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Invited Commentary

To Use or Not to Use Heparin during Abdominal Aortic Aneurysm Repair?

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After its discovery by McLead[1] in 1916, heparin was first used in the 1950s and since then has remained the standard anticoagulant to avoid the risk of intraoperative arterial and venous thromboembolism in cardiovascular surgery. Its use is not without risks, bleeding being the most frequent and severe complication. This is particularly true during major vascular surgery such as open repair of abdominal aortic aneurysm (AAA). However, since thromboembolism may pose the vascular reconstruction and patient's life at risk, currently most of surgeons routinely administer heparin intravenously during elective open and endovascular repair of AAA.[2] In this issue of the Journal, Wiersema et al.[3] reported the results of a systematic review on this issue claiming that intraoperative use of heparin may not be beneficial during repair of AAA. Such conclusions do not apply to repair of ruptured AAA or endovascular treatment because data on these are scarce. The authors did not perform a meta-analysis, but a personal analysis of data on elective open repair of AAA revealed a risk ratio for mortality of 0.984 (95% confidence interval (CI) 0.368–2.629, three studies included), for arterial thromboembolism of 0.966 (95%CI 0.496–1.881, four studies included) and for myocardial infarction of 1.198 (95%CI 0.032–44.414, two studies included). These data indicate that intravenous administration of heparin may not provide any benefit to patients undergoing elective open repair of AAA. However, such results should be viewed in the light of a limited number of studies of small size and suboptimal quality. In particular, selection bias is a major problem with these studies. Furthermore, patients not receiving intravenous heparin may have received a significant amount of diluted heparin in saline in the graft and iliac or femoral arteries, namely uncontrolled heparinisation.[4] My concern regards the potential increased risk of ischaemic colitis[5] and spinal cord ischaemia[6] in patients not heparinised. Similarly, lack of heparinisation may expose patients with coronary artery disease to an increased risk of cardiac events.[7] I believe that intravenous heparinisation was a major achievement in the early years of vascular surgery. In an era of aggressive antithrombotic treatments, it would be difficult to safely prove the non-inferiority or even superiority of a no-heparinisation policy during major vascular surgery.

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