basis. On the basis of a large prospective study of patients undergoing major vascular procedures, we showed that maintaining clopidogrel up to the time of surgery is not associated with increased bleeding complications or transfusion requirements. Therefore, we believe that vascular surgeons must become familiar with procedures performed in patients receiving clopidogrel and teach their residents how to minimize the risk of bleeding in such patients. We also thank Stone and colleagues for their support of our conclusion.

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Regarding "Comparison of outcomes following endovascular repair of abdominal aortic aneurysms based on size threshold"

Keith and colleagues[1] report a comparison of outcomes after endovascular repair in small (4.0-4.9 cm) vs larger abdominal aortic aneurysm (AAA). Endovascular repair was performed in 157 patients with small AAA, of whom 15.3% had complications. Four randomized trials, two of open repair and two of endovascular repair,[2] have demonstrated that there is no benefit from repair of AAA smaller than 5.5 cm, making this one of the best-proven tenets of AAA management. The authors state that they have continued to perform these procedures because of their previous experience of low perioperative morbidity and mortality. Surely, this is not sufficient justification; would reports of low morbidity and mortality after appendectomy in healthy patients lead them to recommend that procedure for all?

A second justification for repair of small AAA offered by Keith and colleagues, that the repair threshold should be lowered because many patients with a smaller AAA will eventually cross it, is fallacious.[3] After lowering the repair threshold from the 5.5 cm established by randomized trials to, for example, 5.0 cm, it could then be argued that many patients with a diameter of 4.5 cm will reach this new threshold in a few years; therefore we must now lower it to 4.5 cm, and so on, until we get to 3.0 cm or lower, in a clear *reductio ad absurdum*. The key point is that many patients will not cross the 5.5 cm threshold and will never need repair.

Their third justification is their concern that if repair is deferred, the patient might no longer be eligible for endovascular repair and then might need open repair, as occurred in the CAESAR trial. However, given that this "harm" did not influence the results of either endovascular small aneurysm trial and that another of the best-proven tenets of AAA management is that endovascular repair offers no long-term advantage over open repair,[4] this justification also fails.

Their implied fourth justification, that patients with small AAA fared better after endovascular repair than did patients with large AAA, is no justification at all—those who do not need a treatment often fare better with it than those who do need treatment. If the concern is that patients would have had worse outcomes if repaired later, this is precisely what the four randomized trials disproved.

Accepting the authors' statement that after excluding ruptures, a fifth of the small AAA were symptomatic (which is surprising because none of the 567 patients with small AAA followed for up to 8 years in the ADAM study[5] had non–rupture-related symptoms), what valid justification remains for repairing the other 80%? What did the authors say to the 15% who suffered complications after a procedure that they may have never needed? Performing endovascular repair on small AAA causes harm and does not confer any benefit, at least not to the patient.

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Reply

It is of no surprise that the article by Keith et al[1] has stirred up some strong opinions regarding size threshold and abdominal aortic aneurysm (AAA) repair, and we appreciate the opportunity to respond. While the comments made in the Letter to the Editor by Dr Lederle are the standard argument made against endovascular AAA repair (EVAR) for small AAAs, the problem is that they represent a static perspective focusing solely on a rigid AAA size threshold of 5.5 cm established on the basis of randomized evidence from clinical trials comparing open repair versus surveillance.[2,3] However, with these studies predating and not including endovascular options, this threshold should not simply be extrapolated to EVAR. There is ample evidence comparing open AAA repair with EVAR to suggest lower overall risk with a different distribution of fewer severe complications favoring EVAR over open repair.[4,5] Although long-term survival curves merge a few years after repair, there is still early survival advantage favoring EVAR, which extends long-term for younger patients.[6] Additionally, another study from our institution demonstrated the early advantage of EVAR over open repair that is sustained over 9 years.[7] Therefore, the need for a different risk-to-benefit crossover point for EVAR on the basis of size threshold is readily supported, especially in patients with a greater life expectancy. In the more recently published randomized trials comparing EVAR and surveillance for small AAAs, both PIVOTAL and CAESAR showed equivalence in terms of rupture risk and mortality, with a significant number of patients in the surveillance group eventually requiring EVAR.[8,9] The underlying implication here is that there is a subgroup of patients with small AAAs who underwent EVAR that is driving equivalence in both of these trials, and this