Interactive CardioVascular and Thoracic Surgery

The risk of arrhythmias following coronary artery bypass surgery: do smokers have a paradox effect?

Nael Al-Sarraf, Lukman Thalib, Anne Hughes, Maighread Houlihan, Michael Tolan, Vincent Young and Eillish McGovern

Interact CardioVasc Thorac Surg 2010;11:550-555; originally published online Aug 16, 2010;
DOI: 10.1510/icvts.2010.242586

doi:10.1510/icvts.2010.242586

INTERACTIVE CARDIOVASCULAR AND THORACIC SURGERY

Interactive CardioVascular and Thoracic Surgery 11 (2010) 550–555

www.icvts.org

Institutional report - Arrhythmia

The risk of arrhythmias following coronary artery bypass surgery: do smokers have a paradox effect?

Nael Al-Sarraf[a,b,*], Lukman Thalib[c], Anne Hughes[a], Maighread Houlihan[a], Michael Tolan[a], Vincent Young[a], Eillish McGovern[a]

[a]*Department of Cardiothoracic Surgery, St. James's Hospital, Dublin 8, Ireland*
[b]*Department of Cardiothoracic Surgery, Chest Disease Hospital, Al-Jabriah, P.O. Box 718, Kuwait city 46308, Kuwait*
[c]*Department of Community Medicine (Biostatistics), Kuwait University, Kuwait*

Received 6 May 2010; received in revised form 19 July 2010; accepted 26 July 2010

Abstract

Smoking is reported to increase the risk of arrhythmias. However, there are limited data on its effects on arrhythmias following coronary artery bypass graft (CABG). This is a retrospective review of a prospective database of all CABG patients over an eight-year period. Our cohort ($n=2813$) was subdivided into: current ($n=1169$), former ($n=837$), and non-smokers ($n=807$). Predictors of arrhythmias following CABG in relation to smoking status were analysed. Atrial arrhythmias occurred in 942 patients (33%). Ventricular arrhythmias occurred in 48 patients (2%) and high-grade atrioventricular block occurred in five patients (0.2%). Arrhythmias were lower in current smokers than former and non-smokers (29% vs. 40% vs. 39%, respectively $P<0.001$). Logistic regression analysis showed 30% arrhythmia risk reduction in smokers compared to non-smokers [odds ratio (OR) 0.7, 95% confidence intervals (CI) 0.5–0.8] and this effect persisted after accounting for potential confounders while former smokers had the same risk as non-smokers (OR 1.04, CI 0.9–1.3). There were no significant differences in mortality. Smokers are less prone to develop arrhythmias following CABG. This paradox effect is lost in former smokers. This effect is possibly due to a lower state of hyper adrenergic stimulation observed in smokers than non-smokers following the stress of surgery.

Keywords: Smoking; Coronary artery bypass graft; Arrhythmia; Atrial fibrillation

1. Introduction

Smoking is a risk factor for cardiovascular and cerebrovascular disease. The effect of smoking on coronary artery bypass graft (CABG) patients has been previously reported [1, 2]. However, data examining the effect of smoking on arrhythmias following cardiac surgery are scarce and conflicting [3–6]. While some of these reports have highlighted a lack of association, others have shown a protective effect. We sought to assess the incidence, pattern and predictors of arrhythmias in smokers and former smokers undergoing CABG.

2. Patients and methods

2.1. Patients

This is a retrospective review of a prospective database (Patient Analysis and Tracking System, Dendrite Clinical, UK). All patients who underwent isolated CABG at St James's hospital between February 2000 and July 2008 were reviewed. Two thousand eight hundred and thirteen consecutive patients were included in the study. Patients were divided into three groups based on their smoking habits: current ($n=1169$, 41%), former ($n=837$, 30%) and non-smokers ($n=807$, 29%). Current smokers included all patients who were active smokers and those who quit smoking within four weeks of surgery. Former smokers included all patients who quit smoking more than four weeks before surgery. Non-smokers included all patients who had never smoked before surgery. Smoking refers to consumption of cigarettes, cigars, or pipes [2, 7]. All data were collected prospectively in a departmental database through the input of a dedicated database manager. The database was strictly controlled with twice yearly departmental reports generated and an annual report of departmental performance generated and submitted to society of cardiothoracic surgeons of UK and Ireland for audit purposes. All missing information was collected by reviewing patients' charts. Outcome measures studied were postoperative arrhythmias and mortality. This study and the database were both approved by our institutional review board. Individual patient consent was obtained for entry into the database. However, our institutional review board waived the need for individual patient consent for this study.

2.2. Cardiopulmonary bypass (CPB) and definitions

All cardiac surgeries were performed through a median sternotomy with CPB as previously described [8]. Definition

*Corresponding author. Tel.: +96-5-66600543; fax: +96-5-4741504.
E-mail address: trinityq8@hotmail.com (N. Al-Sarraf).

of operative priority and in-hospital mortality was previously reported [8, 9].

2.3. Arrhythmias and medications

Postoperative arrhythmias were recorded by using telemetry for 72 h post surgery in patients who remained in sinus rhythm and longer in patients who sustained arrhythmia until they are back in sinus rhythm or until their rate is controlled for 48 consecutive hours. In addition, a 12-lead electrocardiogram is obtained routinely for all patients in the first four postoperative days and prior to discharge. Postoperative atrial fibrillation is defined as new onset atrial fibrillation (both persistent and paroxysmal) in patients with no prior history of same. Patients who were in atrial fibrillation preoperatively and continued to be in atrial fibrillation postoperatively are recorded separately and these are not considered as arrhythmia complication since they were already in that rhythm. Ventricular arrhythmia (i.e. ventricular tachycardia and ventricular fibrillation) are recorded both preoperatively if they occurred prior to surgery and postoperatively if they occurred in postoperative period. Atrioventricular block was recorded similarly. As a routine, all patients were started on beta-blocker medications (unless contraindicated) postoperatively with the dose escalated to achieve heart rate of 70–80 beats/min. Intubated patients and those with contraindication to beta-blockers are treated with amiodarone. As a rule, preoperative anti-arrhythmic medications are restarted postoperatively with the dosage adjusted if required. Our practice remained uniform throughout the study period.

2.4. Data analysis

Data analysis began by exploring the differences between the three groups for clinical, admission, and outcome variables. Categorical variables were compared using the χ^2-test, and continuous variables were compared using analysis of variance or the non-parametric Kruskal–Wallis test based on the satisfaction of the normal assumption (Tables 1 and 2). The effect of smoking on the outcome variables was further analyzed using logistic regression methods (Table 3). Effect size of smoking on each individual outcome variables was quantified by crude odds ratios (OR), followed by adjusted OR after accounting for age, gender and other potential confounders (Table 4). OR, 95% confidence intervals (CI) as well as exact P values are reported. Statistical analysis was performed using SPSS version 17 (SPSS, Chicago, IL, USA). The P values were considered statistically significant when <0.05.

3. Results

Our cohort ($n=2813$) consisted of 558 (20%) females and 2255 (80%) males. Age ranged from 32 to 85 years old with a mean (± 1 S.D.) of 63.5 (± 9.1) years old. In-hospital mortality overall was 2.6% (72 patients). The incidence of postoperative arrhythmia overall was 35% (995 patients). Preoperative factors and patient characteristics among the three groups are summarized in Table 1. As expected, current and former smokers were males with younger age at surgery than non-smokers. In addition, the incidence of chronic obstructive pulmonary disease (COPD), myocardial infarction (MI), triple vessel disease and poor left ventricular function were highest among smokers and former smokers. The incidence of preoperative arrhythmia was highest among former smokers and lowest among current smokers compared to non-smokers. The incidence of preoperative pacemaker was similar in all groups. Current and former smokers had higher emergency and salvage CABG than non-smokers. There was no difference in the number of grafts used among the three groups (Table 1).

Table 2 summarizes postoperative arrhythmias and in-hospital mortality among the three groups. As shown, there was no significant difference in the mortality rate. However, the incidence of postoperative arrhythmias was significantly lower in the current smokers compared to both the former and non-smokers (29% vs. 40% vs. 39%, $P<0.001$). The incidence of atrial arrhythmias was lower in current smokers compared to non-smokers (27% vs. 37%, respectively). Similarly, ventricular arrhythmias were lower in current smokers (1.5% vs. 2%, respectively).

Logistic regression analysis (Tables 3 and 4) showed that current smokers had 30% risk reduction compared to non-smokers (OR 0.7, 95% CI 0.6–0.9) after accounting for potential confounders but no risk reduction was observed in former smokers (OR 0.96, 95% CI 0.8–1.2). In addition, factors that were associated with increased risk of arrhythmias were age, male gender, New York Heart Association (NYHA) class III/IV, preoperative absence of pacemakers and intra-aortic balloon pump (IABP). These factors were all adjusted for potential confounders as summarized at the bottom of Table 4.

4. Discussion

Smoking is a risk factor for coronary artery, peripheral vascular and cerebrovascular diseases. Smoking causes endothelial dysfunction, atherosclerosis and arrhythmias through the combined effects of nicotine, carbon monoxide and polycyclic aromatic hydrocarbons [10]. While this effect has been studied in general population, its applicability to cardiac surgery differs. Cardiac surgery patients are prone to develop arrhythmias by virtue of the surgical stress and the use of inotropic agents [6]. Atrial fibrillation is the commonest arrhythmia observed with an incidence of 30% leading to prolonged hospitalization, increased morbidity and cost [3]. Data examining the effect of smoking on postoperative arrhythmias are limited and conflicting [3–6]. In addition, only atrial fibrillation was examined with no publications examining other arrhythmias (Table 5). We sought to assess the incidence, pattern and predictors of arrhythmias among smokers and former smokers undergoing CABG. By doing so, our understanding on the potential link between smoking and arrhythmias in the surgical setting is improved leading to better understanding of potential interaction that occurs at the mechanistic level.

Do smokers have a paradoxically lower risk of arrhythmias post CABG? As we have shown, the incidence of arrhythmias following CABG was lower in current smokers than non-smokers (29% vs. 39%, $P<0.001$). In addition, logistic regression analysis showed that current smoking is indepen-

Table 1. Sociodemographic and clinical history at admission classified according to smoking status for patients undergoing CABG ($n=2813$)

Variable	Non-smokers ($n=807$)	Current smokers ($n=1169$)	Former smokers ($n=837$)	P-value
Age (years)				
Mean ± S.D.	65.3 ± 8.6	61.3 ± 9.2	64.8 ± 8.8	<0.001*
Gender				
Female	231 (29%)	203 (17%)	124 (15%)	<0.001*
Male	576 (71%)	966 (83%)	713 (85%)	
Angina class (CCS)				
0–2	308 (38%)	498 (43%)	285 (34%)	0.001*
3–4	499 (62%)	671 (57%)	552 (66%)	
NYHA score				
I–II	534 (66%)	806 (69%)	450 (54%)	<0.001*
III–IV	273 (34%)	363 (31%)	387 (46%)	
Congestive cardiac failure				
Yes	73 (9%)	106 (9%)	110 (13%)	0.005*
No	734 (91%)	1063 (91%)	727 (87%)	
Number of MI				
None	469 (58%)	616 (53%)	400 (48%)	<0.001*
One	294 (36%)	438 (37%)	354 (42%)	
Two or more	44 (6%)	115 (10%)	83 (10%)	
Interval of MI to surgery				
None	469 (58%)	616 (52%)	400 (48%)	<0.001*
<90 days	179 (22%)	229 (20%)	195 (23%)	
>90 days	159 (20%)	324 (28%)	242 (29%)	
Diabetes mellitus				
Non-insulin dependent	106 (13%)	177 (15%)	147 (18%)	<0.001*
Insulin dependent	42 (5%)	32 (3%)	52 (6%)	
None	659 (82%)	960 (82%)	638 (76%)	
Hypercholesterolemia				
Yes	602 (75%)	891 (76%)	676 (81%)	0.008*
No	205 (25%)	278 (24%)	161 (19%)	
Hypertension				
Yes	482 (60%)	608 (52%)	539 (64%)	<0.001*
No	325 (40%)	561 (48%)	298 (36%)	
Chronic obstructive pulmonary disease				
Yes	34 (4%)	98 (8%)	85 (10%)	<0.01*
No	773 (96%)	1071 (92%)	752 (90%)	
Cerebrovascular accident				
Yes	37 (5%)	53 (5%)	58 (7%)	0.036*
No	770 (95%)	1116 (95%)	779 (93%)	
Peripheral vascular disease				
Yes	129 (16%)	205 (18%)	150 (18%)	0.539
No	678 (84%)	964 (82%)	687 (82%)	
Preoperative arrhythmia				
Yes	47 (6%)	63 (5%)	72 (9%)	0.011*
No	760 (94%)	1106 (95%)	765 (91%)	
Type of arrhythmia				
Normal sinus rhythm	760 (94.2%)	1106 (94.6%)	765 (91.4%)	0.009*
Atrial fibrillation/atrial flutter	35 (4.3%)	48 (4.1%)	62 (7.4%)	
Complete heart block/paced	9 (1.1%)	9 (0.8%)	3 (0.4%)	
Ventricular fibrillation/tachycardia	3 (0.4%)	6 (0.5%)	7 (0.8%)	
Extra-cardiac arteriopathy				
Yes	32 (4%)	56 (5%)	92 (11%)	<0.001*
No	775 (96%)	1113 (95%)	745 (89%)	
Extent of coronary artery disease				
Single/double	205 (25%)	263 (22%)	152 (18%)	0.002*
Triple	602 (75%)	906 (78%)	685 (82%)	
Left main disease				
Yes	243 (30%)	333 (28%)	285 (34%)	0.027*
No	564 (70%)	836 (72%)	552 (66%)	
Ejection fraction (%)				
<50%	274 (34%)	474 (41%)	292 (35%)	0.004*
≥50%	533 (66%)	695 (59%)	545 (65%)	
Pacemaker				
Yes	11 (1%)	9 (1%)	7 (1%)	0.376
No	796 (99%)	1160 (99%)	830 (99%)	
Preoperative IABP				
Yes	16 (2%)	11 (1%)	7 (1%)	0.057
No	791 (98%)	1158 (99%)	830 (99%)	
Priority				
Elective/urgent	759 (94%)	1139 (97%)	810 (97%)	<0.001*
Emergent/salvage	48 (6%)	30 (3%)	27 (3%)	

Table 1 *(Continued)*

Variable	Non-smokers ($n=807$)	Current smokers ($n=1169$)	Former smokers ($n=837$)	*P*-value
Number of grafts				
≤3	593 (73%)	874 (75%)	646 (77%)	0.208
>3	214 (27%)	295 (25%)	191 (23%)	
Body mass index (kg/m²)				
Mean±S.D.	28.1±5.7	27.8±4.5	28.2±4.2	0.127
Aortic cross-clamp time (min)				
Mean±S.D.	51.1±18.4	51.5±19.6	52.1±17.9	0.563

*Statistically significant ($P<0.05$). S.D., standard deviation; CCS, Canadian cardiovascular society; NYHA, New York Heart Association; IABP, intra-aortic balloon pump; MI, myocardial infarction; CABG, coronary artery bypass graft.

Table 2. Postoperative arrhythmias in current smokers, former smokers and non-smokers following CABG ($n=2813$)

Variable	Non-smokers ($n=807$)	Current smokers ($n=1169$)	Former smokers ($n=837$)	*P*-value
Arrhythmia				
Yes	318 (39%)	339 (29%)	338 (40%)	<0.001*
No	489 (61%)	830 (71%)	499 (60%)	
Type of arrhythmia				
Atrial fibrillation/atrial flutter/ supraventricular tachycardia	303 (37%)	319 (27.2%)	320 (38%)	<0.001*
Ventricular tachycardia/fibrillation	15 (2%)	17 (1.5%)	16 (1.8%)	
Atrioventricular block requiring pacemaker	0	3 (0.3%)	2 (0.2%)	
None	489 (61%)	830 (71%)	499 (60%)	
Status				
Alive	785 (97%)	1147 (98%)	809 (97%)	0.116
Dead	22 (3%)	22 (2%)	28 (3%)	

*Statistically significant ($P<0.05$). CABG, coronary artery bypass graft.

dently associated with a lower risk of postoperative arrhythmias (Table 3) and this effect persisted after accounting for potential confounders (Table 4). This relative reduction was 30% compared to non-smokers (OR 0.7, 95% CI 0.6–0.9). Both atrial and ventricular arrhythmias were significantly lower in current than non-smokers following CABG (Table 2). Previously, authors have reported conflicting results of the risk of developing atrial fibrillation in smokers undergoing cardiac surgery with no data on other types of arrhythmias. A summary of these studies is shown in Table 5. Some of these studies have reported a protective effect of smoking to the risk of developing atrial fibrillation following cardiac surgery [4–6] while others [3] showed no difference. Few points could potentially explain these conflicting results. Firstly, the number of patients studied differed among the studies, Secondly, the specific association that was intended to be looked at also differed e.g. some examined predictors of atrial fibrillation by examining a generalized set of data including smoking [3–5] while others have specifically examined smokers [6]. None of these data examined other arrhythmias apart from atrial fibrillation and none have examined former smokers. In our series, the protective effect observed in current smokers was lost in former smokers (OR 0.96, 95% CI 0.8–1.2).

Table 3. Logistic regression analysis for smoking and arrhythmia (unadjusted, aged adjusted, age and gender adjusted) ($n=2813$)

Postoperative arrhythmias	OR (95% CI)	*P*-value
Smoking (unadjusted)		
Never smoker	Ref.	Ref.
Former smoker	1.04 (0.9–1.3)	0.686
Current smoker	0.6 (0.5–0.8)	<0.001*
Age adjusted smoking		
Smoking		
Never smoker	Ref.	Ref.
Former smoker	1.07 (0.9–1.3)	0.494
Current smoker	0.8 (0.6–0.9)	0.006*
Age at surgery	1.06 (1.05–1.07)	<0.001*
Age and gender adjusted smoking		
Smoking		
Never smoker	Ref.	Ref.
Former smoker	1.01 (0.8–1.2)	0.938
Current smoker	0.7 (0.6–0.9)	0.001*
Age at surgery	1.06 (1.05–1.07)	<0.001*
Gender		
Male	1.6 (1.3–1.9)	<0.001*
Female	Ref.	

*Statistically significant ($P<0.05$). OR, odds ratio.

Why do smokers have lower incidence of arrhythmias following CABG? In chronic nicotine exposure, a sympathetic over activity state occurs which can contribute to a variety of cardiovascular dysfunctions including atherogenesis and thrombogenesis. This autonomic dysfunction may operate through the biologic remodelling of nicotine acetylcholine receptors with resulting down regulation of these receptors secondary to chronic nicotine stimulation which can lead to desensitization as an adaptive response [11]. The overall effect is arrhythmogenic stimulation in smokers [12]. On the other hand, cardiac surgery is a hyper adrenergic condition with a surge in catecholamine release secondary to surgical stress and inotropic drugs [6]. These effects predispose patients to develop arrhythmias following cardiac surgery. In smokers, these mechanisms are potentially altered with a lowered effect on smokers than non-smokers following cardiac surgery. Smokers have a

Table 4. Logistic regression analysis with adjusted odds ratios (OR) for factors predicting postoperative arrhythmia after accounting for potential confounding variables [a] (only significant variables are shown) ($n=2813$)

Variable	Odds ratio (95% CI)	*P*-value
Smoking		
Never smoker	Ref.	Ref.
Former smoker	0.96 (0.8–1.2)	0.679
Current smoker	0.7 (0.6–0.9)	0.001*
Age at surgery (years)	1.06 (1.05–1.07)	<0.001*
Gender		
Male	1.6 (1.3–2.0)	<0.001*
Female	Ref.	Ref.
NYHA class		
I/II	Ref.	Ref.
III/IV	1.2 (1.01–1.5)	0.037*
Preoperative pacemaker		
No	3.0 (1.1–8.3)	0.032*
Yes	Ref.	Ref.
Preoperative IABP		
No	2.4 (1.01–5.7)	0.047*
Yes	Ref.	Ref.

[a]Age, gender, extent of coronary disease, presence of preoperative arrhythmia, type of preoperative arrhythmias, diabetes mellitus, interval between myocardial infarction and surgery, number of previous myocardial infarctions, congestive cardiac failure, angina status, hypertension, hypercholesterolemia, chronic obstructive pulmonary disease, number of grafts, ejection fraction (%), left main stem disease and peripheral vascular disease. *Statistically significant ($P<0.05$). CI, confidence intervals; IABP, intra-aortic balloon pump; NYHA, New York Heart Association.

higher baseline level of sympathetic activity (and higher adrenergic tolerance) and therefore are less likely to respond to postoperative stress in the same fashion as non-smokers do [11]. This might explain the observed lower incidence of arrhythmias in smokers following CABG. These protective effects do not seem to operate in former smokers where the levels of catecholamine stimulation might have returned to normal baseline activity. This is evident where former smokers lose this smoking-related protective effect of arrhythmogenecity post CABG (Tables 3 and 4).

It is important to emphasise that our results should not be taken as an encouragement of people to continue smoking, as the risk of coronary artery disease (CAD), cerebrovascular disease and COPD are significantly higher in smokers than non-smokers (Table 1). In addition, smokers required CABG on an average four years earlier than non-smokers did (Table 1) with a higher incidence of triple vessel disease and poor ejection fraction seen in smokers. We are merely trying to elucidate the association between smoking and risk of arrhythmias following CABG to try to understand the mechanistic link of the two. Non-smokers develop CAD by various reasons including genetic predisposition while active smokers develop CAD by their smoking habits.

5. Limitations of the study

This remains a retrospective study and as such we can only report an association rather than causality which could only be established by a randomized controlled trial. Such a trial would be ethically questionable since one arm would be encouraged to continue to smoke. In addition, the smoking status of our patients was determined on the basis of self-reporting with no biochemical proof. However, self-reported smoking habits have been found to be accurate in

Table 5. Summary of previously published papers examining risk of atrial fibrillation following cardiac surgery

First author (year)	Type of study/number	Main findings	Limitations/comment
Almassi (1997) [3]	Prospective $n=3855$	1) Included CABG, valve and combination. 2) Incidence of A Fib was 30%. 3) Smoking status was not a significant factor neither in univariate nor multivariate analysis among patients with A Fib vs. those with no A Fib.	1) The majority of patients (98%) were males. 2) Study did not examine other types of arrhythmia. 3) The study did not examine former smokers.
Mahoney (2002) [4]	Retrospective $n=10,550$	1) Included CABG, valves and combination. 2) Smoking status was associated with a lower incidence of A Fib on univariate analysis. 3) Smokers had a protective effect in multivariate analysis (OR 0.59, 95% CI 0.37–0.94).	1) The study did not specifically examine effect of smoking on rhythm but rather examined predictors of A Fib post surgery and cost benefit of amiodarone therapy. 2) No other types of arrhythmias were studied.
Wiggins (2006) [5]	Retrospective $n=377$	1) Included CABG patients only. 2) A Fib occurred in 25% of cases. 3) Smoking history was significantly higher in no A Fib group vs. A Fib group (41% vs. 27%). 4) Smoking status was a predictor of postoperative A Fib on multivariate analysis, together with age and NYHA class.	1) Significant data were missing (22.5% of patients studied) which may have an impact on the findings. 2) Former smokers were not examined. 3) Only A Fib was studied.
Mariscalco (2009) [6]	Retrospective $n=3245$	1) Smokers were 15% of population studied. 2) Examined CABG, valves and combination. 3) A Fib occurred in 26% of cases. 4) Smokers had reduced incidence of A Fib compared to non-smokers (27% vs. 20%). 5) Smoking conferred 46% reduction in risk of postoperative A Fib after inotropic support were excluded (OR 0.54, 95% CI 0.34–0.83).	1) Study did not examine former smokers. 2) Study did not have data on the duration of smoking cessation. 3) Only A Fib was studied with no mention of other types of arrhythmia.

A Fib, atrial fibrillation; CI, confidence interval; NYHA, New York Heart Association; OR, odds ratio; CABG, coronary artery bypass graft.

studies of different population [13]. Another limitation is that we could not examine if there was a link in current smokers to arrhythmias based on the number of cigarette smoked or duration of smoking (i.e. number of pack-years smoked) since this information is not routinely collected in our database. However, within these limitations, we believe our work will stimulate more research about the pathophysiology of arrhythmia in smokers and former smokers undergoing CABG.

6. Conclusion

The risks of atrial and ventricular arrhythmias following CABG are less in smokers than non-smokers. This paradox effect tends is lost in former smokers where the risk of arrhythmias is equivalent to non-smokers. This paradox is possibly due to a lower state of hyper adrenergic stimulation observed in smokers than non-smokers following the stress of surgery. Further studies are warranted to elucidate the mechanistic link of smoking to rhythm disturbances in the surgical setting.

References

[1] Van Domburg RT, Meeter K, Van Berkel DFM, Veldkamp RF, Van Herwerden LA, Bogers AJJC. Smoking cessation reduces mortality after coronary artery bypass surgery: a 20-year follow-up study. J Am Coll Cardiol 2000;36:878–883.
[2] Al-Sarraf N, Thalib L, Hughes A, Tolan M, Young V, McGovern E. Effect of smoking on short-term outcome of patients undergoing coronary artery bypass surgery. Ann Thorac Surg 2008;86:517–523.
[3] Almassi GH, Schowalter T, Nicolosi AC, Aggarwal A, Moritz TE, Henderson WG, Tarazi R, Shroyer AL, Sethi GK, Grover FL, Hammermeister KE. Atrial fibrillation after cardiac surgery: a major morbid event? Ann Surg 1997;226:501–511.
[4] Mahoney EM, Thompson TD, Veledar E, Williams J, Weintraub WS. Cost-effectiveness of targeting patients undergoing cardiac surgery for therapy with intravenous amiodarone to prevent atrial fibrillation. J Am Coll Cardiol 2002;40:737–745.
[5] Wiggins MC, Firpi HA, Blanco RR, Amer M, Dudley SC. Prediction of atrial fibrillation following cardiac surgery using rough set derived rules. Conf Proc IEEE Eng Med Biol Soc 2006;1:4006–4009.
[6] Mariscalco G, Engstrom KG. Are current smokers paradoxically protected against atrial fibrillation after cardiac surgery? Nicotine Tob Res 2009;11:58–63.
[7] Al-Sarraf N, Thalib L, Hughes A, Tolan M, Young V, McGovern E. Lack of correlation between smoking status and early postoperative outcome following valve surgery. Thorac Cardiovasc Surg 2008;56:449–455.
[8] Doddakula K, Al-Sarraf N, Gately K, Hughes A, Tolan M, Young V, McGovern E. Predictors of acute renal failure requiring renal replacement therapy post cardiac surgery in patients with preoperatively normal renal function. Interact CardioVasc Thorac Surg 2007;6:314–318.
[9] Al-Sarraf N, Raza A, Rowley S, Hughes A, Tolan M, Young V, McGovern E. Short-term and long-term outcome in low body mass index patients undergoing cardiac surgery. Gen Thorac Cardiovasc Surg 2009;57:87–93.
[10] Heeringa J, Kors JA, Hofman A, van Rooij FJ, Witteman JC. Cigarette smoking and the risk of atrial fibrillation: the Rotterdam study. Am Heart J 2008;156:1163–1169.
[11] Yun AJ, Bazar KA, Lee PY, Gerber A, Daniel SM. The smoking gun: many conditions associated with tobacco exposure may be attributable to paradoxical compensatory autonomic responses to nicotine. Med Hypotheses 2005;64:1073–1079.
[12] Katz A, Grosbard A. Does it all go up in smoke? Cigarette smoking and tachyarrhythmias. J Cardiovasc Electrophysiol 2006;17:937–939.
[13] Patrick DL, Thompson DC, Diehr P, Koepsell T, Kinne S. The validity of self-reported smoking: a review and meta analysis. Am J Public Health 1994;84:1086–1093.

eComment: Could effect of smoking guide us to a new treatment option for atrial fibrillation?

Authors: ***Kaushal K. Tiwari***, *Adult Cardiac Surgery, G. Pasquinucci Heart Hospital, Via Aurelia Sud, 54100 Massa, Italy;* ***Nermir Granov, Stefano Bevilacqua, Mattia Glauber***

doi:10.1510/icvts.2010.242586A

We found the article by Al-Sarraf et al. interesting but provocative [1]. Smoking is always supposed to do harm and cause all sorts of cardiovascular diseases leading to high morbidity and mortality. The potential risks of active smoking are pulmonary complications, prolonged postoperative ventilation and early bypass occlusion due to corrosive and harmful effects of smoke. But its role in decreasing atrial fibrillation in patients undergoing coronary artery bypass graft (CABG) is of real interest [1]. In general, active smoking patients should be advised to terminate smoking at least four weeks before surgery to decrease the volume of airway secretions and pulmonary complications and to improve mucociliary transport [2].

Atrial fibrillation in patients after CABG has several etiologies like: multiple wavelet re-entry in the atria, rapid firing of an atrial focus, and less likely, atrial ischemia. Preoperative clinical predictors of atrial fibrillation after cardiac surgery include increased age, history of hypertension, male gender, previous history of atrial fibrillation or congestive heart failure, peripheral and/or cerebral vascular disease, and severity of coronary artery disease [3]. Although, the authors made a logistic regression analysis and showed that current smokers had a 30% risk reduction compared to non-smokers after accounting for potential confounders, they have not mentioned the status of the thyroid hormone in their patients. Cerillo et al. [4] have shown that low free T3 hormone could potentially predispose to atrial arrhythmias in CABG patients. Klemperer et al. [5] have proposed administration of exogenous T3 to reduce the incidence of postoperative AF in CABG patients. Al-Sarraf et al. [1] have already cautioned not to take their result as an encouragement for smoking. However, this study raises the possibility to think about a well structured randomized control study with nicotine therapy in non-smoker patients undergoing CABG, taking into consideration that nicotine is the main substance in smoking, which probably has protective effects. Ironically, as smoking cannot be advised, the authors might in the future think of proposing a treatment with nicotine patches or tablets to non-smoker patients before CABG to prevent postoperative atrial fibrillation.

References

[1] Al-Sarraf N, Thalib L, Hughes A, Houlihan M, Tolan M, Young V, McGovern E. The risk of arrhythmias following coronary artery bypass surgery: do smokers have a paradox effect? Interact CardioVasc Thorac Surg 2010;11:550–555.
[2] Bluman LG, Mosca L, Newman N, Simon DG. Preoperative smoking habits and postoperative pulmonary complications. Chest 1998;113:883–889.
[3] Aranki SF, Shaw DP, Adams DH, Rizzo RJ, Couper GS, VanderVliet M, Collins JJ Jr, Cohn LH, Burstin HR. Predictors of atrial fibrillation after coronary artery surgery. Current trends and impact on hospital resources. Circulation 1996;94:390–397.
[4] Cerillo AG, Bevilacqua S, Storti S, Mariani M, Kallushi E, Ripoli A, Clerico A, Glauber M. Free triiodothyronine: a novel predictor of postoperative atrial fibrillation. Eur J Cardiothorac Surg 2003;24:487–492.
[5] Klemperer JD, Klein IL, Ojamaa K, Helm RE, Gomez M, Isom OW, Krieger KH. Triiodothyronine therapy lowers the incidence of atrial fibrillation after cardiac operations. Ann Thorac Surg 1996;61:1323–1327; discussion 1329.