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European Journal of Cardio-thoracic Surgery 34 (2008) 55–61

EUROPEAN JOURNAL OF CARDIO-THORACIC SURGERY

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Incidence and pathophysiology of atrioventricular block following mitral valve replacement and ring annuloplasty☆

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Received 27 August 2007; received in revised form 12 March 2008; accepted 31 March 2008; Available online 15 May 2008

Abstract

Background: In this retrospective study we evaluate the causative mechanisms underlying postoperative atrioventricular block (AVB) following mitral valve replacement and mitral valve annuloplasty. **Methods**: Between January 1990 and December 2003, 391 patients underwent mitral valve replacement or ring annuloplasty and quadrangular resection. Exclusion criteria were preoperative AV block, two or three valvular procedures, reoperations and procedures combined with coronary artery bypass grafting. The presence of the postoperative AVB was compared with preoperative and intraoperative variables. On 55 post-mortem specimens the relationship between the AV node, AV node artery and mitral valve annulus was investigated. **Results:** The mean age was 59 ± 14 years and 44% of patients were female. Postoperatively AVB occurred in 92 (23.5%) patients. AVB III was found in 17 (4%) patents, in whom a pacemaker was implanted within median interval of 4 days. Second degree AVB occurred and first degree AVB in five (1.3%) and in 70 (18%) patients respectively. In dry dissected human hearts in 23% of investigated cases the AV node artery was discovered to run close to the annulus of the mitral valve. **Conclusions:** Data collected in this study showed that, sotalol and amiodarone as well as a prolonged cross-clamp time may slightly influence the 23% incidence of postoperative AVB. The morphological investigation showed that the AV node artery runs in close proximity to the annulus in 23% of cases. We speculate that damage of the AV node artery may play a role in development of AVB.

Keywords: Mitral valve surgery; Mitral valve reconstruction; Postoperative rhythm disturbances; Atrioventricular block; Heart conducting system

1. Introduction

The risk of developing conduction disturbances after coronary artery bypass grafting or valve replacement has been well established from previous studies, leading to permanent pacemaker implantation in about 2–3% of patients [1–4] and in 10% of patients undergoing repeat cardiac surgery [5].

Mitral valve reconstruction has recently become the technique of choice in the treatment of patients with mitral regurgitation of degenerative origin [6,7]. This surgical technique is more complex and sometimes results in longer ischemic times. The longer intraoperative ischemia has been postulated as being responsible for the postoperative incidence of the AV node block. However, the mechanism of the postoperative AV node block is still not understood.

Furthermore, it is clear that the mitral valve annulus is anatomically in close proximity to the atrioventricular conduction system, particularly the posterior-medial commissure of the anterior mitral leaflet, which lies close to the atrioventricular node. Despite these anatomical arrangements, the risks of conduction disturbances and permanent pacemaker implantations after mitral valve replacement or reconstruction considering morphological damage of the conducting system have not yet been investigated.

This study examines the incidence, predictors, and the evolution of postoperative atrioventricular block (AVB), and addresses the need for permanent pacemaker implantation after mitral valve surgery considering the morphological and clinical predictors.

2. Methods

We report data from 391 consecutive patients who underwent mitral valve reconstruction, including both annuloplasty and quadrangular resection, or mitral valve replacement in the period between January 1990 and December 2003.

☆ Presented at the 21st Annual Meeting of the European Association for Cardio-thoracic Surgery, Geneva, Switzerland, September 16–19, 2007.

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1010-7940/$ – see front matter
doi:10.1016/j.ejcts.2008.03.051

The operative technique consisted of a median sternotomy with a conventional left lateral atriotomy in all patients. Cardiopulmonary bypass was established between the ascending aorta and bicaval venous cannulation. All procedures were performed under moderate systemic hypothermia (mean rectal temperature 28.4 °C ± 3.5) with combined anterograde and retrograde cardioplegia repeated every 30 min for myocardial protection, using both medium crystalloids and blood.

Patients with permanent or transient cardiac pacing, with preoperative AV block (n = 42), patients undergoing subaortic resection (n = 1), maze procedure (n = 1), and those with two (n = 2) or three valve procedure (n = 2) or additional tricuspid valve surgery (n = 2) were excluded. To assess conduction disturbances, a 12-lead electrocardiogram was recorded three times routinely 24 h before surgery, after surgery, immediately after admission to the intensive care unit (ICU) and as continuous routine monitoring once daily until admission to the wards. Patients in the wards were monitored continuously once daily if arrhythmia, bradycardia, or pacing from epicardic electrodes were present. On the day of discharge, a 12-lead electrocardiogram was also recorded. The diagnosis of a conduction disturbance designated as a left bundle branch block, right bundle branch block, left anterior hemiblock, first degree atrioventricular block (AVB I), second degree (AVB II), or third degree (AVB III) were determined according to the criteria published in the literature [8].

The presence of postoperative AVB was compared with preoperative variables including baseline characteristics, antiarrhythmic drugs taken before surgery, etiology and mechanism of mitral disease and with operative and postoperative variables (Tables 1–6.).

To assess the morphological relations between the mitral valve annulus, the AV node, His bundle and AV node artery anatomical studies were conducted on 55 human hearts. Hearts of persons aged between 6 and 70 years without any pathological alterations were investigated. To study the anatomy of the AV node artery, the left and the right coronary arteries were injected with the contrast material. Subsequently the origin of the AV node artery was determined by selective coronary angiograms. After the preserving procedure was completed, the run off of the AV node artery, the AV node and the His bundle were dissected. Doing this we designed to especially focus on the relationships between the mitral valve annulus and AV node, its artery and the His bundle.

Table 1
Conduction disturbances as compared following mitral valve replacement or mitral valve annuloplasty

Conduction disturbances	Patients
AVB I	70 (17.9%)
AVB II	5 (1.5%)
AVB III	17 (4.3%)
Right bundle branch block	18 (4.6%)
Left bundle branch block	10 (2.6%)
Left anterior hemi block	30 (7.7%)
Total	150 (38.3%)

Conduction disturbances after mitral valve replacement or after mitral valve annuloplasty (n = 391).

3. Statistical analysis

Results are expressed as mean ± standard deviation (SD). The association of preoperative, intraoperative, and postoperative factors with the occurrence of postoperative incidence of the AVB was evaluated with the use of the Mann–Whitney test, the Chi-square test and Fisher's exact test where appropriate. Factors significant in the univariate analysis were included into a stepwise logistic regression (forward selection) to identify independent predictors of postoperative AVB. A p value <0.05 was considered statistically significant.

4. Results

Patients undergoing MVA and MVR alone were included in this study; a total of 391 individuals. Patient ages ranged from 19 to 85 years (mean 59 ± 14 years): 220 (56.3%) were

Table 2
Preoperative data of the patients

Patient characteristics	AV block (n = 92, 23.5%)	No AV block (n = 299, 76.5%)	p-Value
Age (years)	57.6 ± 13.0	59.3 ± 13.8	ns
Female gender (n = 171)	35 (20.5%)	136 (79.5%)	ns
Diabetes mellitus (n = 26)	5 (19.2%)	21 (80.8%)	ns
Systemic hypertension (n = 131)	30 (22.9%)	101 (77.1%)	ns
Renal insufficiency (n = 95)	23 (24.2%)	72 (75.8%)	ns
Myocardial infarction (n = 13)	3 (23%)	10 (77%)	ns
Pulmonary hypertension (n = 151)	27 (17.9%)	124 (82.1%)	0.04
NYHA class			
I (n = 100)	30 (30%)	70 (70%)	ns
II (n = 182)	41 (22.5%)	141 (77.5%)	ns
III (n = 102)	21 (20.5%)	81 (79.5%)	ns
IV (n = 6)	0 (0%)	6 (100%)	ns
Ejection fraction			
EF < 35% (n = 7)	3 (42.8%)	4 (57.2%)	ns
EF 35–55% (n = 97)	24 (24.7%)	73 (75.3%)	ns
EF > 55% (n = 285)	64 (22.4%)	221 (77.5%)	ns

ns: Non-significant.

Table 3
Preoperative antiarrhythmic drugs

Drugs	AV block (n = 92, 23.5%)	No AV block (n = 299, 76.5%)	p-Value
Beta-blocker with sotalol (n = 249)	72 (29%)	177 (71%)	0.01
Amiodarone (n = 24)	11 (45.8%)	13 (54.2%)	0.01
Digoxin (n = 105)	11 (10.4%)	94 (89.5%)	<0.001
Sotalol (n = 142)	48 (33.8%)	94 (71.8%)	<0.001

Table 4
Preoperative electrocardiogram

Preoperative electrocardiogram	AV block (n = 92, 23.5%)	No AV block (n = 299, 76.5%)	p-Value
Atrial fibrillation (n = 6)	3 (50%)	3 (50%)	ns
Left branch block (n = 3)	1 (33.3%)	2 (66.7%)	ns
Complete right branch block (n = 11)	1 (9%)	10 (91%)	ns
Left anterior hemi block (n = 22)	7 (31.8%)	15 (68.2%)	ns

Table 5
Etiology and mechanism of mitral valve disease

Pathology of the mitral valve[a]	AV block (n = 92, 23.5%)	No AV block (n = 299, 76.5%)	p-Value
Barlow's disease (n = 7)	3 (42.9%)	4 (57.1%)	ns
Marfan disease (n = 1)	1 (100%)	0	ns
Degenerative (n = 296)	72 (24.3%)	224 (75.6%)	ns
Rheumatic (n = 59)	10 (16.9%)	49 (83%)	ns
Endocarditis (n = 62)	16 (25.8)%)	46 (69.7%)	ns
Hemodynamical effect of the mitral valve pathology			
Mitral valve stenosis (n = 76)	3 (3.9%)	73 (96.1%)	<0.001
Mitral valve insufficiency (n = 261)[b]	69 (26.4%)	192 (73.6%)	0.001

[a] Missing three values.
[b] Missing 53 values.

Table 6
Operative variable an the incidence of the AV block

Operative variables	AV block (n = 92, 23.5%)	No AV block (n = 299, 76.5%)	p-Value
Cardiopulmonary bypass time (min)	130 ± 67.5	121.7 ± 52.8	ns
Cross clamp time (min)	81 ± 41	55.2 ± 33.3	<0.001
Body temperature	27.7 ± 3.7	28.6 ± 3.5	ns
Procedure in ventricular fibrillation (n = 36)	9 (25%)	27 (75%)	ns
Mitral valve replacement (n = 237)	39 (16.4%)	198 (83.5%)	<0.001
Mitral valve reconstruction (n = 147)	52 (35.4%)	95 (64.6%)	<0.001

male and 171 (43.7%) were females. No patient had a history of cardiac surgery. According to NYHA classification 100 (25.6%) patients were in NYHA class I, 182 (46.7%) patients in NYHA class II, 102 (26.2%) patients in NYHA III and only 6 (1.5%) in NYHA IV class (Table 2).

4.1. Postoperative conduction disturbances

Postoperative conduction disturbances occurred in 142 patients (38.3%). The different types of conduction disturbances are described in Table 1, with AVB being more frequent (23.5%) than bundle branch block (14.8%). Third-degree AVB was found in 17 patients in whom a pacemaker was implanted within a median time of 4 days (range 0–36 days) and was done by the cardiac surgeon in the operating room.

Second-degree AVB occurred in five patients immediately after surgery; where one of these five patients was transient Mobitz-type I, and was resolved before discharge. The mean length of the ICU stay was 3.3 ± 3.3 days. Isolated AVB I was diagnosed in 70 patients in the immediate postoperative period, and most was permanent and present at discharge. Right bundle branch block was found in 18 patients, left bundle branch block in 10, left anterior hemiblock in 30 immediately after surgery. The mentioned bundle branch blocks were of transient nature and resolved before discharge.

The presence of postoperative AVB was compared with variables listed in Tables 1–6.

4.2. Patients' baseline data

Preoperative patient data for both the postoperative AVB group and the no-AVB group are listed in Table 2. Univariate statistical analysis demonstrated that the pulmonary hypertension was significantly less frequent in patients with AVB

($p = 0.04$). The preoperative antiarrhythmic drugs used by patients during the month preceding surgery are shown in Table 3. All patients were still taking these drugs 48 h before surgery. No patient took calcium channel blockers. No patient with postoperative advanced heart block was treated preoperatively with amiodarone. Therapy with atrioventricular nodal blockers administered before the operation was discontinued after development of postoperative AVB II and AVB III. Most of the patients were preoperatively treated with beta-blockers ($n = 249$) where 29% of the patients in the beta-blocker group developed postoperative AVB. Sotalol was the most frequently used beta-blocker (57%, $n = 142$), however (Table 3). The stepwise multiple logistic regressions demonstrated, with modest effect, that sotalol may increase the risk of incidence of postoperative AVB. The preoperative usage of amiodarone also increases, with modest effect, the incidence of postoperative AV block (Table 3). In this group the postoperative AV block developed in 45.8% ($n = 11$) of patients, none of them developed a high-grade block.

Preoperative electrocardiographic findings demonstrated no differences between the AVB and non-AVB groups (Table 4) for both frequencies of *de novo* atrial fibrillation and/or conduction disturbances. The etiology of mitral disease is described in Table 5. Degenerative morphological alteration was the predominant cause for surgical intervention in both groups. In this group no predicted factors were found which may increase the incidence of the postoperative AV block. Mitral valve stenosis ($n = 76$) was significantly less frequent ($p < 0.001$) in patients with AVB.

4.3. Operative variables

Operative variables are summarized in Table 6. Cross-clamp time ($p < 0.001$) was significantly longer in the AVB group during the surgical procedure. The number of patients having a mitral valve reconstruction was higher in the group without the AVB. Similar distribution was found in the group with the mitral valve replacement (Table 6).

4.4. Postoperative variables

There were two perioperative deaths (overall operative mortality rate 1.7%): none in the AVB group and two in the group without AVB. No deaths were related to heart block. One death was due to stroke and subsequent coma and one was related to heart failure. Postoperatively, 180 patients developed atrial fibrillation (46.5%) (those patients with preoperative atrial fibrillation were not included). Postoperative atrial fibrillation with postoperative AVB occurred in 34 (37.4%) patients, but it was transient in all cases. The diagnosis of AVB was then possible during sinus rhythm in most patients before the onset of atrial fibrillation. Overall the incidence of postoperative complications in the AVB group was comparable to the group without AVB.

4.5. Multivariate analysis

The preoperative admission of sotalol and amiodarone as well as a prolonged cross-clamp time were identified as independent risk factors for the incidence of postoperative AVB, whereas the preoperative admission of digoxin proved to be an independent protective factor (Table 7).

Table 7
Multivariate analysis of the incidence of the AV block

Predictor	Odds ratio (95% CI)	*p*-Value
Amiodarone	3.55 (1.43–8.80)	0.006
Digoxin	0.41 (0.20–0.84)	0.015
Sotalol	1.86 (1.11–3.12)	0.019
Cross-clamp time	1.018 (1.011–1.025)	<0.001

4.6. Follow-up

Postoperative follow-up ranged from 6 to 48 months (mean 36 months). There were no recurrences or worsening of conduction disturbances, no further pacemaker implantation, and no late deaths among patients with postoperative AVB.

5. The anatomy of the atrioventricular artery

We found 42 (76.4%) specimens in which the right AV node artery (Fig. 1A and B) and 13 (23.6%) specimens in which the left AV node artery vascularized the AV node. From its origin, the artery ascends onto the superior process of the left ventricle toward the AV node and His bundle (Fig. 1A and 1B). Considering its topography to mitral and tricuspid annulus fibrosus the following morphological variants were determined. In the first subtype, there were 13 (23%) cases where the artery passes along the left lateral margin of the superior process. After reaching the proximal part of the annulus fibrosus of the posterior leaflet of the mitral valve the artery passes just lateral to the postero-medial commissure (Fig. 1A). The second subtype consisted of 27 (40%) cases. The artery runs in the middle of the space between the mitral and tricuspid valve. In the third subtype 15 (18%) cases were identified. The artery passes just adjacent but not in contact to the annulus of the septal leaflet of the tricuspid valve (Fig. 1B).

6. Discussion

The report presented herein demonstrates that the incidence of new AVB develops in 23.5% of the patients being operated on for the mitral valve replacement or reconstruction. Considering all of the conduction disturbances including the branch blocks the incidence rises to 38% (Table 1). These data are comparable to the results reported in the literature where the incidence of these postoperative AVB was postulated as 23–37% of all cases [9–11]. The mechanism of the postoperative AVB was not clear; the permanent blocks were independent of the preoperative, intraoperative and early postoperative factors [9,10].

In the study herein we tried to evaluate the causative mechanism behind the permanent postoperative AVB following the mitral valve replacement and the mitral valve reconstruction. The risk of developing a conduction disturbance is more likely to be an AVB (61.3%) rather than

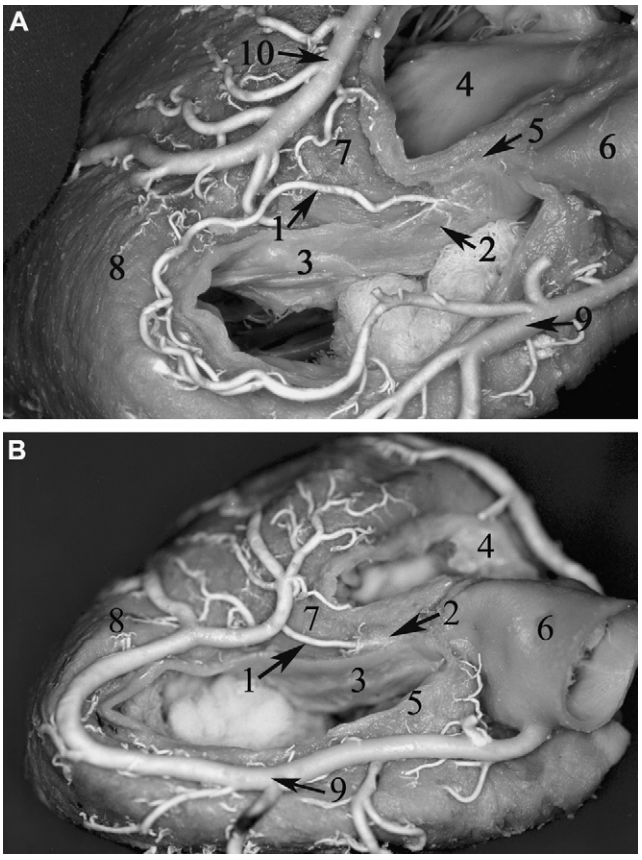

Fig. 1. (A) Dry dissected specimen of the right AV node artery. Note that the right and the left atrium were removed. The presented vascularization type is of left dominance. In the presented case the AV node artery arises from the right coronary artery, reaching the crux the artery runs on the left lateral part of the posterior superior process of the left ventricle. Distally to the area of the posterior-medial commissure the artery runs just near to the mitral valve annulus. (1) AV node artery, (2) AV node, (3) tricuspid valve, (4) mitral valve, (5) right fibrous trigone, (6) aorta, (7) superior posterior process of the left ventricle, (8) right ventricle, (9) right coronary artery, (10) left coronary artery. (B) Dry dissected specimen of the right AV node artery. The right and the left atria were removed just over their attachment. The coronary vascularization is of right type, the AV node artery takes its origin from the right coronary artery. Running toward the AV node on the posterior superior process the artery is found near to the tricuspid leaflet. (1) AV node artery, (2) AV node, (3) tricuspid valve, (4) mitral valve, (5) right fibrous trigone, (6) aorta, (7) superior posterior process of the left ventricle, (8) right ventricle, (9) right coronary artery.

bundle branch block (38.7%). Furthermore 76% of all patients with AVB develop AVB I (Table 1) and only small numbers of patients develop AVB II (5.4%) with some more of the investigated patients representing AVB III (18.4%). All branch blocks were transient, and resolved prior to discharge. In the AVB group, 40% of AVB II (two from five patients) resolved before discharge, the AVB I were permanent and most of them were recorded at discharge. The permanent pacemaker was implanted in all 17 patients developing AVB III in the early postoperative phase. The mean interval of the implantation was 4 days. The decision to implant the permanent pacemaker was made if the block was still present prior to discharge to the ward. In this way we were not able to detect the transient nature of the mentioned AV block, this phenomenon is, in literature, described to be about 40% of the investigated cases [9,12]. This can be explained by the lack of the intermediate unit where the permanent 24 h monitoring following the transmission from the ICU may be performed. However, we believe that the best timing of the pacemaker implantation is after mitral valve replacement or/and reconstruction and should be beyond the first postoperative week but not further.

Preoperative use of the digoxin and beta-blockers associated with other factors such as undergoing artery bypass grafting, intraoperative ischemia and the time of the cross-clamping were in literature postulated as predictor factors of postoperative conduction disturbance [13,14] especially of those with the block nature. However, later in the extensive investigation it was found there is no independent predictors for the incidence of the postoperative AVB following the mitral valve surgery [10]. In our study more than half of the patients were preoperatively taking digoxin and/or beta-blockers before surgery. Digoxin was registered as protective factor. Usage of beta-blockers was not associated with increased incidence of the AVB. Rather the opposite is true; in the patients being treated only with beta-blockers the incidence to develop a conducting disturbance was significantly lower (Table 3). However, the multiple logistic regressions showed that within the beta-blocker group, the use of sotalol may slightly increase the postoperative incidence of the AV block. The same phenomenon was observed in the amiodarone group (Table 7). It is well known that both drugs may have pro-arithmetic affects and may influence the normal function of the conduction system. Regarding the use of both these drugs, we cannot postulate in how many cases the postoperative block were results of the aforementioned drug nature.

The complexity of the mitral valve procedure, performed during surgery such as mitral valve replacement and mitral valve reconstruction were not related to the increased postoperative AVB. The same may be stated for the intraoperatively diagnosed morphological findings of the mitral valve such as Barlow's disease, ischemic or rheumatic degeneration, endocarditis and ischemic dilatation of the annulus. According to the hemodynamical effects of the mentioned morphological alteration, neither the stenotic neither the insufficient valves were identified as predisposing factors for the AVB.

Systemic hypothermia was slightly more pronounced in the AVB group compared to the no-AVB group. Additionally, the cross-clamp time and the cardiopulmonary bypass time were also slightly longer in AVB group. We postulated that these phenomena occurred as a consequence of the longer bypass time and longer cross-clamp periods, which results in a longer warm up interval in the AVB group. However, the measured temperature is the temperature measured in the rectum and as such reflects the hypothermia of the whole body and as such does not reflect the temperature of the heart and its conducting system. To assess the influence of the temperature on the development of the conducting disturbances the local temperature of the myocardium should be measured. In this way the cooling of the different areas of the heart especially that of the AV node and the His bundle would be assessed, and their effect on the development of the AVB would be possible to define.

The mitral valve apparatus is topographically very close to the structures of the atrioventricular conducting systems, especially the proximal part of the posterior leaflet and the

posterior medial commissure. Nevertheless the morphological evidence for conduction disturbance following the manipulations of the annulus fibrosus is unknown. Based on recent literature it is well known that major rhythm disturbances following the mitral valve replacement or reconstruction are AVB of different grades [9,15]. It was considered that the mentioned postoperative complication may be due to the damage of the AV node artery [15]. With regard to the mentioned complication, Meimoun et al. [9] studied the incidence and the predictors of the AVB after mitral valve surgery. However, in this study only the clinical predictors were investigated as potential causes for postoperative AVB, no morphological studies regarding the damage of the AV node or AV node artery were conducted. As a result there was no detectable preoperative or intraoperative predictor of the postoperative incidence of the AVB where the reported incidence of the AVB was 23%.

In the morphological part of the report, we described the topography of the AV node artery and its relation to the mitral valve. We detected that the AV node artery in some cases approaches the annulus fibrosus of the mitral valve. Independent from the dominance or the origin, in 13 (23%) cases the AV node artery approached the P3 section of the posterior leaflet. Thus this morphological data shows that the AV node artery passes frequently just near the posterolateral part of the mitral valve annulus. Therefore damages to the AV node blood supply become very probable during the mitral valve ring annuloplasty or prosthesis implantation. This theoretical assumption on the potential damage of the AV node artery, is supported by detailed analysis of the preoperative, intraoperative and postoperative data of the 391 patients with operations on the mitral valve where, except for the drugs that may influence the nature of the conduction systems, no predictors for postoperative AVB were found.

7. Limitations of the study

The main limitation of this study is that we were not able to investigate the blood supply of the AV node prior to and following the operation. Although theoretically this should be the structure of the study mentioned herein, we have to postulate that this may not be realized yet. The main reason is that we do not have investigation methods where this 2 mm thick artery may be identified. So far this is not even possible with the newer technologies such as multislice CT scans. However, the AV node and its artery may be identified under very precise dry dissection. The aforementioned morphological study gave as a very important input, namely that the artery may run very near to the mitral valve annulus. We believe that in the future we will have very precise diagnostic tools with high resolution, where preoperatively the identification of the very small arteries of the heart conduction system may be possible.

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Appendix A. Conference discussion

Dr C. Mestres (*Barcelona, Spain*): This paper has the value of calling again upon the attention of the reader towards a frequently neglected postoperative problem in cardiac surgery, namely, the need for permanent pacing. It has been more or less established that in around 2—3% of the cases, as the authors have shown, there will be a need for pacing regardless of the type of operation, and this is particularly true after reoperative interventions. There has indeed been some literature in the past addressing this problem after surgery, specifically for myocardial revascularization.

In this series of 434 patients, the authors went to study all possible pre- and intraoperative factors that could eventually influence the appearance of conduction disturbances, of which complete AV block is the most serious. They have not been able to find any variable that can be considered as a predictor for postoperative rhythm abnormalities, but in addition, the authors performed an anatomical study showing, and has been clearly shown in the slides, that in 23% of the cases the AV nodal artery ran close to the mitral annulus. Three different anatomical patterns were also found that relate in different ways the supply of the AV node with the mitral annulus. Thus, it seems that our impression for many years that intraoperative trauma to the arterial supply of the AV node at the time of surgery may play a more important role than other variables like myocardial temperature, cardioplegia, duration of cross-clamp and cardiopulmonary bypass times or associated conditions like coronary artery disease, to mention just a few.

Postoperative conduction disturbances leading to permanent pacemaker implantation may not be critical in terms of patient survival, however, to this discussant have a very important clinical implication, namely, the decision-

making process. Currently, there is an increasing epidemic of pacemakers and also AICD infections due to the huge number of patients implanted worldwide. Device infection is getting more frequent, and data from the literature show that the figures are also increasing. Age, number of leads, manipulations and associated procedures appear to be influencing risk factors for device infection in the form of right-sided endocarditis. As this is a potentially lethal complication, to me, the decision of implanting a cardiac device merits to be carefully evaluated, especially in the setting of a patient receiving a prosthetic valve or a prosthetic ring. As these devices are to be implanted very shortly after a major surgical procedure, we must reduce the chances for a serious complication like infection.

If it is true that a significant factor is surgical trauma when manipulating the mitral annulus, then the chances of reducing the incidence of about 4% of complete AV block may be slim, as sutures have to be placed in the region of the posterior annulus quite often, if not always.

In summary, about 32% of the patients had conduction disturbances, AV block was present in 27% of the series, and complete AV block in 4%. These patients underwent implantation of a device, and I have just a few questions for you.

Number one is, when was complete AV block detected, meaning if it was just coming off bypass, one, two, three, four days after the operation, or so?

Number two, we know that in a number of cases even complete AV block can be transient. We have seen that for many, many years. But the authors implanted a device at an average of .5 days after block was detected, if I am not wrong. The waiting period seemed to be very short. Why didn't the authors wait longer before the decision of implanting a device was made? If there is an extended waiting time in the ward or in the intermittent stepdown unit, would it be possible not to wait longer because of financial reasons or nonmedical or nonsurgical reasons? Do the authors believe it may be worth waiting longer to make sure that they could avoid unnecessary implants?

And finally, regarding the implanted patients, what was the distribution among replacements and repairs?

Perhaps they could look a little deeper at intraoperative variables like hypothermia.

Dr Berdajs: May I have you please repeat the first question?

Dr Mestres: Yes, absolutely. When was complete AV block detected, immediately after coming off bypass, in the following days, because I have not been able to draw that from this information.

Dr Berdajs: Perhaps I should answer the first two questions together. The AV block was detected on the ICU, that means in the period 24 h after the operation, and it is because in our hospital it is something about our internal policy. We don't have an intermediate station, that means between the ICU and the ward, and because of that, immediately, if the AV block of third degree was permanent, still the patient was on the ICU and we did the pacemaker implantation. I believe I answered both questions with the same answer.

And the third one?

Dr Mestres: The point is that when I have to implant a patient with a pacemaker, I have to think, because the morbidity associated with this is tremendous, is getting more and more important today, and I wouldn't mind just to wait one or two or even three weeks. So the point is we are under pressure and sometimes we are forced to do something against our feelings. This is one of the reasons. Would it be medical? Would it be financial? Would you wait three weeks in a row instead of .5 days, at the average, as you reported in your paper?

Dr Berdajs: Of course I totally agree with you, it is logical to wait, because we know from the literature many of the AV blocks from the third degree are transient. But, as I said, it was something about our internal policy. We don't have a transient station.

Dr Mestres: Finally, if you found differences in terms of implantation between replacements and repairs.

Dr Berdajs: You mean if the incidence of the AV block was greater? No, there were no differences, no. We didn't find any difference regarding the distribution in both groups regarding valve replacement or reconstruction. In the reconstruction we were actually focusing on the patients becoming annuloplasty. That means the annulus fibrosis was manipulated in both cases. It is logical. We didn't find any differences.

Dr G. Gerosa (*Padova, Italy*): In your group, all patients undergoing mitral repair received a ring?

Dr Berdajs: Yes.

Dr Gerosa: So there were no patients undergoing mitral repair without a ring?

Dr Berdajs: No.

Dr Gerosa: So you couldn't establish this?

Dr Berdajs: We selected the patients this way. All the patients which were in this group had some kind of manipulation of their annulus fibrosis, because this was our hypothesis, that the manipulation of the annulus fibrosis may damage the AV node artery and this may cause then the incidence of AV block.

Dr Gerosa: In your risk analysis did you include also the operator, the surgeon, among the variables?

Dr Berdajs: No, we didn't look at that.