

Comment on:

Schipke JD, Halkos ME, Kerendi F, Gams E, Vinten-Johansen J. Postconditioning: a brief review. Arch Med Sci 2006; 2:137-45.

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Dear Editor,

We read with great interest the manuscript of Schipke et al. [1] about postconditioning. This review was published at the same time that it was found for the first time that such postconditioning – with a series of mechanical interruptions of reperfusion – also significantly reduces ischaemic damage in the brain [2].

Reperfusion damage is a complex process involving several cell types, soluble proinflammatory mediators, oxidants, ionic and metabolic dyshomeostasis, and cellular and molecular signals. Novel neuroprotective strategies are required to target this form of injury [3]. The neuroprotective potential of ischaemic preconditioning has not been realized in clinical practice because it necessitates intervention applied before the onset of ischaemic stroke, which is difficult to predict. A more amenable approach to neuroprotection is to intervene at the onset of reperfusion, the timing of which is under the control of the operator. In this regard, these new findings of postconditioning in the brain may open a window to improve stroke treatment or prevention [4]. In contrast to preconditioning, which requires a foreknowledge of the ischaemic event, postconditioning can be applied at the onset of reperfusion at the point of clinical service. Interestingly, experimental studies suggest that ischaemic preconditioning and postconditioning activate the same signalling pathway at the time of reperfusion, thereby offering a common target for neuroprotection [2]. Therefore, the pharmacologic recruitment of this signalling pathway at the time of cerebral reperfusion might allow one to harness the neuroprotective potential of ischaemic preconditioning and postconditioning and therefore substantially improve the outcome in EC-IC bypass and neurovascular surgery.

Further research is needed to find new pharmacological agents that would mimic postconditioning in order to treat all patients with ongoing acute ischaemic stroke. Of course, the potential of postconditioning must be rigorously tested in clinical trials, first for its safety and feasibility and then subsequently for its efficacy and therapeutic potential. It will be mandatory in such clinical trials to carefully control for confounding variables such as the size of the area at risk, the duration of the preceding ischaemic insult, and collateral status. Neglect of these confounding variables has probably contributed to the failure to translate experimentally validated principles of neuroprotection to the clinical arena (e.g. adenosine receptor activation) in the past.

References

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4. Schaller B, Graf R, Jacobs AH. Ischaemic tolerance a window to endogenous neuroprotection? Lancet 2003; 362: 1007-8.

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