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ORIGINAL ARTICLE

Significance of Ectopic Beats in Post Aortic Valve Replacement Arrhythmia Patients

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ABSTRACT

The incidence of new onset arrhythmia after conventional aortic valve replacement (AVR) is relatively high whereby atrial fibrillation (AF) in particular (30-40%). Arrhythmias increase postoperative morbidity, mortality and consequently health costs. The need for a reliable method for early detection and discrimination between low and high risk patients is therefore indispensable. **Objective:** For this reason this study examined the possible correlation between electrophysiological abnormalities on continuous ECG recordings and the initiation of arrhythmia directly after surgery. **Methods:** Both ECG and clinical data was collected from the hospitals filing system for all patient (n=107) who underwent surgical Aortic Valve Replacement (AVR) for non-rheumatic aortic valve stenosis or insufficiency for the period from January 2010 to December 2018. Continuous ECG data was converted into ISHNE-format and analyzed by using Synescope™ software. **Results:** Data showed that one minute prior to arrhythmia, AF in particular, an increase of both supraventricular premature beats (SVPB) and missed beats (MB) was detected (n=33; P<0,05). However, there was no correlation between arrhythmia and the overall SVPB incidence (n=33). Twenty-one out of 33 AVR patients developed a de novo intraventricular conductance delay directly after cardioplegic arrest, which persisted in 7 cases. **Conclusions:** Although there is an increase of both SVPB and MB prior to arrhythmia startup butthe true predictive value of these findings are still questionable. Additionally it appeared that a temporarily intraventricular conduction delay (IVCD) is a common finding after AVR.

INTRODUCTION

Aortic valve stenosis is the most common form of valvular heart disease. Aortic valve replacement (AVR) therefore, is a frequently performed procedure [1]. Although AVR improves the quality of life and prolongs its expectancy, valve replacement is known to be associated with severe peri- and postoperative complications like bleeding and infection [2, 3]. The most common post-AVR complications however, are cardiac arrhythmias with an incidence rate of over 70%. These arrhythmias are mainly originating in the supraventricular compartments, whereby new-onset atrial fibrillation (AF) has by far the highest incidence (30-40%) [3, 4, 5]. Patients developing supra ventricular arrhythmias are at significantly higher risk to experience additional co-morbidity such as congestive heart failure and thromboembolic events, including stroke and

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myocardial infarction all leading to longer hospitalization and thereby increasing health costs [5, 7-9]. In addition a greater mortality has been observed in these patients [3, 10, 11]. Although ventricular arrhythmias are not uncommon directly after cardiac surgery, the incidence drops progressively in the course of time making them relatively negligible when compared to arrhythmias arising from the atrial compartments [12].

Several clinical risk factors are known to predispose AVR patients to postoperative arrhythmias, including: obesity, left atrial enlargement, a history of rheumatic heart disease, left ventricular hypertrophy (LVH), hypertension, increased cross-clamp time, previous cardiac surgery, diabetes mellitus and increased age [4, 13-16]. The exact mechanism responsible for the induction and maintenance of an arrhythmia is nevertheless complex and not yet completely understood. For AF in particular however, there are currently two plausible theories. Whereby one theory assumes ectopic foci surrounding the pulmonary veins to fire at a high rate and thereby disturbing sinus rhythm (SR) [6]. Whereas the other theory is based on the re-entry of electric signals, which also seems to be the underlying mechanism in atrial flutter (AFL) [17]. Although there is disagreement on the exact mechanism it has been demonstrated that AF can be progressive and consequently sustain, leading to longer lasting patient discomfort and the latter mentioned co-morbidity. Due to the progressive nature of AF, relatively short episodes already predispose patients [18, 19]. Therefore, it is considered beneficial when also shorter periods of arrhythmia could be foreseen. Tran Thong et al showed there is a detectable increase in supra ventricular premature beats (SVPB) prior to an episode of AF in patients with a history of paroxysmal AF (PAF), a finding which was unrelated to surgery. The increased SVPB rate enabled them to predict whether it was likely that a patient would experience an episode of PAF [20]. An observation that was supported by yet another study in which patients experiencing a relatively high quantity of SVPB were found to be prone to develop PAF [21]. To confirm these results and to argue the predictive value of these supraventricular events prior to an episode AF this study focuses on the incidence of such ectopic events in a postoperative setting by using continuous monitoring. However, as already mentioned, AF is not the only arrhythmia occurring after cardiac surgery. Thus therefore this study aimed to qualify and quantify all various types of arrhythmia during the initial period directly postoperatively in 33 patients who consecutively underwent AVR via conventional on-pump open heart surgery, whereby we focused on the minute prior to arrhythmia startup. Additionally we analyzed the pre- and post-operative 12-lead ECG of these patients whereby markers for LHV in particular since we expected a reduction of left ventricular size in the course of time after surgery due to a decrease in cardiac workload. Whereas we also expected to find an increase in de novo intraventricular conduction delay, whereby mainly left bundle branch blockage (LBBB) as described in current literature [22]. These 12-lead ECG characteristics have also been analyzed for a group of patients undergoing concomitant CABG surgery.

METHODS

Patient Population

All data considered for this study has been collected from the patients file of Queen Alia Heart Institute, Amman, Jordan. Because of this study's retrospective and non-invasive nature, patients did not approve to a waiver of informed consent. All patients (n=107) who consecutively underwent surgical aortic valve replacement for non-rheumatic aortic valve stenosis or insufficiency via conventional on-pump open heart surgery in cardioplegic arrest were included in study during period from January 2009 to December 2018. Patients received either a mechanical (35%) or bio prosthesis (65%) whether or not combined with additional reconstructive cardiac surgery. Patients undergoing repeat

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valvular surgery (n=4) were also included in this study, as were patients already diagnosed with cardiac arrhythmia (n=37) either with or without a pacemaker (n=5). Exclusion criteria for continuous monitoring were concomitant valve replacement or CABG.

CLINICAL DATA

Patients were screened for following risk factors prior to surgery: hypertension, diabetes mellitus (DM), hyperlipidemia, peripheral vascular disease (PVD), transient ischemic attack (TIA) and cerebrovascular accident (CVA), smoking, alcohol, congenital heart disease (CHD) and having a family history of cardiovascular disease (CVD) or sudden cardiac death (SCD). All risk factors are shown in Table 1, which also summarizes relevant medical history and other preoperative characteristics.

12-Lead ECG

Standard 12-lead ECG recordings prior to and after OR have been interpreted for patients solely undergoing AVR and also for patients undergoing concomitant CABG surgery. Recordings have been examined on following characteristics: heart-axis, PR-interval (<200msec), QRS-duration (<100ms), QTc, AV-conductance, intraventricular conduction (i.e (i)LBBB, (i)RBBB, LAFB, LPFB), left and right ventricle hypertrophy, P-wave morphology and length, repolarization and premature complexes. All recordings were crosschecked by a physician and a trained cardiologist to preclude misinterpretation.

Continuous Rhythm Monitoring

In the majority of the cohort (i.e. 88%) data was analyzed from the moment surgery ended. Recordings were however not continuous, meaning consecutive recordings were interrupted by time gaps of 18, 9 ± 26.9 hours. Original data was not compatible with Synescope™ software and had to be converted into ISHNE-format. Analyses were performed on two leads after removing all gross artifacts. Noise filters were adjusted to recording quality and settings used during analyses are shown in Table 2. Minimal and maximal heart rates were determined on two consecutive beats and RR interval respectively. Supraventricular tachycardia with a heart rate faster than 150 bpm (SVT >150bpm) and regular rhythm were automatically detected. Supraventricular premature beats (SVPB) were detected by a 75% sensitivity (i.e. RR-interval <75% of previous two beats) but were not included during periods of arrhythmia. This in contrast to ventricular events (VE), which were detected based on morphology and which were always included. Arrhythmia were detected based on morphology and RR-interval differences, whereby there was no minimal duration threshold meaning that AF episodes of less than 30 seconds were also included.

ST-deviations (i.e. elevations and depressions) have been left out of analyses due to inaccuracy and artifact. Paced rhythms were detected based on either mono- or bipolar electrical spikes.

Statistical Analysis

Endpoints in this study were to determine the correlation between clinical (risk) factors and the incidence of arrhythmia, as also to confirm the postoperative change in LVH markers on 12-lead ECG recordings as described in literature. For which respectively a regression analyses of multiple variables and paired double tailed T-test were used. Another aim was to detect differences in (S) VE on telemetric data during the first minute before arrhythmia startup. This was done by comparing the incidence of the particular events to its average detected per minute by using a paired T-test. Which was also used to determine the correlation between the total number of (S) VE and arrhythmia and to determine the persistence of intraventricular conductance delay (IVCD) directly after cardioplegic arrest. Furthermore a single factor ANOVA test was used to determine possible differences between the various types of initial events at the beginning of an AF episode

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RESULTS

Clinical Data

The above mentioned criteria led to an initial cohort of 108 patients including 67 male and 41 female patients together with an average age of 67yr and BSA of 1.94 ± 0.2 m^2 (1,6-2,6). Patients were hospitalized for an average of 9 ± 7 days. The majority of the study population already received cardiovascular medication before valve replacement, namely: anti-arrhythmic drugs (47%), anti-coagulants(22%), anti-platelet drugs (37%), ACE-inhibitors (40%), statins (36%). Whereby 4% of the study population was already treated with Amiodarone in the past (see Table 2).

DM = Diabetes Mellitus, PVD = Peripheral Vascular Disease, CVA = Cerebro Vascular Accident, TIA = Transient Ischemic Event, SCD = Sudden Cardiac Death, CVD = Chronic Vascular Disease, PCI = Percutaneous Coronary Intervention, CHD = Chronic Heart Disease, CKD = Chronic Kidney Disease, COPD = Chronic Obstructive Pulmonary Disease. *Total (n) = 107. **Other medical history concerning non-cardiovascular related issues.

N	% of Subgroup	% of Total*
Gender (n=107)		
Male 67	62.60	
Female 40	37.40	
Risk Factors (n=84)		
Hypertension 53	63.10	49.50
DM 16	19.10	15.00
Hyperlipidemia 20	23.80	18.70
PVD 13	15.50	12.20
TIA/CVA 12	14.30	11.20
Stopped smoking >1yr 2	2.40	1.90
Smoking present 27	32.10	25.20
Alcohol abuses 0	0.00	0.00
Thyroid disorders 8	9.0	7.50
Family history of SCD 5	6.00	4.70
Family history of CVD 17	20.20	15.90
Cardiac History (n=52)		
History of MI 8	15.40	7.50
PCI 8	15.40	7.0
CABG 5	9.60	4.70
Valve-OK 4	7.70	3.70
Endocarditis 4	7.70	3.70
CHD 4	7.70	3.70
Aortic aneurysm 3	5.80	2.80
Medical History (n=94)		
CKD 10	10.60	9.40
COPD 22	23.40	20.60
Carcinoma 5	5.30	4.70
Varices 4	4.30	3.70
Acute Rheumatic Disease 3	3.20	2.80
Pulmonary Hypertension 3	3.20	2.80
Thromboemsbolic event 2	2.10	1.90
Other** 81	86.20	75.70

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Medication (n=82)		
Antiarrhythmic agents 50	46.70	46.70
Class I 0	0.00	0.00
Class II 47	94.00	43.90
Class III 7	14.00	6.50
Class IV 2	4.00	1.90
Anti-platelet drugs 40	37.40	37.40
Anti-coagulants 24	22.40	22.40
ACE-inhibitors/AG-II 43	40.20	40.20
Statins 39	36.50	36.50
History of amiodarone use 4	3.70	3.70

Table 1: Preoperative characteristics for the AVR cohort considered relevant

Settings
Pause >3000ms
Bradycardia <45bpm
SVT >150bpm
VT >50bpm
SVE prematurity <75%
Acceleration >50%
Deceleration <35%

Table 2: Settings of software

Table 2 shows all relevant software settings and thresholds used during analyses. Percentages concern the previous R-R interval on which the event in dispute is calculated. SVT = Supra Ventricular Tachycardia, VT = Ventricular Tachycardia, SVE = Supra Ventricular Event.

Valve function (n=106)	N	% of Subgroup	ss% of Total*
AS	94	88.70%	87.90%
AI	72	67.90%	67.30%
MS	3	2.80%	2.80%
TI	72	67.90%	67.30%
PI	13	12.30%	12.10%
Aortic Function			
	Mean	**SD**	**Range**
AVA (n=77)	0.8 cm^2	0.43-3.5	0.4
Mean gradient (n=14)	48 mmHg	6-82	19.5
Peak gradient			
(n=85)	77 mmHg	13-145	26,1
Lung Function			
VC% (n=107)	95.40%	3.2-202	26.1
FEV1% (n=107)	92.50%	2-200	26.8
Tiffenau% (n=89)	96.50%	52-133	15.7

Table 3: Preoperative Echo and Pulmonary Function Testing

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Table 3 Q shows all preoperative ultrasound an pulmonary function tests results. AS = Aortic valve Stenosis, AI = Aortic valve Insufficiency, MS = Mitral valve Stenosis, TI = Tricuspid valve Insufficiency, PI = Pulmonary valve insufficiency, AVA = Aortic Valve Area, VC = Vital Capacity, FEV1 = Forced Expiratory Volume. *Total (n) = 107.

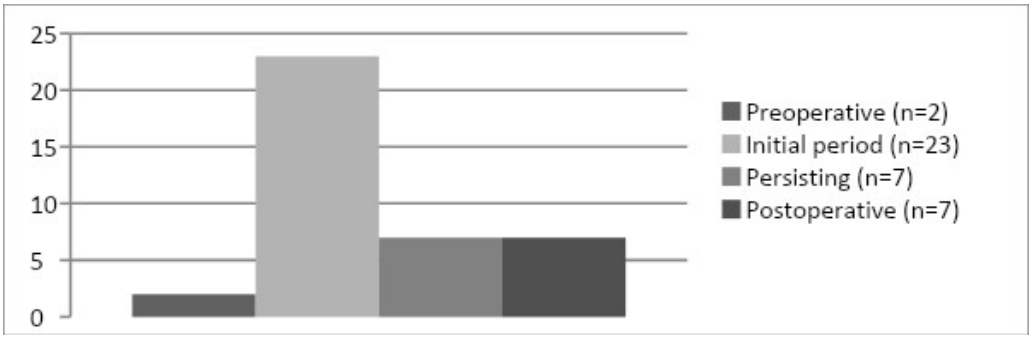

Figure 1: Intraventricular Conductance Delay

Figure 1 shows the overall incidence of the different types of IVCD. Pre- and postoperative IVCD were diagnosed on 12-lead ECG, whereas IVCD directly after surgery were based on continuous monitoring data. Y-axis: number of patients with IVCD. X-axis: moment of diagnosis as shown on the right. Preoperative (n) =21, Initial period (n) = 23, Persisting (n) = 23, Postoperative (n) = 19.

Preoperative echo revealed that 89% of the patients (n=94) suffered from aortic valve stenosis (AS) and 67% (n=72) from aortic valve insufficiency (AI). In 88 patients concomitant valve deficiency had been detected. Patients with congenital heart disease (CHD) were diagnosed with a bicuspid aortic valve, whereas one patient had an additional ventricular septal defect (VSD). The mean aortic valve area and peak gradient were 0.8cm^2 (n=77) and 77mmHg (n=85) respectively. Coronary angiography (CAG) prior to surgery showed that 21% of all patients had a perceptive coronary obstruction (LAD=7, RCX=8, RCA=7), although none of these obstructions were severe enough to undergo concomitant CABG. Spirometry showed a mean population Tiffenau ratio of 97% (n=89), VC of 95% (n=107) and FEV1 of 93% (n=107) as shown in Table 3.

The average time spend in OR was 4.5h whereby 65% received a biological- and 35% a mechanical valve (n=107) together with an average diameter of 23mm. Twenty-nine patients underwent concomitant surgery including MAZE-procedure (n=3), repair of either tricuspid- or mitral valve (n=9) and decalcification/ repair or replacement of the aortic arch and annulus (n=5). In addition there were 3 patients that underwent non-aortic valve related surgery, namely: closure of a VSD, cardiac myectomy and a lung biopsy. Due to a severe decreased cardiac output, 3 patients were in need of an intra aortic balloon pump (IABP) of which 1 patient received the device prior to surgery. Reported complications by the surgeons during OR mainly included arrhythmia (i.e. AF (10%); VT (1%); VF (2%); VER (1%) nodal rhythm (1%); bradycardia (7%); AV-block (14%); LBBB (1%)), bleeding and de novo conductive disorders. Temporary- pre-emptive epicardial pacing electrodes were placed in all patients. Seven patients however directly received either a ventricular or atrioventricular permanent pacemaker due to inadequate intraventricular conduction during surgery.

Postoperative complications predominantly involved bleeding and pleural effusion. Six patients developed hypertension during hospital stay and all patients received supportive cardiovascular medication. Whereby 5 patients received additional inotropic agents due to hemodynamic instability. Re-thoracotomy was performed in 7 cases because of significant blood loss, leading to cardiac tamponade in two cases. Reported was that eighteen out of the 42 patients

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that initially developed AF were successfully converted back to sinus rhythm by either medicinal or electrical cardioversion during hospital stay. A total of two patients had to be reanimated due to insufficient cardiac output, whereas 1 patient died while hospitalized as a result of multiple cerebellar infarcts.

Regression analyses on risk factors, cardiac and medical history as listed in Table 2 was performed on a subgroup of 33 AVR patients in which arrhythmia were detected on continuous cardiac monitoring. Similar analyses were also performed for AF in particular. Results showed that of all risk factors only hypertension and alcohol usage were significantly correlated with the development of AF ($P < 0,05$). None of the other risk- or clinical factors showed a significant correlation to arrhythmia, including AF, on continuous monitoring.

12-Lead ECG

Pre- and postoperative recordings of 82 AVR patients and 31 patients that underwent concomitant CABG were analyzed. Recordings were examined on discrepancies in ECG characteristics before and after surgery. Data of AVR patients was recorded 28 ± 40 days prior to surgery and 67 ± 109 days after surgery. And for the AVR + CABG group 30 ± 67 days and 48 ± 95 days respectively. Analyses revealed a significant postoperative reduction of LVH markers after AVR surgery without concomitant CABG.

Continuous Rhythm Monitoring

Telemetric data of thirty-three patients out of the initial cohort that solely underwent AVR was analyzed for an average of 69.8 ± 30,5 hours per patient directly after cardioplegic arrest (average HR 80 ± 9 bpm.). Table VI shows the mean incidence of all various types of events detected during this postoperative period including the number of patients in which these events were detected. It also shows the total number of isolated (i)SVPB (i.e. SVPB not followed by another SVE or arrhythmia) and overall incidence SVPB, which includes all initial SVE and arrhythmia SVPB. SVT with a rate of >150bpm, lasting longer than 5s, were automatically detected by Synescope software and were therefore also included as SVE in analyses as already mentioned earlier. Three patients showed intermittent ventricular pacemaker activity due to inadequate AV-conduction or because of SR interruption. As was the case in one patient which frequently experienced longer lasting episodes of AF. Both SVE and VE were however still detected during these periods of ventricular stimulation.

The following types of arrhythmia were detected on telemetric data: AFL (n=1), AF (n=13), ventricular escape rhythm (VER; n=1) and unspecified SVT (n=2). Results of regression analyses including the various types of events per thousand beats (‰) and the latter arrhythmia all together or AF. There was no significant correlation between the incidence of any type of event and any type of arrhythmia.There were two remarkable cases, whereby recordings showed an obvious irregular rhythm with a high number of SVE (6-10 SVPB/min) which were considered as sinus arrhythmia. However neither one of these patients experienced episodes of AF or any other arrhythmia. Additionally there was one case in which a patient repeatedly experienced a high number of SV-Runs and short SVT (1.7 hours), which did not develop a definable arrhythmia either.

It must be mentioned that not all episodes of AF were included in the latter analyses, because two patients directly developed AF after cardioplegia. Whereas two other recordings started within an episode of AF, due to which it was not possible to include these arrhythmia in the analyses since there was no regular rhythm prior to the arrhythmia. Additionally there was one patient in which it was impossible to detect the exact moment of AF startup due to inadequate pacemaker activity.

Besides the above listed events, recordings of two patients showed a deceleration right before an episode of AF. And yet another patient was still

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in AF during the minute before the next episode. Whereas in one particular case a patient experienced several episodes of AFL which suddenly turned into AF without an SR interval.

For patients experiencing AF a single factor ANOVA test (P<0.05) was performed to determine the differences between the initial types of events at the beginning of an episode. Whereby results showed that an iSVPB was fare out the most frequently detected event at AF startup.

Intraventricular Conductance

Twenty-three out of the 33 monitored patients initially developed a de novo IVCD directly postoperative (23 ± 51 minutes) which averagely lasted for 29 ± 58 minutes. On telemetric data IVCD were diagnosed based on a widened QRS-complex (>100ms) that morphologically differed from later QRS-complexes. Twenty of these patients also had a concomitant temporarily altered electrical heart axis during this initial period. Pre- and postoperative 12-lead ECG recordings were examined to determine whether patients already had an IVCD prior to AVR (n=2) and to determine if a de novo conductance delay diagnosed on telemetric data sustained (n=7; see Figure 1).The figure 1 already indicates that the majority of IVCD were discontinuous (n=16) and could therefore not been detected on later postoperative 12-lead recordings. An observation that was statistically confirmed by using a T-test, which due to missing ECG-data only included 17 patients. Test results showed a significance difference in the number of IVCD de novo and IVCD diagnosed on 12-lead ECG after 65 ± 128days post-AVR. Suggesting most IVCD are indeed temporarily. Besides, regression analyses of the various types of sustained postoperative IVCD diagnosed on 12-lead ECG (i.e. BBB) did not show a particular intraventricular conduction pathway to be predisposed for blockage.

DISCUSSION

This study confirmed the postoperative incidence rate of AF to be 30-40% as already described in literature. Risk factors associated with AF were alcohol and hypertension. This study also supports the finding of an increased number of SVPB during the period prior to an arrhythmic episode, AF in particular, as was already described for PAF [22]. Whereas analyses additionally revealed an increased incidence of MB during this period. The overall mean incidence of SVPB and MB was however very low, making the true predictive value of this finding questionable. Because already a low number of either SVPB or MB prior to arrhythmia startup easily exceeds the average incidence rate.

Noteworthy was that most episodes of AF started with a SVPB, indicating that the trigger for AF mainly arises from the supraventricular compartments. However when examining the average SVPB incidence per minute during 1h before and after an AF episode there was no marked increase as one might expect. Rather, SVPB seemed to be concentrated in clusters which were randomly spread out with no clear pattern and without any relation to arrhythmic episodes. An observation that however lacks statistical evidence and therefore requires further investigation.

Neither there was a correlation between the overall incidence of SVPB and AF during continuous monitoring, as described by Wallmann*et al.*, [20]. A discrepancy which could be explained by a general increase of SVPB during the turbulent period directly after cardoplegia. Due to which the relative difference in SVPB of AF-patients and non-AF-patients faded. Although it must be mentioned that in three cases in which recordings showed a remarkable high number of SVE none of the patients eventually developed an arrhythmia. Which does make the earlier described correlation questionable.

Analyses of pre- and postoperative 12-lead recordings revealed a significance decrease in markers for LVH after AVR. A finding which could

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perfectly be explained by a decrease in left ventricular workload after valve replacement. However, patients undergoing concomitant CABG surgery did not show a similar reduction, which could be due to remodeling incompetence as a result of long term hypoxemia.

Analyses of intraventricular conduction initially showed a delay in the majority of patients analyzed by continuous monitoring. The IVCD however disappeared after 30 minutes in most patients and was therefore most likely induced by hypothermic and hyperkalemic conditions as also temporal hypoxemia, rather than due to irreversible damage caused during the procedure [24-30]. Change of heart-axis in these patients could be explained in a similar fashion and the assumption that myocardial cells recover in a asynchronously manner after cardioplegic arrest. Twenty percent of these patients however preserved an IVCD, which equally distributed over the various ventricular conduction pathways. Meaning there was no particular pathway (LBBB) predisposed as described earlier [21].

Overall it may be concluded that the true predictive value of an increase in SVPB and MB to predict arrhythmia, AF in particular, still needs to be examined in a more precise manner. By getting a better understanding of startup mechanisms and predisposing events, continuous monitoring might eventually be used in clinical setting to predict which patients are more likely to experience arrhythmia. Combining telemetric data with preoperative echographic- and biochemical markers such as ventricular function and pro BNP could therefore lead to a selection of patients that qualify for prophylactic treatment [33-35, 4]. Such prophylaxis could potentially reduce the incidence of the most common and clinically most relevant arrhythmia, AF, and therefore consequently prevent additional cardiovascular complications [12, 15, 23, 31, 32]. Another possible way to decrease postoperative arrhythmia onset would be to further explore technical possibilities as in minimal invasive surgery whereby cardiac stress and tissue damage are greatly reduced. Certain techniques could possibly also benefit intraventricular conductance and prevent IVCD to occur. Although it must be mentioned that current TAVI techniques do not show an AF incidence reduction.

CONCLUSIONS

Although there is an increase of both SVPB and MB prior to arrhythmia startup, it is still questionable what the true predictive value of these findings is are. Additionally it appeared that a temporarily intraventricular conduction delay (IVCD) is a common finding after AVR.

REFERENCES

[1] Vahanian A, Ottavio A, Felicita A, Antunes MJ, Baro´ n-Esquivias G, Baumgartner H, et. al., (2012).Guidelines on the management of valvular heart disease (version 2012). European Heart Journal.

[2] Paparella D, Brister SJ, Buchanan MR (2004).Coagulation disorders, of cardiopulmonary bypass: a review. Intensive Care Med. 30(10):1873-81.

[3] Filardo G, Hamilton C, Hamman B, Hebeler RF Jr, Adams J, Grayburn P (2010). New-Onset Postoperative Atrial Fibrillation and Long Term Survival After Aortic Valve Replacement Surgery. Ann Thorac Surg. 90(2):474-9.

[4] Banach M, Goch A, Misztal M, Rysz J, Jaszewski R, Goch JH. Predictors of paroxysmal atrial fibrillation in patients undergoing aortic valve replacement.J ThoracCardiovasc Surg. 2007 Dec;134(6):1569-76.

[5] Almassi GH, Schowalter T, Nicolosi AC, Aggarwal A, Moritz TE, Henderson WG, et. al. (1997).Atrial Fibrillation After Cardiac Surgery: A Major Morbid Event? Ann Surg. 226(4):501-11.

[6] Haïssaguerre M, Jaïs P, Shah DC, Takahashi A, Hocini M, Quiniou G, et. al. (1998). Spontaneous initiation of atrial fibrillation

DOI: https://doi.org/10.54393/pbmj.v4i2.76

by ectopic beats originating in the pulmonary veins. N Engl J Med. 339(10):659-66.

[7] Creswell LL, Schuessler RB, Rosenbloom M, Cox JL (1993).Hazards of Postoperative Atrial Arrhythmias. Ann Thorac Surg. 56(3):539-49.

[8] Flegel KM, Hanley J(1989). Risk factors for stroke and other embolic events in patient with nonrheumatic atrial fibrillation. Stroke. 20(8):1000-4.

[9] Ruel M, Masters RG, Rubens FD, Bédard PJ, Pipe AL, Goldstein WG, et. al. (2004).Late Incidence and Determinants of Stroke After Aortic and Mitral Valve Replacement. Ann Thorac Surg. 78(1):77-83.

[10] Schulenberg R, Antonitsis P, Stroebel A, Westaby S (2010). Chronic Atrial Fibrillation Is Associated With Reduced Survival After Aortic and Double Valve Replacement. Ann Thorac Surg. 89(3):738-44

[11] Levy F, Garayalde E, Quere JP, Ianetta-Peltier M, Peltier M, Tribouilloy C (2006). Prognostic Value of Preoperative Atrial Fibrillation in Patients With Aortic Stenosis and Low Ejection Fraction Having Aortic Valve Replacement. Am J Cardiol. 15;98(6):809-11

[12] Chung MK (2000). Cardiac surgery: Postoperative arrhythmias. Crit Care Med. 28:N136-44.

[13] Zacharias A, Schwann TA, Riordan CJ, Durham SJ, Shah AS, Habib RH (2005). Obesity and Risk of New-Onset Atrial Fibrillation After Cardiac Surgery. Circulation. 112(21):3247-55.

[14] Frost L, Hune LJ, Vestergaard P (2005). Overweight and obesity as risk factors for atrial fibrillation or flutter: The Danish Diet, Cancer, and Health Study. Am J Med. 118(5):489-95.

[15] Hogue CW Jr, Hyder ML (2000). Atrial Fibrillation After Cardiac Operation: Risks, Mechanisms, and Treatment. Ann Thorac Surg. 69(1):300-6.

[16] Asher CR, Miller DP, Grimm RA, Cosgrove DM, Chung MK (1998). Analysis of Risk Factors for Development of Atrial Fibrillation Early After Cardiac Valvular Surgery. Am J Cardiol.82(7):892-5.

[17] Saksena S, Hettrick DA, Koehler JL, Grammatico A, Padeletti L(2007). Progression of paroxysmal atrial fibrillation to persistent atrial fibrillation in patients with bradyarrhythmias. Am Heart J. 154(5):884-92.

[18] Jahangir A, Lee V, Friedman PA, Trusty JM, Hodge DO, Kopecky SL, et. al.(2007). Long-Term Progression and Outcomes With Aging in Patients With Lone Atrial Fibrillation : A 30-Year Follow-Up Study. Circulation. 115(24):3050-6.

[19] Thong T, McNames J, Aboy M, Goldstein B (2004). Prediction of Paroxysmal Atrial Fibrillation by Analysis of Atrial Premature Complexes. IEEE Trans Biomed Eng.51(4):561-9.

[20] Wallmann D, Tüller D, Wustmann K, Meier P, Isenegger J, Arnold M, et. al.(2007). Frequent Atrial Premature Beats Predict Paroxysmal Atrial Fibrillation in Stroke Patients: An Opportunity for a New Diagnostic Strategy. Stroke. 38(8):2292-4.

[21] El-Khally Z, Thibault B, Staniloae C, Theroux P, Dubuc M, Roy D, et. al. (2004).Prognostic significance of newly acquired bundle branch block after aortic valve replacement.Am J Cardiol. ;94(8):1008-11.

[22] Tai CT, Chen SA (2009). Electrophysiological mechanisms of atrial flutter. J Chin Med Assoc. 72(2):60-7.

[23] Early treatment of atrial fibrillation for stroke prevention. Available from: http://www.easttrial.org/

[24] O'Connell JB, Wallis D, Johnson SA, Pifarre R, Gunnar RM (1982). Transient bundle branch block following use of hypothermic cardioplegia in coronary artery bypass surgery: high incidence without perioperative

DOI: https://doi.org/10.54393/pbmj.v4i2.76

myocardial infarction.Am Heart J. 103(1):85-91.

[25] Flack JE, Hafer J, Engelman RM, Rousou JA, Deaton DW, Pekow P (1992). Effect of normothermic blood cardioplegia on postoperative conduction abnormalities and supraventricular arrhythmias.Circulation. 86(5):II385-92.

[26] Windsor JS, Rodway GW, Montgomery HE (2010). A review of electrocardiography in the high altitude environment.High Alt Med Biol. 11(1):51-60.

[27] Hansel J, Solleder I, Gfroerer W, Muth CM, Paulat K, Simon Pet. al. (2009).Hypoxia and cardiac arrhythmias in breath-hold divers during voluntary immersed breath-holds.Eur J Appl Physiol. 105(5):673-8.

[28] Krishnaswamy A, Askari AT (2007). A young woman with severe hypoxemia, electrocardiographic changes, and altered mental status.Cleve Clin J Med.74(7):521-8.

[29] Ohmae M, Rabkin SW. (1981). Hyperkalemia-induced bundle branch block and complete heart block.ClinCardiol. 4(1):43-6.

[30] Punja MM, Schneebaum R, Cohen J (1973). Bifascicular block induced by hyperkalemia. J Electrocardiol. 6(1):71-6.

[31] Halonen J, Halonen P, Järvinen O, Taskinen P, Auvinen T, Tarkka M, et. al.(2007). Corticosteroids for the prevention of atrial fibrillation after cardiac surgery: a randomized controlled trial.JAMA. 11;297(14):1562-7.

[32] Halonen J, Hakala T, Auvinen T, Karjalainen J, Turpeinen A, Uusaro A, et. al.(2006). Intravenous administration of metoprolol is more effective than oral administration in the prevention of atrial fibrillation after cardiac surgery.Circulation.114(1):I1-4.

[33] Murtuza B, Pepper JR, Stanbridge RD, Darzi A, Athanasiou T (2008). Does minimal-access aortic valve replacement reduce the incidence of postoperative atrial fibrillation? Tex Heart Inst J.35(4):428-38.

[34] Wazni OM (2004). Plasma B-Type Natriuretic Peptide Levels Predict Postoperative Atrial Fibrillation in Patients Undergoing Cardiac Surgery. Circulation. 110:124-127.

[35] Patton KK (2009). N-Terminal Pro-B-Type Natriuretic Peptide Is a Major Predictor of the Development of Atrial Fibrillation : The Cardiovascular Health Study. Circulation. 120:1768-1774.