Commentary: A case for getting bogged down in the details—Post-coronary artery bypass grafting atrial fibrillation

Siamak Mohammadi, MD, FRCSC, and Dimitri Kalavrouziotis, MD, FRCSC

From the Department of Cardiac Surgery, Quebec Heart and Lung Institute, Quebec City, Québec, Canada.

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Address for reprints: Siamak Mohammadi, MD, FRCSC, Department of Cardiac Surgery, Quebec Heart and Lung Institute, 2725 Chemin Sainte-Foy, Quebec City, Québec, Canada, G1V 4G5 (E-mail: siamak.mohammadi@fmed.ulaval.ca).

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Siamak Mohammadi, MD, FRCSC (left), and Dimitri Kalavrouziotis, MD, FRCSC (right)

Central Message

The pathophysiologic mechanisms involved in new-onset atrial fibrillation after CABG continue to be elusive but may involve diminished atrial myocyte contractility and right heart dysfunction.

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The prevalence of new-onset postoperative atrial fibrillation (POAF) has not changed during the past few decades, and many studies have been performed to identify patients at highest risk and prophylactic strategies to prevent POAF.¹ In this issue of the *Journal*, Bening and colleagues² attempt to determine a “unifying theory” of POAF on the basis of a pathophysiologic model that uses serum and tissue biomarkers, contractile characteristics of left and right atrial appendages removed at surgery, and clinical and echocardiographic variables, between patients who had POAF develop after elective coronary artery bypass grafting (CABG) and those who did not. Two hundred twenty-nine patients without a history of atrial fibrillation were recruited prospectively, and 16.8% had POAF develop. In multivariate analysis, the independent risk factors for POAF after CABG were (1) advanced age, (2) higher left and right atrial areas on 2-dimensional echocardiography, (3) lower tricuspid annular plane systolic excursion, and, finally, (4) reduced left atrial myofilament contractile force in an ex vivo analysis of human tissue. Other inflammatory and fibrotic biomarkers were not associated with POAF.

That the typical patient who had POAF develop after CABG was an elderly hypertensive patient with renal failure and probable left ventricular diastolic dysfunction should come as no surprise. Advanced age is by far the number-one determinant of POAF in a CABG population,³ and it seems to be at the core of the pathogenetic processes involved in POAF, including loss of myocardial fibers, increased interstitial collagen deposition, and atrial remodeling and enlargement.⁴ To what extent the loss of atrial contractile function occurs independently of age in influencing the risk of POAF after CABG cannot be clearly established by the study of Bening and colleagues.² Statistical interactions and collinearity weaken purported independent associations in multivariate analyses; these were not clearly reported. Bening and colleagues² also identified left atrial size as an independent risk factor for POAF. The clinical

prognostic value of left atrial enlargement for adverse cardiovascular outcomes is not new.⁵ Quantifying left atrial size is difficult, however, and current recommendations of the American Society of Echocardiography⁶ suggest that left atrial volume should be indexed to body surface area as a more accurate predictor of clinical outcome than simple, unindexed 2-dimensional atrial area measurements, as used in the study of Bening and colleagues.²

One interesting and novel finding of the study of Bening and colleagues² was the predictive value of right atrial enlargement and lower tricuspid annular plane systolic excursion in development of POAF. Bening and colleagues² did not define a cutoff reference value of atrial area (either left or right), which may serve as a trigger to consider preoperative preventative pharmacologic therapies to decrease the risk of POAF after CABG.³

The demonstration of decreased responsiveness of atrial myocytes to calcium from excised left and right atrial appendages among patients who had POAF develop after CABG versus those who did not is certainly compelling, because it suggests the presence of a long-standing substrate for the subsequent development of POAF in patients without preexisting AF. The use of this criterion as a risk-stratifying tool to predict POAF after CABG is difficult, however, and even impossible in everyday practice. The

central role of atrial functional remodeling in the pathogenesis of atrial fibrillation is well known.⁷ Left atrial strain analysis and volumetric parameters (eg, left atrial ejection fraction) with advanced echocardiographic or cardiac magnetic resonance imaging techniques are future modalities to explore and may help to identify patients at risk for POAF.

The study of Bening and colleagues² supports the contention that, in addition to the usual clinical risk factors, atrial size measurements and functional assessment may become promising tools in risk stratification and preventive decision making for patients undergoing CABG. Further studies are needed to identify precise cutoff values of left and right atrial size and function adjusted to the patient's age and body surface area in predicting POAF, which may allow the administration of individualized prophylactic measures, including pharmacologic therapy³ or perioperative atrial override pacing.⁸

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