



Letter to the Editor

## Left atrial fibrosis after ischemic stroke: How the insular cortex-ganglionated plexi axis interacts?

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We read the interesting study by Balint et al. [1], who reported that selective left or right insular cortex (IC) ischemic stroke in rats resulted specifically in left atrium (LA)-pulmonary vein (PV) border fibrosis, the pathological hallmark of atrial fibrillation (Af). LA-PV border fibrosis was suggested to be originated in local inflammation triggered by pre-ganglionic fibers ending in ganglionated plexi (GP) [1].

Cardiovascular system is regulated by a central autonomic network consisting of IC, anterior cingulate gyrus and amygdala [2,3]. Oppenheimer et al. [4] identified a site of cardiac representation within the human IC. Bradycardia/depressor effect was common after left posterior IC stimulation, while tachycardia/pressor effect was common after right anterior IC stimulation [4]. Thus, left posterior IC was associated with vagal tone, while right anterior IC was associated with sympathetic tone. Vingerhoets et al. found that patients with left posterior IC

ischemia had a higher risk of new-onset Af [5], suggesting that sympathetic-mediated Af was observed after the ischemic stroke. In the study reported by Balint et al. [1], there were no clear descriptions about the impact of distinct patterns of damaged area according to anterior/posterior part of each side of IC on the post-stroke LA-PV border fibrosis.

Until now, there have been few basic reports assessing the IC damage and LA-PV border fibrosis after ischemic stroke according to hemispheric laterality and anterior versus posterior portion of IC. The data presented in the manuscript by Balint et al. [1] would make much more importance if the exact mechanism in relation to the IC-GP axis was provided.

### Declaration of Competing Interest

The authors declare no conflict of interest.

### References

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