

The biomechanics of pathological gait – from muscle to movement

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Clinicians face the daily challenge of assessing and treating patients with gait problems. Musculoskeletal models appear to show potential for assisting with the understanding of complex pathological movements, however they are also complex and reliant on multiple assumptions in order to maintain stability. This paper breaks down the process by which muscles produce movement into a series of steps. The contributions and limitations of modelling each separate step are then considered. The calf muscles serve as an illustration throughout the paper, as these muscles are frequently implicated in the development of pathological gait patterns. An argument is put forward for the development of a range of tools for use in clinical practice, leading to an enhanced appreciation of the importance of joint moments. Improved clinical understanding of the link between muscles and movement will allow clinicians to develop better treatment plans for their patients.

Key words: musculoskeletal modelling, simulation, muscle, triceps surae

1. Background

During normal walking muscles produce forces which act directly on the skeleton. These forces influence whole body movement, as joint contact forces and ground reactions cause the effects of muscle force to be transmitted to segments remote from the muscular contraction. This effect is referred to as “dynamic coupling”. Understanding the complex mechanisms behind normal gait is challenging. For the clinician seeking to treat a patient this complexity is compounded by the need to take account of the effects of pathology.

In recent years, musculoskeletal models have been used to generate simulations of normal and pathological gait patterns [1], [2]. The resulting predictions have often been counter-intuitive, suggesting that models have considerable potential to improve understanding and hence treatment planning for patients.

However, practical models appropriate for routine clinical use still seem a long way off.

This paper will highlight the contributions and limitations of current modelling approaches, using the calf muscles by way of illustration. Many of the arguments developed are based on the gait of children with cerebral palsy. Treatment planning in this group is the most frequent use of clinical gait analysis, so it would seem logical that any clinical tools produced should be appropriate for them.

A case is made for a renewed focus on joint moments, as an accessible method of understanding pathological movement.

2. The calf muscles

Figure 1 is an illustration of the triceps surae taken from the original engravings of Dr Henry Gray (1825–

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1861), *Anatomy of the Human Body* published in 1918. All three component muscles, the medial and lateral head of gastrocnemius and soleus, cross the talo-crural and subtalar joints through their insertion into the Achilles tendon. At the ankle they are considered to act as plantarflexors. The heads of gastrocnemius also cross the knee and are described as knee flexors. However, this simple interpretation does not adequately represent the complex and unresolved function of these muscles. As SALMONS states, in Gray's anatomy (1996) *The relative contributions of*

soleus and gastrocnemius to phasic activity of the triceps surae in walking has yet to be analysed satisfactorily [3].

The table gives the key architectural and morphological characteristics of the triceps surae. The information presented gives some indication of the complementary contributions of the three different components of the triceps surae. The soleus has by far the largest physiological cross sectional area (PCSA) and shortest fibre length suggesting a muscle that is capable of producing large forces over a short range. In contrast, the lateral head of the gastrocnemius has fibres of more than twice the length and has a comparatively small PCSA, suggesting a muscle designed to generate lower forces but capable of contracting at much higher velocities. The medial gastrocnemius has an architecture somewhat between the other two muscles.

3. The role of the calf in gait

With such large differences in structure, it might be expected that the muscles would make different and distinctive contributions to the normal gait cycle. Overall during gait the calf muscles are known to be active in stance phase, with activity rising to a peak in terminal stance before the muscle becomes quiet during swing [4]. Figure 2a, however, shows data taken from the normal database from ORLAU. There are clearly subtle differences in the activation of the component muscles at a self-selected walking speed. Figure 2b shows the contribution of walking speed, where more dramatic changes are revealed. The marked increase in activation of the lateral gastrocnemius with increasing speed suggests a particular role for this muscle in accelerating the lower limb.

A simplistic view would be that the muscle has an action dictated by its morphology and at any time it is known to be active, allowing for the electro-mechanical delay. Muscle action can, however, be more subtle than that. The calf muscles, for example, are thought to contribute to knee extension, through the "plantar flexion/knee extension couple" [5].

The calf muscles are common contributors to the development of pathological gait patterns. Their distal location makes them vulnerable to injury or any more proximal neurological insult. Conditions where calf muscle involvement contributes to the development of a pathological gait pattern include cerebral palsy, muscular dystrophy, spinal injuries and stroke.

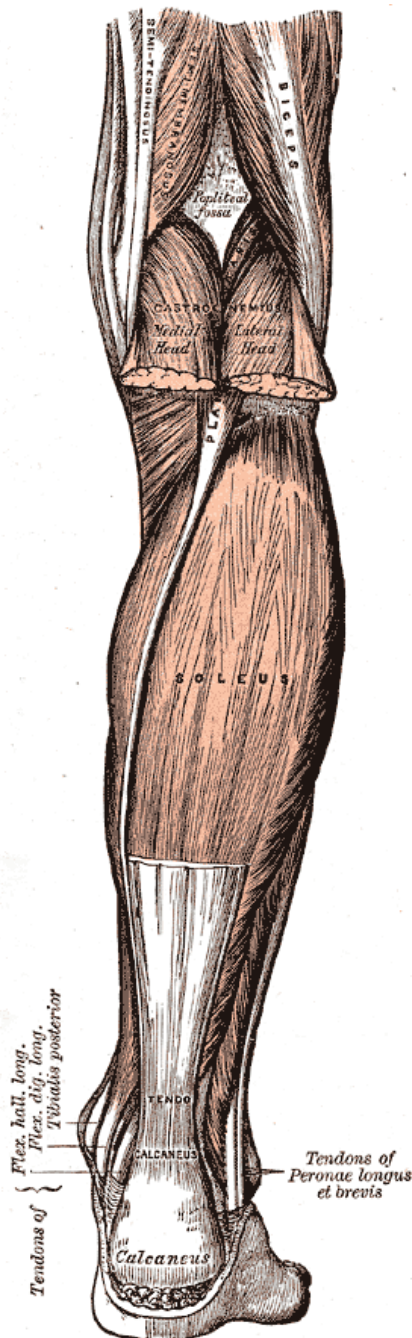


Fig. 1. Illustration of the triceps surae taken from the original engravings of Dr Henry Gray (1825–1861)

Table. Key morphological and architectural parameters for the calf muscle. Moment arm values are given within 10 degrees of the anatomical position

Parameter	Reference	Medial gastrocnemius	Lateral gastrocnemius	Soleus
Volume (cm ³)	FRIEDRICH and BRAND 1990 [28]	137	74	374
	WARD et al. 2009 [29]	114	62	276
Muscle length (cm)	FRIEDRICH and BRAND 1990 [28]	22.2	21.7	33.7
	WARD et al. 2009 [29]	26.9	22.4	40.5
	WICKIEWICZ et al. 1983 [30]	24.3	22.1	31.0
Muscle fibre length (cm)	FRIEDRICH and BRAND 1990 [28]	3.89	6.06	3.03
	WARD et al. 2009 [29]	5.1	5.88	4.40
	WICKIEWICZ et al. 1983 [30]	3.5	5.4	1.95
Pennation angle (°)	FRIEDRICH and BRAND 1990 [28]	6.5	17.5	32
	WARD et al. 2009 [29]	9.9	12.0	28.3
	WICKIEWICZ et al. 1983 [30]	17.5	8	35
Physiological cross-sectional area (cm ²)	FRIEDRICH and BRAND 1990 [28]	35.0	11.7	104.7
	WARD et al. 2009 [29]	21.1	9.7	51.8
Percentage of slow-twitch fibres	DUL et al. 1985 [31]	51	47	88
Knee flexion moment arm (mm)	SPOOR et al. 1990 [32]	27.5	29.5	xxx
	WRETENBERG et al. 1996 [33]	39.6	38.7	xxx
Plantar flexion moment arm (mm)	KLEIN et al. 1996 [21]	52.8		
	HINTERMANN et al. 1994 [34]	47		
Subtalar inversion moment arm (mm)	DUL et al. 1985 [31]	17.3	17.5	14.0
	KLEIN et al. 1996 [21]	5.3		

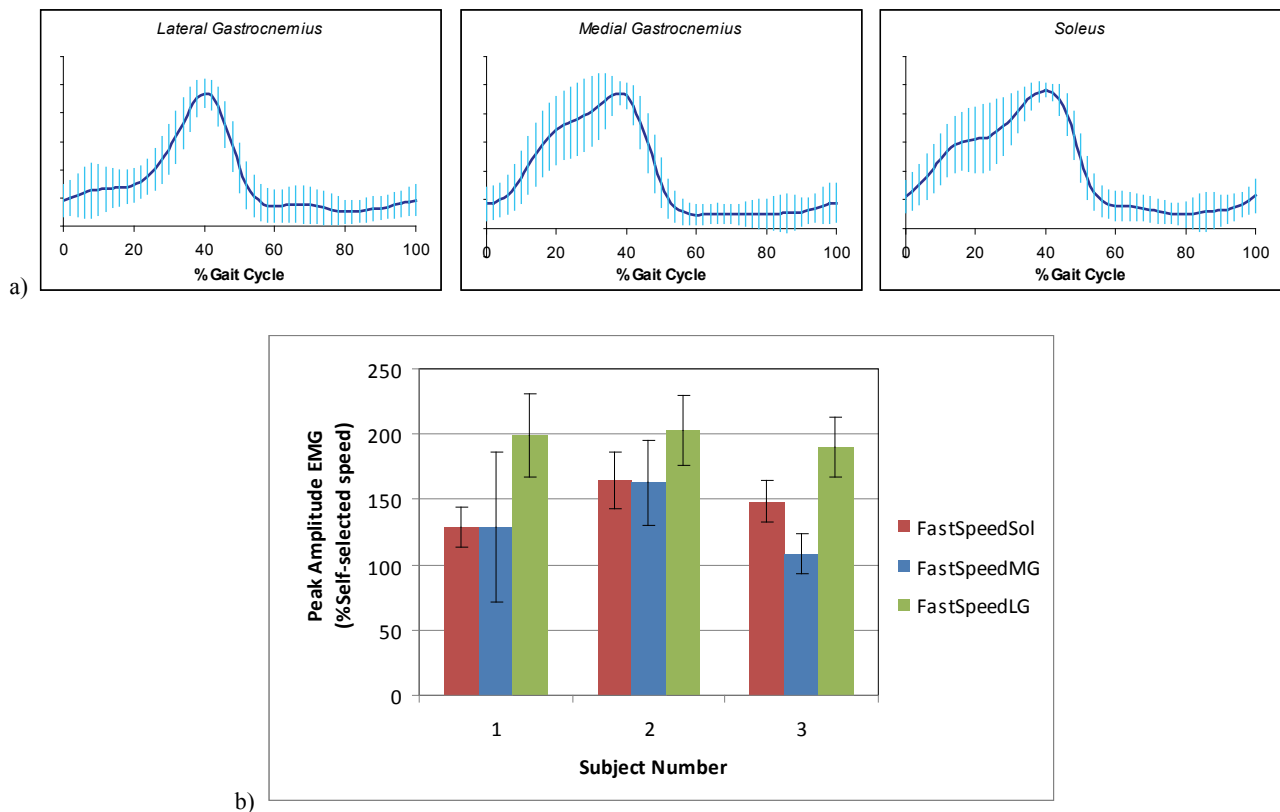


Fig. 2. EMG data illustrating activity in medial gastrocnemius, lateral gastrocnemius and soleus when walking at self-selected speed (data collected in ORLAU) (a).

The effect of fast walking speed on the amplitude of EMG signals in 3 subjects (data collected at Guy's and St Thomas') (b)

4. Biomechanical models

Figure 3 illustrates the steps needed to produce a movement pattern, and hence the steps required by any musculoskeletal simulation. Published results for the calf muscle [1], [6], [7] have yielded somewhat counter-intuitive results, contrary to the classical interpretations of the function of these muscles. In particular the gastrocnemius and soleus appear to have quite distinctive and different actions when considering the accelerations of the limb segments and joints. This is consistent with KIMMEL & SCHWARTZ (2006) [8], whose overview of induced acceleration patterns revealed particularly paradoxical actions for biarticular muscles. The actions of mono-articular muscles tended to be more sympathetic with clinicians' understanding of their roles.

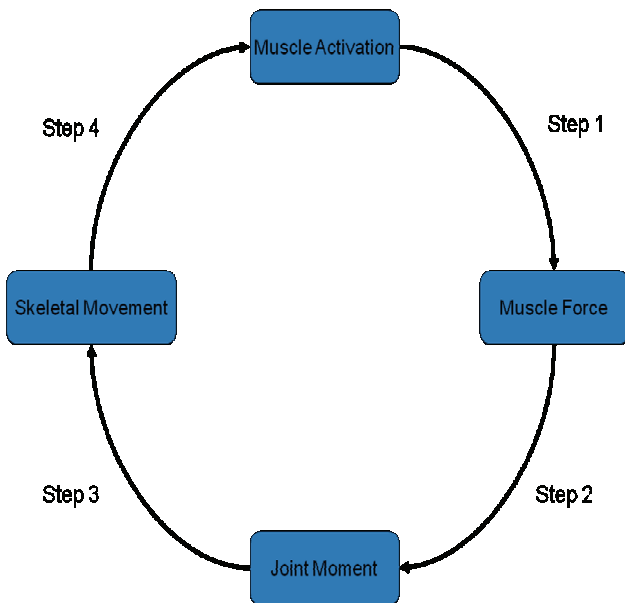


Fig. 3. Diagram showing the steps required in a musculoskeletal model-based simulation of gait

To consider the implications of these potentially surprising results it is necessary to look at each stage in the cycle (figure 3) in further detail.

Step 1: Muscle activation produces muscle force. The most common approach taken to estimate muscle forces is to apply a Hill-type muscle model [9]. Here muscle force predictions depend on the muscle's length, architecture and activation. These three factors will be considered in turn.

One of the first applications of musculoskeletal modelling was to estimate the overall length of the combined musculotendinous unit. This approach al-

lowed a dynamic estimation of muscle contracture in pathology, similar to that performed passively on the examination couch. The technique proved most useful for assessing biarticular muscles where dynamic examination of the posture of a single joint could be misleading. The clinical understanding of dynamic hamstring contracture in cerebral palsy has changed as a result of the musculoskeletal modelling techniques [10]. Children walking in a crouched posture often had hamstring muscles of normal length or longer as the shortening effects of the knee posture were counterbalanced by the lengthening at the hip. Differentiating lengths to obtain velocities may also have a role in understanding hamstring spasticity [11].

The same approach has been used to look at the biarticular gastrocnemius in children with cerebral palsy who walk on their toes [12]. The potential here for changing clinical understanding is much lower as, unlike the hamstrings, the posture of both joints tends to shorten the muscle. An additional difficulty of obtaining these values for the calf is the uncertain influence of the subtalar posture and the need to measure foot deformity.

The limitations of considering overall musculotendinous length are well illustrated by the study of FUKUNAGA et al. [13]. They measured the fascicle and tendon length changes for the medial gastrocnemius during normal walking. Large length changes were observed in the tendon, compared with the muscle, an effect masked by considerations of overall length.

To understand the roles of the muscle belly and tendon, their important architectural properties must be included in the model. The model of ZAJAC [9], which is widely used in musculoskeletal simulations, incorporates the salient features. These include the time constants of activation (determining speed of contraction and representing the fibre type distribution within the modelled muscle); muscle fibre length (determining the width of the force-length and force-velocity relationships); physiological cross-sectional area (determining the force-generating capacity of the muscle) and tendon compliance (contributes to the relationship between muscle activation and force production).

Finally, the electrical activity of the muscle must be included, and here EMG data have an important role to play. Muscle activations are often the subject of optimisation procedures in order to produce a set of muscle forces consistent with the measured joint moments (e.g. [14], [15]).

The creation of models of this type involves the introduction of many assumptions, even for the simulation of normal gait. When pathological gait is consid-

ered the issues are even more pronounced. The main assumptions and limitations are discussed below.

Muscle models tend to be parameterised by the limited data available from cadaveric specimens, with the results heavily dependent on the small number of individuals measured and the techniques used. In reality, architecture may even vary within different compartments of a single muscle. Sarcomere lengths may also differ. Muscles such as soleus have a three-dimensional structure which is not well captured by a single variable such as the physiological cross-sectional area. Another compounding factor when considering the distribution of muscle force is the possibility of myofascial force transmission between muscle groups [16]. This is not generally taken into account when muscle forces are calculated.

When a musculoskeletal model is constructed to represent a specific individual, scaling is required to the new musculoskeletal geometry. It may not be valid simply to scale muscle fibre length by stature or limb length [17]. For subjects with pathology bespoke muscle architectural parameters may be required. This is well illustrated by MALAIYA et al. [18] who used ultrasound to examine the architecture of the medial gastrocnemius in hemiplegia. They revealed shorter muscle belly lengths and smaller muscle volumes on the paretic side. The PCSA values were also different, as reported in figure 4.

Correct architectural parameters are the key to understanding the action of the calf muscle. The results

of FUKUNAGA et al. [13] show how the muscle and tendon properties combine to produce an energy-efficient gait pattern, whilst protecting the muscle bellies from excessive extension. This energy conservation will be disrupted if the properties of the tendon or muscle are altered in pathology.

Joint moments are the sum total of the action of all the muscles crossing that joint. Predictably, there is a high level of redundancy in the system and optimisation routines are often used to distribute muscle forces. In many neurological conditions, however, EMG traces show signs of disordered control. It is difficult to see how an optimisation approach can appropriately predict this pathological activation. Prediction criteria based on minimising functions of muscle activation or stress (e.g. [14], [19]) based on normal function are unlikely to be appropriate in pathology. Children with cerebral palsy, for example, are known to show high levels of co-contraction, causing overall higher levels of muscle activation and lower endurance.

The common practice of scaling EMG signals to reference contractions, normally maximum voluntary contractions, in order to derive muscle activation is also problematic, as patients with neurological problems may not be able to produce an appropriate level of muscle activation volitionally. This phenomenon was explored by STACKHOUSE et al. [20] who revealed both deficits in voluntary muscle activation and increased antagonist co-contraction.

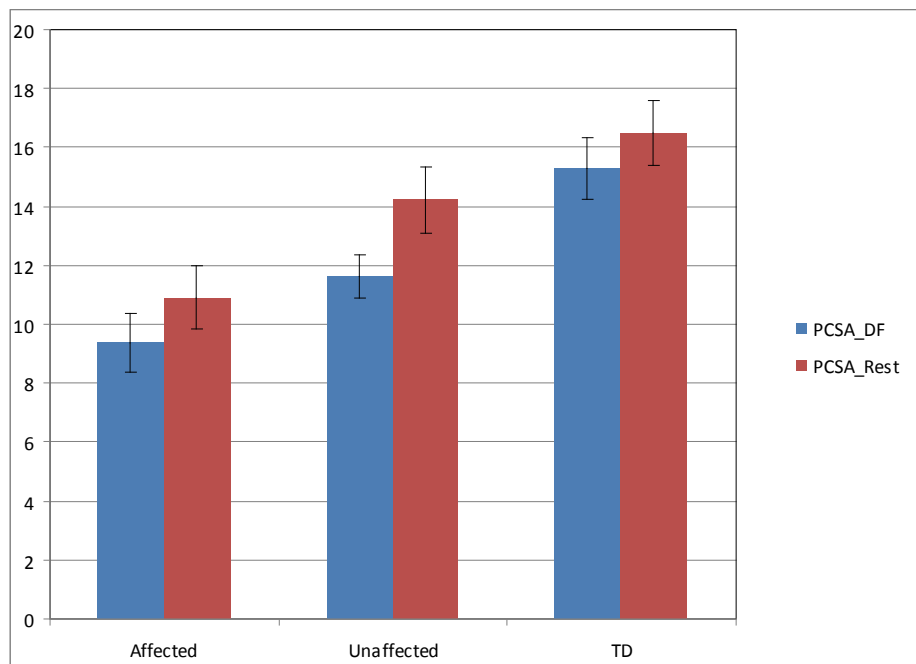


Fig. 4. PCSA values for the medial gastrocnemius for typically developing (TD) children and for the affected and unaffected limbs in children with hemiplegic cerebral palsy measured at Guy's and St Thomas' hospital. Data are presented for the ankle in maximum dorsiflexion (DF) and at rest

Those creating musculoskeletal models have an understandable desire to reduce the overall complexity of their models and, in particular, the number of variables (degrees of freedom). Historically, muscles have been classified into groups, which conveniently suggests combinations of muscles which may be bundled together and given the same architectural properties and activation. From the evidence it seems more likely, however, that muscle groups contain elements with complementary, rather than identical functions. This will allow the muscle to contribute to a wide range of different biomechanical tasks.

Those creating musculoskeletal models need to beware of removing the subtleties of normal function

and variation, or ignoring the changes introduced by pathology. The determination of muscle force is a key step in the cycle illustrated in figure 3. The sensitivity of the whole cycle to decisions made at this stage requires careful investigation.

Step 2: Muscle force produces a joint moment. A joint moment arises from the relationship between the muscle force and the axis of the joint it spans. The relationship between the force and the resultant joint moment is given by the muscle's moment arm, an expression of its leverage. A muscle with a long moment arm will produce a higher moment for a given force, but will also experience a greater change in its length if the joint changes position.

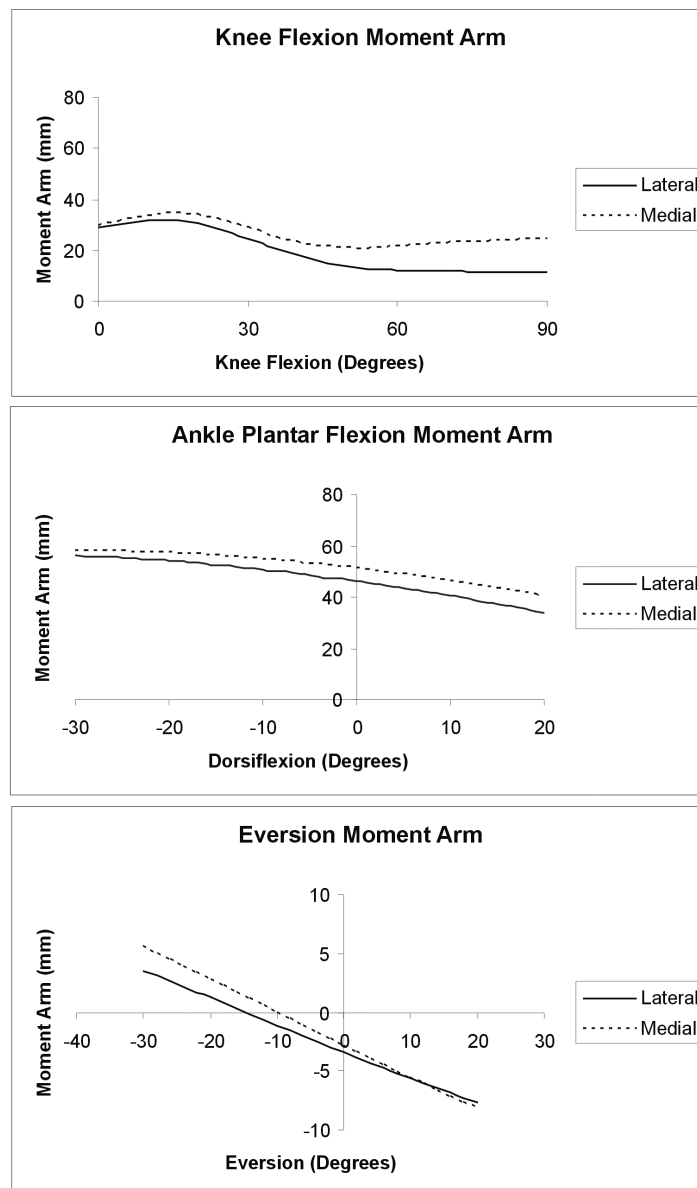


Fig. 5. Illustration of the changes in moment arm which take place across the range of motion at the knee, ankle and subtalar joints for the two heads of gastrocnemius. The data were derived from a scaled musculoskeletal model created in ORLAU

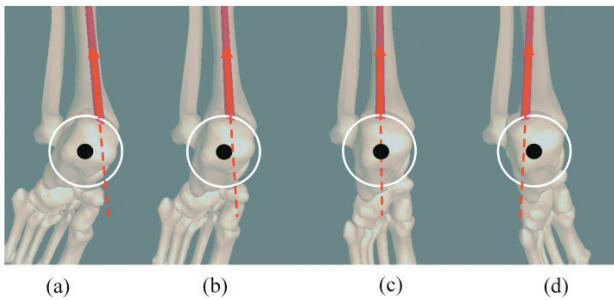


Fig. 6. An illustration of the stabilizing action of gastrocnemius at the subtalar joint. The ankle joint is in neutral and the limb is orientated such that the line of sight is straight down the subtalar axis. The axis location is marked with a black dot. The white circle shows the path of the gastrocnemius insertion as the os calcis and foot rotate around the subtalar axis. For simplicity the gastrocnemius muscle is shown as a single line of action. The four positions shown are (a) 15° eversion, (b) 0° or neutral, (c) 15° inversion and (d) 30° inversion

The table gives the moment arm data for the calf muscles. The moment arm may have important implications for understanding the roles of particular muscles in stabilising a joint. The calf muscles show a particularly interesting pattern. Figure 5 illustrates

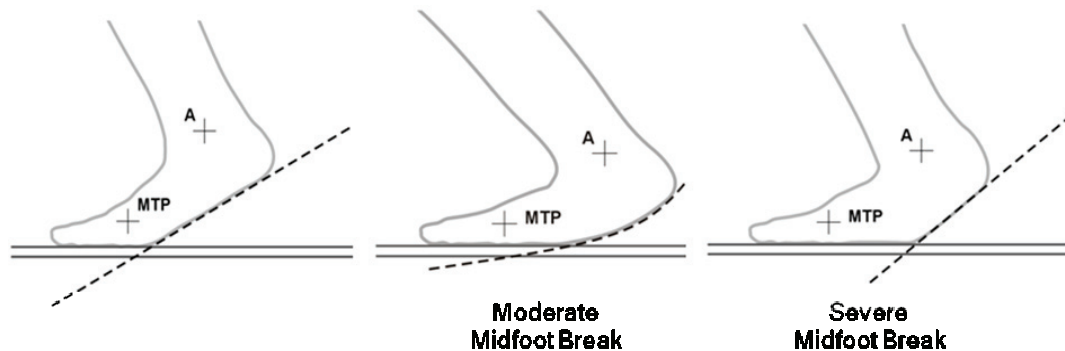


Fig. 7. An illustration of the midfoot break from JARVIS et al. (2008) [35]

the moment arm results for a model created in Oswestry. The action at the subtalar joint is unusual in that the moment arm changes sign across the range of motion. The calf muscles appear to have a stabilising action on the joint, producing an inverting moment in eversion and vice versa. An illustration of this is provided in figure 6. This particular finding is confirmed in an anatomical study [21].

Comparing moment arms with published data is an important step in the creation of musculoskeletal models. Unfortunately, there is little data in the literature that documents muscle moment arms in normal adults and data are even more sparse for children or for individuals with pathology. Often the existing values are measured for one anatomical position and there is considerable variation between studies. The

use of imaging has been shown to have potential for scaling musculoskeletal geometry to a specific individual [22], however determining muscle moment arms for specific limb postures, combining extreme joint ranges at more than one joint, still represents a considerable challenge. In reality, a muscle is also a three-dimensional structure, not a line and so a single moment arm value for a muscle may not truly express the moment arm of individual fibres. This was demonstrated for the gluteus maximus by BLEMKER and DELP [23].

As well as the “internal” moment arm, it can also be helpful to consider the “external” moment arm [7]. The external moment arm for the calf muscles depends on the length of the foot in the plane perpendicular to the axis of joint rotation. Children with cerebral palsy may also have alterations in their external lever arms, caused by valgus foot deformities affecting the mobility and structure of the subtalar joint and midfoot, often referred to as a “midfoot break”, see figure 7. Changes such as this affect the skeletal modelling of joints and ground contacts, an essential part of the next step in the simulation cycle.

Step 3: Joint moments lead to skeletal movement. If the mechanics of the musculoskeletal system can be fully defined, along with any interactions with the environment, then the equations of motion can be written. The relationships between system loads (including muscle forces) and segment accelerations, often described as induced accelerations (IAs), can then be calculated. Whilst IA data are mathematically correct, the main challenges are providing appropriate validation and interpretation.

An agreed conceptual framework would go some way to answering the critics of IA analysis (e.g. [24]). Muscle IAs are the accelerations which result from a perturbation in the force of a single muscle. Approaches using electrical stimulation, providing a close experimental replication of the theoretical analysis

[25], [26], show some potential for providing validation. STEWART et al. 2007's stimulation work showed similar counter-intuitive results for the calf muscles as those reported from IA studies [25].

What IA analysis requires is a full mechanical definition of the musculoskeletal system: its joints, segments and interaction with the environment. This has proved problematic, particular obtaining a satisfactory model for the foot–floor interaction. In common with the other steps described above, an appropriate information for modelling pathology, for example the inertial properties of segments, is lacking.

Forward dynamics takes the analysis a step further by seeking to simulate the movements in time. Numerical techniques are required to solve the non-linear equations of motion, producing segment and joint trajectories. This approach has the appeal of being a genuine simulation, as new motion patterns can be created. The difficulty of producing stable models has, however, limited the number of problems to be addressed by this technique. The computation power and time required to run simulations have also been excessive, though more efficient techniques have been developed using static optimisation to produce muscle activations compatible with the joint kinematics [15].

The vast majority of investigations of pathological gait using forward dynamic simulations have started by solving a tracking problem, based on existing gait data (e.g. [2]). Perturbations are then applied to test pathological function or possible therapeutic intervention. There is little practical validation available in the literature. Because of the complexity and redundancy of the system, achieving accurate tracking alone is not enough to prove that the solution found is the correct one.

It is perfectly possible to perform simulations, both induced accelerations and forward dynamics, using joint moments rather than muscle forces, but examples are rare [27]. Joint moment can be calculated directly for gait laboratory data and so are less prone to error or dependent on assumptions. Although, joint-based moments may not reveal the individual muscles responsible for a normal or pathological movement, understanding the contributions of individual joint moments to support and progression may help the clinician decide of strategies to ameliorate an individual's movement dysfunction.

Step 4: Skeletal movement influences muscle activation. Achieving stable models has proved challenging. Optimisation routines are required to identify the precise set of conditions needed to achieve stability across the cycle. Small residual errors, or deliberate perturba-

tions, lead to large and uncontrolled changes in joint kinematics. In practice, human gait needs to be able to adapt muscle activations responsively in order to achieve stability. Movement in animals is controlled by higher and lower centres in the central nervous system and represent a closed loop system. This is probably the greatest weakness of existing dynamic simulations, that they are essentially open loop and lack the control of the human neuromusculoskeletal system. Incorporation of algorithms that enable control of muscular activation and respond to a loss of stability in the model may help us to generate stable solutions and also to analyse and understand normal and disordered neurological control.

5. Discussion

In individuals with movement disorders such as cerebral palsy or stroke, the calf muscles are often subjected to treatments including physiotherapy (stretching and strengthening), orthotic management (ankle foot orthoses), surgical lengthening and electrical stimulation. Ideally musculoskeletal modelling approaches could contribute to the tailoring of specific treatment packages, which take into account the particular function and characteristics of the different component parts of the muscle. Despite the apparent potential of modelling techniques, clinicians should remain wary.

This paper has described the assumptions and limitations which underlie the different steps of a musculoskeletal model-driven simulation. The result is a series of analytical challenges where each is dependent on the uncertainties of the others. When combined with numerical techniques for optimisation and integration, and the absence of closed loop control, it is not particularly surprising that simulations can lack stability.

In order to create models capable of translation into meaningful clinical tools, the following characteristics should apply. Firstly, models should be **dependable**. That means that each step of the modelling process has a sound anatomical and physiological basis, with careful consideration of the effects of pathology. Every possible aspect of the model should be subject to careful validation and verification by means of practical experiments.

Secondly, models should be **stable**. This means that very small changes in the input data should not lead to large alterations in the model output. Sensitivity analysis of models should reveal whether or not this is the case. Clinical data will always be subject to

small amounts of error and uncertainty and these effects should not have an excessive impact on the results.

Thirdly, models should be **accessible**. For use in clinical practice models need to be quick and simple to run, with clear and friendly interfaces. The requirement for patient data collection should be kept to a minimum, as lengthy assessments using expensive facilities lead to high costs.

Finally, models should be **comprehensible**. This means that clinicians can have a reasonable understanding of the underlying processes. When the results make sense errors can be identified and mechanisms elaborated.

In order for a model to possess these characteristics, it will need to be analytically simple but physiologically sophisticated. This represents a significant challenge to those designing future techniques. What the authors of this paper advocate is the production of a series of models, along the lines of the steps described above, rather than a single model seeking to deal with all steps in one single simulation. We propose that an enhanced appreciation of joint moments is key to the development of better clinical understanding and treatment in the future.

Joint moments sit at the heart of the cycle described in figure 3. They are also measured outputs of clinical gait analysis. Whilst they are subject to measurement error, for example in the determination of joint centres, overall they provide a much more solid and comprehensible basis for simulation than muscle forces. The whole cycle can be summarised using two key questions. How are moments created by muscles? What do moments do to the skeletal system? These two questions can be considered independently, rather than loading them together into one common simulation.

Firstly, how are moments created? A detailed examination of pathological muscle architecture and length during the gait cycle would allow the clinician to predict the force generating capacity of an individual muscle with reasonable confidence. EMG data, where available, is then used to assess the relative activation levels through the gait cycle. The clinician can then assess whether a muscle is weak or acting out of range.

This force data then needs to be combined with information concerning bony deformities and joint axis orientations. The contribution of a specific muscle to the joint moments can hence be established for the joints which the muscle spans. From this the clinician is able to identify muscles which are acting at a mechanical disadvantage. The result is an overall appreciation of an individual muscle's role.

The action of moments on the skeletal system can then be considered separately. The information needed is the induced accelerations of the joint moments for the musculoskeletal system in that particular posture. Calculating induced accelerations, rather than forward dynamic simulations, reduces the complexity and makes the analysis more accessible. IAs based on muscle moments again reduce the complexity and allow the clinician to consider the actions of biarticular muscles at the individual joints or the two joints combined. Is the muscle having an abnormal effect because of the overall posture of the patient?

Current clinical gait analysis is based very heavily on the kinematics of joints. Most treatments are, however, directed at muscles. Even bony surgical procedures aim to realign muscle internal and external moment arms. The joint kinetics are often under-utilised in clinical practice, but they hold the key to unlocking the action of the muscles. What is required is a series of well designed tools, based on musculoskeletal modelling to elucidate both the origin and the dynamic implications of the joint moment graphs. This would allow a more detailed focus on the contribution and pathology of individual muscles.

6. Conclusion

Clinicians need to understand gait pathology in order to treat patients with mobility disorders. Current musculoskeletal modelling approaches have evolved rapidly in an attempt to incorporate all the steps of the cycle portrayed in figure 3. The resulting models have a high degree of complexity, a large number of underlying assumptions, a tendency to instability and lack experimental validation. The intellectual challenge of creating such models is considerable and interesting and useful insights have been obtained, however the models are not currently suitable for translation into clinical practice. We have proposed an alternative modelling approach for use in clinical practice, one based on an enhanced appreciation of joint moments. This would allow the clinician to assess the biomechanics of pathological gait from muscle to movement using data already familiar to them and readily available.

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