Unraveling the mystery of postoperative atrial fibrillation

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Postoperative atrial fibrillation (POAF) continues to be the most common complication after cardiac operations. Despite all the advances in surgical technique, anesthesia, and perioperative care, the incidence of this arrhythmia has remained unchanged during the last 2 decades.¹ Although POAF is for the most part self-limiting, its impact on both short-term outcomes and long-term mortality is well documented.^{2,3} Still, no effective prophylactic measure is in sight. Prophylactic use of antiarrhythmic agents, pulmonary vein isolation, a posterior pericardial window, and postoperative atrial pacing, have had variable success in lowering the incidence of POAF, but none has demonstrated any impact on the postoperative outcomes or survival.⁴ The literature is replete with various theories about the underlying causes and factors responsible for the genesis of POAF; however, the precise mechanism for its development, its underlying causes, and effective preventive measures remain elusive. Older age appears to be the only consistent factor in most large series. Hypertension, obesity, and race are other risk factors that are also common to nonsurgical atrial fibrillation.⁵⁻⁷ The role of cardiopulmonary bypass and the potential mechanisms leading to POAF have long been discussed and reported; however, experience with off-pump coronary artery bypass grafting disproved the theories about the role of cardiopulmonary bypass as the major contributing factor in the development of POAF.⁸

The report by Melby and colleagues⁹ in this issue of the *Journal* is a new look at POAF in the time-related parametric hazard domain. Melby and colleagues⁹ suggest a bimodal hazard for POAF, with distinct risks for each phase that overlap with time. Looking at their data and in line with previous reports on this topic, mitral valve replacement with or without coronary artery bypass

grafting carries the highest risk for development of POAF.¹⁰ The risk remains elevated for a longer interval before returning to the baseline. Melby and colleagues' suggestion⁹ for looking into the mechanisms responsible for this behavior is well taken.

Cardiac surgeons and cardiologists continue to be challenged by POAF and its associated increased morbidity and mortality. In our analysis of the causes of death at 1 year in patients enrolled in the Randomized On/Off Bypass (ROOBY) trial (POAF, n = 551; no POAF, n = 1552), most of the deaths in the POAF group (71.4%) were cardiac related, with only 1 death secondary to a cerebrovascular accident (unpublished data). Current management strategies for POAF are borrowed mainly from the experience with nonsurgical atrial fibrillation. POAF should be regarded as a distinct entity from nonsurgical atrial fibrillation. Efforts should be directed toward elucidating the basic pathophysiologic mechanisms of POAF, with concurrent initiatives directed toward developing effective prophylactic measures to prevent its occurrence and thus improve patient outcomes.

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