Department of Anesthesiology
University of South Alabama Medical Center
Mobile, Alabama

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What is the Etiology of "Reactions" to Vascular Graft Material?

To the Editor:—The report by Roizen *et al.*[1] is a well-documented examination of a legendary phenomenon in the history of vascular surgery, one often hidden under the rubric "loss of preclot." One can only conclude that this phenomenon is related to the particular choice of a highly porous nonvelour material for the initial graft. In recent years, either velour grafts, which preclot more readily and which are more likely to maintain a laminated coating of fibrin and thrombus, or polytef grafts have become the more common choices for these reconstructions, and this pattern of loss of preclot and apparent disseminated coagulopathy seems to have become rare. The exact biocompatability problem, which is nevertheless very unusual for the nonvelour grafts, has not been elucidated to my knowledge, and it has received no recent attention in the vascular surgery literature.

Case 5 as presented may have a different etiology than that suggested, and crucial information to assess the validity of this case is not presented in the manuscript. The patient had a secondary aneurysm at or just distal to the renal arteries. The authors do not note whether suprarenal or supraceliac cross clamping was necessary. I must presume that this was performed. If so, then a different mechanism for disseminated coagulopathy and severe vasodilitation would have occurred. With the reconstruction of the thoracoabdominal aortic segment, where all visceral artery ostia may be occluded for periods up to 1 h, declamping hypotension and a coagulopathy have been commonly noted. The exact mechanism for this phenomenon is yet unclear, but it presumably occurs with intense vasodilatation of the early ischemic intestine and with initial washout of vasoactive substances into the systemic circulation, perhaps exacerbated by the systemic acidosis which may occur during these procedures. The treatment is anticipatory: early bicarbonate replacement, use of cell-saving devices, volume replacement with fresh frozen plasma, preparation for the administration of aminocaproic acid, and vigorous attempts to maintain the highest possible body temperature during the procedure.

JEFFREY L. KAUFMAN, M.D.
Chief, Vascular Surgery,
Albany Veterans Administration Medical Center
Associate Professor of Surgery,
Albany Medical College
Albany, New York 12208

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In Reply:—I believe the phenomenon we described is not related to the loss of preclot or to the choice of a highly porous nonvelour material for the initial graft. As Dr. Kaufman correctly points out, we found this phenomenon both in woven and in nonwoven grafts; it occurred with three different types of grafts. An anaphylactoid reaction is a particularly difficult reaction to prove because one doesn't want to expose the patient to the graft material *in vivo*. The rechallenge, which was associated with the reaction, was performed in the patient whom we could challenge, and it was a rechallenge done *in vitro* with the graft material. Thus this reaction clearly is not related to loss of preclot in the patient whose blood reacted with abnormal generation of kallikrein when rechallenged with the graft. I hope Dr. Kaufman and other readers appreciate this point.

Figure 1 of the manuscript[1] shows that the plasma of patient 3 generated abnormal amounts of kallikrein activity when exposed to the graft *in vitro*. In addition, in comparison with controls, there were abnormal amounts of C3a generated *in vivo* in both patients 1 and 2, the two patients whose blood was available for study.

Our group of three physicians was privileged to give anesthesia for more than 200 abdominal aortic reconstructions per yr for this group of vascular surgeons and we never saw this problem other than the times reported. This phenomenon was rare—not the usual phenomenon slower or less technically competent surgeons experience. Yes, the treatment of hypotension upon opening of a newly revascularized limb is anticipatory, but these surgeons work so fast and are so competent that bicarbonate is virtually never necessary and volume replacement suffices. Clearly, the reason we were able to discover this phenomenon is that something atypical of the normal course of expected events occurred in these patients. Volume replacement and maintenance of normal left ventricular end-diastolic volumes as assessed by echocardiography, or maintenance of left ventricular end-diastolic pressures as measured by pulmonary capillary occlusion pressure, had