Semester project
Biorobotics Laboratory, EPFL

Neuromuscular Model for Gait Pathology

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I. Introduction

Locomotion impairments decrease drastically the quality of life. It is a daily used function strongly associated with social interaction. There is wide range of gait pathologies with some of them more handicapping than others. The aim of the project is to modelize some of them with a biologically inspired model. Modelizing gait allows a better comprehension of the walking mechanism and its affections. We want to assess the biological relevance of the neuromuscular model developed in H. Geyer, et al., (2010) [1] by testing its flexibility. Another purpose of modelizing gait pathology is to use directly the model to design controller of orthosis or prosthesis as *M. F. Eilenber, H. Geyer, et al. (2010)* [2] have done for a active robotic ankle.

We will focus on two locomotion pathology types. First, the muscular dystrophy and in particular Duchenne Disease as it is a spread one and it has well known disease mechanisms. The second type of pathology is the leg hemiparesis due to a stroke. This affects the neural network level so it is a good way to test the neuromuscular model which is a reflexe-based model. These two disease type allow to have stage were the walking ability is still there but impaired. Indeed we need to have walking subject to modelized their gait.

II. Theory

The legged locomotion is cyclic motion which seems simple because walking is something very natural for humans. It is the result of an interaction between the environment and the body. The body is composed of a mechanical structure made of bones linked with ligaments. This structure is activated by muscles attached by tendons. The muscles are controlled accordingly to the environment. The Mechanical part is quite well understood and could be easily modelize, however the control mechanism is still under discussion. Some evidences shows that the cortex is not directly involved in the creation of the cyclic motion pattern. *Grillner, S. (1973)* [3] paper made an experiment with a decebrate cat on the treadmill with a weight support set up. The cat was able to walk using only the sensory feedback and the spinal circuitry.

This tends to think of a low level network controlling the locomotion. This network should use the information coming from the environment (input) to generate propper muscle activation (output). This could be implemented by simple reflex loops (i.e. time delayed linear transformation of the input). Such network has been sucessfully used by *H. Geyer, et al., (2010)* [1] to generate stable walking with lower limb model of human walking. The results were suprisingly close to human walking in terms of joint angles, joint torques and muscle activation patterns highlighting the importance reflexes could play in human locomotion. First they implement the mechanical aspect of the body where they chose the Hill model for the muscles showed in the figure 2. In this model, the muscles are composed of two parallel springs, one shorter than the other, and one in serie of an activator. The spring in serie models the tendons and the parallel springs model the passive element of the muscle. At last the activator represent the active element in a contraction. The force generated by the active component is function of the activation level, actual length and velocity of the muscles. In the two-dimensional version of the model, one leg is actuated by seven muscles as shown in the figure 1. They are stimulated by a neural network encoding the reflexes. In order to obtain a meaningful pattern accordingly to the phase of the gait three, sets of reflexes are used for the following decomposition: the swing, the stance and the double stance phase. The leg is considered in the swing phase if it is not touching the ground. There is two possibility otherwise: the leg is the only touching the ground so it is in the stance phase or both legs are touching the ground and they are in double stance phase. The different reflexe loops are shown in the figure 3 and the resulting stimulation for each muscle in the different phases are describe in *H. Geyer, et al. , (2010)* [1]. During the swing phase the leg should go forward without touching the ground. The

reflexes of the double stance phase aim at changing the body weight of leg. Finally the stance phase is needed to propel the body forward. There is different types of feedback as force or length feedback, positive or negative. There are also stability feedback and knee angle overextension prevention feedback. Once the set of reflexes are determine for each muscles in each phase there is all the parameters characterising these reflexes as the gain or the offset to optimize.

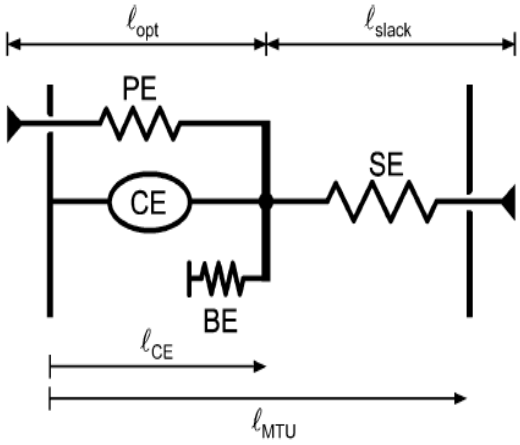

Figure 2 : Hill-type muscle model.

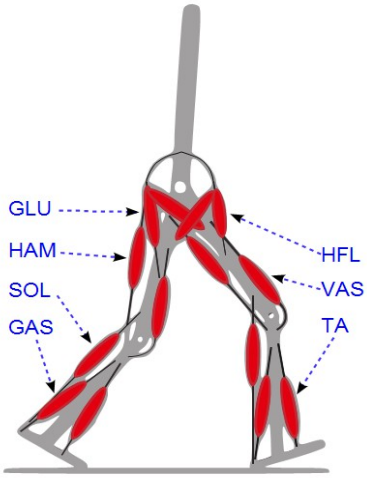

Figure 1 : Muscles distribution.

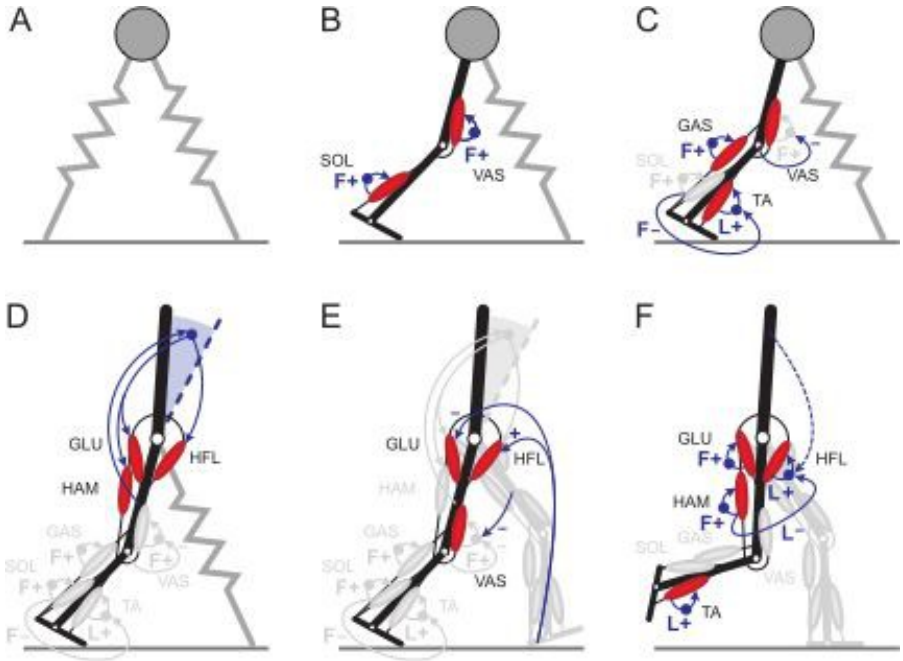

Figure 3 : Reflexes implementation.

III. Biological parametrisation

The neuromuscular model offers a certain flexibility due to its large number of parameters. Furthermore, its strong biological inspiration makes the modeling of different diseases easier. Indeed, we can play with the parameters associated with the different model structures (such as muscle parameters, tendon parameters, joint parameters and reflex gains) to produce different kind of gait. For example, it is possible to reduce the force of the muscles, change theirs springs

characteristics or their geometry. In an other hand, we can tune the reflexes parameters which are part of the neural network representation. At last, there is the possibility of re-optimizing or not the models with the new settings, depending on the kind of disease. Indeed if it is slowly degenerating diseases, the neural network has the time to adapt and optimize its reflexe feedback which is not the case of an event as stroke, at least in the acute phase.

1. Duchenne Muscular Dystrophy

Theory

The duchenne muscular dystrophy is a degenerative disease of the muscles. It is the most common dystrophy and it touches one boy over 3500 birth. It is linked to the X chromosome and it is a recessive mutation of the dystrophin gene. The weakness of the muscles start at the hip and children show difficulties to walk from the age of 2-3 year old. Without treatment the mean age they stop walking is 9 year old due to spreading of the disease to the other muscles [9] .

The weakest muscles in Duchenne diseases are the proximal muscles of the leg, the gluteus maximus muscle in particular (*D'Angelo, M. G. (2009)* [4]). This could be implemented in the model by reducing the maximal force the muscles of the hip and some of the knee can produce. This concerns four muscles among the seven implemented: the gluteus maximus, the hip flexor, the biarticular hamstring muscle group and the vasti muscle group. Additionally, there is a tightness of the hip flexor which could be represented as an increased spring constant of the parallel passive element in the muscle tendon model. As the force of the parallel spring is given by the following equations, the way to increase its constant is to decrease the *w*. Indeed we see that it is inversely proportional to the resulting force of the spring.

$$F_{muscle} = F_{CE} + F_{PE} - F_{BE}$$

$$F_{PE}(l_{CE}) = \begin{cases} F_{max} * \left(\frac{l_{CE} - l_{opt}}{l_{opt}^2 * w}\right)^2, l_{CE} > l_{opt} \\ 0, otherwise \end{cases}$$

With l_{CE} , the length of the active part *CE* as in the figure 2. l_{opt} , the length of the parallel spring *PE* at rest. And *Fmax*, the maximal force of the muscle. The muscle force is composed of the different parts of the Hill-type model. For the other force component refers to *F. Dzeladini, master thesis* [5].

Method

First, we need to identify the concerned parameters in order to obtain a Duchenne muscular dystrophy gait (DMD). As the muscle weakness starts at the hip, the maximal force of muscles acting on the hip should be reduced (GLU, HAM, HFL in the figure 1) . Even more the one for the gluteus maximus (GLU) because this is the most affected muscle. In an other hand, the ω parameter should be reduced to obtain a greater stiffness of the parallel spring component in the hip flexor muscle (HFL). This parameter was set to 0.56 in the healthy gait model as it is shown in the annexes. So we set it to 0.3 in DMD model. In the table 1, we see the different settings changed compared to the healthy model before re-optimizing the reflexes parameter set. Indeed the DMD is progressive, so the neural network has the time to adapt. Due to the neural network plasticity the reflexes evolve accordingly to the disease. The parallel process in the model is the optimization.

F_max	Healthy	Set 1	Set 2	Set 3
GLU	1500	1050 *(70%)*	900 *(60%)*	750 *(50%)*
HFL	2000	1600 *(80%)*	1300 *(65%)*	1000 *(50%)*
HAM	3000	2400 *(80%)*	1950 *(65%)*	1500 *(50%)*
VAS	6000	4800 *(80%)*	3900 *(65%)*	3000 *(50%)*

Table 1: Duchenne parameter sets.

Here in the table, the different sets representing the evolution of the disease as the maximal forces decreases are shown. For each modified values there is the corresponding percentage of the healthy value.

Results

The reflexes parameters were optimized with the three different sets, given in the table 1, and for the original values, as we can see in the annexes. For the set1 and the set2, the model was able to walk, so we present the angles in the figure 5 and 6, respectively. These data were obtained by extracting the gait cycles using footstrike event. Then the time line was normalized to 100. At last the graphs were obtained using the mean and the standard error of different cycles. The biological data of real DMD patient are shown in the figure 4 in order to have a comparaison. However the model was falling for the third set so there is not meaningful data to show for this set.

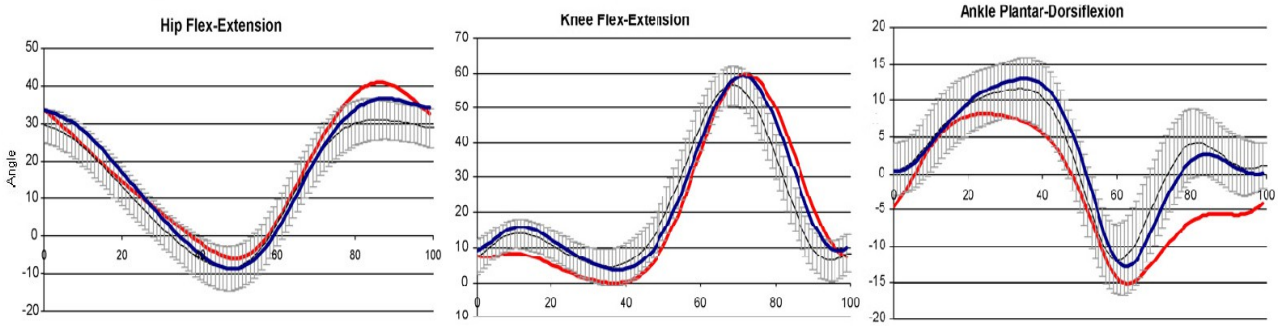

Figure 4 : Biological mean values of joint angles [°] in function of the gait cycle [%] measured in the paper D'Angelo, M. G. (2009) [4] with DMD subject (red), control group (blue). The gray scale come from literature.

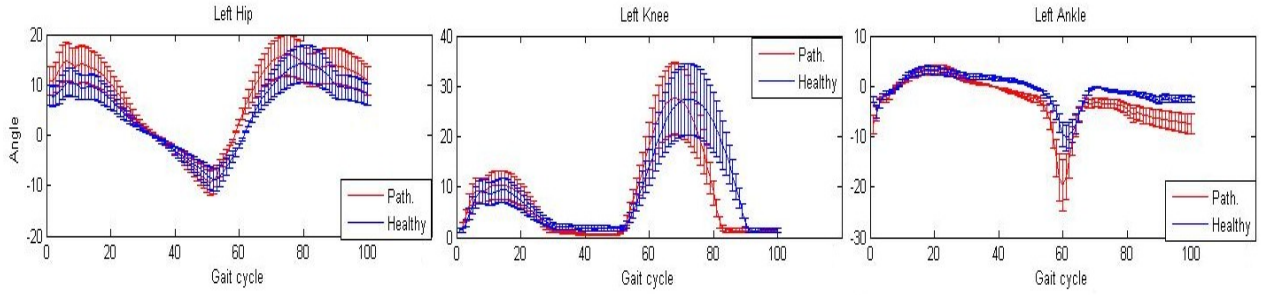

*Figure 5 : Mean values of joint angles [°] of the model in function of the gait cycle [%]. **Set1** settings in red and 'healthy settings in blue.*

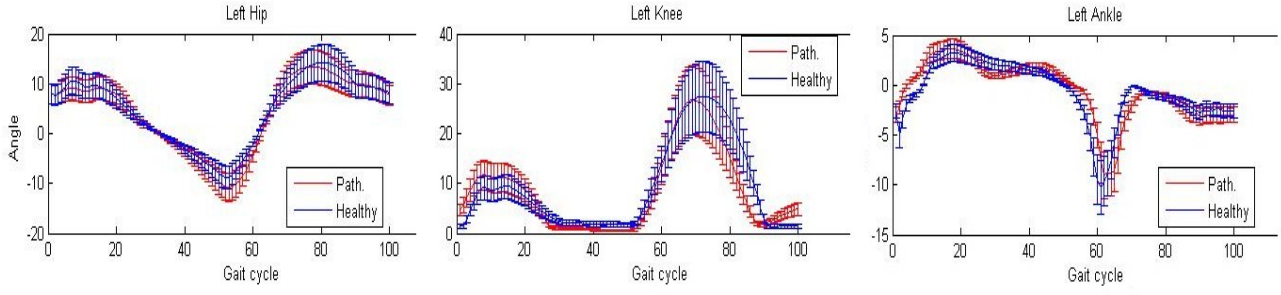

*Figure 6 : Mean values of joint angles [°] of the model in function of the gait cycle [%]. **Set2** settings in red and 'healthy settings in blue.*

We retrieve also the torques of the model and process them in the same way. The figure 8 and 9 shows the results of the knee and the ankle for the set1 and set2, respectively. We have the comparaison for this two joints in the literature but not for the hip.

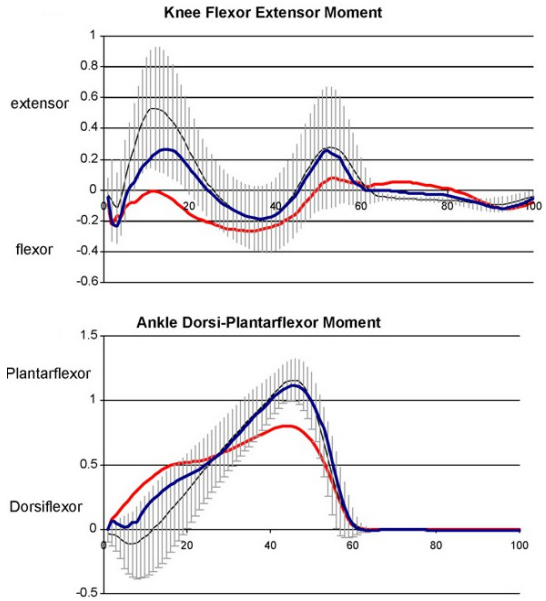

*Figure 7 : Biological mean values of normalized force moments [N*m/Kg] in function of the gait cycle [%] measured in the paper D'Angelo, M. G. (2009)[4] with DMD subject (red), control group (blue). The gray scale come from literature.*

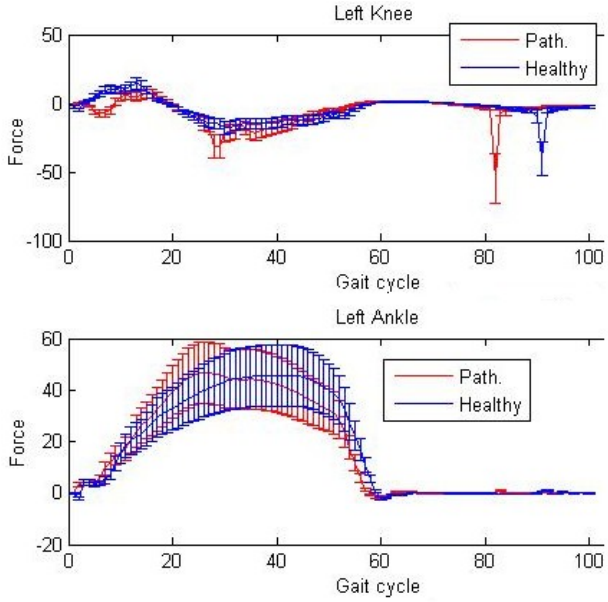

*Figure 8 : Mean values of force moments [N*m] of the model in function of the gait cycle [%]. **Set1** settings in red and 'healthy settings in blue.*

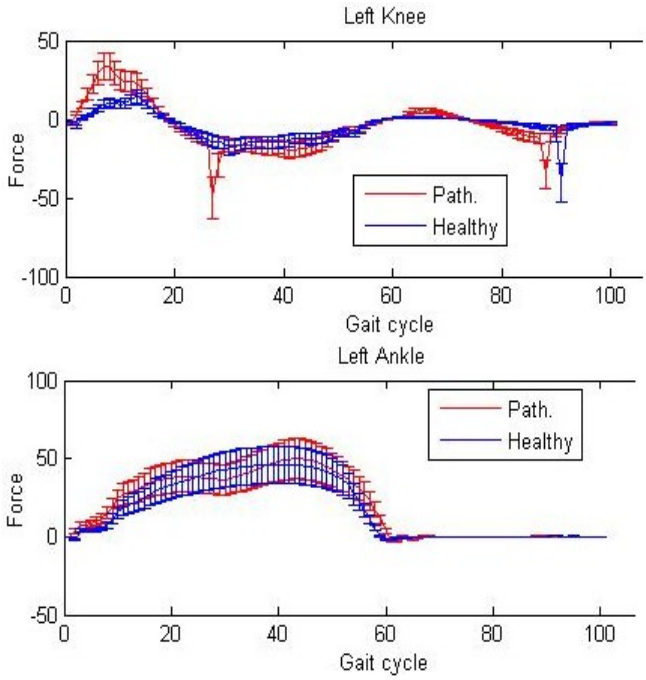

*Figure 9 : Mean values of force moments [N*m] of the model in function of the gait cycle [%]. **Set2** settings in red and 'healthy settings in blue.*

In the Figure 7, the moments are normalized by the masse and the force joints are not normalized moments, but they are proportional to each other so it is possible to compare the type of curves without looking at the scale. At last, we showed only the left side results because they are supposed to be symmetric.

Discussion

In the simulation we see that the first two sets of parameters are walking nicely whereas the third one fall quite immediately. It means that the impairments start being too excessive. For the walking sets, we can assess the differences only quantitatively because we don't have access to the raw data of the literature to quantify the correlation. The pathological angle graphs of the model (Figure5) of the Set1 seem to be closer to the biological data than those of the set2 (Figure6). In particular, we see the same tendency to have bigger hip flexion and bigger ankle dorsiflexion in the second part of the gait cycle. For the joint moments we have also the same type of impairment for the model ankle compared to the literature. The curve is moved toward the beginning of the cycle and smaller at the end. It seems that the ankle pattern is the most characteristic of DMD among the three joints. As in the set2 the model is walking in a quite good manner and the results are close the healthy model, we can suppose that this not only the decrease of force but the relative distribution among the different muscles that is determinant to have a Duchenne muscular dystrophy pattern. Indeed, in the set1 the differences between the GLU muscle (70% of healthy force) and the other impaired muscles (80%) is bigger than in the set2 where there is only 5% and not 10%, as we see in the table 1.

In an other hand we observe a difference of range of angle values for the three joints between the model and the record data. This could be explained by the fact that the model is representing an adult of 1m80 (*see in the annexes*). The group of patient use to obtain the kinematic data have a mean age around 7 year old. It is due to the progression of the disease as they are often not able to walk from 9 year old. So the mass distribution is changed and the geometry of the model is much bigger than the reality. Since Duchenne disease concerns mainly children and toddlers, an improvement of the duchenne disease model could be done by downscaling the adult model to children size.

For each of the settings we have to optimize the model which is taking a lot of time. All of the impairment values were hand tuned, so it would be a good thing to implement a systematic search in order to gain time. This would also permit to have a better overview of the possibility of the model.

2. Stroke gait pathology

Theory

Cerebrovascular accident affect a large range of the population. This often implies permanent motor impairment including locomotion functions. The loss of tissues in the motor cortex area is the reasons of these impairments. It could affect in various way the synergies and the reflexe patterns of the walk. This is why it is hard to modelize, however it is possible to extract common features between the affected patients. In opposition to the Duchenne muscular dystrophy this is mainly asymmetric impairment. The most common observation is the decrease of strength of the affected side [6][7][8]. As the Duchenne muscular dystrophy, this could be implement by reducing the maximal force of the muscles. One issue of the stroke impairment is the spasticity and an increased muscle tonus, in particular of the ankle and of the quadriceps. Moreover, it induces abnormal stretch reflexes. The idea is to model the patient gait soon after their stroke and before the rehabilitation process, so we should implement this impairment in the model without re-optimizing.

Method

At first we should decrease the maximal muscle forces but unlike DMD modelization, this

can't be a big change. Indeed in this case we are not re-optimizing the model with the changes so just a small force change affects drastically the behavior. All the following settings are hand tune in order to keep a model able to walk. Accordingly to the literature [6][7][8], there is not one muscle which have a particular loss of forces, so we can decrease the seven muscle in the same way. If we reduce of 90% compared to the healthy *Fmax* all muscles of the right leg the model falls. However with a decrease to 5% only the model achieve to keep a stability. So the *Fmax* parameters will be set as following.

F_max	Healthy	Affected
GLU	1500	1425 *(95%)*
HFL	2000	1900 *(95%)*
HAM	3000	2850 *(95%)*
VAS	6000	5700 *(95%)*
GAS	1500	1425 *(95%)*
SOL	4000	3800 *(95%)*
TA	800	760 *(95%)*

Table 2: Stroke F_max parameters for each muscles.

Once we reduced the *Fmax*, still keeping the stability, we can modify the reflexe components of the same leg. In the buckling-knee pattern which is one kind of typical pattern after a stroke [6][7][8] we can extract two excessive muscle contraction or spasticity. The first one is the vasti muscle group (VAS) responsible for the knee extension. The second, the gastrocnemius (GAS), is responsible for the ankle dorsiflexion. The parallel we can make in the model is to increase the basal activity of these muscles.The initial values of 0.010 for the VAS and 0.0176 for the GAS are increased to 0.012 and 0.020, respectively. We can also play with the gain of the feedback loop reflexes in order to increase stiffness of the leg. The three positive force feedback loop of SOL, GAS and VAS are describe in *H. Geyer, et al. , (2010)* [1] as encoding for the compliance. So we can tune the assiociated gain to modify the leg behavior. We can expect a higher stiffness for the set1 as it is decrease the feedback which allows compliance.

G_m	Healthy	Set 1	Set 2
SOL	1.38	*1.3*	*1.41*
GAS	0.94	*0.9*	*0.96*
VAS	1.7	*1.65*	*1.73*

Table 3: Stroke parameter sets.

Results

The figures 12 and 14 show the angles for the set1 and 2 respectively with the right leg as the affected one. We can compare them with the angles of *Olney, S. J., & Richards, C. (1996)* [8] in the figure 10. The figures 13 and 15 show the moments of the model compared with the figure 11 from the same paper.

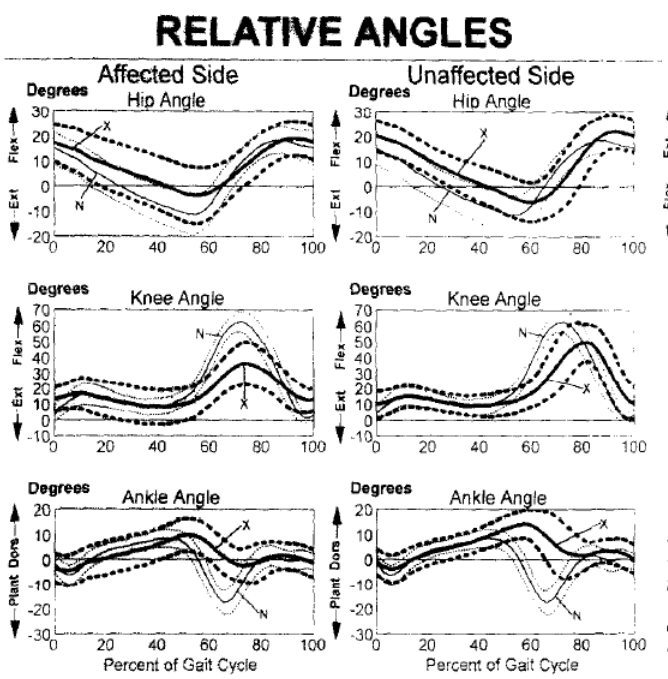

Figure 10 : Biological mean values of joint angles [°] in function of the gait cycle [%] measured in the paper Olney, S. J., & Richards, C. (1996) [8] *with hemiparesis subject (X), control group (N).*

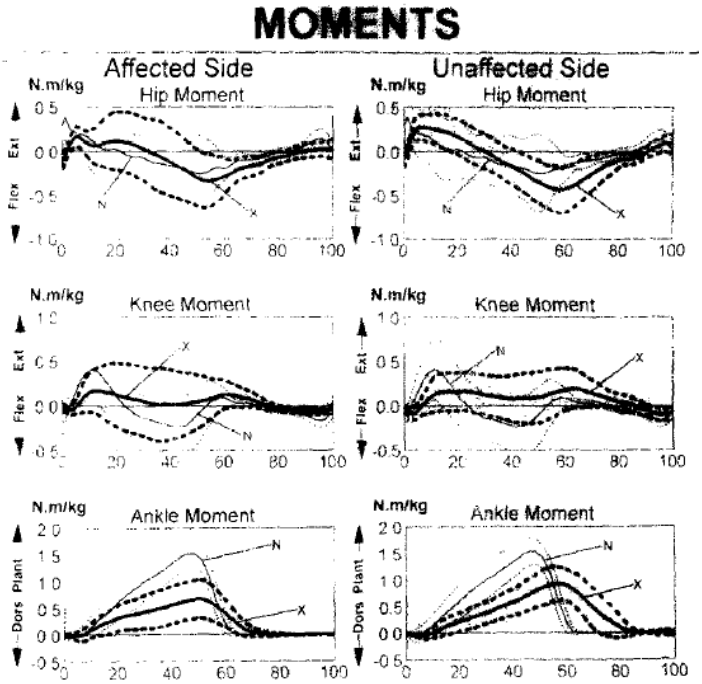

*Figure 11 : Biological mean values of normalized moments [N*m/Kg] in function of the gait cycle [%] measured in the paper Olney, S. J., & Richards, C. (1996)* [8] *with hemiparesis subject (X), control group (N).*

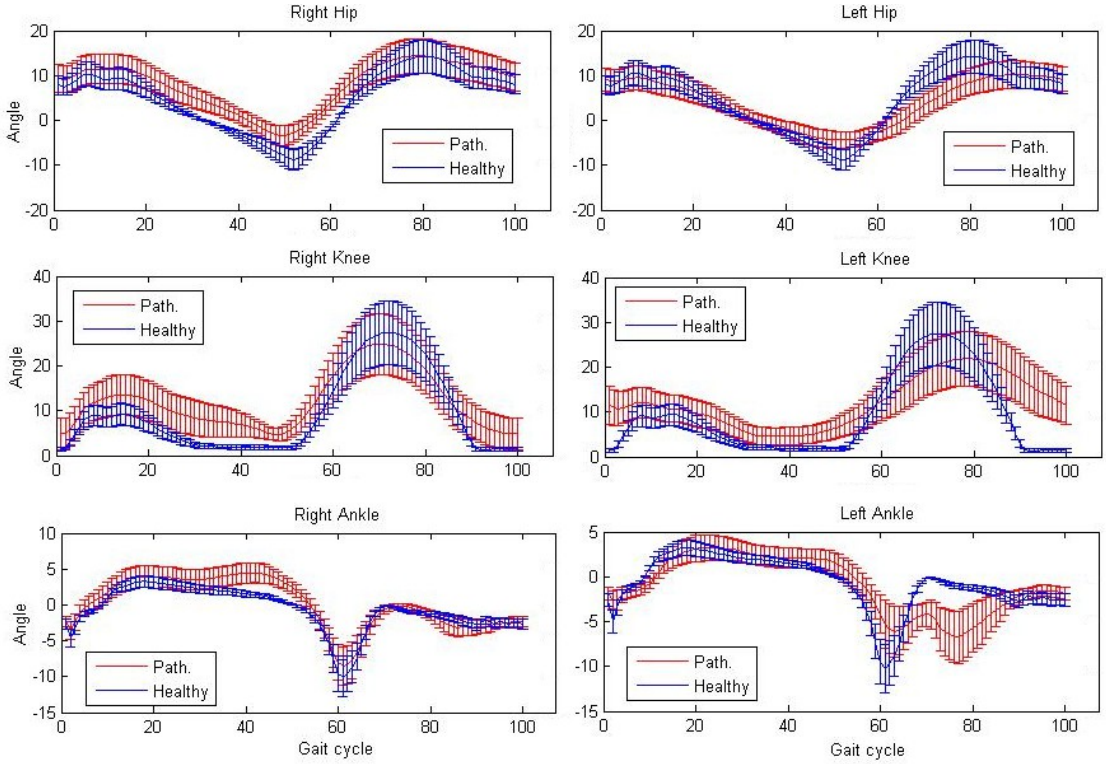

*Figure 12 : Mean values of joint angles [°] of the model in function of the gait cycle [%]. **Set1** settings in red and 'healthy settings in blue.*

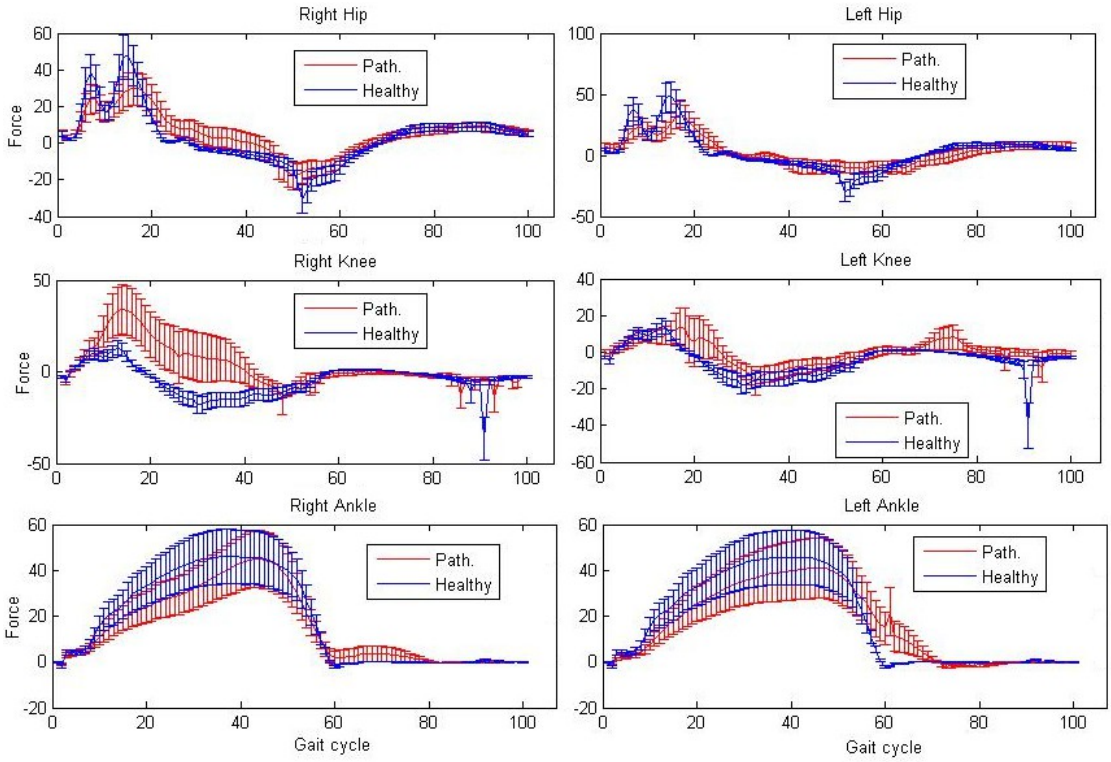

*Figure 13 : Mean values of torques [N*m] of the model in function of the gait cycle [%]. **Set1** settings in red and 'healthy settings in blue.*

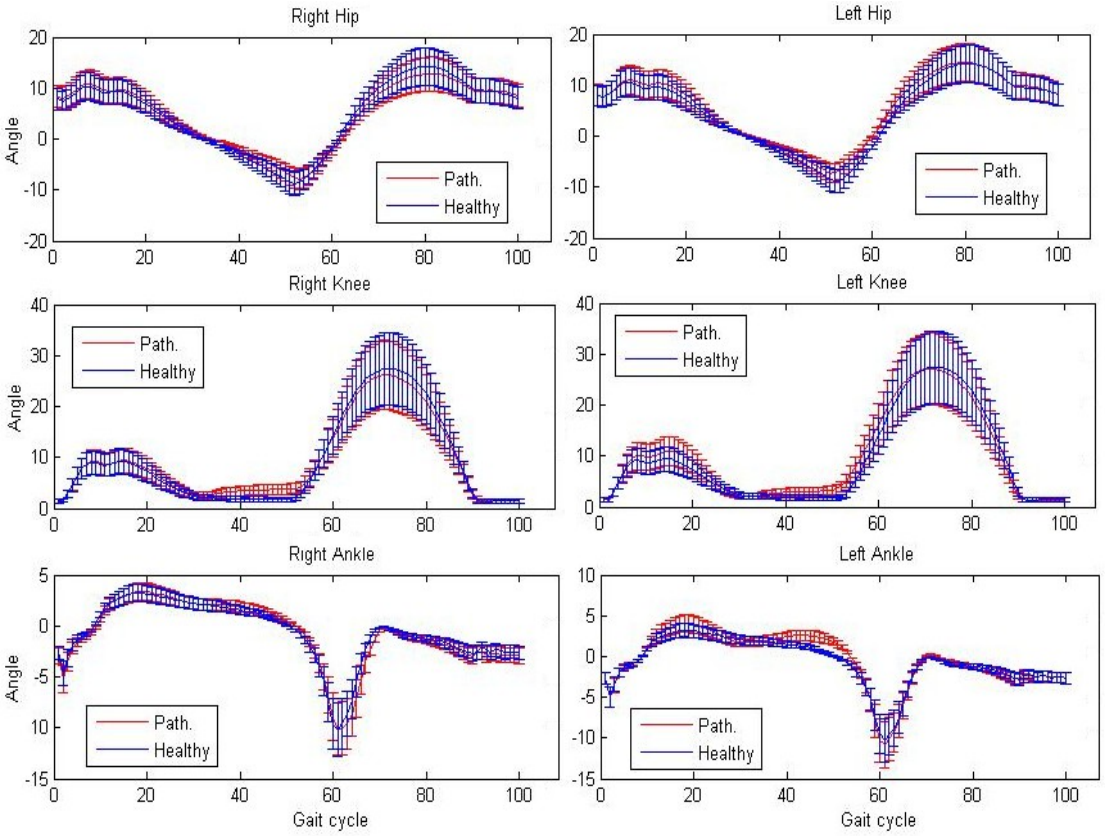

*Figure 14 : Mean values of joint angles [°] of the model in function of the gait cycle [%]. **Set2** settings in red and 'healthy settings in blue.*

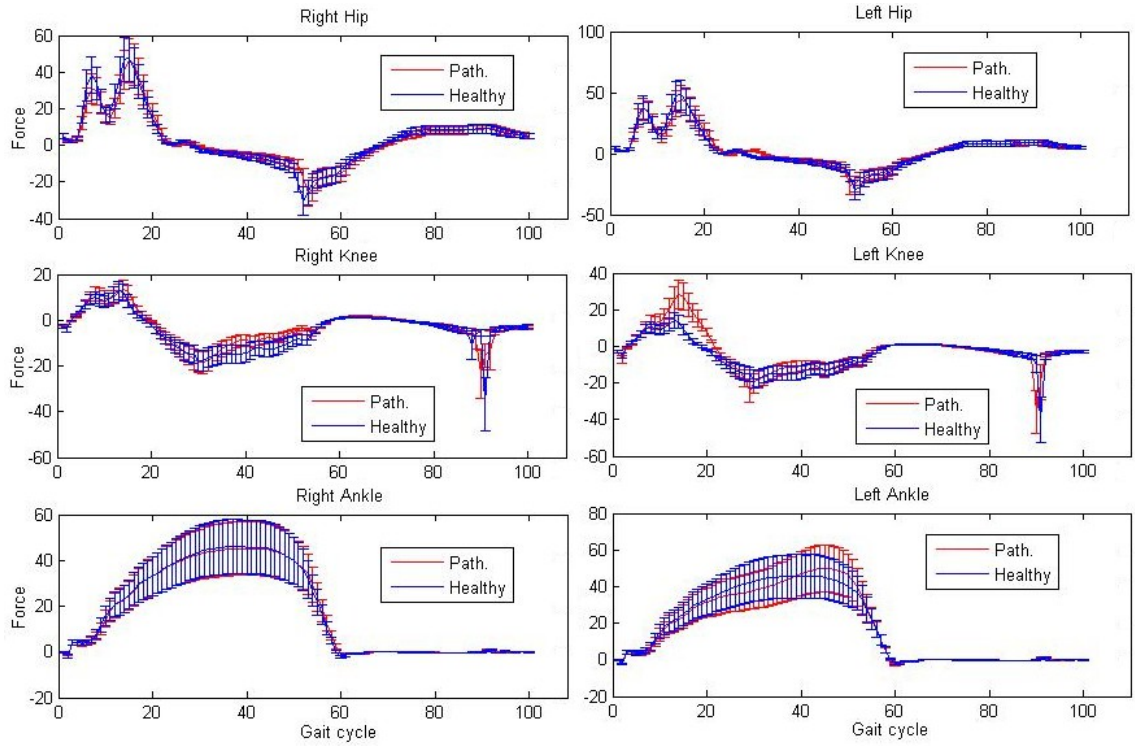

*Figure 15 : Mean values of torques [N*m] of the model in function of the gait cycle [%]. **Set2** settings in red and 'healthy settings in blue.*

Discussion

In the results we can see qualitatively that the results of the set1 are closer to the literature pattern than those of the set2. This confirms that the three positive force feedback loop of the soleus, the gastrocnemius muscle and the vasti muscle group allow the compliance of the leg. Indeed when we decrease the gain of these reflexes the behavior of the leg seems more like the hemiparesis pattern after a stroke which is known to have a higher stiffness.

For the model angles of the first set (Figure12), we see on the affected side hip the same tendency to have a higher flexion than the healthy curve and on the unaffected the swing flexion is retarded (second part of the gait cycle). We see also as in the literature that the knee flexion of the affected side during the swing is slightly decreased and it is also shifted in time for unaffected side. Finally there is the same buckling pattern of the ankle on the unaffected side. In the literature it seems as this pattern is more marked on this side. The moments in this set1 have also some similarity with the recorded biological data. For example the affected knee pattern as a much higher variability and diverge from the healthy curve. The ankles moments for both side are, as the biological data, decreased and shifted in time.

For the second settings the stroke model angles are nearly the same than the healthy model (Figure14). The force moments are as well very close to the healthy model moments (Figure15).

However, all these assessment are just qualitative and would require a deeper study to correlate the curve with raw data comparison. Moreover this type of walk impairment is really dependant of the subject and has a high intersubject variability. It can even evolve in time with rehabilitation. Here we just tried to model the mainly shared impairment components. According to all these constraints we obtain nevertheless a nice pattern of this pathology. This consolidates the biological relevance of the model. In order to design a device for rehabilitation we should be able to adapt and personalized it to each patient.

IV. Biological Data Correlation

Theory

In the case where we have exact biological data, measured for example by motion capture of a subject, another approach to obtain a pathological gait model could be to force the angles of the joints to correspond. This would be useful for personalizing the model for a future device application model. This could be done by optimizing some parameters accordingly to a fitness function taking into account the correlation between the model data and a set of real biological recorded angles. This way we assume that we will obtain a model close to the reality in term of joint angles. Nevertheless, the biological relevance of the model construction is less emphasized with this type of optimization as the parameters are deduce using the real data.

Method

We use biological data of an healthy subject and data of subject with a muscular dystrophy for the optimization[1]. The muscular dystrophy affect the force of the muscles, so we should optimize the maximal force parameters as it is corresponding to muscle strength or weakness. The other parameters are set as the healthy gait. In order to keep a symmetric gait we put one parameter for the both side muscles. As the previous optimization, the first stage consists of maximizing the distance travelled before without falling. Then we try to maximize the angle curves correlation with the muscular dystrophy biological data. This correlation fitness coefficient is given by the following equation:

$$Correlation_{coeff} = \frac{1}{3} * \left(Corr_{hip} + Corr_{knee} + Corr_{ankle} \right)$$

$$Corr_{joint} = \frac{Covar\left(M_{joint}, B_{joint} \right)}{\sqrt{Var\left(M_{joint} \right) * Var\left(B_{joint} \right)}}$$

Where M_{joint} is the model angle for the indicated joint and B_{joint} the same angle joint of the biological data. The correlation was calculated only on one side, as the forces of each side are optimize together, the gait is supposed to be symmetric.

Results

The figure 16 show the healthy and the muscular dystrophy biological joint angles. The figure 17 is the joint angles of the model for a final correlation score of 0.40. The table 4 is showing the obtain maximal force parameters.

F_max	Healthy	Affected
GLU	1500	852 *(57%)*
HFL	2000	1560 *(78%)*
HAM	3000	2635 *(88%)*
VAS	6000	4277 *(71%)*
GAS	1500	1167 *(78%)*
SOL	4000	2995 *(75%)*
TA	800	779 *(97%)*

Table 4: F_max parameters find after optimization for each muscles.

1 Data from Marco Iosa, Foundation Santa Lucia, Laboratory of Experimental Neurorehabilitation, Italy.

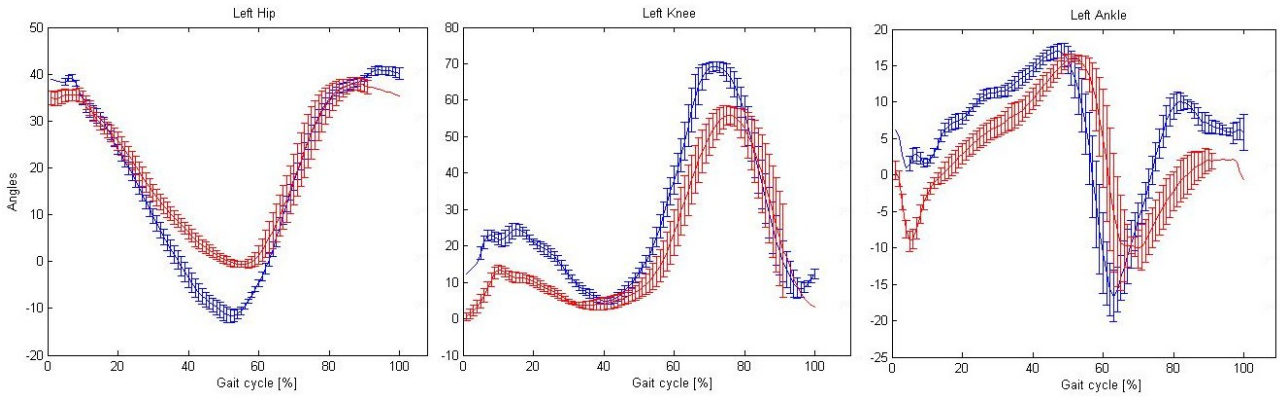

Figure 16 : Biological mean and standard error values of joint angles [°] in function of the gait cycle [%] measured by Marco Iosa with dystrophic subject (red), control group (blue).

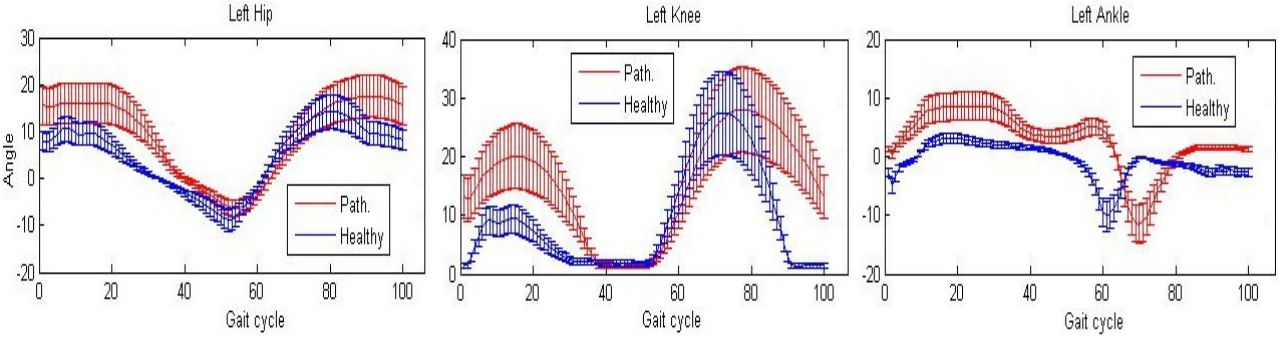

Figure 17 : Mean and standard error values of joint angles [°] of the model in function of the gait cycle [%].

Discussion

The results are not as good as expected because they don't seem exactly similar. Indeed the correlation is quite low as it is 0.4 over 1. We could hope to obtain better results with longer optimisation process. Indeed, it takes a long time for the model to find good parameters allowing to walk enough time to be able to measure the correlation. One way to accelerate the process would be to reduce the authorized range for the optimizing parameters in order to increase the constraints.

However we can see in the maximal force table4 than the optimization permits to lower the forces much more than a equally distributed decrease. As at 90% of the healthy force the model is already falling.

V. Future work

We could to design an active orthosis based on the neuromuscular model of *H. Geyer, et al. , (2010*) [1]. This model have already been use for an active ankle prosthesis by *M. F. Eilenber, H. Geyer, et al. (2010)* [2] where they implement a neuromuscular model to control the robotic device. This model could also used to make an external device assisting the patient to walk and not replace completely the joint. The main principle would be to take into account the local information of the leg to implement the model and to compensate the missing torque caused by a neuromuscular pathology to achieve a healthy gate. This kind of device would be very adaptive and could be use in different type of pathologie as the Duchenne muscular dystrophy where it would serve as a compensation device or even as a rehabilitation tool. Indeed it would be wearable device that a person in acute rehabilitation after a stroke or an accident could use to relearn a natural walking. The advantage is that the gain could be tune to force the patient to increase it is contribution to the walk and be able to adapt to the recovery evolution. In order to implement the external orthosis we

need first to model the pathological gait. In our case we would split the model in two where one would produce the impaired walk and the other will help the first one to recover a normal gait. The following equation shows an example of the effective torques produce with this two parallel neuromuscular models.

$$T_{eff} = T_{path} + a * T_{device}\left(T_{path}(t - t_0), \vec{\theta}\right)$$

where T_{eff} is the effective torque, T_{path} the torque produced by the pathological model and T_{device} the compensatory torque of the device model which depends of the pathological torque and the angles θ provided with sensors. In order to make it realistic we should add a delay t_o to measured pathological torque. Finally, the constant a allows to tune the gain produce by the device model.

VI. Conclusion

The different parts of this project, even if the results were not always as good as expected, show that the neuromuscular model used was flexible. Indeed, we obtain different gait pattern only by tuning its parameters.

A better biological comprehension of the mechanisms of the different modelized pathologies could help to target in the right way the parameters of the model to change. Using the three-dimensional version of the model could also improve the results. We the 2D version we are missing an import aspect of pathological gaits, because the first sign of a gait disorder is an increased motion in the transversal plane.

For the Duchenne muscular dystrophy modelling, we find joint angles and torques with some similarity compared to the literature. It seems as the distribution of the forces between the muscle play a key role in the locomotion pattern. However, the main improvement we can do for this disease modeling is to adapt the model to children as the patient aren't able to walk from 9 year old.

The result for the hemiparesis modelling after a stroke were surprisingly good if we take into account the high variability of existing pattern. Indeed only with few parameters change we retrieve similar modification of the joint angles as in the real recording. This consolidates the reflex-based theory.

The last part of the project will require deeper investigation as the results were not as expected but the variability we see in the angles pattern are encouraging.

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VIII. Annexes

Muscles parameters

	HF	GLU	VAS	HAM	GAS	SOL	TA
Maximal muscle force ($F_{max}[N]$)	2000	1500	6000	3000	1500	4000	800
Maximal speed of the muscle ($v_{max}[m/s]$)	12.0	12.0	12.0	12.0	12.0	6.0	12.0
Optimal length of the muscle ($l_{opt}[m]$)	0.11	0.11	0.08	0.10	0.05	0.04	0.06
Optimal length of the tendon ($l_{slack}[m]$)	0.10	0.13	0.23	0.31	0.40	0.26	0.24
Pennation factor ($\rho[]$)	0.5	0.5	0.7	0.7	0.7	0.5	0.7
Type I fibers percentage	0.5	0.5	0.44	0.54	0.81	0.7	0.5

Lower limbs segments parameters

Segment	Mass (kg)	Length (m)
Trunk	53.5	0.8
Thigh	8.5	0.5
Shin	3.5	0.5
Foot	1.25	0.16
Ankle	0	0.1
Total body	70	1.8

Joint	θ_{min}	θ_{max}
Hip	20°	230°
Knee	45°	175°
Ankle	70°	130°