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ASSOCIATION OF LATE TRICUSPID REGURGITATION PROGRESSION AND INTRACARDIAC
LEAD TYPES IN PATIENTS AFTER PRIMARY IMPLANTATION
OF CARDIAC ELECTRONIC DEVICES

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Aim. To evaluate the factors and degree of progression of tricuspid regurgitation (TR) depending on the type of implanted electrode in patients after primary implantation of a cardiac implantable electronic device (CIED) in the late postoperative period.

Methods. Case histories of 674 patients who underwent primary implantation of an implantable cardioverter-defibrillator (ICD) for the secondary prevention of sudden cardiac death for the period from 2009 to 2019 were analyzed and 75 case histories were selected. Using similar criteria, a comparison group was selected, who were implanted with a permanent pacemaker (PM) for sinus node dysfunction. For maximum comparability of the main and reference groups, pseudo-randomization was used using the nearest neighbor search method; 68 pairs were formed, comparable in terms of the main factors. Multiple logistic regression was used to analyze the predictors of progression of TR in the postoperative period.

Results. In the preoperative period in the ICD group, 34 patients (50%) had a mild degree of TR, moderate and severe TR occurred in an equal number of cases - 2 patients (2.9%) each. In the PM group, mild TR was detected in 24 cases (35.3%), moderate - in 2.9% (n=2) of cases, respectively. In the postoperative period, predominantly mild TR was determined in both groups (in the ICD group in 67.6% (n=46), in the PM group - in 48 patients (70.6%)).

In the analysis of the general group of patients with implanted CIEDs (n=136), in the late postoperative period, an increase in the volume of both atria and the degree of TR was revealed. Left ventricular ejection fraction and end-diastolic volume didn't have significant dynamics. All echocardiographic data obtained in the postoperative period were within the normal range.

According to the results of multiple logistic regression, a history of coronary heart disease (CHD) has the greatest predictive power (hazard ratio 4.170; 95% confidence interval 1.751-9.933, p=0.001).

Conclusion. TR in patients after primary implantation of a CIED in the late postoperative period progresses slightly, regardless of the type of right ventricular electrode. A long history of CHD is associated with the greatest risk of progression to tricuspid valve insufficiency.

Key words: tricuspid regurgitation; endocardial right ventricle electrode; right ventricle; permanent pacemaker; implantable cardioverter defibrillator; defibrillating electrode

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Implantable permanent cardioverter defibrillators (ICD) and pacemakers (PM) are used in modern practice for the treatment of cardiac conduction disorders and life-threatening arrhythmias. Since the first use of PM for permanent pacing in 1959, technology in this area has developed rapidly, resulting in a significant increase in the number of implantable devices [1]. Their widespread use is partly due to the increase in the life expectancy of patients, the increase in the number of cardiac surgical interventions and the expansion of the capabilities of the devices themselves. It is quite logical to state that implantation of an endocardial right ven-

tricular lead can contribute to complications. A number of recent studies have shown that "transtricuspid" lead can cause damage to valvular or subvalvular structures, as well as indirectly lead to electrical and mechanical dyssynchrony of the heart chambers [2, 3]. At the same time, defibrillating lead, as the cause of the development and progression of tricuspid valve (TV) insufficiency, has always stood apart due to its design features, which are based on a larger electrode diameter, less flexibility and the presence of shock coils of the right ventricle (RV) / superior vena cava. This can cause greater interference, adhesion and, as a result, fibrosis of valvular

structures, which can eventually lead to early development and progression of insufficiency [4].

The phenomenon of lead-related tricuspid insufficiency is gaining recognition, but the frequency and clinical significance of this problem have not been reliably established. In addition, the significance of the type of implantable device has not been sufficiently studied.

The aim of the study was to evaluate the factors and degree of progression of tricuspid regurgitation (TR) depending on the type of implanted lead in patients after primary implantation of a cardiovascular implantable electronic device (CIED) in the late postoperative period.

METHODS

We analyzed data from 674 patients who underwent primary ICD implantation for the purpose of secondary prevention of sudden cardiac death for the period from 2009 to 2019. Appropriate approval from the local ethics committee was obtained for this retrospective study. Patients were included in the study if the following conditions were met:

- age 18 and over;
- postoperative follow-up ≥ 3 months;
- availability of two-dimensional echocardiography results in the pre- and late postoperative period;
- primary implantation of ICD/PM;
- implantation of the right ventricular lead only in the inter-ventricular septum;
- left ventricular ejection fraction (LVEF) $\geq 40\%$; systolic excursion of the TV ring (TAPSE, tricuspid annular plane systolic excursion) ≥ 15 mm;
- no history of severe valvular stenosis and open heart interventions, as well as endocardial lead extraction;
- absence of severe pulmonary hypertension (systolic pressure in the pulmonary artery ≤ 50 mm Hg);
- valvular regurgitation, not exceeding moderate values according to preoperative echocardiography;
- lack of indications for cardiac resynchronization therapy (CRT);
- compliance of patients to adequate drug therapy.

As a result, 75 patients were selected. Using similar criteria, a comparison group was selected from the data of 239 patients who underwent PM implantation for sinus node dysfunction. For maximum comparability of the main and reference groups according to the available confounders, pseudo-randomization was used with the search for the “nearest neighbor”. As a result of group alignment, 68 comparable pairs were formed. The main clinical and demographic characteristics of the patient groups are shown in Table 1.

All interventions were carried out according to the ex-

isting recommendations [5-7], according to the standard technique [8, 9]. The following electronic devices were used as implantable PMs: Sensia VR/DR “Medtronic”, Effecta VR/DR “BIOTRONIK SE & Co. KG”, Verity ADx XL DR/SR and Sustain XL DR “St. Jude Medical” with endocardial right ventricular leads with silicone and silicone-polyurethane coatings and active fixation Capturefix® Novus 5076-58cm (Medtronic) and Safio S 60 (BIOTRONIK SE & Co. KG) with a diameter of 2.0 mm (6 Fr), Flexend 2 (Guidant Corporation) 2.4 mm (7.2 Fr), Tendril ST (St. Jude Medical) 2 mm (6 Fr). The following devices were implanted in the ICD group: Maximo DR, Maximo II VR/DR, Protecta VR/DR, Evera S DR, Protecta XT DR “Medtronic”; Lexos VR, Lumax 540 VR, Iforia 3 DR-T, Iforia 5 DR-T “BIOTRONIK SE & Co. KG”; Vitality VR/DR “Guidant Corporation”. The following endocardial defibrillating leads were used: Sprint quattro secure with polyurethane-coated silicone insulation 2.87 mm (8.6 Fr), Sprint quattro secure S 2.73 mm (8.2 Fr) Medtronic; Linx S 65 with silicone coating and 2.6 mm diameter (7.8 Fr) “BIOTRONIK SE & Co. K.G.”

In the pre- and postoperative periods, all patients received optimal drug therapy for the underlying and concomitant diseases, in accordance with modern clinical guidelines. Also in the postoperative period, a routine assessment of the work of the PM and ICD was carried out.

Echocardiographic studies were carried out on the basis of generally accepted modern recommendations [10, 11] using ultrasound diagnostic systems (General Electric) Vivid 9, Vivid 7 Dimension, Vivid 7 Pro with sensors with variable frequency from 1.5/3 to 2.3/4 .6 MHz - for trans-thoracic studies. The progression of TR was considered the appearance or increase in the degree of insufficiency by 1 or more steps.

Statistical analysis of the study results was performed using IBM® SPSS® Statistics version 26 (SPSS, Chicago, IL, USA). Pseudorandomization was used to equalize scores across groups. Logistic regression was

Table 1.

Clinical and demographic characteristics of patient groups

	ICD (n=68)	PM (n=68)	p
Male sex, n (%)	48 (70.6)	46 (67.6)	0.853
Age, years	64.5 [56.0; 70.0]	67.5 [63.0; 70.0]	0.143
BMI, kg/m ²	29.6 \pm 4.8	29.7 \pm 5.4	0.876
Hypertension, n (%)	59 (86.7)	64 (94.1)	0.243
Diabetes mellitus, n (%)	10 (14.7)	10 (14.7)	1.000
CHD, n (%)	22 (32.4)	32 (47.1)	0.114
TIA/CVA, n (%)	4 (5.9)	4 (5.9)	1.000
COPD, n (%)	4 (5.9)	10 (14.7)	0.156
Paroxysmal/persistent AF, n (%)	37 (54.4)	43 (63.2)	0.296
Duration of observation (months)	55.0 \pm 30.2	51.3 \pm 28.4	0.543
V pacing percentage, %	1 [0; 5.75]	2 [0.25; 26.8]	0.091
Mode VVIR / DDDR, n (%)	22 / 46 (32.4 / 67.6)	20 / 48 (29.4 / 70.6)	0.711

Note: BMI - body mass index; CHD - coronary heart disease; TIA - transient ischemic attack; CVA - cerebrovascular accident; COPD - chronic obstructive pulmonary disease; AF - atrial fibrillation.

Table 2.

Comparison of echocardiography parameters in the preoperative and late postoperative periods

	ICD (n=68)			PM (n=68)			ICD + PM (n=136)			P ₁	P ₂
	Preoperative period	Postoperative period	p	Preoperative period	Postoperative period	p	Preoperative period	Postoperative period	p		
LVEF, %	56.8±9.2	55.4±7.8	0.167	59.8±6.7	58.1±8.2	0.090	58.3±8.2	56.7±8.1	0.028	0.030	0.054
LVED volume (ml)	138.1±37.6	131.7±36.3	0.063	129.3±30.1	122.5±34.1	0.011	133.7±34.2	127.1±35.4	0.002	0.135	0.129
MR (degree)	1 [0; 1]	1 [0; 1]	0.748	0 [0; 1]	0 [0; 1]	0.375	0 [0; 1]	1 [0; 1]	0.433	0.013	0.009
LA volume (ml)	75.1±27.9	69.5 [56.0; 100.0]	0.035	76.3±24.7	83.5±27.9	0.008	75.7±26.3	81.3±29.8	0.001	0.765	0.409
TR (degree)	1 [0; 1]	1 [1; 1]	0.010	0 [0; 1]	1 [1; 1]	0.000	0 [0; 1]	1 [1; 1]	0.000	0.115	0.749
Tricuspid annular diameter (mm)	32.4±4.9	33.2±4.2	0.152	32.9±4.7	31.9±5.0	0.021	32.7±4.8	32.6±4.5	0.801	0.547	0.113
RV basal dimension (mm)	26.6±4.6	26.8±4.3	0.687	27.2±3.8	27.1±3.4	0.721	26.9±4.2	26.9±3.9	0.928	0.355	0.629
RA volume (ml)	45.5 [40.0; 64.0]	53.5 [42.0; 72.0]	0.000	52.5 [43.0; 66.0]	54.5 [42.0; 77.0]	0.124	56.2±21.2	61.3±25.8	0.001	0.439	0.905
Tricuspid regurgitation gradient (mmHg)	14.5 [0; 23.0]	20.0 [12.0; 26.0]	0.000	0 [0; 24.0]	21 [12.0; 25.0]	0.000	0 [0; 23.3]	20 [12.0; 25.0]	0.000	0.298	0.698
TAPSE (mm)	19.5±2.6	19.4±2.3	0.716	20.1±1.9	21.1±2.7	0.002	19.8±2.3	20.3±2.6	0.026	0.101	0.000

Note: LVEF - left ventricular ejection fraction; LVED - left ventricular end-diastolic; TR - tricuspid regurgitation, RV - right ventricular, LA - left atrium, RA - right atrium, MR - mitral regurgitation, TAPSE - tricuspid annular plane systolic excursion.

used with the selection of pairs of observations comparable to each other with the closest values of the correspondence index (propensity score, PS). The pairs of observations were aligned according to 12 factors: gender, age, body mass index (BMI), duration of follow-up, median cumulative percentage of right ventricular pacing, type of CIED, history of hypertension, diabetes, coronary heart disease (CHD), transient ischemic attack (and/or acute cerebral circulation), chronic obstructive pulmonary disease, paroxysmal/persistent atrial fibrillation (AF). The distribution of parameters was checked using the Shapiro-Wilk test. In the case of a normal distribution, the arithmetic mean with indication of the standard deviation ($M \pm SD$) was used; in other cases, the median and interquartile range were indicated - the 25th and 75th percentiles. Frequencies and proportions (in %) were used to describe qualitative data. Data from populations with a normal distribution were compared using Student's t-test for independent samples. Comparison of data from populations with a non-normal distribution was carried out using the Mann-Whitney U-test and the χ^2 test (in some cases, the Fisher test was used). For dependent samples, the Wilcoxon test was used. The critical level of statistical significance when testing statistical hypotheses was taken as 0,05.

Multiple logistic regression was used to analyze predictors of the onset/progression of TR in the postoperative period. The recorded fact of TR progression in the long-term period was determined as a dependent variable; independent variables included: age, duration of follow-up, type of device (ICD/PM), BMI, history of paroxysmal/persistent AF, CHD, hypertension.

RESULTS

In the preoperative period, LVEF and the degree of mitral regurgitation (MR) had differences, however, they were within the normal range or slightly exceeded the normal values, as well as other indicators (Table 2). In the ICD group 36 (52.9%) patients had mild TR and 2 (2.9%) patients had moderate valvular insufficiency; in the PM group only 28 (41.2%) patients had an insignificant TR (Fig. 1 a,b).

In the postoperative period, differences in echocardiographic parameters had a similar picture, and the values themselves were also within or slightly exceeded the indexed indicators of age norms (Table 2). In both groups 52 (76.5%) patients were diagnosed with a minor degree of TR (Fig. 1 c, d).

Evaluation of the postoperative dynamics of echocardiography parameters within the studied groups of patients showed a slight progression of TR, regurgitation gradient and volumes of both atria, while in the PM group there was a regres-

sion of parameters of the end-diastolic volume of the LV and the size of the annulus fibrosus TV (Table 2).

In the late postoperative period, there was an increase in the volumes of both atria, an increase in the degree and gradient of TR, and the indicators of LV EF and end-diastolic volume of the left ventricle had a slight reverse trend (Table 2), when analyzing the generalized population of patients with implanted electronic devices ($n=136$). There were no cases of TR increase in the postoperative period by 2 or more steps. All echocardiographic data obtained in the postoperative period were within the limits of generally accepted standards or slightly exceeded them.

A relationship was found between such independent predictors as age, BMI, a history of CHD, and progression of TV deficiency in the long-term period after implantation of an electronic device, according to the results of multiple logistic regression (Table 3).

DISCUSSION

The problem of tricuspid insufficiency in a patient with CIED is not new. Classically, valvular regurgitation is usually divided into functional and structural. If the former is associated with acquired pathology of the heart and is accompanied by a change in its geometry, then the latter is associated with congenital malformations and iatrogenic mechanical damage to the valvular apparatus [12]. Theoretically, in a patient with CIED, one of these mechanisms may predominate, or a combination of both may occur. Secondary (functional) TR was neglected for a long time, considered a minor problem, a consequence of other pathologies of the heart (mitral valve disease, pulmonary hypertension, or AF). Regarding TV, it is not unreasonable that the term "forgotten valve" is found in a number of foreign works [13]. This is primarily due to the significant clinical needs in the treatment of TR [14].

The exact reason for the progression of TV deficiency in patients with CIED is not known, most likely it is the result of a physical impact of the electrode on the valve, less often perforation or rupture of the valve leaflets and/

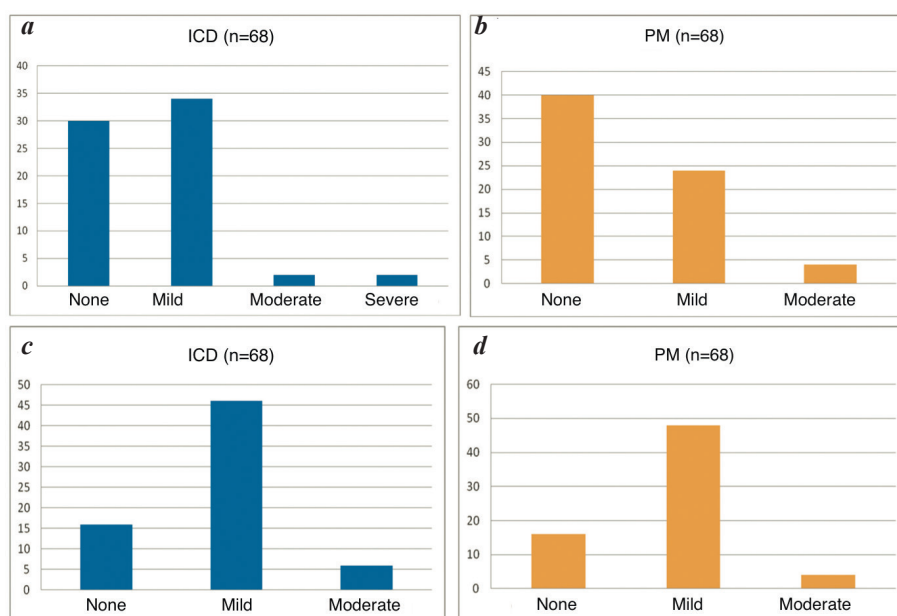


Fig. 1. Prevalence of TR in the preoperative period (a, b). Prevalence of TR in the postoperative period (c, d).

or the formation of fibrous tissue, which subsequently leads to “adhesion” with lead [3]. This postulate is reflected in the study by G.Lin et al. (2005), which showed that in all patients (n=41) with severe TR, who underwent reconstructive surgery of the tricuspid valve, the fact of damage to the valvular structures by the electrode was recorded [15]. Taking into account the selection criteria of our study, as well as the results of postoperative echocardiography, which did not show additional structures and formations in the area of the right ventricular lead, it can be assumed that there is no significant mechanical damage to TV. However, more detailed information about the anatomy and interaction of valve and electrode structures can only be obtained using 3D echocardiography, which was not performed in our study.

If we take into account the fact of the presence of a foreign body (electrode) in the right ventricle and impaired valve sealing function, then several prospective studies have not revealed the development of significantly progressive valvular incompetence in the early (first 3-5 days) and mid-term (6-12 months) periods [8, 9]. In the present work, the analysis of echocardiographic results of the combined group of patients (n=136) showed a slight increase in the volumes of both atria, an increase in the degree and regurgitation gradient of TR in the late postoperative period.

The fact that right ventricular pacing affects the functioning of the left chambers of the heart, due to asynchronous electromechanical activation of the left ventricle, is a generally recognized cause of MR. How this mechanism relates to the development of TR remains a controversial issue. It is suggested that RV remodeling, dilatation of the fibrous ring of TV and, as a result, the appearance of functional TR are the outcome of either systolic dyssynchrony in the case of apical stimulation, or a progressive decrease in systolic and diastolic LV function. This hypothesis is supported by a retrospective study by M. Sadreddini et al. (2014) of patients who underwent primary implantation of PM and CRT. At the same time, it was found that the degree of TR significantly increases after implantation of a two-chamber PM but does not progress against the background of biventricular stimulation, which suggests the “suppression” of the pathophysiological mechanisms of the development of valvular insufficiency in the CRT group [12]. On the contrary, analysis of the PROTECT-PACE study (145 patients, 76 of them with apical stimulation)

showed that after 2 years of follow-up, the degree of TR increased, but the location of stimulation in the RV was not associated with changes in echocardiography parameters of the right heart chambers [16]. Stimulation of the RV septum is also far from physiological, but leads to the appearance of a narrower QRS complex on the ECG and may be characterized by a less negative effect on echocardiographic and hemodynamic parameters of both the left and right ventricles.

Anatomically, the leaflets of TV are held by chordae in three separate regions of the RV. It is reasonable to assume that any change in the timing of the reduction of these areas may change the degree of valve closure, which will eventually lead to the emergence or exacerbation of a pre-existing TR. This view is supported by a small prospective study by M. Vaturi et al. (2009), which included only patients who were independent of the pacemaker. As a result, there was a progression of the degree of TR in the mode of active pacing, but no changes in contractility RV were detected [17]. Along with this, there are also studies indicating that the percentage of right ventricular stimulation does not correlate with the progression of the degree of TR and only the physical presence of the electrode plays the main, if not the only role in TV dysfunction [18]. It should be noted that in one of the prospective studies that assessed the “acute” effect of active right ventricular pacing on the degree of TR and RV contractility, no significant changes in echocardiographic parameters were found [19]. According to the results of this study, in both cohorts of patients, the cumulative percentage of right ventricular pacing was insignificant, so it is impossible to objectively judge the effect of right ventricular pacing on RV or TV function.

If we approach the problem of lead-related TR in a differentiated way, then it is logical to separate patients with ICD from patients with PM, due to the design features of defibrillating lead. Larger and stiffer defibrillator leads are more likely to cause injury or disruption of the coaptation of the TV leaflets. This postulate is reflected in the work of J.B. Kim et al. (2008) [20]. An analysis of large studies has shown that ICD implantation, even with a minimal percentage of RV pacing, is associated with an increased risk of hospitalization for congestive heart failure and death compared with the control group [21, 22]. In other words, in the ICD group, TV failure may be the result of progression of pre-existing significant cardiac pathology.

We were able to exclude patients with severe valvular stenosis and a significantly reduced LV ejection fraction from the study. At the same time, both groups did not differ significantly from each other in the pre- and postoperative period. There were no cases of increase in valvular insufficiency of TV in the postoperative period by 2 or more degrees. Evaluation of postoperative parameters within both groups showed a similar slight increase in the degree of TR.

F.N.Delling et al. (2016), using a multivariate regression analysis of 1245 patients, found a number of variables that led to the progression of lead-related TR: increased age, decreased BMI, increased heart

Predictors of progression of TR. Results of multiple logistic regression (n=136)

	B	Значимость	Exp (B)	95% ДИ
Age	0,064	0,037	1,066	1,004-1,132
BMI	-0,098	0,020	0,906	0,834-0,985
Hypertension	-1,181	0,253	0,307	0,040-2,328
CHD	1,428	0,001	4,170	1,751-9,933
AF	-0,041	0,922	0,959	0,417-2,209
Type of lead	0,580	0,163	1,786	0,790-4,034
DO (months)	-0,001	0,926	0,999	0,983-1,016

Note: BMI - body mass index; CHD - coronary heart disease; AF - atrial fibrillation; DO - duration of observation.

Table 3.

rate, history of mitral valve repair or replacement, severe MR, increased pulmonary systolic pressure arteries ≥ 37 mmHg and RV dilation. Somewhat similar results were obtained in our study. A relationship was found with such independent predictors as age, BMI, and a long history of CHD. At the same time, with each year of the patient's age, the chances of an increase in the degree of TR increased by 6.6%, CHD in history increased the probability of an increase in the dependent variable by 4 times, a decrease in BMI for every 1 kg/m² increased the chances of progression of TR by 10%. Relationships of the dependent variable with such factors as the period of observation, the type of device, and a history of paroxysmal/persistent AF were not found [23].

Study limitations

The limitations of our study include the standard disadvantages of a retrospective study. The selected group of

patients is limited to one center, which does not allow to avoid sampling bias. The intervals between echocardiographic studies were variable. Consequently, chronologically structural and functional changes in the chambers and structures of the heart could not be fully assessed, which certainly requires prospective follow-up.

CONCLUSION

Tricuspid regurgitation in patients after primary implantation of a cardiac electronic device in the late postoperative period progresses slightly, regardless of the type of implanted electrode.

A long history of coronary heart disease in patients with implanted electronic devices is associated with the highest risk of progression of tricuspid valve regurgitation.

REFERENCES

1. Hindricks G, Camm J, Merkely B, et al. The EHRA White Book 2017. The current status of cardiac electrophysiology in ESC member countries. Available from URL https://www.escardio.org/static_file/Escardio/Subspecialty/EHRA/Publications/Documents/2017/ehra-white-book-2017.pdf.
2. Makarova NV, Durmanov SS, Bazilev VV et al. Tricuspid regurgitation associated with right ventricular endocardial electrodes. *Journal of Arrhythmology*. 2016;85: 40-47. (In Russ.). <https://doi.org/10.15275/annaritmol.2017.1.3>.
3. Rasha Al-Bawardy, Krishnaswamy A, Rajeswaran J, et al. Tricuspid regurgitation and implantable devices. *Pacing Clin Electrophysiol*. 2015;38: 259-266. <https://doi.org/10.1111/pace.12530>.
4. Lee RC, Friedman SE, Kono AT, et al. Tricuspid Regurgitation Following Implantation of Endocardial Leads: Incidence and Predictors. *Pacing Clin Electrophysiol*. 2015;38: 1267-74. <https://doi.org/10.1111/pace.12701>.
5. Revishvili AS, Bojcov SA, Davtyan KV, et al. Clinical guidelines for the electrophysiologic studies, catheter ablation and the use of implantable antiarrhythmic devices. The new edition. 2017. Moscow; p. 17-42. (In Russ.) ISBN 978-5-9500922-0-6.
6. Shlyakhto EV, Arutyunova GP, Belenkova YN. National guidelines for risk assessment and prevention of sudden cardiac death (2nd edition). Medpraktika-M. 2018. p. 70-93. (In Russ.) ISBN 978-5-98803-XXX-X.
7. Guidelines on Cardiac Pacing and Cardiac Resynchronization Therapy (ESC) 2021. *European Heart Journal*. 2021;42: 3427-3520. <https://doi.org/10.1093/eurheartj/ehab364>.
8. Glumskov AB, Durmanov SS, Bazilev VV. et al. Is the right ventricular pacemaker lead an independent risk factor for the development of tricuspid regurgitation in the early postoperative period? One-center prospective study. *Annaly Aritmology*. 2017;14(1). (In Russ.). <https://doi.org/10.15275/annaritmol.2017.1.3>.
9. Glumskov AB, Durmanov SS, Bazilev VV et al. Permanent right ventricular lead and its effect on the function of the tricuspid valve. *Journal of Arrhythmology*. 2018;93: 17-23. (In Russ.). <https://doi.org/10.25760/VA-2018-93-17-23>.
10. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging*. 2015; 16(3): 233-70. <https://doi.org/10.1016/j.echo.2014.10.003>.
11. Lancellotti P, Moura L, Pierard LA, et al. European Association of Echocardiography recommendations for the assessment of valvular regurgitation. Part 2: mitral and tricuspid regurgitation (native valve disease). *European Journal of Echocardiography*. 2010;11: 307-332. <https://doi.org/10.1093/ejehocard/jeq031>.
12. Sadreddini M, Haroun MJ, Buikema L, et al. Tricuspid valve regurgitation following temporary or permanent endocardial lead insertion, and the impact of cardiac resynchronization therapy. *Open Cardiovasc Med J*. 2014;31: 113-20. <https://doi.org/10.2174/1874192401408010113>.
13. Elisa E, James DC, Peter JZ, et al. Tricuspid Valve Dysfunction Caused by Right Ventricular Leads. *Cardiac Electrophysiology Clinics*. 2018;10: 447. <https://doi.org/10.1016/j.ccep.2018.05.006>.
14. Lancellotti P, Fattouch K, Go YY. Secondary tricuspid regurgitation in patients with left ventricular systolic dysfunction: cause for concern or innocent bystander? *Eur Heart J*. 2018;39(39): 3593-3595. <https://doi.org/10.1093/eurheartj/ehy522>.
15. Lin G, Nishimura RA, Connolly HM, et al. Severe symptomatic tricuspid valve regurgitation due to permanent pacemaker or implantable cardioverter-defibrillator leads. *J Am Coll Cardiol*. 2005;45(10):1672-5. <https://doi.org/10.1016/j.jacc.2005.02.037>.
16. Saito M, Iannaccone A, Kaye G, et al. Effect of Right Ventricular Pacing on Right Ventricular Mechanics and Tricuspid Regurgitation in Patients with High-Grade Atrioventricular Block and Sinus Rhythm (from the Protection of Left Ventricular Function during Right Ventricular Pacing Study). *Am J Cardiol*. 2015;116: 1875-82. <https://doi.org/10.1016/j.amjcard.2015.09.041>.
17. Mordehay V, Jairo K, Yaron S, et al. Right ventricular pacing increases tricuspid regurgitation grade regardless of the mechanical interference to the valve by the electrode. *European Journal of Echocardiography*. 2009;11: 550-553. <https://doi.org/10.1093/ejehocard/jeq018>.
18. Fanari Z, Hammami S, Hammami MB, et al. The effects

- of right ventricular apical pacing with transvenous pacemaker and implantable cardioverter defibrillator on mitral and tricuspid regurgitation. *J Electrocardiol.* 2015;48: 791-7. <https://doi.org/10.1016/j.jelectrocard.2015.07.002>
19. Glumskov AB, Durmanov SS, Bazilev VV. The effect of active right ventricular pacing on tricuspid regurgitation grade and right ventricular contractility in patients with sick sinus syndrome regardless of the mechanical interference to the valve by the electrode. *Annals of Arrhythmology.* 2019;16(4): 217-225. (In Russ.). <https://doi.org/10.15275/annaritm.2019.4.4>.
20. Kim JB, Spevack DM, Tunick PA, et al. The effect of transvenous pacemaker and implantable cardioverter defibrillator lead placement on tricuspid valve function: an observational study. *J Am Soc Echocardiogr.* 2008;21(3): 284-7. <https://doi.org/10.1016/j.echo.2007.05.022>.
21. Goldenberg I, Moss AJ, Hall WJ, et al. Multicenter Automatic Defibrillator Implantation Trial (MADIT) II Investigators. Causes and consequences of heart failure after prophylactic implantation of a defibrillator in the multicenter automatic defibrillator implantation trial II. *Circulation.* 2006;113(24): 2810-7. <https://doi.org/10.1161/CIRCULATIONAHA.105.577262>.
22. Höke U, Auger D, Thijssen J, et al. Significant lead-induced tricuspid regurgitation is associated with poor prognosis at long-term follow-up. *Heart.* 2014;100(12): 960-8. <https://doi.org/10.1136/heartjnl-2013-304673>.
23. Delling FN, Hassan ZK, Piatkowski G, et al. Tricuspid Regurgitation and Mortality in Patients With Transvenous Permanent Pacemaker Leads. *Am J Cardiol.* 2016;117(6): 988-92. <https://doi.org/10.1016/j.amjcard.2015.12.038>.

