Commentary: Getting to the heart of postoperative atrial fibrillation after cardiac surgery

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Central Message

Much mechanistic study is yet required to develop potential predictors of postoperative atrial fibrillation into a clinically useful risk prediction model.

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Postoperative atrial fibrillation (POAF) is associated with prolonged hospital stay, greater health care costs, and increased mortality after cardiac surgery.¹ Effective prophylactic measures, such as corticosteroids and antiarrhythmics, are not widely used, perhaps because of concerns about side effects and costs.² A clinical prediction model for POAF might encourage targeted use in higher-risk patients, but development of such a model requires improved understanding of POAF pathophysiology and risk factors.

In this issue of the *Journal*, Bening and colleagues³ address an important gap in the literature by investigating predictors of POAF. Casting a wide net, they examine atrial muscle fiber function, echocardiographic measurements of heart chamber size and function, and biochemical markers of inflammation.

Bening and colleagues³ demonstrate an association between impaired left atrial contractility and occurrence of POAF. Measurement of left atrial contractility is a labor-intensive process of harvesting atrial tissue intraoperatively, preparing skinned myocardial fibers, exposing them to increasing concentrations of calcium, and measuring their contractile force. Results are only available postoperatively, whereas evolving evidence suggests POAF prophylaxis should start preoperatively or intraoperatively.^{4,5} The clinical applicability of this unwieldy process is therefore limited. The authors³ also report that echocardiographic evidence of increased right atrial size and depressed right ventricular function may predict POAF, which has not been previously reported in the literature and merits further exploration.

Bening and colleagues³ suggest that patients with impaired left atrial contractility and echocardiographic evidence of right heart dysfunction may benefit from postoperative cardiac monitoring and magnesium and potassium supplementation. At our institution, and we suspect in many other centers, these measures are routinely used for

all postoperative cardiac surgical patients. Although relatively low risk and low cost, they are also ineffective for POAF prophylaxis.²

Clinical prediction models must discriminate between patients who will experience an event and those who will not. Clinicians can use such models to weigh the risks of an event, such as POAF, against the risks of interventions, such as corticosteroids and antiarrhythmics.⁶ Bening and colleagues³ unfortunately do not report cutoff values for atrial contractility or echocardiographic parameters, which would permit discrimination between higher and lower risk patients. Neither do they describe the degree of risk conferred by the presence of each predictor. Future work should examine these predictors in larger populations.

Another area for future work is the mechanistic pathophysiology of POAF. Despite previous research suggesting links between POAF and inflammation,⁷ why were none of the inflammatory biomarkers measured in the study of Bening and colleagues³ predictive of POAF? By what mechanism does right heart dysfunction increase risk of POAF?

Bening and colleagues³ have provided both impetus and direction for exciting future research into the mechanisms of and risk factors for POAF after cardiac surgery. Ultimately, their research moves us closer to a clinical risk prediction model for POAF, which would allow individualized POAF risk management.

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