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Reply to the Editor:

We appreciate the interest in our article and the nice comments of Mr Wong. Our report, however, was intended to bring to light the apparent association among the constellation of clinical findings demonstrated in the patients described. We in no way intended to discuss the complex issues related to the potential mechanisms of spinal cord injury during procedures on the thoracic or abdominal aorta. This is an entirely different matter. Rather, we sought to review the relevant literature regarding spinal cord stroke after cardiac surgery and assemble an educated opinion as to the possible mechanism of this devastating complication.

We stand by our supposition that the mechanism of cord injury involved in these patients is almost undoubtedly plaque rupture and embolization of atheromatous debris via radicular arterioles providing collateral blood flow to the cord. Plaque rupture is known to be facilitated by poorly controlled hypertension (among other things). It is a potential mechanism involved in a number of well-described clinical phenomena, including myocardial infarction, carotid distribution stroke or transient ischemic attack, and the showering of atheromatous debris into the lower extremities, renal, or bowel circulation.¹⁻³ In the cases reported, mean pressure tended to be above 100 mm Hg despite aggressive attempts to lower it, constituting, in

our clinical perception, a hypertensive crisis. Paraplegia appeared to develop in proximity to this event.

The fact that the patients described displayed the outward, clinical manifestations of severe peripheral vascular disease is not at all surprising. In these types of patients, population-based or autopsy studies have clearly demonstrated the inconsistent and therefore unpredictable presence of severe atheromatous involvement of the thoracic and abdominal aorta.⁴ Although it may be true that brief periods of extreme hypertension are well-tolerated in general, a high percentage of patients with thoracic aortic catastrophes, such as aortic dissection or ruptured aneurysm, also have hypertensive crises. There is ample evidence in the literature that supports the notion that extreme hypertension is not well tolerated by the diseased thoracic aorta.⁵ As another example, the clinical entity more recently described as "aortic penetration" or "penetrating aortic ulceration" is also associated with severe hypertension and is not dissimilar (clinically) to type III aortic dissection.⁶ Although relative hypotension and resultant hypoperfusion can certainly lead to neurologic injury in certain circumstances, as we alluded to in our report, there is no evidence that this ever occurred in either patient. On the contrary, both presented the picture of malignant hypertension in the perioperative period that was extremely difficult to treat. Perhaps we did not stress this point clearly enough. It is also interesting to note that since the publication of our paper, others who have observed a similar clinical picture have contacted us. Although spinal cord infarction is, fortunately, unusual after isolated heart surgery, perhaps this syndrome is underreported rather than rare.

Neil J. Thomas, MD
Cardiovascular Research and Care Foundation
2720 North 20th St
Phoenix, AZ 85006

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