

Atrial Flutter after Coronary Artery Bypass Grafting: Proposed Mechanism as Illuminated by Independent Predictors

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Objective: The aim of this study was to evaluate the cause and treatment of atrial flutter after coronary artery bypass grafting (CABG).

Methods: Between July 1999 and July 2000, 96 CABGs were performed at the Cardiovascular Center of Aichi Prefectural Owari Hospital. We compared two groups of patients: those with atrial flutter after CABG (group A) and those without atrial flutter (group B).

Results: We treated the atrial flutter group with medication, electrical defibrillation, and overdrive pacing. Six of these cases later required radiofrequency catheter ablation (RFCA). These were all with common-type atrial flutter, treated by RFCA for the posterior isthmus, without difficulty. The frequency of direct cannulation to the coronary sinus for retrograde cardioplegia was significantly higher in group A.

Conclusions: Incidence of atrial flutter after CABG was influenced by surgical damage arising from direct cannulation to the coronary sinus for retrograde cardioplegia. Preoperative viability of the myocardium (in addition to good myocardial protection during surgery) also seems to be associated with an increased likelihood of postoperative atrial flutter. (*Ann Thorac Cardiovasc Surg* 2003; 9: 50–6)

Key words: atrial flutter, coronary artery bypass grafting (CABG), radiofrequency catheter ablation (RFCA), retrograde cardioplegia

Introduction

Atrial tachyarrhythmias are common early in the recovery period after cardiothoracic surgery. They develop in 11 to 40% of patients after coronary artery bypass grafting (CABG) and in over 50% of patients after valvular surgery.¹⁻⁵⁾ There are many reports of atrial fibrillation subsequent to CABG. However, atrial flutter after CABG is not well documented. The aim of this study was to evaluate the cause and treatment of atrial flutter after CABG.

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Materials and Methods

Between July 1999 and July 2000, 96 consecutive CABGs were performed at the Cardiovascular Center of Aichi Prefectural Owari Hospital. Patients with combined valvular heart operations, Dor operations, or coronary aneurysm operations were excluded, as were patients who received operations for atrial septal defect. The 80 patients who were not excluded by the above criteria were studied retrospectively. We compared patients with atrial flutter after CABG (group A) to those without atrial flutter (group B). Transthoracic echocardiography (TTE) and cardiac catheterization were performed in all patients. All the stable patients had scintigraphy using thallium-201 (Tl-201) and technetium-99m (Tc-99m) before surgery to evaluate the viability of the myocardium. Anesthetic medication was similar in all patients. Median sternotomy was performed in all patients. Cardiopulmonary bypass (CPB) with a roller pump was established after antico-

agulation with heparin (3 mg/kg), and activated clotting time was maintained above 400 seconds. CPB was instituted using ascending aortic cannulation and venous cannulations of the superior and inferior vena cavae; a standard circuit was used. Systemic temperature was allowed to decline naturally during CPB.

In all patients, myocardial protection was achieved by using intermittent tepid hyperkalemic blood cardioplegia (a modified Calafiore method). In our modified Calafiore method, tepid blood cardioplegia with 2 mEq/L potassium chloride was infused at 800 ml/h until cardiac arrest. The first cardioplegia dose was 1,000 ml at a potassium concentration of 20 mEq/L. The second cardioplegia dose was 600 ml at a potassium concentration of 16 mEq/L. The third and subsequent cardioplegia doses were 600 ml at a potassium concentration of 12 mEq/L. Our cardioplegia strategy is as follows. We selected antegrade cardioplegia in cases with 75% stenosis or less. In cases with over 90% stenosis, we selected retrograde cardioplegia, which involved opening the right atrium and placing a retrograde cannula directly in the coronary sinus (open technique) for patients with stenosis of the right coronary artery (RCA) in the presence of viable myocardium. We did not open the right atrium for retrograde cardioplegia (blind technique) for patients with stenosis of the RCA with no viable myocardium. In the case of intact RCA, we selected the blind technique for retrograde cardioplegia. When retrograde cardioplegia via the blind technique was selected, we combined this with antegrade cardioplegia. The retrograde cannula used had a rigid stylet and a manually inflated balloon.

In the blind technique, a 3-0 purse-string suture is placed in the low right atrial wall, and the cannula was introduced through a stab incision. In the open technique, we snared the superior vena cava and the inferior vena cava, made an incision to the right atrium, and placed a 3-0 purse-string suture around the coronary sinus to prevent reflux. We used 4-0 prolene to close the right atrium. Heparin was reversed by protamine (2.5 mg/kg) at the termination of CPB. The patients without arrhythmia did not have prophylactic medication after surgery. Heart rate and rhythm were continuously monitored and displayed on a screen with an automated arrhythmia detector during the first 10 days after surgery. Patients who experienced some arrhythmias were monitored more closely. Twelve-lead electrocardiogram (ECG) recordings were performed before surgery, one hour after surgery, daily until the third postoperative day (POD), and twice a week until hospital discharge. Postoperative TTE was performed

on the eighth to 12th postoperative days. Before hospital discharge, at least one 24-hour Holter ECG was performed.

Definitions

The “number of diseased vessels” was based on the presence of over 50% stenosis. “Left main trunk” was defined as the presence of over 50% stenosis of the left main trunk (LMT). “Severe stenosis” was defined as the presence of over 90% stenosis of the left anterior descending artery (LAD), left circumflex artery (LCX), and RCA, simultaneously. Clinical diagnostic criteria for old myocardial infarction (OMI) were a past history of myocardial infarction and Q wave of >0.04 ms in ≥ 2 leads and/or akinesis or dyskinesis of the ventricular wall by echocardiography. In this study, atrial flutter was defined as continuous atrial flutter recorded over 10 beats. “Electrical defibrillation” means electrical defibrillation or cardioversion within the operating theater after declamping the aorta, or in the intensive care unit or a hospital ward after surgery. “Hospital stay” was defined as the days following surgery. The mortality rate included operative deaths within postoperative days (POD) 30.

Statistical analysis

Univariate analysis was performed. Student's t test was performed to compare age, body surface area (BSA), preoperative left ventricular ejection fraction (LVEF), number of diseased vessels, preoperative creatinine, cross-clamp time, CPB time, esophagus temperature, and hospital stay. Welch's t test was performed to compare preoperative creatinine kinase muscle brain (CK-MB), number of grafts per patient, and postoperative CK-MB peak. Mann-Whitney's U test was performed to compare preoperative New York Heart Association Classification (NYHA class). Chi-square test was performed to compare sex, preoperative OMI, diabetes mellitus, hypertension, hyperlipidemia, LMT, severe stenosis, antegrade cardioplegia, retrograde cardioplegia, direct cannulation to coronary sinus, postoperative atrial fibrillation, intraaortic balloon pumping (IABP), percutaneous cardiopulmonary support (PCPS), postoperative pericardial effusion, and postoperative electrical defibrillation. The variables found to be significant ($p < 0.05$) with univariate analysis (number of grafts per patient, cross-clamp time, direct cannulation to coronary sinus, and postoperative CK-MB peak) and the other variables, such as LMT, severe stenosis, OMI, preoperative CK-MB, CPB time, antegrade cardioplegia, and retrograde cardioplegia were

Table 1. Preoperative characteristics (univariate analysis)

Variable	Group A (n=16)	Group B (n=64)	p value
Age (years old)	65.3±9.9	63.9±8.9	NS
Sex (male)	14	48	NS
BSA (m ²)	1.67±0.17	1.61±0.16	NS
Diabetes mellitus	4	27	NS
Hypertension	8	35	NS
Hyperlipidemia	5	27	NS
NYHA class (I/II/III/IV)	2/10/4/0	8/33/12/11	NS
LVEF (%)	58.9±16.3	60.1±16.5	NS
No. of diseased vessels	3.62±0.72	3.31±0.99	NS
Left main trunk	5	29	NS
Severe stenosis	8	14	NS
OMI	4	25	NS
CK-MB (IU/l)	10.6±5.9	12.9±21.2	NS
Creatinine (mg/dl)	1.03±0.43	0.96±0.35	NS
Normal sinus rhythm	16	63	NS

Data are shown as mean±standard deviation.

NS: not significant (p>0.05)

BSA: body surface area, NYHA class: New York Heart Association classification,

LVEF: left ventricular ejection fraction, OMI: old myocardial infarction,

CK-MB: creatinine kinase MB

Table 2. Intraoperative clinical data (univariate analysis)

Variable	Group A (n=16)	Group B (n=64)	p value
No. of grafts per patient	2.94±0.25	2.64±0.70	NS
Cross-clamp time (min)	104.7±32.4	84.5±33.7	NS
CPB time (min)	196.0±49.0	176.9±61.4	NS
Antegrade cardioplegia	14	50	NS
Retrograde cardioplegia	16	53	NS
Direct cannulation to coronary sinus	14	29	0.0043
Esophagus temperature (°C)	34.1±0.9	34.2±1.3	NS

Data are shown as mean±standard deviation.

NS: not significant (p>0.05)

CPB time: cardiopulmonary bypass time

subjected to multivariate analysis. Discriminant analysis was performed to ascertain their independent roles of the variables. A value of p<0.05 was considered statistically significant.

Results

Eighty consecutive patients who underwent CABG fulfilled the criteria for inclusion in the study. No patient had documented atrial flutter before surgery. Sixteen patients (20%) had documented postoperative atrial flutter (group A). Preoperative characteristics are shown in Table 1. There were no significant differences between the groups in age, sex, BSA, diabetes mellitus, hypertension,

hyperlipidemia, preoperative NYHA class, preoperative LVEF, number of diseased vessels, LMT, severe stenosis, OMI, CK-MB, creatinine, or preoperative ECG. One patient in group B showed atrial fibrillation preoperatively, and had had hyperthyroiditis. He had sustained episodes of ventricular tachycardia postoperatively, and died from mesenteric artery occlusion 32 days postoperatively. This patient had had no postoperative episode of atrial flutter.

Intraoperative clinical data are shown in Table 2. There were no significant differences between the groups in CPB time, antegrade cardioplegia, retrograde cardioplegia, and esophagus temperature. Group A showed a larger number of grafts per patient and a longer cross-clamp time. Direct cannulation to the coronary sinus for retrograde

Table 3. Postoperative clinical data (univariate analysis)

Variable	Group A (n=16)	Group B (n=64)	p value
CK-MB peak (IU/l)	26.5±15.4	46.4±45.5	0.04
Atrial fibrillation	7	2	0.00000937
IABP	2	10	NS
PCPS	1	2	NS
Hemodialysis	0	2	NS
Pericardial effusion	1	4	NS
Electrical defibrillation	2	2	NS
Hospital stay (days)	32.3±15.2	29.7±14.0	NS
Mortality (%)	0	3.1	NS

Data are shown as mean±standard deviation.

NS: not significant ($p>0.05$)

CK-MB: creatinine kinase MB, IABP: intraaortic balloon pumping, PCPS: percutaneous cardiopulmonary support

Table 4. Multivariate analysis for atrial flutter

Variable	Group A (n=16)	Group B (n=64)	p value
Left main trunk	5	29	NS
Severe stenosis	8	14	NS
OMI	4	25	0.0072*
Preoperative CK-MB (IU/l)	10.6±5.9	12.9±21.2	NS
No. of grafts per patient	2.94±0.25	2.64±0.70	NS
Cross-clamp time (min)	104.7±32.4	84.5±33.7	NS
CPB time (min)	196.0±49.0	176.9±61.4	NS
Antegrade cardioplegia	14	50	NS
Retrograde cardioplegia	16	53	NS
Direct cannulation to coronary sinus	14	29	0.00383*
Postoperative CK-MB peak (IU/l)	26.5±15.4	46.4±45.5	0.0407*

Data are shown as mean±standard deviation.

*: A value of $p<0.05$ was considered statistically significant.

OMI: old myocardial infarction, CK-MB: creatinine kinase MB, CPB time: cardiopulmonary bypass time

cardioplegia was performed in 14 of 16 patients in group A and in 29 of 64 patients in group B ($p=0.0043$).

Postoperative clinical data are shown in Table 3. There were no significant differences between the groups in postoperative IABP, PCPS, hemodialysis, pericardial effusion, electrical defibrillation, hospital stay, and mortality. Postoperative CK-MB was increased in group B. The incidence of the postoperative atrial fibrillation was 43.8% (seven patients) in group A and 3.1% (two patients) in group B ($p=0.00000937$). There were two deaths in group B within 30 days of operation. One was an emergent case after acute myocardial infarction resulting in low-output cardiac failure, while the other was from pneumonia.

According to the multivariate analysis, both the lack of OMI and operation by direct cannulation to the coronary sinus for retrograde cardioplegia were independent

predictors of postoperative atrial flutter. Postoperative CK-MB was significantly elevated in group B (Table 4).

Sixteen patients (20%) had documented postoperative atrial flutter (group A). Postoperative data for group A are shown in Table 5. The mean onset of the atrial flutter was 6.88 ± 2.96 POD. Three of the 16 patients (18.8%) with postoperative atrial flutter had spontaneous conversion to normal sinus rhythm. Initial treatment of atrial flutter was by antiarrhythmic drugs (Ia, Ib, Ic), cardioversion, and overdrive pacing. Seven of the 16 patients (43.8%) with postoperative atrial flutter had conversion to normal sinus rhythm with these treatments. However, six of the 16 patients (37.5%) with postoperative atrial flutter, (i.e., 7.5% of the patients after CABG) showed sustained or repeated atrial flutter with some subjective symptoms. The main symptoms were malaise,

Table 5. Postoperative data (group A)

Onset of atrial flutter (POD)	6.88±2.96
Symptoms	Malaise, dysphoria, dyspnea, palpitation
Treatment	Antiarrhythmic drugs (Ia, Ib, Ic), cardioversion, overdrive pacing
Number of RFCA	6 cases
Type of the atrial flutter	Type I (common type)
Catheter ablation	Linear ablation to posterior isthmus
Days of ablation (POD)	32.0±20.8
Complication	None

Data are shown as mean±standard deviation.

RFCA: radiofrequency catheter ablation, POD: postoperative days

dysphoria, dyspnea, and palpitation. These patients required further treatment by electrophysiologic study (EPS) and RFCA. The mean time of ablation was 32.0±20.8 POD. These were all common-type atrial flutter (type I). The six patients underwent radiofrequency linear ablation for posterior isthmus without difficulty; they were discharged from the hospital without recurrence of atrial flutter.

Discussion

Between July 1999 and July 2000, the Cardiovascular Center of Aichi Prefectural Owari Hospital experienced a 20% overall incidence of atrial flutter (as distinct from atrial fibrillation) after CABG. On recognition of this problem, we sought to evaluate the cause of atrial flutter after CABG. While 10 of 16 cases in group A showed temporary atrial flutters that were curable without RFCA, 37.5% of the patients with postoperative atrial flutter (i.e., 7.5% of the patients after CABG) showed sustained or repeated atrial flutter with subjective symptoms.

Although there are many reports of the atrial fibrillation after CABG, atrial flutter after CABG is not well-documented. In this study, we did not pay much attention to postoperative atrial fibrillation. In unpublished data from the Aichi Prefectural Owari Hospital, we indicated that the incidence of postoperative atrial fibrillation was 10% in the cold blood cardioplegia cases, as opposed to 12.8% in tepid blood cardioplegia cases (using the modified Calafiore method). In one study, d'Amato et al.⁶ demonstrated that the incidence of postoperative atrial fibrillation after single vessel bypass surgery is reduced to a very low level after minimally invasive direct CABG when compared with standard CABG. They suggested that the elimination of atrial trauma from cannulation and atrial ischemia from cardiac arrest are the most likely factors. In their prospective randomized study, Ascione et

al.⁷ reported that CPB inclusive cardioplegic arrest is the main independent predictor of postoperative atrial fibrillation in patients undergoing coronary revascularization.

Preoperative factors

Age is consistently the independent factor most strongly associated with postoperative atrial fibrillation.^{1,2} Age associated changes in the atria, such as dilatation, muscle atrophy, and decreased conduction may explain the strong association.⁸ However, in the present study, there was no significant difference in age between the groups.

Patients with a history of atrial fibrillation are at increased risk for postoperative atrial arrhythmia.¹ Preoperatively, all the patients in this study, except one, demonstrated normal sinus rhythm. The single patient in the group B who showed atrial fibrillation preoperatively had had hyperthyroidism. He had sustained episodes of ventricular tachycardia postoperatively and died as a result of mesenteric artery occlusion on POD 32. He had experienced no episode of postoperative atrial flutter.

Neither the degree of ischemia nor the extent of coronary artery disease is a consistent predictor of postoperative atrial tachyarrhythmias.^{1,2} We defined severe stenoses as the presence of more than 90% stenosis of the LAD, LCX, and RCA, simultaneously. There were no significant differences between groups in severe stenosis nor in the presence of more than 50% stenosis of the LMT.

Intraoperative factors

Myocardial protection was designed to reduce the metabolic demand of the ventricular myocardium during the main surgical procedure. Studies of myocardial protection have failed to associate different rates of postoperative atrial tachyarrhythmias with the various techniques.⁹ In the present study, myocardial protection was achieved by using the intermittent tepid hyperkalemic cardioplegia (modified Calafiore method) for all patients. Postop-

erative CK-MB was higher in group B. This result may indicate that those patients with atrial flutter had been given superior myocardial protection.

We selected antegrade cardioplegia in cases of 75% stenosis or less. That is why only antegrade cardioplegia was sufficiently distributed in only these cases. In cases of over 90% stenosis, we selected retrograde cardioplegia because we had judged that antegrade cardioplegia would be insufficient. The veins accompanied with the posterior descending artery (4PD) and the right atrioventricular branch (4AV) drain into the great cardiac vein near the coronary sinus. In the blind technique, we placed the retrograde cannula deeply to prevent reduced flow. So in some cases, the cannula was placed deep in the junction, causing unequal distribution of cardioplegia. Borger et al.¹⁰⁾ reported that right ventricle perfusion was poor with retrograde cardioplegia, as evidenced by transesophageal echocardiography with injection microbubbles. One of our previous patients experienced severe right heart failure after CABG using retrograde tepid blood cardioplegia, independently, by the blind technique. We had inferred that the reason for this was an inadequate cardioplegia, because this patient had "severe stenosis", yet experienced no problem of anastomoses and cardioplegia during the surgery, and the postoperative coronary angiogram was perfect. After this, we changed our strategy to that used in this series. We opened the right atrium and placed the retrograde cannula directly in the coronary sinus (open technique) for patients with stenosis of the RCA with viable myocardium. We did not open the right atrium for retrograde cardioplegia (blind technique) for the patients with stenosis of the RCA with no viable myocardium. In cases of intact RCA, we selected the blind technique for retrograde cardioplegia. When we selected retrograde cardioplegia with the blind technique, we included the antegrade cardioplegia because of anxiety about the unequal distribution of cardioplegia. We isolated and placed the venous cannula in the superior vena cava and the inferior vena cava for all patients.

Myocardial perfusion imaging using Tl-201 has been widely used for the detection of dysfunction in ischemic but viable myocardium.¹¹⁻¹³⁾ Therefore we evaluated the viability of the myocardium with scintigraphy using Tl-201 and Tc-99m before and after surgery.

Postoperative factors

Pericardial inflammation or effusion is present after cardiac surgery before atrial fibrillation develops.¹⁴⁾ However, in the present study, there was no significant difference between the groups in postoperative pericardial ef-

fusion.

An important result of this study is that surgical damage inflicted by direct cannulation to the coronary sinus for retrograde cardioplegia (open technique) might be associated with atrial flutter after CABG. What, then, are the differences between the open technique and the blind technique? In cases in which the open technique were applied, we snared the superior vena cava and the inferior vena cava in concert with the blind technique. It has been said that mechanical contact around the superior vena cava is associated with postoperative unbalance of the autonomic nerve system. However, we snared the superior vena cava in the other procedures and did not experience an increased rate of atrial flutter. Secondly, there are some preoperative differences in the indication for the open technique, such as viability of the myocardium in the area of the RCA and stenosis of more than 90%. According to the multivariate analysis, the severity of the stenosis was not an independent predictor of postoperative atrial flutter. However, the lack of OMI was an independent predictor of postoperative atrial flutter. Additionally, postoperative CK-MB was significantly higher in group B (Table 4). These factors indicate that not only modes of surgical damage, such as the purse-string suture around the coronary sinus and the incision in the right atrium, but also good myocardial protection during the surgery (in addition to the preoperative viability of the myocardium) are related to the incidence of postoperative atrial flutter after CABG.

Olgin et al.¹⁵⁾ reported a flutter circuit involving the coronary sinus with conventional mapping. There are atrium muscles around the coronary sinus, as revealed histopathologically. In our data, all the sustained or repeated cases of atrial flutter were type I (common type) under postoperative EPS. Linear ablation to the posterior isthmus cured all six cases. This suggested to us that the surgical insult of the purse-string suture around the coronary sinus is more to blame than is the incision in the right atrium.

Atrial arrhythmias are most frequent in the first two to three days after cardiothoracic surgery.¹⁾ In particular, most episodes of atrial fibrillation occur within the first few days after surgery, with a peak incidence at POD 2 to 3.¹⁶⁾ Our data shows that postoperative atrial flutter occurred, on average at POD 6.9. We theorize that atrial flutter arises during the healing stage of the myocardium around the coronary sinus and is different from atrial fibrillation.

RFCA is widely used to cure symptomatic patients with common atrial flutter.^{17,18)} The cavotricuspid isthmus, ly-

ing between the inferior vena cava and the tricuspid annulus, is the common target of atrial flutter ablation.^{19,20} In this series, the six patients who underwent RFCA for posterior isthmus did so unproblematically; they were discharged from hospital without recurrence of atrial flutter. RFCA was seen to be safe, effective, and reliable treatment for curing patients with atrial flutter after CABG. The importance of postoperative atrial flutter is the patient's symptoms and psychological anxiety, and delayed discharge due to the need for additional monitoring and treatment. Proper monitoring and the treatment without delay leads to better outcomes. If atrial flutter goes untreated, it can be fatal problem.

Some limitations of this study should be noted. First of all, the number of the patients enrolled was relatively small. Secondly, this study was a retrospective and observational study. The number of cases done using off-pump bypass in our institute is increasing, so it is difficult to evaluate this situation. However, we endeavor to reconsider the open method surgical technique, as well as our strategy for cardioplegia, by which we seek to reduce postoperative atrial flutter. In conclusion, the incidence of atrial flutter after CABG was influenced by surgical damage inflicted by the direct cannulation to the coronary sinus for retrograde cardioplegia. Furthermore, the preoperative viability of myocardium (in addition to good myocardial protection during the surgery) seems also to be related to increases in postoperative atrial flutter. RFCA proved to be a safe, effective, and reliable treatment for reversing atrial flutter following CABG.

Definitive conclusions await the outcome from a larger number of patients in the long term.

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