Postoperative atrial fibrillation: The truth is stranger than fiction

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Disclosures: Author has nothing to disclose with regard to commercial support.

Received for publication March 7, 2017; accepted for publication March 17, 2017.

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J Thorac Cardiovasc Surg 2017;154:499-500

0022-5223/\$36.00

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<http://dx.doi.org/10.1016/j.jtcvs.2017.03.057>

Given its prevalence and the bulk of literature it has inspired, it is quite remarkable how little we know about new postoperative atrial fibrillation (POAF). Theories of pathogenesis are certainly plausible—dispersion of atrial refractoriness, increase in phase 3 depolarization, enhanced automaticity, increased interatrial conduction time, decreased conduction velocity, atrial transmembrane potentials, and fluid and electrolyte shifts¹—although the variety of theories suggests either a complex mechanism, a lack of clear understanding on our part, or both. Although the list of reported risk factors is extensive, some appear more frequently than others—but none is pathognomonic.² The mere fact that some patients have POAF develop despite evidence-based prophylaxis, while a larger portion do not, even without prophylaxis, suggests that it is neither the substrate nor the surgery alone but rather a combination of the two that can best explain the occurrence.³ Even the highly plausible data regarding atrial ischemia and inflammation after surgical insult do not explain a definitive (albeit lower) incidence of POAF after off-pump coronary artery bypass grafting (CABG) or transfemoral transcatheter aortic valve replacement.⁴

How important is it to prevent what ostensibly is a self-limited phenomenon? Here again, data are not clear. Although association with perioperative mortality is commonly reported, POAF's role as an independent predictor is controversial.⁵⁻⁷ Association with morbidity appears clearer; however, current data do not permit determination of causality.

It seems reasonable that if a certain substrate predisposes toward POAF, then that substrate might have long-term prognostic implications. Most but not all studies suggest an increased incidence of late atrial fibrillation, while impact on late mortality remains controversial. Interestingly, long-term follow-up of patients undergoing CABG and aortic valve replacement from the same database during the same period demonstrated that POAF was an independent predictor of late mortality

Origin of dispersion of atrial refractoriness. Reprinted with permission.³

Central Message

Despite conflicting reports, there appears to be a time-related impact of postoperative atrial fibrillation on long-term survival.

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in patients undergoing CABG alone but not those undergoing CABG with valve procedures.^{8,9} The higher overall mortality of the patients undergoing valve procedures may suggest higher competing risks or a difference in how the POAF “substrate” interacts with ischemic versus structural challenges. It is here that the study of Swinkels and colleagues¹⁰ in this issue of the *Journal* is welcome and unique. The 18-year follow-up is the longest to date in the literature. The finding that POAF was not an independent risk factor for late mortality is less secure than it might seem. The *P* value of .052 for the hazard ratio suggests that if the null hypothesis were true, we would expect to find the survival distribution observed only 5.2% of the time—hardly a resounding endorsement. Examination of the Kaplan-Meier plots suggests a clear survival benefit for the population without POAF during the first 10 years, with virtual overlapping curves thereafter. Unfortunately, rather than a more robust landmark analysis, Swinkels and colleagues¹⁰ elected to evaluate the interaction with time in 1-year slices, with a predictable lack of power in any given year to drive significance for the interaction. Nonetheless, the information remains valuable. Whatever the substrate for POAF, it seems that its impact on prognosis may diminish with time. In this confusing arena, any small piece that can contribute to assembling the puzzle is more than welcome.

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