

Re. 'Remote Ischemic Preconditioning to Reduce Contrast-Induced Nephropathy: a Randomized Controlled Trial'

We read with great interest the paper by Menting et al. about the use of remote ischemic preconditioning (RIPC) to reduce contrast medium induced nephropathy (CIN) in patients at risk of CIN.¹ Their results in a group at high risk of CIN are in line with results from Er et al. who showed that use of such a procedure could reduce CIN in high risk patients. Er et al. identified CIN in 20 patients in their control group, but only six in their RIPC group ($p = .002$).² In both studies, RIPC was performed as an adjunct to hydration. However, in our opinion, the role of hydration requires further discussion, especially where different hydration protocols are performed. Zarbock et al. showed that RIPC alone reduced the rate of acute kidney injury and the use of renal replacement therapy among high risk patients undergoing cardiac surgery.³ Therefore, the question remains whether or not RIPC should be used as an adjunct or alone?

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Response to "Re: Remote Ischemic Preconditioning to Reduce Contrast-induced Nephropathy: A Randomized Controlled Trial"

We thank Drs. Koch and colleagues for their comments. Their question, if remote ischemic preconditioning is sufficient on its own to prevent contrast medium induced nephropathy (CIN), cannot be answered with current literature data. In patients undergoing major (non-)cardiac surgery the efficacy of remote ischemic preconditioning (RIPC) remains unclear. Some randomized controlled trials showed a reduction in surgery related acute kidney injury (AKI),^{1,2} whereas others could not confirm this.^{3,4} Regarding the use of remote ischemic preconditioning in patients receiving intravascular contrast media, we feel that there is now suggestive evidence that remote ischemic preconditioning when added to hydration may prevent CIN.^{5,6} However, the routine use of added RIPC in unselected patients cannot be advocated, and better identification of high risk patients is needed. Since hydration is proven to be effective in preventing CIN, and dehydration is associated with higher risk of AKI we would argue against the use of ischemic preconditioning alone in such high risk patients. However, we envisage that RIPC alone may be sufficient to prevent CIN in intermediate risk patients, and could be used to replace intravenous sodium chloride or intravenous sodium bicarbonate. Controlled studies are needed to explore the best strategies for the prevention of CIN.

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Re: 'Endovascular Treatment of Ruptured Abdominal Aortic Aneurysms with Hostile Aortic Neck Anatomy'

We read with great interest the article by Broos and colleagues.¹ The authors reported encouraging outcomes of EVAR in ruptured AAA with hostile aortic neck anatomy (HNA). We congratulate them for such a wonderful result, which may broaden the selection criteria of the current endovascular strategy to include patients previously excluded from EVAR.

As the authors advocated, it is technically feasible and safe to perform EVAR in rupture AAA with HNA at experienced endovascular centres. However, their results may not be generalisable to less experienced centres, and they did not tell inexperienced surgeons what to do under these circumstances. Previously, Brownrigg and colleagues² reported that endovascular aneurysm sealing (EVAS) is effective for AAA with challenging aortic anatomy, which seems suitable for treating a greater proportion of patients than EVAR. As stent length is the only sizing variation, EVAS is of benefit in the emergency setting for ruptured aneurysm repair.³ Ruptured AAA with HNA can be repaired by experienced or inexperienced surgeons with the revolutionary EVAS.

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Response to 'Re: Endovascular Treatment of Ruptured Abdominal Aortic Aneurysms with Hostile Aortic Neck Anatomy'

We would like to thank Zhou et al. for sharing their views on the treatment of ruptured abdominal aortic aneurysms (rAAAs) with hostile infrarenal aortic neck anatomy. The current EVAR devices have proven their safety and efficacy, in both highly specialized and less experienced centers. In contrast, the endovascular aneurysm sealing (EVAS) technique is relatively new and information on intra- and post-operative complications is sparse. For this reason we feel that great care should be taken in emergency use.

The first real world EVAS registry data have only been published recently.¹ These data consist of selected patients treated in internationally renowned vascular centers. Technical success is promising, but secondary interventions were required in 9% of patients within 12 months. These clinical failures were associated with patient selection and deployment techniques. Current literature on EVAS for rAAA treatment consists of a mere nine patients.^{1–4} All patients were treated in large vascular centers with substantial elective EVAS experience.

In addition, endobag filling in EVAS is performed under pressure monitoring of 180–220 mmHg, which allows the correct dosage of the polymer to seal the aneurysm. Theoretically, this pressure guided strategy could increase the aortic tear in a rAAA.

Although EVAS proposes sizing benefits, experience in "less experienced hands" is not available. In contrast, there is extensive experience with standard EVAR devices in treating rAAA. While EVAS aims at treating hostile anatomy, the current instructions for use are the same as those applied in our patients.

We feel that it is too early to speculate on the use of EVAS for rAAA, especially in hostile anatomy and most certainly by less experienced operators. In our opinion, the emergency outside IFU use of any device should only be