to-point representation of each finger to neurons located in the motor cortex in a way that the radial finger area is located laterally and the ulnar finger area medially; recent authors advocate overlapping representation or group activation theories. Isolated weakness of a single finger secondary to a motor cortical lesion observed in our patient seems to agree more with the former theory. Unexpectedly, the MRI lesion was located slightly medial to the center of the hand knob (see the figure), which does not agree with a previous report that predominant weakness of radial-sided fingers is usually caused by laterally located infarcts whereas weakness of ulnar fingers is related to medial lesions in the precentral knob. Perhaps, there may be considerable individual variation of motor topography, which also was observed in the previous report.

Regarding the pathogenesis of stroke, our patient had atrial fibrillation, mobile aortic atheroma, and normal MR angiogram findings except for the posterior cerebral artery occlusion. Moreover, two infarcts developed in succession in different vascular territories. Therefore, an embolism is a likely pathogenic mechanism. This seems to be consistent with the previous observation that paresis of the radial-sided fingers was more often associated with embolic mechanisms as compared with ulnar-sided finger weakness.

References