moved by cerebrospinal fluid circulation. Secondly, CAA is a clearly defined and separate disease entity due to severe amyloid deposits mainly in the leptomeningeal and cortical vessels, leading to repeated cerebrovascular lesions, bleedings as well as infarcts. Mild amyloid deposits in cerebral vessels are common in elderly patients with various neurodegenerative diseases and non-contributive to additional cerebrovascular lesions. In our series, only 5 of the 30 patients classified as non-CAA had mild amyloid deposits in some cortical arterial branches, not spatially related to the SS. Thirdly, CAA is not exclusively a microangiopathy like lipohyalinosis, but it also involves large leptomeningeal arteries, capillaries and even veins. Fourthly, in the cases of SS preceding the occurrence of a lobar haematoma, it cannot be demonstrated or excluded with the routinely used ‘in vivo’ MRI machines that the SS is due to a cortical microinfarct which, with time, undergoes a haemorrhagic transformation with a lobar haematoma as a late complication. In any case, this controversy stimulates a discussion about the significance of SS in CAA and will hopefully also promote further neuropathological and MRI studies on this fascinating topic.

I first wish to thank the commentators for their pertinent remarks and the great interest in this topic. I completely agree that our series could be selective and not overall representative, although our centre has a particular interest in cerebral amyloid angiopathy (CAA). Our divergent opinions on some matters are mainly due to differences in definition. Firstly, the neuropathological substrate of (cortical) superficial siderosis (SS) is non-removable haemosiderin deposition in the subpial space, and not in the subarachnoid space where blood residues can easily be removed by cerebrospinal fluid circulation.