Atrial fibrillation (AF) is a major risk factor for the development of an ischaemic stroke. It has been demonstrated that oral anticoagulation with vitamin K antagonists (VKAs), such as warfarin, is highly effective in preventing thromboembolic events in patients with AF. However, the administration of VKAs is accompanied by haemorrhagic complications and other unfavourable limitations (1). Recently, new oral anticoagulants directly targeting coagulation factors have been introduced and attracted significant interest as they have a better benefit/risk profile than VKAs for stroke prevention. In a large clinical trial, the new oral anticoagulant and direct thrombin inhibitor, Dabigatran etexilate (DE), was revealed to effectively prevent stroke in patients suffering from AF with less intracranial bleeding complications as compared to the VKA, warfarin (2). However, several questions have yet to be resolved. For example, it is still disputed how anticoagulated, acute stroke patients should be treated regarding the use of a thrombolytic therapy (3).

In this issue, Sun et al. have experimentally addressed this interesting point by using a series of various rodent models of cerebral ischaemia, in which mice received either no anticoagulant, warfarin or DE before induction of ischaemic stroke (4). Subsequently, thrombolysis was performed with the recombinant tissue plasminogen activator and mice were analysed for secondary intracerebral haemorrhage after 24 hours. The authors conclusively show that anticoagulation with DE, in contrast to warfarin, did not increase the rate of secondary intracerebral haemorrhage after thrombolysis in experimentally-induced cerebral ischaemia models. Furthermore, the reduced bleeding complications after thrombolysis observed in mice treated with DE as compared to warfarin, was ascribed to a less severe leakage in the blood-brain barrier as determined by Evans blue extravasation. The authors also provide a potential mechanism by showing that increased activation and proteolytic activity of matrix metalloproteinase 9 might mediate the disruption of the blood-brain barrier in warfarin-treated mice (4).

Together, the observations obtained in this experimental study could provide useful information for the management of anticoagulated, acute stroke patients but still have to be confirmed in clinical studies.

Conflicts of interest
None declared.

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References