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Letter to the editor

Lateralization of the control of cardiovascular autonomic function and left atrial injury after selective right and left insular stroke☆

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We thank Dr. Nagai [1] for their interest in our paper showing that selective insular stroke induced by the stereotactic injection of endothelin-1 in the left and right insular cortex of Wistar rats resulted in increased endothelial dysfunction, inflammation, and fibrosis in the left atrium at 28 days after stroke [2]. In our study, we did not specifically investigate differences between anterior vs. posterior regions on the right or left insula. However, we did not find differences in left atrial changes when comparing left vs. right insular cortex strokes [2].

Characterizing the role of the right vs. left insular cortex on autonomic regulation is complex. Studies evaluating the lateralization of autonomic function in the insula in humans and rats have shown conflicting results [3]. For example, the left posterior insular cortex was associated with parasympathetic tone in rats [3] but sympathetic tone in humans [4]. The safest conclusion is that, to date, the lateralization of cardiovascular autonomic function in the insular cortex remains largely unknown, both in humans and rodents. Standardization of autonomic testing in clinical and preclinical research is crucial for narrowing this gap.

Our study constitutes a proof of concept approach demonstrating that damage to the insular cortex results in left atrial injury. Investigating the pathophysiological link between ischemic damage to the insula and left atrial changes induced at the level of ganglionated plexi was beyond its scope but is currently being investigated in our laboratory.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

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☆ In reply to the letter to the editor "Left atrial fibrosis after ischemic stroke: How the insular cortex-ganglionated plexi axis interacts?" (IJC_2019_2786).

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