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Reply to the Letter to the Editor

Reply to Toumpoulis

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We thank Dr Toumpoulis for his nice comments regarding our article entitled 'Ischemic preconditioning and nicotinamide in spinal cord protection in an experimental model of transient aortic occlusion'. He highlights the important point that keeping the blood pressure above 100 mmHg reduces the postoperative neurological dysfunction during the aortic occlusion [1]. We concur completely that hypotension should be avoided during aortic occlusion. This might reduce the collateral circulation. In our model, the tail artery was cannulated for distal aortic pressure. Blood pressure dropped from 80–90 to 10–20 mmHg after cross-clamping. However, our aim was to confirm that cross-clamping was complete. Heartbeats were around 180–190. We did not find any correlation with blood pressure and neurological scoring. However, we discarded four animals that had hypotension due to hemorrhage.

With regard to the comments for improvement in paraplegia from 24 to 48 h, as stated in our article we think that apoptosis might be the underlying mechanism. The necrotic cells in light microscopy might be apoptotic and terminal damage might not occur. We measured the apoptotic cells in 48 h. Another study comparing the histological changes and the number of apoptotic cells at 24 and 48 h of the experiment should be undertaken to explain this finding. However, we did not focus on the mechanism for reduced apoptotic cell death by ischemic preconditioning (IPC) in our study. Recent studies have shown the importance of inhibitors of apoptosis (IAP) following spinal cord injury [2].

Another point that has been raised is the duration of IPC. This has different time scales in various studies [3,4]. We believe that at least 5 min of ischemia should be present for the protective effect of IPC.

In conclusion, IPC is a promising method in spinal protection and further studies are needed to explain the underlying mechanism.

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