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Predictors of atrial fibrillation post coronary artery bypass graft surgery: new scoring system

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To cite: Lotter K, Yadav S, Saxena P, *et al*. Predictors of atrial fibrillation post coronary artery bypass graft surgery: new scoring system. *Open Heart* 2023;**10**:e002284. doi:10.1136/openhrt-2023-002284

Received 15 February 2023
Accepted 23 May 2023

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ABSTRACT

Background Atrial fibrillation (AF) following coronary artery bypass graft surgery (CABG) is common and results in significant increases in hospital stay and financial encumbrance.

Objective Determine and use the predictors of postoperative AF (POAF) following CABG to develop a new predictive screening tool.

Method A retrospective case–control study evaluated 388 patients (98 developed POAF and 290 remained in sinus rhythm) who undertook CABG surgery at Townsville University Hospital between 2016 and 2017. The demographic profile, risk factors for AF including hypertension, age≥75 years, transient ischaemic attack or stroke, chronic obstructive pulmonary disease (HATCH) score, electrocardiography features and perioperative factors were determined.

Results Patients who developed POAF were significantly older. On univariate analysis HATCH score, aortic regurgitation, increased p-wave duration and amplitude in lead II and terminal p-wave amplitude in lead V1 were associated with POAF; as were increased cardiopulmonary bypass time (103.5±33.9 vs 90.6±26.4 min, p=0.001) and increased cross clamp time. On multivariate analysis age (p=0.038), p-wave duration ≥100 ms (p=0.005), HATCH score (p=0.049) and CBP Time ≥100 min (p=0.001) were associated with POAF. Receiver operating characteristic curve demonstrated that with a cut-off of ≥2 for HATCH score, POAF could be predicted with a sensitivity of 72.8% and a specificity of 34.7%. Adding p-wave duration in lead II >100 ms and cardiopulmonary bypass time >100 min to the HATCH score increased the sensitivity to 83.7% with a specificity of 33.1%. This was termed the HATCH-PC score.

Conclusion Patients with HATCH scores ≥2, and those with p-wave duration >100 ms, or cardiopulmonary bypass time >100 min were at greater risk of developing POAF following CABG.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Atrial fibrillation is a common complication following coronary artery bypass graft (CABG) surgery, which is associated with significant increases in cost, morbidity and mortality. Many predictors of postoperative atrial fibrillation (POAF) have been identified, including hypertension, age≥75 years, transient ischaemic attack or stroke, chronic obstructive pulmonary disease (HATCH) score, but no routine screening tools have yet been adopted clinically to identify high risk individuals.

WHAT THIS STUDY ADDS

⇒ By adding two additional variables to the HATCH score, including p-wave duration ≥100 ms and cardiopulmonary bypass time ≥100 min, the HATCH-PC method is a more sensitive tool in predicting POAF following CABG.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ To date, no studies have combined prophylactic therapies in conjunction with a risk stratification model. Future research should be conducted investigating the use of prophylactic therapies in combination with risk stratification of POAF. To facilitate this, a prospective observation study is required to validate a risk stratification tool such as HATCH-PC.

INTRODUCTION

Atrial fibrillation (AF) is a common complication following coronary artery bypass grafting (CABG) surgery. Postoperative AF (POAF) most commonly develops on day 2 or 3 postoperatively, and affects 20%–40% of patients following CABG.[1] Acutely, POAF is associated with increased intensive care and in-hospital stays, and higher readmission rates.[1–4] The financial costs associated with POAF are estimated at US$10 000 –US$11 500 per patient.[3] Thus, POAF has important economic and public health effects, especially with regards to human resources and delaying bed turnover. Additionally, POAF is associated with higher rates of postoperative renal insufficiency, infection, ventricular arrhythmia and stroke.[1 2] Over the long-term POAF is associated with higher rates of permanent AF and stroke.[5 6] Studies have demonstrated that the 30-day mortality rate and long-term mortality rate at 15 years to be significantly higher in patients who develop POAF following CABG.[2 7]

Our study aims to identify risk factors associated with the development of POAF following CABG, and develop a screening tool to help identify patients at high risk of developing POAF. Such identification may allow for more targeted postoperative surveillance and monitoring, and potentially enable targeted prophylactic treatment for high-risk individuals.

METHOD

Study population

POAF was defined as any episode of AF that failed to terminate spontaneously after 5 min, or required pharmacological or electrical cardioversion.

Inclusion criteria

Patients who were over 18 years old and undertook CABG at Townsville University Hospital, a tertiary hospital, between 1 January 2016 and 31 December 2017 were eligible for inclusion, amounting to 498 patients.

Exclusion criteria

Patients were excluded from the study if they had a history of AF. Patients undertaking CABG along with valvular replacement in the same operation were excluded. Patients who died acutely in the perioperative setting were excluded as no postoperative ECG data were available. Following the application of exclusion criteria, a total of 388 people were included.

Sample size

Using Open Epi V.3, a sample size of 193 people was calculated.[8] The prevalence of POAF following CABG was assumed to be 20% with 95% confidence based on the existing literature.[1 4] Type I error was set at 0.05 and power at 0.90.

Demographic factors

Age, gender, weight, height and body mass index along with concurrent comorbidities including hypertension, diabetes, history of stroke or trans-ischaemic attack, cardiac failure, peripheral arterial disease and smoking status were assessed. Using these data, the hypertension, age≥75 years, transient ischaemic attack or stroke, chronic obstructive pulmonary disease, and heart failure (HATCH) score was calculated for each patient (2 points for either a history of transient ischaemic attack/stroke or heart failure, respectively; 1 point for hypertension, age >75 years or chronic obstructive pulmonary disease (COPD), respectively).

ECG parameters

Standard 12-lead ECG acquired at paper speed of 25 mm/s and standard calibration 10 mm/mV, was used for analysis. ECG parameters were obtained from preoperative ECGs and included p-wave duration (ms) and amplitude (mV) measured in Lead II along with the negative deflection of the p-wave in V1 duration (ms) and amplitude (mV) below isoelectric baseline. The terminal p-wave force, that is, the area of negative p-wave deflection in V1, was calculated by multiplying the negative deflections duration and amplitude.[9]

Echocardiogram

Preoperative transthoracic echocardiogram results for left ventricular ejection fraction (LVEF), and the presence of valvular pathology were included.

Perioperative factors

Cardiopulmonary bypass (CPB) time, cross clamp time, number of diseased coronary arteries and haemoglobin levels at the time of CPB were included. Postoperative factors such as intercostal catheter loss in the first 4 hours postoperatively was measured in mL and intensive care unit (ICU) length of stay measured in hours and minutes were also noted.

A retrospective case–control study design was used. All data were gathered by the same assessor to maintain continuity. Twenty-seven ECGs (approximately 1 in 15 patients) were selected and measured by a second independent blind assessor for intraclass correlation.

Statistical analysis

IBM SPSS statistics software (IBM Corp. Released 2017. IBM SPSS Statistics for Windows, V.25.0., IBM) was used for statistical analysis.

Most continuous data were non-parametric, hence were expressed as medians and IQRs. Exceptions included cross clamp time, CPB time and haemoglobin levels at time of CPB, which were parametric and were expressed as mean±SD. Univariate analysis was performed using Student's t-test and Mann-Whitney U test for parametric and non-parametric data, respectively. χ^2 test was used to compare categorical variables. Variables were included for multivariate analysis if p<0.1 on univariate analysis. OR, p<0.05 and 95% CIs were obtained for variables included in multivariate analysis to determine independent predictors of POAF. Receiver operating characteristics curve analysis was performed to assess sensitivity and specificity of various models. The intraclass correlation assessed p-wave length in lead 2, and assumed a two-way mixed model, with absolute agreement, and CIs of 95%. A p<0.05 was considered statistically significant for the study.

RESULTS

Study population

Of the 498 patients who underwent CABG, 388 patients met inclusion criteria (table 1). A majority of the 388 patients were elderly males (78.4%) with a median age of 62 (54–75 years). The two most common comorbidities in this population were a history of smoking (71.9%) and hypertension (79.1%).

Of the 388 patients, 98 (25.3%) developed POAF. Importantly, POAF was associated with increased ICU length of stay (43 hours:31 min vs 27 hours:54 min p<0.05). The preoperative baseline characteristics, echocardiogram and ECG findings of the POAF group and sinus rhythm (SR) group are shown in tables 2 and 3. The

Table 1 Baseline characteristics and comorbidities (N=388)

Variable	Value
Male	304 (78.4%)
Age	62 (54–75)*
BMI	28.37 (24.37–32.16)*
Current smoking status (n=358)	124 (34.6%)
History of smoking (n=370)	277 (74.9%)
HTN	307 (79.1%)
Age >75	37 (9.5%)
TIA/stroke	4 (1.0%)
COPD	67 (17.3%)
CCF (EF<40%)	40 (10.3%)
PAD	31 (8.0%)

Values are presented as n (%).
*Median (IQR).
BMI, body mass index; CCF, congestive cardiac failure; COPD, chronic obstructive pulmonary disease; EF, ejection fraction; HTN, Hypertension; PAD, peripheral arterial disease; TIA, transient ischaemic attack.

POAF group had a significantly older population with a median age of 65 (IQR: 12.5) vs 61 (IQR: 16), p<0.001. Both groups had similar anthropometrics and comorbidity risk.

The development of POAF was associated with higher HATCH scores, p=0.019. However, the median and IQR were the same for both the POAF and SR group, while the mean score in the POAF group was greater than the SR group (1.5±SD=1.018 vs 1.21±SD=1.16).

Increased p-wave duration (ms) and amplitude (mV) in lead II on preoperative ECGs were associated with the POAF group. The intraclass correlation for p-wave duration in lead II was 0.725 (95% CI 0.046 to 0.9, p<0.001), with a Cronbach's alpha of 0.832. In V1 having a deeper terminal p-wave depth (mV) was associated with the POAF group, while terminal p-wave duration (ms) and force (ms×mV) was not associated.

Aortic regurgitation was the only echocardiography risk factor to reach significance with an OR of 2.318 (1.027–5.234).

Table 2 Differences between patients with POAF and sinus rhythm (n=388)

Variable	POAF n=98 (25.3%)	Sinus rhythm n=290 (74.7%)	P value
Demographics			
Age at operation	65 (58–71)	61 (53–69)	0.001
Height (cm)	170 (166–176)	171 (165–177)	0.640
Weight (kg)	85 (98–73)	83 (73–95)	0.792
BMI (kg/m^2)	29.07 (24.75–32.30)	28.37 (24.98–32.32)	0.582
ECG changes			
p-wave duration in lead II (ms)	100 (86.67–113.33)	93.33 (80-100)	<0.001
p-wave amplitude in lead II (mV)	0.1 (0.067–0.1167)	0.1 (0.05–0.1)	0.017
Terminal p-wave duration (ms)	40 (30–60)	40 (40–60)	0.336
Terminal p-wave depth (mV)	0.05 (0.05–0.1)	0.05 (0.025–0.05)	0.046
Terminal p-wave force (ms x mV)	2 (1-4)	2 (1-4)	0.259
Preoperative risk factors:			
HATCH Score	1 (1-2)	1 (1-2)	0.019
LV eection fraction	59 (46–61)	55 (50–60)	0.981
Intraoperative risk factors:			
Cardiopulmonary bypass time total (min)	103.51±33.927	90.64±26.393	0.001
Cross clamp time total (min)	71.98±28.413	63.22±23.529	0.009
Haemoglobin (g/L)	91.93±17.638	91.08±17.898	0.691*
Postoperative variables:			
Intercostal catheter loss at 4 hours postoperative (mL)	320 (169–510)	250 (160–400)	0.093
ICU Length of stay (hour:min)	43:49 (25:05–69.33)	28:00 (23:05–50.31)	0.024

Values expressed as mean±SD or median (IQR).
*Indicates p value where equal variance assumptions were violated, thus could not be assumed.
BMI, body mass index; HATCH, hypertension, age≥75 years, transient ischaemic attack or stroke, chronic obstructive pulmonary disease and heart failure; ICU, intensive care unit; LV, Left ventricle; POAF, postoperative atrial fibrillation.

Table 3 Univariate analysis of risk factors for the development of POAF (n=388)

Variable	OR	95% CI	P value
Demographic			
Gender	1.104	0.628 to 1.942	0.730
Age categorised in 15 years			
<50	1		0.010
50–64	3.756	1.277 to 11.044	0.016
65–79	5.714	1.952 to 16.729	0.001
80+	6.000	1.074 to 33.53	0.041
Comorbidities:			
Hypertension	1.481	0.810 to 2.705	0.200
Age >75	1.695	0.810 to 2.705	0.146
TIA/stroke	9.126	0.938 to 88.781	0.051*
COPD	1.107	0.609 to 2.009	0.739
Heart failure (EF<40%)	1.692	0.845 to 3.389	0.134
Peripheral arterial disease	1.232	0.547 to 2.774	0.614
Current smoking	0.827	0.496 to 1.381	0.486
Smoking history	0.698	0.413 to 1.178	0.176
Statin therapy	1.429	0.741 to 2.754	0.285
Diabetes mellitus	1.267	0.794 to 2.022	0.320
Perioperative risk factors:			
Left main coronary artery stenosis	1.066	0.644 to 1.764	0.803
Cardiopulmonary bypass	1.197	0.469 to 3.057	0.707
p-wave duration 100 ms	2.084	1.027 to 5.234	0.002
Cardiopulmonary bypass ≥100 min	2.205	1.382 to 3.519	0.001
Echocardiogram results:			
Mitral regurgitation	1.485	0.833 to 2.646	0.178
Tricuspid regurgitation	1.994	0.835 to 4.762	0.114
Aortic stenosis	1.485	0.133 to 16.553	0.584*
Aortic regurgitation	2.318	1.027 to 5.234	0.038

All values represent Pearson's χ^2 with continuity correction.
Other abbreviations as in table 1.
*Fisher's exact test due to violation of Pearson's χ^2 test assumptions.
COPD, chronic obstructive pulmonary disease; EF, ejection fraction; POAF, postoperative atrial fibrillation; TIA, transient ischaemic attack.

Intraoperative risk factors

Increased mean CPB time (103.5±33.9 vs 90.6±26.4 min, p=0.001) and mean cross clamp time (72.0±28.4 vs 63.2±23.5 min, p=0.009) were both associated with the POAF group. A CPB time of more than 100 min was associated with a two-fold increased risk of POAF (OR 2.205, 95% CI 1.382 to 3.519, p=0.001).

Our multivariate analysis demonstrated in table 4 had a presentation accuracy in classification of 77.9%. Furthermore, our model shows advanced age to be the most important risk factor for the development of POAF, with p-wave duration in Lead II, HATCH score and CPB time also being significantly, and therefore, independently associated with the development of POAF. The presence of aortic regurgitation on echocardiography failed to reach significance in multivariate analysis.

A HATCH score of ≥2 was associated with a 34.7% sensitivity (Sn) and a 72.8% specificity (Sp) for predicting POAF. Sensitivity was improved while specificity declined when p-wave ≥100 ms (Sn=65.3%, Sp=47.6) and CPB ≥100 min (Sn=83.7%, Sp=33.1%) were added successively, as demonstrated in figure 1. In both situations a score of 1 point was added to the patients' underlying HATCH score for each positive variable, this was termed HATCH-PC. Furthermore, higher scores were associated with improved specificity and reduced sensitivity across all models. In this population where the prevalence of POAF was 25.3% the HATCH-PC model had a negative predictive value (NPV) of 85.71% .

Table 4 Multiple logistic regression analysis—risk factors for the development of POAF (n=388)

	Multivariate		
Variable	**OR**	**95% CI**	**P value**
Age categorised in 15 years			
<50			0.038
50–64	3.938	1.305 to 11.879	0.015
65–79	4.989	1.654 to 15.046	0.004
80+	6.138	1.00 to 37.672	0.050
p-wave duration ≥100 ms	2.027	1.243 to 3.304	0.005
HATCH score	1.301	1.001 to 1.689	0.049
Cardiopulmonary bypass time ≥100 min	2.218	1.359 to 3.620	0.001
Aortic regurgitation	1.605	0.662 to 3.890	0.295

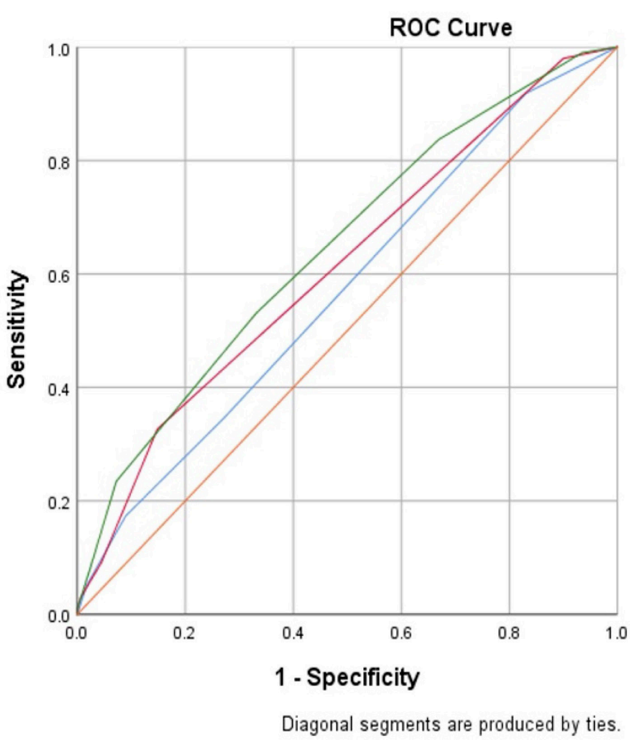
Other abbreviations as per table 2.
HATCH, hypertension, age≥75 years, transient ischaemic attack or stroke, chronic obstructive pulmonary disease and heart failure; POAF, postoperative atrial fibrillation.

DISCUSSION

The prevalence of new-onset POAF following CABG was 25.3% at Townsville University Hospital between 2016 and 2017, which compares with other international studies.[1 4 10] Independent risk factors for the development of POAF included age, p-wave duration in lead II, HATCH score and CPB time.

Patient age was the most significant risk factor and is a well-documented association.[1 10 11] Mathew *et al* estimated that for every 10 years of age, the risk of POAF increased by 75%.[1] Our model assessed the OR at 15-year increments from 50 years old and found a similar positive association with the development of POAF. It should be noted however, that Matthew *et al*'s study included patients undertaking valve replacement, which itself is associated with higher rates of POAF, therefore, making direct comparison difficult.[1 11] Physiologically, it has been postulated that age-associated anatomical remodelling and fibrosis of the conduction pathways may predispose to the development of POAF.[12]

Furthermore, it has been hypothesised that preoperative ECGs may detect underlying atrial conductive abnormalities, particularly those associated with left atrial dilatation.[13 14] Increased p-wave amplitude and duration in lead 2, and the p-wave negative deflection characteristics of terminal p-wave duration (ms), depth (mV) and force (ms×mV) in precordial lead V1 has previously been correlated with increased left atrial size.[9] A 2018 metanalysis and prospective cohort study by Wu *et al* found similar results to our study with pre-operative p-wave duration >105 ms being strongly associated with increased risk of POAF, OR 4.63, 95% CI 2.66 to 8.03 p<0.001.[14] Moreover, a majority of the literature surrounding pre-operative p-wave duration is more than 15 years old, and while their results support our study's findings, more up-to-date literature is needed to reflect the change in clinical and



Score of ≥2	HATCH	HATCH + p-wave ≥100ms (HATCH-P)	HATCH + p-wave ≥100ms + CPB ≥100min (HATCH-PC)
Sensitivity	34.7%	65.3%	83.7%
Specificity	72.8%	47.6%	33.1%
AUC	0.572	0.614	0.646
P	0.34	0.001	<0.001
95% CI (upper)	0.637	0.679	0.709
95% CI (lower)	0.506	0.549	0.583

Figure 1 Receiver operating characteristics curve (ROC) of HATCH score plus add on scoring methods with 1 point allocated for having either p-wave ≥100 ms or CPB ≥100 min for predicting atrial fibrillation after coronary artery bypass graft surgery. AUC, area under the curve; CBP, cardiopulmonary bypass; HATCH, hypertension, age ≥75 years, transient ischaemic attack or stroke, chronic obstructive pulmonary disease and heart failure.

post-operative practice.[15 16] With an intraclass correlation value of 0.725, measuring p-wave duration in lead II is moderately reliable.[17]

The surgical factors of CPB time and cross clamp time were both associated with increased rates of POAF. The ischaemic reperfusion injury associated with cross clamp release, and the proinflammatory state triggered by complement system activation associated with CPB use have both been proposed as possible triggers for the development of POAF.[18 19] Importantly, when CPB time exceeded 100 min the patients' risk of developing POAF doubled. Similarly, the POAF group had significantly longer CPB times, p=0.041, in Tsai *et al*'s 2015 study on isolated CABG patients.[11] However, Hashemzadeh *et al* in a multivariate analysis found increased CPB time was associated with lower rates of POAF (OR 0.984 (95% CI 0.976 to 0.992) p<0.001).[20]

The presence of aortic regurgitation was associated with POAF on univariate analysis, but failed to demonstrate an independent association on multivariate analysis. However, since patients undergoing concurrent valve replacement were excluded from the study, severe valvular disease was unlikely to be seen, hence only the presence of valvular pathology was included rather than disease severity. Aksu *et al* and Abdel-Salam and Nammas supported these findings, finding no association between valvular pathology or LVEF and POAF following CABG.[21 22] Furthermore, no studies could be found assessing tricuspid regurgitation and POAF, making our study potentially the first to assess this variable despite its non-significant finding.

The study population had well-preserved left ventricular function with only 10.3% of the total population having an ejection fraction of <40%, while the median LVEF were 59% (IQR 46–61) in the POAF group and 55% (50–60) in the SR group. A meta-analysis by Yanashita *et al* found both reduced LVEF and history of heart failure (LVEF<40%) to be associated with the development of POAF across 7 and 4 studies, respectively. Yamashita *et al* did highlight significant heterogeneity between data sets especially with respect to LVEF which had an I^2 of 0.79.[23]

The studies investigating the relationship between the HATCH score and POAF are limited.[10 24 25] Selvi *et al* and Emren *et al* had a similar study design being retrospective studies assessing POAF following CABG. Burgos *et al* have produced two papers the first from 2019 which focused on all cardiac surgeries of which only 2% of patients had an isolated CABG; compared with 31% having isolated CABG in their 2021 paper, making comparison difficult.[10 24–26] In our study for every 1 point increase in the HATCH score, the risk of POAF increased by 1.312 times. This was comparable to Selvi *et al* who found an adjusted OR of 1.334 (95% CI 1.022 to 1.741) p=0.034.[10] Similarly, Burgos *et al*'s 2021 paper who found an adjusted OR of 1.18 (95% CI 1.018 to 1.36) p=0.04, while Emren *et al* found a statistically significant relationship in univariate analysis, but failed to perform a multivariate analysis.[24 26]

Both Selvi *et al* and Emren used receiver operator curves to validate the potential use of the HATCH score as a predictive test for POAF. Both studies also used a HATCH score of ≥2. Selvi *et al* found similar results with a sensitivity of 42% and specificity of 70%. These are comparable to our findings of 34.7% sensitivity and a 72.8% specificity. Emren *et al* on the other hand, found a sensitivity of 77% and specificity of 62%. Furthermore, Emren *et al* found a statistically significant relationship between POAF and COPD and lower ejection fraction (<40%), a relationship not supported by either Selvi *et al*, or the findings of our study.[10 24] Our results suggest that by including CPB time ≥100 min and p-wave duration (≥100 ms) in addition to the HATCH score, proved to be more sensitive screening test than HATCH alone. While difficult to directly compare; the HATCH-PC model, which exclusively assesses patients for risk of POAF following CABG, demonstrated higher sensitivity but lower specificity compared with the COM-AF model, a recent model proposed in Burgos et al's 2021 paper for predicting POAF in all postoperative cardiac patients.[26]

Clinical implications

The financial costs associated with POAF are estimated at US$10 000–US$11 500 per patient, with increased ICU length of stay being a contributor.[3] In our study the development of POAF increased median ICU length of stay by 16 hours, costing approximately $A3276 (US$2286) per patient with POAF, based on the 2019 Independent Hospital Pricing Authority estimate of average ICU cost in Australia, and a $1AUD to $0.70USD exchange rate.[27 28] While this is a crude measure of the economic burden, it clearly demonstrates the financial implications of POAF and value in findings measures to reduce its prevalence.

Several prophylactic interventions for POAF have been investigated, however, inconsistent results have limited their adoption. Treatments such as amiodarone and beta-blockers in the early postoperative period have been trialled, but limited by inconsistent evidence and fears that such interventions may cause more harm than good.[29 30] Offering therapy only to high-risk individuals has the potential to reduce adverse effects on a population level with less patients unnecessarily exposed, while providing an intervention with a potential benefit to those most in need. To date, no studies have combined prophylactic therapies in conjunction with a risk stratification model—that is, providing targeted prophylactic intervention. Hence, the utility of the HATCH or HATCH-PC model in combination with prophylactic therapy is an area where future research is required in the form of prospective clinical trials.

Limitations

This study relied on well-documented and accurate medical records, with a major limitation being the availability and reliability of the data. To overcome this challenge, data were assessed from various sections of the medical charts including preoperative surgical notes, ECG data records and discharge letters. An example of information not found was left atrial size, a known risk factor for POAF, instead indirect measures such as p-wave length and amplitude were used. Furthermore, continuity and standards of data collection were maintained by only having one team member performing the data collection. An example where observer measurement error may have taken place includes the measurements of p-waves, where the small measurement values on a standardly calibrated ECG, may have predisposed to error. Furthermore, the wide CI and low number of participants with stroke suggest that this study is likely too underpowered to draw meaningful conclusions regarding this risk factor. Two additional limitations of this study are the single-centre experience and retrospective design meaning that the findings of the study only show an association between the HATCH-PC model and predicting POAF in patients post-CABG. Furthermore this study does not demonstrate that the HATCH-PC model improves outcomes, it only serves to highlight where this model could be used to potentially improve clinical outcomes, formal prospective trials would be required to confirm its clinical utility.

Strengths

This is the largest study to date that has evaluated the HATCH scoring method in relation to POAF in an isolated CABG population, and the first that has combined the HATCH scoring method with additional risk factors to help improve its sensitivity and clinical utility. The addition of p-wave duration and CPB time to the HATCH score provides a scoring system that is easy to calculate and does not require invasive laboratory tests to improve sensitivity, and identify people who are at increased risk of developing POAF following CABG. This information could be used to improve monitoring or commence prophylactic interventions on patients who are identified as being at higher risk.

CONCLUSION

Overall, patients with a higher HATCH score may be at greater risk of developing POAF following CABG. However, with low sensitivity, our study does not support the use of the HATCH score as a screening tool for predicting POAF. Furthermore, our results would suggest that by adding two additional variables to the HATCH score, including p-wave duration ≥100 ms and CPB time ≥100 min, the HATCH-PC method is a more sensitive tool in predicting POAF following CABG. Future research should be conducted investigating the use of prophylactic therapies in combination with risk stratification of POAF.

Contributors KL: data collection, statistical analysis, lead author, guarantor. SY: manuscript and editing. PS: manuscript and editing. VV: statistical analysis support and project supervision. BJ: team leader, supervisor and corresponding author.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Not applicable.

Ethics approval Ethics and site-specific approval were granted by the Townsville District Human Research Ethics Committee and the Townsville Hospital and Health Service Research Governance Office, HREC/18/QTHS/155. The study was in accordance with the Queensland Government Public Health Act and undertaken as part of the research collaboration between Townsville Hospital and Health Service and James Cook University. A deidentified patient list was provided by Queensland State-wide Cardiac Clinical Informatics Unit. All data were gathered retrospectively using the 'ieMR' electronic record system.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement All data relevant to the study are included in the article or uploaded as online supplemental information.

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REFERENCES

1 Mathew JP, Fontes ML, Tudor IC, *et al*. A multicenter risk index for atrial fibrillation after cardiac surgery. *JAMA* 2004;291:1720–9.
2 Phan K, Ha HSK, Phan S, *et al*. New-onset atrial fibrillation following coronary bypass surgery predicts long-term mortality: a systematic review and meta-analysis. *Eur J Cardiothorac Surg* 2015;48:817–24.
3 Yadava M, Hughey AB, Crawford TC. Postoperative atrial fibrillation: incidence, mechanisms, and clinical correlates. *Cardiol Clin* 2014;32:627–36.
4 Thorén E, Hellgren L, Ståhle E. High incidence of atrial fibrillation after coronary surgery. *Interact Cardiovasc Thorac Surg* 2016;22:176–80.
5 Benedetto U, Gaudino MF, Dimagli A, *et al*. Postoperative atrial fibrillation and long-term risk of stroke after isolated coronary artery bypass graft surgery. *Circulation* 2020;142:1320–9.
6 Conen D, Wang MK, Devereaux PJ, *et al*. New-onset perioperative atrial fibrillation after coronary artery bypass grafting and long-term risk of adverse events: an analysis from the CORONARY trial. *JAHA* 2021;10.
7 El-Chami MF, Kilgo P, Thourani V, *et al*. New-onset atrial fibrillation predicts long-term mortality after coronary artery bypass graft. *J Am Coll Cardiol* 2010;55:1370–6.
8 Dean A, Sullivan K, Soe M. *OpenEpi: Open Source Epidemiologic*.
9 Huang Z, Zheng Z, Wu B, *et al*. Predictive value of P wave terminal force in lead V1 for atrial fibrillation: a meta-analysis. *Ann Noninvasive Electrocardiol* 2020;25:e12739.
10 Selvi M, Gungor H, Zencir C, *et al*. A new Predictor of atrial fibrillation after coronary artery bypass graft surgery: HATCH score. *J Investig Med* 2018;66:648–52.
11 Tsai Y-T, Lai C-H, Loh S-H, *et al*. Assessment of the risk factors and outcomes for postoperative atrial fibrillation patients undergoing isolated coronary artery bypass grafting. *Acta Cardiol Sin* 2015;31:436–43.
12 Camm AJ, Kirchhof P, Lip GYH, *et al*. Guidelines for the management of atrial fibrillation: the task force for the management of atrial fibrillation of the European society of cardiology (ESC). *Eur Heart J* 2010;31:2369–429.
13 Chandy J, Nakai T, Lee RJ, *et al*. Increases in P-wave dispersion predict postoperative atrial fibrillation after coronary artery bypass graft surgery. *Anesth Analg* 2004;98:303–10.
14 Wu F, Wu Y, Tao W, *et al*. Preoperative P-wave duration as a predictor of atrial fibrillation after coronary artery bypass grafting: a prospective cohort study with meta-analysis. *Int J Nurs Sci* 2018;5:151–6.
15 Chang CM, Lee SH, Lu MJ, *et al*. The role of P wave in prediction of atrial fibrillation after coronary artery surgery. *Int J Cardiol* 1999;68:303–8.
16 Klein M, Evans SJ, Blumberg S, *et al*. P-wave signal-averaged electrocardiogram to predict atrial fibrillation after coronary artery bypass surgery. *Am Heart J* 1995;129:895–901.
17 Fisher RA. *Statistical Methods for Research Workers, 12th ed*. Edinburgh: Oliver and Boyd, 1954.
18 Velioglu Y, Yuksel A. Predictors of postoperative atrial fibrillation after beating-heart coronary artery bypass surgery: is cardiopulmonary bypass a risk factor. *Acta Cardiol Sin* 2019;35:468–75.

19 Maesen B, Nijs J, Maessen J, *et al*. Post-operative atrial fibrillation: a maze of mechanisms. *Europace* 2012;14:159–74.
20 Hashemzadeh K, Dehdilani M, Dehdilani M. Postoperative atrial fibrillation following open cardiac surgery: predisposing factors and complications. *J Cardiovasc Thorac Res* 2013;5:101–7.
21 Abdel-Salam Z, Nammas W. Incidence and predictors of atrial fibrillation after coronary artery bypass surgery: detection by event loop recorder monitoring from a contemporary Multicentre cohort. *Acta Cardiol* 2017;72:311–7.
22 Aksu U, Gulcu O, Aksakal E, *et al*. Comparison of the three conventional methods for the postoperative atrial fibrillation prediction. *Echocardiography* 2017;34:831–4.
23 Yamashita K, Hu N, Ranjan R, *et al*. Clinical risk factors for postoperative atrial fibrillation among patients after cardiac surgery. *Thorac Cardiovasc Surg* 2019;67:107–16.
24 Emren V, Aldemir M, Duygu H, *et al*. Usefulness of HATCH score as a predictor of atrial fibrillation after coronary artery bypass graft. *Kardiol Pol* 2016;74:749–53.
25 Burgos LM, Seoane L, Parodi JB, *et al*. Postoperative atrial fibrillation is associated with higher scores on predictive indices. *J Thorac Cardiovasc Surg* 2019;157:2279–86.
26 Burgos LM, Ramírez AG, Seoane L, *et al*. New combined risk score to predict atrial fibrillation after cardiac surgery: COM-AF. *Ann Card Anaesth* 2021;24:458–63.
27 Independent Hospital Pricing Authority. National pricing model: technical specifications 2019–20; 2019.
28 Hicks P, Huckson S, Fenney E, *et al*. The financial cost of intensive care in Australia: a multicentre registry study. *Med J Aust* 2019;211:324–5.
29 Budeus M, Hennersdorf M, Perings S, *et al*. Amiodarone prophylaxis for atrial fibrillation of high-risk patients after coronary bypass grafting: a prospective, double-blinded, placebo-controlled, randomized study. *Eur Heart J* 2006;27:1584–91.
30 Gillespie EL, White CM, Kluger J, *et al*. A hospital perspective on the cost-effectiveness of beta-blockade for prophylaxis of atrial fibrillation after cardiothoracic surgery. *Clin Ther* 2005;27:1963–9.