Evaluation of Right Atrial and Biatrial Temporary Pacing for the Prevention of Atrial Fibrillation After Coronary Artery Bypass Surgery

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OBJECTIVES The purpose of this study was to determine if atrial pacing is effective in reducing postoperative atrial fibrillation (AF).

BACKGROUND Atrial fibrillation after coronary artery bypass grafting (CABG) is a common problem for which medical management has been disappointing. Atrial-based pacing has become an attractive nonpharmacologic therapy for the prevention of AF.

METHODS Sixty-one post-CABG patients (mean age 65 years) were randomized to one of three groups: no atrial pacing (NAP), right atrial pacing (RAP) or biatrial pacing (BAP). Each patient had one set of atrial wires attached to both the right and left atria, respectively, at the conclusion of surgery. Patients in the RAP and BAP groups were continuously paced at a rate of 100 pulses per minute for 96 h or until the onset of sustained AF (>10 min). All patients were monitored with Holter monitors or full disclosure telemetry to identify the onset of AF. The primary end point of the study was the first onset of sustained AF.

RESULTS There was no significant difference in the proportion of patients developing AF in the three groups (NAP = 33%; RAP = 29%; BAP = 37%; p > 0.7). However, for the subset of patients on beta-adrenergic blocking agents after CABG, there was a trend toward less AF in the paced groups. There were no serious complications related to pacing, although in three patients the pacemaker appeared to induce AF by pacing during atrial repolarization.

CONCLUSIONS Continuous right or biatrial pacing in the postoperative setting is safe and well tolerated. We did not find that post-CABG pacing prevented AF in this pilot study; however, the role of combined pacing and beta-blockade merits further study. (J Am Coll Cardiol 1999;33:1981–8) © 1999 by the American College of Cardiology

Atrial fibrillation (AF) after coronary artery bypass graft (CABG) surgery is a common and vexing problem. Treatment with electrical cardioversion, antiarrhythmic drugs and anticoagulation adds significant morbidity and cost (1,2). Prophylactic pharmacologic treatment has been disappointing. Although a recent trial found a significant reduction in postoperative AF with amiodarone treatment (3), the incidence of AF in the treatment group was still 25%, and concerns about potential morbidity exist.

Nonpharmacologic strategies for prevention of atrial fibrillation have become more attractive because they avoid the toxicity of antiarrhythmic drugs. There is a growing body of evidence that atrial-based pacing is effective in reducing recurrences of paroxysmal AF. Retrospective and prospective studies have suggested that patients who receive atrial or dual chamber pacemakers experience fewer episodes of AF than those receiving traditional ventricular demand pacemakers (4–12). New evidence has recently emerged suggesting that biatrial pacing can be more effective than standard right atrial pacing in decreasing recurrences of paroxysmal AF (13). Although investigation of these strategies is ongoing, they are currently limited to patients undergoing pacemaker implantation for other indications, usually sick sinus syndrome.

Patients undergoing CABG surgery may provide a model to examine the impact of prophylactic atrial pacing, since they have temporary atrial and ventricular pacing wires implanted at the time of surgery, and a high incidence of
Patient population. Patients 18 years of age and older who were scheduled for elective CABG at the University of Massachusetts Medical Center (UMMC) were approached for enrollment in the study. All patients had to be in sinus rhythm before surgery and on no antiarrhythmic medications. Recruitment occurred between April 1, 1995 and December 31, 1995. Patients were randomly assigned in a single blinded fashion to one of three groups: no atrial pacing (NAP), right atrial pacing (RAP) or biatrial pacing (BAP). Patients were randomized before surgery and followed for up to 96 h postoperatively.

Patients were excluded from the study if they had a known history of AF or atrial flutter requiring antiarrhythmic medications, had renal or hepatic dysfunction (serum creatinine $>3$, liver enzyme tests $>3\times$ normal) or were unable to give informed consent. In addition, patients in whom epicardial pacing wires could not be placed during surgery, or patients who developed ventricular arrhythmias requiring therapy with oral or intravenous antiarrhythmic agents other than intravenous lidocaine after surgery were excluded from the study group. Patients who required temporary pacing immediately after surgery due to hemodynamic compromise remained in the study.

Baseline characteristics and arrhythmia history were ascertained from direct patient interviews and review of the medical record. The ejection fraction was calculated by left ventriculography in the right anterior oblique projection during preoperative cardiac catheterization. P-wave duration was measured from lead II of the preoperative electrocardiogram.

The protocol was approved by the Human Subjects Committee of UMMC, and all patients signed an informed consent before participating.

Study protocol. All patients had one set of ventricular and two sets of atrial bipolar pacing wires implanted at the conclusion of surgery (Medtronic model #6500, Minneapolis, Minnesota). One pair of atrial wires was implanted in the standard location 1 cm apart along the high lateral right atrium near the sinus node. A second pair was attached to the posterior surface of the left atrium between the right superior and inferior pulmonary veins. These two pairs of wires were connected by a Y connector to the atrial input of the temporary pacemaker for biaatrial pacing. The ventricular wires were attached to the right ventricular apex in the standard fashion.

Patients in the NAP group were paced in the ventricular single-chamber pacing mode at a backup rate of 50 pulses per minute (ppm) while in the surgical intensive care unit, and had no pacing while in the hospital ward as is the standard practice at UMMC. Patients in the RAP and BAP groups were paced with a temporary external dual chamber pacemaker (Medtronic model #5346) in the AV universal (DDD) mode at a lower rate limit of 100 ppm with an atrioventricular (AV) delay of 220 ms to establish continuous atrial pacing at rest. Pacemaker settings including an upper-rate limit of 140 ppm, a postventricular atrial refractory period of 175 ms, atrial sensitivity of 0.5 mV, ventricular sensitivity of 2 mV and maximum atrial and ventricular pacing output of 20 mA. Pacing was continued for 96 h or until the first sustained episode of AF (>10 min). Patients in the NAP group requiring pacing due to hemodynamic compromise were paced in the atrial single-chamber pacing or AV universal mode at the discretion of the attending physician, and were subsequently returned to their assigned mode when their condition allowed this to occur.

Physicians were instructed to continue beta-adrenergic blocking agents postoperatively in all patients receiving preoperative beta-blocker therapy. Preoperative beta-blockers were continued through the morning of surgery, and metoprolol 25 mg orally twice daily was instituted postoperatively as soon as all intravenous inotropes were discontinued. The dose was titrated upward at the discretion of the attending surgeon. Patients not on beta-blockers preoperatively were not started on beta-blockers in the postoperative period. Patients prescribed verapamil, digoxin or diltiazem preoperatively received alternative medications postoperatively with no effect on AV nodal conduction.

Patients were continuously monitored during the study period with a full disclosure telemetry system (HP CareVue, Hewlett-Packard Co., Burlington, Massachusetts) or continuous Holter monitoring. Both surface lead II and electrograms from the right atrial electrode pair were recorded on the Holter monitors (Fig. 1). Bipolar pacing and sensing thresholds for both atrial leads were checked after arrival at the intensive care unit and daily thereafter to ensure atrial
The underlying heart rhythm and rate were documented daily. When considered stable, patients were transferred out of the intensive care unit to monitored beds in the general hospital ward where pacing was continued for the remainder of the study period.

The primary end point of the study was the initial occurrence of AF or atrial flutter with a ventricular rate greater than 100 beats/min for 10 consecutive minutes, or completion of the 96-h monitoring period. An investigator reviewed the hospital chart and full disclosure telemetry at least once daily to monitor the cardiac rhythm and establish the time of onset of AF. The exact time of onset was confirmed using the Holter monitor.

Previous studies have suggested that the use of beta-blockers is associated with a reduced incidence of postoperative AF. Therefore, it was predetermined that the effects of beta-blockers on the incidence of AF would be analyzed for each group. Patients were defined as being on beta-blockers only if they were taking the medication for at least 24 h before the onset of AF. All cardiac medications administered over the previous 24 h were documented daily by an investigator throughout the study period.

**Data analysis.** All values are expressed as mean ± standard deviation. Baseline characteristics of the study groups were compared using Student t test or analysis of variance for continuous variables, and the chi-square test for discrete variables. The principal study outcomes were examined using a logistic multivariate regression analysis that controlled for baseline variables that were possible confounders (p < 0.1). These controlling variables included a history of prior myocardial infarction (MI) and intraoperative cross-clamp time. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated in the standard manner. Predictors of AF were initially determined by univariate analysis with chi-square or Student t test, and then entered into a multivariate regression model to ascertain independent predictors.

We used Holter monitoring to detect all episodes of AF, both clinically apparent as well as that detected by electrocardiography alone, and we anticipated a 50% incidence of AF. Enrolling 20 patients per group allowed us 80% power to detect a reduction of AF to 10% in either pacing arm compared with no pacing.

Statistics were calculated using SAS version 6.12 (Cary, North Carolina). All patients were initially analyzed in their original randomized groups, on an intention-to-treat basis, and then based on treatment received.

**RESULTS**

A total of 65 patients were enrolled in the study. Four patients were dropped from the study after randomization based on the predefined criteria: one patient developed AF intraoperatively and was started on procainamide, one patient did not have a second set of atrial wires implanted for technical reasons, one patient had a postoperative course complicated by multiorgan system disease and was eliminated at the request of the attending physician and one patient asked to be dropped from the study on the first postoperative day because of hiccups that were attributed to pacing. No patients were withdrawn because of ventricular arrhythmias. The final study population consisted of 61 patients.

Of the 61 patients enrolled, 45 (74%) were men with an average age of 65 years. Twenty-one patients were randomly
assigned to the NAP and RAP groups each, and 19 to the BAP group. Baseline characteristics of the groups are presented in Table 1. Patients in the RAP group included a significantly greater proportion of patients with prior MI; patients in the BAP group had a significantly shorter operative cross-clamp time. There was no significant difference between preoperative and postoperative beta-blocker use.

Right atrial and biatrial pacing were well tolerated. Mean pacing threshold for the RAP group was 2.0 mV. In the BAP group, mean left atrial pacing threshold was significantly greater than mean right atrial pacing threshold (3.2 vs. 1.6 mA; p < 0.001). Some patients in the BAP group initially complained of discomfort from diaphragmatic pacing; however, this was easily corrected by decreasing the pacemaker output.

No complications were attributed to the implantation of the second set of atrial wires in the left atrium. One patient died in the intensive care unit on the 3rd postoperative day due to respiratory arrest. No other patients died during the study period.

Three patients crossed over to other groups after randomization. One patient in the BAP group returned to the operating room on the first postoperative day because of mediastinal bleeding, and did not have the atrial wires reimplanted. This patient remained unpaced for the remainder of the study period. A second patient who was in the RAP group had their pacemaker turned off on the second postoperative day to relieve uncomfortable diaphragmatic pacing. The third patient, who was in the NAP group, was paced from the right atrium for the entire 96-h study period due to persistent bradycardia. In addition, four patients in the NAP group required temporary pacing in the postoperative period due to bradycardia: three until postoperative day 1, and one until postoperative day 2.

Pacing efficacy. There was no significant difference in the proportion of patients developing AF in the three groups (NAP = 33%, RAP = 29%, BAP = 37%; SAP vs. NAP adjusted OR = 1.01, 95% CI 0.25, 4.07; BAP vs. NAP adjusted OR = 1.31, 95% CI 0.32, 5.38; p > 0.7) (Fig. 2). The mean time to onset of AF was significantly shorter in the BAP group than the other two groups (NAP = 39 ± 19 h, RAP = 54 ± 29 h, BAP = 27 ± 8 h; p < 0.02). There was no significant difference in the total time spent in the intensive care unit (NAP = 49 ± 54 h, RAP = 64 ± 137 h, BAP = 47 ± 25 h; p > 0.6), or in the total hospitalization time (NAP = 158 ± 99 h, RAP = 164 ± 195 h, BAP = 163 ± 113 h; p > 0.5) among the groups. Occurrences of atrial fibrillation were equally distributed throughout the 96-h monitoring period (Fig. 3). No patients in the study developed atrial flutter.

Among patients receiving beta-blocker therapy there was a trend toward fewer episodes of AF in the BAP group (Fig. 2). No patients on beta-blocker therapy who actually received BAP developed AF. One patient randomized to the BAP group did develop AF, but this patient was never paced because the wires had been removed on postoperative day 1.

On-treatment analysis. When patients were analyzed according to the pacing treatment they actually received, there was again no statistically significant difference in the proportion of patients developing AF among the three groups (NAP = 36%, RAP = 29%, BAP = 33%; SAP vs. NAP adjusted OR = 1.13, 95% CI 0.28, 4.58; BAP vs. NAP adjusted OR = 1.72, 95% CI 0.43, 6.80; p > 0.4). However, for patients receiving beta-blocker therapy (n = 39), there was a statistically significant reduction in the proportion of patients developing atrial fibrillation in the paced groups (NAP = 38%, RAP = 15%, BAP = 0%; p < 0.05). This reduction remained of borderline statistical significance when a multivariate regression analysis was

Table 1. Sample Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>NAP (n = 21)</th>
<th>RAP (n = 21)</th>
<th>BAP (n = 19)</th>
<th>p Value</th>
</tr>
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<tbody>
<tr>
<td>Age (mean ± SD, y)</td>
<td>65 ± 13</td>
<td>65 ± 10</td>
<td>65 ± 10</td>
<td>NS</td>
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<td>Hypertension* (%)</td>
<td>67</td>
<td>62</td>
<td>74</td>
<td>NS</td>
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<td>History of alcohol use† (%)</td>
<td>19</td>
<td>33</td>
<td>26</td>
<td>NS</td>
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<tr>
<td>Chronic obstructive pulmonary disease (%)</td>
<td>5</td>
<td>0</td>
<td>5</td>
<td>NS</td>
</tr>
<tr>
<td>Prior myocardial infarction* (%)</td>
<td>33</td>
<td>67</td>
<td>26</td>
<td>0.02</td>
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<tr>
<td>Diabetes mellitus (%)</td>
<td>38</td>
<td>29</td>
<td>21</td>
<td>NS</td>
</tr>
<tr>
<td>History of atrial fibrillation or flutter (%)</td>
<td>0</td>
<td>5</td>
<td>11</td>
<td>NS</td>
</tr>
<tr>
<td>P-wave duration (mean ± SD, ms)</td>
<td>108 ± 17</td>
<td>104 ± 20</td>
<td>107 ± 16</td>
<td>NS</td>
</tr>
<tr>
<td>Preoperative beta-blocker use (%)</td>
<td>71</td>
<td>81</td>
<td>68</td>
<td>NS</td>
</tr>
<tr>
<td>Postoperative beta-blocker use (%)</td>
<td>71</td>
<td>76</td>
<td>58</td>
<td>NS</td>
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<tr>
<td>Ejection fraction (mean ± SD)</td>
<td>63 ± 11</td>
<td>54 ± 13</td>
<td>60 ± 13</td>
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<tr>
<td>Operative data</td>
<td></td>
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<td>Cross clamp time (mean ± SD, min)</td>
<td>79 ± 30</td>
<td>78 ± 25</td>
<td>59 ± 18</td>
<td>0.02</td>
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<tr>
<td>Bypass time (mean ± SD, min)</td>
<td>128 ± 45</td>
<td>127 ± 36</td>
<td>107 ± 25</td>
<td>NS</td>
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</tbody>
</table>

*Determined by patient interview and review of medical record. †More than 1 oz. of alcohol per day.
performed which controlled for cross-clamp time and history of MI (p = 0.06).

Predictors of AF. Overall, 20 (33%) of the patients developed postoperative AF, which agrees with previous published reports (1). In a univariate analysis, patients who developed AF were significantly older, had a longer p-wave duration on baseline electrocardiogram, were more likely to have a history of hypertension and were less likely to receive beta-blockers than those who remained in sinus rhythm.

Figure 2. The proportion of patients developing atrial fibrillation (AF) among all the patients, and among the subset of patients on beta-blockers using an intention-to-treat analysis. There was a trend toward a reduction in the incidence of AF among the patients on beta-blockers receiving biatrial pacing (BAP). The one patient who developed AF in the BAP group (asterisk) was never actually paced because of postoperative complications. The numbers above the bars indicate the absolute number of patients in each category. Hatched bars = no atrial pacing; black bars = right atrial pacing; white bars = BAP.

Figure 3. Cumulative incidence of atrial fibrillation (AF) throughout the study. Each bar represents the cumulative incidence of AF through 24, 48, 72 and 96 h, respectively. Although the peak incidence of AF was during the 24 to 48 h period, patients continued to develop AF until the study was completed after 96 h.
Total hospital time was significantly longer among patients developing atrial fibrillation. This was mainly due to extra time spent on the hospital ward (see Table 2). After surgery, there was no difference in mean heart rate between patients who went on to develop AF compared with those who did not (AF vs. no AF; 81 ± 14 vs. 85 ± 15 beats/min; p > 0.3). A multivariate regression analysis was performed which included history of prior MI, cross-clamp time, age, p-wave duration, history of hypertension and postoperative beta-blocker use as potential predictors of AF. The only factors significantly associated with development of postoperative AF were patient age greater than 65 years (adjusted OR = 6.01, 95% CI 1.03, 35.08, p < 0.05) and beta-blocker use (adjusted OR = 0.07, 95% CI 0.01, 0.44, p < 0.01).

**Initiation of AF.** We examined the onset of AF in all patients with good quality recordings to try and gain some insight into the mechanism of AF initiation. Holter monitor or telemetry data were available to examine the onset of AF in 17 of the 20 patients. In 11 patients, AF began after an APB (Fig. 1A). In three patients, AF began after a wide complex ventricular beat which may have been either a ventricular premature beat or an atrial premature beat (APB) with aberrant conduction. In three patients, one in the RAP group and two in the BAP group, the pacemaker may have inadvertently initiated AF. There were no cases where AF developed after a long pause.

The three cases where the pacemaker may have initiated AF are shown in Figure 4. In one case, transient loss of atrial sensing caused the atrial pacemaker spike to fall progressively later after atrial depolarization until AF was initiated (Fig. 4A). In two cases, failure of the pacemaker to sense an APB led to an atrial pacemaker spike during atrial repolarization, which appeared to initiate AF (Fig. 4B and C). These spikes occurred during the initial part of the QRS complex and would have been difficult to delineate without the Holter recording of the atrial electrograms.

**DISCUSSION**

Most evidence suggests that AF is a reentrant rhythm consisting of multiple wandering wavelets of electrical activity (14,15). It is often initiated by an APB encountering areas of slow conduction and unidirectional block (16). There are many reasons why one might expect atrial pacing to be effective in preventing AF. Increasing atrial rate suppresses the APBs which may initiate AF. A prospective randomized trial found that AF recurrences are reduced in patients receiving right atrial pacing compared with ventricular pacing (12). Biatrial pacing is a new technique that may have improved efficacy in preventing AF by shortening intra-atrial conduction delays, decreasing dispersion of refactoriness and increasing the conduction velocity and refractory period of APBs (17). Papageorgiou et al. (18) found that the posterior triangle of Koch is a critical area of slow conduction, and that coronary sinus (i.e., left atrial) pacing prevented the induction of AF by high right atrial APBs. Saksena and colleagues (13), in a small randomized crossover trial, found that biatrial pacing was useful clinically in reducing the time to first recurrence of AF in patients receiving permanent pacemakers. Slow conduction and increased dispersion of refactoriness have also been implicated in the pathogenesis of postoperative AF (19). This trial is the first to examine whether right atrial or biatrial pacing strategies are safe and effective for preventing AF in the postoperative setting.

In our study, temporary right or biatrial pacing after CABG surgery had no effect in reducing occurrences of AF.
increase the incidence of AF in some patients, eliminating the beneficial effects of pacing. This may explain our finding that patients in the BAP group who developed AF had a shorter time to onset of AF than the other groups. Permanent atrial pacing systems have better sensing characteristics than the temporary pacemakers used in the intensive care unit. Nevertheless, the possibility of pacemaker-induced AF should be recognized, and may need to be addressed in future trials of prophylactic pacemaker therapy.

Role of beta-blockers. The beneficial effect of beta-blockers in preventing postoperative AF has been well described (20,21). We also found a significant reduction in postoperative AF among patients taking beta-blockers. Although the population of patients on beta-blockers in our study was not large enough to show a statistically significant benefit from pacing, the fact that there were no occurrences of AF in patients on beta-blockers who received BAP is encouraging. Excess catecholamines in the postoperative patients may be one reason why pacing was not effective in suppressing AF. Perhaps by blocking catecholamines and reducing the resting heart rate and number of APBs, beta-blockers may enhance the ability of pacemakers to prevent the initiation of AF and minimize the likelihood of pacemaker-induced AF.

Risk factors for AF. Our results agree with larger retrospective studies (1,2) which found age to be an independent risk factor for postoperative AF. We did not find cross-clamp time or p-wave duration in lead II to be an independent predictor of AF. The patients in our study who developed AF spent nearly 100 h more in the hospital than those who did not. Most of this time was spent in the hospital wards, presumably waiting for therapeutic anticoagulation. This highlights the need for continued investigation into methods of AF prevention and early hospital discharge in these patients. Patients over age 65 years are a particularly high risk group that may benefit from targeted therapy.

Limitations. In this pilot study, the patient population was small. Therefore, our findings of no reduction in AF in the entire patient population, and a trend toward a reduction of AF in paced patients on beta-blockers, are less definitive than they would have been had a larger group of patients been studied. In particular, we cannot exclude a smaller benefit to right atrial or biatrial pacing compared with no pacing in the entire patient population than we were able to detect. These data suggest that a study in a larger group of patients, all of whom are on beta-blockers, should be performed to confirm these preliminary observations. We did not include an enrollment registry in our study, therefore we cannot comment on differences between the population we studied and those patients who declined to enroll. Since consecutive patients were approached for participation, and the baseline characteristics of our population are similar to that reported by large observational research. 

Figure 4. Holter monitor tracings recorded from three patients in whom atrial fibrillation (AF) appeared to be initiated by the pacemaker. The top tracing is a bipolar recording from the right atrial epicardial wires showing the atrial pacing spikes, and the bottom tracing is a recording from surface lead I. In A, a failure of pacemaker sensing caused the atrial pacing spike to fall progressively later after atrial depolarization until AF was initiated when a pacing spike (open arrow) occurred during a critical period of atrial repolarization. In B and C an atrial premature beat (filled arrow) occurs which is not sensed appropriately by the pacemaker. The next pacing spike (open arrow) occurs during atrial repolarization and the rhythm converts to AF. A ventricular spike also occurs due to safety pacing.

However, there was evidence that those patients treated with both beta-blockade and pacing had a trend toward fewer occurrences of AF. Pacing was well tolerated in all patients and did not increase hospitalization time. There were no complications related to the placement of left atrial pacing wires at the conclusion of surgery.

There are several possible reasons why our results differed from those of previous studies. The initiation of AF in the postoperative setting is likely different than in patients with sick sinus syndrome or pacing-induced AF. Although Cox and colleagues (19) have demonstrated that the development of postoperative AF is related to slow conduction and increased dispersion of refractoriness in the atria, postoperative patients also have a high catecholamine state, postoperative pericarditis, frequent fluid and electrolyte shifts and respiratory compromise. These factors may alter the electrophysiologic substrate for AF, thereby making pacing less effective in its prevention. It is possible that therapies helpful in the prevention of outpatient AF may be different than those required in the postoperative setting.

We detected some cases of AF that appeared to have been triggered by the pacemaker after failure to sense an atrial depolarization. In patients with frequent APBs, inappropriate pacing during atrial repolarization could paradoxically
studies (2), we think that the study group was a representa-
tive sample of patients undergoing CABG.

The temporary pacemakers in the paced groups were set
to a rate of 100 beats/min to try to ensure continuous pacing
without causing hemodynamic compromise. Some patients
with an intrinsic heart rate greater that 100 beats/min may
not have been continuously paced. Our records indicate that
continuous pacing occurred >90% of the time in the paced
patients, however, making this an unlikely confounder.
There were no patients randomized to pacing who devel-
oped atrial fibrillation while the pacemaker was inhibited
due to sinus tachycardia.

Cox and colleagues have demonstrated that the duration
of cross-clamp time is a risk factor for the development of
postoperative AF (22). This raises the question of whether
the shorter cross-clamp time in the BAP group could be
responsible for a reduction of AF in that group. Cross-
clamp time is only a significant risk factor for AF in patients
undergoing aortic valve replacement, a group excluded from
our study. Furthermore, the trend toward less AF in
patients with BAP on beta-blockers persisted even after
correcting for this confounder, making it an unlikely expla-
nation for our results.

Conclusions. Continuous right or btrial pacing in
the postoperative setting is safe and well tolerated. In this pilot
study, we find that temporary right or btrial pacing did not
prevent postoperative AF. Temporary pacing may paradox-
ically induce AF in some patients if inappropriate sensing
leads to pacing during atrial repolarization. The role of
combined pacing and beta-blockade shows some promise
for reducing the incidence of postoperative AF and deserves
further study.

Acknowledgments
We thank the cardiothoracic surgeons at UMMC for
contributing their patients to the study; the nurses from
3-ICU and 3-West for their cooperation with the study;
Robert Goldberg, PhD for statistical review and Ellen
Cowan for secretarial support.

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