Biomechanics and muscle function during gait

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Gait disorders in patients with cerebral palsy (CP) initially lead to functional deformities which later become structural. The deformities may greatly increase energy consumption and thus limit function. Under normal conditions, however, energy consumption is optimal [1]. Correction of biomechanics towards normality improves energy consumption [2, 3]. For this reason, correction of deformities aims at normality. As, in principle, normal function rather than normal anatomy is the goal, biomechanics and muscle function need to be understood in normal and pathological situations.

Gait analysis has yielded deep insights into these issues. Kinematics show the interplay of joint movements at a given time, but only the additional use of kinetics can help us to understand muscle function. A major problem in this respect is the anatomical bias of agonist–antagonist interplay: the main motors of motion. When interpreting kinetic gait data, it becomes obvious that this approach applies poorly to the functional situation. Joint moments are, to a great extent, the product of the ground reaction force (GRF) times its distance to the center of the joint. However, segment inertia and acceleration also contribute and should not be underestimated. The external moment is neutralized by an internal moment created by the muscles on the opposite side of the joint in order to maintain stability. As a consequence, under load, the functional antagonist to the muscle is the external moment. Thus, hip flexion (for instance) may not be due to hip flexor activity. The external moment may flex the hip and inadequate hip extension thus results from hip extensor weakness. In this case, the extensor side of the joint needs to be addressed.

Another consequence concerns the direction of muscle activity. Anatomically, a muscle pulls from insertion to origin, moving the distal body segment towards the proximal one. Under load, however, the distal body segment is the more stable of the two segments, as it is relatively fixed to the floor; the proximal one has more freedom of movement. This condition inverts the direction of muscle pull, and the proximal segment moves with respect to the distal one. Thus, the interpretation differs depending on whether a loaded (stance phase) and unloaded (swing phase) situation is considered.

One example of such a situation is internal rotation of the leg (seen as hip internal rotation) resulting from excessive dorsiflexion in the subtalar joint and the midfoot. Foot dorsiflexion is a compound movement of the ankle, subtalar, and midfoot joints. The contribution of each joint varies and depends on the length of the triceps surae muscle–tendon complex. The result is an oblique dorsiflexion axis that combines pure dorsiflexion with external rotation and pronation with respect to a fixed shank. If, however, the foot is fixed to the floor by loading and friction, the same movement rotates the leg internally [4, 5]. When the hip flexes and adducts, the pelvis also rotates and tilts anteriorly. This phenomenon becomes more important as the triceps surae shortens and the midfoot break becomes more pronounced. Severe cases present with a malalignment syndrome. Under normal conditions, however, adequate activity of the tibial muscles stabilizes the foot, avoiding this phenomenon. Another physical effect was described as a plantar flexion–knee extension couple [6, 7]. Only under load is the triceps muscle capable of holding the tibia back and in this way extending the knee (Fig. 1). Due to joint connections and inertia, the effect of this single muscle only travels proximally and leads to hip flexion, internal rotation, and adduction, together with

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Fig. 1 Schematic drawing of the plantar flexion–knee extension couple (working only under load)

pelvic retraction, forward tilting, and elevation, as well as respective movements in the spine [8]. All of these movements are simple physical effects and thus should be seen whenever there is an increased plantar push. Avoidance of these movements is an active compensation by the patient as a consequence. Overactivity of the plantar flexors may hyperextend the knee in this way (pathological plantar flexion–knee extension couple) [6, 9].

Nevertheless, this plantar flexor–knee extension couple plays an important role in knee control during gait. The knee extensors become relatively insufficient towards full knee extension [10]. They control only the first phase of knee extension during the response to loading. The second phase—resulting in maximal knee extension in healthy individuals—is contributed by the plantar flexors, as described (Fig. 2). When the first phase is missing, toe walking occurs, with loss of the plantar flexion–knee

Fig. 2 Kinematic curve of the knee in the sagittal plane, normal ± 1 standard deviation. The first phase of knee extension after accepting the load of the body is performed by the knee extensors, and the second phase by the plantar flexors (by the plantar flexion–knee extension couple). The crossover between the two mechanisms may differ among individuals

extension couple (as in CP patients with a flexed knee gait), whereas a missing second phase leads to a mild crouch at least (as in spina bifida patients, despite having normal strength of the quadriceps). Both phases are necessary for normal control of the loading response at the knee with well-controlled muscle interplay, which probably varies among individuals. The loss of both phases of knee extension, on the other hand, is known as crouch gait in CP.

As described earlier, an important role of muscle activity is controlling the external moment, including gravity or the ground reaction force during motion. It is thus highly questionable that a patient with a hip flexion posture should have hip flexor activity (if he did, he would collapse). There may be the idea of co-contraction of the hip extensors at the same time. However, with the external moment (including gravity) as a synergist, the flexors would still lead to a collapse. Moreover, if we look more closely, agonist and antagonist do not necessarily work against each other: in the knee joint for instance, the anatomy highlights an important difference between the two muscle groups. Whereas the knee extensors mainly consist of the monoarticular vasti (80 %) at the knee, the ischiocrural muscles (except for the short biceps belly) are biarticular, extending over knee and hip joint. This arrangement allows hamstring activity to be modified under load. With a bent knee, the ischiocrural muscles are potent knee flexors due to the lever arm situation at the knee under load. If their power is requested at the hip, however, simultaneous contraction of the vasti locks the

knee and shifts the power of the hamstring to the hip [11]. This happens in normal (e.g., at the start of a sprint) and in pathological (crouch gait) situations. These examples show that muscles may have distant effects that are not obvious, and that co-contractions may be physiological.

The effect of load is especially interesting when we consider the ischiocrural muscles. Unloaded, they are potent knee flexors, but they become important hip extensors under load as long as the knee is not bent too much [11]. As they are active under both conditions, they control both joints. At initial contact, they actively contract in order to flex the knee and thus induce knee flexion during the response to loading. Without this knee flexor activity, the leg would extend due to the acceleration of the pelvis and thigh and a pelvic inclination moment when pushing off, derived from the contralateral leg [12]. As the leg is loaded, the direction of activity of the ischiocrural muscles changes and they become hip extensors. The simultaneous contraction of the vasti increases this effect. They shut off after loading, and may show only mild and inconstant activity in pre-swing. At that point in time, the leg is unloaded and the ischiocrural muscles flex the knee. Their next activity during the gait cycle is to dampen knee extension towards the end of swing. While this list of physical reactions, co-contractions, and load effects is certainly incomplete, these findings afford important insights into muscle function and help us to understand normal and pathological gait.

Understanding joint control under gravity helps us to assess normal and pathological gait. In principle, there are two ways to control a joint: some muscles act directly on the segments connected by the joint and for example produce extension, and other muscles control the external moments (with a contribution from the GRF); for instance, an extending moment derives from a GRF on the extensor side of the joint. The movement of the ankle is crucial in midstance: the triceps surae locks the ankle joint and thus produces an external extending moment at the knee as the GRF advances. In the case of triceps surae weakness, the GRF is held close to the center of joint rotation, thereby reducing the external dorsiflexing moment, and dynamic instability of the leg can only occur at terminal stance/pre-swing when the body needs to move forward. At the knee joint there are three options for control: the knee extensors, the plantar flexion–knee extension couple, and the external moment. The latter is often used for compensation: the trunk of the patient leans forward in order to shift the GRF in front of the knee, creating an extending moment. This position is controlled by the hip extensors (including the ischiocrural muscles) as the hip is passively flexed (Fig. 3). Finally, hip extension is provided by either the hip extensors (including the ischiocrural muscles) or by a backward trunk lean that shifts the GRF behind the center of the hip

joint, creating an external extending moment (and an external flexion moment at the knee that requires knee extensor activity). The control of adduction in the case of abductor weakness is well known as a Duchenne limp.

In normal gait, there are at least three crucial phases. First, there is initial contact, which is prepared during terminal swing and is thus described at the end of the step. Stability during single limb support is important in order to ensure that the opposite leg is cleared and the leg can be swung forward. In stance, the plantar flexors control the ankle. The knee is extended first by the knee extensors and second by the plantar flexors over the external moment including the GRF (the plantar flexion–knee extension couple). The hip is first extended by the hip extensors (including the ischiocrural muscles) and secondly by the external moment including the GRF, in a similar manner to the knee. As leg stability is crucial during this phase, compensations aim at extending the knee: premature plantar flexor activity and hyperactivity [13], avoiding knee flexion during loading response, and forward leaning of the trunk with co-contraction of the knee extensors and ischiocrural muscles in the case of knee flexion. The next important phase is pre-swing, when the leg is accelerated as a biarticular pendulum that folds and extends passively during swing. This acceleration is normally generated by the triceps pushing off and hip flexor activity. Weakness in either part may shift the load to its partner, and in severe cases the ischiocrural muscles will be activated as well,

Fig. 3 Schematic drawing showing how indirect control of knee extension is achieved by shifting the center of mass anteriorly. Posture control in this situation is performed by the hip extensors (the hip flexors are not active)

acting as knee flexors in the increasingly unloaded leg. The role of the rectus, however, remains unclear. Overactivity at this time, often together with the vasti, may be one reason for delayed peak knee flexion during swing, with the consequence being inadequate extension at terminal swing. Adequate knee extension at this time is crucial to achieving heel contact. At terminal swing, the ischiocrural muscles normally avoid hyperextension of the knee.

Plantar flexor overactivity is a common problem in neurological cases with spasticity. It has been shown, however, that such overactivity can be seen in flaccid paralysis or even non-neurological cases as well, where it is most closely correlated with muscle weakness anywhere in the leg [13]. Patients with spastic cerebral palsy always present with weakness too, and it remains difficult to decide whether muscle overactivity is part of the compensation for weakness, spastic reflex activity, or simply due to a generally high muscle tone. Unfortunately, neither kinematic nor kinetic nor electromyographic data from gait analysis are of any help in this respect. Plantar flexor hyperactivity is a particular problem in patients with spasticity and high tone, as they have a high risk of developing a structural equinus deformity. This deformity can initially be compensated for by knee hyperextension. In this situation, the knee extensors are mainly inactive. As knee hyperextension is limited, however, further foot deformity would force the patient to fall backwards. At that moment at the latest, the knees must be bent to recover balance. The knee extensors must then cope with gravity after a long time without activity. The poor preparation for this situation may be one reason for the increased rate of knee flexion in these patients.

Following on from the biomechanical and functional descriptions given above, the paragraphs below provide a rough overview of the compensations for function in pathological situations:

- Restrictions on the ranges of motion of joints and muscle shortness affect either flexion or extension. Both situations functionally affect leg length: the leg is short in stance or long in swing. Both situations are compensated for in a similar way to leg length discrepancies, according to the possibilities of the locomotor system.
- Leg length discrepancies lead to a short leg in stance and a long leg in swing contralaterally. In stance, the short leg shows increased plantar flexion and knee and hip extension in order to lengthen the leg, whereas the contralateral leg shows the opposite. If the leg remains long in swing, there may even be vaulting by the plantar flexors or circumduction.
- Muscle weakness of any cause affects joint control. Either synergists can be activated (like the flexor

hallucis for weak triceps surae) or the center of mass is positioned in such a way as to avoid working the weak muscle (e.g., Duchenne lateral trunk lean).

- The situation in cases with spasticity is more difficult. Spasticity leads to inadequate joint positions at particular moments in time (too flexed, too extended), which may be absolute (limited motion) or relative (delayed motion). These positions again affect leg length, but at the same time the muscles are weak, which usually affects the legs globally. All of these factors reduce the stability of the legs, which again deteriorates gait function. This combination makes it very difficult to understand cause and compensation in individual patients.

This list of pathological situations and compensations is incomplete; it may be that not all of the possibilities are known yet. However, understanding the biomechanics of normal gait and muscle function provides the necessary basis for detecting and understanding pathological situations. Instrumented three-dimensional gait analysis is an essential tool for assessing functional problems and thus determining the appropriate treatment.

Conflict of interest None.

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