Dear Sir

I really appreciate the case report by Flury et al. [1] picking up the important problem of anticoagulation in a haemorrhagic affection such as carotid artery dissection.

However, the goal of this article is misleading and the whole point regarding heparin treatment in spontaneous internal carotid artery dissection (sICAD), unfortunately, is not taken. Diagnosing sICAD and assessing the degree of vessel stenosis is usually made by imaging (MRI or US) and represents a momentary picture. Fluctuating degree of stenosis with recurrent pseudoocclusion in the early phase following acute ICAD is a well-known phenomenon with, but also without anticoagulant therapy [2]. This is also to be expected regarding the dynamic nature of a mural haematoma (changes of composition, diffusion, etc.) and the degree of volume changes involved/necessary. A mural ICA haematoma of e.g. 4 cm longitudinal extension with a volume of only 0.3 ml may cause 50% stenosis regarding area reduction of normal ICA lumen (around 20 mm² with a diameter of 5 mm). An increase of the haematoma volume by only another 0.3 ml is necessary to cause pseudoocclusion. But, most important, whether heparin leads to ICA pseudoocclusion or not is irrelevant – local thrombosis and subsequent distal embolism have to be prevented, which is the cause in >98% of all strokes occurring in patients with sICAD [3]. What is the risk of a progressive mural haematoma? No one has ever reported an ICA rupture following anticoagulant treatment of a patient with sICAD. A hypothetically increased frequency of local symptoms such as pain, Horner’s syndrome or lower cranial nerve palsies has not been reported either. On the other hand, recurrent (unexpected) ICAD in patients already under anticoagulant treatment seems to have an especially benign prognosis [4]. Unless someone will prove that the recanalisation rates in dissected ICAs with pseudoocclusion (at any time point) are reduced there is no need to be afraid of mural haematoma progression. In the latter case, furthermore, reduced recanalisation rates would have to be weighed against the benefit from embolism protection provided by heparin therapy. To conclude, my learning point would rather be: Don’t worry about progression of mural haematoma and even of ICA stenosis in the early phase of sICAD under heparin treatment.

References