these new potential confounders in the fully adjusted Cox regression analysis, being ≥65 years and on a traditional perioperative protocol remained the only factors independently associated with prolonged hospital stay. This finding supports the important role of the ERP in functional recovery after RP abdominal aortic aneurysm repair.

Second, they referred to the left kidney "down" technique as having potential for bleeding, developing a plane anterior to the kidney, whereas they prefer a left kidney "up" approach, except in case of retroaortic left renal vein. However, in the RP approach for both aneurysmatic and Leriche diseases, we did not experience bleeding during the dissection to maintain the left kidney down. Instead, although it is more time-consuming, we do prefer such a technique because it is less traumatic as the kidney is not displaced and it seems easier to get further distal on the right renal artery.

Third, they mentioned that many surgeons performing RP surgery see the clear physiologic benefits it offers over the transperitoneal (TP) route and speculate that the advantages of the RP one may have little to do with ERPs but may be primarily due to the approach itself. We agree that RP surgery offers such benefits over the TP approach; however, all patients included in our study were operated on through the RP approach, and control group patients showed slower functional recovery and longer hospital stay as opposed to patients who were on an ERP.

Finally, although one of the general strategies of enhanced recovery methodology is to reduce the surgical trauma, we do agree that ERP is most likely to demonstrate greatest benefit for vascular operations involving the peritoneal cavity. As the magnitude of the surgical stress response increases with the invasiveness of the surgical operation, it seems sensible to implement strategies to attenuate such a response (ie, ERPs), in particular when the trauma of the operation cannot be reduced (eg, open TP surgery).

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Regarding "Outcomes of arterial resection during pancreatectomy for tumor"

We read with interest the paper by Glebova et.[1] The study included a considerable number of patients compared with the other reports in the literature and gave valuable information about the results of arterial resections during pancreatectomy. We have a limited experience in arterial resections during pancreatectomies, including two right-sided (Whipple) and five left-sided pancreatic resections (distal pancreatectomy). Two patients who underwent Whipple operation with arterial resection died on postoperative days 5 and 35. However, all of the five patients who underwent distal pancreatectomy with celiac axis resection survived. We believe that perioperative morbidity and mortality are not the same after left- or right-sided pancreatectomies with arterial resection, as the extent of surgery in these operations is markedly different. Whereas vascular reconstruction is generally required in the right-sided resection, it is not generally required in the left-sided pancreatectomy.

In the paper by Glebova et al, there were no data about in-hospital mortality. Their objective was mentioned as examining the safety and efficacy of arterial resections during pancreatectomy. It is clear that the most important determinant of safety is the postoperative mortality. In addition, the results of arterial resection could be evaluated after either left-sided or right-sided pancreatectomies separately. Could the authors please give information about the perioperative mortality? Was the perioperative mortality different after arterial resection in the right-sided and left-sided pancreatectomies?

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REFERENCE

1. Glebova NO, Hicks CW, Tosoian JJ, Piazza KM, Abularrage CJ, Schulick RD, et al. Outcomes of arterial resection during pancreatectomy for tumor. J Vasc Surg 2016;63:722-9.

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Reply

We thank Professors Ozer and Kayaalp for the question. There were two perioperative deaths among patients with arterial reconstructions in our group. One was intraoperative, and the other occurred on postoperative day 29. Both patients had pancreaticoduodenectomies. Thus, in our series, the perioperative mortality (defined as occurring within 30 days of surgery) was 9% (2/22 patients) for pancreaticoduodenectomy and 0% (0/9 patients) for distal pancreatectomy. We would like to caution about generalization of these data, as the numbers of patients are quite small in both groups for a statistically sound comparison.

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Regarding "Prospective evaluation of postimplantation syndrome evolution on patient outcomes after endovascular aneurysm repair for abdominal aortic aneurysm"

We read with great interest the recent publication of Arnaoutoglou et al[1] demonstrating that postimplantation syndrome (PIS) after endovascular aneurysm repair (EVAR) seems to correlate with the presence of a cardiovascular or any other adverse event during the first year after EVAR. The authors evaluated the kinetics of three common inflammatory markers (highly sensitive C-reactive protein, white blood cells, and interleukin [IL]-6) that were found increased early after EVAR in the PIS group. The striking finding of the correlation between PIS and cardiovascular risk makes the need for full elucidation of the pathophysiologic mechanism of the syndrome mandatory to facilitate a potential therapeutic strategy focusing on inflammation reduction.

Current literature fails to clarify these uncharted waters because the majority of authors did not consent to the definition of PIS, and they do not evaluate the same markers.[1-3]

The increase in IL-6 levels is indicative of the activation of macrophages as antigen-presenting cells in a cell-mediated inflammatory response as well as activation of endothelial cells, probably because of the endograft-aortic wall interaction. IL-6 also induces the expression of acute phase reactants (C-reactive protein) and fibrinogen in the liver.[4] Of interest, the authors did not demonstrate increased fibrinogen expression in PIS patients. This might imply that the increase in IL-6 levels is short and locally induced rather than systemic. It is known that the major source of IL-6 after EVAR is the aneurysmal thrombus.[3,4]

The possibility of systemic response to endograft could have been evaluated by tumor necrosis factor expression as evaluated by other studies[2] because it crosstalks with macrophages and causes a systemic inflammatory response including fever and neutrophil and endothelial cell activation (TRADD-TRAF pathway). Similarly, the evaluation of interferon-γ, IL-2, and CD40L levels would indicate the involvement of the T_H1 pathway of cell-mediated immunity (JAK-STAT and Toll-like receptor pathway) and T-dependent antibody production (CD40L). In the same frame, the expression of IL-10 and tumor growth factor-β that act as anti-inflammatory cytokines could also have been evaluated,[2-4] which can counterbalance the proinflammatory response of IL-6 and explain the deterioration of systemic inflammatory response syndrome/PIS during the first 5 days after EVAR.

Moreover, it would be of interest if the authors had presented the stratification of leukocyte subtypes throughout the follow-up period because the increase in white blood cells alone is not informative. We suspect that early after EVAR, neutrophils should be the dominant subtype, and during the follow-up, lymphocytes and monocytes should represent the majority (CD4 and CD45 marker).

High expression of markers such as CCR7 and CD62L is indicative of immune memory, which would be a striking finding because it implies that the patients with PIS have already been exposed to antigens similar to endograft (mimicry), thus demonstrating a more robust inflammatory response.

As far as humoral response is concerned, the authors did not evaluate the role of the sympathetic/adrenomedullary system and the hypothalamic-pituitary-adrenal axis that should have been activated.[4,5] Fig summarizes the potential mechanism of systemic inflammatory response syndrome/PIS after EVAR.