Interatrial block is an independent risk factor for new-onset atrial fibrillation after cardiac surgery

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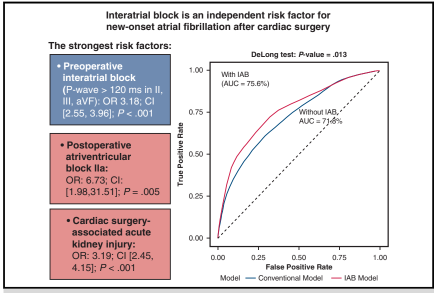
ABSTRACT

Objectives: This study aims to investigate the association between interatrial conduction block and postoperative atrial fibrillation, which can precipitate acute cardiopulmonary instability and is associated with subsequent heart failure, stroke, and mortality following cardiac surgery.

Methods: Perioperative 12-channel electrocardiograms from 3405 patients undergoing myocardial revascularization, valve surgery, aortic surgery, or combinations thereof, were considered. Clinical and electrographic parameters were compared between patients with and without atrial fibrillation, and significant variables were analyzed using univariate and multivariate logistic regression.

Results: Among 2108 analyzed patients, 764 (36.2%) developed atrial fibrillation. Preoperative interatrial block was a strong independent risk factor (3.18; 95% CI, 2.55, 3.96; $P < .001$), significantly improving area under the receiver operator characteristics curve from 71.8% to 75.6% (Delong's test: $P = .013$). Other risk factors included advanced age (1.05; 95% CI, 1.03, 1.07; $P < .001$), female gender (1.86; 95% CI, 1.45, 2.38; $P < .001$), history of cardiogenic shock (1.44; 95% CI, 0.99, 2.09; $P = .057$), reduced left ventricular ejection fraction <40% (1.57; 95% CI, 1.06, 2.33; $P = .024$), cessation of preoperative β-blockers (1.17; 95% CI, 0.95, 1.46; $P = .145$), score for clinical prediction rules for estimating the risk of stroke in people with non-rheumatic atrial fibrillation ($CHAS_2DS_2$-VASc) and European System for Cardiac Operative Risk Evaluation II score (0.87; 95% CI, 0.79, 0.97; $P = .01$) and (1.04; 95% CI, 0.99, 1.11; $P = .138$), preexisting left bundle branch block (1.59; 95% CI, 0.92, 2.74; $P = .097$), cardiopulmonary bypass time (1.00; 95% CI, 1.00, 1.00; $P = .049$), bicaval cannulation (1.45; 95% CI, 0.88, 2.41; $P = .035$), cardiac surgery-associated acute kidney injury (3.19; 95% CI, 2.45, 4.15; $P < .001$), and postoperative atrioventricular block (1.20; 95% CI, 0.96, 1.51; $P = .105$), particularly Mobitz I (6.73; 95% CI, 1.98, 31.51; $P = .005$).

Conclusions: Perioperative electrocardiogram-derived parameters, especially interatrial block, are associated with postoperative atrial fibrillation. Further research is needed to clarify the link between conduction abnormalities and postoperative atrial fibrillation, enabling targeted prophylactic therapies for high-risk patients. (JTCVS Open 2024;22:345-53)



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CENTRAL MESSAGE

Risk stratification of postoperative atrial fibrillation can be augmented by inclusion of easily obtainable ECG-based parameters, including interatrial block.

PERSPECTIVE

The integration of ECG-derived parameters, particularly interatrial block, improves risk assessment of postoperative atrial fibrillation after heart surgery. Further research is needed to elucidate the association between conduction abnormalities and postoperative atrial fibrillation to facilitate specific delivery of targeted prophylactic therapies in high-risk patients.

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Abbreviations and Acronyms

AF	= atrial fibrillation
AUC	= area under the curve
CPB	= cardiopulmonary bypass
CSA-AKI	= cardiac surgery associated acute kidney injury
ECG	= electrocardiogram
IAB	= interatrial block
MVR	= mitral valve replacement
POAF	= postoperative atrial fibrillation

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Postoperative atrial fibrillation (POAF) is frequent complication after cardiac surgery, affecting 15% to 50% of patients.[1-4] Despite often being asymptomatic and brief, it correlates with adverse outcomes like chronic atrial fibrillation (AF), stroke, heart failure, hospitalizations, and increased health care costs.[1,5-7]

Persistent POAF necessitates prolonged anticoagulation and antiarrhythmic therapy, carrying risks of bleeding, stroke, arrhythmias, or thyroid dysfunction.[2,8] Prophylactic surgical interventions like left atrial appendage closure, as adjunctive therapy in patients at high risk for POAF, showed controversial results, incur financial costs, and might cause complications deterring routine use.[1,8-10]

Cardiac surgery presents a distinct scenario that increases the risk of POAF by causing injury to atrial myocardium, such as through manipulation during valve surgeries or inflammation and ischemia.[1,3] This injury induces conduction abnormalities and cardiac autonomic derangement, promoting dynamic re-entry and POAF.[1,7,11]

Changes in the P-wave, especially interatrial conduction block (IAB) has been related to higher incidence of AF in various settings, reflecting atrial structural changes like fibrosis and remodeling.[11-17] To date, 1 study investigated the link between IAB and POAF after cardiac surgery.[18] The aim of this study was to identify significant risk factors for POAF during the perioperative period, including electrocardiograph (ECG) changes such as IAB.

METHODS

Study Population

This retrospective observational study analyzed prospectively collected data from patients undergoing elective cardiac surgery at the Department of Cardiac Surgery at Klinikum Nürnberg-Paracelsus Medical University (Nuremberg, Germany), from January 2015 to January 2021. Patients underwent aortic valve replacement, coronary artery bypass grafting, mitral valve replacement (MVR) or repair, tricuspid valve replacement or repair, ascending aorta replacement, or combinations thereof. Subgroup analysis included MVR/repair combined with any other procedure, aortic valve replacement combined with any other procedure apart from MVR/repair, and aortic surgery (isolated/or combined with any other procedure). Inclusion criteria included patients aged 18 years or older, and sinus rhythm before surgery. Exclusion criteria were preexisting AF, missing preoperative and/or postoperative ECG, amiodarone use within the past 12 months, advanced atrioventricular blocks (IIb and III), pacemaker rhythm, emergency surgery, endocarditis, off-pump surgery, and prior cardiac surgery (Figure 1).

Definitions

POAF was defined as occurrence of an irregular heart rhythm, without detectable P-waves, lasting more than 30 seconds.[19] IAB was defined as P-wave duration $\geq$120 msec in the inferior leads (II, III, augmented vector foot) and classified according to the P-wave shape (Figure 2).[16,17] Preexisting AF was defined as any documented episode of AF before surgery either in ECG or in the patient's records. Pulmonary hypertension was defined as ECG-estimated pulmonary artery systolic pressure >35 mm Hg. Cardiogenic shock was defined as condition with signs of end-organ hypoperfusion and/or hypotension.[20] Cardiac surgery-associated kidney injury (CSA-AKI) was defined as increase in creatinine level by $\geq$0.3 mg/dL from baseline within 48 hours or as increase to $\geq$1.5 times baseline within 7 days postoperatively.[21,22]

Ethical Statement

The study complied with the Declaration of Helsinki, Finland, and the Oviedo Convention, Asturias, Spain. The institutional review board of Paracelsus Medical University approved the study protocol with a registry No. IRB-2020-028 approved on January 5, 2021. Due to retrospective nature of the study and complete anonymization no informed consent was obtained.

Data Collection

Medical records of 3405 patients were reviewed. Preoperative ECGs of the patients were examined for arrhythmias and conduction abnormalities. Patients were followed throughout their clinical stay and documents, including ECGs, were screened for arrhythmic events. Scanned ECG recordings were analyzed by 5 independent examiners with medical degree. Preoperatively, at least 1 12-lead ECG was recorded 1 day before surgery. Postoperatively, all ECGs until discharge were analyzed. Patients were continuously monitored for the first 4 postoperative days and received at least 1 ECG per day for the first 5 days. At clinical suspicion for POAF, additional 12-lead ECG was recorded.

Statistical Analysis

Categorical variables were presented as frequencies/percentages, and scalar variables as means (SD). Patients' characteristics with/without POAF were compared using exact Fisher or χ^2 test for categorical and Student *t* test for scalar variables. Logistic regression was performed to assess the effect on POAF, reporting odds ratios (OR) with 95% CI and *P* values. Hazard ratios of cardiopulmonary bypass (CPB) time refers to change of 10 minutes for better readability. Multivariate regression considered correlations among variables, selecting those with $P < .1$ using a stepwise-forward approach with Akaike information criteria. Two models were constructed: a model with conventional risk factors and IAB model incorporating preoperative IAB. For training and testing we applied, bootstrapping (100 draws) to mitigate overfitting and achieve more robust results.[23] Receiver operating characteristic curves and areas under the curves (AUC) were calculated and DeLong's test was used for comparison. Calibration was assessed with Hosmer-Lemeshow test. Statistical analysis was conducted using R version 4.0 (R Foundation for Statistical Computing).

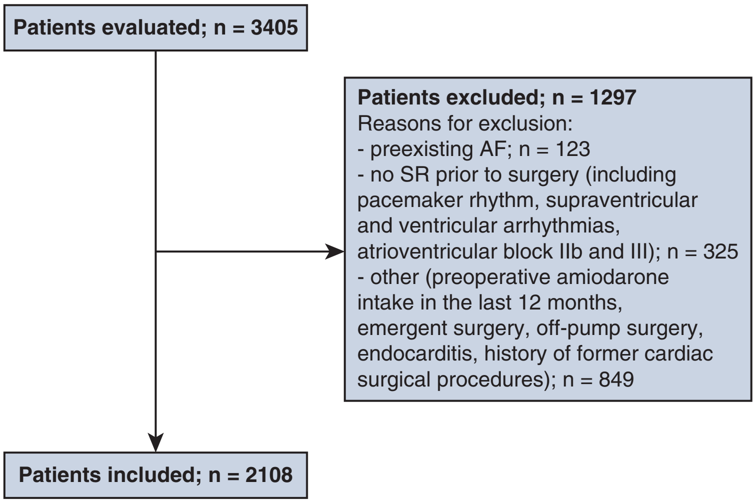


FIGURE 1. The flow chart displays exclusion process of the patients. *AF*, Atrial fibrillation; *SR*, sinus rhythm.

RESULTS

Baseline Characteristics and Clinical Parameters

Out of 3405 patients, 2108 patients were amenable to analysis (Figure 1) and 764 (36.2%) developed POAF. Recurrences occurred in 365 (17.3%) cases and 48 (2.3%) patients were discharged in AF. During hospital stay, 47 (2.2%) patients died. Patients in POAF group were significantly older (69.8 ± 8.5 years vs 65.2 ± 10.1 years; $P < .001$). POAF was more common among women 248 (32.4%) versus 349 (26.0%); $P = .002$. Preoperative and postoperative IAB, including all subtypes were more common in the POAF group (Table 1).

In patients with newly introduced β-blocker postoperatively, the POAF incidence was lower: 32.8% (317 out of 966 cases) compared with 39.1% (447 out of 1142 patients); $P = .003$. For patients with discontinued β-blockers perioperatively, there was no significant difference in POAF: 35.9% (418 out of 1163) compared with 36.6% (346 out of 945 patients); $P = .784$.

Regression Analysis and Calibration

In multivariable stepwise regression, 2 models were constructed. In terms of influence, the following variables had the highest ORs: preoperative IAB (OR, 3.18; 95% CI,

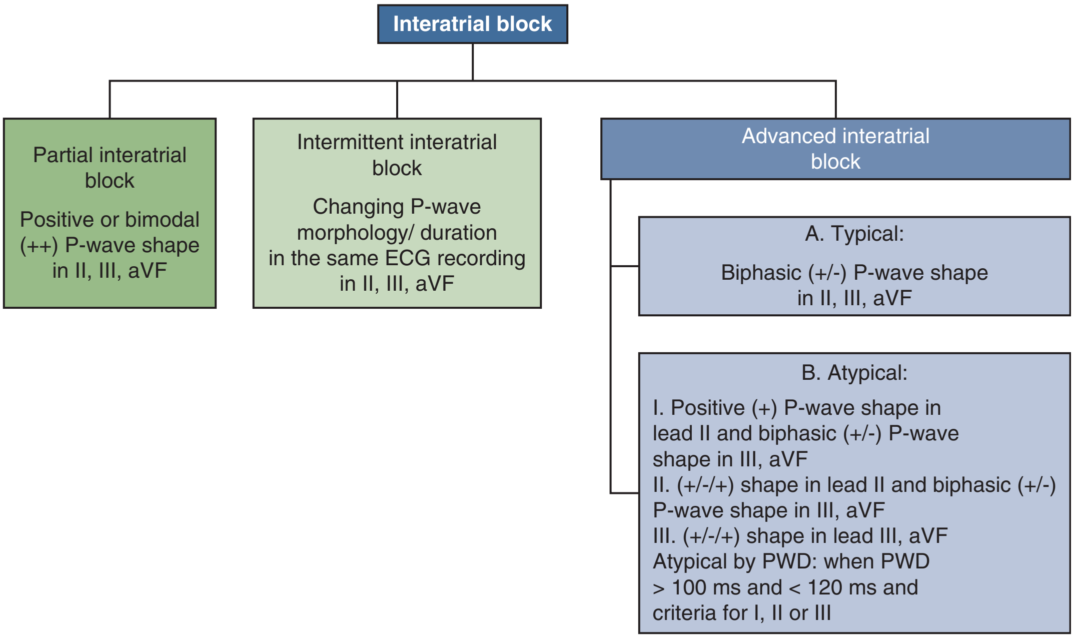


FIGURE 2. Interatrial block subtypes according to Bayes de Luna.[17] *aVF*, Augmented vector foot; *ECG*, electrocardiogram; *PWD*, P-wave duration.

TABLE 1. Baseline characteristics and clinical parameters

Baseline characteristics	POAF group (n = 764)	SR group (n = 1344)	*P* value	Univariate analysis Odds ratio (95% CI)	*P* value
Preoperative characteristics					
Age (y)	69.8 ± 8.5	65.2 ± 10.1	<.001	1.06 (1.05-1.07)	<.001
Female sex	248 (32.5)	349 (26.0)	.002	1.37 (1.13-1.66)	.001
BMI	27.9 ± 4.7	28.3 ± 7.0	.114	0.99 (0.97-1.00)	.157
Arterial hypertension	697 (91.2)	1183 (88.0)	.022	1.44 (1.07-1.95)	.018
History of smoking	338 (44.2)	673 (50.1)	.004	0.79 (0.66-0.95)	.010
Pulmonary hypertension	57 (7.5)	44 (3.3)	<.001	2.37 (1.59-3.57)	<.001
Hyperlipidemia	568 (74.3)	103 (76.9)	.217	0.87 (0.71-1.07)	.198
CHA_2DS_2-VASc	3.4 ± 1.5	2.9 ± 1.5	<.001	1.24 (1.16-1.31)	<.001
NYHA functional class III or IV	2.5 ± 0.8	2.4 ± 0.8	<.001	1.22 (1.09-1.37)	<.001
COPD GOLD (3-4)	104 (13.6)	142 (10.6)	.043	1.33 (1.02-1.75)	.037
LVEF	55.5 ± 10.9	56.6 ± 10.3	.016	0.99 (0.98-1.00)	.014
LVEF <40%	82 (10.7)	78 (5.8)	<.001	1.78 (1.30-2.43)	<.001
History of stroke	45 (5.9)	103 (7.6)	.155	0.76 (0.52-1.08)	.132
Vascular disease	416 (54.5)	730 (54.3)	.963	1.01 (0.84-1.21)	.927
EuroScore II	3.4 ± 3.2	2.5 ± 1.9	<.001	1.16 (1.12-1.21)	<.001
Diabetes mellitus (II)	218 (28.5)	391 (29.1)	.824	0.97 (0.80-1.18)	.786
History of cardiogenic shock	104 (13.6)	87 (6.5)	<.001	2.28 (1.69-3.08)	<.001
Bicuspid aortic valve	90 (11.8)	215 (16.0)	.010	0.69 (0.52-0.91)	.008
β-Blockers cessation	394 (51.6)	626 (46.6)	.031	1.22 (1.02-1.46)	.028
Statins	403 (52.7)	733 (54.5)	.455	0.93 (0.78-1.11)	.428
Calcium antagonist	176 (23.0)	300 (22.3)	.746	1.04 (0.84-1.29)	.706
Aldosterone antagonists	60 (7.9)	65 (4.8)	.006	1.68 (1.16-2.41)	.005
ARBs	204 (26.7)	336 (25.0)	.419	1.09 (0.89-1.34)	.390
ACE inhibitors	269 (35.2)	488 (36.3)	.646	0.95 (0.79-1.15)	.613
Creatinine (>1.4 mg/dL)	74 (9.7)	69 (5.1)	<.001	2.00 (1.42; 2.81)	<.001
AVB I and IIa	124 (16.2)	137 (10.2)	<.001	1.71 (1.32-2.22)	<.001
RBBB	41 (5.0)	68 (5.1)	.828	1.07 (0.71-1.58)	.749
LBBB	41 (5.4)	37 (2.8)	.003	2.01 (1.28-3.17)	.003
IAB of any type	413 (54.1)	349 (26.0)	<.001	3.35 (2.78-4.04)	<.001
Partial IAB	232 (30.4)	228 (17.0)	<.001	2.13 (1.73-2.63)	<.001
Intermittent IAB	92 (12.0)	70 (5.2)	<.001	2.49 (1.80-3.45)	<.001
Advanced IAB	87 (11.4)	54 (4.0)	<.001	3.07 (2.17- 4.39)	<.001
Intraoperative characteristics					
CPB time* (min)	103.1 ± 53.3	95.2 ± 54.4	.001	1.04 (1.02-1.07)	.003
Aorta clamping time (min)	66.42 ± 29.5	61.5 ± 32.1	<.001	1.01 (1.00-1.01)	<.001
Bicaval cannulation	109 (14.3)	84 (6.3)	<.001	2.48 (1.84-3.36)	<.001
Blood cardioplegia	702 (91.9)	1248 (92.9)	.529	0.89 (0.64-1.24)	.474
Crystalloid cardioplegia	55 (7.2)	86 (6.4)	.533	1.14 (0.80-1.61)	.475
Isolated AVR	195 (25.5)	327 (24.3)	.577	1.07 (0.87-1.31)	.542
Isolated MVR/MVr	53 (6.9)	33 (2.5)	<.001	2.96 (1.91-4.66)	<.001
Isolated CABG	246 (32.2)	603 (44.9)	<.001	0.58 (0.48-0.70)	<.001
Combined AVR procedures	151 (19.8)	222 (16.5)	.069	1.25 (0.99-1.57)	.056
Combined MVR/MVr procedures	56 (7.3)	48 (3.6)	<.001	2.10 (1.41-3.15)	<.001
Aortic surgery	66 (8.6)	113 (8.4)	.919	1.04 (0.75-1.42)	.812
Postoperative characteristics					
IAB of any type	307 (40.2)	283 (21.1)	<.001	2.52 (2.07-3.06)	<.001
Partial IAB	153 (20.0)	144 (10.7)	<.001	2.09 (1.63-2.68)	<.001
Intermittent IAB	55 (7.2)	59 (4.4)	.008	1.69 (1.16-2.47)	.007
Advanced IAB	112 (14.7)	92 (6.8)	<.001	2.34 (1.75-3.14)	<.001
IAB stage new or increase	131 (17.1)	166 (12.4)	.003	1.47 (1.15-1.88)	.002
IAB stage decrease	26 (3.4)	14 (1.0)	<.001	3.35 (1.77-6.63)	<.001
AVB	311 ± 40.7	380 ± 28.3	<.001	1.75 (1.45-2.10)	<.001

(Continued)

TABLE 1. Continued

Baseline characteristics	POAF group (n = 764)	SR group (n = 1344)	*P* value	Univariate analysis Odds ratio (95% CI)	*P* value
AVB I	278 ± 36.4	355 ± 26.4	<.001	1.60 (1.32-1.94)	<.001
AVB IIa	14 ± 1.	3 ± 0.2	<.001	8.37 (2.72-36.38)	<.001
AVB IIb	4 (0.5)	5 (0.4)	.867	1.41 (0.35-5.35)	.608
AVB III	45 (5.9)	36 (2.7)	<.001	2.28 (1.46-3.59)	<.001
CSA-AKI	244 (31.9)	149 (11.2)	<.001	3.76 (3.00-4.74)	<.001

Values are presented as mean ± SD or n (%). *POAF*, Postoperative atrial fibrillation; *SR*, sinus rhythm; *BMI*, body mass index; *$CHAS_2DS_2$-VASc*, score for estimating the risk of stroke in people with non-rheumatic atrial fibrillation; *NYHA*, New York Heart Association; *COPD*, chronic obstructive pulmonary disease; *GOLD*, global initiative for chronic obstructive lung disease guidelines classification; *LVEF*, left ventricular ejection fraction; *EuroScore II,* European System for Cardiac Operative Risk Evaluation II; *ARBs*, angiotensin receptor blockers; *ACE inhibitors*, angiotensin-converting-enzyme inhibitors; *AVB*, atrioventricular block; *RBBB*, right bundle branch block; *LBBB*, left bundle branch block; *IAB*, interatrial block; *CPB*, cardiopulmonary bypass; *AVR*, aortic valve replacement; *MVR*, mitral valve replacement; *MVr*, mitral valve repair; *CABG*, coronary artery bypass grafting; *CSA-AKI*, cardic surgery associated acute kidney injury, defined as increase in creatinine level by ≥0.3 mg/dL from baseline within 48 hours postoperatively or as increase in creatinine to ≥1.5 times baseline within 7 days after surgery. *Hazard ratio refers to a change of 10 minutes of CPB time.

2.55-3.96; *P* < .001), CSA-AKI (OR, 3.19; 95% CI, 2.45-4.15; *P* < .001) and postoperative AVB IIa (OR, 6.73; 95% CI, 1.98-31.51; *P* = .005). A model with conventional risk factors yielded an AUC of 71.8%, whereas adding IAB to the model improved the AUC to 75.6% (Table 2 and Figure 3), DeLong test *P* = .013. The calibration curves (Figure 4) revealed a fair agreement between the observed and expected rates of POAF in both test (*P* = .483) and training sets (*P* = .478) in a conventional and IAB model; *P* = .507 versus *P* = .497 (Table 3). IAB model was better calibrated than the conventional model for the part where most of the data samples are found.

TABLE 2. Multivariable analysis for conventional model and conventional model with interatrial block (IAB)

Clinical parameters	Stepwise analysis Odds ratio (95% CI)	*P* value	Stepwise analysis with IAB Odds ratio (95% CI)	*P* value
Preexisting IAB			3.18 (2.55-3.96)	<.001
CSA-AKI	3.13 (2.43- 4.04)	<.001	3.19 (2.45-4.15)	<.001
Age	1.06 (1.04-1.07)	<.001	1.05 (1.03-1.07)	<.001
History of cardiogenic shock	1.52 (1.05-2.20)	.025	1.44 (0.99-2.09)	.057
Postoperative AVB IIa	7.01 (2.12-31.92)	.004	6.73 (1.98-31.51)	.005
CPB time	1.00 (1.00-1.01)	.022	1.00 (1.00-1.00)	.049
Postoperative AVB of any type	1.41 (1.14-1.75)	.002	1.20 (0.96-1.51)	.105
Female sex	1.53 (1.21 1.93)	<.001	1.86 (1.45-2.38)	<.001
LVEF <40%	1.53 (1.05-2.25)	.028	1.57 (1.06-2.33)	.024
CHA_2DS_2-VASc	0.87 (0.79-0.96)	.008	0.87 (0.79-0.97)	.010
EuroScore II	1.06 (1.00-1.12)	.053	1.04 (0.99-1.11)	.138
Preexisting LBBB	1.57 (0.93-2.65)	.090	1.59 (0.92-2.74)	.097
Bicaval cannulation	1.53 (0.93-2.51)	.092	1.45 (0.88-2.41)	.145
β-Blockers cessation	1.18 (0.96-1.46)	.188	1.17 (0.95-1.46)	.145
AUC	0.718		0.756	
Accuracy	0.702		0.736	
Accuracy lower	0.653		0.688	
Accuracy upper	0.747		0.779	
Sensitivity	0.381		0.486	
Specificity	0.881		0.875	

CSA-AKI, Cardic surgery associated acute kidney injury, defined as increase in creatinine level by ≥0.3 mg/dL from baseline within 48 hours postoperatively or as increase in creatinine to ≥1.5 times baseline within 7 days after surgery; *AVB*, atrioventricular block; *CPB*, cardiopulmonary bypass; *LVEF*, left ventricular ejection fraction; *$CHAS_2DS_2$-VASc* score, clinical prediction rules for estimating the risk of stroke in people with non-rheumatic atrial fibrillation; *EuroScore II*, European System for Cardiac Operative Risk Evaluation II; *LBBB*, left bundle branch block; *AUC*, area under the receiving operating characteristic curve.

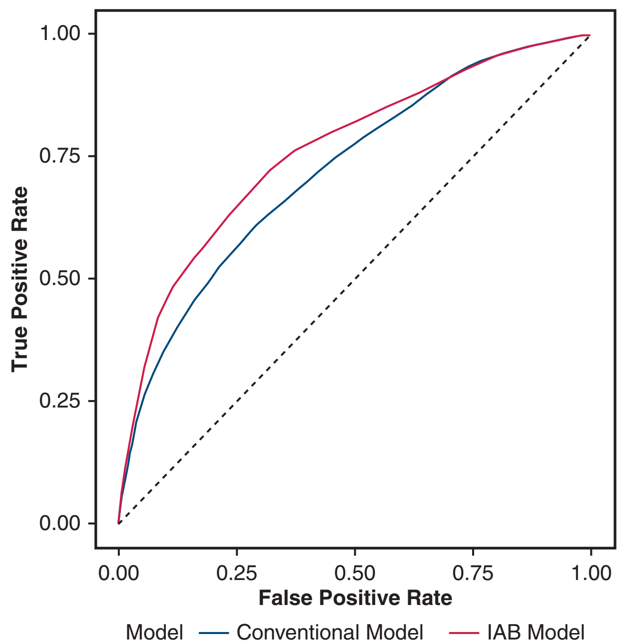


FIGURE 3. Receiver under the operating characteristic curve for conventional and interatrial block model. *IAB*, Interatrial block.

DISCUSSION

POAF rate in this mixed surgery cohort is 36.2%, which is consistent with prior studies.[1,3,5,11] Perioperative IAB and its subtypes were associated to a higher incidence of POAF. Moreover, preoperative IAB emerged as a strong independent risk factor of POAF with OR, 3.18; 95% CI, 2.55-3.96; $P < .001$ and enhanced accuracy when added to the conventional model (AUC, 71.8% vs 75.6%; DeLong $P = .013$).

IAB is associated with atrial tachyarrhythmias and mirrors atrial structural changes akin to those in AF, arising from Bachmann bundle blockage,[15,17,24] intra-atrial conduction delay, or left atrial enlargement.[14] Cellular-level changes in IAB include atrial remodeling, edema, and fibroblast proliferation,[15] whereas proposed pathophysiological mechanisms include inflammation, ischemia, and degeneneration.[25] Although patients with preexisting IAB exhibit higher POAF incidence,[18] its role in POAF is not fully understood.[25] Regardless of the inability to link the observations with the underlying structural alterations predisposing to POAF,[1,3,25] our study demonstrates that the properties of atrial substrate, reflected in ECG as IAB allude to preoperative electromorphological sinus rhythm deterioration potentially facilitating POAF onset.

Although the role of left bundle branch block in POAF genesis remains contentious, this study suggests a positive association between AF and left bundle branch block[26,27] (OR, 1.59; 95% CI, 0.92-2.74; $P = .097$). Similarly, previous research indicates positive correlation between atrioventricular block, especially atrioventricular block II, and AF.[28] atrioventricular block IIa was a significant risk factor of POAF (OR, 6.73; 95% CI, 1.98-31.51) in this study. Conduction disorders indicate atrial fibrosis and degeneration, disrupting myocardial fiber connections and slowing electrical conduction that promotes abnormal conduction and arrhythmias due to differing electrical properties between fibrotic and normal cardiomyocytes.[29] Although conduction disorders and AF share anatomical substrates, their direct causal association remains unclear.[17,26]

Age is a major risk factor of POAF[1,3,8,29] (OR, 1.05; 95% CI, 1.03-07; $P < .001$) linked to fibrosis and collagen buildup making atria more prone to surgical stresses like inflammation, ischemia, hypovolemia, and autonomic nervous system hyperactivation.[3,29] Indeed, age, PR-interval (subcomponent of IAB definition), and cardiac autonomic derangement were the strongest independent POAF risk predictors with comparable AUC in 1 of our previous prospective studies.[11]

Along with others, reduced left ventricular rejection fraction <40% carries a high risk for POAF (OR, 1.57; 95% CI, 1.06-2.33; $P = .024$).[29,30] The pathophysiological mechanisms linking heart failure to POAF involve atrial remodeling, neurohormonal activation, and oxidative stress, creating a substrate for the initiation and maintenance of AF.[1,3] Additionally, hemodynamic alterations in heart failure, such as atrial stretch and elevated filling pressures, further predispose to (PO)AF.[3]

Controversially, female gender was a risk factor in this study.[1,3,4] Higher risk for advanced atrial remodeling, predisposition for valvular disease, hormonal fluctuations, and electrophysiological changes could influence this finding.[31,32]

Cessation of preoperative β-blocker use was associated with a higher risk of POAF (OR, 1.17; 95% CI, 0.95-1.46; $P = .145$). In patients with new β-blockers postoperatively, the incidence of POAF was significantly lower. Patients with other medical conditions predisposing them to POAF, such as heart failure, were more frequently prescribed β-blockers preoperatively.[8] Given β-blockers represent currently 1 of the most effective preventive therapies,[1,33] discontinuation of β-blockers perioperatively significantly increases the risk of POAF, highlighting the importance of early reintroduction of β-blockers to mitigate excessive sympathetic nervous system activation after surgery.[33,34]

The CHA_2DS_2-VASc score (a score for clinical prediction rules for estimating the risk of stroke in people with nonrheumatic atrial fibrillation) emerged as another risk factor[35] with a seemingly protective effect (OR, 0.87; 95% CI, 0.79-0.97; $P = .01$). This unanticipated observation may stem from the score's composite nature, originally designed to inform antithrombotic therapy decisions in patients with chronic AF and excludes other pertinent surgery-related risk factors.[4]

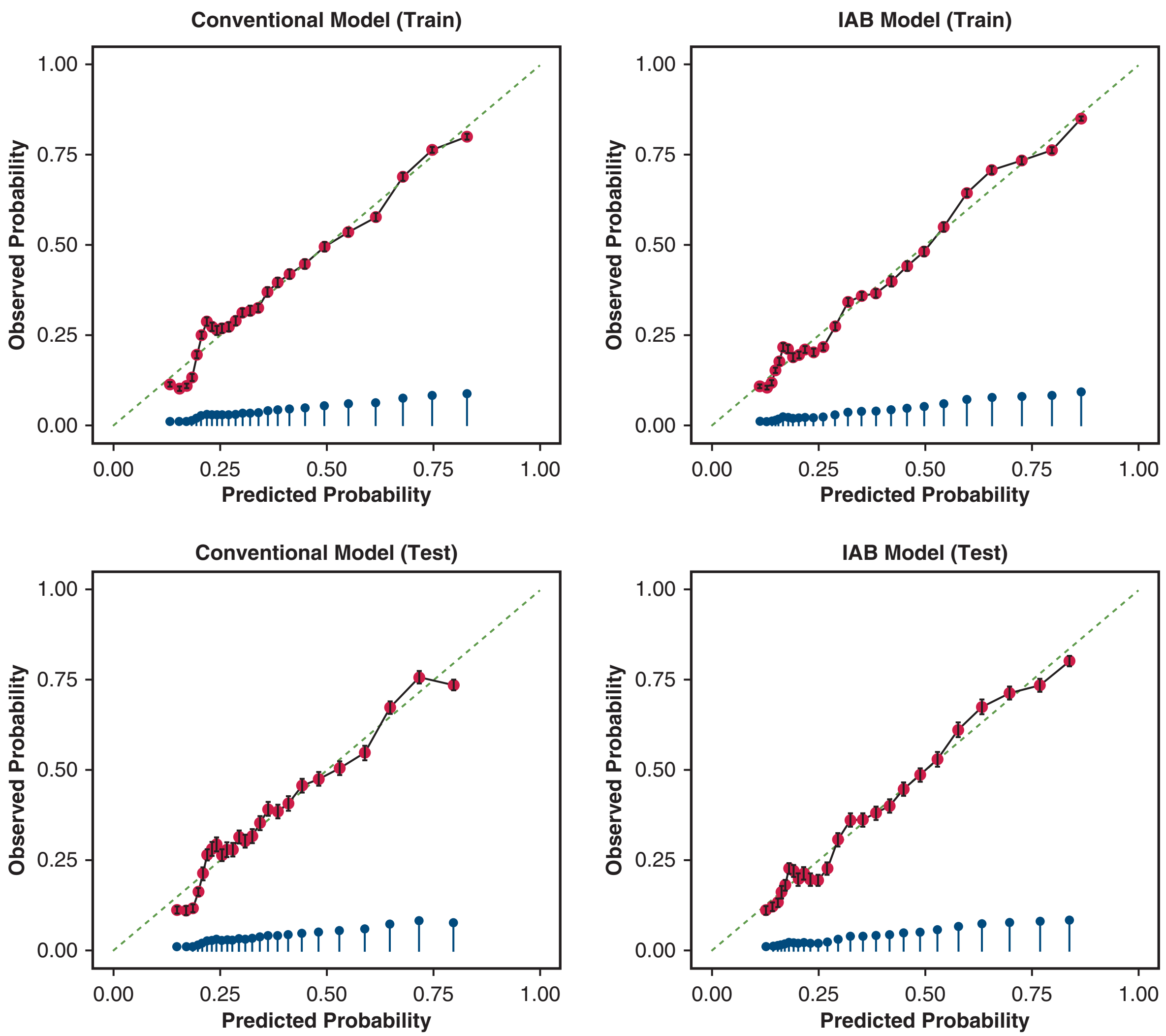


FIGURE 4. Calibration curves for test and train data sets for conventional and interatrial block (*IAB*) model with corresponding lollipop bars representing the distribution of the samples.

Prolonged CPB may sustain atrial electrical activity aggravated by vasoplegic syndrome-induced ischemia leading to induction of arrhythmias, including POAF.[1,36,37] Although the cannulation of the atrium and CPB per se were not associated with higher incidence of POAF in the large, randomized Five-Year Outcomes after On-Pump and Off-Pump Coronary-Artery Bypass (ROOBY) trial,[38] prolonged CPB was associated with higher incidence of POAF in this study. Suggested mechanisms include neurohormonal and inflammatory activation, heightening sympathetic tone, shortening atrial refractory periods, altogether increasing susceptibility for POAF.[1,3,4] CSA-AKI increases POAF risk in this study (OR, 3.19; 95% CI, 2.45-4.15; $P < .001$), likely due to shared inflammation pathways.

TABLE 3. Calibration with Hosmer-Lemeshow tests and results of linear regression analysis of observed versus expected calibration plot*

	Training set			Test set		
Univariate analysis	***P* value**	**Slope**	**Intercept**	***P* value**	**Slope**	**Intercept**
Conventional model	.478 (.426-.530)	1.00 (1.00-1.00)	−0.00 (−0.00 to −0.00)	.483 (.430-.536)	0.95 (0.92-0.97)	0.01 (0.01-0.02)
IAB model	.497 (.439-.555)	4.91 (4.90-4.92)	−2.44 (−2.44 to −2.43)	.507 (.445-.569)	4.52 (4.39-4.64)	−2.28 (−2.32 to −2.23)

Higher *P* values-slope approaching 1 and intercept approaching 0 indicate better calibration. *IAB*, Interatrial block. *Slopes and intercepts from linear regression analysis with corresponding 95% CI.

Older patients and those with chronic kidney disease are prone to CSA-AKI from inflammation, cytokine imbalance, and oxidative stress.[3]

Overall, the results of our study are consistent with similar risk estimation models based on perioperative risk factors.[3,4,11,13,24] This study recognises various perioperative risk factors, including ECG parameters. Further research is needed to estimate their significance and create an accurate prediction model for POAF.

The primary clinical relevance of accurately predicting POAF lies in the potential to refine prophylactic strategies. An effective prediction model would enable clinicians to identify high-risk patients who would benefit most from prophylactic interventions, thus allowing for the meticulous application of treatment only in those with a high probability of developing POAF. Rate control agents could be used prophylactically to prevent re-entry and tachycardia onset.[39] The choice of the agent should depend on comorbidities, and the potential for side effects and interactions. Further options such as amiodarone, anti-inflammatory therapies (like colchicine), can be strategically employed based on the refined risk assessment provided by the model.[2,40] This selective approach minimizes unnecessary exposure to the risks associated with prophylactic therapies, which may have adverse effects, especially in intermediate or low-risk patients.

Limitations of the Study

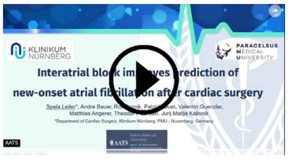
This retrospective single-center study acknowledges inherent limitations. External validation is mandatory. Further limitations include nondetermination of left atrium size, (other) ECG parameters, and consistently defined heart failure or postoperative complications (eg, exact timing of β-blocker withdrawal and reintroduction, timeframe for catecholamine need, pericardial or pleural effusion, potassium levels, and medication during the early postoperative period).[1,8,30] Moreover, absence of continuous ECG monitoring until discharge may have led to underestimation of asymptomatic POAF episodes, highlighting the need for cautious interpretation.[4,11] To understand the relevance of specific risk factors recognized in this study, further in-depth analyses are necessary to separate between confounding factors such as CPB use and duration and complex procedures (eg, coronary artery bypass grafting + MVR).

CONCLUSIONS

Prophylactic therapy for POAF should be tailored to individual patients due to the variability in side effects and efficacy of treatments. An effective risk assessment model, incorporating ECG parameters and intraoperative and postoperative risk factors, can enhance this individualization. Further research is required to explore the relationship between conduction disorders, such as IAB, and POAF, and to enhance prophylactic treatment strategies.

Webcast

You can watch a Webcast of this AATS meeting presentation by going to: https://www.aats.org/resources/interatrial-conduction-block-p-7378.



DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work the authors used ChatGPT to improve scientific English. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Conflict of Interest Statement

The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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Key Words: atrial fibrillation, cardiac surgery, electrocardiogram, interatrial block