Table 2.4). In six RA patients with

**Figure 2.6** (A) severe foot deformity in rheumatoid arthritis; (B) skeleton foot model showing anatomical landmarks for skin-surface markers and tracking markers for a typical multi-segment kinematic foot model used in rheumatoid arthritis; (C) the same patient with segments (geometry defined by cones) for the shank, rearfoot, forefoot and hallux segments from standing foot pose.
advanced subtalar disease and pes planovalgus deformity, Marshall et al. (1980) detected a more plantarflexed foot prior to ground contact to assist the foot in landing flat accompanied by prolonged ankle dorsiflexion and delayed heel-rise. These sagittal plane features about the ankle joint were consistent with a slow shuffling gait and prolonged double-support, mechanisms thought to lessen pain and enhance stability. Similarly, Locke et al. (1984) showed that increased dorsiflexion was accompanied by more valgus (eversion) motion in the frontal plane during stance in five patients with painful ankle and hindfoot joints.

In pes planovalgus, these abnormal motion patterns are consistent with the observed deformity and we confirmed these early findings in larger cohorts with early and more flexible foot deformity (Woodburn et al. 1999, 2002, Turner et al. 2003). Furthermore, we undertook 3D measurements and were, therefore, able to demonstrate the coupled motion pattern between excessive ankle joint complex eversion and internal leg rotation (Woodburn et al. 2002, Turner et al. 2003) (Fig. 2.7). In all of these studies, however, measurement is restricted to the ankle joint or the ankle joint complex (ankle and subtalar joint) using active marker systems based on electrogoniometry or electromagnetic tracking.

The utility of gait analysis has been well demonstrated by the National Institutes of Health group in the USA (Siegel et al. 1995, O’Connell et al. 1998). Starting with a single rigid body model of the foot, they were able to relate forefoot disease to altered foot function and separate two cases by severity. The contrast in foot function between cases with near-rigid hindfoot varus and flexible pronated foot was also clearly demonstrated, the former showing < 5˚ of total movement about an inverted position, the latter showing 10˚ of total movement about an everted position (Siegel et al. 1995). This group then focused specifically on forefoot disease and showed diminished ankle plantarflexion in late stance and delayed heel rise, deficits associated with loss of forefoot rocker function (O’Connell et al. 1998).

In 2004, the first proof of concept for a multi-segment kinematic foot model for RA, based on the Oxford foot model (Carson et al. 2001) was presented (Woodburn et al. 2004). This provided a more complete description of foot motion deficits in RA adding to the work already presented for patients with forefoot disease to altered foot function and separate two cases by severity.

### Table 2.4 A summary table of foot kinematic studies in rheumatoid arthritis with reference to measurement technique, defined foot model and kinematic parameters measured.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Measurement technique</th>
<th>Model</th>
<th>Kinematic parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Locke et al. (1984)</td>
<td>Two single axis electrogoniometers</td>
<td>Ankle joint complex*</td>
<td>Ankle dorsiflexion/plantarflexion</td>
</tr>
<tr>
<td>Keenan et al. (1991)</td>
<td>Two single axis electrogoniometers</td>
<td>Ankle joint complex (tibiotalar and subtalar joints)</td>
<td>Ankle dorsiflexion/plantarflexion</td>
</tr>
<tr>
<td>Siegel et al. (1995)</td>
<td>3D video-based motion analysis system</td>
<td>Shank and foot</td>
<td>Joint angular displacement of the foot relative to the leg in all three planes</td>
</tr>
<tr>
<td>O'Connell et al. (1998)</td>
<td>3D video-based motion analysis system</td>
<td>Shank and foot</td>
<td>Joint angular displacement of the foot relative to the leg in all three planes</td>
</tr>
<tr>
<td>Woodburn et al. (1999, 2002, 2003) and Turner (2003)</td>
<td>Electromagnetic tracking</td>
<td>Ankle joint complex</td>
<td>Joint angular displacement of the rearfoot relative to the leg in all three planes</td>
</tr>
<tr>
<td>Woodburn et al. (2004)</td>
<td>3D video-based motion analysis system</td>
<td>Shank, rearfoot, forefoot and hallux</td>
<td>Joint angular displacement of the rearfoot relative to the leg, the forefoot relative to the rearfoot in all three planes and the hallux in flexion/extension relative to the forefoot</td>
</tr>
</tbody>
</table>

*The ankle joint complex comprises the tibiotalar and subtalar joints*
foot pain and pes planovalgus. In the current model, presented in Figure 2.6, we are now able to measure 3D motion between four foot segments and record the rise and fall of the medial longitudinal arch by tracking a marker on the highest point of the arch at the tuberosity of the navicular. For RA, this is a functionally relevant model since we can expect motion deficits to occur between the rearfoot and shank, and forefoot and rearfoot segments associated with disease in and around the ankle and tarsal joints. The need to extend markers off a wand to enable reliable tracking precludes measurement at all five metatarsophalangeal joints. Nevertheless, the hallux is an important segment to track given the prevalence, severity and impact on function of 1st MTP disease in RA (Spiegel & Spiegel 1982, Shrader & Siegel 2003). This model is currently used at Leeds in clinical gait analysis to assist treatment planning and evaluation in complex cases and in clinical research to understand more fully dysfunctional movement in the foot joints.

In the Leeds gait laboratory, a real-time 3D motion capture (MOCAP) system is used and this is summarized in Figure 2.8. Briefly, we start by palpating and marking surface landmarks on the leg and foot to which we attach small reflective markers. Some markers are attached directly to the skin, others on wands extending from the heel and the hallux and four attached to a rigid plate mounted on a Velcro ankle...
From the process described above, one can automatically identify for both the static and walking trials. Each trial is then processed in a proprietary gait analysis software package: the four segments with the relevant geometry, correct orientation and segment embedded reference frames defined. These reference frames provide a local coordinate system that is fixed and moves with each segment (assumed to be rigid). For each frame of the captured walking sequence, computations are made of the orientation of the coordinates of two segments forming each joint in the software and the joint angles determined. Different computational processes are available for this but the joint angles are finally expressed in clinically meaningful terms such as dorsiflexion/plantarflexion. Intermediate steps in the process include filtering of the raw motion data to remove noise, and data reduction using averaging across trials for the stance period only (heel-strike to toe-off) using the onset and end of the force signal recorded as the foot strikes and leaves the force-plate. Finally, a standard report is generated that provides time normalized angle diagrams for each segment by axis of rotation and summary data, including minimum and maximum joint angles, range of motion and angles and their timings at key events such as heel-strike, mid-stance, heel-rise and toe-off (Fig. 2.8).

Since the process described above is mostly automated, routine application of multi-segment foot kinematics as a clinical investigation tool is now feasible. Our approach uses gait analysis to understand more fully the relationship between inflammatory joint disease, impairment to foot structure and function and the compensatory gait strategies used by patients. For example, in Figure 2.9, a 54-year-old male with long-standing resistant RA presented with severe pes planovalgus and marked forefoot deformity. Managed on anti-TNF therapy his treatment was problematic because of recurrent plantar MTP ulceration. Bed rest followed by custom orthoses and off-the-shelf extra-depth shoes eventually healed the ulcers, but no improvement was noted for his extremely painful and disabling rearfoot and forefoot deformities. Using a multi-segment kinematic foot model the change in the motion parameters could easily be detected and explained in the context of the clinically detected joint stiffness (reduced range of motion) and deformity (altered joint position from which motion occurs).

The rearfoot (Fig. 2.9A), was dorsiflexed at initial foot contact and continued in this direction to late stance. Heel-lift was delayed and plantarflexion severely limited during propulsion, characteristic of the loss of the forefoot rocker function. In the frontal plane the rearfoot was excessively everted and stiff and the motion pattern is closely related to the observed valgus heel deformity. The rearfoot was also more externally rotated than normal and this represents the coupled internal leg rotation with rearfoot eversion described earlier. The forefoot (Fig. 2.9B) was dorsiflexed relative to the rearfoot and this is consistent with the collapsed medial longitudinal arch and no plantarflexion of the forefoot was measured during the propulsion phase. The ‘twisting’ about the forefoot is characterized by the large inverted forefoot position relative to the everted heel position with only a small amount of forefoot eversion during terminal stance. In the transverse plane the forefoot abduction, characteristic of a pronated foot type, was identified as a fixed deformity because of the absence of late stance forefoot adduction. In Figure 2.9C, we can see the collapse of

Figure 2.8  Schematic diagram for kinematic data capture using a multi-segment foot model. MOCAP – motion capture system. Signal processing is undertaken to smooth the motion trajectories. Event detection uses force plate data to identify heel-strike (start) and toe-off (stop) to normalize the kinematic variables in the time domain.
Figure 2.9 Multi-segment foot kinematics in a patient with severe pes planovalgus. (A) Rearfoot-to-shank motion; (B) forefoot-to-rearfoot motion; (C) vertical height of the medial longitudinal arch and (D) hallux-to-forefoot motion. Grey shaded area represents mean±1 SD for five age- and sex-matched able-bodied persons. The represent motion in the sagittal, frontal and transverse planes respectively. In (C), the line represents the vertical arch height for the patient.
the medial longitudinal arch, confirmed in walking by
the ground contact made by the motion marker on the
tuberosity of the navicular from the foot-flat to heel-
rise period of stance. Finally, in Figure 2.9D, the gross
deformity of the hallux is captured well during walk-
ing with evidence that the toe is slightly flexed and
stiff in flexion/extension, rotated in valgus in the
frontal plane and abducted ~90˚ to the forefoot.

This set of motion patterns is typical for pes
planovalgus in rheumatoid arthritis, although extreme
in this case. The multi-segment approach shows the
extent of the functional impairment arising from per-
sistent inflammation in the foot joints and this patient
had evidence of synovitis in the ankle and tarsal joints
and MTP joints. It could be argued that such close
agreement between clinically detected impairment and
the abnormal motion patterns negates the need for gait
analysis. However, it was eventually decided that the
patient undergo forefoot reconstruction surgery and
triple arthrodesis in the rearfoot, and the information
assisted the surgical planning. Moreover, the findings
improve our understanding of foot function in this
disease. What effect does arthroplasty and arthrode-
is procedures have on foot function in rheumatoid
arthritis? Our post-surgery repeat analysis may help us
determine this and further assist design and evaluation
of new orthoses and footwear.

The real benefits for gait analysis will emerge if we
can identify abnormal function in early disease. In
terms of joint motion in the foot, the current challenge
is fraught with difficulties, as we have already identi-
fied considerable variation in able-bodied adults.
Given that the motion also has a temporal element
over the walking cycle we also need to identify the key
parameter, be it a peak value, the timing of that peak
value, or the duration a certain value persists above
a normal level or a combination of these. At the
moment, in early cases, it is not possible to diagnose
abnormal motion on the basis of identifying any one
parameter which lies outside two standard deviations
for the mean value in normal subjects. Pragmatically,
we currently look for a combination of factors, includ-
ing localization of disease activity within the foot,
clinical red flags and an overall trend in the motion
pattern towards abnormal function.

For example, in the following case – a 31-year-old
female patient with well-controlled disease (3 years) but
persistent right foot problems – three features stand out:

1. MRI confirmed synovitis of the ankle and subtalar
joint and tenosynovitis of tibialis posterior and
flexor digitorum longus.

2. Patient self-reported change to foot posture, ‘I can
feel my foot rolling in.’

3. Pain and swelling localized to the tendons passing
the ankle medially with increased subtalar joint
eversion on passive examination, tender when
stressed at end range of motion.

In Figure 2.10, swelling can be detected along the
course of the medial ankle tendons and the heel is in
valgus when standing. Passive range of motion during
the examination of the subtalar joint tended towards
an increased range of motion, especially in eversion.
The MRI findings are consistent with the clinical pic-
ture. From our current understanding of normal func-
tion, the motion pattern is within normal limits,
according to standard diagnostic criteria based on
means and standard deviation. However, we believe
the foot posture has changed according to the self-
reported evidence and it is that change which may be
of more interest in terms of a biomechanical factor
associated with the persistent soft-tissue pathology.
Cause and effect is not clear, but the association
between the pathology, the clinical picture and the
associated movement pattern is notable. Furthermore,
the preserved range of motion should facilitate foot
orthotic therapy using corrective devices aimed at
reducing the amount of eversion and its timings
through stance.

Excessive and prolonged rearfoot eversion is the
hallmark motion pattern in patients with progressive
pes planovalgus and it has been demonstrated in a num-
ber of kinematic studies (Locke et al. 1984, Keenan et al.
al. 2003). It is characterized by progressive shift towards
an eversion motion envelope as the subtalar joint
becomes unstable. Keenan et al. (1991) described three
subtalar motion abnormalities for 10 patients with a
mean disease duration of 25 years: abnormal eversion of
the calcaneus at heel-strike, everted subtalar alignment
through the entire stance phase of gait and insufficient
inversion motion during propulsion to establish a neu-
tral or inverted subtalar joint alignment. Importantly, we
have found strong evidence to support early motion
changes (disease duration ~5 years) in patients with
active peri-talar disease (Woodburn et al. 2002)
(Fig. 2.11). Here, the inversion/eversion motion patterns
were consistent with the observations of Keenan and
others, but with less severe deformity. The frequency of	ibiotalar joint involvement in RA is less than the sub-
talar or midtarsal joints (Lehtinen et al. 1996, Bouyssset
et al. 1987), but reports show decreased ROM and
change in motion pattern favouring dorsiflexion (Locke
et al. 1984, Woodburn et al. 2002). This may be associ-
ated with secondary stresses from other joints, particu-
larly when the subtalar joint is abnormally aligned
(Klenerman 1995).
The subtalar joint has a ‘torque converter’ role, which couples subtalar joint inversion with external leg rotation, and eversion with internal leg rotation. The pronated foot is associated with excessive motion for the latter two and frequently propagated as an injury mechanism for a number of common musculo-skeletal complaints in the lower limb and foot. This coupling effect has been demonstrated in the pes planovalgus foot, showing that the ankle joint complex failed to reach a neutral or inversion alignment and that leg rotation reached a neutral or externally rotated alignment, under the barefoot walking condition (Turner et al. 2003). As a potential injury mechanism, interestingly, valgus heel and knee deformities are frequently observed together in RA and a distal-proximal causal relationship has been proposed but never studied seriously.

What is happening inside the RA foot to cause these motion pattern changes? The medial ankle and tarsal joint ligaments have important motion guiding and stabilizing functions and tibialis posterior is considered the major muscle maintaining the medial longitudinal arch. Persistent or repeated inflammation at these sites may lead to collapse of the arch (pes planus) or valgus of the hindfoot (pes valgus) or both (pes planovalgus) depending on the patterns of joint and soft-tissue involvement. Under physiological loads, the largest tarsal joint rotations are found at the talonavicular joint, especially in the frontal plane (Lundberg et al. 1998). This joint remains stable because the inferior calcaneonavicular (the spring) ligament and the superomedial calcaneonavicular ligament are force-bearing and resist medial and plantar displacement of the talar head, assisted by the expansive insertion and blending of the tibialis posterior to the tuberosity of the navicular.

Histopathological analysis suggests this region is an ‘enthesis organ’ comprising the osteotendinous...
junction (the enthesis), the superomedial part of the calcaneonavicular ligament (which may fuse with the tendon), the tendon sheath, and associated accessory bones (Morrigl et al. 2003). Rich in fibrocartilage, degenerative changes associated with inflammation in RA may target the enthesis itself or adjacent locations. Indeed, MRI studies confirm coexisting insufficiency in the inferior calcaneonavicular ligament and chronic tibialis posterior dysfunction (Yao et al. 1999). Using stab incisions in this region to represent longitudinal fibre tears and focal degeneration, frontal plane changes in talonavicular joint orientation were found in a cadaver foot experiment (Woodburn et al. 2005). Combined with similar attenuation to the medial ankle tendons, followed by cyclic loading at physiological levels, the simulated damage resulted in gross postural changes consistent with pes planovalgus. When tibialis posterior is dysfunctional, the midfoot

Figure 2.11 Three patients with disease duration of <1 year and all presenting with self-reported ankle/rearfoot or medial longitudinal arch instability and clinical evidence of active peri-talar joint disease. In (D) the frontal kinematics of the rearfoot are shown for each patient. Patient A had the mildest deformity and motion approaching the outer one standard deviation from the mean pattern (shaded zone). Patient B shows abnormal motion outside one standard deviation from normal to foot flat to toe-off. Patient C had the most significant deformity with abnormal motion most prominent shortly after loading through to heel lift, but shows good inversion motion through the propulsive phase.
loses its rigidity and stability during the latter part of stance (Coakley et al. 1994, Yao et al. 1999). The powerful gastro-soleus complex then acts across the talonavicular joint as well as the forefoot during propulsion and the resultant motion is thought to stretch the calcaneonavicular and medial plantar ligaments (Coakley et al. 1994, Yao et al. 1999). Visualization of these structural changes in foot geometry was demonstrated through 3D MRI reconstruction of the tarsal joints in pes planovalgus feet (Woodburn et al. 2002). In the talonavicular joint region, very accurate measurement of bone–bone orientation and separation showed an increased distance between the calcaneus and navicular with plantar drift of the talar head. There was strong evidence of ligamentous insufficiency, and half the patients had synovitis, about one-quarter erosive joint changes and over one-third tenosynovitis of tibialis posterior. A reconstruction is shown in Figure 2.12.

Although tibialis posterior tendon rupture is uncommon (a series of three imaging studies found the prevalence to be < 5% amongst cases with pes planovalgus due to rheumatoid arthritis), the tendon may attenuate within its structure through inflammatory damage, and thus become dysfunctional (Coakley et al. 1994, Masterton et al. 1995, Jernberg et al. 1999, Premkumar et al. 2002). The gait changes presented earlier show coupled motion around the joints of the rearfoot. In the cadaver study reported earlier, the tibionavicular, anterior tibiotalar and tibiocalcaneal portions of the medial deltoid ligament were also attenuated on the basis that the tibiotalar and subtalar joints are also involved in rheumatoid arthritis (Lehtinen et al. 1996). This resulted in further changes in eversion orientation and small amounts of internal tibial rotation through the tibiotalar and the tarsal joints. More recently, attention has been paid to degeneration of the interosseous talocalcaneal and cervical ligaments associated with inflammation in the sinus tarsi region (Jernberg et al. 1999, Bouysset et al. 2003). These ligaments are important stabilizers of the talocalcaneal joint and when diseased may contribute to the valgus heel.

Figure 2.12  Two sagittal slices of a post-gadolinium MR sequence showing disease activity in the tarsal joint region (A). The clinical picture is that of severe pes planovalgus (B). In (C) the 3D rendition of the calcaneus, cuboid, talus and navicular is presented. The distance between the geometric centroids of the calcaneus and navicular is significantly greater than normal owing to the insufficiency of the plantar calcaneonavicular ligament and this creates a gap into which the talar head accommodates. This can be measured by an increase in the angle formed between the principle axes of the talus and calcaneus.
deformity described earlier (Kjaersgaard-Andersen et al. 1988). The potential for widespread changes in tarsal structure and function does exist as soft-tissue compromise on the medial side of the foot resulted in small changes in joint orientation at the calcaneocuboid joint (Woodburn et al. 2005). The 3D MRI reconstructed model confirms this, as do the measurements showing flattening also along the lateral aspect of the foot (Woodburn et al. 2002).

The approach to foot kinematics described so far relate only to barefoot walking conditions. Using an electromagnetic tracking technique that allows sensors to be applied to the skin through ‘windows’ cut in a set of laboratory shoes, 3D ankle joint complex kinematics have been measured in-shoe (Woodburn 1999, 2002). Less variability was reported between repeated trials possibly related to improved patient comfort when wearing shoes. The standard shoe had a heel height of 4 cm and served to increase the anterior-posterior pitch of the shoe so that plantarflexion motion was increased for the groups studied. Joint range of motion was not appreciably changed and a small decrease in dorsiflexion was noted. Most interestingly, the stiff medial counter in the test shoe served to invert the subtalar joint and externally rotate the leg, bringing about partial correction of the valgus deformity. No assumptions could be made on the closeness of match between the laboratory standard shoe and those worn by patients day-to-day. Nevertheless, the observed motion control has some interesting implications towards the design of therapeutic footwear.

The final clinical scenario for RA is the acutely painful foot and a patient is featured in whom rheumatoid arthritis was suspected, but where the diagnostic classification criteria had not been fulfilled. This lady presented with an acutely painful and stiff right ankle and exquisitely tender MTP joints 1–3. On examination, synovitis was suspected at the ankle and subtalar joint and she was markedly tender behind the lateral malleolus along the course of the peroneal tendons and in the sinus tarsi. Tenosynovitis of both peroneal tendon sheaths was confirmed by ultrasonography. Her ankle was stiff and very painful when moved into dorsiflexion and her heel was in mild varus when standing. All MTP joints were tender on palpation, notably the medial three. Her spatial and temporal gait parameters were within normal limits, but she commented that she was: ‘Putting up with it but holding . . . (her) . . . foot out the way to make the ball of . . . (her) . . . foot less painful.’

In Figure 2.13, the lateral ankle and medial forefoot swelling is obvious. The peroneal tendon sheath pathology is evident on ultrasound. Her heel is in varus on standing and during walking a ~10˚ inverted position was measured during the entire stance phase. Careful attention to the chronological order of the symptomology suggests her gait pattern is a compensatory effort to avoid loading the medial MTP joints. The complete picture, mapping the disease process to clinical history and the impairment of structure and function provides a more complete understanding of foot function in this case and the information was used to plan the conservative treatment plan as an adjunct to the medical intervention, following subsequent confirmation of the diagnosis.

There has been limited use of joint kinematic analysis as a functional outcome in rheumatoid arthritis. Using a modified standard shoe ankle joint complex, 3D kinematics were measured with and without a custom functional foot orthosis in patients with painful correctable valgus heel deformity (Woodburn et al. 2003). The devices changed the motion pattern towards normal with the main effect being a statistically significant change in eversion motion throughout the stance phase. The orthoses re-established a normal inverted heel-strike position, allowed eversion though the mid-stance phase and increased inversion though propulsion. The devices had no significant effect on reducing internal leg rotation or ankle joint complex dorsiflexion. Beneficially, the changes in kinematic parameters were sustainable over a 30-month period accompanied by improvement in symptoms.

### KEY POINTS

- Kinematics describes the motion in the joints of the foot regardless of the forces causing that motion
- It is not possible to simultaneously measure the movement in all the small joints of the foot during walking. Joints must be grouped into functional units typically comprising the shank, rearfoot, forefoot and hallux
- In pes planovalgus, excessive and prolonged eversion coupled with internal leg rotation and dorsiflexion are notable features. Medial longitudinal arch collapse and forefoot inversion, dorsiflexion and abduction are components of the abnormal motion patterns
- It is possible to demonstrate, in individual cases, close association with sites of inflammation, clinical symptoms, impairment to structure and function and abnormal foot joint motion patterns
- In RA, foot motion can be changed through the use of custom orthoses.
Joint kinetics

Imagine patients with RA walking to the shops to undertake an errand, each time the foot strikes the ground the patient applies a force to the ground. At the same time the ground applies to the patient a reaction force of the same magnitude, but in the opposite direction (the ground reaction force). In the gait analysis laboratory we use a force platform set flush with the floor to measure the ground reaction forces (GRF). Our system allows us to visualize the GRF vector in relationship to the 3D geometry of the leg and foot and to study the three orthogonal components of the force vector designated $F_X$, $F_Y$, and $F_Z$ (Fig. 2.14). During stance phase, the path of the point of application of the force vector within the area of foot contact can be tracked and this is referred to as the centre of pressure (COP). For all these variables, the data can be normalized from 0 to 100% of stance.

The vertical $F_Z$ component shows a spike immediately after initial foot contact and the characteristic double hump separated by a middle valley. The two peaks are approximately 110–140% body weight because of the added effect of vertical acceleration on body weight.

Figure 2.13  (A) Swelling along the course of the peroneal tendons and the medial three MTP joints; (B) varus heel on weight bearing; (C) tenosynovitis on high-resolution ultrasound of peroneus longus and brevis; (D) the inversion/eversion motion pattern in walking (mean ±1 SD from five trials) indicated by the line in comparison with normal range in able-bodied subjects (grey region).
The forces generated parallel to the walking surface, sometimes referred to as the shear forces, also exhibit typical patterns during stance. During initial contact, as the foot comes down and inwards onto the ground, the plate pushes outwards in a lateral direction on the foot; therefore, the initial signal is positive in \( F_X \). For most of stance phase the plate pushes inwards acting in a medial direction (negative \( F_X \)). For the first half of stance phase, the anterior-posterior \( F_Y \) component is negative for the first half of the cycle as the foot drives forward and into the plate. In the second half, the force becomes positive as the patient drives the foot backwards on the plate during the propulsive phase. The magnitude of the \( F_X \) and \( F_Y \) forces is approximately one-tenth and one-third of the vertical GRF respectively.

As noted earlier, walking speed in patients with RA is often slow and this serves to flatten the \( F_Z \) pattern since momentum and vertical acceleration are both reduced. This is a typical finding in patients with well-established forefoot pain with accompanying short stride-length and slow walking speed (O’Connell et al. 1998). This group showed a diminution of both the \( F_Y \) and \( F_Z \) force components during stance phase. For \( F_Y \), the negative force component directed towards the heel was significantly less negative, particularly at the 2nd peak occurring near 90% of the stance phase. Both the double peaks for the \( F_Z \) component were blunted in early and late stance and the COP tended to be closer to the ankle joint and was delayed in anterior progression. This is illustrated for a typical case in Figure 2.15.

In RA as the number and severity of lower limb and foot impairments increase, gait is adapted to compensate, and the GRFs change. GRF data are used in a further calculation, known as inverse dynamics, to determine the internal forces and moments that act across all the lower limb joints in response to external forces including the GRFs. However, the force plate acts as a single force sensor and the spatial distribution of the GRF on different segments of the foot cannot be determined. Therefore, until recently, a major limitation in this approach is that the foot has to be modelled as a single rigid body restricting analysis to the ankle joint. Furthermore, armed with estimates of internal moments and forces and image sequences, such as an ankle MRI, it may be tempting to infer cause and effect between abnormal joint loading and diseased tissue. However, net joint forces and moments cannot tell us how loading is shared amongst important structures that bridge a joint such as the capsule, ligaments and muscle-tendon units and are ultimately diseased in RA. The major structures that contribute to the net moments of force, or torque, are the muscle forces, so our data tell us something about the mechanical output of the controlling muscles. How well these muscles act to produce and control limb movement can be determined from joint power analysis. Joint power is calculated by multiplying the net moment of the force by the joint angular velocity. When profiled over stance, the time integral of the power curve tells us the positive and negative mechanical work done. Our
assumption is that all bone and muscle forces are reduced to a single vector resultant force and moment and that these can be expressed, in a 3D analysis, about each axis of rotation. Power and work are scalar terms but for the purposes of clinical relevance can also be conveniently partitioned by each body plane.

The net torque generated by the muscles crossing the ankle joint, the internal moment, is predominantly plantarflexion for most of stance phase. Initially, however, a small dorsiflexion torque is developed during initial contact as the GRF vector is located posterior to the ankle joint centre (Fig. 2.16). Here, the ankle rapidly plantarflexes under control from the ankle dorsiflexor muscles acting eccentrically. This is the first rocker function of the foot and ankle. The COP then rapidly advances from its initial point of contact towards the ankle joint centre coinciding with a reduction of the dorsiflexor moment to zero and the onset of a plantarflexor moment. As the COP advances forward from the ankle joint centre, the moment arm increases and the plantarflexor moment increases to a peak in late terminal stance (~60% stance). During this period the plantarflexor muscles act eccentrically to control the forward rotation of the leg over the foot (the second rocker function) and then concentrically to generate a rapid push-off where the COP, located at the MTP joints, is furthest from the ankle centre (the third rocker function). Towards toe-off, a small but functionally important third peak occurs, a small dorsiflexor moment to effect toe clearance from the ground. The power profile shows the typical power absorption phase from initial contact (80% of stance) where typically ~ 10 J of negative work is undertaken through to the large and rapid power generation phase towards toe-off, where typically ~ 30 J of positive work is undertaken (Buczek et al. 1994).

These normal ankle moments are shown in Figure 2.16, contrasted with a patient who presents with markedly swollen and tender MTP, midtarsal, subtalar and tibiotalar joint and probable medial tendon tenosynovitis consistent with a flare in his disease (DAS score was 5.73). His walking speed was slow (0.54 m/s), stride length short at 0.68 m and double support time prolonged at 24.4% of the stance phase.
His impairments are closely related to underlying gait function; his foot is placed carefully on the ground, with loss of first rocker function. The COP remains in the heel and midfoot region for a prolonged period during stance (notice the dither and posterior progression in the midfoot) and towards toe-off the COP remains posterior to the location found in otherwise healthy individuals such that the moment arm during stance is much reduced. The sagittal plane moments are significantly lower than normal and the power generated in terminal stance is about one-fifth the normal value. These findings are typical in RA patients, especially those with forefoot pain and this has been clearly demonstrated by the work of O'Connell et al. (1998). This group hypothesized that the third rocker function of the foot would most likely be affected in RA patients with symptomatic forefoot disease. They confirmed this by showing a significant reduction in the peak plantarflexion moment accompanied by a delay in the anterior progression of the COP in comparison with normal, as typically demonstrated by the case presented within.

Similarly, changes can be detected in the moment and power profiles in both early and well-established disease. In Figure 2.17A, this patient was assessed prior to total ankle joint replacement and demonstrated characteristic reduction in both the moment and power profile. By contrast, in Figure 2.17B, this patient with early disease has adopted an antalgic gait in response to forefoot pain and subsequently developed inflammation in the subtalar joint and peroneal
tenosynovitis. We noted in the clinical examination severe pain on ankle dorsiflexion and to avoid this, the patient is maintaining the foot in a plantarflexed pose during gait. This is captured by the near normal plantarflexion moment and the generation of power from about 30% of stance through to toe-off, indicating active plantarflexion of the gastrocnemius-soleus complex to maintain the compensatory joint pose.

In a complete 3D model, reference should also be made to the internal moments and power profiles about the secondary plane axes, but these are less reliable than the sagittal plane. The frontal plane is important in RA as we have previously seen the kinematic changes associated with pes planovalgus and varus heel deformity. Eng and Winter (1995) showed an evertor moment during initial contact and terminal stance,
with an invertor moment during mid-stance. In the transverse plane, a small external rotation moment was observed during initial contact, then again with a higher peak during propulsion. In both planes, small and highly variable power phases, accounting for ~7% of the total work for the ankle joint, were observed. Interestingly, Siegel et al. (1995) compared two cases with rigid varus and mobile valgus rearfoot deformity. The patient with the varus deformity showed an evertor muscular moment for ~75% of stance phase, opposing the inverted foot position in comparison with a large invertor moment controlling the everted foot position, with the COP remaining lateral to the midline of the foot in the mobile valgus rearfoot case. No data for the transverse plane were presented. In Figure 2.18, the frontal plane moments and power are shown for a patient with a flexible collapsing pes planovalgus. The net muscular moment in the frontal plane is predominantly invertor, opposing the everted position of the foot during stance. Currently, we have made no observation on transverse plane net muscular moments or power profiles in feet of people affected by RA.

**KEY POINTS**

- The ground reaction force (GRF) is equal and opposite in direction to force applied to the ground each time a patient’s foot strikes the ground.
- In the gait laboratory this can be measured using a force plate.
- The GRF vector has three orthogonal components designated $F_x, F_y, F_z$.
- In RA each component of the GRF can be affected according to impairments encountered.
- The internal moment is the net torque generated by the muscles crossing the ankle joint. From this joint power can be calculated.
- In RA, the sagittal net muscular torque and joint power can be significantly reduced where the ankle is stiff, or the forefoot is impaired, primarily by pain and deformity.

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**Figure 2.18** Frontal plane ankle joint moment (A) and power (B) profile in a 43-year-old patient with 9 years’ disease duration. His disease was active on the day of gait assessment and he presented with a collapsing but flexible pes planovalgus deformity (C) and probable tenosynovitis of the medial ankle tendons located around tibialis posterior and flexor digitorum longus.
Plantar pressure distribution measurement is the most frequently used gait analysis technique employed in the study of RA. The equipment is readily available, relatively easy to install and use, and it provides information that can be both visually interpreted for clinical use and processed for more robust analyses. As described earlier, the GRF has a COP point location within the area of contact of the foot that changes though the stance phase of gait. If the contact area is known over which the force vector is distributed we can calculate pressure, defined as the force per unit area and expressed in kPa (Fig. 2.19). In the Leeds laboratory two pressure measurement systems are used: a plate device, similar to our force plate, but with a matrix array of small \((5 \times 5 \text{ mm})\) capacitance-based transducers or sensors, and, using the same technology, a flexible in-shoe pressure sensing insole (sensors in the array vary in size \(\sim 10 \times 5 \text{ mm}\)). The former permits higher resolution measurement at the foot/plate interface and is able to detect pressure over small discrete anatomical regions such as the metatarsal heads. In-shoe measurement is useful to study step-to-step variability within a walking sequence, to analyse pressure distribution during activities of daily living such as walking, standing and stair climbing, and, importantly, it permits measurement at the interface between the foot and the shoe or foot and shoe-orthosis. Measurements are generally undertaken using both systems to allow assessment of foot function and to assist planning or evaluating some therapeutic interventions as part of the overall assessment protocol. In each case, the visual output also serves as a powerful education tool to explain to patients how their foot shape has changed, why certain regions of the foot are tender if located at a site of prominent deformity and high pressure, or why a callus or ulceration is present and how a shoe or orthosis is working to redistribute pressure from these sites (Fig. 2.20).

Most pressure distribution measurement systems have software tools to permit detailed analysis of foot function. The walking footprint can be used to study the geometry of the foot: the algorithm overlaying a series of lines determined from subdivisions of the foot contact area defined by anatomical landmarks. In Figure 2.21, one of these footprints is shown for a patient with a severe pes planovalgus foot. In comparison with normal (Fig. 2.21A), the collapsed medial longitudinal arch and severely abducted forefoot can easily be visualized (Fig. 2.21B) with a significant increase in the subarch angle (case 148˚ versus 105˚ in able-bodied subject), defined by the angle formed between the points RLN (see Figure 2.21). Other parameters are useful in relation to typical forefoot changes in RA and include the hallux angle (case 72˚ versus 8˚ in able-bodied subject), and spreading of the metatarsals using a co-efficient of the forefoot width/foot length (case 0.4 versus 0.36 in able-bodied subject). In a preliminary analysis, several of these footprints parameters were correlated with 3D structural variables derived from MRI reconstructions in pes planovalgus (Woodburn 2002). These findings are encouraging and suggest that important structural information may be gained from simple footprint parameters avoiding the unnecessary use of radiographic techniques.

Having gained information on the overall foot shape, analysis can then determine how pressure is
distributed, in terms of magnitude and timing, in plantar regions of interest. This can be simply visualized in ‘playback’ mode and in Figure 2.22 a profile is shown for a patient with sharp focal pressures over the MTP joints. The diagram depicts the profile across the whole foot every 0.06 s for 15 frames (foot contact time = 0.90 s). A small spike of pressure appears in the second frame located at the medial calcaneal tubercle, which was prominent when directly palpated. In the midfoot a normal medial arch profile is present for the entire stance phase duration. Peak pressure values can be determined for each region of interest when masks are overlaid on the footprint. The major abnormality, in this case, existed in the forefoot with sharp spikes of pressure, in excess of normal upper limits, across the 2nd-to-5th MTP regions (Fig. 2.23). Not only are these pressures abnormally high, but they develop around 0.36 s and last until 0.84 s, a contact time of 0.48 s, representing over 50% of the stance phase. Lesser toe
contact is made, but only for 0.18 s in late stance and function is diminished as the peak pressure values are under normal limits for this region. Interestingly, pressure distribution is normal in the 1st metatarsal head region and the hallux.

The utility of these data emerges when we consider the clinical picture (Fig. 2.24). This 59-year-old male patient presented with longstanding bilateral forefoot pain, deformity of his toes and self-reported changes to his walking style (slow, avoidance of uneven surfaces, and careful foot placement). On examination, the MTP and interphalangeal joints were retracted with claw-toe deformity, although range of motion was within normal limits. The fatty-fibro padding was displaced anteriorly and dorsally and moderate callus and adventitious bursae were observed over the 2nd–5th MTP joints (Figure 2.24A and B). He tested positive to the metatarsal squeeze test, and all 5 MTP joints were tender on direct palpation. Radiographically, all 5 MTP joints scored 5 on the Scott modification of the Larsen index, indicating severe erosion and deformity. The tarsus and ankles of both feet were unremarkable. The sites of high pressures are entirely consistent with the pathology and clinical features at the MTP joints (Fig. 2.24C).

The foot pressure distribution pattern in able-bodied persons is variable and Hughes et al. (1991), using a discriminant analysis technique, classified four basic patterns (medial, medial-central, central, lateral). In RA, these basic patterns are influenced by the extent of forefoot pain and deformity, and any compensation strategy to off-load sites and by the transfer of load forward from the heel and midfoot particularly when these sites are deformed and painful. In Figure 2.25, we noticed our patient was uncomfortable when standing and was rolling the foot outwards to off-load the medial forefoot region. The patient, with 2 months of disease, was tender at the medial three MTP joints, the first and second of which had synovitis confirmed by ultrasonography. Early in the disease there were no major foot deformities, but the daylight sign confirmed spreading of the forefoot. She had no plantar pressure lesions, and when the peak pressures were averaged the loading pattern was medial according to the Hughes Classification. However, when individual steps were considered, two other patterns were observed on subsequent steps and we believe this demonstrates a variable off-loading pattern in response to the medial forefoot symptoms.

Clinical utility of plantar pressure measurement has been demonstrated in a number of studies. Sharma et al. (1979) for instance, showed that forefoot loading was associated with lesser toe deformity and increased severity of clinical symptoms and radiographic joint damage. In three cohorts of patients

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**Figure 2.23** (A) masking technique for regional pressure analysis. (B) distribution and magnitude of peak pressure across the MTP joint regions in comparison with normal range (area between dashed lines represent the normal range [mean ±2SD]).
Figure 2.24  Forefoot clinical features showing lesser toe deformities, prominent metatarsal heads, and bursa and callosities overlying the MTP joints (A) and (B). Peak pressure profile in presented case (C) and foot of age and sex matched normal subject (D).

Figure 2.25  Patient with early disease off-loading the medial forefoot region, which was both tender and inflamed. The daylight sign is seen on standing at the 1st and 2nd toe clefts. The pressure distribution pattern is variable ranging from central to medial. The symptoms (RAI: Ritchie Articular Index scores for tenderness ranging in severity from 0 to 3), presence of inflammation (HRUS: high-resolution ultrasound), and peak plantar pressure (PP) values for each MTP joint are presented in the table.
presenting with progressively worse radiological damage (not based on any current scoring system), loading on the hallux, second and lateral toes (regional analysis) were markedly reduced in comparison with normal with significantly less loading in the lateral toe regions (Sharma 1979). The area of toe contact and the force generation capacity of the lesser toes appear critical in relation to abnormally high peak pressures in the forefoot (Collis & Jayson 1972, Sharma 1979, Simkin 1981, Minns & Craxford 1984, Soames et al. 1985). In Figure 2.26, a series of six feet are presented with varying severity of forefoot impairment characterized, if viewed from left to right, with increasing loss of toe function. In each case, focal areas of high pressure with a sharp gradient from adjacent sites were observed and consistent with MTP deformity including hallux valgus and claw and hammer toe. These patterns are observed in patients who have predominantly forefoot disease and in all cases a well-defined medial longitudinal arch is present. This serves to decrease the contact area proximal to the metatarsal head region in some cases and this is well demonstrated in the right most foot.

The case series above perfectly illustrates why it is impossible to attempt to define a typical pattern of forefoot loading in patients with RA. In addition to deformity, plantar pressure measurement may be affected by local factors such as skin and soft-tissue thickness, plantar callosities (Sharma 1979, Woodburn & Helliwell 1996) and the extent of bony erosion at the MTP joints (Sharma 1979, Soames et al. 1985, Tuna et al. 2004). Sites of forefoot plantar callus are proxy indicators of localized high pressure and these are useful to detect during clinical examination. In most instances, the callus has an underlying adventitious bursa and both serve to increase the contact area over which the forces are distributed. Hence the debate as to whether these lesions are protective or harmful (Woodburn et al. 2000, Davys et al. 2005). We do know, however, that elevated focal pressures are associated with the development of ulceration in some feet and three pressure profiles are shown in Figure 2.27. In each case, the peak pressures are in excess of normal values, and are experienced for prolonged periods during stance. Extensive deformity, bone erosion and fatty-fibro padding displacement are important in all three cases and, characteristically, the ulcer sites have steep pressure gradients from the adjacent normal or under-loaded skin sites. Toe function is universally non-existent.

Attention to underlying structure is important and four groups (Sharma et al. 1979, Soames et al. 1985, Tuna et al. 2004, Davys et al. 2005) noted higher pressures in patients with more erosive disease in the MTP joints. These joints are frequently subluxed or dislocated and through erosion have irregular surfaces and sharp spikes. When the fatty-fibro padding is displaced it is of little surprise that these feet have sharp focal pressures, severe symptoms and are at risk from ulceration. The pathomechanics of this process has been well described by Stainsby and others in rheumatoid arthritis (Dixon 1969, Mann & Coughlin 1979, Stainsby 1997, Briggs 2003). Briefly, synovitis and effusion leads to capsular stretching, which results in loss of integrity of the collateral ligaments (hypothetically with greater involvement of the medial collateral ligaments that leads to fibular toe drift) and capsule itself. The plantar plate moves distally around the metatarsal head (see

Figure 2.26 Peak pressure distribution profiles for six patients with rheumatoid arthritis with varying severity of foot impairment characterized from left to right with increasing loss of toe function and decreased forefoot contact area.
When foot pressures are measured, the duration of pressure experienced at each site must be considered alongside the absolute peak pressure value and this is generally longer in RA patients than normal, so moderate-high pressures may also be experienced for longer in discrete regions such as the forefoot. If plotted as a function of time, the pressure integral or impulse can also be higher (Turner et al. 2003, Otter et al. 2004). This, however, mostly depends on symptoms because those patients with severe forefoot pain at sites of high peak pressure can compensate by delaying load transfer into the forefoot and then rapidly off-loading by eliminating the third rocker function (Simkin 1981, Soames et al. 1985). This can be assessed by measuring the time spent by and the velocity of the COP through the heel, midfoot, forefoot and toe regions of the foot. A typical case is shown in Figure 2.29. In the able-bodied subject, the first rocker function of the foot is shown by the rapid forward progression of the COP in the heel region, followed by forward progression of the COP between 0.3 and 0.5 m/s through the midfoot, forefoot and toe at approximately 22%, 42% and 85% of stance phase respectively. By contrast, the patient has no first rocker function and maintains the COP in the heel region until 70% of stance. The velocity rises sharply through the mid- and forefoot regions, peaking at 2.0 m/s, with the forefoot and toe contact together only making 11% of stance phase contact. The foot is

![Figure 2.27](image_url)  
**Figure 2.27**  
Peak pressure distribution profiles for three patients with forefoot ulceration. Ulcer sites are indicated with a red circle.

![Figure 2.28](image_url)  
**Figure 2.28**  
The planovalgus foot in RA reveals interesting changes in pressure distribution and these have been described in a number of studies (Stockley et al. 1990, Woodburn & Helliwell 1996, Turner et al. 2003). Stockley’s group found an association between valgus heel deformity and elevated medial forefoot pressures in patients who had undergone forefoot arthroplasty (Stockley et al. 1990). Since these patients fared worse surgically than their counterparts with normal heel alignment, they concluded that load transfer from the heel to forefoot was an important factor in foot function. A later study confirmed this showing a similar medial distribution of forefoot peak pressures and callus patterns in patients with valgus heel deformity (Woodburn & Helliwell 1996). More recently, a

Figure 2.29  Progression of the centre of pressure (COP) through the foot. The velocity of the COP is plotted as a function of time in (A) a typical able-bodied individual and (B) a patient with severe forefoot impairment (pain located to the central MTP joints with overlying callosities and claw toe deformity).

lifted from the ground indicating loss of the second and third rocker functions.

Further work is necessary to elucidate further the compensation strategies adopted by patients to alleviate symptoms. They appear to be highly variable. The case above is predominantly compensating, using a sagittal plane strategy, but contrasts that with two further patients shown in Figure 2.30. These patients both had persistent and severe forefoot symptoms starting early in the course of their disease and had compensated by holding the foot in a stiff varus position to off-load the more painful medial side. The long-term consequence is fixed varus foot deformity with elevated pressures and new symptoms at the lateral forefoot.
comprehensive study of 23 cases with clinically defined pes planovalgus incorporated spatial-temporal and 3D ankle joint complex kinematics with plantar pressure measurement (Turner et al. 2003). Typical pronatory motion for the ankle joint complex was found with changes to the plantar pressure and force distribution patterns. The patients walked slower and remained in double-support longer and these compensatory mechanisms reduced the amplitude of the pressure and force variables, but may have served to increase the cumulative load because of the longer contact time. This was demonstrated in the heel region and the effect may be beneficial to reduce painful symptoms in the peri-talar region during the loading response. As expected, medial longitudinal arch collapse resulted in a large increase in the contact area in the midfoot and medial forefoot as the soft-tissue and bony architecture changed. Furthermore, higher midfoot peak pressures and pressure-time integrals may be related to tissue viability problems and skin callosities were noted over the talonavicular joint area in several patients with severe deformity. Internal joint loads may be harmful in the midfoot region where peak force, force-time integrals and contact time are increased and this was demonstrated in the patients. In agreement with others, forefoot loading was altered from a central to a medial pattern, peak pressure and pressure- and force-time integrals were greater, and lateral forefoot off-loading was present in the group with RA (Fig. 2.31). The findings from this study confirm the observation that pes planovalgus may have detrimental effects on the entire structure and function of the foot.

The progression of pes planovalgus has been estimated in radiographic studies, but these techniques are not advisable for routine clinical use. Plantar pressure measurement offers quick assessment of overall foot geometry and loading characteristics. From the Turner et al. study of 2003 six cases are presented in Figure 2.32 to show the progressive features of pes planovalgus. From left to right, progressive off-loading in the lateral heel and forefoot regions with increased loading medially and increased contact area in the midfoot region can be observed. Two extreme cases on the far right are shown with severe midfoot collapse with high focal pressures in the navicular-medial cuneiform region consistent with sites of pain and overlying callus.

Plantar pressure measurement is useful in determining functional changes in our patients who have multiple impairments throughout the foot. Figure 2.33 shows the profile from a female patient in her 60s with over 25 years of disease duration. Although her disease is well controlled on biologic therapy she has persistent forefoot pain. She presented with hallux valgus fixed at ~15° of dorsiflexion, claw toes, which were retracted and non-weightbearing when standing, and a valgus heel deformity with low medial longitudinal arch profile. Her walking speed was slow at 0.78 m/s, cadence 113 steps/min, cycle time 1.06 s and stride length 0.83 m. In the forefoot the normal geometry of the MTP parabola had been disturbed through a combination of disease activity (erosion and deformity) and surgical intervention (arthrodesis and arthroplasty). The sharp distal margins of the eroded 5th MTP and arthrodesed 1st MTP were evident on plain X-ray. The fatty-fibro padding was displaced anteriorly and a callus was present over the 1st MTP joint. The heel fat pad was atrophied and a firm palpable mass consistent with a nodule or bursa present over the medial calcaneal tubercle. From her pressure profile a number of features can be determined. The superimposed COP shows normal progression in the heel region, delay in the midfoot and rapid progression through the forefoot consistent with an off-loading pattern described earlier. The elevated medial heel pressures are located as the site of the plantar nodule or bursa. Although her arch profile is low, it has stiffened in that posture rather than collapse as the pressure profile shows a normal arch distribution.
Armed with this information one can go about systematically linking structural and functional impairment with knowledge of the disease pathology, symptoms, compensation strategies and effects of past interventions. In the forefoot, the heel position has resulted in high medial pressures concentrated over the first metatarsal head, which is dorsiflexed and immobile. This is further exacerbated by the fixed dorsiflexed position of the 1st MTP with the hallux undergoing minimal ground contact. Laterally, the eroded 5th
metatarsal head may be responsible for the spike of pressure at the MTP joint region. The 3rd metatarsal head was slightly longer and prominent on palpation and the toe made no ground contact explaining the elevated pressure here. Pain is significant enough to slow the walking speed and shorten the stride length.

The pressure information from this case was used to assist surgical planning and it is here that PPM has been used as a functional outcome tool (Betts et al. 1988, Phillipson et al. 1994, Dereymaeker et al. 1997, Bitzan 1997). Findings have been variable and difficult to compare due to methodological differences with the pressure measurement and variance in the patient cohorts, surgical procedures undertaken and follow-up time. As a rule, detrimental peak pressures can be reduced in the forefoot with arthroplasty procedures and these tend to be associated with clinical improvement in symptoms and function (Betts et al. 1988, Dereymaeker et al. 1997, Bitzan 1997). Betts and colleagues found that pre- and post-operative pressure analysis allowed them to appraise their surgical techniques after encountering good pressure reduction in the central metatarsal heads, but not at

Figure 2.33  (A) The heel is in valgus. (B) The medial longitudinal arch height is low but not fully collapsed and the midfoot joints are stiff. (C) The lesser toes are retracted, deformed and non-weight bearing. (D) The 1st MTP is arthrodesed, the 2nd, 3rd and 4th MTP joints are severely eroded, and the 5th MTP has undergone excision of the metatarsal head and proximal phalanx. (E) Peak pressure profile with vertical ground reaction and centre-of-pressure line superimposed.
the 1st or 5th (Betts et al. 1988). They attributed this to a shallow arc created during the metatarsal head resection thereby transferring load from the central metatarsal heads outwards to the first and fifth. Dereymaeker et al. (1997) detected a reduction of abnormal high pressure areas in just over 50% of cases with increased toe loading and resolution of plantar callosities at 35 months post-surgery. After a similar period, by contrast, Phillipson and colleagues found an increase in both peak pressure and the pressure-time integral in 15 patients, although not all were RA. This was partly attributed to increased walking speed following surgery, although this change was not formally reported and the greatest changes were reported under the first metatarsal head and associated with recurrent callosities. Clearly, more controlled studies with well-defined end points are required to clarify some of the anomalies arising from the current literature.

Elsewhere, in-shoe pressure measurement has been used to assess the off-loading properties of custom and prefabricated orthotic devices. Shrader and Siegel, from the National Institutes of Health, present a very detailed and excellent case history, showing the effective reduction of peak pressure and pressure-time integral in the forefoot using a custom-fabricated orthosis (Shrader & Siegel 2003). The pressure relief was accompanied by improvement to symptoms and ability to perform activities of daily living requiring standing and walking. In more controlled studies, both prefabricated and custom orthoses have been shown to be effective for reducing forefoot pressure and improving symptoms with moderate evidence to suggest custom devices are more effective (Hodge et al. 1999, Li 2000). The Leeds randomized controlled trial of custom orthoses manufactured in carbon-graphite, followed 101 patients with early disease and mobile correctable valgus heel deformity for 30 months (Woodburn 2001). Results showed effective off-loading of the medial forefoot region, increased contact area and force in the midfoot and decreased pressure in the heel region, all favourable changes for the foot type being treated.

Noteworthy is the use of padded hosiery as these too, in the short term, have pressure-relieving function similar to orthotics. In Leeds, the effect of scalpel debridement on forefoot pressures at callus sites has also been studied and showed no significant difference in the change between a real and sham procedure (Davys et al. 2005). Again, these areas will benefit from further studies that are well controlled, adequately powered to detect pressure differences and appropriately disease-staged.

**KEY POINTS**

- Plantar pressure (P) is defined as the force (F) per unit area (A) \( P = \frac{F}{A} \).
- Plantar pressure distribution can be measured using platform or in-shoe systems.
- The pressure footprint can be used to determine the plantar geometry.
- Peak pressure and the pressure-time integral are frequently higher than normal in the forefoot in RA and are associated with impairment including pain, stiffness and deformity.
- In pes planovalgus collapse of the medial longitudinal arch is associated with increased contact and force in the midfoot region.
- Plantar pressure relief, pain reduction and functional improvement have been reported following surgical and non-surgical interventions for symptomatic forefoot disease in RA.

**MUSCLE FUNCTION (SEE ALSO CHAPTERS 3 AND 4)**

Determining muscle function from electromyography has been rarely studied in RA. Perry (1992) suggested that the loss of muscle strength was a major pathomechanical factor in foot disease in RA. Two mechanisms were postulated: (1) inflammation causes joint and soft-tissue pain that inhibits muscle function, thereby reducing the force transmission at painful joint sites and (2) through the above action motion is decreased, which results in reduced activity and the development of secondary muscle weakness. This was demonstrated in a single RA patient with gastrocnemius and soleus weakness secondary to MTP joint inflammation showing loss of third rocker function as described earlier (Perry 1992). The same group used fine-wire electromyography (EMG) to study extrinsic foot muscle function in patients with RA with and without valgus deformity of the foot (Keenan et al. 1991). This work eloquently showed increased intensity and duration of activity of the tibialis posterior, supposedly in an effort to support the medial longitudinal arch in those patients with valgus heel deformity. This group believed the compensatory action of tibialis posterior was due to primary weakness in calf muscles that, despite increased activity on EMG testing, were weak on manual muscle testing. Combined with motion, structural and clinical data, the integrated approach demonstrated in this work facilitates a better understanding of changes in foot function resulting from primary pathology. Electromyography
is one of the more difficult techniques to undertake during routine gait analysis and we have yet to fully integrate this technique to the approaches described above.

ENERGY CONSUMPTION

When patients who have RA walk with painful joints and undertake compensatory strategies we can postulate that the gait is less energy efficient than normal. Measurement of metabolic energy expenditure is a useful global measure of overall gait performance and allows the physiological cost of pathological gait to be estimated. Measurement techniques are not routinely performed since they typically involve collection and analysis of blood gases and heart rate whilst walking or exercising. The physiological cost index (PCI) was previously proposed as an objective measure of disability and as an outcome for intervention studies in RA (Steven et al. 1993, Kavlak et al. 2003). The index is based on the observation that any voluntary increase in walking speed demands an increase in energy expenditure that is relatively disproportionate in disabled persons. Past studies have shown that NSAID therapy leads to a statistically significant improvement in the PCI, but that the improvement was not correlated with conventional clinical measurements such as tender/swollen joints, pain score or ESR (Steven et al. 1983). Kavlak and colleagues (2003) found a significant improvement in foot pain, step and stride length, and the physiological cost index after 3 months of foot orthotic therapy. Beyond these two applications, the PCI has not been widely adopted in clinical research and this is understandable when disability can be easily measured using valid and reliable global and foot-specific questionnaires.

CONCLUSIONS

This chapter has summarized currently available gait analysis techniques that are available to the clinician and researcher. We have appraised the available information and where evidence was lacking appropriate cases were presented to illustrate the use of the various techniques in our own laboratory. Plantar pressure measurement probably represents the extent that most clinical units will invest in gait equipment and we believe this is reasonable given its ease of use and affordability. This technique yields valuable information on foot structure and function in RA. We are steadily refining other techniques and using them for clinical research and practice. 3D joint kinematic and kinetic analyses are more difficult to perform and interpret, but we have shown some valuable uses in patients at various stages of the disease to detect changes associated with impairment and as compensation strategies. We hope that gait analysis will become more readily available in the future, as it has important uses for determining the structural and functional changes in the foot brought about by inflammation in the joints and soft-tissues of the foot. Moreover, treatment planning and evaluation can be greatly aided by gait information, especially in complex cases undergoing surgical intervention. In terms of clinical research, gait analysis will be used to drive experimental work aimed at further advancing our core knowledge and, in translational studies, inform future development of customized approaches to footwear and orthosis manufacture as well as foot surgery.

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Further reading

