

Role of trunk sensorimotor cortex in promoting cortical plasticity associated with successful robot rehabilitation training in adult rats spinalized as neonates

Chintan S. Oza and Simon F. Giszter, *Member, IEEE*

INTRODUCTION

Recovery of function after spinal cord injury (SCI) and rehabilitation training involves cooperation between novel skill development and intrinsic spinal mechanisms. Rats with complete SCI at T8-T10 at post-natal day 1-6 (NTX) as adults generate some reflex stepping motions. A subset of these rats (~ 20%) achieves autonomous weight support (WS). Motor reorganization of trunk/hindlimb sensorimotor cortex is believed to play an important role. Lesions to trunk/hindlimb cortex in WS NTX rats' results in loss of WS function [1]. Using trunk robot rehabilitation, we previously showed that NTX rats significantly improve function and associated plasticity of trunk motor cortex. Further, NTX rats with lesions to trunk sensorimotor cortex prior to the start of robot rehabilitation did not improve function with training. Here, we test if synergies and representation in NTX rats with cortical lesion are altered by robot training as in non-lesioned NTX rats with robot rehab. We hypothesized that disrupting sensorimotor integration in lesioned NTX rats that results in a failure to improve function will also result in lack of plastic changes in the motor cortex.

METHODS

25 female Sprague-Dawley rats received neonatal transection at post natal day 5/6. Post weaning (~ 4 weeks) until adult age (~ 8-10 months) rats received moderate treadmill training (5 min/day, 3 days/week) after which they were implanted with a pelvic orthosis as described in [2]. Using a heat cautery we lesioned trunk/hindlimb sensorimotor cortex in the lesion NTX group (n=12) whereas the control NTX group (n=13) had no cortical lesion. Both groups were subsequently trained quadrupedally on the robot which applied isotropic elastic forces (stiffness 45 N/m) at the pelvis during treadmill locomotion for 20 min/day, 5 days/week for 4-5 weeks. Following training we used intracortical microstimulation to map the motor cortex representations in all NTX rats (n=7 lesion group, n=8 control group). For analysis of motor maps we considered motor sites from at and above the bregma line (rostral to lesioned cortex) in all NTX rats. Measures used to assess changes in motor maps were synergy (coactivation), segmental level of representation, and representation topography. These were compared in cortex lesioned and non-lesioned robot trained NTX rats. Measures of robot body weight support, Antri Orsal Barthe stepping measures and percentage of WS steps were used to assess function in the lesioned and non-lesioned groups.

RESULTS / CONCLUSION

All non-lesioned NTX rats showed significant improvement of function with robot training. Lesioned NTX rats did not improve function. Results from motor maps show that rehabilitation failure in NTX rats with lesioned cortex also associates with a lack of any plastic changes in the motor cortex. Specifically NTX rats with cortical lesions in trunk overlap cortex, elsewhere lack the emergence of novel complex synergies between different trunk segments, fractionation of trunk and forelimb representation, expansion of caudal trunk areas and a caudal displacement of trunk motor representation in the cortex, all seen in successful rehab. Trunk sensorimotor overlap cortex is therefore an apriori necessary element required for functional improvement in robot rehabilitation in NTX rats, in that lesion abolishes both recovery of function and motor plasticity in other areas and it thus also may play an important role in mediating or supporting other aspects of cortical plasticity also associated with successful robot rehabilitation training. We speculate that this area's key role is in skill representation needed to integrate trunk control between voluntary and spinally controlled regions of trunk.

REFERENCES

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