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The Impact of Differential Atrioventricular Delays on Arterial Distensibility Measured by Pulse Wave Velocity in Patients with Dual-chamber Pacing

Çift Odacıklı Kalp Pili olan Hastalarda Değişken Atriyoventriküler Gecikme Zamanlarının Nabız Dalga Hızı ile Ölçülen Arteriyel Genişleyebilirlik Üzerine Etkisi

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Abstract

Objective: Atrioventricular (AV) delay optimization improves hemodynamics and clinical parameters of patients treated with dual-chamber (DDD) pacemakers. Pulse wave velocity (PWV) is an index of arterial stiffness and a marker of cardiovascular events. Increased levels of PWV is an indicator of diffuse atherosclerosis and rigid arteries. We aimed to investigate the impact of differential AV delay intervals (100, 150 and 200 ms respectively) on arterial distensibility measured by carotid-femoral PWV in patients with DDD pacing.

Method: A total of 40 patients with DDD pacing were enrolled in our prospective study. PWV was measured for each AV delay (100 ms, 150 ms and 200 ms respectively) with 10 minute resting intervals. Pacing was programmed at least 10 beats/minute above the resting heart rate. PWV was automatically calculated by using the device, Complior Colson (France), which allows online pulse wave recording according to this formula: PWV(m/s) = distance (meter)/transit time (second).

Results: There was a statistically obvious positive correlation between carotid-femoral PWV and prolongation of AV delay interval ($p<0.001$). According to gender analysis; weight, height and waist-hip ratio values were significantly higher in male group. Additionally, there was not a statistically significant difference between groups in terms of age, body mass index, mean, systolic and diastolic blood pressure, pulse pressure and PWV measured at differential AV delay intervals.

Conclusion: Prolongation of AV delay interval increased carotid-femoral PWV values thereby decreased arterial distensibility in patients with DDD pacing. Therefore, optimum AV delay adjustment may provide better hemodynamics in these patients.

Keywords: Atrioventricular delay interval, dual-chamber pacemaker, pulse wave velocity

Öz

Amaç: Atriyoventriküler (AV) gecikme optimizasyonu, çift odacıklı (DDD) kalp pilleri ile tedavi edilen hastaların hemodinamiklerini ve klinik parametrelerini iyileştirir. Nabız dalga hızı (NDH), arteriyel sertliğin bir indeksi ve kardiyovasküler olayların bir belirtecidir. Artan NDH seviyeleri, yaygın ateroskleroz ve sertleşmiş arterlerin bir göstergesidir. Diferansiyel AV gecikme aralıklarının (sırasıyla 100, 150 ve 200 ms) DDD pacingli hastalarda karotis-femoral NDH ile ölçülen arteriyel esneyebilirlik üzerindeki etkisini araştırmayı amaçladık.

Yöntem: Prospektif çalışmamıza DDD pacingli toplam 40 hasta alındı. NDH, her AV gecikmesi için (sırasıyla 100 ms, 150 ms ve 200 ms) 10 dakikalık dinlenme aralıklarıyla ölçüldü. Pacing, dinlenme kalp hızının en az 10 vuruş/dakika üzerine programlandı. NDH çevrimiçi nabız dalgası kaydına izin veren Complior Colson (Fransa) cihazı kullanılarak, şu formüle göre otomatik olarak hesaplandı: PWV(m/s) = mesafe (metre)/geçiş süresi (saniye).

Bulgular: Karotis-femoral NDH ile AV gecikme aralığının uzaması arasında istatistiksel olarak belirgin pozitif korelasyon vardı ($p<0,001$). Cinsiyet analizine göre; ağırlık, boy ve bel-kalça oranı değerleri erkek grubunda anlamlı olarak daha yüksekti. Ayrıca yaş, vücut kitle indeksi, ortalama, sistolik ve diyastolik kan basıncı, nabız basıncı ve diferansiyel AV gecikme aralıklarında ölçülen NDH açısından gruplar arasında istatistiksel olarak anlamlı bir fark yoktu.

Sonuç: AV gecikme aralığının uzaması karotis-femoral NDH değerlerini artırdı ve bu nedenle DDD pacingli hastalarda arteriyel distensibiliteyi azalttı. Optimum AV gecikmenin ayarlanması bu hastalarda daha iyi bir hemodinami sağlayabilir.

Anahtar kelimeler: Atriyoventiküler gecikme aralığı, çift odacıklı kalp pili, nabız dalga hızı

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Introduction

Newer generation dual-chamber (DDD) pacemakers have been widely used as they are considered to be more physiological (1). However, DDD pacemakers require more timers than other devices as they include several types of intervals, such as the basic and atrioventricular (AV) delay intervals.

Arterial stiffness is an indicator of atherosclerosis and caused by loss of elasticity and thickening of the arterial wall. Increased arterial stiffness is not only an indicator of vascular aging, but also a predictor of target organ damage and increased cardiovascular events (2). Due to the insidious nature of the atherosclerotic process, early recognition of arterial changes and functional and/or structural lesions may help to identify patients with high risk for clinical complications.

Pulse wave velocity (PWV) is one of the most important parameters used to evaluate elastic properties of large arteries (3,4). Also, using PWV measurements provides an easy and reproducible method to assess arterial stiffness (5). PWV is measured by using two ultrasound or pressure sensitive transducers fixed transcutaneously on the trace of a couple of artery at a certain distance from each other (such as carotid-femoral or brachial-radial arteries) (3). Increase in PWV values, which is defined as the velocity of the pressure wave over an arterial segment, is an important indicator of the diffuse atherosclerotic process (6). Arterial PWV is inversely proportional to arterial compliance and PWV propagation time (PWVPT) (3,4).

In addition, increased heart rate causes an increase in arterial PWV (7); thereby a decrease in arterial distensibility and compliance. Previous studies have shown that changes in AV delay interval affect both the left ventricular filling time and stroke volume of the patients (8,9). It is unknown whether other different factors such as AV conduction time affects PWV or not in case of a fixed heart rate.

Therefore, we assumed that PWV values calculated at different AV delay intervals may guide optimal programming to provide better hemodynamics in patients with DDD pacemakers. Due to the lack of data regarding this issue, we aimed to investigate the effect of differential AV delay intervals (100, 150 and 200 ms, respectively) on arterial distensibility measured by the carotid-femoral PWV in patients with DDD pacing.

Materials and Methods

Fourty consecutive patients with DDD pacemakers who were admitted to our outpatient clinic between January to July 2015, were recruited in our single-center and prospective clinical study. Written informed consent was taken from each participant. Our study was conducted in concordance with the Declaration of Helsinki and approved by the Local Ethics Committee of İstanbul University (09/07/2014, no: B.08.06.YOK.2.I.U.E.50.0.05.00/10).

None of the patients had a pacemaker other than DDD mode. Exclusion criteria included patients with coronary artery disease, cerebrovascular disease, peripheral artery disease, heart failure, liver or kidney failure, atrial fibrillation and patients using non-steroidal anti-inflammatory or steroid drugs during the study. We also excluded patients with body mass index (BMI) ≥35 kg/m² and waist-hip ratio (WHR) ≥1.

Patients were asked to rest in a supine position for 10 minutes before starting the measurements. Following each 10 minute period of haemodynamic stabilization, AV delay intervals were gradually prolonged and programmed as 100, 150 and 200 ms, respectively. Three different AV delays were used for testing by AV sequential pacing. Carotid-femoral artery PWV and blood pressure (BP) were measured by same researcher. Pacing was programmed at a minimum of 10 beats/minute above the resting heart rate. Also, pacing rate was further increased when continuous capture could not be obtained (8). Weight measurements of the patients were made by scale, whereas height measurements by meter. BMI was calculated by dividing the weight in kilogramme by the height calculated in square meters (kg/m^2). The waist circumference (cm) was measured from the mid point of the distance between the lowest rib and the crista iliaca when the patient was in upright position. The hip circumference (cm) was measured from the major trochanter femoris level. WHR was calculated by dividing waist circumference by hip circumference. Arterial BP was measured with a standard mercury manometer in supine position following a 10-minute period of rest. Systolic BP (SBP) was accepted as the value at which Korotkoff sounds were heard first and diastolic BP (DBP) as the value at which sounds disappeared (phase 5). Pulse pressure (PP) was calculated by subtracting DBP from SBP. Mean BP (MBP) was determined using the formula MBP = SBP + 2xDKB/3.PWV measurements were taken immediately after obtaining the brachial BP. Aortic PWV was calculated using the Complior device (Createch Industrie, France), which allows automatic online pulse wave recording

and automatic calculation of PWV (3). Common carotid and femoral artery pressure waveforms were measured non-invasively using a pressure sensitive transducer TY-306 Fukuda (Fukuda, Tokyo, Japan). An average of ten repeated measurements was taken for final analysis. PWV was automatically calculated with the formula ΔD/Δt [ΔD: distance traveled by the pulse wave on the body surface (meters) between two recording points, Δt: Pulse wave transit time (seconds) automatically determined by the Complior device].

Statistical Analysis

Statistical analysis was performed by using the SPSS version 15.0 (SPSS Inc., Chicago, Illinois, USA) packet program. Continuous variables were reported as mean ± standard deviation while categorical variables were presented as numbers and percentages. The significance between different measurements of the same participant was determined by using the non-parametric Friedman test. If significance was detected in the Friedman test, the non-parametric Wilcoxon test was used to determine the source of significance. As AV delay parameters were non-normally distributed, Friedman and Wilcoxon tests were used. The non-parametric Mann-Whitney U test was used for comparison between the female and male groups. Student t-test was used for normally distributed data. The relationship between PWV and other variables was evaluated by Pearson correlation test. Values of $p<0.05$ were considered statistically significant.

Results

Of the 40 cases included in the study, 21 (52.5%) were male and 19 (47.5%) were female. Demographic characteristics and anthropometric measurements of the patients were shown in Table 1. Type II diabetes mellitus was present in 7 (36.8%) of 19 female cases and 5 (23.8%) of 21 male cases. Hypertension was found in 15 (78.9%) patients among women and 11 (52.3%) patients among men. Eight (42.1%) female and five (23.8%) male patients had hyperlipidemia.

The change in BP with respect to increasing AV delay intervals was shown in Table 2. No statistically significant difference was found between SBP, DBP, MBP and PP values measured at differential AV delay intervals ($p=0.105$, $p=0.264$, $p=0.449$ and $p=1.000$, respectively).

A statistically significant increase in PWV and a decrease in PWVPT were detected with the prolongation of AV delay intervals ($p<0.001$ and $p<0.001$, respectively) and graphically shown in Figure 1A and 1B. When the groups were compared among themselves by the Wilcoxon test; a statistically significant difference was found between PWV 100-150 ms, 100-200 ms and 150-200 ms (for PWV 100 ms vs. 150 ms, $p<0.001$; PWV 100 ms vs. 200 ms, $p<0.001$; PWV 150 ms vs. 200 ms $p=0.001$). Similarly, statistically significant results were obtained in the comparison between the groups in terms of PWVPT (for PWVPT 100 ms vs. 150 ms, $p=0.002$; PWVPT 100 ms vs 200 ms, $p<0.001$; PWVPT 150 ms vs. 200 ms $p<0.001$, respectively). A strong positive correlation ($p<0.001$, $r=0.590$) was found between age and the PWV values taken at 100 ms. Likewise, there was a strong positive correlation between age and PWV values taken at 150 ms and 200 ms (respectively $p<0.001$, $p<0.001$; $r=0.611$, $r=0.562$).

According to gender analysis that was given in Table 3; height, weight and WHR values of men were significantly higher than those of women ($p<0.001$, $p=0.016$ and $p=0.001$, respectively). Also, there was no statistically significant difference between women and men in terms of age, BMI, SBP, DBP, MBP, PP, PWV and PWVPT values measured at differential AV delay intervals (100, 150 and 200 ms, respectively).

Discussion

To the best of our knowledge, this is the first study to investigate the effect of different AV delay intervals on arterial distensibility measured by carotid-femoral PWV in patients with DDD pacing. Our study demonstrated a statistically significant increase in arterial PWV and thereby a decrease in PWVPT with gradually increasing AV delay intervals (100, 150 and 200 ms). In this respect, the results of our study suggest that prolongation of AV conduction time may increase arterial stiffness in patients with classical DDD pacemakers.

Nishimura et al. (10) examined the patients with dilated cardiomyopathy into two groups according to PR intervals whether above or below 200 milliseconds. A significant increase in cardiac output of patients in long PR interval group was noted with AV interval optimization. Likewise, Manisty et al. (8) evaluated hemodynamic changes occurred at different AV delay intervals (40-120 ms) in 19 patients with permanent pacemaker, and observed an acute improvement in hemodynamic parameters (SBP, DBP, MBP, PP and stroke volume) as the AV delay interval approached from non-physiological (40 ms) to physiological values (120 ms). Contrary to this study, no significant difference was found in our study in terms of hemodynamic parameters. This may be due to the fact that

we recorded our measurements in a more physiological range of AV delay, such as 100 ms to 200 ms.

Patients whose arterial PWV values increase may have worse cardiac functions due to the increased workload in front of the left ventricle. Also, decrease in arterial distensibility may lead to decreased effective coronary blood flow and increased myocardial ischemia (11). Various studies have suggested that increased PWV values may be related to increased adverse cardiovascular events and mortality (3,6,11,12).

PWV may be affected by different parameters such as age, heart rate, SBP, DBP, MBP, and PP (12,13). In the present study, a significant relationship was found between PWV and age. As the age increases, the elastic tissue of the aorta gradually decreases and therefore stiffness increases. Additionally, increasing the right ventricular pacing rate may increase arterial stiffness. Yıldız et al. (14) compared 17 patients with classical DDD pacemakers with healthy subjects in terms of arterial distensibility determined by PWV, and found an increase in arterial PWV and a decrease in distensibility in patients with pacemakers in terms of gender and age compared to the control group. Similarly, Wilkinson et al. (15) reported a linear relationship between heart rate and arterial stiffness in patients with permanent atrial or dual pacemakers. Krishnamoorthy et al. (16)

Table 1. Demographic characteristics and anthropometric measurements of the study group

	Mean ± SD; or n (%)	Range
Age (years)	62.4±15.9	(23-84)
Male gender	21 (52.5)	
Body height (cm)	165.0±8.12	(150-180)
Body weight (kg)	75.3±12.02	(48-97)
Waist circumference (cm)	96.43±10.39	(67-110)
Hip circumference (cm)	105.95±7.75	(87-125)
WHR	0.90±0.00	(0.73-0.99)
BMI (kg/m^2)	27.63±3.75	(34.17-17.63)
Diabetes, n (%)	12 (30)	
Hypertension, n (%)	26 (65)	
Hyperlipidemia, n (%)	13 (32.5)	

BMI: Body mass index, SD: Standard deviation, WHR: Waist-to-hip ratio, SD: Standard deviation

Table 2. Blood pressure values according to different AV delay intervals

	AV delay 100 ms	AV delay 150 ms	AV delay 200 ms	p-value
SBP (mmHg)	129.50±20.12	128.75±19.90	128.75±19.37	0.105
DBP (mmHg)	78.75±10.42	78.25±10.10	78.25±9.84	0.264
MBP (mmHg)	95.66±12.78	95.08±12.56	95.08±12.15	0.449
PP (mmHg)	50.25±13.68	50.50±13.77	50.50±13.77	1.000

AV: Atrioventricular, DBP: Diastolic blood pressure, MBP: Mean blood pressure, PP: Pulse pressure, SBP: Systolic blood pressure

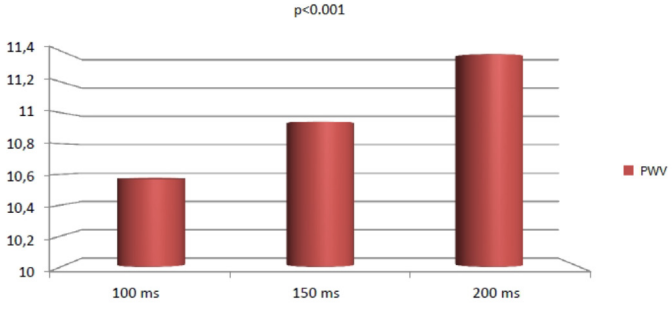

Figure 1A. PWV values measured at different AV delay intervals

PWV: Pulse wave velocity, AV: Atrioventricular

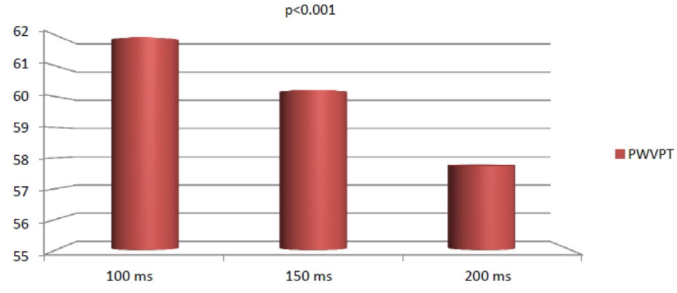

Figure 1B. PWVPT values measured at different AV delay intervals

AV: Atrioventricular, PWVPT: Pulse wave velocity propagation time

Table 3. Comparison of anthropometric and hemodynamic measurements between the groups

	Female	Male	p-value
Body weight (kg)	70.16±12.04	79.95±10.17	0.016
Body height (cm)	158.11±4.58	171.24±4.86	<0.001
BMI (kg/m^2)	28.04±4.44	27.25±3.22	0.523
WHR	0.87±0.06	0.93±0.03	0.001
SBP-100 msec (mmHg)	132.63±21.56	126.67±18.80	0.361
SBP-150 msec (mmHg)	131.05±21.32	126.67±18.80	0.469
SBP-200 msec (mmHg)	131.05±20.25	126.67±18.80	0.486
DBP-100 msec (mmHg)	80.00±11.06	77.62±9.95	0.503
DBP-150 msec (mmHg)	78.95±10.49	77.62±9.95	0.668
DBP-200 msec (mmHg)	78.95±9.94	77.62±9.95	0.668
MBP-100 msec (mmHg)	97.53±13.67	93.97±12.00	0.376
MBP-150 msec (mmHg)	96.32±13.37	93.97±12.00	0.486
MBP-200 msec (mmHg)	96.32±12.52	93.97±12.00	0.503
PP-100 msec (mmHg)	52.63±14.85	48.10±12.50	0.333
PP-150 msec (mmHg)	52.11±14.37	49.05±13.38	0.520
PP-200 msec (mmHg)	52.11±14.37	49.05±13.38	0.520
PWV-100 msec (m/s)	10.82±2.67	10.35±2.05	0.630
PWV-150 msec (m/s)	11.29±2.97	10.62±2.04	0.421
PWV-200 msec (m/s)	11.81±3.30	11.00±2.36	0.537
PWVPT-100 msec (m/s)	59.36±14.82	64.23±11.68	0.145
PWVPT-150 msec (m/s)	57.21±15.06	62.90±11.78	0.083
PWVPT-200 msec (m/s)	55.10±15.12	60.14±12.53	0.226

BMI: Body mass index, WHR: Waist-to-hip ratio, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MBP: Mean blood pressure, PP: Pulse pressure, PWV: Pulse wave velocity, PWVPT: Pulse wave velocity propagation time

divided 101 patients with DDD pacemakers into two groups according to the presence of atrial high-rate episodes. An increase in arterial stiffness was detected in the group with atrial high velocity episodes. These studies were not conducted at a constant heart rate unlike our study, in which the effect of heart rate was excluded.

The effect of differential AV delay intervals on arterial stiffness still remains controversial. Differential AV delay intervals may lead to hemodynamic adaptation and lengthen the time of systole or diastole (17). The prolongation of systolic phase, occured by AV conduction programming, may cause an increase in left ventricular stroke volume and thus in the amount of blood passed and consequently an increase in aortic pressure. Chan et al. (18) investigated the changes in vascular function with PR (AV delay interval) prolongation in 88 healthy individuals. As the AV interval prolonged, a decrease in flow-mediated dilatation and an increase in PWV was observed. This also indicates that PR prolongation may a negative effect on arterial mechanics.

Evaluation of arterial stiffness is increasingly important in patients with high cardiac risk such as the presence of a permanent pacemaker. Based on this, measuring arterial expandability besides hemodynamic changes when programming the pacemaker seems to be reasonable in these patients. If their devices are programmed for the optimum AV delay interval, these patients may experience improvement in large artery mechanics such as arterial expansion.

Study Limitations

There were some limitations to be noted in our study. First of all, our results were based on a single-center study and relatively small number of patients were analyzed. Secondly, most of the patients have coexistent medical conditions such as diabetes mellitus and hypertension; therefore our results might not accurately reflect endothelial dysfunction associated with AV delay interval prolongation. Antihypertensive and antidiabetic drugs also may effect our results.

Conclusion

In conclusion, the results of this study indicated that lengthening AV delay interval increased carotid-femoral

PWV in patients with DDD pacemakers. Thus, optimum AV delay adjustment may provide better hemodynamics in these patients. Nevertheless, further prospective studies are needed to confirm our findings in a larger cohort of patients.

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Ethics

Ethics Committee Approval: Fourty consecutive patients with DDD pacemakers who were admitted to our outpatient clinic between January to July 2015, were recruited in our single-center and prospective clinical study. Our study was conducted in concordance with the Declaration of Helsinki and approved by the Local Ethics Committee of İstanbul University (09/07/2014, no: B.08.06. YOK.2.I.U.E.50.0.05.00/10).

Informed Consent: Written informed consent was taken from each participant.

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Authorship Contributions

Concept: B.B.K., G.Ç., M.Y., Design: B.B.K., G.Ç., M.Y., Data Collection or Processing: B.B.K., G.Ç., Analysis or Interpretation: B.B.K., G.Ç., M.Y., Literature Search: B.B.K., G.Ç., Writing: B.B.K., G.Ç.

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