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# Bachmann bundle pacing reduces atrial electromechanical delay in type 1 myotonic dystrophy patients

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## Abstract

**Background** Atrial electromechanical delay (AEMD) is an echocardiographic parameter correlated with the onset of supraventricular arrhythmias in several clinical conditions. Inter-atrial septal pacing in the region of Bachmann's bundle (BB) has been shown to be safe and feasible in myotonic dystrophy type 1 (DM1) patients, with a low rate of sensing and pacing defects. The aim of this study was to assess the impact of temporary BB pacing compared with right atrial appendage (RAA) pacing on AEMD in DM1 patients undergoing pacemaker (PM) implantation for cardiac rhythm abnormalities.

**Methods** The study enrolled 70 consecutive DM1 patients undergoing PM implantation for cardiac rhythm abnormalities in accordance with the current guidelines. Seventy age- and sex-matched non-DM1 patients undergoing dual-chamber PM implantation for cardiac rhythm abnormalities were used as controls. The atrial pacing lead was temporarily positioned in the RAA and on the right side of the inter-atrial septum in the region of Bachmann's bundle. For each site (BB and RAA), temporary atrial pacing in the AAI mode was established at 10 beats per minute above the sinus rate and a detailed trans-thoracic echocardiogram with tissue Doppler (TDI) analysis was recorded after at least 10 min of atrial pacing to evaluate AEMD.

**Results** Temporary RAA pacing did not show statistically significant differences in inter-AEMD (48.2 ± 17.8 vs 50.5 ± 16.5 ms; $P = 0.8$), intra-left AEMD (43.3 ± 15.5 vs 44.6 ± 15.8 ms; $P = 0.1$), or intra-right-AEMD (14.1 ± 4.2 vs 15.4 ± 5.8 ms; $P = 0.9$), in comparison with sinus rhythm. Temporary BB pacing determined a significantly lower inter-AEMD (36.1 ± 17.1 vs 50.5 ± 16.5 ms; $P = 0.001$) and intra-left AEMD (32.5 ± 15.2 vs 44.6 ± 15.8 ms; $P = 0.001$) values in comparison with temporary RAA pacing. No statistically significant difference was found in intra-right AEMD (12.2 ± 4.6 vs 15.4 ± 5.8 ms; $P = 0.2$). In the control group, neither temporary RAA pacing nor temporary BB pacing showed statistically significant differences in inter-AEMD, intra-left AEMD, or intra-right AEMD values in comparison with sinus rhythm.

**Conclusions** In DM1 patients undergoing dual-chamber PM implantation, atrial pacing in the Bachmann bundle region is associated with significantly lower echocardiographic indices of atrial electromechanical delay (inter-AEMD and intra-left AEMD) in comparison with RAA pacing.

**Keywords** Myotonic dystrophy · Atrial fibrillation · Bachmann bundle · Pacemaker · Atrial electromechanical delay · Neuromuscular disorders · Arrhythmias

## 1 Introduction

Myotonic dystrophy type 1 (DM1) is the most common muscular dystrophy in adult life. Cardiac involvement is recorded in about 80% of cases, and this often precedes the involvement of skeletal muscle [1]. Several studies have associated baseline electrocardiographic abnormalities with the risk of sudden death in DM1 patients [2]. This has prompted pacemaker (PM) implantation in 4.1 to 11% of patients or the use of an implantable cardioverter defibrillator (ICD) in 1.1 to 5.3%, in order to prevent fatal events [3]. Paroxysmal atrial arrhythmias (atrial fibrillation, atrial flutter, atrial tachycardia) frequently occur in DM1 patients [4–8] with a prevalence of

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up to 25% in DM1 patients and seem to increase mortality in this population [9, 10]. Given the high risk of supraventricular arrhythmias and their consequences, clinical strategies for reducing the risk of atrial fibrillation are of pivotal importance to the optimization of clinical management. Inter-atrial septal pacing in the region of Bachmann's bundle (BB) has been shown to be a safe and feasible procedure in DM1 patients, with a low rate of sensing and pacing defects [11, 12]. However, no data are available on the effects of BB pacing on the atrial electromechanical delay (AEMD), an echocardiographic parameter correlated with the onset of supraventricular arrhythmias in several clinical conditions [13–18]. The aim of this study was to assess the impact of temporary BB pacing compared with right atrial appendage (RAA) pacing on AEMD in DM1 patients undergoing PM implantation for cardiac rhythm abnormalities.

## 2 Materials and methods

### 2.1 Study population

From a large cohort of 270 DM1 patients undergoing cardiac evaluation at our hospital, we prospectively enrolled 80 consecutive DM1 patients undergoing PM implantation for cardiac rhythm abnormalities in accordance with the current guidelines [19]. The diagnosis of DM1 was firstly based on family history, clinical evaluation, and detection of myotonic discharges on electromyography and it had been subsequently confirmed by molecular genetic testing form peripheral blood, to evaluate the CTG triplet expansion. DM1 patients with cardiac systolic dysfunction, patent foramen ovale, atrial septal aneurysm, severe mitral stenosis or regurgitation, complete atrioventricular block, left atrial enlargement (antero-posterior left atrial diameter > 40 mm, left atrial indexed volume > 28 ml/m$^2$), history of atrial fibrillation (AF)/atrial arrhythmias, or previous surgery involving the right atrium (coronary bypass or valvular heart surgery) were excluded from the study. Seventy age- and sex-matched non-DM1 patients undergoing dual-chamber PM implantation for cardiac rhythm abnormalities were used as controls.

### 2.2 Study protocol

This was a single-center, acute observational study of DM1 patients undergoing PM implantation at our hospital. A standard technique for the insertion of a dual-chamber PM system was used. Percutaneous subclavian vein cannulation was performed in all cases. First, the right ventricular lead was positioned in the apex, under fluoroscopic guidance. The atrial pacing lead was positioned in the RAA and on the right side of the inter-atrial septum in the region of Bachmann's bundle. For each site, the correct location was confirmed by means of fluoroscopic imaging and paced P wave configuration on standard surface ECG. Sensing values, pacing thresholds, and impedance were measured for each site. The average of three measurements for each site was calculated. The optimal atrial pacing site was defined as the location with the lowest pacing and highest sensing thresholds. From each optimal atrial site (BB and RAA), temporary atrial pacing in the AAI mode was established at 10 beats per minute (bpm) above the sinus rate, and a detailed trans-thoracic echocardiogram with tissue Doppler (TDI) analysis was recorded after at least 10 min of atrial pacing to evaluate AEMD. The study was conducted in accordance with the Declaration of Helsinki. Written informed consent, as approved by the local ethics committee, was obtained from the patients before enrollment.

### 2.3 Echocardiographic measurements

Images were gathered by a standard ultrasound machine (Vivid 9, GE Medical Systems, Milwaukee, WI, USA) with a 3.5–4-MHz phased-array probe (M3S). All patients were examined in the supine position by means of precordial M-mode, two-dimensional, Doppler, and tissue Doppler echocardiography. A 1-lead electrocardiogram was continuously recorded. Left ventricular (LV) diameter and wall thickness were measured from the two-dimensional targeted M-mode echocardiographic tracings in the parasternal short axis. Ejection fraction was measured by means of a modified Simpson's biplane method. Each representative value was obtained from the average of three measurements. LV mass was determined and indexed to body surface area. All the echocardiographic studies were digitally stored and all the measurements were taken off-line by two independent observers who were blinded to the clinical status of the subjects. Pulsed-wave Doppler examination was performed to obtain the following indexes of LV diastolic function: peak mitral inflow velocities at early (E) and late (A) diastole and E/A ratio. Average values of these indexes obtained from five consecutive cardiac cycles were used for analysis. Left atrial size was determined by M-mode in the parasternal long axis projection; the cavity area of both atria was measured planimetrically in the apical four-chamber view. Pulsed-wave tissue Doppler echocardiography was performed at transducer frequencies of 3.5–4.0 MHz, by adjusting the spectral pulsed Doppler signal filters up to a Nyquist limit of 15–20 cm/s, and using the minimal optimal gain. The monitor sweep speed was set at 50–100 mm/s to optimize the spectral display of myocardial velocities. In the apical four-chamber view, the pulsed Doppler sample volume was subsequently placed at the level of the left ventricular lateral mitral annulus, septal mitral annulus, and right ventricular tricuspid annulus. The tissue Doppler pattern is characterized by a positive myocardial systolic wave (S) and two negative diastolic waves, early (E) and atrial (A). Time intervals from the onset of the P wave on surface ECG to the beginning

of the A wave (PA), representing atrial electromechanical delay, were obtained from the lateral mitral annulus, septal mitral annulus, and right ventricular (RV) tricuspid annulus and were named lateral PA (Fig. 1), septal PA (Fig. 2), and RV PA (Fig. 3), respectively. The timing of mechanical activation of each reference point, namely lateral mitral, septal mitral, and RV tricuspid annuli, depends on the distances of these points from the sinus node during sinus rhythm or from the pacing site during RAA and BB pacing; the RV tricuspid annulus is the earliest point to be activated by the impulse arising from the sinus node, while the lateral mitral annulus is the latest. Therefore, it is hypothesized that the difference between any two reference points reflects the mechanical delay between these two points. The difference between septal PA and RV PA was defined as the intra-right atrial electromechanical delay (septal PA-RV PA); the difference between the lateral PA and the septal PA was defined as the intra-left atrial electromechanical delay (lateral PA-septal PA), and the difference between the lateral PA and the RV PA (lateral PA-RV PA) was defined as the inter-atrial electromechanical delay [20]. Intra- and inter-observer coefficients of variation for AEMD variables were found to be less than 5% and not significant.

### 2.4 Assessment of left atrial mechanical functions

Left atrial (LA) volumes were obtained from apical four-chamber views by means of the modified Simpson's method. The maximum LA volume (*V*max) in the end-systolic phase, the minimum LA volume (*V*min) in the end-diastolic phase, and the LA volume before atrial systole (*V*p) (origin of P wave on electrocardiography) were measured and all volumes were indexed to body surface area (BSA) and expressed in ml/m$^2$. Parameters of LA function were calculated as follows: LA passive emptying volume (LAPEV): *V*max *V*p; LA passive emptying fraction (LAPEF): [(*V*max*V*p)/*V*max] × 100; LA active emptying volume (LAAEV): *V*p*V*min; LA active emptying fraction (LAAEF): [(*V*p *V*min)/*V*p] × 100; LA total emptying volume (LATEV): *V*max*V*min; LA ejection fraction (LAEF): [(*V*max *V*min)/*V*max] × 100.

### 2.5 Statistical analysis

Continuous variables were expressed as mean values ± standard deviation (SD). Statistical analysis was performed by means of Student's *t* test for paired data and one-way analysis of variance (ANOVA) coupled with Newman-Keuls post hoc test for multiple comparisons. The Pearson chi-square test was used to compare categorical variables between the groups. In all statistical tests, calculated *P* values of less than 0.05 were considered statistically significant. Statistical comparisons were made by means of the statistical software package SPSS 10.01 (SPSS Inc., Chicago, IL, USA).

## 3 Results

### 3.1 Study population

From the initial cohort of 80 DM1 patients, 10 patients were excluded owing to suboptimal acoustic windows into the atria ($n = 6$), current AF during the procedure ($n = 2$), and poor patient compliance ($n = 2$). The study therefore comprised

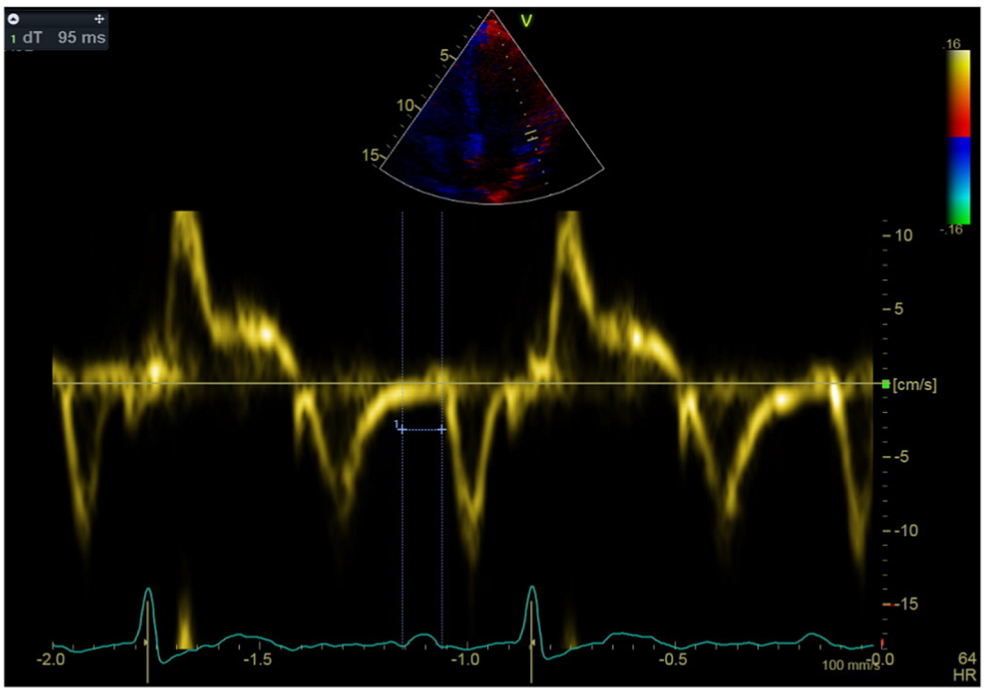


**Fig. 1** Time interval from the onset of the *P* wave on surface ECG to the beginning of the A wave (PA), representing atrial electromechanical delay obtained from the lateral mitral annulus (lateral PA)

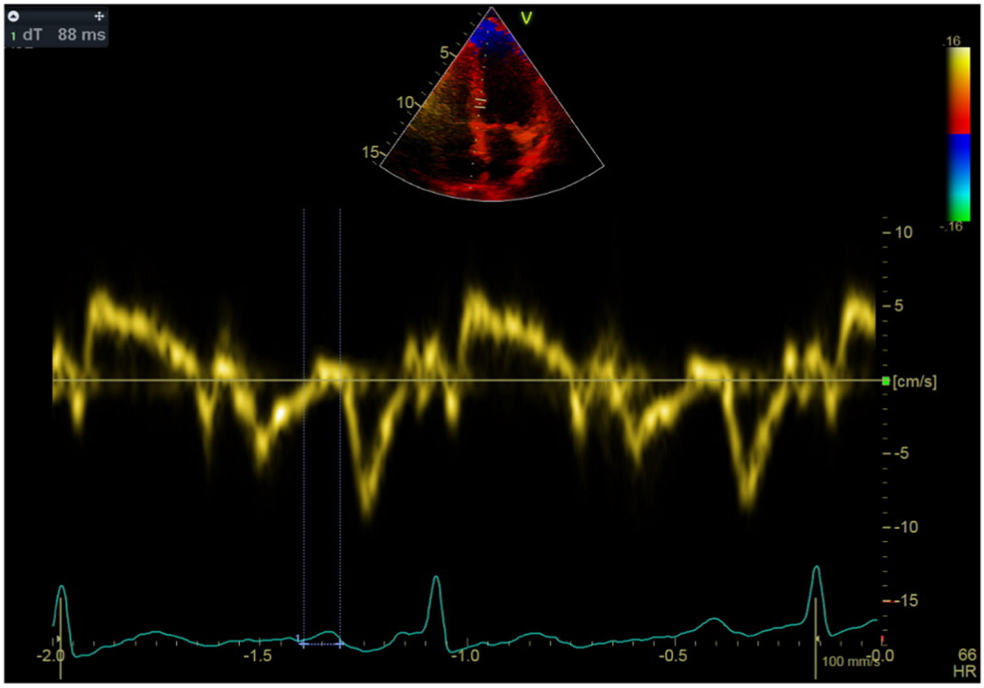


**Fig. 2** Time interval from the onset of the P wave on surface ECG to the beginning of the A wave (PA), representing atrial electromechanical delay obtained from the septal mitral annulus (septal PA)

70 consecutive DM1 patients (mean age 54.2 ± 11.4 years, 45 M) undergoing dual-chamber PM implantation at our hospital. As a control group, we used 70 age- and sex-matched non-DM1 patients with similar clinical and echocardiographic features undergoing dual-chamber PM implantation for cardiac rhythm abnormalities. The DM1 patients enrolled showed normal values of left ventricular posterior wall end-diastolic thickness (LVPWEDT 9.1 ± 0.5 mm), inter-ventricular septum end-diastolic diameter thickness (IVSEDT 7.1 ± 1.1), left ventricular end-diastolic diameter (LVEDD 50.5 ± 1.1 mm), left ventricular end-systolic diameter (LVESD 30.1 ± 3.1 mm), left atrial diameter (38.2 ± 1.3 mm), LA area (20.1 ± 2.9 $cm^2$), and indexed volume (22.1 ± 2.1 ml/m2). They also showed conserved systolic and diastolic cardiac function, with normal values of left ventricle ejection fraction (EF 55.3 ± 5.9%), E wave (81.7 ± 14.9 cm/s), A wave (58.9 ± 12.6 cm/s), E/A ratio (1.3 ± 1.1), and E wave deceleration time (Edt, 192.1 ± 9.5 ms). Basal inter- and intra-left AEMD values

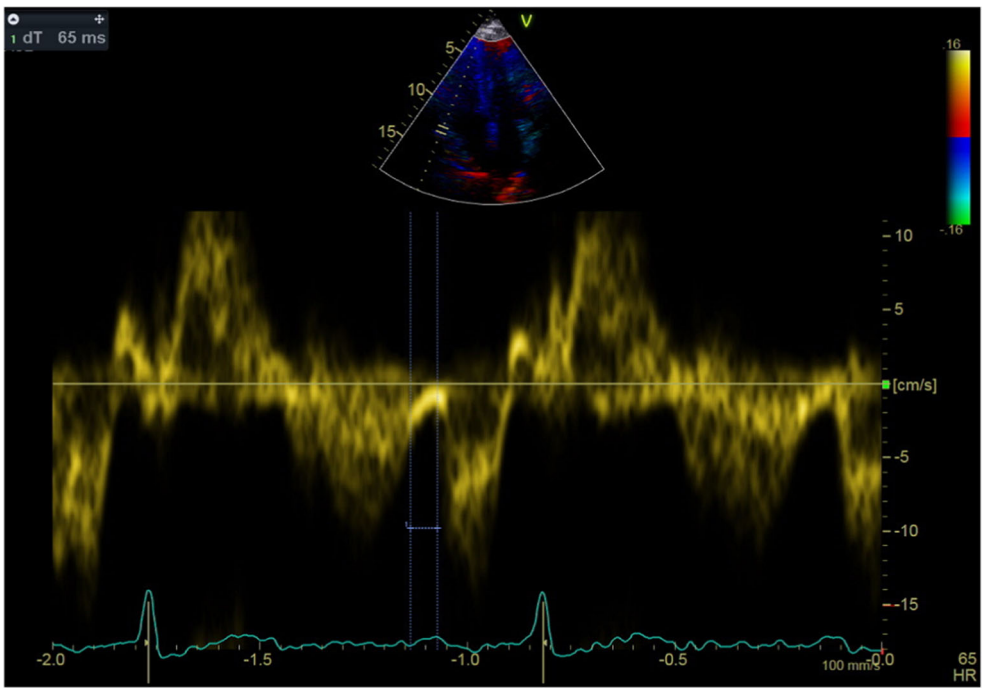


**Fig. 3** Time interval from the onset of the P wave on surface ECG to the beginning of the A wave (PA), representing atrial electromechanical delay obtained from the right ventricular tricuspid annulus (RV PA)

recorded in sinus rhythm were significantly higher in DM1 patients than in the control group (48.2 ± 17.8 vs 32.8 ± 9.9 ms, $P = 0.03$, and 43.3 ± 15.5 vs 29.4 ± 9.5 ms, $P = 0.03$); no statistically significant differences in intra-right AEMD were observed (14.1 ± 4.2 vs 12.8 ± 7.1 ms; $P = 0.5$). Table 1 summarizes the clinical and echocardiographic characteristics of the study population.

### 3.2 Left atrial mechanical function

The DM1 patients presented normal LA *V*max, LA *V*p, LA *V*min, LA ejection fraction, LA total emptying volume, LA active emptying volume, LA passive emptying volume, LA active emptying fraction, and LA passive emptying fraction values (Table 1).

### 3.3 Effect of different atrial pacing sites on AEMD

In DM1 patients, temporary RAA pacing did not show statistically significant differences in inter-AEMD (48.2 ± 17.8 vs 50.5 ± 16.5 ms; $P = 0.8$), intra-left AEMD (43.3 ± 15.5 vs 44.6 ± 15.8 ms; $P = 0.1$), or intra-right AEMD (14.1 ± 4.2 vs 15.4 ± 5.8 ms; $P = 0.9$) in comparison with sinus rhythm. However, temporary BB pacing determined significantly lower inter-

**Table 1** Clinical, electrocardiographic, and echocardiographic characteristics of the study population

| | DM1 group | Control group | *P* values |
|---|---|---|---|
| Patients (*n*) | 70 | 70 | |
| Age (years) | 54.2 ± 11.4 | 53.1 ± 12.2 | 0.2 |
| BMI (Kg/m$^2$) | 19.5 ± 3.6 | 20.1 ± 1.2.2 | 0.2 |
| Sex (male/female) | 45/25 | 40/30 | |
| SBP (mmHg) | 120.8 ± 10.1 | 119.1 ± 10.8 | 0.5 |
| DBP (mmHg) | 66.6 ± 8.1 | 60.8 ± 9.1 | 0.6 |
| HR (bpm) | 55.1 ± 5.1 | 70.2 ± 1.7 | 0.4 |
| EF (%) | 65.3 ± 5.9 | 63.5 ± 4.5 | 0.4 |
| FS (%) | 31.5 ± 3.2 | 33.5 ± 1.9 | 0.4 |
| LVEDD (mm) | 50.5 ± 1.1 | 47.1 ± 1.9 | 0.4 |
| LVESD (mm) | 30.1 ± 3.1 | 27.7 ± 2.6 | 0.5 |
| IVSEDT (mm) | 7.1 ± 1.1 | 6.5 ± 1.1 | 0.6 |
| LVPWEDT (mm) | 9.1 ± 0.5 | 8.1 ± 0.6 | 0.4 |
| LVM/H 2.7 (g/m 2.7) | 35.7 ± 1.1 | 33.4 ± 4.2 | 0.4 |
| LVEDV (ml) | 105.5 ± 19.2 | 96.8 ± 19.9 | 0.4 |
| LVESV (ml) | 38.2 ± 12.5 | 36.9 ± 13.1 | 0.4 |
| E wave (cm/s) | 81.7 ± 14.9 | 85.8 ± 11.1 | 0.4 |
| A wave (cm/s) | 58.9 ± 12.6 | 52.2 ± 9.1 | 0.4 |
| E/A ratio | 1.3 ± 1.1 | 1.4 ± 1.2 | 0.4 |
| Edt (ms) | 192.1 ± 9.5 | 190.1 ± 9.6 | 0.4 |
| Left atrial diameter (mm) | 38.2 ± 1.3 | 35.9 ± 2.8 | 0.5 |
| Left atrial area (cm$^2$) | 20.1 ± 2.9 | 18.8 ± 3.8 | 0.4 |
| Right atrial area (cm$^2$) | 20.5 ± 3.3 | 19.6 ± 1.9 | 0.4 |
| LAVI (ml/m$^2$) | 22.2 ± 2.1 | 21.6 ± 1.8 | 0.4 |
| LA *V*max, ml/m$^2$ | 32.4 ± 7.1 | 32.2 ± 7.8 | 0.4 |
| LA *V*min, ml/m$^2$ | 13.1 ± 1.9 | 11.8 ± 4.2 | 0.4 |
| LA *V*p, ml/m$^2$ | 22.1 ± 3.9 | 23.2 ± 4.8 | 0.5 |
| LA EF, % | 32.8 ± 7.5 | 33.3 ± 7.1 | 0.4 |
| LATEV, ml/m$^2$ | 23.3 ± 4.7 | 23.5 ± 5.8 | 0.4 |
| LAAEF, % | 0.46 ± 0.08 | 0.44 ± 0.06 | 0.4 |
| LAAEV, ml/m$^2$ | 8.1 ± 3.7 | 9.2 ± 3.2 | 0.4 |
| LAPEF, % | 0.39 ± 0.05 | 0.39 ± 0.06 | 0.5 |
| LAPEV, ml/m$^2$ | 12.9 ± 3.5 | 13.5 ± 3.8 | 0.4 |
| P wave duration, ms | 100.4 ± 20.9 | 65.9 ± 8.2 | 0.03 |
| Sino-atrial node block, *n* (%) | 18 (25.7) | 0 (0) | |
| First-degree AV block, *n* (%) | 32 (45.7) | 0 (0) | |
| Second-degree AV block, *n* (%) | 9 (12.8) | 15 (21.4) | |
| Combined AVN and SAN block, *n* (%) | 11 (15.7) | 0 (0) | |
| Cardioinhibitory vasovagal syncope (%) | 0 (0) | 55 (78.6) | |

*BMI* body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HR* heart rate, *EF* ejection fraction, *FS* short fraction, *LVEDD* left ventricular end-diastolic diameter, *LVESD* left ventricular end-systolic diameter, *IVSEDT* inter-ventricular septum end-diastolic diameter thickness, *LVPWEDT* left ventricular posterior wall end-diastolic thickness, *LVM/H 2.7* left ventricular mass indexed to height to the power of 2.7, *LVEDV* left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume, *Edt* E wave deceleration time, *LAVI* left atrial indexed volume, *LA Vmax* left atrium maximum volume, *LA Vmin* left atrium minimum volume, *LA Vp* left atrium volume before atrial systole, *LAEF* left atrium ejection fraction, *LATEV* left atrium total emptying volume, *LAAEF* left atrium active emptying fraction, *LAAEV* left atrium active emptying volume, *LAPEF* left atrium passive emptying fraction, *LAPEV* left atrium passive emptying volume

**Table 2** Atrial electromechanical delay values according to different atrial pacing sites in DM1 patients

| | Sinus rhythm | RAA pacing | BB pacing | *P** |
|---|---|---|---|---|
| Inter-AEMD (ms) | 48.2 ± 17.8 | 50.5 ± 16.5 | 36.1 ± 17.1 | 0.001 |
| Intra-left AEMD (ms) | 43.3 ± 15.5 | 44.6 ± 15.8 | 32.5 ± 15.2 | 0.001 |
| Intra-right AEMD (ms) | 14.1 ± 4.2 | 15.4 ± 5.8 | 12.2 ± 4.6 | 0.2 |

*Inter-AEMD* inter-atrial electromechanical delay, *Intra-left AEMD* intra-left atrial electromechanical delay

**P* interaction between AEMD values of BB and RAA pacing

AEMD (36.1 ± 17.1 vs 50.5 ± 16.5 ms; $P = 0.001$) and intra-left AEMD (32.5 ± 15.2 vs 44.6 ± 15.8 ms; $P = 0.001$) values than temporary RAA pacing. No statistically significant difference was found in intra-right AEMD (12.2 ± 4.6 vs 15.4 ± 5.8 ms; $P = 0.2$) (Table 2; Fig. 4). There was not a statistically significant difference in P wave amplitude (2.96 ± 0.8 vs 2.76 ± 1.4 mV; $P = 0.2$), atrial pacing threshold (0.46 ± 0.14 vs 0.66 ± 0.46 V; $P = 0.2$), or atrial bipolar impedance (634.79 ± 122.15 vs 567.56 ± 281.87; $P = 0.2$) between BB and RAA pacing. In the control group, neither temporary RAA pacing nor temporary BB pacing showed statistically significant differences in inter-AEMD, intra-left AEMD, or intra-right AEMD values in comparison with sinus rhythm (Table 3).

## 4 Discussion

In the present study, we compared the impact of temporary pacing from the inter-atrial septum in the region of Bachmann's bundle with that of right atrial appendage pacing on atrial electromechanical delay in DM1 patients undergoing PM implantation for cardiac rhythm abnormalities. Studying the atrial electromechanical delay in DM1 patients with conserved left ventricular systolic function and normal left atrial mechanical function offers an interesting clinical opportunity to exclude the influence of LA enlargement, LV overload, and other possible comorbidities on the evaluation of the inhomogeneous atrial propagation of sinus impulses and to determine the relationship between the different atrial pacing sites and AEMD indices. According to previous reports in DM1 populations, atrial lead placement in the region of BB is affected by a low rate of sensing and pacing defects [11, 12] and offers the advantage of reducing the oversensing of the R wave on the atrial lead [21]. The role of BB pacing in the prevention of atrial fibrillation is still controversial [22–26]. Bailin et al. [22] found that BB pacing reduced progression to chronic atrial fibrillation when compared with RAA pacing in patients with sinus bradycardia and paroxysmal atrial fibrillation. Yu et al. [23] showed that single-site pacing at Bachmann's bundle was more effective than biatrial or dual-site atrial pacing in preventing the induction of AF. Nevertheless, in a larger multi-center, randomized study, the Atrial Septal Pacing Efficacy Clinical trial (ASPECT) [24], the atrial septal lead location did not reduce AF frequency or burden in spite of reducing premature atrial complexes. These results were confirmed by Hakacova et al. [25] in a small group of patients with drug-refractory paroxysmal AF undergoing pacemaker implantation, and by Katsivas et al. [26] in a randomized controlled study that included patients requiring atrial pacing for sinus node dysfunction without documented AF episodes in the 3 months before pacing.

The duration of atrial electromechanical delay, as evaluated by TDI echocardiography, can be used for the non-invasive measurement of inter-atrial and intra-left atrial conduction times. In particular, intra- and inter-AEMD are useful parameters for assessing the risk of AF in some clinical conditions [13–18]. In our DM1 patients, the atrial electromechanical delay indices (inter-AEMD and intra-left AEMD) were significantly higher than in age- and sex-matched healthy controls. A cutoff value of 39.2 ms for intra-left AEMD displayed a sensitivity and specificity of 90% in identifying DM1 patients at high risk of AF. A cutoff value of 57.7 ms for inter-AEMD

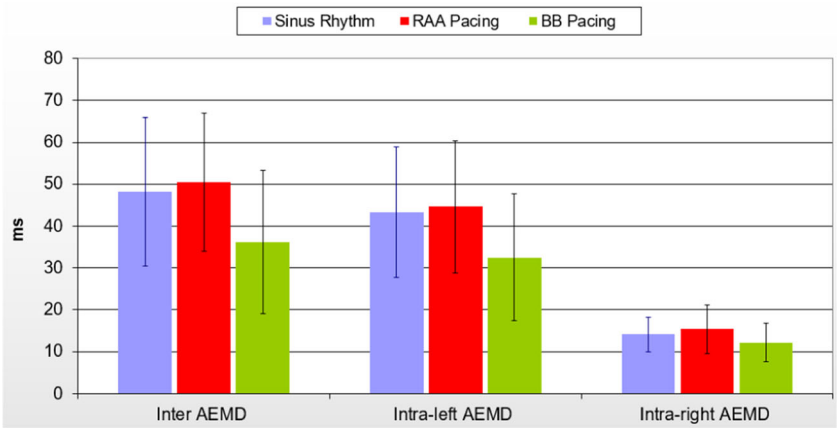


**Fig. 4** Effect of different atrial pacing sites on AEMD parameters in DM1 patients

**Table 3** Atrial electromechanical delay values according to different atrial pacing sites in the control group

| | Sinus rhythm | RAA pacing | BB pacing | *P** |
|---|---|---|---|---|
| Inter-AEMD (ms) | 32.8 ± 9.9 | 31.9 ± 9.8 | 32.1 ± 9.7 | 0.2 |
| Intra-left AEMD (ms) | 29.4 ± 9.5 | 29.5 ± 10.2 | 30.1 ± 10.1 | 0.2 |
| Intra-right AEMD (ms) | 12.8 ± 7.1 | 14.4 ± 5.8 | 14.2 ± 5.9 | 0.2 |

*Inter-AEMD* inter-atrial electromechanical delay, *Intra-left AEMD* intra-left atrial electromechanical delay

**P* interaction between AEMD values of BB and RAA pacing

had a sensitivity of 84.2% and a specificity of 93.5% in identifying DM1 patients at high risk of developing AF [27]. Choosing the optimal atrial lead position according to the electrical parameters and to AEMD values could be a useful means of reducing the AF risk in DM1 patients. To the best of our knowledge, ours is the first study to assess the effect of BB and RAA pacing on echocardiographic parameters that reflect heterogeneity of atrial depolarization in DM1 patients undergoing pacemaker implantation.

### 4.1 Main findings

In our DMI population, temporary BB pacing was associated with significantly lower inter-AEMD and intra-left AEMD values than temporary RAA pacing, when sensing and pacing electrical parameters were equal. No statistically significant difference was found in intra-right AEMD. Moreover, the lack of significant differences in the control group between AEMD values during temporary BB pacing and RAA pacing in comparison with sinus rhythm suggests that the decreased AEMD seen in DM1 patients during BB pacing may be related to the greater proximity of this pacing site to the areas of slow conduction, such as the BB region and the left atrium. Further studies, including the electro-anatomical mapping of the atrial chambers, are necessary to better define the distribution of fibrosis pattern and to assess the clinical benefits of alternative atrial pacing sites on AF burden reduction in DM1 patients.

### 4.2 Limitations

The present study assessed only the acute effects of atrial pacing from different sites (BB and RAA) on AEMD in DM1 patients, and it did not consider the long-term effects of BB pacing on AEMD in this population. We are therefore unable to comment on whether the changes observed would be maintained or become progressive. Further studies are necessary to address the clinical benefits of alternative atrial pacing sites on AF burden reduction in DM1 patients. The echocardiographic measurements were performed in the only supine position because of the need of keeping the patient in this position during the PM implantation; this represents a limitation of the present study, considering that the optimal apical four-chamber view can be obtained only with left lateral position. The Doppler measurements were made setting the monitor sweep speed at 50–100 mm/s. The decision of the minimum speed to adopt was left to the physician's choice and experience, however only for a small percentage of DM1 patients; we used a speed lower than 100 mm/s ($n = 3/70$, 4.3%); in the remaining part of the study population (66/70, 95.7%), we used a speed of 100 mm/s. Moreover, if on the one hand the presence of first-degree AV block in 45.7% of the study population determined the overlap of tissue E and A waves, making onset of A difficult to find, on the other hand, the intra- and inter-observer coefficients of variation were found to be less than 5% and not significant.

## 5 Conclusions

According to our results, the atrial pacing in the Bachmann bundle region is associated with significantly lower echocardiographic indices of atrial electromechanical delay (inter-AEMD and intra-left AEMD) than right atrial appendage pacing in the DM1 population undergoing dual-chamber PM implantation. Further studies should be conducted to confirm these results in a long-term follow-up evaluation and to correlate the decreased atrial electromechanical delay with the atrial fibrillation burden.

## Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

**Research involving human participants** All procedures performed in studies involving human participants were carried out in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

**Informed consent** Informed consent was obtained from all individual participants included in the study.

## References

1. Pelargonio G, Dello Russo A, Sanna T, et al. Myotonic dystrophy and the heart. Heart. 2002;88:665–70.

2. Groh WJ, Groh MR, Chandan S, et al. Electrocardiographic abnormalities and sudden death in myotonic dystrophy type 1. N Engl J Med. 2008;358:2688–97.
3. Laurent V, Pellieux S, Corcia P, Magro P, Pierre B, Fauchier L, et al. Mortality in myotonic dystrophy patients in the area of prophylactic pacing devices. Int J Cardiol. 2011;150:54–8.
4. Russo V, Di Meo F, Rago A, Papa AA, Molino A, Mosella M, et al. Paroxysmal atrial fibrillation in myotonic dystrophy type 1 patients: P wave duration and dispersion analysis. Eur Rev Med Pharmacol Sci. 2015;19:1241–8.
5. Russo V, Papa AA, Rago A, Nigro G. Which is the true epidemiology of atrial fibrillation in myotonic dystrophy type 1 patients? Pacing Clin Electrophysiol. 2016 Dec;39(12):1418–9.
6. Russo V, Nigro G, Rago A, Antonio Papa A, Proietti R, Della Cioppa N, et al. Atrial fibrillation burden in myotonic dystrophy type 1 patients implanted with dual chamber pacemaker: the efficacy of the overdrive atrial algorithm at 2 year follow-up. Acta Myol. 2013;32:142–7.
7. Russo V, Rago A, Papa AA, Politano L, Golino P, Russo MG, et al. Does a high percentage of right ventricular pacing influence the incidence of paroxysmal atrial fibrillation in myotonic dystrophy type 1 patients? Kardiol Pol. 2013;71:1147–53.
8. Russo V, Nigro G, Papa AA, Rago A, Della Cioppa N, Cristiano A, et al. Adenosine-induced sinus tachycardia in a patient with myotonic dystrophy type 1. Acta Myol. 2014;33:104–6.
9. Brembilla-Perrot B, Schwartz J, Huttin O, Frikha Z, Sellal JM, Sadoul N, et al. Atrial flutter or fibrillation is the most frequent and life-threatening arrhythmia in myotonic dystrophy. Pacing Clin Electrophysiol. 2014 Mar;37(3):329–35.
10. Russo V, Rago A, Nigro G. Sudden cardiac death in neuromuscolar disorders: time to establish shared protocols for cardiac pacing. Int J Cardiol. 2016 Mar 15;207:284–5.
11. Nigro G, Russo V, Vergara P, D'Andrea A, Di Gregorio G, Politano L, et al. Optimal site for atrial lead implantation in myotonic dystrophy patients: the role of Bachmann's bundle stimulation. Pacing Clin Electrophysiol. 2008;31:1463–6.
12. Nigro G, Russo V, Politano L, Della Cioppa N, Manfredi D, Chianese R, et al. Right atrial appendage versus Bachmann's bundle stimulation: a two-year comparative study of electrical parameters in myotonic dystrophy type-1 patients. Pacing Clin Electrophysiol. 2009;32:1191–6.
13. Antoni ML, Bertini M, Atary JZ, Delgado V, ten Brinke EA, Boersma E, et al. Predictive value of total atrial conduction time estimated with tissue Doppler imaging for the development of new-onset atrial fibrillation after acute myocardial infarction. Am J Cardiol. 2010;106:198–203.
14. Ari H, Ari S, Akkaya M, Aydin C, Emlek N, Sarigül OY, et al. Predictive value of atrial electromechanical delay for atrial fibrillation recurrence. Cardiol J. 2013;20(6):639–47.
15. Calik AN, Ozcan KS, Cağdaş M, Güngör B, Karaca G, Gürkan U, et al. Electromechanical delay detected by tissue Doppler echocardiography is associated with the frequency of attacks in patients with lone atrial fibrillation. Cardiol J. 2014;21(2):138–43.
16. Russo V, Rago A, Di Meo F, Papa AA, Ciardiello C, Cristiano A, et al. Atrial septal aneurysms and supraventricular arrhythmias: the role of atrial electromechanical delay. Echocardiography. 2015;32:1504–14.
17. Rago A, Russo V, Papa AA, Ciardiello C, Pannone B, Mayer MC, et al. The role of the atrial electromechanical delay in predicting atrial fibrillation in beta-thalassemia major patients. J Interv Card Electrophysiol. 2017 Mar;48(2):147–57.
18. Russo V, Di Meo F, Rago A, Mosella M, Molino A, Russo MG, et al. Impact of continuous positive airway pressure therapy on atrial electromechanical delay in obesity-hypoventilation syndrome patients. J Cardiovasc Electrophysiol. 2016 Mar;27(3):327–34.
19. Brignole M, Auricchio A, Baron-Esquivias G, Bordachar P, Boriani G, Breithardt OA, et al. 2013 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy: the Task Force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). Eur Heart J. 2013 Aug;34(29):2281–329.
20. Deniz A, Sahiner L, Aytemir K, Kaya B, Kabakci G, Tokgozoglu L, et al. Tissue Doppler echocardiography can be a useful technique to evaluate atrial conduction time. Cardiol J. 2012;19:487–93.
21. Russo V, Nigro G, Antonio Papa A, Rago A, Di Meo F, Cristiano A, et al. Far field R-wave sensing in myotonic dystrophy type 1: right atrial appendage versus Bachmann's bundle region lead placement. Acta Myol. 2014;33:94–9.
22. Bailin SJ, Adler S, Giudici M. Prevention of chronic atrial fibrillation by pacing in the region of Bachmann's bundle: results of a multicenter randomized trial. J Cardiovasc Electrophysiol. 2001;12:912–7.
23. Yu WC, Tsai CF, Hsieh MH, Chen CC, Tai CT, Ding YA, et al. Prevention of the initiation of atrial fibrillation: mechanism and efficacy of different atrial pacing modes. Pacing Clin Electrophysiol. 2000;23:373–9.
24. Padeletti L, Pürerfellner H, Adler SW, Waller TJ, Harvey M, Horvitz L, et al. Combined efficacy of atrial septal lead placement and atrial pacing algorithms for prevention of paroxysmal atrial tachyarrhythmia. J Cardiovasc Electrophysiol. 2003;14:1189–95.
25. Hakacova N, Velimirovic D, Margitfalvi P, Hatala R, Buckingham TA. Septal atrial pacing for the prevention of atrial fibrillation. Europace. 2007;9:1124–8.
26. Katsivas A, Manolis AG, Lazaris E, Vassilopoulos C, Louvros N. Atrial septal pacing to synchronize atrial depolarization in patients with delayed interatrial conduction. Pacing Clin Electrophysiol. 1998;21:2220–5.
27. Russo V, Rago A, Ciardiello C, Russo MG, Calabrò P, Politano L, et al. The role of the atrial electromechanical delay in predicting atrial fibrillation in myotonic dystrophy type 1 patients. J Cardiovasc Electrophysiol. 2016 Jan;27(1):65–72.