OP- Scientific 1-2

Which brain lesions produce spasticity?

Seong Hoon Lim[1*†], Kyoung Bo Lee[1], Bo Young Hong[1], Joon Sung Kim[1], Bomi Sul[1], Won Jin Sung[1]

St Vincent`s Hospital, College of Medicine, The Catholic University of Korea, Department of Rehabilitation Medicine[1]

Objective
Spasticity is an important barrier that can hinder the restoration of function in stroke patients. Although several studies have attempted to elucidate the relationship between brain lesions and spasticity, the effects of specific brain lesions on the development of spasticity remain unclear. Thus, the present study investigated the effects of stroke lesions on spasticity in stroke patients.

Materials and Methods
The present retrospective longitudinal observational study assessed 45 stroke patients using the modified Ashworth Scale to measure muscle spasticity. Each patient was assessed four times: initially (within 2 weeks of stroke) and at 1, 3, and 6 months after the onset of stroke. Brain lesions were analyzed using voxel-based lesion symptom mapping (VLSM) with magnetic resonance imaging images.

Results
The present study analyzed 45 patients (mean age: 57.2 ± 12.6 years; 22 women and 23 men). Of these patients, 19 had left hemiplegia and 26 right hemiplegia, and the mean lesion volume was 60587.13 ± 72617.56 voxels. Spasticity scores significantly increased between the initial assessment and 3 months but did not differ between 3 and 6 months after onset ($p < 0.05$). The interaction between spasticity of the upper and lower limbs with time was not significant ($p = 0.373$).
An overlay of the lesions of all subjects is presented in Figure 1. The VLSM method with NPM revealed that lesions of the superior corona radiata, internal capsule posterior limb, posterior corona radiata, thalamus, putamen, premotor cortex, and insula were associated with spasticity in the upper limbs (Fig. 2), whereas lesions of the superior corona radiata, internal capsule posterior limb, caudate nucleus, posterior corona radiate, thalamus, putamen, and external capsule were associated with spasticity in the lower limbs (Fig. 3).

Conclusion
The involvement of white matter tracts and the striatum influences the development of spasticity in the upper and lower limbs of patients with stroke. These results may be useful for planning rehabilitation strategies and understanding the pathophysiology of spasticity in stroke patients.

Acknowledgment
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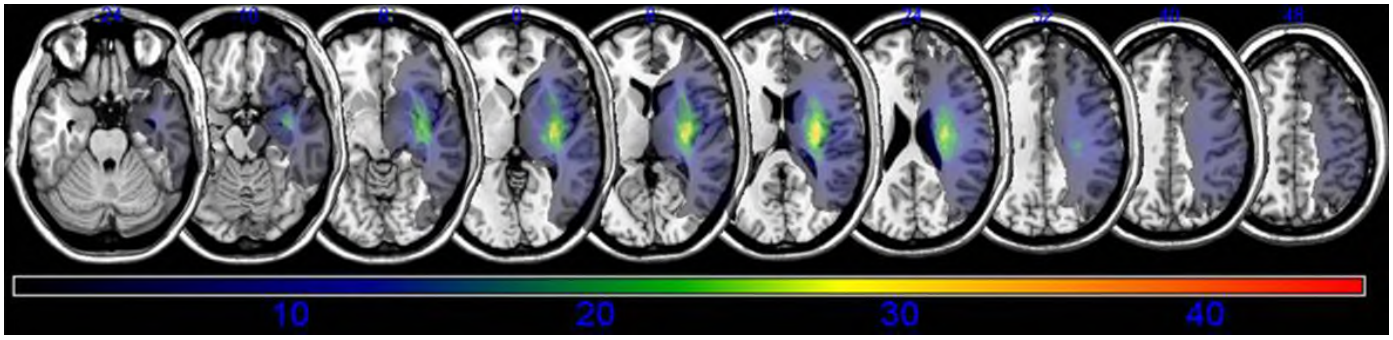

Figure 1. Overlay of lesions in all the subjects with stroke (n = 45). The color indicates the frequency of overlap.

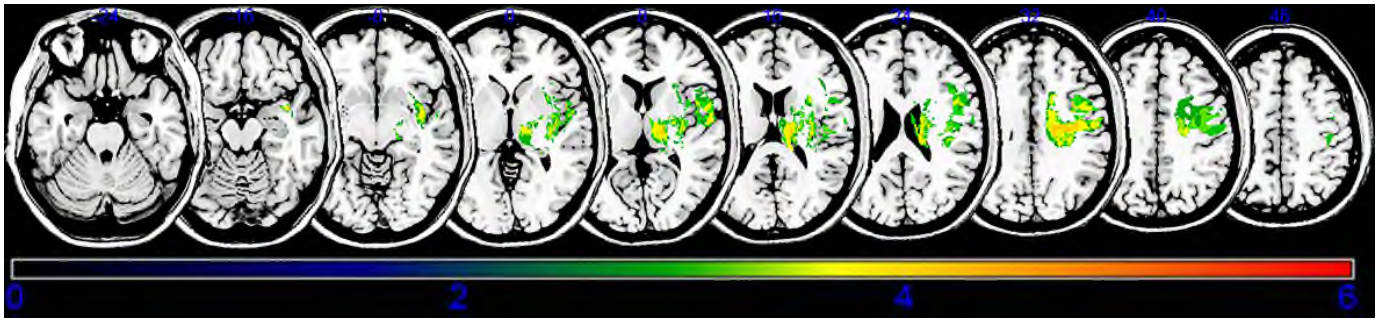

Figure 2. Statistical voxel-based lesion-symptom mapping for upper limb spasticity. The nonparametric Brubber Munzel statistical analysis was used for the continuous severe poststroke upper limb spasticity. Color scale indicates Brunner–Munzel rank order z-statistics. Only voxels significant at P<0.05 are shown. Colored bar represents the z statistics. The statistical map is displaying voxels with a minimum Z score of 2.4083. This matches the false discovery rate threshold. We set the maximum range of the Z score as 6, which be shown as being the maximum brightness.

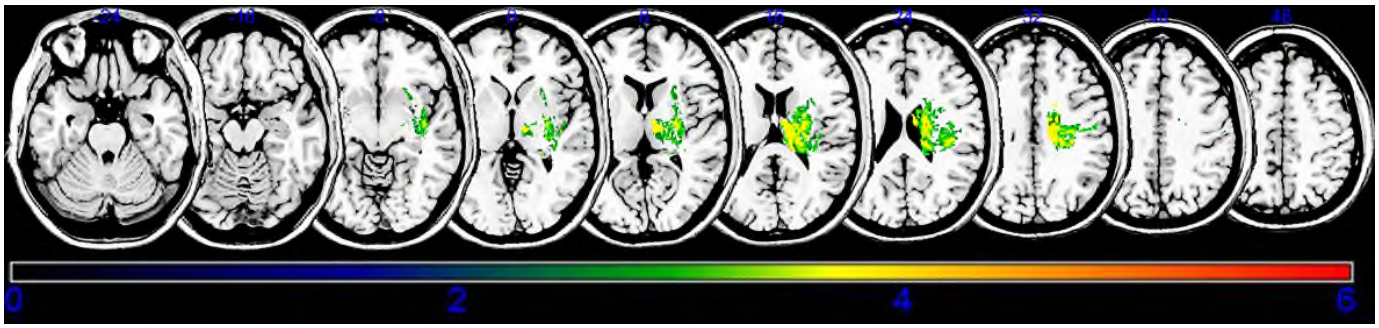

Figure 3. Statistical voxel-based lesion-symptom mapping for lower limb spasticity. The nonparametric Brubber Munzel statistical analysis was used for the continuous severe poststroke lower limb spasticity. Color scale indicates Brunner–Munzel rank order z-statistics. Only voxels significant at P<0.05 are shown. Colored bar represents the z statistics. The statistical map is displaying voxels with a minimum Z score of 2.5742. This matches the false discovery rate threshold. We set the maximum range of the Z score as 6, which be shown as being the maximum brightness.