CAN ELECTROMYOGRAPHIC ASYMMETRIES DURING GAIT BE EXPLAINED BY LIMB DOMINANCE?

Matthew K. Seeley [1], Brian R. Umberger [2], and Robert Shapiro [3]

[1] Brigham Young University, Provo, UT, USA
[2] University of Massachusetts, Amherst, MA, USA
[3] University of Kentucky, Lexington, KY, USA
E-mail: matt_seeley@byu.edu

INTRODUCTION

Subtle asymmetries exist in kinematic, kinetic, and electromyography (EMG) data describing able-bodied gait, yet the causes of these asymmetries remain unclear (Sadeghi *et al*., 2000). Some scientists have suggested that these bilateral asymmetries may reflect differences in functional roles of the lower limbs, with the non-dominant (ND) limb contributing more to support, while the dominant (D) limb contributes more to propulsion (Sadeghi *et al.,* 2000). However, bilateral symmetry has been demonstrated for impulses due to vertical (i.e., support) and propulsive ground reaction forces during normal gait (Seeley *et al.,* submitted) indicating that, at a global level, each lower limb contributes equally to support and propulsion. Symmetrical impulses, however, do not necessarily imply "local" bilateral symmetry for measures such as EMG or joint kinetics. Sadeghi et al. (2003) proposed that such local asymmetries may represent compensatory strategies that help ensure global symmetry of lower-limb function.

The purpose of this study was to determine if local asymmetries in EMG are related to hypothesized functional differences (support and propulsion) of the lower limbs during normal gait, and to see if these differences depend on walking speed. Bilateral EMG was monitored for specific muscles throughout intervals of the gait cycle during which these muscles have been shown to contribute to support or propulsion (Anderson & Pandy, 2003; Neptune *et al.,* 2004). Two general hypotheses were expected to be supported if EMG asymmetries are related to functional asymmetry. First, support-related muscles would be more active for the ND limb and propulsion-related muscles would be more active for the D limb. Second, with increases in walking speed, D-limb muscle activity related to propulsion would increase disproportionately compared to ND-limb counterparts. This limb × speed interaction was not predicted for support-related muscles, as gravity is independent of speed.

METHODS

Bilateral surface EMG data were collected (1200 Hz) from the gluteus maximus (GMX), gluteus medius (GMD), vastus lateralis (VLA), semitendinosis (SMT), and soleus (SOL) during walking for 20 subjects (age = 25 ± 3 yr; ht = 1.7 ± 0.1 m; mass = 70 ± 14 kg) at three speeds: preferred (1.61 ± 0.01 m/s), -20% (1.37 ± 0.01 m/s), and +20% (2.02 ± 0.04 m/s). EMG data were normalized to maximal voluntary isometric contraction amplitude and time normalized to a full gait cycle. Mean muscle amplitudes during intervals of the gait cycle for which those muscles have been shown to contribute to support or propulsion were averaged across five trials for each subject, limb, and speed. Mean amplitudes associated with support were GMX, GMD, VLA, and SOL during the first 30% of the gait cycle. Mean amplitudes associated with propulsion were SMT during the first 30% of the gait cycle, and SOL between 30 and 50% of the gait cycle. A repeated measures ANOVA ($\alpha = 0.05$) was performed to detect effects of limb and walking speed on dependent variables. Bonferroni-Holm *post hoc* analyses were conducted to detect bilateral differences at each walking speed.

RESULTS AND DISCUSSION

No significant limb × speed interactions were indicated, so data pooled from each speed were compared bilaterally for each dependant variable. GMD activity related to support was 25% less for the ND limb. SOL amplitudes related to support and propulsion were 55% and 31% less for the ND limb, respectively (Table 1).

The data offered little support for the first hypothesis and no evidence for the second hypothesis. Support-related muscles (GMX, GMD, VLA, and SOL during the first 30% of the gait cycle) were expected to be greater for the ND limb. However, only GMD and SOL were bilaterally different, and these differences were opposite to the predictions. Propulsion-related muscles (SMT between 0 and 30%, and SOL between 30 and 50%) amplitudes offered limited support for the first hypothesis. SMT activity was not bilaterally different, yet, as was predicted, mean propulsion-related SOL amplitude was greater for the D limb. Present data failed to support the second hypothesis, as no limb × speed interactions were indicated. During gait, propulsion requirements increase disproportionately in comparison to support requirements as walking speed increases. This is because gravity remains constant. Consequently, propulsive D-limb SMT and SOL activity were expected to increase disproportionately in comparison to ND-limb counterparts as walking speed increased, yet this was not observed.

Table 1: Mean EMG amplitudes (%MVIC) during specific intervals of the gait cycle (see text for details). *Asterisks indicate statistical significance.

	Mean Amplitude	
Support	**ND**	**D**
GMX	8.6 ± 0.9	11.8 ± 1.9
GMD* ($p = 0.02$)	8.1 ± 0.5	10.1 ± 0.7
VLA	8.8 ± 1.1	7.7 ± 0.7
SOL* ($p = 0.02$)	10.8 ± 0.9	16.7 ± 1.9
Propulsion		
SMT	4.9 ± 0.4	5.5 ± 0.6
SOL* ($p = 0.03$)	31.8 ± 2.8	41.6 ± 4.4

SUMMARY/CONCLUSIONS

Present results indicate that bilateral EMG asymmetries are not likely related to lower-limb task differences, as conceptualized within the functional asymmetry framework (Sadeghi *et al.,* 2000). Only one of the six bilateral comparisons supported the first hypothesis that was based on functional asymmetry. Additionally, the walking speed manipulation failed to support the idea that EMG asymmetries are related to lower-limb functional differences. Other plausible causes of local EMG asymmetries during normal gait including lower-limb morphological asymmetry or environmental issues should now be explored. Elucidation of this issue may advance our understanding regarding the role of the neuromuscular system during cyclic activities such as gait.

REFERENCES

Anderson F. & Pandy M. (2004). *Gait Post,* **17,** 159-169.
Neptune R. et al. (2004). *Gait Post,* **19,** 194-205.
Sadeghi, H. et al. (2000). *Gait Post,* **12**, 34-45.
Sadeghi, H. (2003). *Gait Post,* **17**, 197-204.
Seeley, M. et al. (submitted). *Gait Post.*