Gait improvement with decreased tibialis anterior recruitment after botulinum toxin injections into peroneus longus in very young children with hemiparetic cerebral palsy

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**Introduction**

In very young cerebral palsy (CP) children, peroneus longus (PL) overactivity by spastic cocontraction (Gracies, 2005; Vinti 2018) is a major contributor to dynamic equinovalgus during swing phase (SW) and at initial contact (IC) (Boulay, 2012). This study assessed the effects of abobotulinumtoxinA injections into PL.

**Methods**

*11 male children with hemiparesis (7 right; age 3.1±0.6 yo)*  
*only one injection in Peroneus Longus, without GastroSoleus Complex(GSC) injection*  
*GSC was assessed for functional length (X1), and spasticity (X2)*  
*Gait analysis videos were evaluated using the Edinburgh Visual Gait Score (EVGS)-validated in CP): pre vs post-injection values were compared using paired t-tests.*  
*EMG monitored tibialis-anterior (TA), gastrocnemius-medialis (GM) and PL during gait.*  

Swing phase was divided into three equal periods (T1, T2, T3) to measured: during SW (SW T1, SW1, SW2, SW3): GM and PL cocontractions and TA recruitment and during standing: on tiptoes (EMGmaxPL, EMMaxGM, on heels(EMGmaxTA)

**Results**

For each measured EMG variable (SW and standing), efficacy index (normalized) was assessed using the ratio:  
\[
\text{Efficacy index} = \frac{\text{EMG}_{\text{variable X POST}}}{\text{EMG}_{\text{variable X PRE}}}
\]

Each efficacy index was compared with an inefficacy theoretical index of 0.

**Conclusions**

*no side effect (no taulus, no drastic paretic effect)*  
*Foot clearance* is improved, *hindfoot* is decreased  
*In knee extended, GSC spasticity (X2) (NON INJECTED) switched to the dorsal flexion after PL injection which is correlated with a significant knee re-extension in terminal SW (EVGS).*  
*It was associated with reductions of PL and GM cocontractions*  
*TA is the antagonistic muscle of PL.*  
*This constitutes an argument supporting that increased TA recruitment in children with hemiparesis may be an attempt by the nervous system to compensate for plantar flexor cocontraction rather than the opposite (increased plantar flexor cocontraction due to increased TA recruitment)*  
*we can discuss a central action of abobotulinumtoxin A*

**References**

Gracies JM, Muscle and Nerve, 2005  