Postoperative Atrial Fibrillation

Incidence, Mechanisms, and Clinical Correlates

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KEYWORDS

• Atrial fibrillation • Postoperative atrial fibrillation • Beta-blockers • Coronary artery bypass grafting

KEY POINTS

- Atrial fibrillation is often encountered after cardiac and noncardiac surgical procedures.
- Atrial fibrillation is associated with an increased hospital stay and stroke risk, and a reduced in-hospital and long-term survival.
- Understanding the underlying pathophysiology of POAF remains elusive; however, numerous risk factors predisposing to its development have been identified, including advanced age, structural damage to the heart, left ventricular dysfunction, hypertension, and valve surgery.
- Further investigation into the mechanisms underlying POAF, and the effects of various therapeutic modalities, will enable a better understanding of this phenomenon.
- Risk stratification, and targeted interventions for high-risk patients, may hold the key to mitigating the morbidity and financial burden associated with this arrhythmia.

INTRODUCTION

Atrial fibrillation (AF) is a commonly encountered arrhythmia in clinical practice, and is a well-recognized complication of cardiac surgery. The occurrence of postoperative AF (POAF) is associated with an increased length of stay, stroke risk, health care costs, and mortality.[1–14] The incidence of POAF varies from 15% to 60%,[3,5–11,15–19] with the highest rates observed in patients undergoing valve surgery (37%–60%).[5,7,17] POAF has also been known to complicate noncardiac surgery, especially esophagectomy, lung surgery, and large colorectal surgery.[13,20–22] The onset of POAF peaks on the second day after surgery and declines to 2% at discharge.[16,23] Although usually self-limiting in nature, the risks of hemodynamic compromise and thromboembolism exist.[4,5,24]

MECHANISM

Although the exact pathophysiology of POAF remains incompletely understood, it is likely multifactorial in cause. Patient-related factors known to contribute include atrial dilatation, age-related fibrosis, structural damage to the heart, hypertension, and other comorbid conditions.[6,18,25] The concept of (structural) predisposition for AF seems

Conflicts of Interest: None reported.
Disclosures: None reported.
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Cardiol Clin 32 (2014) 627–636
http://dx.doi.org/10.1016/j.ccl.2014.07.002

to be true for vulnerability of certain patients to AF after cardiac surgery.[26] The electrophysiologic substrate may be pre-existing or may develop because of heterogeneity of refractoriness after surgery. Furthermore, the role of ectopic beats from the pulmonary veins in the development of POAF, as in nonsurgical patients, is yet to be delineated. This represents an area of significant interest, because such sites may be amenable to isolation at the time of surgery.

Several factors related to the surgical procedure also potentially contribute to the development of AF. These include operative trauma from surgical dissection and manipulation, pericardial lesions (pericarditis), atrial dilatation (caused by left ventricular dysfunction and intraoperative volume overload), perioperative use of catecholamines, parasympathetic activation, and electrolyte imbalances.[3,5,6,16,25,27,28] Current cardioplegia techniques and inadequate atrial cooling may be responsible for atrial ischemia. This has led some to postulate that ischemic injury and subsequent oxidative stress on reperfusion are potential triggers for POAF.[6,29,30] However, there have been conflicting reports regarding the effect of cardiopulmonary bypass time and aortic cross-clamp time on the incidence of the arrhythmia.[5,6,28,31,32]

The time course of development of POAF corresponds with the activation of the complement system, as evidenced by the release of proinflammatory cytokines and an increase in inflammatory markers.[14,33–37] This suggests an inflammatory component to the development of POAF. Inflammation is often related to the development of varying degrees of pericarditis. In support of this theory, some studies have demonstrated a benefit of drugs with anti-inflammatory action, including corticosteroids and statins, in decreasing the incidence of POAF.[38–41]

There seems to be a significant increase in sympathetic tone postoperatively in patients that subsequently develop POAF.[42–45] Withdrawal of preoperative β-blockers and nonuse of perioperative β-blockers are associated with a higher rate of AF,[7,18,46–48] thereby reinforcing the hypothesis of increased sympathetic tone being a facilitating factor in the development of POAF.

EPIDEMIOLOGY

The reported incidence of POAF varies depending on the type of surgery, definition of arrhythmia used, and method of arrhythmia surveillance.[16,17,49] Highest rates have been observed with combined valve surgery and coronary artery bypass grafting (CABG; 62%).[3,26,50] A lower incidence has been observed in patients undergoing isolated CABG (15%–40%),[5,7,50] cardiac transplantation (11%–24%),[5,17,51] and noncardiac surgery (0.3%–13.7%).[13,20–22] The different modalities used to monitor for arrhythmias after surgery also contribute to the variation in reported incidence. When intermittent 12-lead electrocardiograms are used for detection, a rate of 11% has been reported, compared with greater than 40% when diagnosis is based on continuous Holter monitoring.[15,31]

Advanced age has consistently been described as the most significant predictor of developing AF after cardiac and noncardiac surgery.[3,5–7,9,18,28,52,53] Structural changes of the heart with age, such as atrial fibrosis and dilatation, and age-related comorbidities, are likely responsible for this increase in incidence with age.[54,55] Villareal and colleagues[3] found age greater than 65 years to be an independent risk factor for developing POAF after revascularization (odds ratio [OR], 2.4; 95% confidence interval [CI], 2.06–2.74; $P<.0001$). In another study done on 2588 patients, the incidence of POAF in patients undergoing thoracic surgery increased with age as follows: age 50 to 59 years (relative risk [RR], 1.70; 95% CI, 1.01–2.88), age 60 to 69 years (RR, 4.49; 95% CI, 2.79–7.22), age 70 years or greater (RR, 5.30; 95% CI, 3.28–8.59).[53]

Patient-related risk factors for developing POAF also include left ventricular dysfunction and presence of congestive heart failure (CHF), obesity, hypertension, chronic obstructive airways disease, and severe underlying coronary artery disease.[3,6,56,57] The $CHADS_2$ and CHA_2DS_2-VASc scores were also found to be predictive of AF after cardiac surgery.[58]

Several studies have also described a difference in the incidence of POAF based on geographic region and race.[8,49] Higher rates were observed in the Middle East (41.6%), Canada (36.6%), Europe (34%), and United States (33.7%), with relatively lower rates being reported in South America (17.4%) and Asia (15.7%).[8] Whether this divergence represents a racial predisposition of whites toward the development of POAF, comorbid conditions, or a disparity in arrhythmia surveillance and reporting remains unknown. However, racial differences in POAF incidence have previously been described, with white race/ethnicity found to be an independent risk factor for POAF (OR, 1.8; 95% CI, 1.5–2.0; $P<.0001$).[49]

Because structural factors, such as fibrosis, scarring, and dilatation of the atria, predispose to the development of POAF, it has been hypothesized that electrophysiologic measurements before surgery may help predict the development of AF after surgery. The measurement of