tion of the Internal Thoracic Artery," which appeared in the April 2003 issue of the *Journal*.¹ This report resurrects a technique I described years ago in an article titled "Carbodissection of the Internal Thoracic Artery Pedicle," which appeared in *The Annals of Thoracic Surgery*.² I was pleased that Dr Özkan used this technique with the same success I have observed since 1988 but was disappointed that, perhaps through an oversight, Dr Özkan chose not to reference my work. The important issue is not who performed the procedure first but that Dr Özkan's observation validates the efficacy of the technique.

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Reply to the Editor:

We thank Dr Lee for responding to our article and for his comments.

Carbon dioxide insufflation, as a method of preparation of the internal thoracic artery, is not novel. Dr Lee first introduced this technique in 1988.¹ Later, Bognolo and associates² used the same technique in 1995. We also have used the carbon dioxide insufflation technique since 2002 without citing Drs Lee and Bognolo.³ After this omission was noticed, we referred to Drs Lee and Bognolo in our next article,⁴ titled "Effect of Carbon Dioxide Insufflation on Free Internal Thoracic Artery Flows: Is it a Vasodilator?" which was published in the *Journal*. We congratulate Dr Lee for introducing this technique to heart surgery.

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Early ischemic preconditioning provides transient protection To the Editor:

We read with great interest the study by Toumpoulis and colleagues,¹ "Superiority of Early Relative to Late Ischemic Preconditioning in Spinal Cord Protection After Descending Thoracic Aortic Occlusion" published in the November 2004 issue of the *Journal*. In their experimental model, they revealed the superiority of early ischemic preconditioning (IP) in reducing spinal cord injury caused by thoracic aortic occlusion. IP is a new concept against spinal cord ischemic injury. Although previous reports have demonstrated its beneficial effects, there are still controversial issues including the fundamental mechanisms by which it provides protection and the duration of reperfusion between 2 ischemic insults.

Recent experiments revealed a delayed protective effect of IP, termed the "second window of protection," which appears more than 24 hours after the initial ischemic insult. A subgroup of protein family, called "stress proteins," which are crucial for the maintenance of cell integrity under unfavorable conditions, are accepted as an important cause of the "second window" provided by IP. These stress proteins, in particular, heat shock proteins (HSPs), are able to alter the resistance of the tissues to subsequent ischemic and nonischemic insults.^{2,3}

We have also examined this known strategy, but our experience differs from that of Toumpoulis and colleagues. We used a rat model of spinal cord ischemia and revealed that hyperthermic IP (HIP) before transient aortic occlusion resulted in improved neurologic and histopathologic outcomes. In the IP group, we used an early IP model with a reperfusion interval of 30 minutes between 2 ischemic

insults. In the HIP group, rats were heated to 41°C and maintained at this temperature for 15 minutes. Twenty-four hours later, the described early IP model was also applied to this group of rats. The spinal cord was extracted, and the lumbosacral region was examined under light microscopy to assess necrosis and under electron microscopy to determine HSP-ubiquitin positivity.

The neurologic evaluation of rats performed on the first day did not reveal a statistically significant difference between the IP and HIP groups. However, on the second day, we noticed a delayed neurologic deterioration in the early IP group. The neurologic scores of the HIP group were significantly higher than those of the IP group at the end of 48 hours ($P < .05$). Histologic evaluation correlated well with the neurologic outcome with lesser cellular damage in the HIP group. Ubiquitin positivity was present only in hyperthermia-pretreated animals.

On the basis of our experience, we believe that a model with a short reperfusion interval does not provide the delayed anti-ischemic effect of IP, termed the "second window," which is possibly related to the expression HSPs. Our results suggest that HSP-ubiquitin induction by heat stress may be responsible for the delayed spinal cord protection seen in this model. Whole-body hyperthermia may have important clinical implications, and further studies will delineate the fundamental mechanisms of hyperthermia-induced neuroprotection.

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