Does Disconnected Cerebral Cortex Become Atrophic Post Cervical Spinal Cord Injury?

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Purpose
Functional neuroimaging has demonstrated adaptive changes in the cerebral cortex of spinal cord injury (SCI) patients (1). To our knowledge, regional volumetric changes in the brain following SCI have not been described. Our objective was to use MR imaging to look for evidence of gray matter volume changes in the motor cortex of SCI patients who, as a result of their injury, have limited or no use of their upper extremities.

Materials & Methods
T1-weighted SPGR volumetric acquisitions were performed in 31 chronic (1-160 months) cervical SCI patient hemispheres and 28 controls. Mean time from injury was 47 months. Patients with head injury were excluded. The characteristic “knob” of the upper extremity motor cortex in the precentral gyrus was identified according to Yousry(2).

Two different methods were used for volumetric analysis under blinded conditions: 1) A manual algorithm was developed to assess cortical area along a fixed distance of the precentral knob using 3D analysis software provided by GE Medical Systems (AWW 4.0). Measurements were averaged between two independent observers. The result was compared with the total brain gray matter volume derived from Statistical Parametric Mapping 1999 (SPM99) (3); 2) Automated voxel-based morphometry (VBM) (4) using SPM99 was applied to look for regional differences in gray matter throughout the cortex including, but not limited to, the precentral knob.

Results
The first analysis method revealed a mean knob cortical area of 48 mm² ± 6 in the patient group, versus 50 mm² ± 8 in the controls (p = 0.354). There was no significant difference in total gray matter volumes between patients and controls (p = 0.19). The second analysis using VBM showed no focal areas of gray matter reduction in the cerebral cortex.
in the SCI patients.

**Conclusion**
A multimethod approach searching for global and regional volumetric changes in the cerebral gray matter of chronic SCI patients failed to show any differences compared to controls. Absence of focal cortical atrophy in the motor cortex controlling upper limb function in these movement impaired patients is not surprising in view of our previous observations that this cortex becomes coactive with movement of structures above the level of the cervical injury, i.e., tongue movement (1). It appears that atrophy is not a sequella of de-efferentation/de-afferentation in this group of patients.

**References**
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