LETTERS TO THE EDITOR

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Paraplegia after coronary artery bypass operations: Relationship to severe hypertension and vascular disease

To the Editor:

Thomas and Harvey¹ are to be congratulated on their article concerning the rare occurrence of paraplegia arising during isolated coronary artery bypass grafting (CABG), but their conclusion that hypertension and peripheral vascular disease are related needs further analysis.

Paraplegia in cardiac surgery is most commonly associated with repairs of descending or thoracoabdominal aortic aneurysms and dissection where there is ischemic injury to the spinal cord through disruption of the artery of Adamkiewicz at T9 through T12.^{2,3} However, increases in the intraspinal pressure and systemic hypotension in the perioperative period may also be potential mechanisms of injury.⁴⁻⁶ Other etiologic factors such as central disk prolapse, cord hemorrhage, or epidural hematomas⁷ have not been reported after cardiac surgery.

Isolated CABG with cardiopulmonary bypass is an exceptional cause of paraplegia. It is known to occur as a result of aortic dissection produced by aortic crossclamping, cannulation of the aorta, the creation of an aortotomy for the aorto-saphenous vein anastomosis, or the use of an intra-aortic balloon pump (IABP).^{3,8} An IABP may by itself produce paraplegia by disrupting fragile plaques in the descending aorta and causing spinal cord emboli.⁹ This mechanism of spinal cord injury has also been reported when IABP is used during CABG.⁸ An IABP was used in one of the two cases reported by Thomas and Harvey, and since no evidence of aortic dissection was found, embolism into the radicular arteries of the spinal cord may be a contributory factor. Thus the literature seems to support the assertion that peripheral vascular disease is a significant risk factor in the absence of aortic dissection.

Thomas and Harvey also suggested that hypertension is a risk factor by causing plaque rupture, the raising of an intimal

flap, or embolization of debris into the spinal cord circulation. However, this contention is not supported by any study. On the contrary, acute hypertension is usually well tolerated for short periods of time, and cerebral injury arises from acute hemorrhage.¹⁰

In the cases reported, antihypertensive treatment was instituted at a mean arterial blood pressure of 100 mm Hg. This may not be abnormally high in patients with chronic hypertension and may be necessary for normal organ perfusion in these patients. However, in hypertensive patients, sudden lowering of blood pressure could cause strokes and paraplegia in watershed areas,^{11,12} and this is implicated as a mechanism of paraplegia in thoracoabdominal aortic surgery.¹³

It is also interesting that sodium nitroprusside has been shown to be detrimental to spinal cord perfusion,¹⁴ and sodium nitroprusside was used as the antihypertensive agent in the authors' study.

Thus the occurrence of neurologic levels in watershed regions in both case reports may be compatible with hypoperfusion. Therefore postoperative hypertension may be necessary to maintain spinal cord perfusion in some patients and may not be an etiologic factor in paraplegia after CABG.

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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.

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