the selective adenosine A2 receptor agonist CGS 21680 on in vitro electrophysiology, cAMP formation and dopamine release in rat hippocampus and striatum. J Pharmacol Exp Ther 1990; 252: 1134-41.


Reply to the Editor:

It is somewhat surprising and quite unusual to receive a letter about an article that we published in the Journal more than 3 years ago. The fast-paced developments in neuroscience make it somewhat difficult to put the findings of an older study in perspective. In our article titled "Profound Systemic Hypothermia Inhibits the Release of Neurotransmitter Amino Acids in Spinal Cord Ischemia" (J Thorac Cardiovasc Surg 1995;110:27-35), we simply described the findings of a study designed to clarify the role of excitotoxicity in spinal cord ischemia and to provide a possible mechanism for the protective effect of hypothermia. For the first time in the spinal cord, amino acid levels were measured under conditions simulating the clinical situation, and several of the findings broke new ground in this field. More recent studies put those findings in a proper perspective, describing mechanisms that quite simply were unknown at the time of publication of our article.

Our paper is criticized for not discussing references that appeared in the literature after the paper was published. The authors of the letter also claim that they re-analyzed statistically our data and reached different results, indirectly implying that our statistical methods were flawed. We wonder, however, how they were able to perform statistical analysis without having the raw data. In the data table, we provided mean values and standard deviations for the various experimental groups. The statistical analysis was performed by taking into consideration the results from individual experiments. Any analysis that compares only mean values of different groups is simply not accurate. We also wish to point out that sham animals served for validation of methodology only, without being the samples constituting an experimental group. It is a commonly accepted practice to use baseline values as control, so long as they do not differ statistically among the experimental groups.

The field of ischemia and neuronal cell death has expanded the horizons of neuroscience tremendously in recent years. We believe our article provided some insights into the pathophysiology of spinal cord ischemia.

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Left subclavian artery as a site of proximal aortic perfusion for hypothermic repair of thoracic and thoracoabdominal aneurysms

To the Editor:

Retrograde aortic perfusion through the femoral artery with deep hypothermic circulatory arrest is a valuable adjunct for thoracic and thoracoabdominal aneurysms. However, retrograde perfusion of the brain through an atheromatous or dissected aorta carries the risk of cerebral embolism or malperfusion. To avoid these fatal complications, Westaby and Katsumata proposed a proximal aortic perfusion through the ascending aorta or aortic arch via an extended left thoracotomy. However, the possibility exists that cerebral emboli may be produced even by external manipulation of an atheromatous aorta or aneurysm. Hence we have recently adopted a proximal aortic perfusion technique using a prosthetic graft attached to the left subclavian artery via a left posterolateral thoracotomy for hypothermic thoracic and thoraco-abdominal aneurysm repair (Fig 1). The left subclavian artery with easy access from a thoracotomy has been used for a systemic-pulmonary arterial shunt using a prosthetic graft in neonates or small infants. Moreover, the axillary artery is also widely known as an alternative site for proximal arterial

![Fig 1. Left subclavian artery (LSA) cannulation technique through a posterolateral thoracotomy. After proximal and distal control of the LSA is obtained, an 8-mm sealed woven Dacron graft is attached to the LSA, followed by cannulation with a polyvinyl arch cannula. Venous drainage is through a straight venous cannula placed in the main pulmonary artery (PA).](image-url)
perfusion during cardiopulmonary bypass in patients with severe atherosclerotic or aneurysmal disease. From our experience, we believe cannulation of the left subclavian artery through a prosthetic graft to be a safe, easy, and effective way of arterial cannulation for cardiopulmonary bypass in patients undergoing hypothermic repair of thoracic or thoracoabdominal aneurysms. In addition, a prosthetic graft used for arterial perfusion is also available for later reconstruction of the left subclavian artery system when necessary.

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REFERENCES

The sympathetic denervation induced by transmyocardial laser revascularization protects the affected tissue

To the Editor:

Having a strong interest in the mechanism of transmyocardial laser revascularization (TLR), I appreciated the article by Kwong and associates about the sympathetic denervation of canine myocardium induced by TLR. The authors believe that such denervation contributes to the angina relief observed in clinical situations. Their results (the absence of tyrosine hydroxylase in the denervated myocardium) also suggest, however, that the denervation blocks not only afferent nociceptive stimuli but also possible efferent sympathetic overstimulation. This hypothesis is supported by the following facts: (1) efferent cardiac sympathetic tone and circulating catecholamines are elevated in advanced heart failure; (2) increased efferent cardiac sympathetic tone and circulating catecholamines induce arrhythmias, dysfunctional myocardial contractions, contraction band necrosis, and infarct-like myocardial lesions; and (3) these functional disturbances and lesions are often present in end-stage heart disease. In this situation, the sympathetic denervation induced by TLR, being able to abrogate injurious increased intramyocardial catecholamine release, provides a partial protection for the denervated myocardium.

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REFERENCES

Cytokines and myocardial injury: Is there a scope for magic bullets?

To the Editor:

In recent issues of the Journal, two studies suggested that cytokines such as interleukin-6 (IL-6) and IL-8 play a pivotal role in inducing myocardial ischemia-reperfusion injury. Inasmuch as both cytokines are released during and after cardiopulmonary bypass (CPB), the question was raised as to