that AF and cTNT are markers of the whole quality of the surgical procedure that has to be adapted to patients’ characteristics, but also to the quality of the postoperative haemodynamic management.

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REFERENCES


eComment. Postoperative atrial fibrillation: a robust human model of atrial fibrillation genesis?

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I read with great interest the article by Koolen et al. [1] The authors have studied the association between perioperative cardiac troponin T (cTNT) release and the development of postoperative atrial fibrillation (AF) in a large cohort of coronary artery bypass grafting (CABG) patients. Although elevated levels of cTNT were associated with the development of postoperative AF in univariate analysis, the finding was not stable in multivariate models, and the authors conclude that postoperative cTNT levels are not predictive of postoperative AF, and that both postoperative AF and cTNT are associated with negative outcome. This is, to my knowledge, the largest study so far of cardiac enzyme release and postoperative AF, and the regression models are well presented and transparent. The authors are to be congratulated for a nice study in an interesting subject.

In the present study, postoperative AF was defined as an episode of AF during hospital stay. While this is a common definition in clinical databases for practical reasons, it introduces an information bias. Patients with a longer hospital stay, for whatever reason, have a longer observation time leading to a higher probability of detection of an episode of AF than patients discharged within the normal time (who may experience undetected episodes of AF at home during the same time). As a longer hospital stay is correlated to complications like prolonged intensive care and sepsis, with a higher risk of postoperative AF, this will inevitably lead to a link between postoperative complications and postoperative AF [2], as found in the present study. One way to correct for this information bias is to define postoperative AF as AF occurring during a specific period of time (in this case, the ‘normal’ hospital stay) in which all patients can be observed. In this way, we might perhaps get closer to answering the question: if we were able to prevent postoperative AF, would this lead to less complications?

Researchers studying pathophysiology often look for animal models, which have a genetic tendency to develop the disease of interest, and in which different kinds of interventions are possible to study. In this context, the development of postoperative AF in patients undergoing cardiac surgery is a fascinatingly robust model of AF genesis in humans. Despite numerous efforts to reduce the incidence of postoperative AF by pharmacological or interventional treatment, the reduction has been only moderate [3]. The timely linkage between the inflammatory response after
cardiac surgery and the incidence of postoperative AF is intriguing, and both steroid and colchicine treatment have been shown to reduce the incidence of postoperative AF [4, 5]. The precise mechanism of inflammatory induction of postoperative atrial fibrillation is, however, still unknown. As postoperative AF has negative long-term consequences, further studies are needed.

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**References**


