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Editorial

Acute AF is a New Name for an Old Condition: An Editorial on Postoperative Atrial Fibrillation

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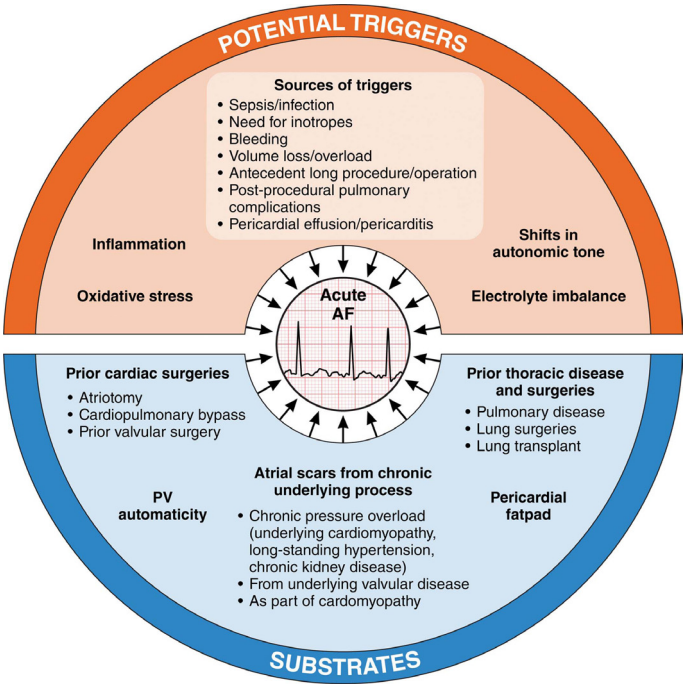
Despite its ubiquity, atrial fibrillation (AF) remains etiologically enigmatic. In fact, it is tempting to want to think of AF as a multitude of conditions, depending on it's the setting, the patient (acute or chronic), its onset (paroxysmal or persistent), and whether it is symptomatic or clinically silent. The American Heart Association (AHA) has recently introduced a new umbrella term to encompass all forms of AF encountered in the acute care setting: "*acute AF*."[1] Formerly called "*secondary AF*," the new terminology recognizes that AF may occur de novo in an acute setting, may be a pre-existing condition, or may emerge due to pre-existing risk factors.

Putting the condition formerly known as postoperative AF (POAF) under the acute AF umbrella may be helpful, because it forces clinicians to consider POAF as AF in the acute setting first, and then as a consequence of a specific surgical intervention second. POAF can occur following both cardiac and noncardiac surgery; POAF complicates recovery and has been associated with higher rates of morbidity[2-5] and mortality.[3-5] Data are alarming. Approximately 32% of coronary artery bypass graft (CABG) surgery patients and 49% of combined CABG and aortic valve replacement and 64% of CABG combined with mitral valve replacement experience acute AF after surgery,[6] which may recur.[7-9] POAF is likely undertreated and often goes underestimated, both quantitatively and qualitatively. As the AHA states in its position paper, "*regardless of the hospital setting, acute AF is not benign.*"[1]

Risk stratification for POAF has always been challenging, in part because there are many heterogeneous factors that have been implicated in raising the risk of POAF, ranging from demographics (age, sex) and genetics to pre-existing hypertension, bleeding, and respiratory complications.[10] The etiology of POAF has previously appeared to vary by type of surgery, causing clinicians to sometimes think of POAF as multiple conditions.

The AHA has devised a framework for all types of acute AF that considers the interplay of two distinct types of factors: physiologic substrates and triggers.[1] See Figure 1.

Figure I. A model of how triggers and substrates can combine and interact to result in acute AF. Courtesy of the American Heart Association.



Substrates that would support acute AF may be atrial scars that are present for any number of reasons: prior cardiac and/or thoracic surgeries, thoracic disease, underlying processes such as cardiomyopathy, pulmonary vein automaticity, and the pericardial fat pad. The triggers that can act upon these substrates are inflammatory processes, oxidative stress, shifts in autonomic tone, or electrolytic imbalances. Such triggers may arise from surgery, infection, or other causes. In this way, this new paradigm of acute AF brings together a "*recipe*" that better explains the range of AF experienced in the acute care setting.

In simple terms, acute AF occurs when a vulnerable substrate encounters one or more triggering factor(s) during an acute hospitalization. This may be a good example of how the principle of Occam's Razor can be applied to medicine. One shortcoming is that the occurrence of acute AF during hospitalization does not provide information helpful in order to differentiate between a pre-existing but not yet diagnosed AF or the emergence of a new arrhythmia.[1] Such differences may not be clinically important for diagnosis and treatment. Indeed, the diagnosis of a pre-existing AF (even if clinically silent) is an important factor in pretreatment of surgical patients,[11] but once POAF occurs, the arrhythmia is officially on the record and can be treated.

Triggers may work alone or in concert. Previous thinking has inclined clinicians to view POAF as a multi-trigger event, but the new acute AF paradigm allows that POAF might commence with one single trigger.[12] Whether or not this occurs in real-world clinical practice, the notion of single-trigger POAF may nevertheless reshape our thinking about POAF. Based on the new AHA paradigm of acute AF, much attention should be given to inflammatory processes. Inflammation has been recognized as playing a role in POAF, which is associated with increases in C-reactive protein (CRP), white blood cell (WBC) counts, and interleukins.[13-16] A study of 110 primary CABG patients found the -174G/C interleukin-6 promoter gene variant played a role in the regulation of postsurgical inflammatory response and this genotype was the only independent predictor of POAF in this study.10 This polymorphism would elevate interleukin-6 (IL-6) levels. And so, this new acute AF definition helps to shed light on possible genetic factors associated with POAF. Such genetic predispositions to POAF have been known but may not have been adequately emphasized.

The high rates of POAF following cardiac-thoracic surgery are likely because such procedures involve a diseased heart, some degree of manual manipulation, elevated pressures, and localized inflammatory response, all of which have been associated with AF. It is thought that POAF after cardiac surgery remits spontaneously when the inflammation and elevated pressures subside following surgery.[17] The new acute AF definition would caution clinicians not to assume the automatic remission of POAF.

The lower rates of POAF after noncardiac surgeries are explained in part by the fact the above conditions (diseased heart, manipulation, high pressures, and localized cardiac inflammation) do not apply to noncardiac surgery. In real-world clinical practice, POAF can and does occur after noncardiac surgery, but other triggers are likely involved, such as oxidative stress, bleeding, loss of volume, respiratory or pulmonary complications, changes in autonomic tone, and an electrolyte imbalance.[17] POAF following noncardiac surgery is likely underdiagnosed because the cardiac activity of such patients is not routinely and consistently monitored.

Another cause of POAF may be hypoxia. Hypoxia can cause atrial myocardial ischemia, which, in turn, can derange the native cardiac conduction pathways and lead to arrhythmias such as AF. Hypoxia may also cause POAF or other arrhythmias by constricting the pulmonary veins, increasing right ventricular pressure and stretch over the right atrium.[11,18]

While it is doubtful that this new definition of "*acute AF*" will eliminate the need for our specific terminology relating to POAF, by grouping together all types of AF in the acute setting, this new paradigm from the AHA has exposed their commonalities-which are considerable. Rather than viewing various risk factors, the new model forces clinicians to consider the role of substrate and trigger and the ways in which surgery can trigger a known or unknown substrate in the patient.

We think the new term "*acute AF*" is an important step toward better understanding POAF, which we argue should lead to more accurate and timely diagnoses and better treatments.

CONFLICTS OF INTEREST

None.

REFERENCES

1. Chyou JY, Barkoudah E, Dukes JW, et al. Atrial fibrillation occurring during acute hospitalization: A Scientific Statement from the American Heart Association. *Circulation*. 2023; 147(15): e676-e698. doi: 10.1161/CIR.0000000000001133

2. Siontis KC, Gersh BJ, Weston SA, et al. Association of New-Onset Atrial Fibrillation after Noncardiac Surgery with Subsequent Stroke and Transient Ischemic Attack. *JAMA*. 2020; 324(9): 871-878. doi: 10.1001/jama.2020.12518

3. Conen D, Alonso-Coello P, Douketis J, et al. (2020) Risk of stroke and other adverse outcomes in patients with perioperative atrial fibrillation 1 year after non-cardiac surgery. *Eur Heart J*. 2020; 41(5): 645-651. doi: 10.1093/eurheartj/ehz431

4. Huynh JT, Healey JS, Um KJ, et al. Association Between Perioperative Atrial Fibrillation and Long-term Risks of Stroke and Death in Noncardiac Surgery: Systematic Review and Meta-analysis. *CJC Open*. 2021; 3(5): 666-674. doi: 10.1016/j.cjco.2020.12.025

5. AlTurki A, Marafi M, Proietti R, et al. Major Adverse Cardiovascular Events Associated With Postoperative Atrial Fibrillation After Noncardiac Surgery: A Systematic Review and Meta-Analysis. *Circ Arrhythm Electrophysiol*. 2020; 13(1): e007437. doi: 10.1161/CIRCEP.119.007437

6. Creswell LL, Schuessler RB, Rosenbloom M, et al. Hazards of postoperative atrial arrhythmias. *Ann Thorac Surg*. 1993; 56(3): 539-549. doi: 10.1016/0003-4975(93)90894-n

7. Abdelmoneim SS, Rosenberg E, Meykler M, et al. The Incidence and Natural Progression of New-Onset Postoperative Atrial Fibrillation. *JACC Clin Electrophysiol*. 2021; 7(9): 1134-1144. doi: 10.1016/j.jacep.2021.02.005

8. Ahlsson A, Fengsrud E, Bodin L, et al. Postoperative atrial fibrillation in patients undergoing aortocoronary bypass surgery carries an eightfold risk of future atrial fibrillation and a doubled cardiovascular mortality. *Eur J Cardiothorac Surg*. 2010; 37(6): 1353-1359. doi: 10.1016/j.ejcts.2009.12.033

9. Melduni RM, Schaff HV, Bailey KR, et al. Implications of new-onset

atrial fibrillation after cardiac surgery on long-term prognosis: A community-based study. *Am Heart J*. 2015; 170(4): 659-668. doi: 10.1016/j.ahj.2015.06.015

10. Gaudino M, Andreotti F, Zamparelli R, et al. The -174G/C interleukin-6 polymorphism influences postoperative interleukin-6 levels and postoperative atrial fibrillation. Is atrial fibrillation an inflammatory complication? *Circulation*. 2003; 108 Suppl 1: 195-199. doi: 10.1161/01.cir.0000087441.48566.0d

11. Bessissow A, Khan J, Devereaux PJ, et al. Postoperative atrial fibrillation in non-cardiac and cardiac surgery: An overview. *J Thromb Haemost*. 2015; 13 Suppl 1: S304-S312. doi: 10.1111/jth.12974

12. Elahi MM, Flatman S, Matata BM. Tracing the origins of postoperative atrial fibrillation: the concept of oxidative stress-mediated myocardial injury phenomenon. *Eur J Cardiovasc Prev Rehabil*. 2008; 15(6): 735-741. doi: 10.1097/HJR.0b013e328317f38a

13. Abdelhadi RH, Gurm HS, Van Wagoner DR, et al. Relation of an exaggerated rise in white blood cells after coronary bypass or cardiac valve surgery to development of atrial fibrillation postoperatively. *Am J Cardiol*. 2004; 93(9): 1176-1178. doi: 10.1016/j.amjcard.2004.01.053

14. Amar D, Goenka A, Zhang H, et al. Leukocytosis and increased risk of atrial fibrillation after general thoracic surgery. *Ann Thorac Surg*. 2006; 82(3): 1057-1061. doi: 10.1016/j.athoracsur.2006.03.103

15. Bruins P, te Velthuis H, Yazdanbakhsh AP, et al. Activation of the complement system during and after cardiopulmonary bypass surgery: Postsurgery activation involves C-reactive protein and is associated with postoperative arrhythmia. *Circulation*. 1997; 96(10): 3542-3548. doi: 10.1161/01.cir.96.10.3542

16. Fontes ML, Amar D, Kulak A, et al. Increased preoperative white blood cell count predicts postoperative atrial fibrillation after coronary artery bypass surgery. *J Cardiothorac Vasc Anesth*. 2009; 23(4): 484-487. doi: 10.1053/j.jvca.2009.01.030

17. Vallurupalli S, Shanbhag A, Mehta JL. Controversies in postoperative atrial fibrillation after noncardiothoracic surgery: clinical and research implications. *Clin Cardiol*. 2017; 40(5): 329-332. doi: 10.1002/clc.22652

18. Danelich IM, Lose JM, Wright SS, et al. Practical management of postoperative atrial fibrillation after noncardiac surgery. *J Am Coll Surg*. 2014; 219(4): 831-841. doi: 10.1016/j.jamcollsurg.2014.02.038