Response to Letter by Metso et al

Response:
We thank Metso et al for their interesting comments, which we will address in this letter. Metso et al first remarked that there was no effect of complete recanalization of narrowed or occluded cervical internal arteries in patients with spontaneous internal carotid artery dissection (SICAD) on the clinical outcome.1 Randomized, controlled trials in patients with acute ischemic stroke have shown that the effect of thrombolysis, and thus of recanalization, declines rapidly over the first 4.5 hours of symptom onset.2 Recanalization that occurs later seems to be beneficial for some patients with persisting perfusion/diffusion mismatch.3 We have shown that recanalization of the cervical internal carotid artery usually occurs within days, weeks, or even months after the onset of SICAD symptoms.4 It is thus not surprising that we found no effect of recanalization on the functional outcome of our patients with ischemic stroke. Metso et al interpreted our findings shown in Table 3 as typographic “errors.”1 This “error” changes into a misunderstanding after the lecture of the title of this table, because it states that the table reports the findings per SICAD, and one SICAD can lead to more than one ischemic symptom, for example, amaurosis fugax followed by an ischemic stroke, which may be accompanied by local symptoms and signs. Metso et al wondered about the higher rate of complete recanalization in the 79 patients who presented with local symptoms and signs. We have shown that SICAD causing carotid territory ischemia (ischemic group) had more often stenoses or occlusions of the cervical internal carotid artery compared with SICAD that caused no ischemia but either led to local symptoms and signs only or remained asymptomatic (nonischemic group).4 Furthermore, SICAD causing no ischemia was more often associated with Horner syndrome and/or palsies of the caudal cranial nerves on the side of the dissection.4 One explanation of these findings was that SICAD in the ischemic group is located between the intima and the media, whereas SICAD in the nonischemic group is located between the media and the adventitia.5 However, pathological specimens from autopsy and surgical material of patients with SICAD causing severe carotid stenosis or occlusion and cerebral ischemia have shown that the dissection hematoma was located in the media, between the media and adventitia, or in the intima.6–8 Consequently, the plane of the dissection hematoma seems not to be essential for the development of internal carotid artery stenosis or occlusion.4 As a result, our group hypothesized that dissection hematomas in the ischemic group extended essentially toward the arterial lumen and cause more stenoses and occlusions, which would lead to more ischemic events in the brain or retina.9 On the other hand, hematomas in the nonischemic group would extend more often outward and compress more often the surrounding sympathetic and/or caudal cranial nerves, which could explain the higher frequency of local symptoms and signs.4 We agree with Metso et al that epidemiological and therapeutic studies often need a large number of patients and that Cervical Artery Dissection in Ischemic Stroke Patients (CADISP) has great potential to bring light into the mechanism and treatment of SICAD.

None.

Disclosures

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