Reply to the Editor:

We are gratified that our experimental work in the field of aortic graft infection¹ has stimulated a critical review from an experienced and renowned proponent of homograft vascular reconstruction. Vascular infection occurs in the de novo setting (ie, mycotic aneurysm, endocarditis) and after reconstruction of the aorta or cardiac valves. In the case of aortic surgery, graft infection of a vascular prosthesis appears to be more common than primary mycotic aneurysm and provokes the question: "How do these grafts get infected?" Relying on the large clinical experience with cardiac valves, it is apparent that a prosthesis may become infected if it is placed in an infected field or if it is exposed to a bacteremia. Postoperative bacteremia is established as a primary cause of prosthetic valve endocarditis. Early prosthetic valve endocarditis reflects a nosocomial bacteriologic pattern and occurs early after valve replacement, before endothelialization of the prosthesis.² In a similar fashion, vascular graft infection occurs after placement in an infected field or after exposure to a bacteremia. We chose to study the latter question, the ability of a graft to resist infection. Reported clinical experience identifies infection of a sterile prosthesis in a sterile field as a common clinical circumstance.^{3,4} The porcine model of graft infection, with endothelial characteristics similar to those observed in human beings, combined with an early postoperative bacteremia, was chosen on this basis. Intraoperative bacteremia, at the doses required to provoke graft infection, proved to be uniformly lethal in our study. Furthermore, there is probably a direct relationship between the ability of a graft material to resist bacteremia before endothelialization occurs and its ability to resist established bacteremia and infected tissues at the time of implantation. Strictly speaking, our study dealt with the resistance of graft infection and not the treatment of infected grafts. However, the two issues are related and remind one of the question, "Which came first, the chicken or the egg?" Further experimental work must be done to directly investigate de novo native infection or vascular replacement in an infected field.

Antibiotics were not used in this study for two reasons. It was our intent to remove as many variables as possible and in the experimental setting to determine which graft was best in resisting infection. Clinical series report an infinite variability of antibiotic treatments before and after vascular graft infection, which may confuse the results.³ Furthermore, we suppose that most vascular graft infections occur in response to a nosocomial bacteremia long after the perioperative antibiotics have been discontinued, but during the vulnerable period before endothelialization.

At present, thoracic aortic infection remains a dangerous complication. In contrast to other cardiovascular operations, deaths often occur after the 30-day postoperative period because of persistent sepsis and progressive multiple organ

failure. Coselli, Cuneit, and LeMaire⁴ report a current series of thoracic aortic infections with a 30-day and hospital mortality of 11% and 42%, respectively. In our article, we made a firm distinction between the thoracic aorta and the abdominal aorta and distal circulation. Comparisons with clinical series in which the majority of reconstructions occur below the diaphragm are irrelevant and were not reviewed in our manuscript.^{3,5} Surgical options for the abdominal aorta are quite different from those for the thoracic aorta. The availability of extra-anatomic reconstruction and omental coverage and the avoidance of extracorporeal circulation, visceral ischemia, and embolization define the relative simplicity of abdominal aortic surgery. Similarly, a canine model of infrarenal aortic graft infection may not be applicable to the thoracic aorta, despite the observation that only antibiotic-treated cryopreserved allograft, but not untreated allograft or gelatin-sealed polyester graft, had superior resistance to infection.⁶

The debate persists over which material is best to use for reconstruction of the thoracic aorta or the cardiac valves. Despite the intuitive notion that biologic tissue is better, the popularity of autogenous reconstruction, and the numerous favorable clinical series, our experimental observation has supported the use of artificial prostheses. Further investigation will be required to provide a more comprehensive answer.

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