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Reply to the Editor:

I appreciate Murphy and colleagues' letter to the editor,¹ discussing the differences in our robotic surgical methods.^{2,3} To be sure, both his group and ours have attained excellent results with robotic mitral valve repairs. He is correct in that we use different sites for working-port and camera incisions, and this difference definitely facilitates hand-tying of intracardiac sutures by the patient-side surgeon. Nevertheless, we are traveling toward a totally endoscopic mitral operation, and the necessity for patient-side intracardiac assistance to perform the operation requires 2 experienced surgeons. Thus, if we are to become asymptotic to "real endoscopic cardiac mitral surgery," experienced surgeons, such as Dr Murphy, must strive to achieve repairs using all robotic techniques, which would lessen the need for an experienced patient-side surgeon. Are these points moot or are they important? When good surgeons disagree, each becomes more circumspect and these dialogs help us advance our specialty—"our destiny." To this end, I appreciate and respect both Dr Murphy and colleagues' letter and opinions.

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doi:10.1016/j.jtcvs.2006.11.046

Which cell is transferred hepatocyte growth factor gene?

To the Editor:

I read with interest the article by Shi and associates,¹ titled "Nonviral Gene Transfer of Hepatocyte Growth Factor Attenuates Neurologic Injury After Spinal Cord Ischemia in Rabbits." It is already known that hepatocyte growth factor (HGF) functions as a powerful angiogenic factor, as well as a potent neurotrophic factor,² and HGF is not expressed in the spinal cord in the early phase after the ischemia.³ Thus I agree that gene transfer of HGF could induce tolerance against a severe spinal cord ischemic insult. Furthermore, intrathecal injection of HGF gene is less invasive than viral injection to the spinal cord. However, I have one question. The authors have not demonstrated that HGF was induced in any cells. Johnson and associates⁴ demonstrated that flunarizine has a protective effect on neurologic recovery after spinal cord ischemia, and the mechanism of protection involves inhibition of calcium accumulation rather than a direct effect on vascular smooth muscle using spinal cord blood flow measurements. Furthermore, Hayashi and associates⁵ demonstrated that prevention of delayed CA1 neuronal death after cerebral ischemia in the gerbil by subarachnoid HGF gene transfer is due to the inhibition of apoptosis through the blockade of Bax translocation from the cytoplasm to the nucleus. These results suggest that HGF gene into intrathecal injection may have a protective effect against neuronal apoptotic cascade rather than angiogenic effect. Therefore, to elucidate the mechanism of neuronal protection, the authors should demonstrate temporal profiles of HGF in histochemical study or in situ hybridization study.

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Reply to the Editor:

My colleagues and I thank Dr Sakurai for his insightful comments on our recently published article. Tolerance induced by gene transfer of hepatocyte growth factor (HGF) against ischemia insults has been well described in the literature, including our study,^{1,2} in which the question about which cell was the target for gene transfer and then expressed with HGF was eclipsed by the emphasis on the assessment of neuroprotective effects and its possible mechanisms. We appreciate that the question raised by Dr Sakurai is very important. Human HGF was detected in the pia matter and parenchyma when HGF was transferred by intracisternal injection using the same vector. It seemed that multiple cells might secrete HGF, including neurons.³ Of course, only further study is the best answer. Spinal cord ischemia can induce two kinds of motor neuron death: necrosis and apoptosis. It is thought that the delayed and selective motor neuron death, which occurs a few days after a relatively short period of spinal cord ischemia (less than 20 minutes in the rabbit model) accompanied by delayed paraplegia, is due to neuronal apoptosis, whereas necrosis usually occurs after acute ischemia.⁴ In the current study, rabbits were subjected to a 30-minute period of ischemia, and most animals were completely paraplegic 1 day after transient ischemia. The previous work of our department showed that a 30-minute period of ischemia induced almost a total loss of motor neurons 2 days after reperfusion.⁵ Therefore, the motor neuron damage in the current study was mediated mainly through

necrosis, but not apoptosis. We agree that HGF might induce neuroprotective effects through inhibition of apoptotic cascade. However, it was highly impossible that the observed neuroprotection of HGF in our study is due to its antiapoptotic effects.

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Apical cannulation for aortic perfusion

To the Editor:

I read with interest the article, "Transapical aortic cannulation for cardiopulmonary bypass in type A aortic dissection operations," by Wada and associates¹ in the November 2006 issue of the *Journal*. I agree with the authors that this time-tested method is indeed most suitable and safe in the management of type A aortic dissections. However, its applicability may be further enhanced by two additional maneuvers: The first technique is use of a special double-lumen catheter; the larger-caliber channel should have an apical port for perfusion and the shorter, smaller-caliber channel should have a port located about 10 cm proximal to the tip and be positioned

Figure 1. Application of the special "double-padded" clamp and perfusion cannula. The jaws of the instrument may be brought into a position to fully occlude the clamped aorta but still allow the passage of the unobstructed flow through the intra-aortic cannula. The double-barreled cannula allows perfusion through the larger, longer channel tip, which is placed into the aorta, and venting through the shorter, smaller channel, which terminates within the left ventricle. (From Robicsek F. Apical aortic cannulation: Application of an old method with new paraphernalia. *Ann Thorac Surg*. 1991;51:330-32. Reproduced with permission from The Society of Thoracic Surgeons).

in the left ventricle for removal of blood, which may be seeping into the left side of the heart. This arrangement eliminates the need for a separate vent through the right pulmonary vein as applied by the authors. The second technique is application of a "special" aortic crossclamp, the padded jaws of which hermetically occlude the aortic lumen but allow passage of the perfusion catheter.² This arrangement provides added versatility to apical perfusion, making continuous or intermittent arch perfusion possible even with the proximal aorta open (Figure 1).

The issue of bleeding that the authors experienced while placing a purse-string

suture into the apex may be effectively addressed by introducing the perfusion catheter through a simple stab wound and placing a 3-pledgeted purse-string suture after the heart is cold and fully relaxed.

The authors' addition transesophageal echo control of the catheter placement adds additional safety to the method.

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Can the consensus statement on surgery journal authorship be implemented?

To the Editor:

The "Consensus statement on surgery journal authorship" is of great interest, not only for the surgical scientific community, but for all biomedical research areas. Most important journals have it in some form and all agree on the fundamentals. This indicates that the problem, which is addressed, is widespread. Some questions, however, have to be added.

How will this statement be implemented? Can one rely on the honesty of the coauthors? Is a simple statement of the authors for which part they are responsible sufficient?

The attitude in many universities "to publish or to perish" is a grim reality and forms a serious obstacle for honesty. It is an incentive for one to have his or her name on as many articles as possible, because career opportunities and promotions depend on it.

This is especially a problem in a hierarchy where heads of departments have to authorize for funds and materials, or supervise but without taking part in the research as such. Young researchers, who want to build a career, usually depend on their head