



CrossMark

Prognosis of patients with implanted pacemakers in 4-year follow-up

Impact of right ventricular pacing site

For over 50 years, pacing has been the method of choice in the treatment of electrical conduction disorders [1–5]. The apical location for pacing was preferred since it provided the possibility to stabilize the location of the ventricular lead. The currently available active-fixation leads offer freedom of choice regarding the right ventricular (RV) pacing site. This is important because of the adverse consequences of long-term pacing of the RV apex [6, 7].

It was reported that apical pacing leads to impaired segmental shortening, reduction in the speed of pressure increase in the initial contraction period, abnormalities in contraction geometry, and hemodynamic disorders [8–10]. In addition, apical pacing extends the time of relaxation and shortens the time of filling of the left ventricle, which can cause ineffective contraction of the interventricular septum (as in the case of left bundle branch block) and leads to a decrease in stroke volume, impairment of mitral valve function, and an increase in telesystolic volume [11].

In addition to the aforementioned disorders, there are also adverse changes in coronary flow [12]. These changes have negative metabolic consequences for the heart muscle, which are evident in increased oxygen-free metabolism [13, 14] and decreased left ventricular ejection fraction (LVEF) [15].

Although the majority of studies confirm the adverse consequences of long-term pacing, especially of the RV apex [16–19], unequivocal indications for the

optimal location of the right ventricular lead tip (RVLT) are still not defined.

Non-apical pacing of the right ventricle (RV) diminishes the influence of adverse pacing on the contraction desynchronization of the left ventricle and the associated poor electrical and hemodynamic cardiac function [20–24].

The RV outflow tract still remains the most frequently selected alternative site for ventricular lead implantation [25–27].

A meta-analysis of 14 studies confirmed the protective influence of non-apically stimulated RV with regard to echocardiographic and laboratory parameters as well as physical sufficiency and life quality [28]. However, data on the relation between the site of RV pacing and long-term prognosis are still scarce and ambiguous.

The objective of this study was to evaluate whether there is an interrelationship between the site of the RVLT and prognosis in patients with implanted pacemakers over a 4-year observation period, taking other cardiovascular risk factors into consideration.

Methods

The clinical data of subsequent patients who had single- or dual-chamber (VVI, DDD) pacemakers implanted for typical indications during the period 2006–2008 at the II Department of Cardiology of the Medical University of Silesia were retrospectively analyzed.

The observation period of the study was 4 years. The following inclusion

criteria were applied: patients aged 18–80 years with typical indications for heart pacing (atrioventricular block, sick sinus syndrome, atrial fibrillation with slow ventricular action); LVEF greater than 40%.

The exclusion criteria were: patients with concurrent diseases significantly influencing the predicted lifespan (active cancer disease, severe kidney failure, persons abusing alcohol or narcotics, poorly controlled mental health disorders, acute coronary syndrome in the last 6 months). The study endpoint was all-cause mortality. Information on death was obtained from the National Health Fund database.

Study parameters

The demographic data collected included information on the patients' sex and their age at the time of implantation. The clinical data comprised indications for implantation and concurrent diseases (arterial hypertension, ischemic heart disease, diabetes, history of brain stroke).

Use of the following pharmaceuticals was also noted: beta-blockers (BB), angiotensin-converting enzyme inhibitors (ACE-I), angiotensin receptor blockers (ARB), thiazide and loop diuretics, statins, acetylsalicylic acid (ASS), oral anticoagulants (AVK), digitalis, oral anti-diabetic drugs, insulin therapy, and aldosterone antagonists (AA).

Further evaluations included laboratory parameters (hemoglobin, creatinine, cholesterol, and sodium concentrations) as well as echocardiographic parameters

Table 1 Baseline patient data and survival analysis (uni- and multivariable Cox regression)

	Entire group			Univariable Cox regression analysis			Multivariable Cox regression analysis		
	Entire group	Survival	Death	<i>p</i>	HR	95% CI	<i>p</i>	HR	95% CI
No. of patients	450 (100%)	359 (79.78%)	91 (20.22%)	–	–	–	–	–	–
Female	223 (49.6%)	193 (53.8%)	30* (33.0%)	0.001	0.463	0.30–0.72	0.000	0.426	0.27–0.67
Mean age at implantation	69.16 ± 9.63	68.14 ± 10.10	72.30*** ± 6.95	0.000	1.063	1.03–1.10	0.004	1.047	1.02–1.08
DDD	302 (67.11%)	256 (71.31%)	46*** (50.55%)	0.000	0.441	0.29–0.66	0.004	0.526	0.34–0.82
VVI	148 (32.89%)	103 (28.69%)	45*** (49.45%)	–	–	–	–	–	–
VVI-ARVLL	100 (22.22%)	66 (18.38%)	34*** (37.36%)	–	–	–	–	–	–
DDD-ARVLL	196 (43.56%)	166 (46.24%)	30* (32.97%)	–	–	–	–	–	–
n-ARVLL (all)	154 (34.2%)	127 (35.4%)	27 ^{ns} (29.7%)	0.259	0.772	0.49–1.21	–	–	–
SSS	197 (43.78%)	161 (44.85%)	36 ^{ns} (39.56%)	0.356	0.821	0.54–1.25	–	–	–
A-V block	193 (42.89%)	148 (41.23%)	45 ^{ns} (49.45%)	0.149	1.354	0.90–2.04	–	–	–
FA with brady-cardia	60 (13.33%)	50 (13.93%)	10 ^{ns} (10.99%)	0.457	0.779	0.40–1.50	–	–	–
CAD	141 (31.33%)	115 (32.03%)	26 ^{ns} (28.57%)	0.455	0.841	0.53–1.32	–	–	–
HA	213 (47.33%)	179 (49.86%)	34 ^{ns} (37.36%)	0.029	0.623	0.41–0.95	0.291	0.771	0.48–1.25
DM-2	98 (21.78%)	74 (20.61%)	24 ^{ns} (26.37%)	0.214	1.344	0.84–2.14	–	–	–
Stroke	24 (5.33%)	17 (4.74%)	7 ^{ns} (7.69%)	0.299	1.504	0.70–3.25	–	–	–
Hemoglobin (g/dl)	13.27 ± 1.65	13.31 ± 1.53	13.12 ^{ns} ± 2.05	0.303	0.933	0.82–1.06	–	–	–
Creatinine (μmol/l)	96.26 ± 8.10	84.12 ± 23.78	110.53 ^{ns} ± 57.24	0.000	1.008	1.01–1.01	0.003	1.005	1.00–1.01
Cholesterol (mg%)	185.30 ± 47.06	186.35 ± 47.64	180.6 ^{ns} ± 44.50	0.343	0.998	0.99–1.00	–	–	–
Sodium (mmol/l)	138.90 ± 4.35	139.01 ± 3.95	138.29 ^{ns} ± 5.67	0.140	0.963	0.91–1.01	–	–	–
LVEDV (ml)	88.43 ± 28.53	87.48 ± 26.44	92.93 ^{ns} ± 36.95	0.164	1.007	1.00–1.02	–	–	–
LVEF (%)	49.47 ± 7.73	49.48 ± 7.87	49.43 ^{ns} ± 7.13	0.913	0.998	0.96–1.04	–	–	–
BB	253 (56.22%)	213 (59.33%)	40* (43.96%)	0.005	0.550	0.36–0.83	0.500	0.838	0.50–1.40
ACE-I	227 (50.44%)	189 (52.65%)	38 ^{ns} (41.76%)	0.051	0.660	0.44–1.00	0.524	0.840	0.49–1.44
ARB	29 (6.44%)	26 (7.24%)	3 ^{ns} (3.30%)	0.182	0.456	0.14–1.45	–	–	–
Statins	174 (38.67%)	155 (43.18%)	19*** (20.88%)	0.000	0.371	0.22–0.62	–	–	–
TD	80 (17.78%)	69 (19.22%)	11 ^{ns} (12.09%)	0.094	0.583	0.31–1.10	0.643	0.846	0.42–1.71
TLD	76 (16.89%)	55 (15.32%)	21 ^{ns} (23.08%)	0.070	1.571	0.96–2.56	0.059	1.750	0.98–3.13
ASS	210 (46.67%)	177 (49.30%)	33* (36.26%)	0.020	0.601	0.39–0.92	0.143	0.675	0.40–1.14
AVK	63 (14.00%)	57 (15.88%)	6*** (6.59%)	0.027	0.391	0.17–0.90	0.017	0.330	0.13–0.82
Digoxin	41 (9.11%)	29 (8.08%)	12 ^{ns} (13.19%)	0.124	1.611	0.88–2.96	–	–	–
AA	74 (16.44%)	60 (16.71%)	14 ^{ns} (15.38%)	0.747	0.911	0.51–1.61	–	–	–
Oral anti-DM drugs	52 (11.56%)	42 (11.70%)	10 ^{ns} (10.99%)	0.840	0.934	0.48–1.80	–	–	–
Insulin therapy	27 (6.00%)	15 (4.18%)	12** (13.19%)	0.000	3.030	1.65–5.57	0.003	2.832	1.44–5.58

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

ns Not significant, *DDD* dual-chamber pacemaker, *VVI* single-chamber pacemaker, *VVI-ARVLL* single-chamber pacemaker with apical right ventricular lead location, *DDD-ARVLL* dual-chamber pacemaker with apical right ventricular lead location, *n-ARVLL* non-apical right ventricular lead location, *SSS* sick sinus syndrome, *A-V* atrial-ventricular block (degree II or III), *FA* atrial fibrillation, *CAD* coronary artery disease, *HA* arterial hypertension, *DM-2* type 2 diabetes, *LVEDV* left ventricular end-diastolic volume, *LVEF* left ventricular ejection fraction, *ACE-I* angiotensin-converting enzyme inhibitor, *ARB* angiotensin receptor blocker, *TD* thiazide derivative diuretic, *TLD* loop diuretic, *ASS* acetylsalicylic acid, *AVK* oral anti-coagulant, *AA* aldosterone antagonist, *anti-DM* antidiabetic

(left ventricular end-diastolic volume, LVEF).

Finally, data on the implantation procedure were also collected, i.e., the pacing mode (VVI/DDD), and the RVLT location. The decision concerning the location of the RVLT was left up to the operator.

Statistical analysis

Constant variables with normal distribution are shown as means with standard deviation; the median value as well as lower and upper quartiles are presented. Qualitative parameters are presented as number of cases and percentages.

The comparative analysis of constant variables was conducted using the Student *t* test, whereas dichotomous variables were compared using the chi-square test with Yates's correction.

A Cox regression model was used to define risk (hazard ratio) of death. Initial evaluation of the significance of potential prognostic factors was performed using the univariable Cox regression model. Factors with a prognostic value of $p < 0.1$ were included in the multivariable analysis. In the uni- and multivariable analyses, the risk of failure is presented as a hazard ratio (HR) with a 95% confidence interval (CI). The probability of event-free survival during the observation period for the selected variables was analyzed using the Kaplan–Meier method; statistically significant differences in the curves were evaluated using a log-rank test, including complete and censored data.

Data on the mortality rate are presented as raw data. Analysis using age as a layer (age-adjustment) was also conducted, but the results did not differ from the raw data. Statistical significance was set at $p < 0.05$. All statistical analyses were conducted with the STATISTICA 10.0 program.

The patients were divided into two groups for analysis: those who survived the 4-year observation period and those who died during that time; the patients were further divided into female and male subgroups. Additionally, a comparison of the distribution of the selected vari-

Herz 2018 · 43:315–324 <https://doi.org/10.1007/s00059-017-4561-6>
© The Author(s) 2017

K. Krzemień-Wolska · A. Tomasiak · E. Nowalany-Kozielska · W. Jacheć

Prognosis of patients with implanted pacemakers in 4-year follow-up. Impact of right ventricular pacing site

Abstract

Background. Pacing remains the method of choice for treatment of cardiac electrical conduction disorders.

This study examined the interrelationship between the site of the right ventricular lead tip and patient prognosis in association with other cardiovascular risk factors over a 4-year follow-up period.

Patients and methods. The study comprised 450 consecutive patients (223 women; aged 69.16 ± 9.63 years) who had their first SSI or DDD pacemaker implanted for typical indications.

Results. During follow-up, 91 (20.2%) patients died. The positive prognostic factors were: female sex (hazard ratio [HR] = 0.426), DDD pacemaker (HR = 0.526), oral anticoagulant use (HR = 0.330; all groups), sodium concentration (HR = 0.926), oral anticoagulant (HR = 0.115) and statin (HR = 0.260) use

(female group), and non-apical location of the right ventricular lead tip (HR = 0.549; male group). Risk factors for death were: age (HR = 1.063), diabetes requiring insulin (HR = 2.832), creatinine concentration (HR = 1.005; all groups), age (HR = 1.11; female group), and elevated creatinine level (HR = 1.012; male group). In all patients, the non-apical location of the right ventricular lead tip was associated with an 18.92% reduced mortality rate during the 4-year follow-up, which was statistically significant for the male group. **Conclusion.** The non-apical location of the right ventricular lead tip was a positive prognostic factor and was statistically significant in the male subgroup.

Keywords

Arrhythmias · Right ventricle · Mortality · Heart conduction system · Cardiac pacing, artificial

Prognose von Patienten mit implantiertem Schrittmacher im 4-Jahres-Verlauf. Einfluss des rechtsventrikulären Stimulationsorts

Zusammenfassung

Hintergrund. Ein Schrittmacher bleibt die Methode der Wahl für die Behandlung kardialer elektrischer Erregungsleitungsstörungen. In der vorliegenden Studie wurde die Beziehung zwischen der Lage der rechtsventrikulären Elektrodenspitze und der Prognose des Patienten in Zusammenschau mit anderen kardiovaskulären Risikofaktoren im Verlauf einer 4-jährigen Nachbeobachtungsphase untersucht.

Patienten und Methoden. An der Studie nahmen 450 konsekutive Patienten teil (davon 223 Frauen; Alter: $69,16 \pm 9,63$ Jahre), bei denen erstmals ein SSI- oder DDD-Schrittmacher aus typischer Indikation implantiert wurde.

Ergebnisse. Während der Nachbeobachtungsphase verstarben 91 (20,2%) Patienten. Die positiven prognostischen Faktoren bestanden aus: weiblichem Geschlecht (Hazard Ratio, HR: 0,426), DDD-Schrittmacher (HR = 0,526), orale Antikoagulanziengabe (HR = 0,330; alle Gruppen), Natriumkonzentration (HR = 0,926), Gabe von oralen Antikoagulanzen (HR = 0,115) und Statinen

(HR = 0,260; Gruppe der Frauen) und nicht-apikaler Lokalisierung der rechtsventrikulären Elektrodenspitze (HR = 0,549; Gruppe der Männer). Risikofaktoren für Tod waren: Alter (HR = 1,063), insulinpflichtiger Diabetes (HR = 2,832), Kreatininkonzentration (HR = 1,005; alle Gruppen), Alter (HR = 1,11; Gruppe der Frauen) sowie erhöhter Kreatininspiegel (HR = 1,012; Gruppe der Männer). Bei allen Patienten war die nichtapikale Lage der rechtsventrikulären Elektrodenspitze mit einer 18,92% verminderten Mortalitätsrate während der 4-jährigen Nachbeobachtungsphase assoziiert, was für die Gruppe der Männer statistisch signifikant war.

Schlussfolgerung. Die nichtapikale Lage der Elektrodenspitze im rechten Ventrikel stellte einen positiven prognostischen Faktor dar und erwies sich in der männlichen Studienkohorte als statistisch signifikant.

Schlüsselwörter

Arrhythmien · Rechter Ventrikel · Mortalität · Kardiales Erregungsleitungssystem · Herzschrittmacherstimulation

Table 2 Baseline female patient data and survival analysis (uni- and multivariable Cox regression)

Female	Survival	Death	Univariable Cox regression analysis			Multivariable Cox regression analysis		
			<i>p</i>	HR	95% CI	<i>p</i>	HR	95% CI
No. of patients	193 (86.55%)	30 (13.45%)						
Mean age at implantation	69.36 ± 9.58	75.23 ^{***} ± 4.47	0.001	1.144	1.05–1.24	0.007	1.116	1.03–1.21
DDD	138 (71.50%)	15 [*] (50.00%)	0.018	0.421	0.21–0.86	0.252	0.637	0.29–1.38
VVI	55 (28.50%)	15 [*] (50.00%)	–	–	–	–	–	–
VVI-ARVLL	40 (20.73%)	11 ^{ns} (36.67%)	–	–	–	–	–	–
DDD-ARVLL	99 (51.30%)	11 ^{ns} (36.67%)	–	–	–	–	–	–
n-ARVLL	54 (28.0%)	8 ^{ns} (26.7%)	0.938	0.968	0.43–2.18	–	–	–
SSS	104 (53.89%)	14 ^{ns} (46.67%)	0.458	0.762	0.37–1.56	–	–	–
A-V block	61 (31.61%)	13 ^{ns} (43.33%)	0.202	1.600	0.78–3.29	–	–	–
FA with bradycardia	28 (14.51%)	3 ^{ns} (10.00%)	0.511	0.671	0.20–2.21	–	–	–
CAD	48 (31.4%)	8 ^{ns} (26.7%)	0.886	1.061	0.47–2.38	–	–	–
HA	96 (49.74%)	12 ^{ns} (40.00%)	0.290	0.674	0.32–1.40	–	–	–
DM-2	36 (18.65%)	10 ^{ns} (33.33%)	0.062	2.063	0.97–4.41	0.070	2.380	0.93–6.07
Stroke	11 (5.70%)	3 ^{ns} (10.00%)	0.377	1.712	0.52–5.65	–	–	–
Hemoglobin (g/dl)	12.82 ± 1.44	12.27 [*] ± 1.71	0.045	0.811	0.66–0.99	0.255	0.867	0.68–1.11
Creatinine (μmol/l)	78.54 ± 25.06	108.49 [*] ± 85.24	0.001	1.007	1.00–1.01	0.208	1.003	1.00–1.01
Cholesterol (mg%)	192.38 ± 47.87	179.0 ^{ns} ± 46.63	0.174	0.994	0.98–1.00	–	–	–
Sodium (mmol/l)	139.37 ± 4.11	137.39 ^{ns} ± 7.39	0.022	0.917	0.85–0.99	0.047	0.926	0.86–1.00
LVEDV (ml)	79.65 ± 21.71	84.85 ^{ns} ± 34.04	0.358	1.010	0.99–1.03	–	–	–
LVEF (%)	50.21 ± 7.04	48.79 ^{ns} ± 7.74	0.426	0.970	0.90–1.04	–	–	–
BB	110 (56.99%)	15 ^{ns} (50.00%)	0.411	0.740	0.36–1.52	–	–	–
ACE-I	88 (45.60%)	9 ^{ns} (30.00%)	0.106	0.524	0.24–1.15	–	–	–
ARB	20 (10.36%)	1 ^{ns} (3.33%)	0.259	0.315	0.04–2.34	–	–	–
Statins	82 (42.49%)	4 ^{**} (13.33%)	0.005	0.220	0.08–0.63	0.019	0.260	0.08–0.80
TLD	43 (22.28%)	2 ^{ns} (6.67%)	0.067	0.260	0.06–1.10	0.126	0.318	0.07–1.38
TD	34 (17.62%)	9 ^{ns} (30.00%)	0.118	1.866	0.85–4.08	–	–	–
ASS	84 (43.52%)	9 ^{ns} (30.00%)	0.155	0.567	0.26–1.24	–	–	–
AVK	38 (19.69%)	1 ^{ns} (3.33%)	0.063	0.148	0.02–1.11	0.036	0.115	0.02–0.87
Digoxin	20 (10.36%)	6 ^{ns} (20.00%)	0.127	2.009	0.82–4.92	–	–	–
AA	32 (16.58%)	5 ^{ns} (16.67%)	0.998	0.999	0.38–2.61	–	–	–
Oral anti-DM drugs	16 (8.29%)	2 ^{ns} (6.67%)	0.803	0.833	0.20–3.50	–	–	–
Insulin therapy	8 (4.15%)	5 [*] (16.67%)	0.005	3.966	1.52–10.38	0.053	3.277	0.99–10.90

p < 0.05, ***p* < 0.01, ****p* < 0.001

ns Nonsignificant, *DDD* dual-chamber pacemaker, *VVI* single-chamber pacemaker, *VVI-ARVLL* single-chamber pacemaker with apical right ventricular lead location, *DDD-ARVLL* dual-chamber pacemaker with apical right ventricular lead location, *n-ARVLL* non-apical right ventricular lead location, *SSS* sick sinus syndrome, *A-V* atrial-ventricular block (degree II or III), *FA* atrial fibrillation, *CAD* coronary artery disease, *HA* arterial hypertension, *DM-2* type 2 diabetes, *LVEDV* left ventricular end-diastolic volume, *LVEF* left ventricular ejection fraction, *ACE-I* angiotensin-converting enzyme inhibitor, *ARB* angiotensin receptor blocker, *TD* thiazide derivative diuretic, *TLD* loop diuretic, *ASS* acetylsalicylic acid, *AVK* oral anti-coagulant, *AA* aldosterone antagonist, *anti-DM* antidiabetic.

ables was made between groups on the basis of the RVLT location.

Our study was a retrospective analysis, not a medical experiment, and therefore did not require the approval of the ethics committee.

Results

Among 621 patients who had a pacemaker implanted for the first time in the period 2006–2008, 450 met the inclusion criteria (223 women) at the age of 69.16 ± 9.63 years. During the observation pe-

riod, 91 patients died (20.2%), including 30 women.

Patients who survived the 4-year follow-up, compared with those who died during the follow-up period, more often had dual-chamber pacemakers implanted, were younger in terms of

Table 3 Baseline male patient data and survival analysis (uni- and multivariable Cox regression)

Male	Survival	Death	Univariable Cox regression analysis			Multivariable Cox regression analysis		
			p	HR	95% CI	p	HR	95% CI
No. of patients	166 (73.13%)	61 (26.87%)						
Mean age at implantation	67.05 ± 10.48	71.29 ^{**} ± 7.31	0.005	1.051	1.02–1.09	0.160	1.023	0.99–1.06
DDD	118 (71.08%)	31 ^{**} (50.82%)	0.002	0.459	0.28–0.76	0.080	0.620	0.36–1.06
VVI	48 (28.92%)	30 ^{**} (49.18%)	–	–	–	–	–	–
VVI-ARVLL	26 (15.66%)	23 ^{***} (37.70%)	–	–	–	–	–	–
DDD-ARVLL	67 (40.36%)	19 ^{ns} (31.15%)	–	–	–	–	–	–
n-ARVLL	73 (46.4%)	19 ^{ns} (31.1%)	0.051	0.582	0.34–1.00	0.042	0.555	0.31–0.98
SSS	57 (34.34%)	22 ^{ns} (36.07%)	0.815	1.064	0.63–1.80	–	–	–
A-V block	87 (52.41%)	32 [†] (52.46%)	0.972	1.009	0.61–1.67	–	–	–
FA with bradycardia	22 (13.25%)	7 ^{ns} (11.48%)	0.703	0.858	0.39–1.89	–	–	–
CAD	67 (40.3%)	18 ^{ns} (18.3%)	0.119	0.645	0.37–1.12	–	–	–
HA	83 (50.00%)	22 ^{ns} (36.07%)	0.064	0.610	0.36–1.03	0.352	0.745	0.40–1.38
DM-2	38 (22.89%)	14 ^{ns} (22.95%)	0.951	1.019	0.56–1.85	–	–	–
Stroke	6 (3.61%)	4 ^{ns} (6.56%)	0.395	1.553	0.56–4.28	–	–	–
Hemoglobin (g/dl)	13.88 ± 1.42	13.59 ^{ns} ± 2.08	0.180	0.894	0.76–1.05	–	–	–
Creatinine (μmol/l)	89.90 ± 20.94	111.5 ^{***} ± 39.17	0.000	1.018	1.01–1.02	0.001	1.012	1.01–1.02
Cholesterol (mg%)	179.4 ± 46.59	181.5 ^{ns} ± 43.78	0.837	1.001	0.99–1.01	–	–	–
Sodium (mmol/l)	138.6 ± 3.71	138.8 ^{ns} ± 4.54	0.852	1.006	0.94–1.08	–	–	–
LVEDV (ml)	95.39 ± 28.45	96.12 ^{ns} ± 38.06	0.358	1.010	0.99–1.03	–	–	–
LVEF (%)	48.69 ± 8.52	49.72 ^{ns} ± 6.95	0.426	0.970	0.90–1.04	–	–	–
BB	103 (62.05%)	25 ^{**} (40.98%)	0.003	0.463	0.28–0.77	0.147	0.639	0.35–1.17
ACE-I	101 (60.84%)	29 ^{ns} (47.54%)	0.058	0.615	0.37–1.02	0.775	0.911	0.48–1.73
ARB	6 (3.61%)	2 ^{ns} (3.28%)	0.823	0.851	0.21–3.48	–	–	–
Statins	73 (43.98%)	15 [†] (24.59%)	0.007	0.450	0.25–0.81	0.611	0.836	0.42–1.67
TLD	26 (15.66%)	9 ^{ns} (14.75%)	0.717	0.877	0.43–1.78	–	–	–
TD	21 (12.65%)	12 ^{ns} (19.67%)	0.142	1.605	0.85–3.02	–	–	–
ASS	93 (56.02%)	24 [†] (39.34%)	0.018	0.539	0.32–0.90	0.425	0.781	0.43–1.43
AVK	19 (11.45%)	5 ^{ns} (8.20%)	0.402	0.676	0.27–1.69	–	–	–
Digoxin	9 (5.42%)	6 ^{ns} (9.84%)	0.200	1.736	0.75–4.03	–	–	–
AA	28 (16.87%)	9 ^{ns} (14.75%)	0.718	0.878	0.43–1.78	–	–	–
Oral anti-DM drugs	26 (15.66%)	8 ^{ns} (13.11%)	0.588	0.814	0.39–1.71	–	–	–
Insulin therapy	7 (4.22%)	7 ^{ns} (11.48%)	0.017	2.609	1.19–5.74	0.124	2.024	0.82–4.97

[†] $p < 0.05$, ^{**} $p < 0.01$, ^{***} $p < 0.001$

ns nonsignificant, *DDD* dual-chamber pacemaker, *VVI* single-chamber pacemaker, *VVI-ARVLL* single-chamber pacemaker with apical right ventricular lead location, *DDD-ARVLL* dual-chamber pacemaker with apical right ventricular lead location, *n-ARVLL* non-apical right ventricular lead location, *SSS* sick sinus syndrome, *A-V* atrial-ventricular block (degree II or III), *FA* atrial fibrillation, *CAD* coronary artery disease, *HA* arterial hypertension, *DM-2* type 2 diabetes, *LVEDV* left ventricular end-diastolic volume, *LVEF* left ventricular ejection fraction, *ACE-I* angiotensin-converting enzyme inhibitor, *ARB* angiotensin receptor blocker, *TD* thiazide derivative diuretic, *TLD* loop diuretic, *ASS* acetylsalicylic acid, *AVK* oral anti-coagulant, *AA* aldosterone antagonist, *anti-DM* antidiabetic

statistical significance, and more often had arterial hypertension. In this group, more patients used statins, ASS, and AVK, while fewer received insulin therapy and loop diuretics. In a subgroup of patients who died, higher creatinine concentrations were observed. No differences in non-apical right ventricular lead location (n-ARVLL) were observed

in the whole study group, whereas in the subgroup of patients with VVI pacemakers, the apical lead location prevailed.

As also seen in the main study group, women who survived when compared to women who died during the 4-year follow-up were younger initially, more often had a DDD pacemaker implanted, more often received statins, and less

often required insulin therapy. In the group of women who died during the follow-up period, higher concentrations of creatinine and lower concentrations of hemoglobin were observed as compared with those who survived the observation period.

In the male group, patients who survived the 4-year observation period were

Table 4 Study group characteristics according to sex and RV lead location

	F n = 223			M n = 227			F vs. M
	ARVLL n = 161	n-ARVLL n = 62		ARVLL n = 135	n-ARVLL n = 92		p < 0.01
Mean age at implantation	70.03 ± 9.32	70.47 ± 9.23	ns	68.94 ± 10.14	67.10 ± 9.48	ns	p < 0.01
DDD	110 (68.3%)	43 (69.4%)	ns	86 (63.7%)	63 (68.5%)	ns	ns
SSS	76 (47.2%)	42 (67.7%)	p < 0.05	42 (31.1%)	37 (40.2%)	ns	p < 0.001
A-V block	62 (38.5%)	12 (19.4%)	ns	79 (58.5%)	40 (43.5%)	p < 0.05	p < 0.001
FA with bradycardia	23 (14.3%)	8 (12.9%)	ns	14 (10.4)	15 (16.3%)	ns	ns
CAD	42 (26.1%)	14 (22.6%)	ns	51 (37.8%)	34 (37.0%)	ns	p < 0.01
HA	78 (48.4%)	30 (48.4%)	ns	66 (48.9%)	39 (42.4%)	ns	ns
DM-2	31 (19.3%)	15 (24.2%)	ns	33 (24.4%)	19 (20.7%)	ns	ns
Stroke	9 (5.56%)	5 (7.94%)	ns	3 (2.22%)	7 (7.61%)	p < 0.05	ns

p < 0.05, **p < 0.01, ***p < 0.001

ARVLL apical right ventricular lead location, n-ARVLL non-apical right ventricular lead location, ns nonsignificant, DDD dual-chamber pacemaker, SSS sick sinus syndrome, A-V block atrioventricular block (degree II or III), FA atrial fibrillation, CAD coronary artery disease, HA arterial hypertension, DM-2 type 2 diabetes, F Female, M Male

younger initially, had lower creatinine concentrations, and more often used beta-blockers, acetylsalicylic acid, and statins. They also more often had an n-ARVLL.

Detailed characteristics of the study group and both the female and male subgroups, along with survival rates and the results of uni- and multivariable analysis in Cox hazard regression, are presented in [Tables 1, 2, and 3](#).

n-ARVLL was more frequent in women with sick sinus syndrome. The female subgroups selected on the basis of lead location did not differ significantly in the type of implanted stimulator, frequency of atrioventricular block, atrial fibrillation with low ventricular action, coronary artery disease, arterial hypertension, type 2 diabetes, and brain stroke in their medical history.

In the male subgroup, n-ARVLL was significantly dominant in the group of patients with atrioventricular block and in patients with a history of brain stroke. No statistically significant differences were observed in the remaining parameters.

Women were older than men, they suffered from coronary disease less frequently, and significantly more often had the RV lead in the apical location. Women had a pacemaker implanted more often because of sick sinus syndrome and less often because of atrioventricular conduction disorders ([Table 4](#)).

Univariable Cox regression

In the entire group of patients, univariable Cox hazard analysis indicated that the following factors were of positive prognostic significance: female sex (HR = 0.463); DDD pacemaker implantation (HR = 0.441); concurrent arterial hypertension (HR = 0.623); BB application (HR = 0.550); statins (HR = 0.371); ASS (HR = 0.601); ACE-I (HR = 0.660); thiazide-derivative diuretic: (HR = 0.583); and AVK (HR = 0.391).

The increased risk of death in the entire group potentially concerned the patients of more advanced age (HR = 1.063), with higher creatinine concentrations (HR = 1.008), requiring loop diuretics (HR = 1.571), and requiring insulin therapy (HR = 3.030).

In the female subgroup, the following factors had potentially protective significance: DDD pacemaker implantation (HR = 0.421); thiazide-derivative diuretics (HR = 0.260); statins (HR = 0.220); and AVK (HR = 0.148) as well as higher hemoglobin concentrations (HR = 0.811) and lower sodium concentrations (HR = 0.917). A potential indicator of worse prognosis was age (HR = 1.144), creatinine concentration (HR = 1.007), and diabetes (HR = 2.063) especially diabetes requiring insulin therapy (HR = 3.966).

In the male subgroup, the following factors were of potentially advantageous importance: DDD pacemaker implan-

tation (HR = 0.459); n-ARVLL (HR = 0.582); arterial hypertension (HR = 0.610); BB application (HR = 0.463); ACE-I (HR = 0.615); statins (HR = 0.450); and ASS (HR = 0.539). Age was also a potential indicator of worse prognosis (HR = 1.051), as was creatinine concentration (HR = 1.018) and diabetes requiring insulin therapy (HR = 2.609).

Multivariable Cox regression

In the entire patient group, multivariable Cox regression showed that the following factors were of positive prognostic significance: female sex (HR = 0.426); DDD pacemaker implantation (HR = 0.526); and the application of oral anti-coagulants (HR = 0.330). Age (HR = 1.047), insulin therapy (HR = 2.832), and higher creatinine concentration (HR = 1.005) had a statistically significant influence on the risk of early death in the entire patient group.

Sodium concentration had positive prognostic significance in the female subgroup (HR = 0.926), as did oral anticoagulant (HR = 0.115) and statin use (HR = 0.260). An adverse prognostic factor was age (HR = 1.116).

In the male subgroup, a protective influence of n-ARVLL was noted (HR = 0.555), with a worse prognosis seen in patients with elevated creatinine levels (HR = 1.012).

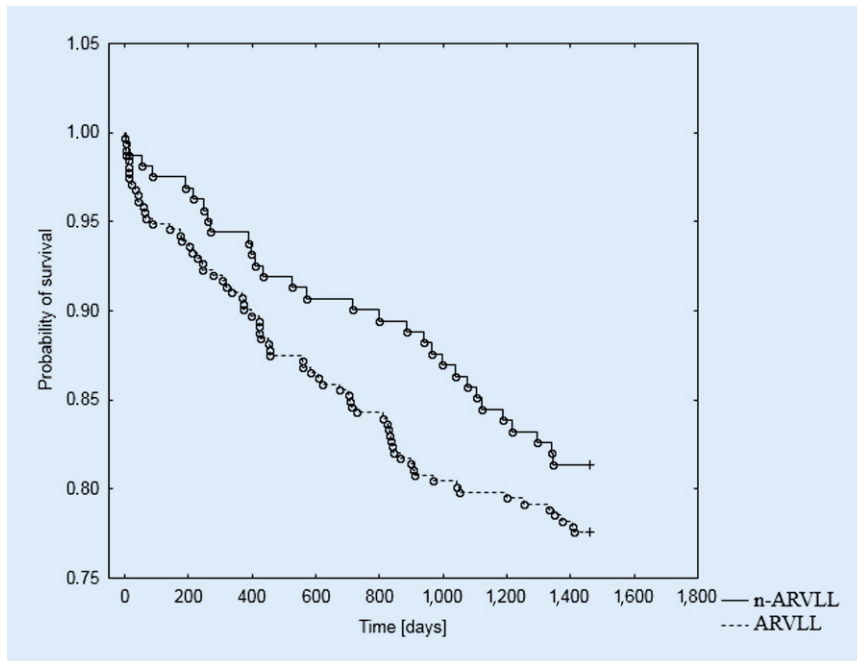


Fig. 1 ▲ Probability of survival in the study group depending on the location of the right ventricular lead; log-rank $p = 0.287$. ARVLL apical right ventricular lead location, n-ARVLL non-apical right ventricular lead location

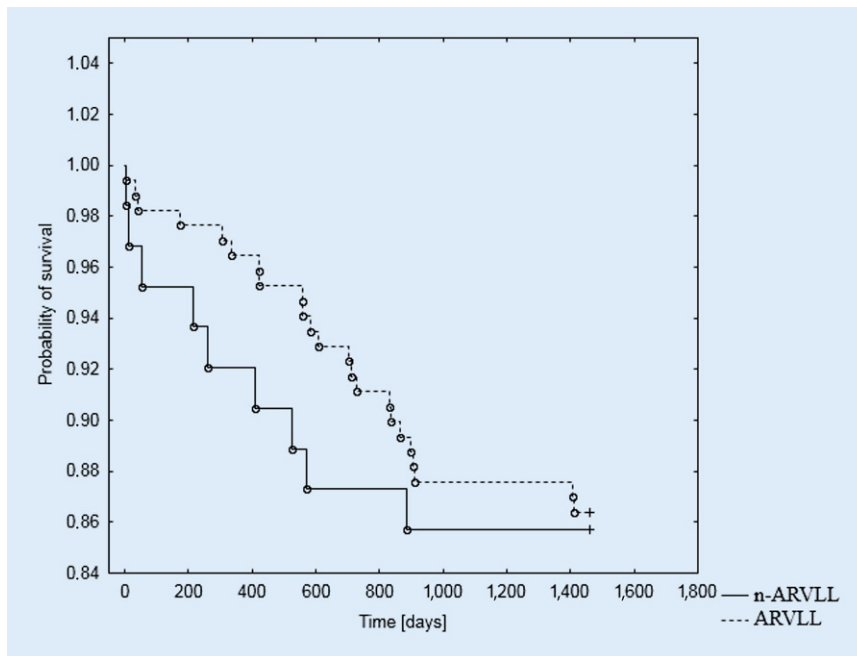


Fig. 2 ▲ Probability of survival in the female subgroup depending on the location of the right ventricular lead; log-rank $p = 0.823$. ARVLL apical right ventricular lead location, n-ARVLL non-apical right ventricular lead location

Kaplan–Meier survival curves and log-rank analysis

In the entire patient group, despite the distribution of the survival curves, log-rank analysis did not indicate a statisti-

cally significant influence of RVLT location on survival in the 4-year observation period ($p = 0.287$).

The log-rank analysis indicated a statistically significant influence of the RVLT location in the distribution of the curves,

showing the survival time during the 4-year observation in the male subgroup ($p = 0.028$). However, no prognostic significance was found for RVLT location in the female subgroup ($p = 0.823$). The Kaplan–Meier survival curves for the female and male groups according to RVLT location are presented in **Fig. 1, 2 and 3**.

Discussion

In this case-control study, we found that non-apical pacing has a positive influence on the survival rate, with statistically significant differences seen in the male subgroup.

The number of studies was smaller in comparison with large clinical studies examining the influence of electrotherapy on the cardiovascular system [29, 29–31]. The patients analyzed here were younger than the population evaluated in other studies [28, 29]. However, the indications for pacemaker implantation and the observation period were similar to those in other studies [29, 31, 32]. In the present study group, a significant percentage of patients had dual-chamber pacemaker implantation (67.11%), which differentiates it from previous papers, especially those with a longer observation period [28, 30].

The mortality rate during the 4-year observation period was similar to the rate reported for the study by Udo et al., in which the survival rate after 3 and 5 years was 81% and 69%, respectively [31], and lower than the one reported by Shiomoto et al., where 67.2% of patients survived in the 4-year observation period [32]. The population evaluated by Pyatt et al. was almost two times larger and approximately 8 years older than the one in our study; the men constituted 51.9% of the research group, there were more patients with atrioventricular conduction disorders, and thus pacing in DDD mode was dominant. Fewer patients suffered from ischemic heart disease, diabetes, or past neurological incidents. Older age at the time of pacemaker implantation, cardiomyopathy in medical history, valve defects, VVI pacing, as well as male sex were among the factors with negative prognostic influence [29]. In the study by Udo et al., the indicators of total mor-

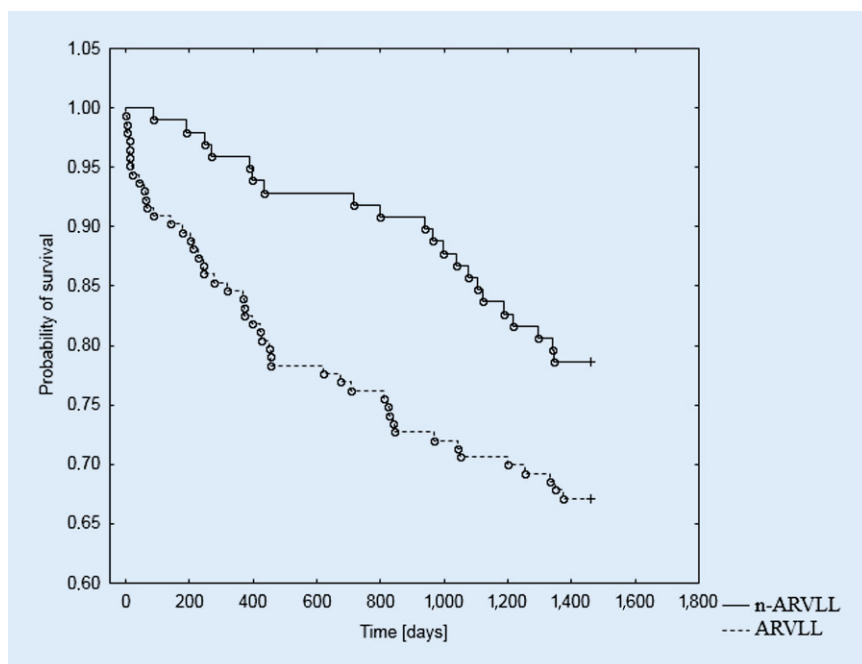


Fig. 3 ▲ Probability of survival in the male subgroup depending on the location of the right ventricular lead; log-rank $p = 0.028$. ARVLL apical right ventricular lead location, n-ARVLL non-apical right ventricular lead location

tality rate were older age, male sex, BMI, concurrent ischemic heart disease, circulatory insufficiency, and atrioventricular conduction disorders [31].

In the analysis conducted by Brunner et al., concerning the survival of 6505 patients during an 8-year observation period, the negative influence of the following parameters on the survival rate was reported: older age at the time of device implantation, male sex, earlier period of pacemaker implantation, pacing in VVI mode, concurrent atrial fibrillation, fainting or total lack of fainting accompanying bradycardia, and lack of pre-MAS symptoms prior to stimulator implantation [28].

As in other studies, in our entire patient group, female sex (despite older age among women) [28, 29, 31], dual-chamber pacing, and anti-coagulant AVK therapy [33, 34] had prognostic significance, whereas the indicators of poor prognosis were higher creatinine concentration [35], loop diuretic application, and diabetes requiring insulin therapy [36]. Although the univariable analysis indicated a positive prognostic influence of BB, ACE-I, and statin application, this was not reflected in the multivariable model. However, in the female group, the appli-

cation of statins and AVK improved the prognosis, and the following factors had negative prognostic significance: age and diabetes requiring insulin therapy as well as lower sodium concentration. In the male subgroup, the non-apical location of the RV lead had a positive influence on survival, whereas high creatinine concentration was a negative factor.

In contrast to other studies, we did not find any influence of coronary artery disease, arterial hypertension, and atrial fibrillation on the long-term prognosis of our study group [28, 31]. This can be explained by the fact that individuals with stable cardiovascular system disorders, receiving optimal pharmacotherapy, were included in the research. In addition, the patients included in the study had left ventricular contraction function.

Studies comparing single- and dual-chamber pacing indicate that physiological pacing is associated with an improved life quality and physical efficiency as well as with a lower frequency of atrial fibrillation episodes [16, 18, 28, 37], brain strokes, and heart insufficiency [12, 16].

The positive aspects of this type of pacing can be hampered by the unnecessarily high frequency of pacing [16, 17] or ventricular contraction desynchroniza-

tion connected with the apical localization of the ventricular lead. The partially different research results [16, 18, 28, 38] on the prognosis can be due to the varied percentages of ventricular pacing, which, along with its increase, led both to an increase in hospitalization risk due to heart failure [16, 17] and in a lower survival rate [17].

In the present study group, almost 70% of patients had a dual-chamber pacemaker implanted and the percentage of patients with the implanted device working in VVI mode was low along with a maintained sinus rhythm. Owing to the retrospective nature of the study, we do not have data available on the percentage of ventricular pacing; however, the significant majority of implanted dual-chamber pacing devices had an active self-organizing atrioventricular search algorithm, which is especially important in patients with sick sinus syndrome [16, 18, 39–41].

The multivariable analysis conducted in the entire patient group did not show a significant prognostic significance for RVLT location. Its non-apical location was the only parameter with positive prognostic significance in the male subgroup. The literature on the prognostic importance of the RV pacing site is not extensive and does not provide consistent results. In our opinion, the positive prognostic effect of non-apical pacing in the male subgroup was associated with sex, which can be the result of the more frequent occurrence of atrioventricular conduction disorders requiring a higher percentage of pacing as compared with the female subgroup in which sick sinus syndrome was dominant.

Dąbrowska Kugacka et al., conducting a study of 122 patients aged 69 ± 11 years, with typical indications for pacemaker implantation randomized to subgroups with apical vs. non-apical RV pacing, did not report a statistically significant reduction in general mortality rate as well as death due to cardiovascular reasons in patients without RVA vs. RVA pacing. In the multivariable analysis, male sex and older age at the time of pacemaker implantation were among factors contributing to a worse prognosis of general mortality rate [42]. Similarly, in the

study conducted by Kypty et al. on 98 patients with atrioventricular block including 53 patients with n-ARVLL, a total of nine patients died in the course of an 18-month observation period, among whom were five in the RVNA subgroup, reaching a statically nonsignificant difference in the survival analysis.

In turn, a positive prognostic effect of RVOT (right ventricle outflow tract) pacing was reported in the study by Vanerio et al. The authors stated that in a group of 150 patients with implanted pacemakers, 18 patients with RVOT pacing and 49 patients with apical pacing died during a middle- and long-term observation period, obtaining a statistically significant result in the log-rank analysis ($p = 0.02$) [43].

Despite the restrictions, the results we have obtained are complementary to current research and confirm the positive influence of non-apical pacing on long-term prognosis. In order to make the aforementioned observations objective, it is necessary to conduct further randomized prospective studies in this field.

Study limitations

This study's limitations include the retrospective nature of the research and the lack of randomization as well as data on the percentage of RV pacing. The heterogeneity of the evaluated population in terms of primary diseases is a further limitation.

Conclusion

The non-apical location of the RVLV has a positive influence on patient prognosis, with statistical significance found for the male subgroup in this study. However, these observations require verification in further randomized studies.

Corresponding address

W. Jacheć, PhD

Second Department of Cardiology, Medical University of Silesia
ul. Skłodowskiej 10, 41-800 Zabrze, Poland
wjachec@interia.pl

Funding. The study was supported by the Medical University of Silesia grant no. KNW-1-057/N/6/K.

Open access funding provided by Śląski Uniwersytet Medyczny.

Compliance with ethical guidelines

Conflict of interest. K. Krzemień-Wolsk, A. Tomasiak, E. Nowalany-Kozielska, and W. Jacheć declare that they have no competing interests.

This article does not contain any studies with human participants or animals performed by any of the authors.

Open Access This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made.

References

1. Cooley DA (2000) In memoriam: tribute to Ake Senning, pioneering cardiovascular surgeon. *Texas Heart Inst J* 27:234
2. Nelson GD (1993) A brief history of cardiac pacing. *Tex Heart Inst J* 20:12
3. Larsson B, Elmqvist H, Rydén L, Schüller H (2003) Lessons from the first patient with an implanted pacemaker: 1958–2001. *Pacing Clin Electrophysiol* 26(1 Pt 1):114–124
4. Elmqvist R (1978) Review of early pacemaker development. *Pacing Clin Electrophysiol* 1(4):535–536
5. Furman S (2003) The early history of cardiac pacing. *Pacing Clin Electrophysiol* 26(10):2023–2032
6. Koch E (1920) Der Kontraktionsablauf an der Kammer des Froschherzens und die Form der entsprechenden Suspensionscurve, mit besonderen Ausführungen über das Alles-oder-nichts-Gesetz, die Extrasystole und den Herzalternans. *Pflügers Arch Ges Physiol* 181:106–114
7. Wiggers CJ (1925) The muscular reactions of the mammalian ventricles to artificial surface stimuli. *Am J Physiol* 73C:275–282
8. Kutarski A (2005) Nastęstwa stymulacji wierzchołka prawej komory – czas na sformułowanie praktycznych wniosków? *Folia Cardiol* 12(9):613–626
9. Gułaj M, Słodowski T, Kutarski A (2007) Dwupunktowa stymulacja prawej komory. Opcja awaryjna w stymulacji resynchronizującej? Korzystniejsza opcja dla pacjentów z granicznymi wskazaniami do stymulacji resynchronizującej? Metoda mniej szkodliwa stymulacji komorowej. *Folia Cardiol* 2(7):276–284
10. Badke RF, Boinay P, Covell JW (1980) Effects of ventricular pacing on regional left ventricular performance in the dog. *Am J Physiol* 238:H-858–H-867
11. Grines CL, Bashore TM, Boudoulas H, Olson S, Shafer P, Wooley CF et al (1989) Functional abnormalities in isolated left bundle block. *Circulation* 79:845–853
12. Prinzen FW, Peschar M (2002) Relation between the pacing induced sequence of activation and left ventricular pump function in Animals. *Pacing Clin Electrophysiol* 25:484–498
13. Prinzen FW, Augustijn CH, Arts T, Alessie MA, Reneman RS et al (1990) Redistribution of myocardial fiber strain and blood flow by asynchronous activation. *Am J Physiol* 259:H300–H308
14. Delhaas T, Arts T, Prinzen FW, Reneman RS (1994) Regional fibre stress-fibre strain area as estimate of regional oxygen demand in the canine heart. *J Physiol* 477:481–496
15. Tse HF, Lau CP (1997) Long-term effect of right ventricular pacing on myocardial perfusion and function. *J Am Coll Cardiol* 29:744–749
16. MODe Selection Trial (MOST) Investigators, Michael O, Sweeney MD, Hellkamp AS et al (2003) Adverse effect of ventricular pacing on heart failure and atrial fibrillation among patients with normal baseline QRS duration in clinical trial of pacemaker therapy for sinus node dysfunction. *Circulation* 107:2932–2937
17. Wilkoff BL, Cook JR, Epstein AE (2002) Dual chamber and VVI Implantable defibrillator trial investigators. Dual-chamber pacing or ventricular back-up pacing in patients with an implantable defibrillator: the Dual Chamber and VVI Implantable Defibrillator (DAVID) trial. *JAMA* 288:3115–3123
18. Connolly SJ, Kerr CR, Gent M et al (2000) Effects of physiologic pacing versus ventricular pacing on the risk of stroke and death due to cardiovascular causes. Canadian Trial of Physiologic Pacing Investigators. *N Engl J Med* 342:1385–1391
19. Andersen HR, Nielsen JC, Thomsen PE et al (1997) Long term follow-up of patients from a randomized trial of atrial versus ventricular pacing for sick sinus syndrome. *Lancet* 350:1210–1216
20. Karpawich PP, Justice CD, Chang CH, Gause CY, Kuhns LR et al (1991) Septal ventricular pacing in the immature canine heart: a new perspective. *Am Heart J* 121:827–833
21. De Cock CC, Meyer A, Kamp O, Visser CA (1992) Hemodynamic benefits of right ventricular outflow tract pacing. *Eur J CPPE* 2(suppl. 1A):126
22. Guidici MC, Thornburg GA, Buck AL et al (1994) Permanent right ventricular outflow tract pacing improves cardiac output – comparison with apical placement in 58 patients. *Eur J CPPE* 4(suppl. 4):332
23. Tse HF, Yu C, Wong KK et al (2002) Functional abnormalities in patients with permanent right ventricular pacing: the effect of sites of electrical stimulation. *J Am Coll Cardiol* 40:1451–1458
24. Katsujji I, Hideki O et al (2010) Right ventricular pacing from the septum avoids the acute exacerbation in left ventricular dyssynchrony and torsional behavior seen with pacing from the apex. *J Am Soc Echocardiogr* 23(2):195–200
25. Wen-Hao L (2010) Right Ventricular outflow tract pacing causes intraventricular dyssynchrony in patients with sick sinus syndrome: a real-time three-dimensional echocardiography study. *J Am Soc Echocardiogr* 23(6):599–607
26. Stambler B, Ellenbogen K, Zhang X (2003) Right ventricular outflow versus apical pacing in pacemaker patients with congestive heart failure and atrial fibrillation. *J Cardiovasc Electrophysiol* 14:1180–1186
27. Avi Shimony, Eisenberg MJ, Filion KB, Guy A (2012) Beneficial effects of right ventricular non-apical vs. apical pacing: a systematic review and meta-analysis of randomized-controlled trials. *Europace* 14:81–91
28. Brunner M, Olschewskib M, Geibela A, Bodea C et al (2004) Long-term survival after pacemaker implantation. Prognostic importance of gender and baseline patient characteristics. *Eur Heart J* 25:88–95

29. Pyatt JR, Somauroo JD, Jackson M, Grayson AD et al (2002) Long-term survival after permanent pacemaker implantation: analysis of predictors for increased mortality. *Europace* 4:113–119
30. Jahangir A, Shen WK, Neubauer SA, Ballard DJ et al (1999) Relation between mode of pacing and long-term survival in the very elderly. *J Am Coll Cardiol* 33(5):1208–1216
31. Udo EO, van Hemel NM, Zuithoff NP, Doevendans PA et al (2013) Prognosis of the bradycardia pacemaker recipient assessed at first implantation: a nationwide cohort study. *Heart* 99(21):1573–1578
32. Amikam S, Lemer J, Roguinn N, Peleg H (1976) Long-term survival of elderly patients after pacemaker implantation. *Am Heart J* 91:445
33. ESC Guidelines for the management of atrial fibrillation (2010) *Kardiologia Polska* tom 68
34. Hart RG, Pearce LA, Aguilar MI (2007) Meta-analysis: antithrombotic therapy to prevent stroke in patients who have nonvalvular atrial fibrillation. *Ann Intern Med* 146:857–867
35. Brinker J (2005) Assessing cardiovascular risk in patients with chronic kidney disease. *Adv Stud Med* 5(7A):S710–S714
36. Smooke S et al (2005) Insulin-treated diabetes is associated with a marked increase in mortality in patients with advanced heart failure. *Am Heart J* 149:168
37. Lamas GA, Lee KL, Sweeney MO, Silverman R et al (2002) Mode selection trial in sinus-node dysfunction. ventricular pacing or dual-chamber pacing for sinus-node dysfunction. *N Engl J Med* 346(24):1854–1862
38. Lamas GA, Orav EJ, Stambler BS et al (1998) Quality of life and clinical outcomes in elderly patients treated with ventricular pacing as compared with dual-chamber pacing. *N Engl J Med* 338:1097–1104
39. Nielsen JC, Kristensen L, Andersen HR et al (2003) A randomized comparison of atrial and dual-chamber pacing in 177 consecutive patients with sick sinus syndrome: echocardiographic and clinical outcome. *ACC Curr J Rev* 42(4):614–623
40. Linde C, Nordlander R, Rosenqvist M (1994) Atrial rate adaptive pacing: what happens to AV conduction? *Pacing Clin Electrophysiol* 17(10):1581–1589
41. Dąbrowska-Kugacka A, Lewicka-Nowak E, Tybura S, Wilczek R et al (2009) Survival analysis in patients with preserved left ventricular function and standard indications for permanent cardiac pacing randomized to right ventricular apical or septal outflow tract pacing. *Circ J* 73:1812–1819
42. Kypta A, Steinwender C, Kammler J et al (2008) Long-term outcomes in patients with atrioventricular block undergoing septal ventricular lead implantation compared with standard apical pacing. *Europace* 10:574–579
43. Vanerio GJLV, Fernández Banizi P, Banina Aguerre D et al (2008) Medium- and long-term survival after pacemaker implant: Improved survival with right ventricular outflow tract pacing. *J Interv Card Electrophysiol* 21(3):195–201

Modellprojekt zur Fernbehandlung startet

Ab März wird die ambulante Versorgung in Baden-Württemberg um eine Facette reicher. In zwei Regionen startet ein Projekt zur Fernbehandlung. Bei „Doc Direkt“ bekommen GKV-Patienten binnen 30 Minuten qualifizierten ärztlichen Rat.

Der Landkreis Tuttlingen im Schwäbischen wird zusammen mit Stuttgart die Modellregion bilden, in der Vertragsärzte bundesweit erstmals – ohne vorherigen Kontakt – telemedizinisch beraten und behandeln. Das Projekt der Kassenärztlichen Vereinigung Baden-Württemberg soll im März starten. Ziel des „Doc Direkt“ genannten Projekts ist letztlich die Patientensteuerung. Die KV will im kleinen Maßstab in Erfahrung bringen, ob ein ärztlich besetztes Callcenter Versicherte davon abhalten kann, das Krankenhaus aufzusuchen. Hintergrund ist das viel diskutierte Phänomen, dass Patienten teilweise auch mit Befindlichkeitsstörungen die Notaufnahme aufsuchen. Die KVBW kooperiert dazu mit der Münchener TeleClinic GmbH.

Ersteinschätzung durch MFA

Im KV-Projekt können GKV-Versicherte in den beiden Modellregionen ab März bei Gesundheitsbeschwerden ein Callcenter anrufen, und zwar werktags zwischen 9 und 19 Uhr. Dort erfolgt durch eine speziell geschulte MFA eine Ersteinschätzung: Im Falle eines Notfalls wird der Anrufer sofort an die Rettungsleitstelle weitergeleitet. Anderenfalls wird der Versicherte binnen 30 Minuten von einem Arzt zurückgerufen. 89 Vertragsärzte haben sich bisher beworben, als Telearzt zu arbeiten. Ergibt sich aus dem Gespräch, dass die Beschwerden nicht abschließend geklärt werden können, wird dem Patient am gleichen Tag ein Termin in einer Vertragsarztpraxis in der Nähe vermittelt.

Bereits 72 PEP-Praxen

Diese teilnehmenden PEP-Praxen (Patientennah erreichbare Portalpraxen) sind über eine webbasierte telemedizinische Plattform mit dem Callcenter verbunden. Sie müssen also keine zusätzliche Software auf den Praxisrechnern installieren. 72 Praxen haben bisher ihr Interesse bekundet, als PEP-Praxis zu agieren. Davon stammen 51 aus Stuttgart und sieben aus dem Landkreis Tuttlingen. 14 weitere Praxen sind in ganz Baden-Württemberg verteilt. 25 Euro Honorar erhält der

Telearzt im Projekt „Doc Direkt“ für jeden Anruf – extrabudgetär.

Der Ausschluss eines Arztes soll insbesondere bei „wiederholter Nichterreichbarkeit“ während der abgestimmten Ansprechzeiten sowie bei „begründeten Patientenbeschwerden“ möglich sein. Wichtig zudem: Die Berufshaftpflicht der teilnehmenden Ärzte muss auch die spezifischen Risiken telemedizinischer Beratung und Behandlung abdecken und eine Deckungssumme von mindestens drei Millionen Euro aufweisen. Vorgesehen ist, dass die PEP-Praxis für jeden über „Doc Direkt“ vermittelten Patienten einen extrabudgetären Fallwertzuschlag in Höhe von 20 Euro erhält. Ihre Leistungen kann die Praxis zudem außerhalb des RLV abrechnen. Die Fälle müssen durch Verwendung einer Pseudo-GOP gekennzeichnet werden. Der Telearzt, der das Erstgespräch führt, soll jeden Anruf mit 25 Euro extrabudgetär honoriert bekommen. Das auf zwei Jahre angelegte Projekt enthält eine Verlängerungsoption – doch dürfte sich relativ rasch abzeichnen, welche Bedeutung die Fernbehandlung in der vertragsärztlichen Versorgung spielen kann. Ausdrücklich heißt es, die Einbindung weiterer Regionen in das Projekt „ist vorgesehen“.

**Quelle: Ärzte Zeitung
(www.aerztezeitung.de)**