

Conservation of Limb Function after Peripheral Nerve Injury in Rat Locomotion

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Introduction

The capacity for locomotion persists after neuromuscular injury to the limbs via the principle of compensation--a change in some local properties such that a greater goal may continue to be met. Yet there is a dearth of knowledge on general compensation mechanisms and how they relate to specific neuromuscular pathologies. The elucidation of common features in locomotor compensation and their correlation to different injuries could yield insights into general limb control strategies during healthy and impaired locomotion.

Though rodents have emerged as the overwhelming model of choice for spinal cord and peripheral nerve injury studies, current locomotor assays in rats lack the resolution to study subtle motor deficits associated with recovery from nerve injury. Investigators typically characterize rat locomotion with subjective survey tools (e.g. BBB locomotor rating scale), or by spatio-temporal stride parameters (e.g. Sciatic Function Index) [4]. Unfortunately these methods cannot provide data on interjoint compensation or kinematics of the proximal joints, and have been demonstrated to incorrectly imply full recovery despite visual evidence of motor impairment [5].

Joint kinematics provides more comprehensive understanding of limb function and recovery, but optical kinematics derived from skin markers are not accurate due to skin movement relative to the underlying joints [6]. We have built a high-speed x-ray video system to directly measure accurate whole limb kinematics from bone positions. Our previous results revealed errors in knee angle as large as 40° at foot contact compared to optical methods. This system permits us to comprehensively examine basic compensation mechanisms to a variety of peripheral nerve injuries.

We denervated major ankle plantarflexor muscles in rats to quantify specific locomotor deficits and patterns of interjoint coordination over the time course after injury. Muscle-specific changes were

observed in similar surgeries on cats depending on the multiarticular character of the involved muscles [1,2]. Asymmetries in stride parameters such as duty factor have also been observed [3]. Peripheral nerve cut injuries clearly disrupt local variables such as joint angle trajectories, but this may be a necessary condition such that the values of other limb properties can be conserved from the pre-injured state. We hypothesized that the locomotor deficits observed after muscle denervation would correspond to a stabilization of leg length and leg orientation as global variables for walking in rats.

Methods

Surgical denervations were performed on three male Sprague-Dawley rats under aseptic conditions. Incisions were made in the posterior popliteal area of the left hindlimb and the branches of the tibial nerve leading to the selected ankle extensor muscles were isolated with retractors. Then the nerves were cut close to the muscle and as large a section as possible was completely removed. This peripheral nerve injury protocol removed innervation to the entire triceps surae group (lateral gastrocnemius, medial gastrocnemius, and soleus) and plantaris.

We captured sagittal plane x-ray video images of the rats at 200 Hz as they walked on a treadmill at 40 cm/s. Data were collected from each rat on PID (post-injury day) 7, 10, 14, 21, and 28. Distal limb joints were tracked using custom Matlab software (T. Hedrick, UNC-Chapel Hill) with radio-opaque tantalum markers due to minimal skin movement, whereas the hip knee and pelvis were manually digitized from bony landmarks. Analyses consisted of calculating joint angle trajectories, interjoint coordination patterns, toe-to-hip leg length and orientation, and stride parameters of the injured hindlimb. The data were then compared to data from four healthy control rats at the same speed.

Results and Discussion

Joint angle trajectories from all three injured rats on PID 7 show that the ankle and MTP joints were notably altered after denervation (>50° difference

from control data for most of the cycle, **Fig. 1**). Knee and hip joint trajectories were moderately altered with increased extension ($\sim 20\text{-}40^\circ$).

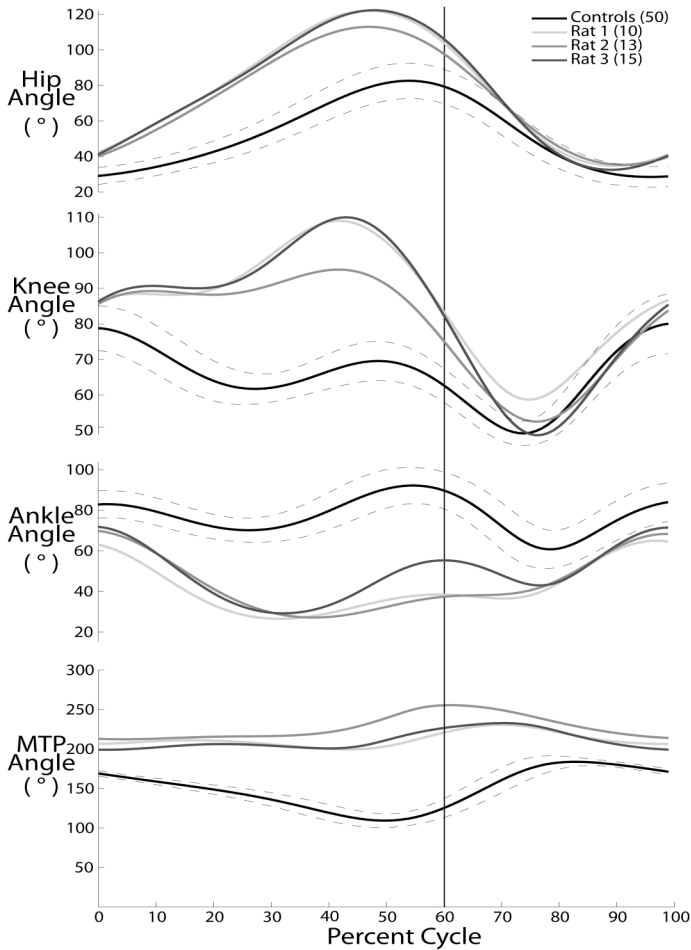


Fig. 1: Mean joint angle trajectories at PID 7 versus mean of all controls. Dashed lines are ± 1 SD of control, #step cycles in parentheses. Vertical line indicates end of stance phase.

In contrast, preliminary results for the same rats indicate leg length and orientation trajectories were unchanged (**Fig. 2**), confirming our hypothesis. There were no significant differences in mean values of leg length and leg orientation between the injured and control rats over the entire normalized gait cycle ($p < 0.01$). The new joint configurations from injured animals taken together are consistent, or goal-equivalent, with a limb configuration that preserves leg length and orientation trajectories from the pre-injured state. Interjoint compensation patterns were visibly disrupted except for hip-knee coordination, which maintained the same shape over greater ranges.

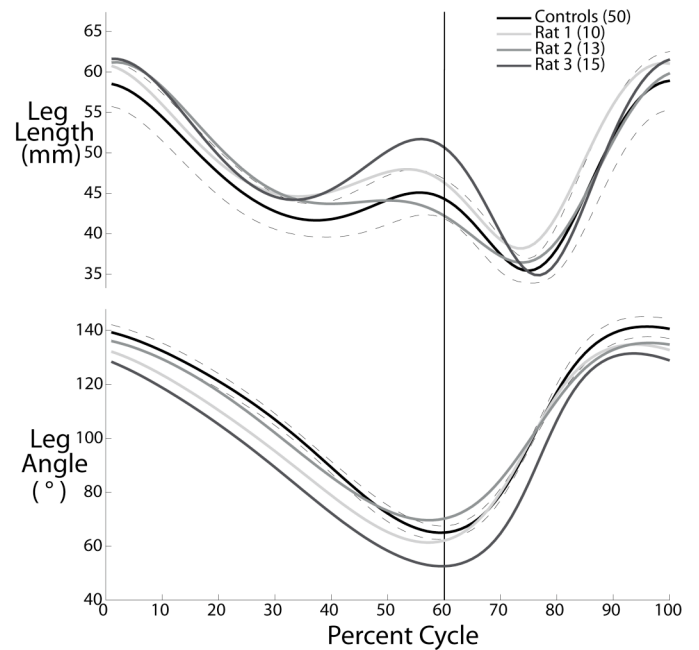


Fig. 2: Mean leg length and orientation trajectories for 3 rats at PID 7 versus mean of all controls. Legend is same as Fig. 1

Conclusion

Surgical denervation of all four ankle plantarflexor muscles resulted in an expected large-scale disruption of joint angles and stride parameters. Notably, values of leg length and orientation were indistinguishable from controls after injury, despite paralysis of all major ankle extensors. Along with the results of this study, a systematic mapping of compensation patterns to different nerve injuries will ultimately yield a valuable baseline for evaluating the efficacy of reinnervation (nerve repair) surgeries, as well as provide insights into basic principles of locomotor compensation.

References

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