ORIGINAL ARTICLE

Predictors of atrial tachyarrhythmias in adults with congenital heart disease

Esther Cambronero-Cortinas[1], Pedro Moratalla-Haro[2], Ana E. González-García[3], Pablo Ávila[4], Alexandros Papachristidis[5], Montserrat Bret-Zurita[3], José Ruiz-Cantador[3], Natalia Rivero-Jiménez[6], Rafael Peinado[3], José M. Oliver-Ruiz[4]

1 Cardiology Department, University Clinical Hospital of Valladolid, Valladolid, Spain
2 University of Castilla-La Mancha, Albacete, Spain
3 La Paz University Hospital, Madrid, Spain
4 Gregorio Marañón General University Hospital, Gregorio Marañón Health Research Institute, Faculty of Medicine, Complutense University of Madrid, Madrid, Spain
5 King's College Hospital NHS Foundation Trust, London, United Kingdom
6 Ramón y Cajal University Hospital, Madrid, Spain

KEY WORDS
atrial tachyarrhythmia, congenital heart disease, pressure and volume overload, right atrial dilatation

ABSTRACT

BACKGROUND Atrial tachyarrhythmias (ATs) represent the major late complications of congenital heart diseases (CHDs) following surgery. Little is known about the association between echocardiographic parameters and AT.

AIMS This study aimed to investigate a potential correlation among clinical, echocardiographic, and electrocardiographic parameters and AT as well as to analyze outcomes in adults with CHD and AT.

METHODS A retrospective case-control study was performed in adults with CHD. We included 71 patients with AT and 71 control individuals matched by sex, age, and the type of CHD without AT, all from the same institute. Medical records, electrocardiograms, and echocardiograms were reviewed. Adverse cardiovascular events were recorded and defined as cardiovascular mortality, admission for heart failure, or stroke. The univariate and multivariate logistic regression analysis of possible risk factors and the Kaplan–Meier analysis of adverse cardiovascular events were performed.

RESULTS Subpulmonary ventricular systolic pressure ≥40 mm Hg (hazard ratio [HR], 6.8; 95% CI, 2.4–18; P <0.001), right atrial dilatation ≥21 cm^2 (HR, 3.1; 95% CI, 1.2–7.6; P = 0.01), and significant tricuspid regurgitation (HR, 4; 95% CI, 1.3–10; P = 0.02) were identified as the main risk factors for AT. Patients with AT had worse outcomes, more frequently developed adverse cardiovascular events (86% vs 14%; P <0.01), and exhibited a 58% event-free survival rate compared with 98% of the patients without AT after 8 years of follow-up (log rank, 6.6; P = 0.01).

CONCLUSIONS Among patients with CHD, the main risk factors for AT include right atrial dilatation, high subpulmonary ventricular systolic pressure, and significant tricuspid regurgitation. The presence of AT may increase the risk of adverse cardiac events.

Correspondence to:
Esther Cambronero-Cortinas, MD, MSc, Cardiology Department, University Clinical Hospital of Valladolid, Avenida de Ramón y Cajal, número 3, 47003 Valladolid, Spain, phone: +34 983420000, email: ecambronero@saludcastillayleon.es
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INTRODUCTION Adults with congenital heart disease (CHD) constitute a growing population of patients.[1] Atrial tachyarrhythmias (ATs) are the most common types of arrhythmia and complications in this population, with a mean prevalence of 25% depending on the type of CHD (from 4% in simple CHD and up to 50% in complex CHD).[2-5] They also most significantly contribute to morbidity and are related with an increased risk of sudden cardiac death.[3,6-8]

Atrial tachyarrhythmias have been described in a variety of pathologies such as tetralogy of Fallot (ToF),[9,10] atrial septal defect (ASD),[11] the Fontan procedure,[12] or transposition of great arteries repaired with the Mustard or Senning procedures.[13] The natural history of AT has been described in recent publications.[9,14,15] However, there are relatively scarce data on echocardiographic variables associated with AT in adults with CHD.

WHAT'S NEW?

The natural history of atrial tachyarrhythmias (ATs) has been described in a variety of congenital heart diseases. However, data on echocardiographic variables associated with AT in adults with congenital heart diseases are relatively scarce. Our large retrospective registry of this population demonstrated the usefulness of echocardiographic variables in the prediction of AT. We concluded that the presence of right atrial dilatation, elevated subpulmonary ventricular systolic pressure, and significant tricuspid regurgitation may predict AT. Moreover, the presence of AT increases the incidence of adverse cardiovascular events. These findings highlight the significance of close monitoring in these patients in order to identify and treat hemodynamic disturbances that may predispose to AT.

The aims of this study were as follows: 1) to investigate a potential correlation of clinical, echocardiographic, and electrographic parameters with AT; 2) to analyze long-term outcomes in adults with CHD and AT.

METHODS

Study population A registry of patients treated and followed up at the adult CHD unit in La Paz Hospital in Madrid was established in December 1989. The patients entered the registry on the date of their first visit. The complexity of CHD was evaluated using the Bethesda classification and regarded as simple, moderate, or severely complex.[15] A total of 3311 patients with complete demographic data were registered between January 1990 and December 2013.

Using that database, a retrospective case-control study was performed. A group of 152 patients was found to develop AT during the analyzed period. Among the 152 patients, 81 were not included in the study: 78 patients were excluded due to lack of recent echocardiographic examination (performed earlier than a year from the first episode of AT), incomplete echocardiographic data, or loss to follow-up, and 3 cases because of suboptimal ultrasound image quality. All echocardiographic data were re-evaluated for the purpose of this study and a good correlation with previous measurements was noted. Therefore, 71 patients with AT whose data were entered in the registry between January 2007 and December 2013 were included in this study. A total of 71 control patients were selected from the same database and matched by age (±5 years), sex, and the type of CHD. Controls had no AT documented at follow-up. Two controls with AT during the follow-up were replaced with patients without AT, and the 2 subjects with AT were added to the group of cases (FIGURE 1).

Atrial tachyarrhythmia was defined as a fast abnormal atrial rhythm in which the electrical impulse originates in atrial tissue other than that of the sinoatrial node and the atrial waves have a constant cardiac cycle length. An irregular atrial rhythm with clear beat-to-beat variation was classified as atrial fibrillation. No distinction was made between macroreentrant or focal tachycardias. Only adults (above 18 years old) with at least a single episode of documented sustained (>30 seconds) and/or symptomatic AT during the follow-up were included in the study. Early postoperative (within 30 days following the surgery) and asymptomatic nonsustained AT episodes during Holter monitoring or device interrogation were excluded from the analysis.

Atrial tachyarrhythmias were recorded by a 12-lead surface electrocardiogram, a rhythm strip, an ambulatory electrocardiogram, or an implantable cardiac device. Site investigators (JMO-R, AEG-G, and RP) reviewed source data to determine the origin and type of arrhythmia.[16]

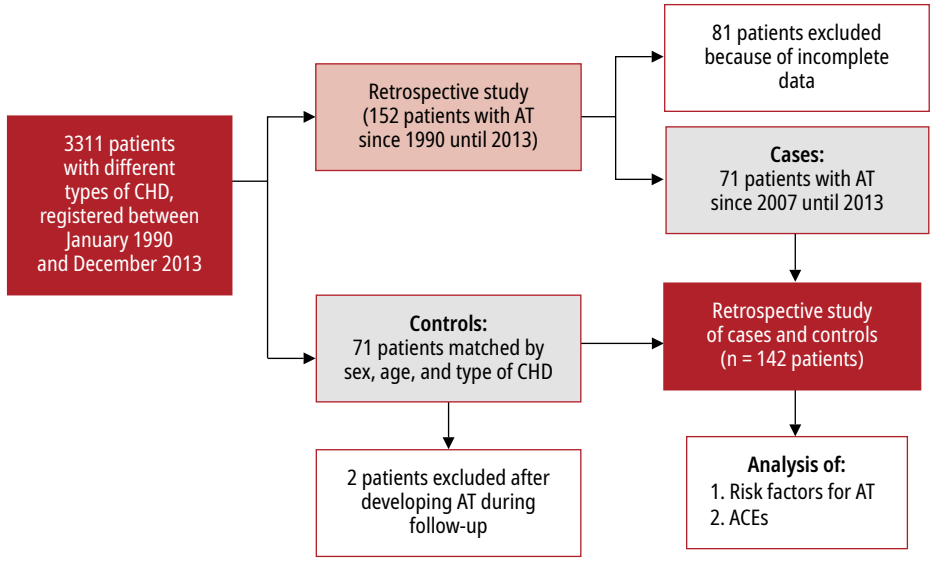


FIGURE 1 Flowchart of all patients with congenital heart disease in this cohort study
Abbreviations: ACEs, adverse cardiovascular events; AT, atrial tachyarrhythmia; CHD, congenital heart disease

Data collection **Demographic and clinical variables** Demographic data (sex, date of birth, and body surface area), anatomy, surgical history or catheter interventions, medical therapy (β-blockers, digoxin, antiarrhythmic agents, and anticoagulants), arrhythmias, New York Heart Association functional class, as well as comorbidities such as hypertension, dyslipidemia, coronary artery disease, diabetes, and previous or current smoking status were recorded.

Resting 12-lead surface electrocardiography was performed in all study patients before arrhythmia occurrence. Heart rate, rhythm, and QRS duration were obtained from electrocardiograms preceding the arrhythmia. Electrocardiograms recorded during arrhythmia occurrence were also analyzed.

We collected data on the treatment of the first AT episode and recurrences. Other arrhythmias such as atrial fibrillation and sinus node dysfunction (SND) were recorded as well. The latter condition was defined as abnormal sinus bradycardia or chronotropic incompetence with inappropriate heart rate responses to physiologic demands during activity.

Echocardiography Transthoracic echocardiograms were analyzed by a physician (ECC) blinded to clinical data and electrocardiograms, according to the American Heart Association / European Association of Cardiovascular Imaging guidelines.[17] For the purposes of this study, ventricular and atrial dimensions, diastolic function, and ejection fraction were reassessed. Left ventricular ejection fraction was calculated by the Simpson biplane method for systemic ventricles with left ventricular morphology and by fractional area change, tricuspid annular plane systolic excursion, myocardial performance index, and systolic tissue Doppler imaging (TDI) for ventricles with right ventricular morphology. The ejection fraction was considered normal if greater than 55%, mildly reduced if between 45% and 54%, moderately reduced if between 35% and 44%, and severely impaired if below 35%.

Color, continuous-wave, and pulsed-wave Doppler imaging data for all valves as well as tissue Doppler imaging data were recorded. Valvular dysfunction was graded as absent, mild, moderate, significant (moderate-to-severe regurgitation), or severe according to the published guidelines.[18] The diastolic function of the left ventricle was assessed based on mitral inflow and tissue Doppler imaging data, in accordance with the current guidelines.[19] The diastolic function of the right ventricle was evaluated using free-wall tricuspid annulus TDI and tricuspid inflow Doppler imaging velocities. Restrictive right ventricular physiology was defined as the presence of diastolic forward flow in the main pulmonary artery coinciding with atrial systole (A wave), throughout the respiratory cycle.[20]

Subpulmonary ventricular systolic pressure (SPVSP) was determined by continuous-wave Doppler echocardiography of mitral or tricuspid regurgitation depending on the morphology of the subpulmonary ventricle. Atrial pressure was estimated according to the dimension of the inferior vena cava and its respiratory variation, when the subpulmonary ventricle was connected to the right atrium (RA). If the subpulmonary ventricle was connected to the left atrium, atrial pressure was considered to be 15 mm Hg.

The RA area was measured by 2-dimensional planimetry in the apical 4-chamber view. In patients with Ebstein anomaly, the RA measurements included the atrialized portion of the right ventricle. In patients with a history of the Mustard and Senning procedures, the RA area was assessed by tracing the systemic or subpulmonary intracardiac pathways. The RA area was considered normal if ≤18 cm^2, mildly dilated if between 18 cm^2 and 24 cm^2, moderately dilated if between 24 cm^2 and 28 cm^2, and severely dilated if >28 cm^2.

Outcomes All study patients were monitored in our CHD outpatient clinic until December 2015, in line with the current guidelines.[21,22] Adverse cardiovascular events (ACEs) were defined as cardiovascular mortality, admission for heart failure (HF),[23] or stroke. An ACE was considered as the first occurrence of any of the 3 clinical outcomes during the follow-up period. Long-term AT recurrences after the first documented episode of AT and surgical or percutaneous interventions were recorded as well.

Statistical analysis Continuous variables were tested for normal distribution using the Kolmogorov–Smirnov test. Normally distributed variables were expressed as mean (SD) and compared with the unpaired or paired *t* test, as appropriate. The analysis of variance was used for comparisons of more than 2 groups. Nonparametric variables were expressed as median (interquartile range) and compared using the Mann–Whitney test. Group percentages were compared using the χ^2 test or the Fisher exact test, as appropriate. The association of AT with time to ACE was assessed using the Kaplan–Meier curves and the log-rank test. Receiver operating characteristic curves were used to provide the optimal area under the curve (AUC) for possible risk factors for AT.

To assess the predictors of AT in both study groups, univariate and multivariate analyses were performed. Hazard ratios (HRs) with significant 95% CIs were calculated using Cox proportional hazard models. Variables with a *P* value below 0.2 were entered in a multivariate forward logistic regression model. Variables were adjusted by confounding factors (diabetes and

TABLE 1 Baseline and follow-up characteristics of the study patients with atrial tachyarrhythmias and controls

Characteristics		All patients (n = 142)	With AT (n = 71)	Without AT (n = 71)	HR	95% CI	*P* value
Baseline characteristics							
Age, y, mean (SD)		42 (13)	41 (14)	42 (12)	1.9	10–13.6	0.75
Female sex		78 (55)	39 (55)	39 (55)	1	0.5–2	>0.99
Diabetes		6 (4)	6 (8.5)	0	0.5	0.4–0.6	0.01
Hypertension		11 (8)	8 (11)	3 (4)	2.9	0.7–11	0.12
Hypercholesterolemia		15 (10)	7 (10)	8 (11)	0.9	0.3–2.5	0.8
Smoking status		9 (6)	0	9 (13)	0.5	0.4–0.6	<0.01
Ischemic heart disease		3 (2)	2 (2.8)	1 (1.4)	2	0.2–23	0.56
Prior surgical treatment							
Previous surgery		118 (83)	61 (52)	58 (49)	1.5	0.6–3.6	0.37
Shunt		18 (13)	9 (13)	9 (13)	1	0.4–2.7	>0.99
Right atriotomy		70 (49)	41 (59)	29 (41)	2	1.1–3.9	0.04
Number of surgeries	0	24 (17)	8 (33)	16 (67)	4.7	1.1–1.3	0.02
	1	79 (56)	40 (51)	39 (49)	–	–	–
	2	30 (21)	16 (53)	14 (47)	–	–	–
	≥3	9 (6)	7 (11)	2 (3)	–	–	–
Electrocardiography							
QRS, ms, mean (SD)		124 (32)	128 (32)	120 (31)	9.2	13–20	0.09
Heart rate, bpm, mean (SD)		65 (13)	68 (14)	64 (12)	3.4	0.7–8	0.11
Pacemaker		17 (12)	15 (21)	2 (3)	9	2–42	<0.01
LBBB		7 (5)	6 (8.5)	1 (1.4)	6.5	0.8–55	0.05
Sinus node dysfunction		16 (11)	11 (15)	5 (7)	0.7	0.5–0.9	<0.01
Chest X-ray							
Cardiomegaly (>50%)		77 (54)	47 (66)	30 (42)	2.7	1.4–5.3	<0.01
NYHA functional class							
I and II		123 (87)	58 (82)	65 (92)	6	1.7–2	<0.01
III and IV		19 (13)	13 (18)	6 (8)	–	–	–
Medical treatment							
No treatment		92 (65)	30 (42)	62 (87)	8.9	3.8–21	<0.001
β-Blockers		40 (28)	32 (45)	8 (11.3)	–	–	–
Amiodarone		5 (4)	5 (7)	0	–	–	–
Dronedarone		1 (1)	1 (1.4)	0	–	–	–
Digoxin		2 (2)	2 (3)	0	–	–	–
Sotalol		1 (1)	1 (1.4)	0	–	–	–
Adverse cardiovascular events during follow-up							
Any		14 (10)	12 (17)	2 (3)	6.8	1.5–13	<0.01
Death		2 (1)	1 (1.4)	1 (1.4)	1	0.1–16	>0.99
Heart failure		10 (7)	9 (13)	1 (1.4)	10	1.3–82	<0.01
Stroke		5 (4)	4 (6)	1 (1.4)	4.2	0.5–38	0.17
Procedures during follow-up							
Pacemaker implantation		4 (3)	4 (6)	0	0.5	0.4–0.6	0.04
Surgery		21 (15)	17 (24)	4 (6)	5.3	1.6–17	<0.01

Data are presented as number (percentage) of patients unless otherwise indicated.

Abbreviations: HR, hazard ratio; LBBB, left bundle-branch block; NYHA, New York Heart Association; others, see FIGURE 1

TABLE 2 Baseline echocardiographic characteristics of the study patients with atrial tachyarrhythmias and controls

Echocardiographic variables	All patients (n = 142)	With AT (n = 71)	Without AT (n = 71)	HR	95% CI	*P* value
2-dimensional examination						
LVEDV, ml	110 (54)	102 (49)	118 (57)	16.6	1.2–36	0.07
LVESV, ml	48 (32)	48 (35)	49 (29)	1.2	0.4–12	0.82
Systemic ventricular EF, %	56 (11)	54 (13)	58 (7)	3.8	0.2–7	0.04
Basal RV diameter, mm	45 (9)	47 (9)	43 (1)	3.6	0.3–0.4	0.03
RV diastolic area, cm^2	31 (9)	32 (10)	30 (8)	1.5	1.9–5	0.4
RV systolic area, cm^2	19 (7)	20 (8)	18 (6)	1.6	0.9–4.2	0.25
Subpulmonary ventricular EF, %	61 (12)	60 (12)	62 (11)	1.9	6–2.4	0.4
FAC, %	39 (9)	38 (10)	39 (8)	1.8	1.5–5.2	0.3
TAPSE, mm	17 (6)	16 (6)	19 (5)	2.6	0.6–4.6	0.01
RA area, cm^2	23 (9)	28 (11)	20 (6)	7.4	4.5–10.3	<0.001
LA area, cm^2	18 (7)	19 (8)	18 (6)	1.4	0.8–3.6	0.2
Doppler imaging						
Systolic wave TDI, cm/s	9 (3)	8 (3)	10 (3)	1.4	0.3–2.5	0.01
Restrictive RV, n (%)	16 (11)	16 (23)	0	0.4	0.3–0.5	<0.001
Restrictive LV, n (%)	12 (9)	11 (16)	1 (1.4)	12	1.6–101	0.02
Significant MR, n (%)	6 (4)	4 (7)	2 (1)	5.2	0.6–46	0.1
Significant TR, n (%)	23 (16)	18 (25)	5 (7)	4.5	1.6–13	<0.01
Severe PR, n (%)	13 (9)	7 (10)	6 (9)	0.8	0.3–2.7	0.1
SPVSP, mm Hg	41 (17)	47 (19)	35 (13)	11.6	5–17	<0.001

Data are presented as mean (SD) unless otherwise indicated.

Abbreviations: EF, ejection fraction; FAC, fractional area change; LA, left atrium; LV, left ventricle; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; MR, mitral regurgitation; PR, pulmonary regurgitation; RA, right atrium; RV, right ventricle; SPVSP, subpulmonary ventricular systolic pressure; TAPSE, tricuspid annular plane systolic excursion; TDI, tissue Doppler imaging; TR, tricuspid regurgitation; others, see FIGURE 1 and TABLE 1

smoking status) in both study groups. Statistical significance was regarded as a 2-tailed *P* value less than 0.05. Statistical analysis was performed with the SPSS statistical package, version 21.0 (SPSS, Chicago, Illinois, United Sates).

Written informed consent was obtained from all participants. The study was approved by the local ethics committee.

RESULTS Study population characteristics

A total of 71 patients with AT were included in the study. Simple CHD was reported in 19 patients (26%), moderate in 26 (37%), and severe in 26 (37%). Five types of CHD were observed in 72% of the patients from the AT group (ToF, transposition of great arteries, univentricular physiology, ASD, and Ebstein anomaly) (Supplementary material, *Table S1*).

Clinical predictors The baseline clinical characteristics of the study patients are presented in TABLE 1. The majority of patients was female (55%), and the mean (SD) age was 41 (14) years. Patients with AT more frequently underwent surgery (*P* = 0.02) and right atriotomy (*P* = 0.04) compared with the control group. Diabetes was more common in the AT group, whereas smoking history, in controls. There was no difference between the study groups in terms of the remaining demographic and clinical variables. Apart from that, the AT group had a worse New York Heart Association functional class (*P* <0.01). The most common symptoms during tachyarrhythmias included palpitations (67%), dyspnea (11%), syncope or presyncope (4%), and chest pain (3%). However, 14% of the patients were asymptomatic during the AT episode.

Need for pacemaker implantation was more common in patients with AT than in controls (*P* = 0.04), as was SND (*P* = 0.03). There were no differences in the QRS duration (*P* = 0.09). Atrial fibrillation was found in 16 patients (22.5%) with AT. Cardiomegaly on chest X-ray was also more frequent in this group (*P* <0.01).

Echocardiographic variables The echocardiographic characteristics of the study groups are presented in TABLE 2. Systemic ventricular impairment (*P* = 0.04) and diastolic dysfunction of the RV (*P* <0.001) and the left ventricle (*P* = 0.02) were more common in the AT group. The same applied to RA dilatation

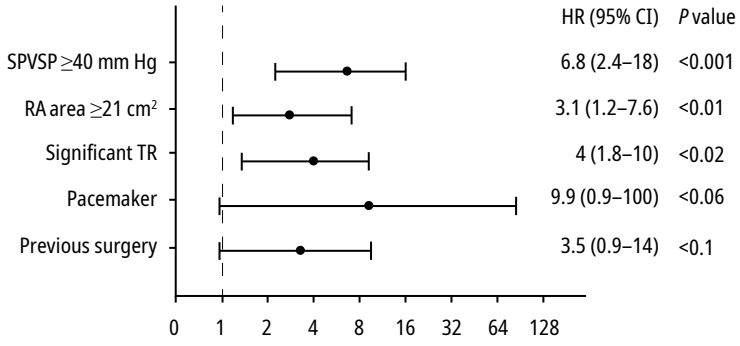


FIGURE 2 Multivariate analysis of atrial tachyarrhythmia predictors
Abbreviations: see TABLES 1 and 2

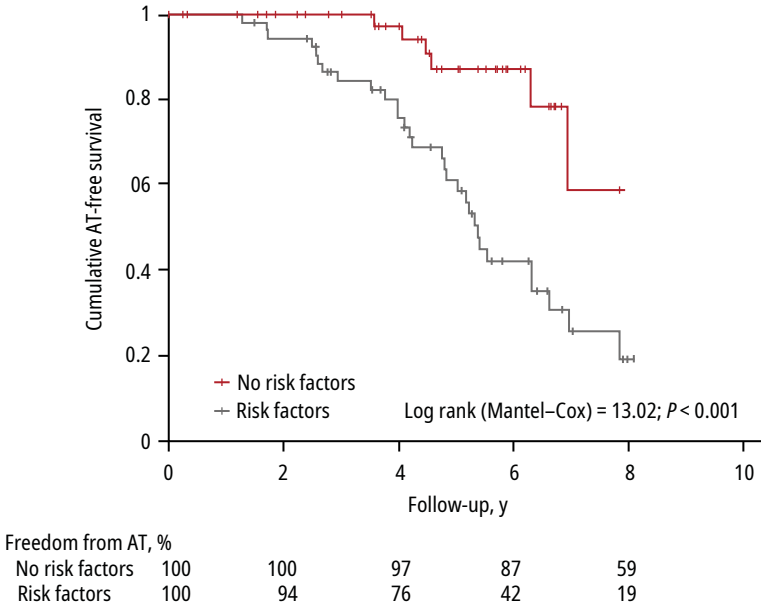


FIGURE 3 Survival free of atrial tachyarrhythmias according to the presence of risk factors from multivariate analysis. Patients with risk factors more frequently developed atrial tachyarrhythmias than those without.
Abbreviations: see FIGURE 1

(P <0.001), significant tricuspid regurgitation (TR) (P <0.01), and SPVSP (P <0.001). The tricuspid valve was systemic in 14 patients with AT and controls (19% of the total study cohort). The groups did not differ in terms of mitral and pulmonary regurgitation.

Receiver operating characteristic curves were plotted and the best cutoff value was 40 mm Hg for SPVSP (AUC, 0.81; 95% CI, 0.72–0.9; P <0.001; sensitivity, 0.75; specificity, 0.44) and 21 cm^2 for the RA area (AUC, 0.76; 95% CI, 0.66–0.86; P <0.001; sensitivity, 0.73; specificity, 0.4).

Predictors of atrial tachyarrhythmias Factors associated with AT in the multivariable analysis were as follows: SPVSP ≥40 mm Hg (HR, 6.8; 95% CI, 2.4–18; P <0.001), RA area ≥21 cm^2 (HR, 3.1; 95% CI, 1.2–7.6; P = 0.01), and significant TR (HR, 4; 95% CI, 1.3–10; P = 0.02) (FIGURE 2). During an 8-year follow-up, AT was diagnosed in almost 81% of patients with these risk factors compared with 41% of patients without these risk factors (P <0.001) (FIGURE 3).

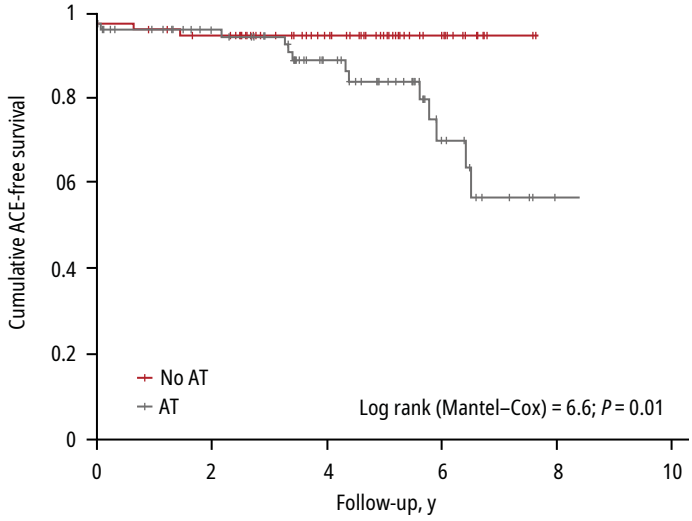
Treatment Electrical and/or pharmacological cardioversion was performed in 50% of patients in the context of acute treatment, heart rate control strategy was used in 20%, and no treatment was initiated in 30%. Radiofrequency catheter ablation (RFCA) was performed in 15 patients, 73% of the patients in whom antiarrhythmic treatment was not started. During the follow-up, pharmacological treatment was changed in 78% of cases (β-blockers in 52%, amiodarone in 19%, flecainide in 3%, calcium antagonists in 2%, and digoxin in 2%).

Radiofrequency catheter ablation was performed in 40 patients. As many as 60% of the RFCA procedures were carried out in patients in whom pharmacological treatment failed to maintain sinus rhythm. Cavotricuspid isthmus (CTI) ablation was performed in 30 patients (75%). Cavotricuspid isthmus ablation combined with other arrhythmia ablations was conducted in 8 patients (20%), and 2 patients (5%) underwent RFCA for incisional tachycardias, which were non–CTI-dependent. Successful ablation during the procedure was achieved in 35 patients (87.5%). The recurrence of AT after successful RFCA was reported in 6 patients (15%): 4 of those patients were treated with the second RFCA procedure, a single patient with electrical cardioversion, and no additional treatment was required in the remaining individual.

Adverse cardiovascular events The median (IQR) follow-up period was 4.4 (2.4–5.8) years. Adverse cardiovascular events were reported in 14 patients with or without AT and were 6-fold more common in patients with AT (86% vs 14%; P <0.01). Patients with AT more frequently presented with HF (13% vs 1.4%; P <0.01), but there was no significant difference in mortality or stroke. Stroke was documented in 4 patients with univentricular physiology or systemic RV and in a single patient with simple CHD. During the follow-up period, 17 cardiac surgeries and 2 percutaneous treatment procedures were performed in patients with AT compared with 4 operations in patients without AT (P <0.01). In both study groups, a similar ACE rate was noted during the first 2 years of follow-up. However, after a follow-up of 8 years, 58% of patients with AT remained free of ACEs compared with 98% of those without AT (P = 0.01; FIGURE 4).

DISCUSSION

The main findings of our study in adult patients with CHD were as follows: 1) the independent risk factors for AT included RA dilatation, elevated SPVSP, and significant



Freedom from ACE, %

No AT	100	98.5	98	98	98
AT	100	98	91	77	58

FIGURE 4 Survival free of adverse cardiovascular events in patients with and without atrial tachyarrhythmias (ATs). Patients with AT more frequently developed adverse cardiac events (cardiac death, stroke, or heart failure) than those without AT.
Abbreviations: see FIGURE 1

TR; 2) an increased rate of ACEs and a higher risk of redo surgery or percutaneous treatment during follow-up were observed in patients with AT.

Remodeling of the right atrium Macroreentry circuits represent the main etiology of AT.[3] These circuits could be CTI-dependent,[6,7] but there is also a well-established association between these arrhythmias and the presence of postsurgical incisions, scars, patches, or other anatomical barriers (ie, tricuspid annulus, crista terminalis, and Eustachian ridge).[23,24] Our study showed that RA dilatation, SPVSP, and TR are risk factors for developing AT in the population of patients with CHD.

Initially, the RA responds to atrial wall stress with RA hypertrophy and RA ejection fraction could be increased to maintain cardiac function. At some point, these adaptive mechanisms fail, the RA dilates, and RA ejection fraction progressively decreases. The histological examination of the RA in this setting has shown extensive fibrosis, the largest myocyte diameters, the longest capillary distances, and more prominent inflammatory cell infiltration.[23] These histopathological changes may develop even in the absence of a previous surgical intervention and they seem to progress in time in the context of increasing pressure and RA volume overload.[23]

Published data have demonstrated that AT could be related to RA remodeling,[25] and RA myocardial hypertrophy may also cause the disruption of atrial myocardial fibers resulting in changes in the atrial refractory period, SND, and slow conduction.[24] During this remodeling process, slow conduction zones (barriers of conduction) and/or complex conduction patterns (fractional electrograms, double potentials) may be formed,[26] which could be the substrate of atrial reentry circuits. Moreover, a correlation between slow conduction zones and fractional electrograms has been reported in patients with AT.[27]

In previous studies, a history of cardiac surgery, older age, and pulmonary regurgitation were regarded as risk factors for AT.[26-28] However, these findings were not confirmed in our study, maybe due to the fact that we matched controls by age, sex, and the type of CHD and enrolled a different number of study participants.

Diastolic dysfunction In our study cohort, restrictive RV physiology correlated with AT in the univariate analysis yet not in the multivariate analysis. However, its assessment was limited by the use of echocardiography, as invasive hemodynamic studies may sometimes be useful[29]; these studies were not performed in our cohort.

Restrictive RV physiology has been well described in ToF or pulmonary atresia with postsurgical pulmonary regurgitation,[19] but only a few cases of these conditions were noted in our study cohort. Indeed, restrictive RV physiology was present only in 23% of the patients with AT.

Therapeutic implications Previous studies have shown that AT may be related to embolic complications,[30] HF,[31] and sudden death.[32] In our cohort, AT increased the risk of HF by 9-fold. Also, patients with AT may need surgical or percutaneous procedures more frequently during the follow-up.

Severely complex CHD with univentricular physiology or systemic RV tends to be susceptible to AT, which affects patients' hemodynamic status and leads to high mortality and/or morbidity.[33] It is of key importance to monitor these patients closely in order to identify and treat, as soon as possible, residual or new structural abnormalities, such as valve disease or shunts, which may have an impact on hemodynamics and predispose to AT.

During cardiac surgery, atrial incision is performed to access cardiac chambers. However, a good understanding of cardiac anatomy, careful manipulation of sutures in relation to the AV node, interatrial septum, and natural anatomical barriers (crista terminalis, valve annulus, and vena cava) could decrease AT prevalence.[34,35]

Treatment for AT should be directed by individual differences in anatomical, hemodynamic, and electrophysiologic characteristics. The use of antiarrhythmic agents and/or atrial pacing to suppress AT have been unsatisfactory, including potent antiarrhythmics such as amiodarone.[36] Additionally, antiarrhythmic drugs could have potential adverse effects

in this young population of patients. Hence, RFCA has been suggested to be the first-line treatment.[21,37] However, patients with complex anatomies and several previous surgical procedures present with diverse locations of reentry circuits. A better understanding of the arrhythmogenicity substrate and anatomy are essential for RFCA to be successful in these patients. Nowadays, the use of modern electroanatomical mapping systems, potentially in conjunction with magnetic resonance imaging or computed tomography, could improve the final outcome and also help in planning the ablation procedure so as to avoid complications.[24]

Limitations Here, we presented a single-center retrospective study, which had its inherent limitations and bias. The sample size was relatively small and a low number of events reported did not allow us to draw firm conclusions. Moreover, 78 patients were excluded because of incomplete follow-up and echocardiographic data. However, the baseline characteristics of the 2 study groups were homogeneous and the patients represented our completed series of 152 patients with AT, but very short, asymptomatic AT episodes were not recorded, so the true prevalence of these arrhythmic complications might have been underestimated.

Conclusions Our study demonstrated the main risk factors for AT in patients with CHD. These included RA dilatation (remodeling), elevated systolic pressure of the subpulmonary ventricle (pressure overload), and significant tricuspid regurgitation (volume overload). Of note, the presence of AT may increase the risk of adverse cardiac events.

SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/kardiologiapolska.

ARTICLE INFORMATION

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CONFLICT OF INTEREST None declared.

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