

Letter to the Editor

The role of early ischemic preconditioning in spinal cord protection after transient aortic occlusion

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I read with great interest the article titled 'Ischemic preconditioning and nicotinamide in spinal cord protection in an experimental model of transient aortic occlusion' [1]. I congratulate Isbir and associates on their study of ischemic preconditioning (IPC) and nicotinamide utilization to provide spinal cord protection.

IPC is a biphasic phenomenon with an early and a late phase of protection. These two phases have been documented in the spinal cord [2–4]. In this study Isbir et al. evaluated the effect of either early IPC (group 2) or nicotinamide (group 3) or combined IPC and nicotinamide (group 4) on spinal cord protection in a rat model. In this model, rats underwent 5 min IPC, followed by 45 min of reperfusion before a 45 min duration of infrarenal aortic cross-clamping. They found that early IPC or combined IPC and nicotinamide reduced spinal cord injury at 48 h when compared with the controls (mean Tarlov score: 2.75 for early IPC, 3.13 for combined IPC and nicotinamide vs. 0.88 in the control group; $P = 0.004$).

In a recent published study, our group demonstrated that early IPC without hypotension prevents spinal cord injury in a porcine model of descending thoracic aortic occlusion [2]. We used 20 min IPC, 80 min of reperfusion and the duration of descending thoracic aortic occlusion was 35 min. In our study, it was very important to maintain the arterial systolic blood pressure higher than 100 mmHg during the 80 min reperfusion interval. Two animals had an arterial systolic blood pressure of 80–90 mmHg during the reperfusion period. Although they had a Tarlov score of 4 at 24 h postoperatively, these two animals became paraplegic at 48 h.

In another study by Caparrelli et al. [5], in a rabbit model (5 min brief ischemia, 30 min of reperfusion and 20 min of infrarenal aortic occlusion), when six animals with early IPC were compared with seven controls, IPC failed to protect the spinal cord at either 24 or 48 h. In this study it is shown that there was a level of hypotension during the reperfusion interval in the IPC group, and this hypotension may be an explanation for the failure of early IPC to protect the spinal cord.

In most studies of spinal cord injury after aortic occlusion there is an aggravation in neurologic scores from 24 to 48 h [2–5]. In the study of Isbir et al. there is an improvement in paraplegia from 24 to 48 h (groups 2 and 3 from 25% to 12% and group 4 from 12% to 0%). How do the authors explain this improvement? Also, it is mentioned in the published manuscript that the arterial blood pressure and heartbeat were continuously monitored throughout the procedure. It would be of great interest if the authors could provide the proximal to the aortic occlusion arterial blood pressure.

References

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