LETTER TO THE EDITOR

Reply: Hyperglycaemia and the outcome of stroke

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Sir, we thank Dr Metso and Dr Murros for their interest and valuable comments on our study. Our study explored the effect of blood glucose levels in two different types of ischaemic stroke, and was not intended to guide acute stroke therapy. Whatever the underlying mechanism (stress or other causes) there is substantial evidence that hyperglycaemia in acute non-lacunar ischaemic stroke is associated with a worse clinical outcome. By using magnetic resonance imaging and spectroscopy, Parsons and colleagues nicely showed that hyperglycaemia in acute non-lacunar stroke leads to increased lactate production in the penumbra and adversely affects clinical outcome (Parsons et al., 2002). These data are in agreement with several observations made in animal models of stroke (Anderson et al., 1999; Li et al., 2000; Lin et al., 1998; Prado et al., 1988). We demonstrated that the effect of hyperglycaemia is different in patients with lacunar ischaemic stroke.

In the GIST-UK trial (Gray et al., 2007), both non-lacunar and lacunar strokes (representing approximately one in five of the included patients) were treated in the same manner. Whether this affected study results is unlikely, because the investigators performed a post-hoc analysis on stroke subtypes. However, a beneficial effect in non-lacunar stroke may have been missed because the median onset to treatment time was approximately 13 h, and only 8 of the 933 patients were treated within 3 h. Thus, for most patients, this late start of treatment may have been insufficient to prevent hyperglycaemia-induced irreversible damage in the penumbra. We propose that ongoing and future trials aimed at controlling glucose levels in acute ischaemic stroke should differentiate between stroke subtypes and start sooner after symptom onset.

References