OpenSim Assignement

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Question 7

The gluteus maximus muscle is represented by multiple muscle-tendon actuators (glut_max1, glut_max2, and glut_max3). Which other muscles in the model are divided into multiple compartments? Propose an explanation.

Other muscles with multiple compartments are Glut_med (1, 2, 3) in purple in the figure (fig1) and bifem (short and large) in green in the figure. Glut_max (1, 2, 3) are in red in the figure.

In OpenSim, muscle-tendon paths are represented by a series of points connected by line segments. Most muscles are modelled by a single-line muscle-tendon actuator from origin to insertion.

However, for more complex muscles we need multiple lines of action to model their structure, they are represented by several actuators. Since they are part of the same muscle, they originate close to each other and stay in the same action area.

For example, the gluteus medius and gluteus minimus muscles contribute to pelvic stability and lower extremity function. The gluteus medius is implicated in hip extension, lateral rotation and abduction, meaning that the muscle performs complex movements, and does not only contract along a line. To model it we need more than one muscle-tendon actuator.

In addition, muscles that are wrapped around structures and bones, constrained, or have particular shapes need more than the origin and the insertion point to model the path: the model provides several intermediate wrapping points.

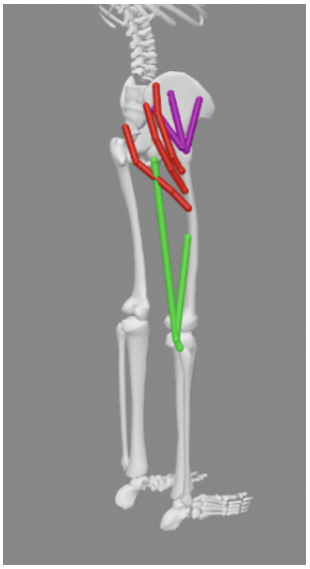


figure 1 : muscles divided into multiple compartments in model_gait

Question 9

Follow the same method to plot the left biceps femoris long (bifemlh_l) and biceps femoris short (bifemsh_l) moment arms about the left knee flexion with the left hip

at resting position and with the left hip flexed above 100° on the same plot. What do you observe? Provide the plot and propose an explanation.

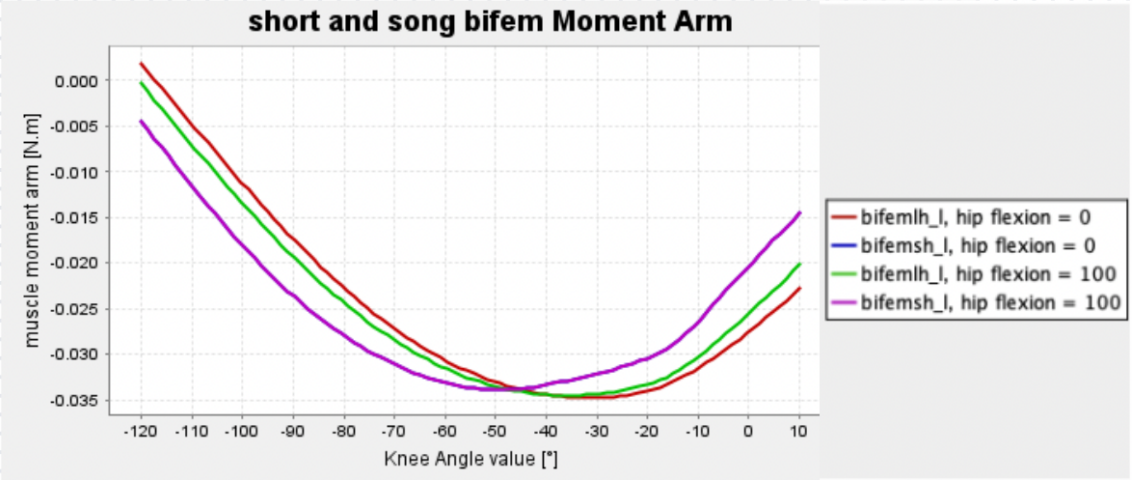


Figure 2: left long and short biceps femoris moment arms about the left knee flexion regarding the left hip flexion angle. Red and blue curve represent hip flexion = 0°; Green and pink curve have hip flexion = 100°

Firstly, it can be observed that the moment arm is negative. This can be explained by observing that, as for the ankle flexion arm, the muscle is behind the axis of rotation rigid-body and therefore behind the "zero" coordinate that starts at the axis of rotation.

Another way to understand it is to observe that the knee flexion is a negative angle in Opensim.(fig3) Thus knee extension must be actuated by a negative torque. Knowing that the Torque is defined as T = R x F and that the force F is positive, then the moment arm R must be negative.

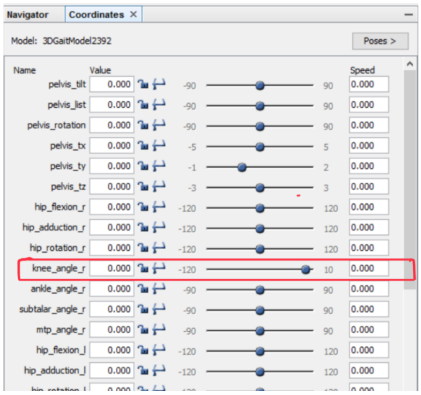


Figure 3: The knee flexion angle is negative.

Long and short biceps femoris have mainly the same bell-shape behaviour: starting at +10° the moment arms increases (in absolute value) until a certain max point where it then decreases back to 0 towards -120°. This is understandable, as the muscle is first stretched and is put backwards by the knee flexion until it bends inwards towards the rotation axis and has therefore a lower moment arm, as can be seen in Figure 4.

As the short biceps femoris is not attached to the hip, hip flexion has no impact on its behaviour, and we can see a clear overlap between the moment arms with 0° and 100° (blue and pink curves in Figure 2). However, hip flexion slightly shifts the MAX of the long biceps femoris to a lower angle. This is probably due to the fact that being fixed to the hip, its position is influenced by the hip rotation and is more stretched during hip flexion.

(a) -120°, maximum flexion, 0 moment arm (b) -20,5°, maximum moment arm

Figure 4: Illustration of the knee flexion moment arm

Question 16

Plot the hip flexion-extension, knee flexion-extension and ankle plantar flexion-dorsiflexion angles of the right leg on a single graph. Search curve references for healthy gait in the literature and compares them with the shape of your curves. Please discuss your results.

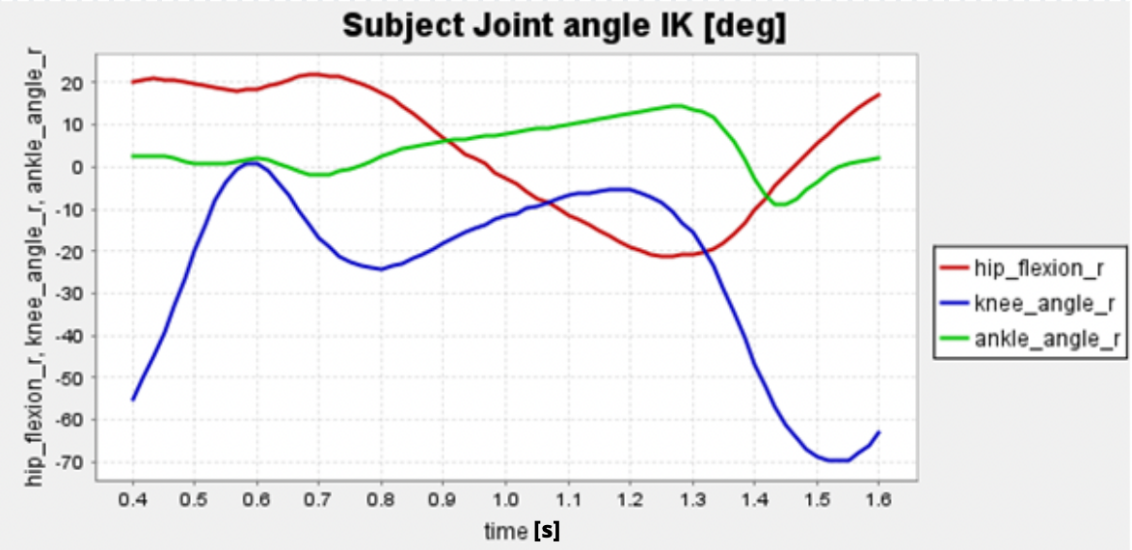


Figure 5: Hip, knee and ankle flexion-extension for scaled model

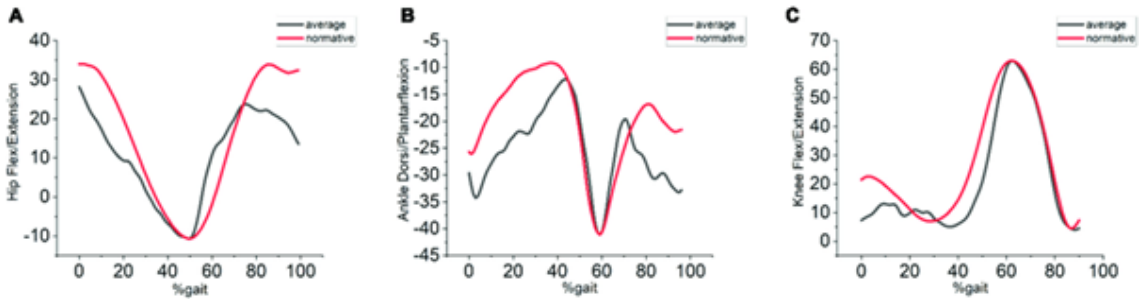


Figure 6: Hip, knee and ankle flexion-extension in healthy gate according to the literature [found on www.researchgate.net]

Generally, it must be noted that fig.5 and 6 have overall very similar shapes. Some minor differences were however noted, perhaps due to individual specificity and general imprecisions of the model:

- Hip: Hip flexion is slower with later and lower minima in Fig. 5, but retains the general shape
- Ankle: The ankle flexion shows an overall similar pattern, with a 40° difference on the overall gate. Nevertheless, the transition between dorsi and plantar flexion is slower in Fig. 5.
- Knee: Knee angle (inverse standard for angle, must make *-1) roughly follows a similar shape in two phases, one going up 20/25°, the other up to 65/70°. The most striking difference is observed in the transition to the first phase, which is much slower in Fig. 5.

Rmq: the same plot as in Fig. 5 has been made on OpenSim on the original 3DGaitModel before scaling, and it yielded very similar results.

Question 19

After executing ID, the muscle moments are exported in subject_walk_id.sto in the inverse_dynamics folder. Plot the hip flexion-extension, knee flexion-extension and ankle plantar flexion-dorsiflexion moments of the right leg on a single graph (similar method as above for IK results). Search curve references for healthy gait in the literature and compares them with the shape of your curves. Please discuss your results.

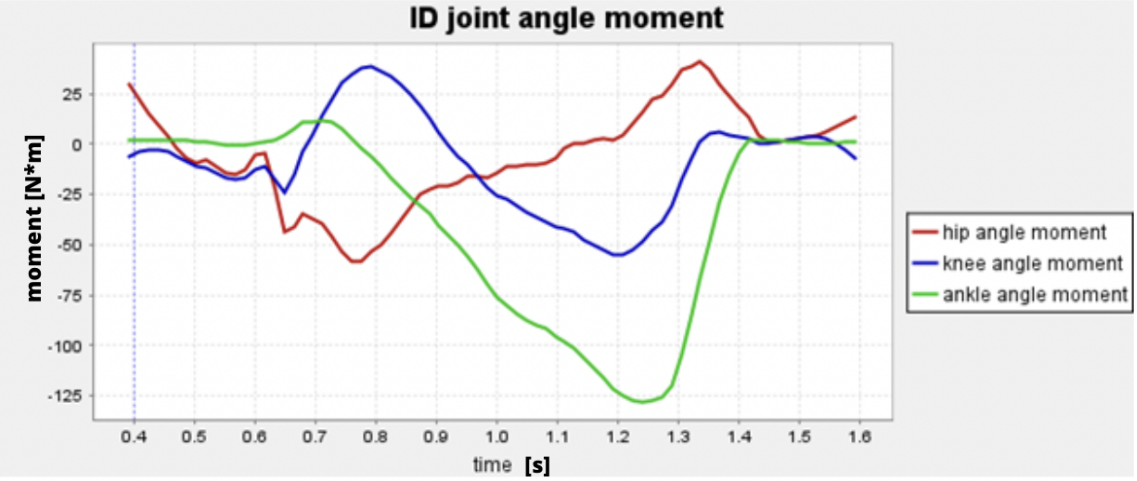


Figure 7: Hip, knee and ankle angle moment for scaled model [N*m]

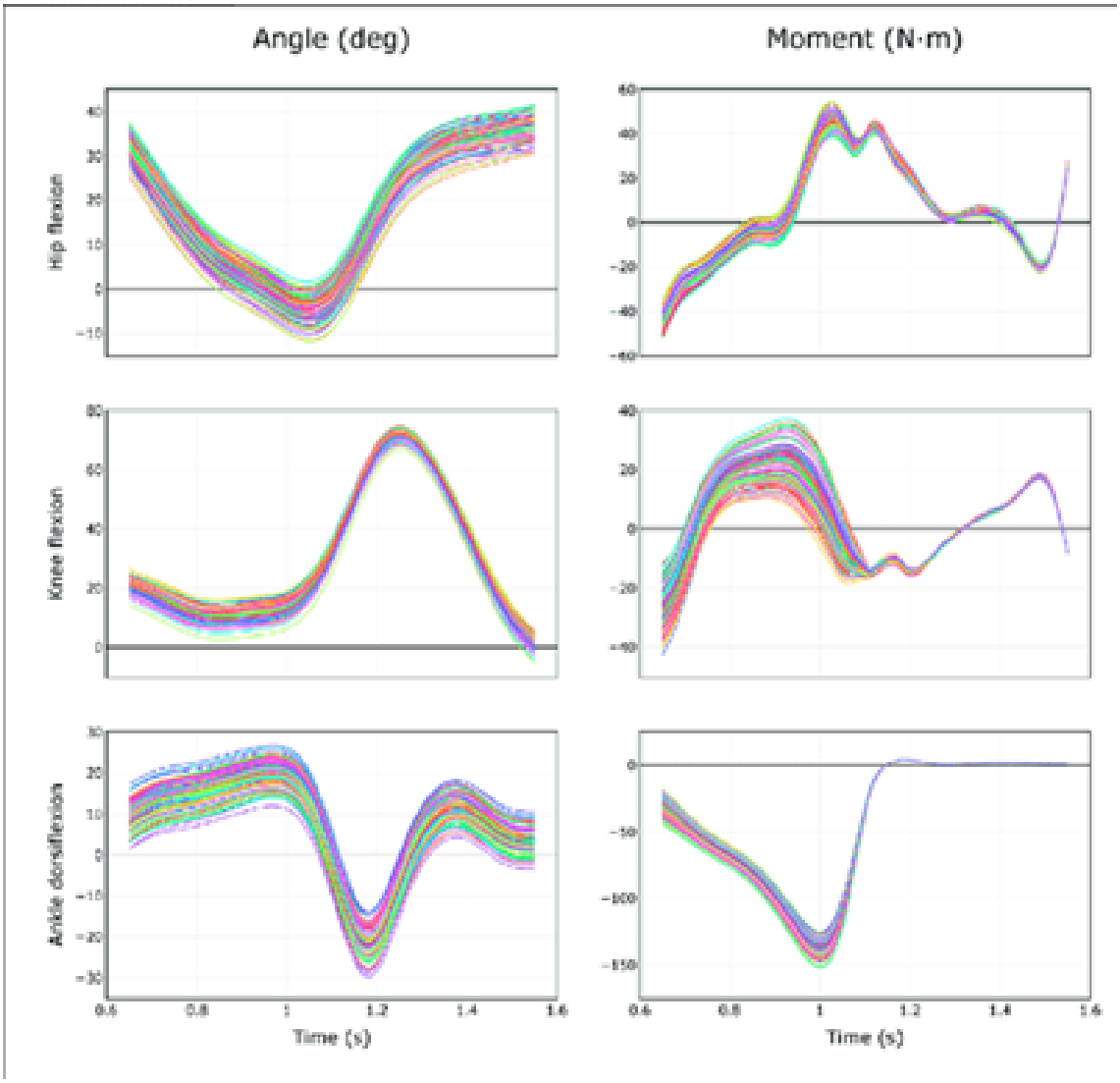


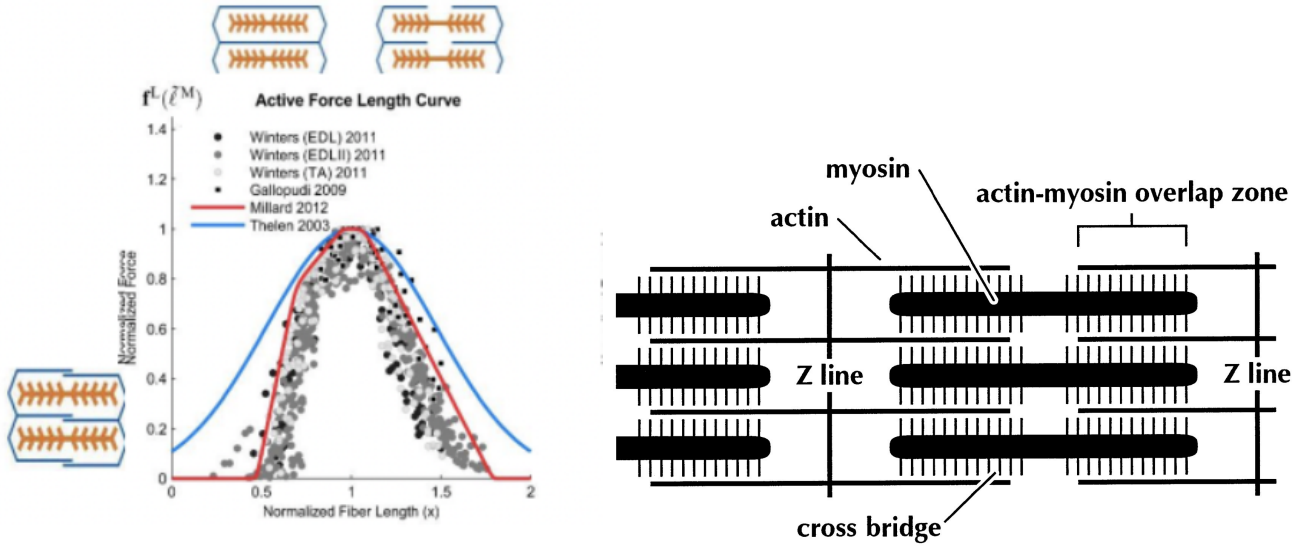
Figure 8: Hip, knee and ankle angle moment in healthy gate according to the literature [found on www.researchgate.net]

Here again, the general patterns are quite similar, with some small differences: The hip does not show a nice camel shape and seems to happen later relative to the rise in knee moment, compared to Fig. 8. There is also a lacking small humphback in the knee pattern, situated between the two major peaks.

Question 20

Why does the force-length curve of the contractile element of the muscle have a bell shape? Please elaborate.

In graph fig. 9, we can effectively see that the force-length curve of the contractile element (sarcomeres) of the muscle has a bell shape.



(a) active force-length curve, bell-shaped (from the course)

(b) scheme of a sarcomere [https://journals.physiology.org/doi/full/10.1152/jappl.1999.86.5.1445]

Figure 9: Influence on the length of the muscle

The sarcomeres are composed of myosin fibre and actin fibres. (see Fig9.b) Myosin fibres can slide between actin fibres, changing the length of the unit and therefore of the muscle. Z discs link parallel myosin/actin pairs and also limit the myosin movement in the direction of the fibre. Myosin and actin fibre overlap and are connected by myosin heads. Myosin heads are present on the extremity of the myosin fibres, in the overlapping zone, and not at the centre of the fibre. Each myosin head produces the same amount of force. Therefore, the force generated by the sarcomere is linearly related to the number of attached myosin heads.

When the normalized fibre length is 1, it means that the optimal fibre length is reached, therefore the generated active force is maximal.

When the muscle is stretched, the overlap between the myosin and the actin fibres decreases. There are fewer and fewer myosin heads that are recruited. At one point, there is no more overlap

and no force can be generated. It explains the right side of the curve.

When the muscle is compressed (never happens in healthy normal use) the number of attached myosin heads does not change because there are none of them at the centre of the fibre: the generated force can't increase. However, overlapping actin filaments will rub against each other, which will decrease the force. After a certain compression, the myosin filament will hit the Z disk, producing an opposite force. No force can be generated. This explains the left side of the curve.

Question 21

What is the meaning of the force-velocity shape of the contractile element? How does it relate to physiology?

To generate a force, the sarcomere needs to recruit myosin heads. To allow the conformal change responsible for myosin head attachment and cycling, time is needed.

Firstly, we notice a positive hyperbolic correlation between fibre velocity and force. (Fig 10. a)

- When the velocity is zero, the contraction is isometric.
- When the velocity decreases (v¡0), we have a concentric contraction. The muscle is shortened, so the possible active force decreases. The orientation of the myosin head changes. When the velocity increase (absolute value) fewer myosin heads can bind to the actin filaments, so the total force decrease.
- When the velocity increases, we have an eccentric contraction (v¿0). The muscle is stretched (lengthening), and the force increase because it needs to stretch passive structures and lengthen the muscle. The orientation of the myosin head change and are faster and easier to recruit. A plateau is reached after a certain velocity.

Fig 11 illustrates isometric, concentric and eccentric contraction.

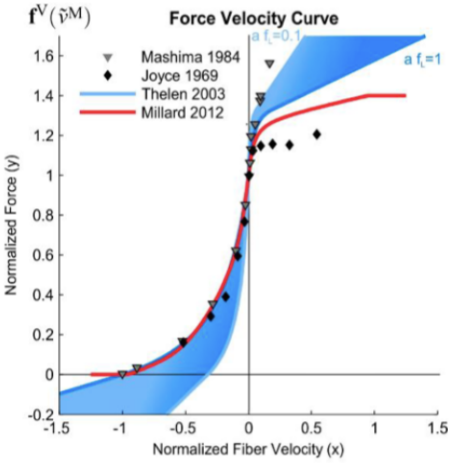


Figure 10. a: force-velocity curve

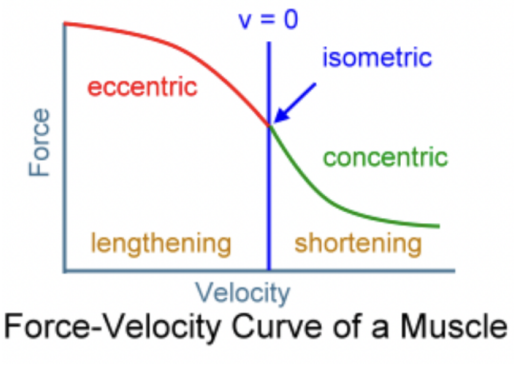


Figure 10. b: force-velocity curve

[Wilmore, J.H., Costill, D.L., Kenney, W.L. (2008). Structure and function of exercising muscle. Physiology of Sport and Exercise Fourth Edition. USA Human Kinetics]

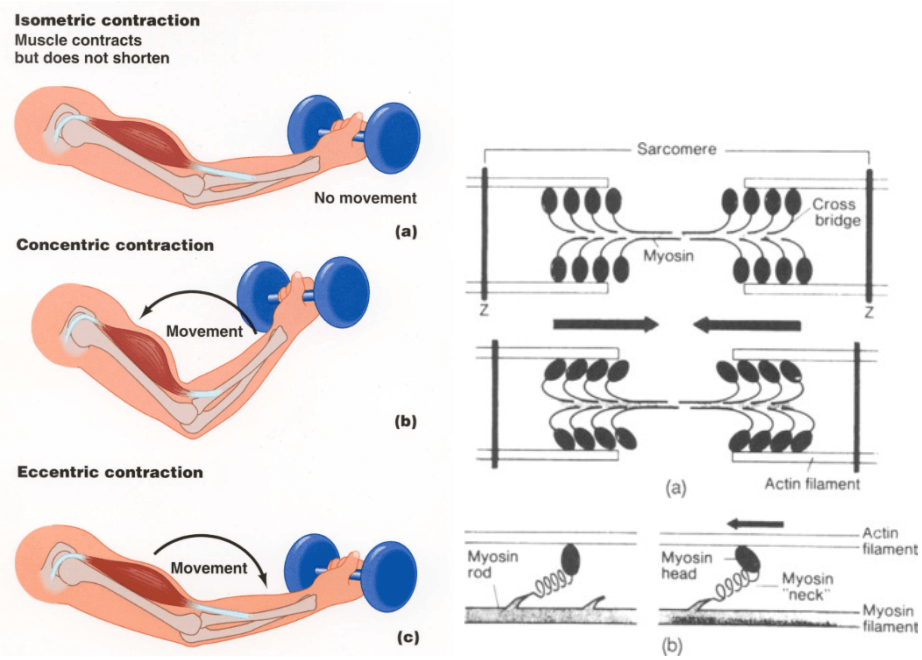


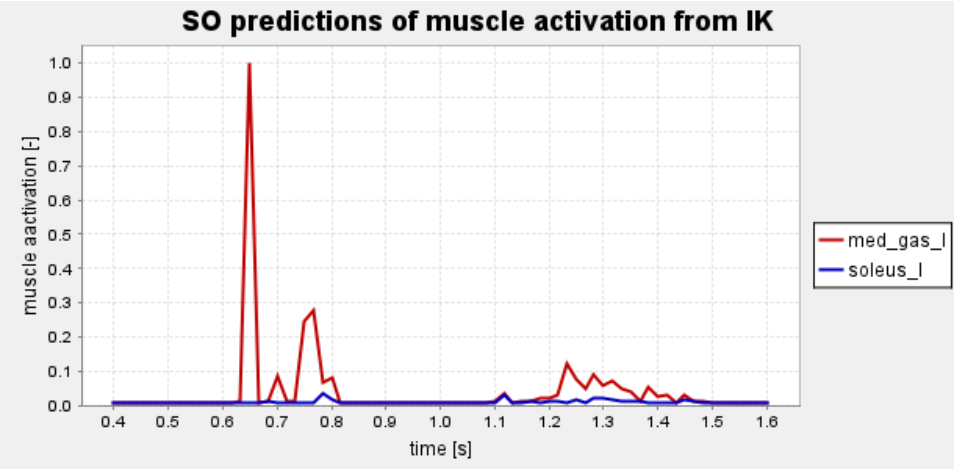
Figure 11: myosin head orientation while contracting [https://colebradburn.com/2013/02/06/biomechanical-work/] and [https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/myosin-head]

Question 22

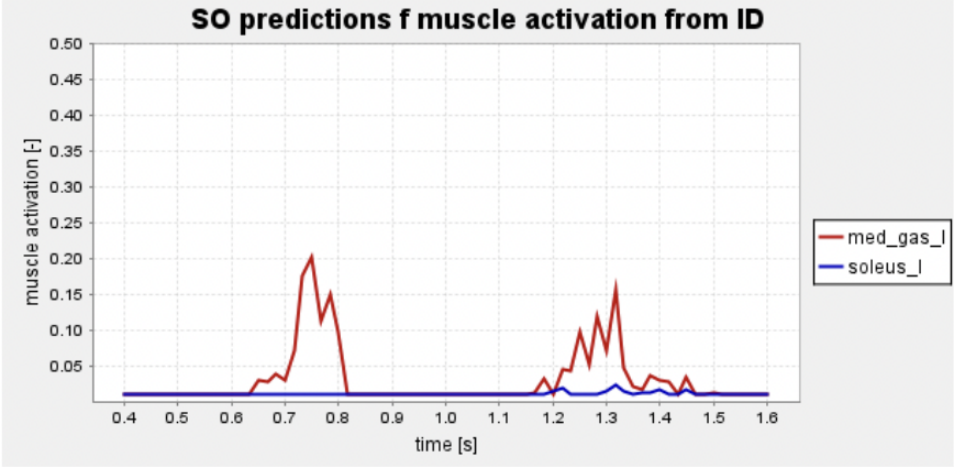
After executing SO (it can take a few minutes), the muscle activations are exported in subject_SO_activation.sto in the static_optimization folder. Plot and compare the activations predicted by SO with the EMG on–off timings reported by [Perry and Burnfield, 2010a] reported in [Bruel, 2022] (additional resources below) for the left gastrocnemius (med_gas_l) and soleus (soleus_l). Please discuss your results.

Static Optimisation (SO) is a frame-per-frame method that here calculates at each step in the simulation the muscle activation required to obtain the gait, either from the calculated coordinates of the Inverse Kinematic (IK) method or either from the calculated torques obtained with the Inverse Dynamic (ID) method.

Here we plotted in Figure 12 the muscle activation of the left gastrocnemius and soleus muscles obtained from ID and IK results and compared them with other simulations in the literature that are in Figure 13.



(a) prediction from IK results



(b) prediction from ID results

Figure 12: SO prediction for the left gastrocnemius and soleus

We observe that the activation picks obtained in Opensim range from 0.2 (i.e. 20%) to 1 (i.e. 100%). We have to be critical against these results, as for the gastrocnemius 100% activation obtained from IK is too much. We should not expect the muscle to be exploited to its maximum capacities during a simple walking gait, but rather during intense cases, jumping or sprinting for instance.

When comparing the timings, we have a good activation timing of the gastrocnemius at the end of the stance phase, it correspond to the fact of bringing the heel up to promote forward motion by pushing on the toes.

The On-Off EMG timings reported in [Bruel, 2002] are shown as grey lines in figure 13.

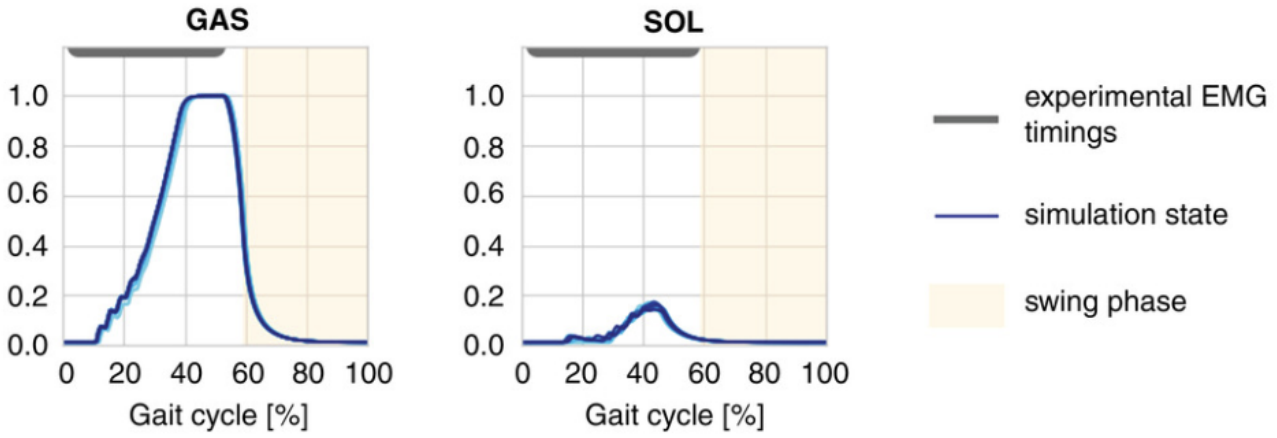


Figure 13: result from the paper

But what is the most striking in these results of Figure 12 is that we observe no activation of the soleus in the stance phase, and significant activation of the med_gas muscle in the swing phase, compared to the EMG signals given in the paper. Indeed we don't expect these muscles to be contracted in the swing phase.

We think that these errors are due to problems of optimisation: because the problem is ill-posed and has a lot of dimensions, very small variations in the model can lead to very different results. In fact, we can have different muscular activation for the same movement (we have a high redundancy of muscle in our body), depending on the position, the anatomy and the context. It is possible that the computation finds another minimum than the paper.

Remark: We could validate that the second pick of activation from 1.2 to 1.5 seconds corresponds to the swing phase by comparing the timing of the plot with the animated model in the visualiser window (see Figure 14).

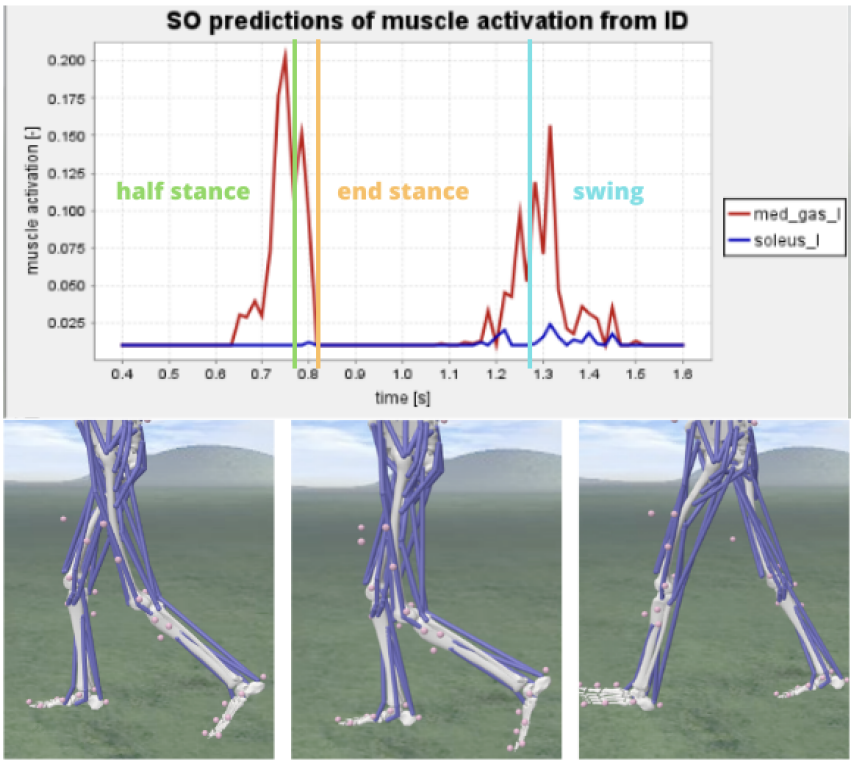


Figure 14: result at different moments of the gate cycle

Question 23

What is the consequence of the objective function used in SO in terms of effort (maximisation, minimisation, no effect, other)? And in terms of antagonist muscles co-contraction? Is it an interesting approach? Justify.

The objective function here minimises the muscle activation level across all muscles.

Activating a muscle requires effort, so more activation means more effort. In that sense, this objective function aims to minimise the total activation and thus the total cost of walking.

Since the objective function uses the sum across all muscles, it will also minimise antagonist muscle co-contraction. In fact, because antagonist muscles can compensate for their effect, they are multiple activation schemes that can create the same movement. But using both the antagonist and agonist muscle with too much co-contraction will lead to a higher global activation level, which needs to be minimised.

It is indeed an interesting approach because it minimises the effort at the level of the neuron's activation, which is natural from a physiological point of view. However, it is unclear whether the body will minimise these neural activations more than the effective muscle stress in the limbs (metabolic energy), even if it is related.

Question 24

Is this objective function used in SO appropriate for studying pathological conditions such as Parkinson's or Cerebral Palsy disease? Propose another objective function (the concept is important, and the formula is optional).

This objective function used in SO relies on joint activation levels, where the activation level is defined by the number of active motor units and the frequency of action potentials received.

This function relies on the neural signal to estimate muscle activity.

In the case of neuropathology such as Parkinson's or Cerebral Palsy disease, this measure may be noisy or irrelevant. In fact, Parkinson's disease leads to tremors (i.e. involuntary and rhythmic shaking), and neuronal signals will not necessarily be transmitted properly down to the muscle. Thus, whatever the optimal muscle activation, the effective contraction of the muscle might be different, so it is not the best objective function in that case. Similarly, Cerebral Palsy affects balance and posture and can lead to tremors and muscle constant contraction.

An objective function relying on muscle force may be more relevant because the patient is still trying to minimise energy.

$$f(F_m) = \sum_{m=1}^{nm} F_m$$

Figure 15: Objective function formula minimising muscle force - from the course (from the course)

It could also be interesting to rely on a combination of factors such as muscle force, stress and activation. The weight of each component can be adapted to match the disease we are trying to model.

$$J = \frac{1}{d}\int_0^{t_f} \left(\underbrace{w_1 \|\dot{E}\|_2^2}_{\text{Metabolic energy rate}} + \underbrace{w_2 \|a\|_2^2}_{\text{Muscle activity}} + \underbrace{w_3 \|u_{\text{dv,lt}}\|_2^2}_{\text{Joint accelerations}} + \underbrace{w_4 \|T_{\text{p}}\|_2^2}_{\text{Passive torques}} + \underbrace{w_5 \|e_{\text{arms}}\|_2^2}_{\text{Arm excitations}} \right) \mathrm{d}t,$$

Figure 16: objective function combining different factors (from the course)

Moreover, we have to take into account the pain of the patient. A function measuring the pain, and the stress on the patient could be interesting, even if it is a subjective measure.

We can also use the same function but choose a specific muscle set for the patient, for example, avoiding muscles with too much-unwanted activation. More generally, take into account the over-activation of muscles.

Question 25

In addition to morphological scaling and muscle force scaling (seen in class but not performed here), which other parameter of the musculoskeletal model would you personalise to a specific person?

In addition to morphological scaling and muscle force scaling, several other parameters could be scaled to individualise the model.

We can match joint range, general tendon and muscle flexibility, muscle architecture or ligament properties, and the fatigue factor of certain muscles.

Other pathological properties such as spasticity, atrophy, hyporeflexia/areflexia, and fasciculation could also be taken into account, especially when we want to study a pathological condition (e.g. tendonitis).

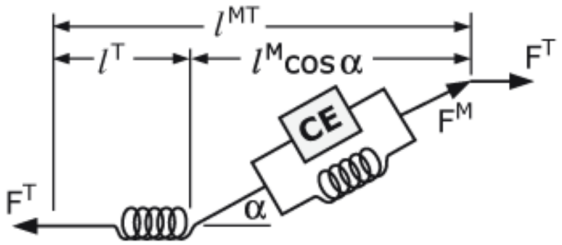
Question 26

How would you model muscle contracture that is common in Cerebral Palsy or Spinal Cord Injury conditions (in the muscle model described above)?

Muscle contraction is defined as a permanent tightening or shortening of muscle, tendons, tissues or joints, which leads to additional constraints that must be considered in the musculoskeletal model. Naturally, the tendon slack length and optimal muscle fibre length should be shortened, which will lead to an adjustment of the pennation angle.

Reduced joint mobility could then also be taken into account. Coordinate could also be fixed to a certain range during the simulation, accounting for the impossibility to reach certain angles or even totally blocked articulations.

Muscle activation may then also be impacted, and should be taken into account.



$$f^M = f_o^M \left(a f^L(\tilde{l}^M) f^V(\tilde{v}^M) + f^{PE}(\tilde{l}^M) \right)$$

$$\frac{da}{dt} = \frac{u - a}{\tau(a, u)}$$

$$f^T = f^M \cos \alpha$$

Figure 17: generic Hill-type model for muscle

Question 27

What does the EMG measure?

EMG (Electromyography) measure the electrical muscle response to a stimulus.
When a signal travels from the motor nerve to the muscle, the muscle fibre depolarizes producing an electrical activity, that can be recorded using electrodes.

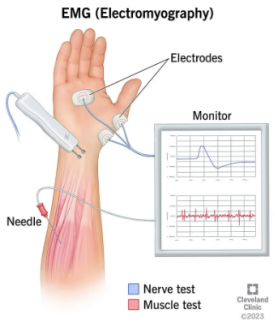


Figure 18: Set-up for EMG measurement

Question 28

Model validation is an important issue. The envelope of EMG recordings can be compared to the model muscle activation signals. Let us suppose that you have a dataset containing various lower limb kinematics and the corresponding EMGs for a specific patient. Propose a detailed protocol to validate the model.

In order to validate the model, we would try to compare the measured EMG with the one simulated by OpenSim with the static optimisation tool.

At first, we would preprocess the kinematic and EMG data, filtering out noise and removing potential artefacts. An option to compare easily the EMG data in a succeeding step would be to take a low-pass filter to extract the EMG envelope.

We would then develop a new musculoskeletal model in OpenSim based on the kinematic markers and scale it to the patient's morphology. We should simulate kinematics in OpenSim and extract the muscle activation signals with the static optimisation tool, with the help of previously run Inverse Kinematics results or Inverse Dynamics results.

To compare the results with the EMG, a qualitative analysis can be done, looking at the timing and amplitude of activation, but also a more quantitative analysis using root mean square error (RMSE). Correlation or coherence of the envelope could also be done. If the difference is not significant, the model could be validated.