long-term outcomes in this high-risk patient population are warranted.

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Commentary: Postoperative atrial fibrillation: An old foe in a new light

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Postoperative atrial fibrillation (POAF) continues to plague the work of cardiac surgeons as one of the most commonly reported complications. Although often self-limited and partially preventable,¹ the reported incidence after bypass and valve surgery is as high as 40%.²

In this edition of the *Journal*, Björn and colleagues³ report their results on 782 patients undergoing isolated surgical aortic valve replacement (SAVR), the incidence of new-onset atrial fibrillation (NOAF), persistence of atrial fibrillation after hospital discharge, and associated mortality over a median follow-up of 5.4 years. Of note, all patients in their cohort received

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CENTRAL MESSAGE

Postoperative atrial fibrillation (POAF) portends an increased risk of long-term mortality after aortic valve replacement. The extent to which POAF directly causes morbidity and mortality remains to be determined.

perioperative and postoperative beta-blocker medication. In addition, all patients, including those receiving a bioprosthetic valve, were received routine anticoagulation with a vitamin K antagonist for at least 90 days postoperatively.

A documented episode of atrial fibrillation of 10 minutes or longer was used to define NOAF, which had an incidence of 42% during the index hospitalization and 32% in the outpatient setting, with a higher incidence in patients receiving a bioprosthetic valve, who

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comprised an older and more morbid population known to have a higher rate of atrial fibrillation than in the general population.

The study has some limitations owing to its retrospective nature, reliance on hospital charts to determine both exposure and outcomes, and potential for recall bias. The authors appropriately acknowledge the difficulty of establishing causality and identify NOAF as a risk marker. Their data support an independent association of NOAF and all-cause mortality, with a hazard ratio (HR) of approximately 2. Importantly, the cause of death was not related to a stroke but rather to cancer, infections, or heart failure. So, what can we learn from this?

Other investigators have previously suggested an association between NOAF and increased long-term mortality after aortic valve surgery, with an HR of ~ 2 .⁴ Björn and colleagues add to the growing body of evidence suggesting that the occurrence of NOAF portends a worse prognosis. What is less clear, however, is how NOAF fits into various causal pathways. In other words, is NOAF a risk marker or a risk factor for increased morbidity? Long-standing atrial fibrillation has adverse consequences for ventricular remodeling, and in patients with hypertrophied ventricles it impacts diastolic filling and cardiac output even in the short term. On the other hand, in the absence of atrioventricular valve disease and prior atrial remodeling, AF is arguably better considered a response to a systemic (eg, sepsis) or local (eg, surgery) inflammatory response in addition to the patient's age. This view is substantiated by the dramatically lower incidence of NOAF after transcatheter aortic valve replacement (TAVR) compared with SAVR in all trials comparing the 2 AVR modalities.⁵ Minimally invasive SAVR techniques also may be associated with a lower incidence of NOAF compared with the standard sternotomy approach.⁶

However, even after minimizing invasiveness and thereby NOAF incidence, the implication for long-term survival is no less severe. A recent analysis of intermediate and low-risk patients undergoing surgical and transcatheter AVR showed that NOAF was associated with a similar 2-fold increased HR over 2-years of follow-up in patients undergoing TAVR.⁷

The totality of evidence suggests that NOAF often heralds new or worsening trouble, especially in patients after cardiac procedures. We congratulate Björn and colleagues on their interesting analysis of the association of NOAF with long-term survival. Future studies should address the directionality of the association and the attributable risk of atrial fibrillation for all-cause and cause-specific morbidity and mortality.

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