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Reply to the Editor:

We thank Miyamoto and Miyamoto for their interest in our recent article.[1] It is a fact that ketamine is a widely known *N*-methyl-D-aspartate receptor antagonist that has demonstrated neuroprotective properties in vitro and in vivo.[2] Therefore the observed neuroprotective effect in our study is the result of ketamine and riluzole effects. Ketamine has been widely used by most investigators working with the rabbit model of spinal cord ischemia, with minor spinal cord protection afforded by this drug.[2,3] In our study, all rabbits received the same dose of ketamine, with huge differences in neurologic recovery among experimental groups. Thus, although the potentiating effect of ketamine cannot be ruled out, it is unlikely that this drug by itself could account for the dramatic differences in neurologic recovery among experimental groups. For definitive assessment of the neuroprotective effects of riluzole, we have performed a new study with a spinal cord ischemia model in the rat.[4] All animals were anesthetized with halothane 1.5% only, and they received riluzole before aortic crossclamping and at the onset of reperfusion. Rats were allowed to recover for 24 (n = 15), 48 (n = 10), or 96 hours (n = 5). In this study, riluzole again prevented neuronal necrosis and apoptosis and cytoskeletal proteolysis.

Regarding neurologic recovery of rabbits, we did not observe worsening of neurologic status in riluzole-treated rabbits between 24 and 120 hours after the operation. Rabbits were scored at 6 hours, 24 hours, and then daily. Riluzole-treated rabbits typically began to recover motor and sensory function between 6 and 24 hours after the operation, whereas control rabbits did not. Neurologic scoring before 6 hours would have been inappropriate considering the anesthetic properties of riluzole. In our recent study, control rats remained severely paraplegic after the operation, whereas riluzole-treated rats began to recover at 6 hours and had either completely normal neurologic function or a mild to moderate deficit up to 96 hours postoperatively.

This result is of importance because some neuroprotective drugs can modify the early recovery but finally fail to alter the mid-term and late recovery after spinal cord ischemia.[5] Riluzole, however, not only has a transient effect, but also may really and consistently prevent spinal cord ischemic injury when given before aortic crossclamping.

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Measurement of chest wall forces on coughing with the use of human cadavers

To the Editor:

We read with interest the recent article by McGregor and associates[1] on biomechanical testing of median sternotomy closures performed on human cadavers. This work provides the first measurement in the literature of chest wall forces on coughing. One needs to know the magnitude of forces across a sternotomy to test methods of sternotomy closure properly. The norm in biomechanics is that a closure should be able to withstand twice the potential maximum stresses.

These first measurements of chest wall forces on coughing are provided only indirectly. The article says that a force of 220 ± 40 N produced a lateral displacement of 1.85 ± 0.14 mm (ie, 260 N displaces 1.99 mm). Also, an intrathoracic pressure of 63 ± 21 mm Hg displaced 2.14 ± 0.11 mm (ie, 42 mm Hg displaces 2.03 mm). Therefore, indirectly, a cough generating 42 mm Hg produces a lateral force across a sternotomy of 260 N (~26 kg).

This work validates our mathematical model,[2] which describes the force placed across a sternotomy closure. With our model, where P is the distending pressure, r is the radius, l is the height of the chest, and T is the force across the sternotomy,

$$T = rlP = 0.17m \times 0.25m \times 5.6\ kPa = 238\ N \sim 24\ kg$$

the pressure of 42 mm Hg (~5.6 kPa) results in a predicted disrupting force of 24 kg as compared with the measured value of 26 kg. However, a normal cough reaches 100 mm Hg, producing a force of 56 kg, whereas maximal coughing can generate a pressure of 300 mm Hg,[3] producing a force of 168 kg.

We do not share McGregor and associates' enthusiasm for human cadavers. The problem in using whole human sternums for biomechanical testing is in the wide biologic variation (eg, osteoporosis, metastases, age) in the samples and the difficulty in quantifying this variation. Hence a large number of samples is required to include bones of different quality.

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Also, multiple re-use of sternums weakens the bone, introducing further error. Therefore we use sheep sternum as our biologic model of sternotomy closures.

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Reply to the Editor:

We appreciate the comments of Casha, Yang, and Cooper regarding our biomechanical analysis of sternotomy closure. It is interesting and reassuring that our measured results agree with what is predicted by the formula that they have developed. Both studies show that physiologic amounts of force can disrupt traditional sternal closure. We agree that a human cadaver is not a perfect model, but neither is the sheep sternum. The size and shape of the ovine sternum and the anatomic dimensions of the ovine thoracic cage are quite different from those of the human being. In this regard, polyurethane foam has been used as a bone analog in orthopedic research and may provide an option for additional studies in this area.

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