Response to Letter by Tsuda

Response:

We thank Dr Tsuda for his insightful comments regarding our recent report on cardiac dysfunction after focal ischemia. Our data showed that left focal cerebral ischemia can lead to global heart dysfunction and that this myocardial dysfunction is related to the extent of left insular cortex damage. Moreover, we showed that plasma and myocardial levels of norepinephrine (NE) were raised in mice with heart dysfunction suggesting that excess NE may mediate cardiac functional impairment.1 We agree with Dr Tsuda that the underlying mechanisms that result in excess NE levels after focal ischemia involving the insular cortex should be investigated further. Dr Tsuda rightly points out that data exist suggesting that bradykinins may be involved in the pathophysiology of cerebral ischemia and may be involved in elevating NE levels.2–7 It is entirely possible that ischemia involving the left insular cortex might trigger elevations in bradykinin levels that in turn may increase NE release, which may in turn lead to cardiac dysfunction. Studies to test this hypothesis are ongoing.

Disclosures

None.

Jiangyong Min, MD
Arshad Majid, MD
Division of Cerebral Vascular Diseases
Sparrow Health System and
Department of Neurology and Ophthalmology
Michigan State University
East Lansing, Mich

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Jiangyong Min and Arshad Majid

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