Editor—Dent and Lekic relate in their letter to an important topic, namely hypercoagulability with adverse events after discontinuation of unfractionated heparin. In our study we observed the majority of ischaemic events clustered around day 0 or 1 after surgery. We think it is reasonable to assume that the above phenomenon contributes to the overall high rate of events—as does postoperative hypercoagulability without earlier heparin administration. Certainly, neither effect can be proven by our study and remains speculation. Additionally, we emphasize that despite >80% of the study population taking antiplatelet drugs until the day before surgery, the rate of ischaemic events was high. More and more evidence is mounting that we need standardized tests to adequately monitor and titrate anticoagulant and antiplatelet drugs on an individual basis in the perioperative scenario.

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Unfractionated heparin and coronary artery stenting

Editor—We read with interest Vicenzi and colleagues’ paper concerning coronary artery stenting and non-cardiac surgery. We were concerned by the high cardiac complication rate they reported, particularly in patients receiving unfractionated heparin (UFH) as a component of their anticoagulant regime (14 patients out of 16) compared with low molecular weight heparin (LMWH) (32 out of 87). The authors, while noting this association, warn against interpreting this as a significant effect, as the heparin regime was not subject to randomization in the study design. We believe, however, that this is further evidence of ‘heparin rebound’—a period of hypercoagulability after abrupt cessation of an infusion of UFH. This can be associated with ischaemic events when UFH is used in the management of unstable angina and myocardial infarction. This effect has been attributed to an increase in thrombin activity and activation of platelets during UFH infusion which persist for many hours after cessation of infusion, whilst the protective anticoagulant effects decline rapidly because of the short half-life of UFH. Ischaemic events in Theroux and colleagues’ study were clustered around a median time of 9.5 h after cessation of UFH—was there any temporal relationship between UFH administration and cardiac events in the authors’ study?

LMWH which has a longer half-life than UFH and does not activate platelets is not associated with an increase in ischaemic events and should, perhaps, be considered the drug of choice in this setting.

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Adverse events in anaesthetic practice

Editor—I read with interest the article by Smith and colleagues on adverse events in anaesthetic practice. I have recently completed an audit in our anaesthetic department to ascertain the reason why critical incidents are under-reported. My audit relied on both consultants and registrars completing an anonymous questionnaire, the results of which are summarized in Table 1. I was pleasantly surprised to see that we are overcoming the era of ‘blame culture’ and that triviality was the most common reason for under-reporting. I, as do some of my colleagues anaesthetists, agree that the definition of ‘criticality’ is ambiguous. As a result most of us would not regard situations such as laryngospasm and circuit disconnection as a ‘critical’ incident. Anaesthesia as a speciality is fraught with life-threatening...