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The Authors reply: Comment on: "Experimental ischaemic stroke induces transient cardiac atrophy" by Veltkamp et al.

We would like to thank Scherbakov and Doehner for their insightful letter[1] regarding our manuscript.[2]

The impact of brain injury on the heart has been the subject of many case series and clinical studies which includes particular syndromes such as Takutsubo cardiomyopathy and the broken heart syndrome. Our study[2] examined the effects of large or small brain infarcts on cardiac function and structure in mice using in vivo microechocardiography as well as post-mortem studies. Using this reverse translational, bedside to bench approach, we found transient cardiac injury, cardiac dysfunction, and transient structural alterations in young mice after extensive brain infarction. Some of our observations resemble features that have been described in stroke patients.[3] Moreover, we identified potential signalling pathways between the brain and the heart that may also contribute to injury in individual patients and be amenable to personalized therapeutic intervention in the future.

We share Sherbakov's and Doehner's opinion that carefully conducted, prospective clinical studies are needed to better characterize the brain–heart relationship after stroke in patients. So far, the focus of research has been on the heart as a potential source of embolism to the brain. Atrial fibrillation is a major risk factor for stroke. Because prevention of embolism with anticoagulants is a particularly effective strategy for stroke prevention, extensive efforts have been made to detect paroxysmal AF after stroke and TIA. Although the majority of stroke patients with AF has pre-existing heart disease,[4] there is some evidence that AF first diagnosed after a stroke may be the consequence rather than the cause of ischemic infarction. This example underlines the complexity of the clinical setting because stroke patients—in contrast to mice—often have comorbidities including coronary and other structural heart disease. Similar to our findings in mice, about 20% of acute stroke patients in the TRELAS study[5] had an elevated high sensitivity troponin as evidence of cardiac injury. This may result from acute coronary heart disease or represent a response of cardiomyocytes to massive autonomic dysregulation secondary to brain injury. Ongoing multidisciplinary projects such as the PRAISE study will shed more light on the complex interaction between the brain and the heart.

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