# Spasticity, muscle, and surgery

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The title contains three topics which are important for treating locomotor disorders in patients with cerebral palsy. Although linked, they need to be considered in more detail.

Spasticity is a poorly defined concept of increasing resistance against fast movements. It comprises a static picture of lost control on reflexes, and primitive reflexes are unmasked. This view is too simple. Reflexes can be modulated by the individual over time, which means, that increase and decrease are possible. Also, some control on reflex activity can be regained. Especially the affection of the sensory parallel to the motor pathways can explain the difficulties to build up a better control. A lot of time and movement experience are required, reflected in developmental delay and improvement of spasticity in at least some patients over time.

The diagnosis of spasticity creates a bias when deciding on the type of muscle activity during function. Although there is no tool available up to date to diagnose spasticity during gait, aberrant muscle activity shown by dynamic EMG recordings is often regarded of spastic origin. Toe walking due to gastrosoleus overactivity is hence treated by reducing spasticity which mainly means reducing muscle strength. Comparing the EMG activities of gastrocnemius and tibialis anterior in different diseases leading to an equinus gait pattern, however, reveals the same control pattern also in patients without spasticity. There are different reasons for this pattern of premature gastric activity with shutoff of the tibialis anterior in late swing: a primitive gait pattern (like toddlers) in developmental retardation, lack of confidence in the leg stability, compensation for a leg length discrepancy, or weakness, amongst others. In a study performed in 2013, we showed that this gait pattern was significantly more frequent in any case of weakness, due to any disease. Spasticity may be one more cause, and it may lead to early deformity when combined with other causes. A second phenomenon is prolonged activity of knee extensors (vasti) and hamstrings. This activity is, however, usually characterized by clear activity and pause. This implies an effective motor control. The posture during gait explains at least the major part of this pattern. Similarly, the co-contraction of vasti and hamstrings needs a closer analysis: This is not a true situation of agonist and antagonist as the vasti are monoarticular, the hamstrings biarticular muscles. This way, the simultaneous contraction of the vasti modifies the effect of the hamstrings in respect of knee flexion and hip extension. This interaction is difficult to assess as the EMG does not reflect muscle force, and the net joint moment only reflects the total of the acting moment. A conclusion on single muscle groups is most often not possible. Considering these points, spasticity may be less important during gait than expected. In another study we showed that the importance of weakness greatly overrides the one of spasticity. Nevertheless, this does not exclude the need to treat impeding muscle overactivity.

Muscles in patients with spasticity undergo a structural change towards smaller muscle bellies, more stiffness, sarcopenia, slower fibers, fibrosis, as well as genetic and metabolic changes. These changes depend on the severity of the affection. They lead to weakness, slower contraction and relaxation, and more resistance against passive movement. They are increasingly seen as adaptations at the altered use of these muscles during daily activities. Weakness may also arise from less neuromotor input and a loss of lever arm, and a combination of all factors. Spasticity and muscle contracture in contrast may help to compensate.

Surgery to lengthen the muscle-tendon unit must be indicated carefully for this reason. Tenotomies done in the 1980ies proved to be excessive resulting in stiff and slow gait. The consequence was to change to intramuscular tenotomies or aponeurotomies. The effect of these procedures is less dramatic. Immediately after the cut the distal fibers are torn off the fibers closer to the origin of the muscle. This produces some length and loss of strength by stretching postoperatively. The cut is filled by scar tissue which erases part of the effect. This applies also to the more modern version of this type of lengthening, the fibromyotomy or the Ulzibat procedure. The main difficulty of soft tissue surgery is the poor control on the amount of lengthening and loss of force by our surgical techniques. Procedures which correct bony deformities and address muscle-tendon overlength seem to do better in respect of dynamics and outcome. Muscle-tendon lengthenings should be indicated cautiously.

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