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Research Article

Echocardiography Parameters Predicting Postoperative Atrial Fibrillation: Their Influence on Early Left Atrial Remodelling and Right Ventricular Function after Heart Valve Surgery

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Abstract

Objective: To identify relationship between echocardiographic parameters variations and risk of postoperative atrial fibrillation (POAF) after cardiac valve surgery.

Design: Prospective. Case-control study.

Setting: A university hospital.

Participants: We analysed the incidence of POAF in 90 patients undergoing elective heart valve surgery. POAF was considered when episodes equal or longer than 5 minutes, and those under 5 minutes with hemodynamic disturbances.

Interventions: None.Measurements and main results: POAF incidence was 36.7%. Preoperative echocardiographic study showed higher systolic pulmonary pressure (p: 0.047) and longer atrial electromechanical interval (AEI) (0.049) in POAF group. Postoperative echocardiographic evaluation revealed higher TAPSE decreasing related with preoperative values (8.18±4.33 mm in No-POAF vs. 10.35±3.83 mm in POAF group) (p: 0.026). In multivariate logistic regression POAF correlated with age> 65 years (p: 0.007) OR: 4.80; IC 95% (1.52-15.14), longer preoperative AEI (p: 0.042) OR: 1.029 IC 95% (1.001-1.059), higher TAPSE reduction (p: 0.040) OR: 1.15 IC 95% (1.006-1.316) and postoperative left atrial volume index> 36 ml / m2 (p = 0.0203) OR: 3.63; 95% CI (1.23-11.92).

Conclusions: After heart valve surgery, POAF favoured right ventricular dysfunction (evidenced by higher postoperative TAPSE decreasing) and impaired early left atrial remodelling. In older patients and those with preoperative longer AEI, biatrial pacing and pharmacological prophylaxis might prevent these undesirable POAF effects.

Keywords: atrial fibrillation; echocardiography; cardiac surgery

Introduction

Up to 50% of patients undergoing heart valve surgery presents postoperative atrial fibrillation (POAF).[1-3] Atrial structural remodeling is considered a risk factor for their appearance. Variations in some echocardiographic parameters have been related to increase risk for atrial fibrillation (AF) after coronary artery bypass graft (GABG) surgery. Longer A-wave and atrial electromechanical interval (AEI), increased E/A ratio, and higher diameter and indexed left atrial volume were more prevalent in patients suffering from POAF.[4-6] Patients with preoperative abnormal right ventricular performance index were more likely to present POAF after GABG.[7] Anyway, their predictive value has not been adequately validated after heart valve procedures.

Cardiac surgery leads to atrial remodeling due to the cumulative effect of fibrosis, scarring, ischemia and myocite necrosis.[8-10] In patients with heart valve disease, these factors were associated with increased atrial volume and diameter.[8-10]

We aimed to evaluate characteristics and predictive value of echocardiographic parameters associated with POAF in patients undergoing valve surgery. Their preoperative identification would permit to apply prophylactic measures in order to prevent POAF and its related complications in this group of patients.

Materials and Methods

Clinical and echocardiographic records were prospectively analyzed in 90 patients with primary diagnosis of heart valve disease (isolated or associated with coronary artery disease) satisfying requirements for heart surgery under cardiopulmonary bypass (CPB). *Informed consent* was *obtained* from *each patient* and the *study protocol, conforms* to the *ethical guidelines* of the *1975 Declaration* of *Helsinki*, as *reflected* in a *priori approval* by the *institution's human research committee.*

Previous episodes of AF, preoperative beta-blocker therapy, severe left ventricular dysfunction (left ventricular ejection fraction< 30%), renal failure (creatinine levels> 2 mg/dl) or urgent surgery were considered exclusion criteria.

Correlation between preoperative and postoperative echocardiographic data and prediction of POAF was included as primary end point. We considered POAF episodes equal or longer than 5 minutes and those less than 5 minutes, with hemodynamic disturbances. Clinical and echocardiographic variables were preoperatively collected. After surgical procedure, all patients had continuous electrocardiographic monitoring until seventh POD (inclusive). Events related to cardiac rhythm disturbances were recorded daily and analyzed by a cardiologist independent of cardiac patient clinical management.

Before hospital discharge (sixth POD) all patients underwent postoperative transthoracic echocardiography with Doppler imaging analysis [Vivid 7; GE Medical Systems, Vingmed, Norway].

Echocardiographic methodology (definitions)

Atrial Electromechanical Interval (AEI): time (in milliseconds) from the onset of electrocardiographic P wave to the beginning of atrial systole (movement backward in the atrioventricular plane) measured in the lateral face of left atrium. Four chambers were obtained in apical view, from the mitral lateral ring in transthoracic echocardiography and measured as the average of 3 cardiac cycles.A-wave: late peak ventricular filling velocity during atrial systole (m/s). It was measured using pulse-wave Doppler across the mitral valve plane in 4-chamber view and calculated from the average speed of 5 consecutive beats in M-Mode.

E-wave: doppler peak wave velocity during early diastolic ventricular filling.

E/A ratio: ratio of the peak early (E-wave) and late (A-wave) ventricular filling velocities.

TAPSE (tricuspid annular plane systolic excursion) assessed with M-mode in an apical 4-chamber view (0º angle related to right ventricular free margin) describes longitudinal right

ventricular myocardial shortening. It measure the amount of longitudinal displacement of tricuspid annulus at peak-systole. (Normal value > 16 mm).

Left atrial measures: Left atrial linear dimension was measured in 2-D mode and parasternal long-axis view and left atrial volume, in apical 4-chamber view at the maximal atrial dimension, using modified Simpson's rule.

Statistical methods

Statistical analysis was conduced with SPSS 22.0 software. Quantitative variables were expressed as mean ± standard deviation or as median, for asymmetric distributions. Qualitative variables were expressed as absolute value and percentage. Association between variables were identified using χ^2 or Fisher exact test when qualitatives and t of Student or U of Mann Whitney in quantitatives. Association between risk factors and analyzed events in univariate analysis ($p < 0.2$) were introduced in logistic multivariate regression. We analyzed risk factors and actuarial survival free of atrial fibrillation using Cox logistic regression proportional risk model and Kaplan–Meier test. A p value less than 0.05 (2-tailed) was considered significant.

Results

90 patients were prospectively recruited between 2011 and 2013. Incidence of POAF was 36.7 %. In all patients, POAF developed during 2º POD, with a median of 45 hours after surgery. Clinical, demographic and surgical variables are detailed in Tables 1 and 2.

VARIABLE	NO POAF GROUP n (%)	POAF GROUP n (%)	p
Female sex	19 (33.3)	12 (36.4)	0.711
Smokers	9 (15.8)	3 (9.1)	0.524
Arterial hypertension	33 (57.9)	19 (57.6)	0.976
Peripheral arteriopathy	0 (0)	0 (0)	-
Diabetes	8 (14)	10 (30.3)	0.063
Dislipemia	26 (45.6)	11 (33.3)	0.254
Renal failure	0 (0)	0 (0)	-
COPD	8 (14)	3 (9.1)	0.740
Previous AMI	0 (0)	0 (0)	-
Obesity (BMI>30)	18 (31.6)	7 (21.2)	0.290
Stroke	1 (1.8)	1 (3)	1
Left main coronary disease	0 (0)	0 (0)	-
NYHA III-IV	11 (19.3)	5 (15.2)	0.620
Moderate left ventricular dysfunction	3 (5.3)	3 (9.1)	0.483
Preoperative treatment:			
□ Digoxin	1 (1.8)	0 (0)	1
□ ACE inhibitors	26 (45.6)	19 (57.6)	0.274
□ Calcium antagonists	2 (3.5)	1 (3)	1
□ Atorvastatin	27 (47.4)	20 (60.6)	0.226
	Mean (SD)	**Mean (SD)**	
Age (years)	63.57 (12.61)	71.64 (7.21)	**0.001**
Weight (kg)	74.66 (14.7)	72.46 (10.24)	0.408
Height (cm)	163.46 (9.83)	162.75 (10.89)	0.746

COPD, chronic obstructive pulmonary disease; AMI: Acute myocardial infraction; BMI: body mass index; ACE: angiotensin-converting enzyme; SD: standard deviation.

Table 1. Demographic profile and clinical characteristics.

PERIOPERATIVE VARIABLES	NO POAF GROUP n (%)	POAF GROUP n (%)	p
Valve surgery + CABG	3 (5.3)	6 (18.2)	0.069
Mitral valve replacement	3 (5.3)	2 (6.1)	0.356
Aortic valve replacement	54 (94.7)	30 (90.9)	
Mitro-aortic valve replacement	0 (0)	1 (3)	
Bicaval canulation	3 (5.3)	3 (9.1)	0.665
PROSTHESIS			
Mechanical	32 (56.1)	8 (24.2)	0.006
Bioprosthesis	28 (38.6)	34 (72.7)	
Valve repair	3 (5.3)	1 (3)	
DRUGS AFTER SURGERY			
No	17 (31.5)	5 (15.2)	0.418
Inotropics (0,5-10 mcg/kg/min)	9 (15.8)	5 (15.2)	
Inotropics (> 10 mcg/kg/min)	2 (3.5)	2 (6.1)	
Vasodilators	2 (3.5)	4 (12.1)	
HEART RHYTHM AFTER DECLAMPING			
Sinus	55 (96.5)	32 (97)	0.147
Atrial fibrillation	0 (0)	1 (3)	
Atrio-ventricular block	2 (3.5)	0 (0)	
	Mean (SD)	Mean (SD)	
CBP time (min)	102.72 (43.57)	100.18(31.89)	0.575
Aortic clamp time (min)	78.79 (38.26)	76.12 (28.10)	0.663
Electrical cardioversion	2.15 (2.57)	2.09 (2.38)	0.901
RBC transfusion (units)	1.18 (2.95)	1.6 (1.5)	0.010

Table 2. Operative data.

CABG: coronary artery bypass graft; CBP: cardiopulmonary bypass; RBC; reed blood cell.

Patients who developed *POAF* were *significantly older*, with a median of 74 years vs. 66 years in patients who did not present arrhythmia. No clinical or surgical analyzed risk factors were related with atrial arrhythmia except an increased trasfusional demands. In multivariate logistic regression, POAF correlated with age> 65 years (p: 0.007) OR: 4.80; IC 95% (1.52-15.14), longer preoperative AEI (p: 0.042) OR: 1.029 IC 95% (1.001-1.059), higher TAPSE postoperative reduction (p: 0.040) OR: 1.15 IC 95% (1.006-1.316) and postoperative left atrial volume index> 36 ml / m2 (p = 0.0203) OR: 3.63; 95% CI (1.23-11.92).

In all patients, left atrial diameter (LAD) and left atrial volume index (LAVI) were increased and pulmonary hypertension (at least moderate) was identified. Preoperative echocardiography parameters showed a certain grade of diastolic dysfunction, which seems more related to advanced age than to hypertension. Although E/A ratio values were normal, A-wave was greater than E-wave. Age ≥ 65 years correlated with an increased A-wave (p: 0.035) RR 5.56 (95% CI 1.12-28.82) and increased systolic pulmonary artery pressure (p: 0.016) RR 4.47 (95% CI 1.25-15.93).

When comparing both groups, a significant increasing in AEI delay, systolic pulmonary artery pressure and gradient between right atrium and ventricle were observed in POAF group (Table 3).

VARIABLE	PREOPERATIVE			POSTOPERATIVE		
	NO-POAF GROUP Mean (SD)	POAF GROUP Mean (SD)	p	NO POAF GROUP Mean (SD)	POAF GROUP Mean (SD)	p
LVEF (%)	62.98 (7.62)	64.87 (7.37)	0.314	62.25 (8.63)	63.55 (8.54)	0.505
PASP (mm Hg) TAMG (mm Hg.)	34.27 (5.31) 55.81 (23.26)	37.18 (5.36) 54.92 (21.81)	0.047 0.268	34.62 (4.95) 21.05 (8.80)	39 (6.58) 21.02 (6.54)	0.005 0.988
TMMG (mm Hg.)	5.19 (3.41)	3.63 (1.13)	0.462	4.63 (2.13)	4.93 (2.17)	0.838
Aortic area (cm^2)	0.83 (0.63)	0.89 (0.45)	0.057	1.45 (0.28)	1.57 (0.67)	0.347
Mitral area (cm^2)	2.73 (1.51)	2.56 (0.59)	0.831	2.64 (0.70)	3.08 (1.41)	0.531
LAD (mm)	42 (6)	44 (6)	0.230	42.19 (5.91)	44.92 (4.91)	0.052
A wave (m/s)	0.96 (0.35)	1.05 (0.34)	0.209	1.18 (1.82)	1.07 (0.34)	0.754
E wave (m/s)	0.84 (0.32)	0.87 (0.25)	0.666	1.06 (0.88)	1.14 (0.28)	0.663
AEI (ms)	65.53 (17.43)	73.42 (17.86)	0.049	55.46 (20.18)	61.32 (16.81)	0.214
LAV (ml)	74.37 (28.63)	75.69 (26)	0.581	66.24 (21.28)	76.61 (20.41)	0.040
LAVI (ml/m^2)	41.35 (16.20)	42.99 (15.25)	0.637	36.98 (12.04)	43.45 (10.66)	0.017
E/A ratio	0.92 (0.39)	0.93 (0.45)	1.000	1.14 (0.45)	1.14 (0.34)	0.973
TAPSE	21.09 (3.26)	21.95 (3.58)	0.280	12.77 (2.90)	11.78 (2.42)	0.118
RA-RV gradient (mm Hg)	25.25 (5.72)	28.39 (4.79)	0.034	25.56 (5.63)	28.80 (6.15)	0.040

Table 3. Preoperative and postoperative echocardiographic variables.

LVEF: left ventricular ejection fraction; PASP: pulmonary artery systolic pressure; TAMG; transaortic mean gradient; TMMG: transmitral mean gradient; LAD: left atrium diameter; AEI: atrial electromechanical interval: LAV: left atrium volume; LAVI: left atrium volume index; TAPSE: tricuspid annular plane systolic excursion; RA-RV: right atrium-right ventricleHowever, there was no difference in A-wave velocity or in E/A ratio, traditionally associated with atrial remodeling and proposed as incremental risks factor for POAF.

In postoperative echocardiogram, decrease in LAD and LAVI were observed, nevertheless, early atrial remodeling was only statistically significant in No-POAF patients. When calculating (for each patient), the difference between pre and postoperative LAVI, this value decreased in No-POAF group and increased when POAF was present (3.62 ± 8.15 ml / m2 vs. -1.09 ± 11.88 ml / m2, respectively) (p = 0.043). Average rate of echocardiographic left atrial reverse remodeling was 5.82 ± 20% in No-POAF vs. -7.67 ± 30.2% in POAF group (p: 0.002). In conclusion, there was no significant reverse atrial remodeling when POAF appeared. Early atrial remodelling was significantly reduced over 65 years (3.22±8.4 ml/m2 vs. -2.4±10.04 ml/m2) (p: 0.036) and was not influenced by age under 65. No influence of pulmonary hypertension was observed on atrial remodeling. Right ventricle functionality was indirectly assessed by TAPSE measurement. Concerning preoperative values, TAPSE decreased in both groups with no significant differences (Table4).

VARIABLE	NO-POAF GROUP			POAF GROUP		
	PREOP Mean (SD)	POSTOP Mean (SD)	p	PREOP Mean (SD)	POSTOP Mean (SD)	p
E wave (m/s)	0.9 (0.24)	1.1 (0.28)	<0.0001	0.9 (0.37)	1.1 (0.45)	0.08
AEI (ms)	65.7 (17.9)	55.1 (20.2)	0.001	75 (16.8)	61 (17.09)	0.004
E/A ratio	0.9 (0.37)	1.1 (0.45)	<0.0001	0.84 (0.35)	1.1 (0.33)	<0.0001
TAPSE	21 (3.16)	12.8 (3.03)	<0.0001	22.3 (3.54)	11.9 (2.39)	<0.0001

Table 4. Preoperative and postoperative comparison between E-wave, E/A ratio, AEI and TAPSE in POAF and No-POAF patients.

However, when analyzing the difference between pre and postoperative TAPSE values in each patient, *showed* statistically *significant reduction when* POAF occurred (10.35 ± 3.83 mm vs. 8.18 ± 4.33 mm, respectively) (p = 0.026). Although TAPSE decrease is independent of patient age, POAF impact over right ventricular function was higher in patients younger than 65 years (11.42±4.27 vs. 7.36±5) (p: 0.083). No other factors influencing TAPSE reduction were found and no changes related to age were observed in the rest of echocardiographic variables considered.

Atrial fibrillation during follow-up

Follow-up was conducted in 100% of survival patients. Median follow-up was 49 months (interval: 32-64 months). Only 1 patient died during follow-up due to a late prosthetic valve endocarditis. No mortality or neurological events related with new onset AF were identified. In 14 patients (15.9%) at least one episode of AF was observed during follow-up. AF was more frequently detected in patients with previous POAF episodes (29.1% vs. 8.9%, respectively) (p: 0.031). In multivariate analysis, age (p: 0.0218) OR: 1.11; IC 95% (1.01-1.22) and longer postoperative AEI (p: 0.0273) OR: 1.07 IC 95% (1.0078-1.14), were identified as incremental risk factors for AF during follow-up.

Discussion

The interpretation of the influence of preoperative echocardiographic variables on the appearance of POAF in patients with valve disease is complex, since we analyze values unusual among the normal population. In heart valve disease, preoperative diastolic dysfunction is considered frequent and manifested by an increased A-wave value, although the E/A ratio maintains within the normal range. Relationship between diastolic dysfunction and impaired atrial electrical activity is uncertain, but it is known that risk of AF is higher when E-wave is greater than 0.84 m/s, as baseline data in our sample.[11] Furthermore, increased baseline atrial volumes and mean values of pulmonary systolic pressure were common. These changes were especially significant over 65 years, although in the logistic regression analysis, only the longer duration of AEI was an independent preoperative risk factor for POAF appearance.

In the same way than previous works, age over 65 was main risk factor for POAF after heart valve surgery in our population.[9,13-15] Ageing is associated with atrial fibrosis and structural changes[9,15,16] which impair and/or slow stimulus conduction between sinus and atrioventricular node and prolong AEI.[6,8] Increasing in E and A-waves velocity, E/A ratio and LAD, positively correlated with higher incidence of AF, both among general population and after cardiac surgery.[4-6,17,18] However, our results suggest that valve disease and age-related *changes* in *atrial* function could modify these preoperative values[8-10,19] invalidating their effectiveness as predictors of POAF.

It seems that, after heart valve surgery, POAF genesis is mainly related to atrial conduction impairment, instead of to a contractility disorder. Prolonged preoperative AEI is the only independent predictor of POAF. Atrial electromechanical delay reflects structural atrial changes that retard transmission of intra- and interatrial electrical impulse and might be due to diastolic dysfunction. AEI duration among 115-147 ms is related to adequate prediction of POAF after coronary artery bypass graft surgery.[4,6,17,20] In the analyzed population, AEI was significantly lower, probably due to the exclusion of patients with preoperative beta-blocker therapy (with a recognized prophylactic effect on POAF) in our sample.[4,6,17,20] Postoperative echocardiographic controls showed an early reduction of LAVI and LAD after valve surgery.[21,22] Early atrial reverse remodeling was also appreciated in our sample, but only among No-POAF patients. This phenomenon was significantly higher among elderly patients. Consequently, in patients older than 65 with preoperative increased A-wave and AEI, it seems appropriate to apply pharmacological POAF prophylaxis and/or consider biatrial pacing during the first 48 hours after surgery. Right ventricular dysfunction is a strong predictor of developing AF in patients with acute decompensate heart failure.[23] Shymony[7] has confirmed the association between impaired right diastolic ventricular contractility and increased risk of POAF after CABG. No influence of preoperative right ventricular function on POAF was demonstrated in our analyzed population; nevertheless, using each patient as a case and control, TAPSE decreasing after surgery was significantly related to POAF. It is difficult to establish the real cause-effect relationship between these two variables. The etiology of postoperative right ventricular dysfunction is considered multifactorial: basal substrate altered by preoperative pulmonary hypertension, ischemia during CPB, trauma associated to surgical procedure, postoperative changes related to volume overload, effects of inotropic drugs…etc.[24,25] Probably, postoperative TAPSE decreasing is a secondary effect of previously mentioned factors. However, considering that its effect was higher in younger patients, in whom POAF was less frequent, we might conclude that POAF contributed to postoperative right ventricular function impairment and was associated to increased TAPSE decline after heart valve surgery.

According to aforementioned data, POAF after valve surgery in patients with preoperative sinus rhythm, impaired early atrial reverse remodeling and contributed to postoperative right ventricular dysfunction, not only in aged patients.

Conclusions

Age is main risk factor for POAF related to heart valve surgery. The etiology of this disorder is supposed to be multifactorial. Echocardiographic parameters previously identified as risk factors for AF were ineffective predictors after heart valve surgery in our population. Increased AEI reflected an atrial conduction disorder and was the only preoperative risk factor identified in this group of patients.

Study limitations

This is a single-center clinical trial, conducted in a small group of patients. The findings may be affected by reduced sample size.

References

1. Mariscalco G, Biancari F, Zanobini M et al. Bedside tool for predicting the risk of postoperative atrial fibrillation after cardiac surgery: the POAF score. J Am Heart 2014 2014 Mar 24;3(2):e000752.
2. Shen J, Lall S, Zheng V et al. The persistent problem of new-onset postoperative atrial fibrillation: a single-institution experience over two decades. J Thorac Cardiovasc Surg 2011; 141: 559–570.
3. Mariscalco G, Engström KG. Atrial fibrillation after cardiac surgery: risk factors and their temporal relationship in prophylactic drug strategy decision. Int J Cardiol 2008; 129: 354–362.
4. Elawady MA, Bashandy M. Clinical and echocardiographic predictors of postoperative atrial fibrillation. Asian Cardiovasc Thorac Ann 2013; 22: 655-659.
5. *Osranek M, Fatema K, Qaddoura F et al. L*eft atrial volume predicts the risk of atrial fibrillation after cardiac surgery: a prospective study. *J Am Coll Cardiol 2006; 48: 779-86.*
6. Roshanali F, Mandegar MH, Yousefnia MA et al. Prediction of atrial fibrillation via atrial electromechanical interval after coronary artery bypass grafting. Circulation 2007; 116: 2012-2017.
7. Shimony A, Afilalo J, Flynn AW et al. Usefulness of right ventricular dysfunction to predict new-onset atrial fibrillation following coronary artery bypass grafting. Am J Cardiol 2014; 113: 913-918
8. Casaclang-Verzosa G, Gersh BJ, Tsang TS. Structural and functional remodelling of the left atrium. Clinical and therapeutic implications for atrial fibrillation. J Am Coll Cardiol 2008; 51:1-11
9. Almassi GH, Schowalter T, Nicolosi AC et al. Atrial fibrillation after cardiac surgery. A major morbid event? Ann Surg 1997; 226: 501-513.
10. Hatam N, Aljalloud A, Mischke K et al. Interatrial conduction disturbance in postoperative atrial fibrillation: a comparative study of P-wave dispersion and Doppler myocardial imaging in cardiac surgery. J Cardiothorac Surg 2014; 9:114-123.
11. Stork T, Mockel M, Danne O et al. Age-related hemodynamic changes during diastole: a combined M-mode and Doppler echo study. Int J Card Imaging 1990; 6: 23-30.
12. Schnabel RB, Sullivan LM, Levy D et al. Development of a risk score for atrial fibrillation (Framingham Heart Study): a community-based cohort study. Lancet 2009; 373: 739-745.
13. Zaman AG, Archbold A, Helft G et al. Atrial fibrillation after coronary artery bypass surgery: a model for preoperative risk stratification. Circulation 2000; 101; 1403-1408.
14. Ahlsson A, Fengsrud E, Bodin L et al. Postoperative atrial fibrillation in patients undergoing aortocoronary bypass surgery carries an eightfold risk of future atrial fibrillation and a doubled cardiovascular mortality. Eur J Cardiothorac Surg 2010; 37:1553-59.
15. Andrade J, Khairy P, Dobrev D et al. The clinical profile and pathophysiology of atrial fibrillation: relationships among clinical features, epidemiology, and mechanisms. Circ Res 2014; 114:1453-68
16. Auer J, Weber T, Berent R, Ng CK et al. Risk factors of postoperative atrial fibrillation after cardiac surgery. J Card Surg 2005; 20:425-31.
17. Takahashi S, Fujiwara M, Watadani K et al. Preoperative tissue doppler imaging-derived atrial conduction time can predict postoperative atrial fibrillation in patients undergoing aortic valve replacement for aortic valve stenosis. Circ J 2014; 78: 2173-2181
18. Spach MS. Mounting evidence that fibrosis generates a major mechanism for atrial fibrillation. Circ Res 2007; 101: 743-745.
19. Ari H, Ari S, Akkaya M et al. Predictive value of atrial electromechanical delay for atrial fibrillation recurrence. Cardiol J 2013; 20: 639-47.
20. DiDomenico RJ, Massad MG. Pharmacologic strategies for prevention of atrial fibrillation after open heart surgery. Ann Thorac Surg 2005; 79: 728–40
21. Hornero Sos F, Montero Argudo JA, Cánovas López S et al. Anatomic atrial remodeling after mitral valve surgery in permanent atrial fibrillation. Rev Esp Cardiol 2003; 56:674-81.
22. Hyllén S, Nozohoor S, Meurling C et al. Left atrial reverse remodeling following valve surgery for chronic degenerative mitral regurgitation in patients with preoperative sinus rhythm: effects on long-term outcome. J Card Surg 2013; 28: 619-26.
23. Aziz EF, Kukin M, Javed F et al. Right ventricular dysfunction is strong predictors of developing atrial fibrillation in acutely decompensate heart failure patients, ACAP-HF data analysis. J Card Fail 2010; 16:827-834.
24. Stein KL, Breisblatt W, Wolfe C et al. Depression and recovery of right ventricular function after cardiopulmonary bypass. Crit Care Med 1990; 18: 1197-1200.
25. Roshanali F, Yousefnia MA, Mandegar MH et al. Decreased right ventricular function after coronary artery bypass grafting. Tex Heart Inst J 2008; 35: 250-255.