Appeared in: 31st International Workshop on Qualitative Reasoning (QR'18), Stockholm, Schweden, 2018

A Qualitative Gait Model for Diagnosis of Juvenile Idiopathic Arthritis

Peter Struss[1] and Robert Reiser[1] and Florian Kreuzpointner[2]
Technische Universität München, [1]Department of Informatics/
[2]Department of Sport and Health Sciences, Munich, Germany
email: {struss, robert.reiser, kf}@tum.de

Abstract

Juvenile idiopathic arthritis is an autoimmune disorder that causes inflammation and pain in joints and, hence, may lead to posture and movement modifications and muscular imbalance with reduced range of motion in the affected joints. In order to support diagnosis and selection of adequate therapy, certain features of the gait of patients are recorded, based on several markers attached to their legs. In our work, we attempt a model-based solution to the diagnostic interpretation of the recorded gait parameters. The model is a compositional one, with bones, joints, muscles, and the "control" of the muscles (the central nervous system) as the building blocks, and captures deviations of parameters from the nominal range. We present a first version of the model library under certain simplifications and restrictions, esp. the limitation to 2d.

1. Introduction

Component-oriented model-based diagnosis (Struss, 2008) has mainly been designed for and applied to artifacts. One of the main reasons for this is that they (usually) have a clear (designed!) structure and are often constructed using a set of building blocks, which allows for compositional modeling based on a library of reusable models. In contrast, medical diagnosis faces very complex systems, often with no obvious structure, spatially distributed organs and processes etc. In this paper, we describe an attempt to apply qualitative modeling and model-based diagnosis techniques to a medical problem that appears to be closer to the traditional applications.

The context is given by data acquisition and diagnostic techniques in the area of juvenile idiopathic arthritis (JIA), where young patients suffer from inflammation of joints, which may seriously harm their motion capabilities. In particular, their gait may be affected leading to abnormal motions. The commonly used 3d gait analysis records deviations from normal gait as a basis for diagnosis and monitoring of the patients. In order to support this task, we started the development of a compositional (simplified and qualitative) model of human legs and gait, which can be exploited by model-based diagnosis.

Of course, two legs in motion form a complex mechanical system. Inspired by the kind of expert interpretation of the patient data, we start with a qualitative, purely geometrical 2d model of a leg, ignoring the complex kinetics and its control by the central nervous system (CNS). This is what we present in this paper.

We start by describing JIA and its diagnosis and therapy and, in particular, standard 3d gait analysis. Section 3 and 4 introduce the foundations of the model related to the geometrical and mechanical aspects and the control of the motion. The models based on these constraints are presented in section 5. Then we discuss first diagnostic results and open questions and future work.

2. Diagnosis and Therapy of JIA

The EULAR (the European League Against Rheumatism) proposed the term juvenile idiopathic arthritis for the heterogeneous group of disorders that manifest as juvenile arthritis (inflammation of a joint). The definition implies that the arthritis begins before the age of 16 years and lasts for at least 3 months (Sherry et al., 2011).

2.1. The Disease

JIA is the most common chronic arthritis in childhood and adolescence. It is an autoimmune and autoinflammatory disease, i.e. there is an immune response against the constituents of the body's own tissues.

This reaction is characterized by joint inflammation, pain and swelling which have an impact on the muscle function and influences the human gait (Hartmann et al., 2010).

According to the classification of the International League Against Rheumatism (ILAR), there are seven subclasses of JIA (Petty et al., 2004). All have a different impact on the muscle function because they cause restrictions at different levels. As JIA is a very heterogeneous disease, it is very important to know the various factors that influence muscle function.

2.2. Diagnosis and Therapy

The etiology of the disease is not clear yet. Additionally, there is no consistent pattern of joint involvement. This

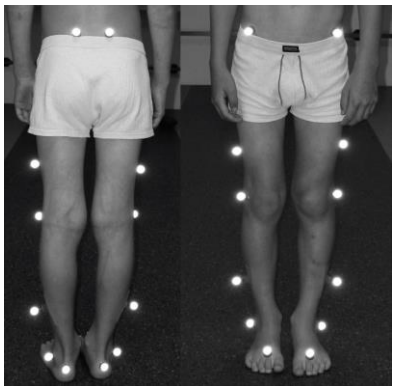


Figure 1 Reflecting markers (Hartmann et al., 2010)

means that under a functional point of view, the disease is very heterogeneous. One subclass, polyarticular JIA (this means that the patient has more than five involved joints), can have six involved joints on the lower extremity or six involved joints on the upper extremity.

The joint involvement has a big impact on the muscle function, which determines the motion sequence of a movement. If the muscle function is restricted there is an anomaly in the motion which limits the patient, for example, in the way they walk.

Patients with JIA are treated within their therapy in a multimodal way. Two major parts of treatment are both medical therapy and functional therapy like physiotherapy and exercise therapy. Drugs treat the inflammatory process, and functional therapy treats the resultant restrictions of the involved joint.

Joint inflammation causes degenerative effects within the joint, which leads to changes of the muscle function.

Pain, dysfunction of the agonist muscles or bony restrictions can affect the ordinary gait function. In terms of treatment planning it is necessary to have a closer look at the outcome of a gait analysis.

2.3. Gait Analysis and its Contribution to Diagnosis and Therapy

Three dimensional gait analysis (3dGA) is a very powerful method to quantify muscle function during walking or running. In general, a standard 3dGA consists of kinematic and kinetic analysis. Some labs use electromyography (EMG), as well. Kinematic parameters are related to joint angular displacements. Kinetic parameters include external ground reaction forces during movement. One can calculate joint loadings with both parameters using inverse dynamics. EMG analyzes the muscular activation that is necessary for quantifying the neuromuscular processes or human locomotion. The most commonly used outcome parameters are kinematic parameters. They are expressed by joint angle displacements.

Today, (3d) gait analysis is a part of a routine procedure in a clinical setting used to quantify movement restrictions to individualize physiotherapy. Basically, gait analysis is performed on an around 10 m long and 3 m wide gait floor in a lab environment. The lab is equipped with a 3d-motion analysis system including at least six infrared cameras, measuring at 120 Hz (e.g. Vicon, MX3) and at least one 3d ground reaction force plate (1080 Hz) (AMTI). The patients are marked in accordance with the Plug-in-Gait Model for the lower extremities (Davis, 1997) with 16 reflecting markers (⌀ = 14 mm) (Figure 1).

After post processing the data, the marker trajectories are translated into joint movements during walking, which is normalized to a gait cycle from 0-100%. A gait cycle lasts from heel strike of one side to the next heel strike of the same foot. The main focus lies in the interpretation of the sagittal plane. Figure 2 shows the movement pattern of the ankle joint over one gait cycle in the sagittal plane (i.e. the yz-plane where z denotes the vertical axis and y is the walking direction) which indicates the range of the joint angle during different phases. In this Figure, the vertical axis represents the plantar- and dorsiflexion angle of the ankle joint. The gray-shaded area is the standard deviation around the black solid line, which is the mean of a healthy age matched control group. This is compared to a single patient with JIA. Again the red shaded red area is the standard deviation of the patient with the black solid line which is the mean out of five gait trails. Combining multiple trails of a single patient is done to cover the variability of the gait patterns. In this example, one can see that the patient has a limited maximum plantar flexion, which takes place around push off (vertical line in the Figure). This means that the patient has less potential to accelerate the center of mass during push off.

One of the difficulties of the interpretation is that different causes (ranging across different time scales) may cause the same abnormalities. For instance, if the flexion of a joint is reduced the cause may be

- an inflammation in the joint causes pain for certain extreme ranges of joint angles. As a response, the CNS attempts to confine the range of the joint angle in order to avoid the pain.

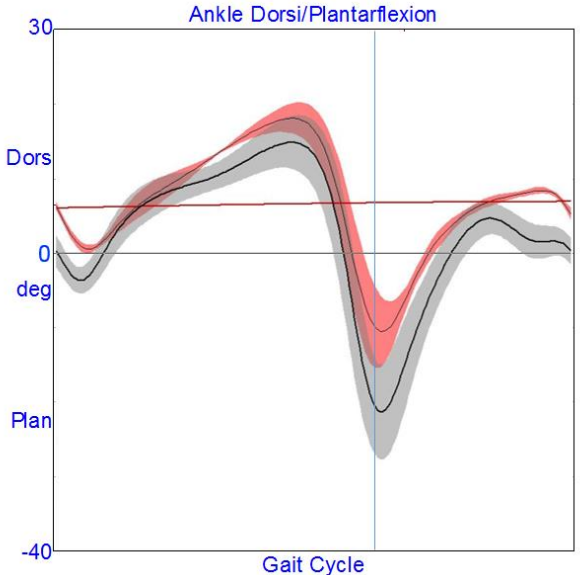


Figure 2 Ankle joint motion in the sagittal plane during walking

- After some time, the modified control regime may actually persist, although the former origin, the inflammation, is no longer present.
- Finally, if the muscle has been restricted in its performance for some time, it may lose its capabilities to operate beyond these restrictions, for instance, achieving a complete contraction.

These causes cannot be discriminated without further information, such as history of the patient's disease, inflammatory values, and additional tests

3. Model-based Gait Analysis – Foundations

It is obvious that computer-based support diagnosis based on the data outlined above has to be knowledge-based, as opposed to data-driven, and the objective of our work is exploring whether and to what extent model-based diagnosis can contribute to the interpretation of the measurements by identifying potential causes for observed deviations of joint study.

In this chapter, the foundations and essential constraints of our model are introduced.

3.1. Component-oriented Modeling

Our approach to modeling the gait is a qualitative, compositional, component-oriented one. This means, we identify different types of entities that have a specific behavior and several instances in the system. These instances ("components") interact with each other, and their combined behaviors establish the overall behavior of the entire system, in a normal or disturbed way.

Developing a compositional model (rather than a black-box model of the entire leg or pair of legs) provides transparency in terms of a clear model structure, supports modifying and extending the model and bears the advantage of re-using model fragments for other purposes (such as differential diagnosis or therapy generation) and other parts of the body.

In our application, the obvious main component types involved in the mechanics of human gait are

- **Bones**
- **Joints**
- **Muscles**

Of course, there are many other parts of the leg involved, but since they are not considered as potential problem sources in our domain, they are neglected. Tendons, for instance, can be treated as part of the muscle or could be seen as the passive connections between bones and muscles, manifested by the respective **terminals** that connect the models of the components and allow sharing of information between them.

In addition to the three main component types, there is the

- **Central nervous system (CNS)**

as the element that determines the gait by controlling the muscles.

3.2. Modeling Decisions and Assumptions

In principle, the human gait is an incredibly complex dynamic process involving, for instance, the interaction of agonist and antagonist muscles (flexor and extensor muscle) in a 3d space. Detailed and numerical modeling has been attempted e.g. in (Koning et al. 2015). Using such models for our purposes appears to be prohibitive due to its complexity. It would also be overly detailed and useless, because the required numerical parameters for tailoring it to an individual patient, and fail to relate to the human diagnostic reasoning. Our model involves several significant simplifications. The expectation that it will serve its purpose is based on the consideration of how human experts perform diagnostic reasoning.

- The **content** of the model: At least a major part of the human diagnostic reasoning seems to ignore the kinetics of the gait. This is why our first model exclusively captures the **geometrical interdependencies only** and ignores forces, momentum, acceleration etc.
- The **granularity** of the model: the description of the gait of an individual patient is based on an abstraction of the numerical measurements to a qualitative level, in determining whether and in which direction angles or positions deviate from envelopes around the nominal curves.
- **Structural reduction**: as already stated above, we obviously have to omit many elements of the body from the model. In particular, we consider **only one pair of muscles for each joint** (muscles come in pairs with one causing the flexion and the other one the extension of the joint when contracting). In reality, there are more muscles affecting the joint. However, in the JIA domain, considering them separately is not feasible and would not contribute to a refined diagnosis, anyway. Another simplification, which is likely to be dropped in the future, is that we **do not consider muscles that work across two joints**.
- **Two dimensions** only: we consider the sagittal plane only, i.e. the one spanned by the vertical axis and the one in the direction of the gait. Also this simplification has to be overcome in more sophisticated models, since avoidance of pain is often achieved by or leads to distortions in the 3rd dimension.

The currently implemented model is additionally limited to

- **one leg** only, which is a restriction, because a deviating motion of one leg may have an impact on the motion of the other one.
- the periods of the gait where it **carries the load** of the body, which is justified by the fact that abnormalities are more likely to show under a load, rather than during the swing phase.

There are other, more or less restrictive modeling assumptions, which we will make explicit when presenting the models in the following.

3.3. The Involved Constraints

3.3.1. Deviation Models

In order to reflect the representation of the gait based on the measurements and its qualitative interpretation as described in section 2, we consider deviations from nominal

coordinates and angles, more specifically, deviations of maximal or minimal angles.

As in (Struss, 2004), for a variable x, we define the **deviation** Δx as the sign of the difference between an actual (or hypothesized) value and a nominal one

(1) $\Delta x := sign\,(x_{act} - x_{nom})$.

For monotonically increasing (decreasing) functions M+ (M-, resp.), we will exploit

(2) $v1 = M^{+}(v2) \Rightarrow \Delta v1 = \Delta v2$,
$v1 = M^{-}(v2) \Rightarrow \Delta v1 = -\Delta v2$

Based on this, we present the basic constraints that will form the core of the component models, both for the magnitudes of the involved variables (which will not appear in the model, since they are numerical interdependencies) and those for their qualitative deviations that are obtained from them.

3.3.2. Geometric Definitions

In our representation, the vertical axis is z, while x denotes the axis in the direction of the motion (Figure 3). In the representation used, components have terminals as connection points to other components, carrying their shared variables, such as positions. And components have local (state) variables, for instance, the angle of a bone. In our notation, for instance, $Bone.T_{prox}.\Delta x$ refers to the deviation of x in the proximal terminal of Bone, and $Bone.\Delta\alpha$ the deviation of the angle of Bone. Here, the **proximal** terminal (or adjacent component) of a component is the one towards the center of the body, while the other one (towards the foot) is the **distal** one.

- The **bone angle** α is the angle between the vertical axis with the origin placed in its terminal T_{prox} and the bone in counter-clockwise direction (Figure 3).
- The **joint angle** ϵ is the angle between the two connected bones, also in counter-clockwise direction.

3.3.3. Position of a Bone

Based on these definitions, we have for the **magnitudes** of the coordinates of the terminals and the bone angle

(3) $T_{dist}.z = T_{prox}.z + cos(\alpha) * Bone.length$
(4) $T_{dist}.x = T_{prox}.x - sin(\alpha) * Bone.length$

Where Bone.length is the length of the bone.

We introduce landmarks 0, 90, 180 and 270 and the intervals between them as the qualitative domain of α and use $\ominus$ for subtraction in the sign domain. Because cos and sin are piecewise monotonic, if the deviations in α do not cause a crossing of minima or maxima, we obtain the simple constraint between the deviations of α and the coordinates.

(5) $Bone.\alpha_{act} \in \{(0,90), 90, (90,180)\}$
$\wedge\, Bone.\alpha_{nom} \in \{(0,90), 90, (90,180)\}$
$\Rightarrow Bone.\Delta\alpha = Bone.T_{prox}.\Delta z \ominus Bone.T_{dist}.\Delta z$
$etc.$
$\Rightarrow Bone.\Delta\alpha = Bone.T_{dist}.\Delta z \ominus Bone.T_{prox}.\Delta z$

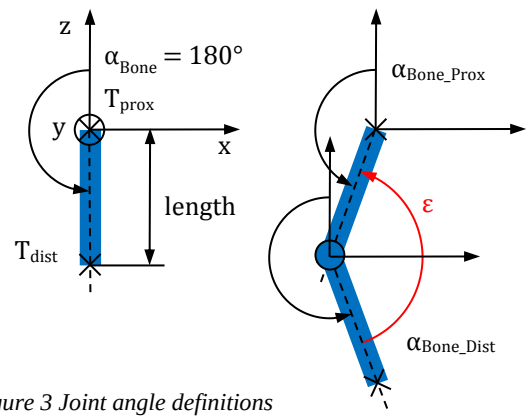


Figure 3 Joint angle definitions

3.3.4. Angles at Joints

According to its definition (see Figure 3), the magnitude of the joint angle ϵ is

(6) $Joint.\epsilon = 180° + Bone_{prox}.\alpha - Bone_{dist}.\alpha$

and its deviation $\Delta\epsilon$

(7) $Joint.\Delta\epsilon = Bone_{prox}.\Delta\alpha \ominus Bone_{dist}.\Delta\alpha$

3.3.5. Impact of Muscle Length

Muscles apply forces to the bones and can modify their relative angles. While we do not explicitly represent the forces in our model, we do represent the monotonic relation between the length of a muscle and the angle difference of the bones it is attached to. If $Muscle_{Front}$ is the one acting on the front side, i.e. in the direction of the motion, and $Muscle_{Back}$ the one in the back, we obtain for the magnitude of ϵ

(8) $Joint.\varepsilon = M^{+}(Muscle_{Front}.length)$
$Joint.\varepsilon = M^{-}(Muscle_{Back}.length)$

Which yields

(9) $Joint.\Delta\varepsilon = Muscle_{Front}.\Delta length$
$Joint.\Delta\varepsilon = -Muscle_{Back}.\Delta length$

for the deviation. Note that this holds independently of which muscle acts as the flexor or extensor, respectively. (This is captured by the possible range of ϵ).

3.3.6. Combination of Muscle Pairs

(8) and (9) indicate that the lengths of the corresponding front and back muscles are coupled; one cannot contract if the other one does not expand. We introduce a variable ∂length to represent the difference to the rest length of a muscle. ∂length=– means contraction (which happens actively), ∂length=+ characterizes an extension (caused by another force, usually the contraction of the antagonist muscle acting on the same joint). Being connected to opposite sides of the same joint, the muscles of a pair are negatively coupled:

(10) $Muscle_{front}.\partial length = -\,Muscle_{back}.\partial length$

And so are the deviations, as captured by (9).

This creates a modeling problem. We cannot simply model the normal behavior of a muscle by stating that it never has a deviating length: if its counteracting muscle suffers from an abnormality and, hence, creates a deviation of the joint angle, the length of the other muscle will be abnormal, too. Thus, it cannot be represented which one is **causing** the deviation. Therefore, we introduce a variable $\Delta length_{pot}$ that represents the **potential** of a muscle regarding its length. If the muscle is working correctly, this deviation regarding the capability of the muscle, will be zero, but its

actual length may deviate, because the potential of the other muscle is abnormal. Only if both potential lengths are normal, the resulting lengths for both will be nominal. This is captured by

(11) $Muscle_{Front}.\Delta length =$
$Muscle_{Front}.\Delta length_{pot}$
$IF\ Muscle_{Front}.\Delta length_{pot} \neq 0$
$-Muscle_{Back}.\Delta length_{pot}$
$IF\ Muscle_{Back}.\Delta length_{pot} \neq 0$
0 ELSE

4. Gait Phases

The human gait is a cyclic movement, which is normally represented by a gait cycle (whitch starts with a heel strike and lasts until the upcoming heel strike of the same side). Therefore, a gait cycle consists of a stance phase and a swing phase. Currently, we only consider the stance phase. This phase is further divided into 5 subphases:

(1) Initial contact (I)
(2) Loading response (Load. Resp.)
(3) Midstance (SiSp. Mid)
(4) Terminal stance (SiSp. Term)
(5) Pre-swing

The characterization of these phases in terms of the model variables (angles and coordinates) and their deviations is shown in Table 2.

During the different gait phases, the contribution of the muscles to the respective motion varies. In principle, all muscles are somehow involved in all phases. However, some are crucial to achieving the essential dynamics of a gait phase, while others play a secondary role, e.g. by stabilizing the motion. For instance, during the Midstance phase, an essential motion is stretching the knee, which is achieved by contraction of the – in our model – KneeFrontMuscle, which is the m. quadriceps fermoris. Essential muscle contributions are not confined to contraction. Also, a controlled extension of a muscle may be crucial. While, in a muscle contraction, (neglecting external forces) the fibers themselves can only shorten or remain static, it is also possible that a muscle contracts eccentrically. This happens under an acting external force that is greater than the muscle force when the fibers lengthen while actively creating a resistive force. For example, during the Loading Response Phase the same KneeFrontMuscle, by a resistive extension, prevents a too sudden bending of the knee due to the body weight.

Diagnosis considerations suggest that the muscles that are strongly engaged in achieving the characteristic motion are also the ones that may cause significant modification of the gait when they function or are controlled in a wrong way. In addition, the antagonist muscles of contracting ones may disturb the motions by counteracting in an abnormal way. Hence, we represent each gait phase also by characterizing patterns of muscle activities, which are summarized in Table 3, which lists the essential commands to the different muscles for each gait phase.

5. Component Models

In this section, we introduce the types of terminals that are attached to the component and the domains of the variables used and, based on this, the different component types.

5.1. Domains and Terminal Types

The defined domains are shown in Table 1. CmdDomain captures the different ways the CNS stimulates the muscles. Besides the ones that occur in the normal gait as shown in Table 3, there are commands that aim at limiting the contraction or extension of a muscle in order to limit the resulting joint angle.

PainDomain allows describing whether and in which position pain occurs in a particular joint. Although this is not visible in gait analysis, information about painful motions can be obtained by separate examinations of individual joints.

Table 1 Domains

Domain	Values
PainDom	NoPain, StretchPain, BendPain, BothPain
Sign	-, 0, +
AngleDom	0, (0, 90), 90, (90, 180), 180, (180, 270), 270, (270, 360)
CmdDom	none, Contraction, ResistiveExtension, LimitedContraction, LimitedExtension

There are two types of terminals in the physical system:

- BoneJoint (BJ): Enables sharing of end positions and angle α of bones between them and joints
- JointMuscle (JM): Communicates (deviations of) muscle lengths to the joint and, hence, captures the respective variables

The CNSis connected to joints and muscles through the following terminals:

- Pain (PT) to receive an indication of pain from joints
- Command (CT) to transmit control commands to muscles

The decomposition of the leg model into components of different types (summarized in Table 4) does not reflect exactly the physical objects that constitute the leg. The main reason for this lies in the nature of a joint, which represents the interaction of two bones and (in our simplified version) one muscle pair.

5.2. Joint

A **joint** is considered as a unit comprising the ends of two bones, has two BoneJointTerminals, and determines the relative position and motion of the two connected bones (via constraints (7)). Furthermore, it is connected to one back and one front muscle by two JointMuscleTerminals and mediates their interaction according to constraint (10), although, in reality, the muscles may not be attached close to the joint. The rationale behind this is that we do not want to model forces on the bones and the torque produced (because their endpoints are fixed by the joint).

Table 2 Definition of gait phases

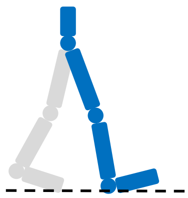
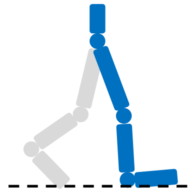
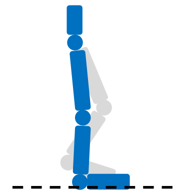
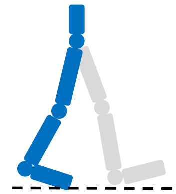
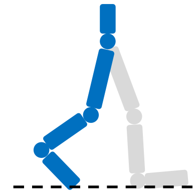
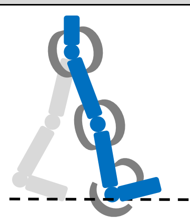
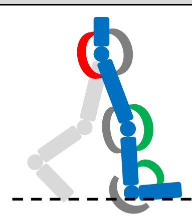
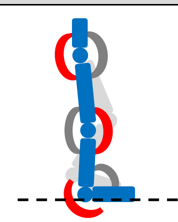
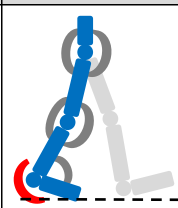
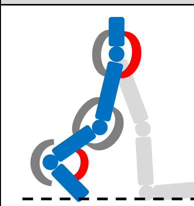
	(1) Init. Contact	(2) L. Response	(3) SiSp. mid	(4) SiSp. term	(5) Pre-swing
					
Pelvis	$\alpha = 180$ $\Delta\alpha = 0$ $Tprox.z = +$ $Tdist.z = +$	$\alpha = 180$ $\Delta\alpha = 0$ $Tprox.z = +$ $Tdist.z = +$	$\alpha = 180$ $\Delta\alpha = 0$ $Tprox.z = +$ $Tdist.z = +$	$\alpha = 180$ $\Delta\alpha = 0$ $Tprox.z = +$ $Tdist.z = +$	$\alpha = 180$ $\Delta\alpha = 0$ $Tprox.z = +$ $Tdist.z = +$
Thigh	$\alpha = (180,270)$ $Tprox.z = +$ $Tdist.z = +$	$\alpha = (180,270)$ $Tprox.z = +$ $Tdist.z = +$	$\alpha = 180$ $Tprox.z = +$ $Tdist.z = +$	$\alpha = (90,180)$ $Tprox.z = +$ $Tdist.z = +$	$\alpha = (90,180)$ $Tprox.z = +$ $Tdist.z = +$
Shank	$\alpha = (180,270)$ $Tprox.z = +$ $Tdist.z = 0$ $Tdist.\Delta z = 0$	$\alpha = (180,270)$ $Tprox.z = +$ $Tdist.z = 0$ $Tdist.\Delta z = 0$	$\alpha = 180°$ $Tprox.z = +$ $Tdist.z = 0$ $Tdist.\Delta z = 0$	$\alpha = (90,180)$ $Tprox.z = +$ $Tdist.z = +$	$\alpha = (90,180)$ $Tprox.z = +$ $Tdist.z = +$
Foot	$\alpha = (270,360)$ $Tprox.z = 0$ $Tprox.\Delta z = 0$ $Tdist.z = +$	$\alpha = (270,360)$ $Tprox.z = 0$ $Tprox.\Delta z = 0$ $Tdist.z = +$	$\alpha = 270$ $Tprox.z = 0$ $Tprox.\Delta z = 0$ $Tdist.z = 0$ $Tdist.\Delta z = 0$	$\alpha = (180,270)$ $Tprox.z = +$ $Tdist.z = 0$ $Tdist.\Delta z = 0$	$\alpha = (180,270)$ $Tprox.z = +$ $Tdist.z = 0$ $Tdist.\Delta z = 0$

Table 3 Discrete muscle activities for the gait phases

	(1) Init. Contact	(2) L. Response	(3) SiSp. mid	(4) SiSp. term	(5) Pre-swing
					
Pelvis stretch	(No phase)	Contraction	Contraction	none	none
Pelvis bend	(No phase)	none	none	none	Contraction
Knee stretch	(No phase)	resistiveExtension	Contraction	none	none
Knee bend	(No phase)	none	none	none	none
Ankle stretch	(No phase)	none	Contraction	Contraction	none
Ankle bend	(No phase)	resistiveExtension	none	none	Contraction

As a consequence, the angle between the bones at the joint is determined by the length of the muscles as indicated by constraint (9), and only one terminal is needed for the connection of each muscle. Also the determination of the muscle length deviations based on the deviations of the potential lengths happens through the joint and, hence, constraint (11) is part of its model (see Figure 4). Finally, the joint model identifies the coordinate deviations on its two BoneJointTerminals. All these constraints hold unconditionally, i.e. independently of the behavior mode of the joint. These modes are only distinct w.r.t. the existence and type of pain in the joint, which is shared with the central nervous system via the PainTerminal, and accordingly, the

"faut modes" are BendPain, StretchPain, BothPain (fixing Tpain.pain to the respective value) and OK (NoPain).

5.3. Bone

A **bone** simply represents the rigid connection of its ends, i.e. the respective joints, carries the resulting constraints (5) (see Table 4) and has no fault models.

5.4. Muscle

A **muscle** is connected to one joint, as depicted in Figure 4, and receives commands from the CNS via its CommandTerminal. The constraints for its different behavior modes have to capture the response of the muscle to these different commands which is then shared with the connected joint. For instance, in its nominal model, we have the tuple

(Tcmd.command=Contraction, ∂length=-, $\Delta length_{pot}$=0),

i.e. the muscle contracts as requested, while the fault mode LimitedContraction contains

(Tcmd.command=Contraction, ∂length=-, $\Delta length_{pot}$=+),

i.e. it does not fully contract. They share the tuple

(Tcmd.command=LimitedContraction, ∂length=-, $\Delta length_{pot}$=+).

While this deviation only shows when the muscle is actively commanded, the failure mode LimitedExtension may have an impact also when it is passive (i.e. receives command none): when its antagonist is contracting, it will restrict its effect due to its limited length. The model contains

$\partial length=+ \Rightarrow \Delta length_{pot}=-$,

which holds under the command ResistiveExtension, but also for Command = none, if the antagonist muscle contracts and causes an extension.

5.5. Central Nervous System

The CNS controls the gait by commanding the muscles appropriately in the various gait phases. It issues commands to the muscles via six CommandTerminals and receives pain signals from the joints via three PainTerminals. Its model is an association of muscle activation patterns with gait phase potentially influenced by indications of pain. Table 3 can be read as the proper association if there is no pain signaled. Otherwise, dependent on which joint indicates which kind of pain, the commands are modified, primarily to their limited version. For instance, in our example, if there is BendPain in the ankle, in gait phase 5, the command to the ankle.FrontMuscle, i.e. the flexor muscle can be modified to LimitedContraction (cf. Table 3).

Table 4 Components

	Joint	Bone	Muscle
Terminals	Tprox (BJ), Tdist (BJ), Tfront (JM), Tback (JM), Tpain (PT)	Tprox (BJ), Tdist (BJ)	Tjoint (JM), Tcmd (CT)
State Vars.	$\Delta\epsilon$ (Sign)		
Constraints	(7), (9), (10), (11)	(5)	

Currently, the only fault model of the CNS is unrestricted behavior, which covers any inadequate control, e.g. due to previously necessary, but meanwhile obsolete compensatory actions. Incorporating this kind of information would require exploiting the anamnesis of the individual patients. CNS are connected to the respecitve muscles (see Figure 4).

6. Diagnosis with the Model – First Results

The model library described above has been implemented in Raz'r (OCC'M Software GmbH, 2011). The overall system model is established as a sequence of bones and joints from the hip to the foot with muscles connected to the back and front of each joint. In addition, the PainTerminal of each joint is connected to the CNS to transmit pain experienced, and the six CmdTerminals of the CNS are connected to the respective muscles.

Furthermore, we introduced a virtual component, "Examination", that serves as a container to add observations obtained from specific examinations and lab data. Technically, it is connected to the terminals of the other components, esp. the PainTerminals. Currently, it captures inflammatory values (if they are not abnormal, this implies the absence of inflammations and, hence, pain in the joints) and the results of tests of individual joints that would reveal pain in specific positions. In future extensions, this may also include explicit information about individual muscles. All this is meant to help confining the diagnoses obtained from the interpretation of the gait analysis results alone.We evaluated the model using Raz'r's consistency-based diagnosis engine based on several scenarios that correspond to qualitative deviations of joint angles (in case they lie outside the normal range, as illustrated in Figure 2) and positions that can be extracted manually or, in future solutions, automatically from 3dGA data of patients. This is done for the various gait phases described in section 4. Additional observations can be added via the Examination component explained above. Such cases were

- Gait Phase 4, Ankle.$\Delta\epsilon$ = –
- Gait Phase 2, Knee.$\Delta\epsilon$ = –
- Gait Phase 4, Hip.$\Delta\epsilon$ = +
- Gait Phase 2, Ankle.$\Delta\epsilon$ = +

Despite the simple structure of the model and the involved modeling assumptions, the results obtained so far suggest that the purely geometrical model is able to reproduce an

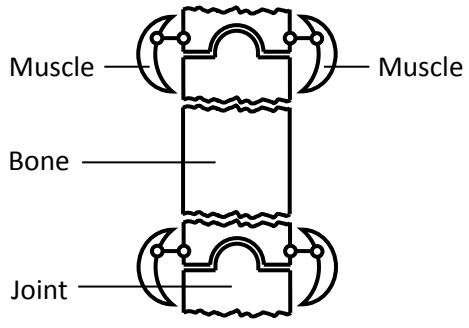


Figure 4 Components

essential part of the conclusions drawn from the data by human experts. The model is able to generate plausible diagnoses, usually expectedly alternative ones. For instance, in the second of the above cases, model-based diagnosis hypothesizes that the muscle KneeFront suffers from LimitedExtension or WrongControl of CNS (excluding a response to pain in the knee). This delivers the kind of information needed to determine additional examinations (in the documented case, the doctor's conclusion was "Function check of both knee muscles").

The introductory case from section 2 (reduced angle at the ankle in phase 4) illustrates limitations of the current solution: the conclusion about the reduction of the push-off force and, hence, of the step width is beyond the scope of the current model: it does not capture forces and momentum, does not handle the swing phase, and would only be able to yield deviations within a single gait phase.

After consolidating the model, we will start an evaluation based on a larger set of cases that have been recorded together with the results of their interpretation by human experts at the German Center for Pediatric and Adolescent Rheumatology, Garmisch-Partenkirchen.

7. Discussion and Future Work

As mentioned above, the model is based on a number of modeling decisions and simplifying assumptions. Some of them appear appropriate for gait analysis and not restrictive. Others are, and we plan to drop them in order to overcome limitations. This will not follow academic motivations related to completeness or theoretical correctness, but be driven by necessities for obtaining better und more useful diagnoses. The planned evaluation will yield criteria for prioritizing modifications and extensions. Some candidates for restrictions to be overcome are, in the order of our current priorities,

- **Restriction** to 2d: sometimes, the impact of an abnormality or the attempt to avoid resulting pain can only be described and observed in the dimension we ignored in our model (e.g. bending a foot inward). Even more fundamentally, the 2d description of the motion of the hip seems quite inadequate.
- **Independent analysis of the two legs**: it seems quite obvious that an abnormality of one leg can affect the other one. For instance, if a leg in the Midstance phase is not fully stretched the swinging of the other leg may have to be modified.
- **Muscles across one joint only**: there are relevant contributions by muscles that act on non-adjacent bones, i.e. have an impact on two joints.
- **Independent analysis of gait phases of one leg**: a deviation created in a certain gait phase may carry over to or affect the following one. It may even be the case that certain characteristic parts of the motion of a gait phase may shift in time and into the next gait phase.
- **Restriction to the load phases**: we have not yet applied the model to the swing phases of the leg, because they usually do not exhibit significant abnormalities (in our context). Actually, this may challenge the model, because it lacks a notion of forces and momentum. Actually, the fact that the foot is fixed during the load phases adds a strong constraint to the model and the observations.

Acknowledgements

The authors would like to thank Josephine Merker, Matthias Hartmann (Motion Analysis Lab) and Prof. Dr. J.-P. Haas (Medical Director) from the German Center for Pediatric and Adolescent Rheumatology for providing one example from a patient (Figure 2) and the discussions. Also special thanks to Florian Grigoleit for his input.

References

(Davis, 1997) Davis, R.B., 1997. Reflections on clinical gait analysis [online]. *Journal of Electromyography and Kinesiology*, **7**(4), 251-257. Available from: 10.1016/S1050-6411(97)00008-4

(Hartmann et al., 2010) Hartmann, M., F. Kreuzpointner, R. Haefner, H. Michels, A. Schwirtz, and J.P. Haas, 2010. Effects of juvenile idiopathic arthritis on kinematics and kinetics of the lower extremities call for consequences in physical activities recommendations. *International journal of pediatrics*, **2010**.

(Koning et al. 2015) Koning, B.H.W., Krogt, Marjolein M. van der, C.T.M. Baten, and B.F.J.M. Koopman, 2015. Driving a musculoskeletal model with inertial and magnetic measurement units [online]. *Computer Methods in Biomechanics and Biomedical Engineering*, **18**(9), 1003-1013. Available from: 10.1080/10255842.2013.867481

(OCC'M Software GmbH, 2011) OCC'M Software GmbH, 2011. FMEA, Testing, Monitoring and Diagnosis (on-board and off-board) are typical applications [online]. Raz'r Model Editor Version 3.0.1107.0 [viewed 2 February 2018]. Available from: http://www.occm.de/

(Petty et al., 2004) Petty, R.E., T.R. Southwood, P. Manners, J. Baum, D.N. Glass, J. Goldenberg, X. He, J. Maldonado-Cocco, J. Orozco-Alcala, A.-M. Prieur, M.E. Suarez-Almazor, and P. Woo, 2004. International League of Associations for Rheumatology classification of juvenile idiopathic arthritis: second revision, Edmonton, 2001. *The Journal of rheumatology*, **31**(2), 390-392.

(Sherry et al., 2011) Sherry, D.D., A.R.S. Bhaskar, M. Poduval, C.E. Rabinovich, F. Talavera, M.S. Kocher, L.K. Jung, and B.L. Myones, 2011. *Juvenile Idiopathic Arthritis* [online] [viewed 2 February 2018]. Available from: https://emedicine.medscape.com/article/1007276-overview#aw2aab6b2b4aa

(Struss, 2008) Struss, P., 2008. Model-based Problem Solving. In: F. von Harmelen, V. Lifschitz, and B. Porter, eds. *Handbook of knowledge representation.* Amsterdam: Elsevier, pp. 395-465.

(Struss, 2004) Struss, P., 2004. Models of Behavior Deviations in Model-based Systems. In: R.L.d. Mántaras, ed. *Proceedings / ECAI 2004, 16th European Conference on Artificial Intelligence. August 22 - 27, 2004, Valencia, Spain.* Amsterdam: IOS Press, pp. 883-887.