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Arrhythmological sudden death in athletes - electrophysiological assessment and management

Negru G Alina[1,2], Ivanică Gabriel[2], Sorca-Albișoru Lavinia[2], Matei Raluca[2], Petrescu Lucian[1,2], Ionac Adina[1,2], Negru Serban[1], Șt. I. Drăgulescu[1,2]
[1]*University of Medicine and Pharmacy Victor Babeș, Timișoara, Romania*
[2]*Institute of Cardiovascular Medicine Timișoara, Romania*

Abstract. *Background*. The assessment by electrophysiological study of sudden cardiac death risk of pure arrhythmological origin in the athletes with positive history of tachycardia was not previously described by the literature. *Method and results.* A prospective study conducted between 2010 and 2014 which included 62 patients, all athletes, performing category IIA sports or higher according to Bethesda Classification of sports, with positive history of arrhythmia (ECG documentation; syncope or aborted cardiac arrest; suggestive symptoms). All patients were evaluated by non invasive (ECG, effort testing) and invasive testing (electrophysiological study). The radiofrequency ablation was done in selected patients. The mean age of all patients was 17.62±6 years. There were found 29 patients (46.77%) with sudden cardiac death risk, from which 15 patients (24.19%) had only symptoms, not previous ECG documentation of arrhythmia and were discovered with high sudden death risk at electrophysiological study. The most common types of arrhythmia associated with sudden death risk were found to be the Wolff-Parkinson-White syndrome, atrioventricular nodal reentrant tachycardia and atrioventricular reciprocating orthodromic tachycardia due to concealed accessory pathways, ventricular tachycardia and premature ventricular contractions with degeneration into ventricular tachycardia. *Conclusions*. The sudden death risk is much more spread than thought among the athletes. This is due to cardiac remodeling at one hand and to associated discrete pathologic conditions on the other hand. Even if there is no documented tachycardia but the symptoms are suggestive for arrhythmic events, further investigation by effort testing and electrophysiological study in selected cases must be done. The two mechanisms of death that we assessed in association with the above described arrhythmias are degeneration into VF or hypo diastolic shock with hemodynamic collapse due to very fast tachycardia. In almost all cases, the radiofrequency ablation was the complete curative method, permitting the continuation of sports.
Key words: *athletes, sudden cardiac death, supraventricular tachycardia, ventricular tachycardia, electrophysiological study, screening.*

Introduction

The sudden cardiac death (SCD) is considered to be the unexpected death caused by cardiac pathology, occurred within one hour from the onset of symptoms in a patient without any known fatal condition (1). It is well known that the repeated athletic training and competitions result in changes of the heart structure and function (2). This process is known as exercise-induced cardiac remodeling and consists in left ventricle enlargement and concentric or eccentric hypertrophy, enlarged right ventricle (RV) mostly in endurance athletes, enlargement of the aortic root, enlargement of the left atria (3,4). Remodeling condition, underlying pathologic substrate (accessory pathways (APs), concealed pathways, double nodal pathways, ectopic foci) or the combination of both, can lead to life threatening arrhythmias.

Atrial fibrillation (AFIB) in the presence of a Wolff-Parkinson-White (WPW) syndrome with a short anterograde effective refractory period (AERP) accessory pathway, or even AFIB alone may be particularly problematic in the professional athlete. Cardiac arrest and SCD of arrhythmological causes in the athlete is the subject of continuous debates and research.

The electrocardiogram (ECG) and the treadmill test are mandatory for the periodical evaluation of the athlete in order to obtain the visa for continuing the sports activity, according to the international guidelines. However, up to 40% of the ECGs will have an abnormal pattern, the

consequences of athletic conditioning itself and representing a component of the "athletic heart" (left ventricular (LV) hypertrophy by voltage: RV1/2 + S V5/6>35mm, corrected QT interval in 0.44-0.49s range, resting sinus bradycardia <50/min) (5). On the other hand, the false positive or false negative results in effort testing or even in standard 12 lead ECG recorded at rest represent limitation of the screening in this specific population. Usually, the underlying arrhythmias are identified combining routine screening (medical history, physical examination, ECG, effort testing), present symptoms or history of symptoms (palpitations, syncope, etc.) and family history of SCD. Sometimes, the incidental finding is the only way of diagnosing a SCD predisposing arrhythmia. The echocardiography is always useful to assess associated cardiac structural conditions.

Aim of the study. During the last three years we experienced large athlete addressability and we tried to identify the most common arrhythmias (including the risk of SCD by cardiac arrest, characterizing every type of arrhythmia, the distribution by age and type of sport), to study the arrhythmogenic conditions by electro-physiological study (EPS) and to proceed to the treatment by radiofrequency current catheter ablation (RFA) in the selected cases.

This study aims to prove also that the catheter ablation can cure the arrhythmia in almost all cases, offering the patient the chance to continue the athletic training and competition shortly after procedure.

It has to be mentioned that this study follows our electrophysiological experience with trained athletes and is the first study revealing the SCD risk of common arrhythmias by invasive procedures like EPS.

This is also the first study performed in the athletes aiming to demonstrate the underlying mechanisms of all types of arrhythmia with sudden death risk (SDR), not only the ventricular ones, in this specific population. This study is the first revealing the SCD risk of pure arrhythmological origin in the athlete.

Material and Method

The study included athletes (n=62) selected between a larger number of addressed patients (n=74), with age between 8 and 32 years, referred to our center between March 2010 and March 2014 for advanced cardiac evaluation, EPS and possible RFA.

Athletes were defined as those engaged in professional sport activities and classified according to the Bethesda classification of sports (5).

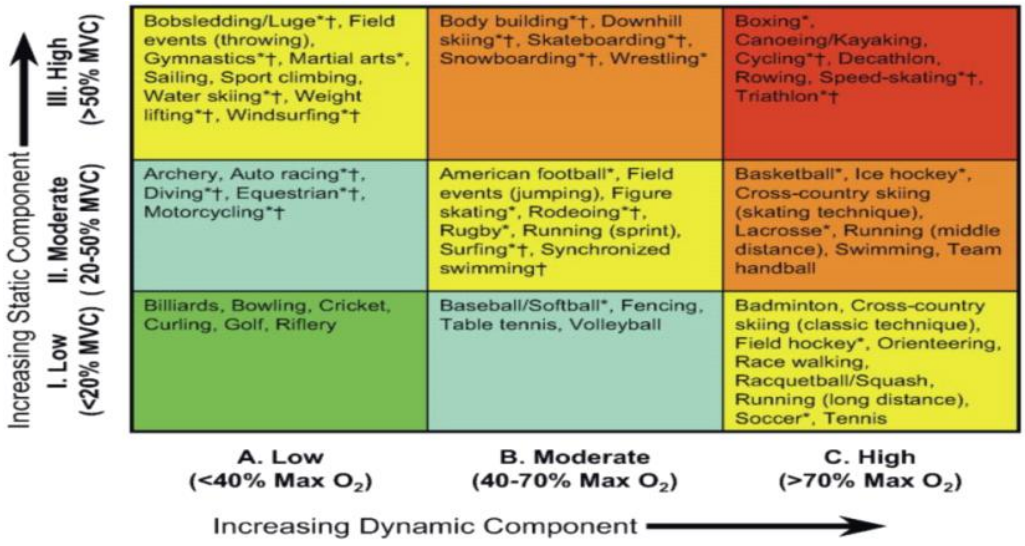

Figure 1.Classification of sports
(From: Bethesda Conference Report, 36th Bethesda Conference: Eligibility Recommendations for Competitive Athletes with Cardiovascular Abnormalities Task Force 8: Classification of sports. *J Am Coll Cardiol.* 2005; 45(8):1364-1367*)*

In a detailed questionnaire patients were asked about the type of sport, number of sports participation, the number of years of participation in different competitions, the number of episodes of arrhythmia experienced and associated symptoms.
Inclusion required were: (i) ECG documented arrhythmia ± structural heart disease; (ii) Syncope or aborted cardiac arrest; (iii) Suggestive symptoms (sudden onset and termination of palpitations) alone or associated with familial sudden death with or without ECG tracing documentation; (iv) History of arrhythmia; (v) Category IIA sport or higher, according to the Bethesda classification (exception-5p included in the study performing dance sport, sport that is not included into Bethesda classification).
The exclusion criteria were: missing data; ablation procedures performed in other centers; patient refusal of undergoing invasive exploration.
The informed consent was obtained from all 62 patients included in the study. The patients were differentiated by the level of difficulty associated to their athletic performance resulting in 51 endurance athletes (running, rugby, rowing, football, basketball, handball, judo, climbing) and 11 patients performing lighter sports (equestrian, gymnastics, dance sport, motocross).

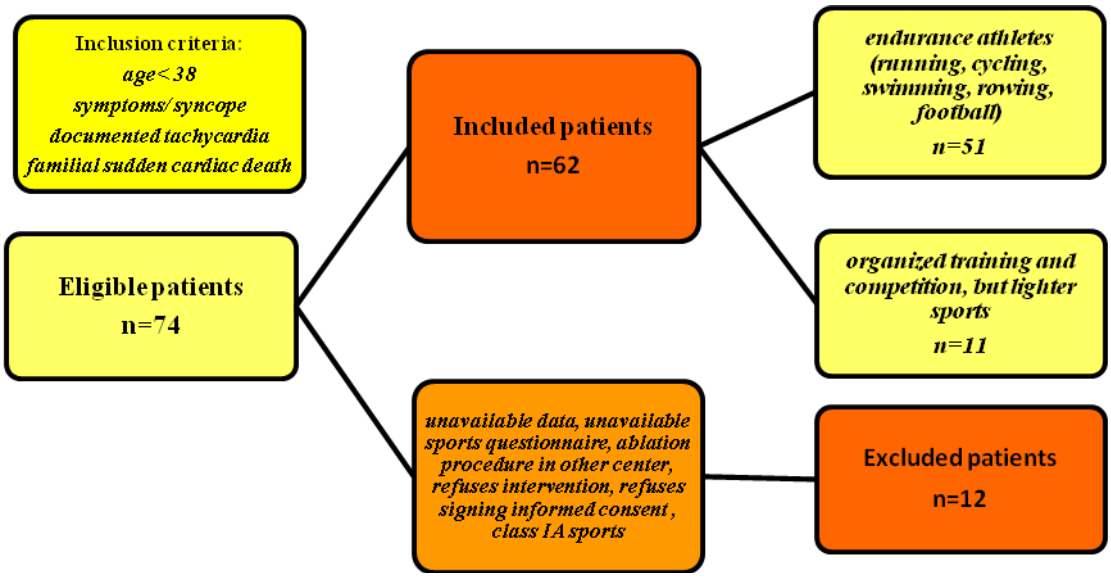

Figure 2. Chart of selection process

Non invasive and invasive screening
Every patient was evaluated by 12 lead rest ECG, bicycle effort testing, and echocardiography.
After the confirmation of normal blood tests, the EPS was performed.
Patients were in fasting condition and with an intravenous access installed in the electrophysiology laboratory.
Electrodes were attached in order to provide a continuous 12 leads surface ECG during procedure. A blood pressure cuff and a pulse-oximeter were also attached. Defibrillation electrodes were attached where if defibrillation would be required to be performed with minimal discomfort and no interference with the sterile field.
Indifferent electrode was placed in the thoraco-lumbar skin area for the expected possibility of RFA. Right groin local anesthesia was achieved with 5ml Lidocain 1%, then three femoral vein punctures were performed using the modified Seldinger technique.
A Siemens electrophysiology system and a Röentgen-Siremobil 2000-Siemens X-ray imaging system were used for heart stimulation, respectively for guiding the catheters placement.

The diagnostic catheters were placed by right femoral venous approach.
A quadripolar catheter with 5-5-5-mm spacing was advanced from the right femoral vein and placed at the right ventricular apex. A steerable quadripolar catheter with 2-2-2-mm electrode spacing was placed on the His bundle, a quadruple catheter with 5-5-5-mm electrode spacing on the lateral wall of the right atrium. For the coronary sinus we used a decapolar catheter with 2-8-2-8-2-8-2-8-2-mm spacing which results in five closely spaced dipoles 1cm apart.
Basic electrophysiological data were recorded. The basic intervals were measured during sinus rhythm, at a stable heart rate: the PP interval (the interval between two consecutive P waves of sinus rhythm), PA interval (interval between onset of surface P wave and earliest endocavitary atriogram), the AH interval (the interval between atrial electrogram recorded by the His catheter and the beginning of the His electrogram), H-delta interval (interval between the onset of His electrogram and the delta wave) or HV interval (interval between His deflection and ventricular electrogram).
Then, for every patient we proceeded to incremental atrial and ventricular stimulation and to atrial and ventricular extrastimulus testing, with one, two and three extrastimuli, in order to evidence the changes suggesting different types of arrhythmias and to trigger the suspected forms of tachyarrhythmia.
Radiofrequency catheter ablation
After the diagnostic invasive maneuvers were done, we eventually established the type of rhythm trouble and proceeded to RFA in selected cases. We used a Biosense-Webster ablation generator together with a 6 French (F) and 7F Biosense-Webster deflectable ablation catheters. The delivered power varied between 20 W and 40 W, adjusted to achieve the target temperature of 50-55 degrees Celsius. The duration of energy deliverance varied between 30 and 60 seconds for one radiofrequency application and we recorded a mean number of 3 radiofrequency applications per patient.

Results

There were a number of 62 patients included in the study, 15 women and 47 men between 8 and 32 years. Mean age of all patients was 17.62±6 years. Most patients (88.7%) had prior symptoms, concordant with the tachycardia episodes.
The baseline of echocardiography characteristics of all athletes showed: end diastolic diameter 4.52±0.61cm, end systolic diameter 2.72±0.57cm, interventricular septum 1±0.18cm, posterior wall of the LV 0.97±0.17cm, ejection fraction of the LV 66.39±6.96% and left atrial surface 18.45±2.72 cm^2.
Apparently, the LV diameters and walls seem to be normal, but taking into account the young age of a large part of the patients included in the study (39 patients with ages ≤18 years) those values are rather at the superior limit of the normal range of measurements.
The LV diameters and the wall thickness are parameters concordant with higher risk of arrhythmogenesis due to cardiac remodeling.
We defined the SCD risk by cardiac arrest as fast degeneration into ventricular tachycardia (VT) and ventricular fibrillation (VF) of an apparently stable tachycardia/arrhythmia with alteration of the hemodynamic status, primary VT associated with very fast heart rate >250/min, the coexistence of rapid AFIB and WPW syndrome with a short anterograde effective refractory period (AERP<220ms) AP with high risk for degeneration into VF, or the hypo-diastolic shock associated with hemodinamic collapse or loss of consciousness due to very high heart rate in some forms of supraventricular tachycardia.
In this light, we recorded 29 patients (46.77%) with SCD risk from which 3 asymptomatic patients discovered incidentally prior to the hospital admission by a routine medical examination and ECG recording.
Sixteen patients (25.8%) presented with syncope and hemodynamic deterioration during tachycardia, from which 15p (24.19%) were discovered with high risk for SCD at the EPS.
The sport types revealed a large number of professional football players (21p, 33.87%), 8 basketball players (12.9%), 5 dance sport (8.06%), 2 equestrian (3.22%), 5 rugby players (8.06%), 2 judo (3.22%), 6 performing athletics running (9.67%), 3 gymnastics (4.83%), 1 motocross (1.61%), 2 climbing (3.22%), 4 rowing (6.45%) and 3 handball players (4.83%).

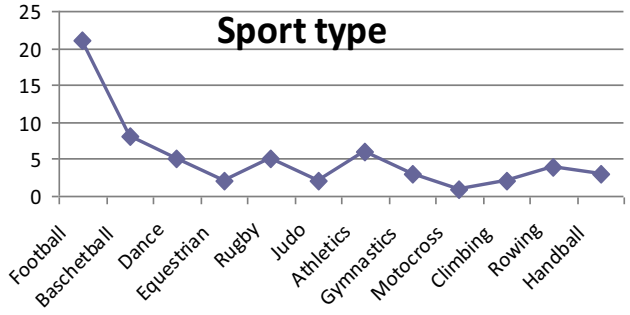

Figure 3. Sport activities distribution

The EPS performed in all patients revealed different types of arrhythmia:

- WPW syndrome (22.5%) from which 21,42% of the WPW group (4,83% of all patients) presented anterograde conduction through an accessory pathway with a short AERP;
- concealed APs with atrio-venticular reciprocating orthodromic tachycardia (AVROT) (14.51%);
- double nodal pathway with atrioventricular nodal reentrant tachycardia (AVNRT) (22.5%) equal to the WPW syndrome incidence;
- ventricular tachycardia (VT) (17.74%);
- premature ventricular contractions (PVCs) (6.45%);
- AFIB (9.67%);
- focal atrial tachycardia (AT) (3.22%);
- other arrhythmia including right atrial flutter, Mahaim fascicle (3.22%).

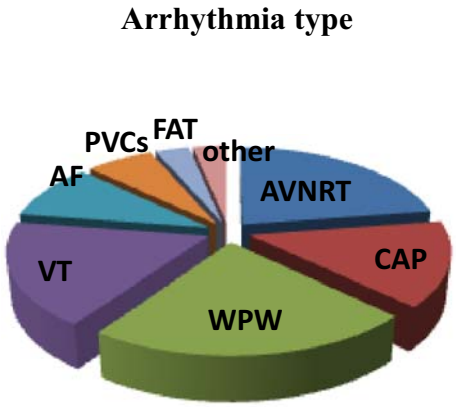

Figure 4. Types of arrhythmia diagnosed in the entire study population**.**
AVNRT-atrioventricular nodal reentrant tachycardia, CAP- concealed accessory pathways with atrio-ventricular junctional reciprocal tachycardia, Wolff-Parkinson-White syndrome, VT- ventricular tachycardia, AF-atrial fibrillation, PVCs- premature ventricular contractions and FAT- focal atrial tachycardia.

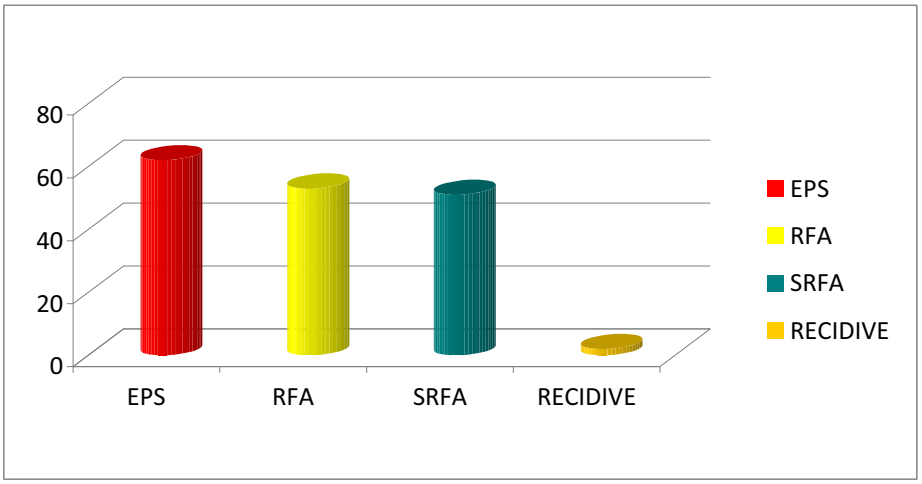

Figure 5. Distribution of the procedures and recidivate after ablation**.**
EPS- electrophysiological study, RFA- radiofrequency ablation, SRFA-successful radiofrequency ablation.

If EPS evaluation was done in all 62 patients included in the study, the RFA was performed in 53 patients (85.48%) and the success was obtained in 51 patients (82.25% of all and 96.22% of the ablation group). Two patients had recidivate after ablation.
After all screening, diagnostic and therapeutic procedures were performed we assessed a number of arrhythmias with SDR.
The total number of patients found with SDR is 29 (46.77%) divided as it follows:

- 9p (14.51% of all patients, 64.28% of the WPW group)with WPW syndrome:
  - 4 cases (6.45% of all, 28.57% of the WPW group) with short AERP APs associated with AFIB, and degeneration into VF at the EPS;
  - 3 cases (4.83% of all, 21.42% of the WPW group) with apparently benign WPW;
  - 2 cases (3.22% of all, 14.28% of the WPW group) of WPW syndrome with changing properties of the APs during radiofrequency applications;
- 5p (8.06% of all, 55.55% of the "concealed" AP group) with AVROT and AVNRT associated with very fast heart rate resulted into cardiogenic hypo diastolic shock with hemodynamic collapse;
- 9p (14.51% of all, 81.81% of the VT group) with RVOT sustained tachycardia;
- 2p (3.22% of all, 18,18% of the VT group) with repetitive nonsustained right and left VT;
- 3p (4.83% of all, 75% of the PVCs group) with PVCs with systematized pattern.

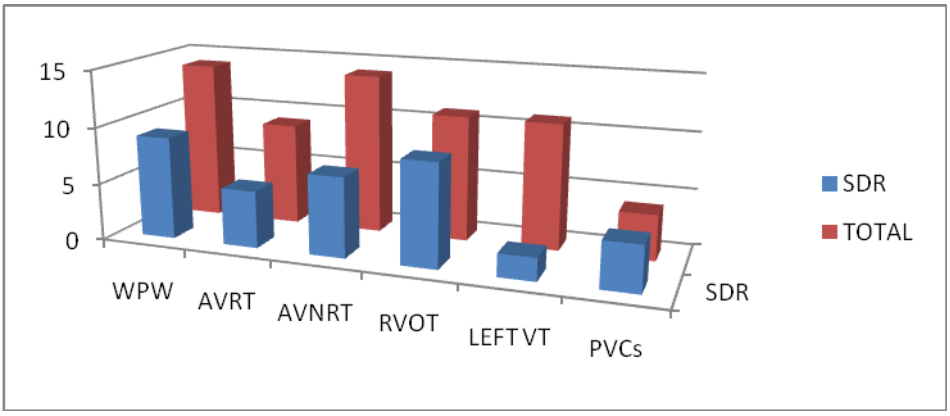

Figure 6.Arrhythmia chart comparing total number (second row) of each arrhythmic subtype and the number of arrhythmias from each subtype characterized by SDR (first row).
WPW- Wolf- Parkinson-White syndrome, AVRT-atrio-ventricular reciprocating tachycardia, AVNRT- atrio-ventricular nodal reentrant tachycardia, RVOT- right ventricular outflow tract ventricular tachycardia; VT- ventricular tachycardia, PVCs- premature ventricular contraction

Discussions

The assessment of the arrhythmic SCD risk during both EPS and RFA was a priority for our study. Regarding this aspect, we distinguished multiple mechanisms of cardiac arrest linked to the following specific arrhythmias: WPW syndrome associated with AVROT, atrio-ventricular reciprocating antidromic tachycardia (AVRAT), rapid ventricular response over the AP during AFIB, concealed APs associated with very fast AVROT, sustained VT, short, repetitive VT, VE and fast AVNRT.

It is mandatory a brief characterization of the mentioned arrhythmias.

The WPW syndrome and "concealed "conduction Aps. The WPW syndrome is a direct electric connection between atria and ventricles (cavities normally separated and isolated by a fibro-adipose ring) competing with the normal conduction pathway. Those aberrant communications are called Kent fibers conducting very fast and without detrimental pattern. There are also described Kent fibers with decremental properties (very few).

Usually, the AP in WPW syndrome has bidirectional conduction possibilities.

When the conduction happens exclusively in retrograde direction the AP is called to have "concealed conduction"(concealed AP).

The Kent bundle can be the origin of multiple tachycardias: AVROT using the normal atrioventricular pathway anterogradely and the Kent bundle or the concealed AP retrograde. The AVROT can be associated with concealed conduction APs also. This type of tachycardia has narrow QRS complexes, usually fast and associated with high repetitivity. In our study, the AVROT were associated with high heart rate determined by the high cathecolaminergic load during training or competitions, causing hemodynamic collapse or syncope. The AVRAT uses the Kent bundle anterogradely and the normal atrioventricular

conduction pathway retrograde. It is a large QRS tachycardia, less common than the AVROT. Other types of arrhythmias associated with the WPW syndrome are the atrial flutter and the AFIB. These arrhythmias can rapidly degenerate into VF in the presence of an AP with anterograde conduction and short AERP. The ventricles are activated very fast by the atrial flutter or fibrillation waves which are passing directly through the fast conduction AP shortcutting the normal atrioventricular pathway with decremental conduction (8).

From the 14 patients with WPW syndrome, 4 presented with a short AERP of the AP, an anterograde blocking cycle in the accessory pathway measuring less than 250 ms and a critical RR preexcited interval of the AP shorter than 220ms. In 3p of this subgroup of patients we evidenced associated AFIB. Other 3 patients in the WPW group had apparently benign intermittent anterograde conduction through the AP. The progressively increasing frequency ventricular stimulation revealed a block in the 4th phase of repolarisation. The preexcitation became permanent and the anterograde conduction block in the AP occurred under the cycle of 250ms. Two cases showed changes in the properties of the APs after the beginning of radiofrequency delivery, thus the benign WPW became malign with shortening of the AERP under local inflammation condition caused by a difficult approach of the target point and multiple RFA lesions in the AP's area, before the permanent status and extension of lesions to completely destroy the pathway.

It has to be also mentioned the possibility confirmed by our experience, that an intermittent, benign WPW syndrome (proved to be benign at an initial EPS), can change its electrophysiological properties and become malign in conditions of localized myocardial inflammation caused by a different mechanism (viral myocarditis or fever, for example).

The AVNRT is a tachycardia that originates in the atrioventricular node, having as substrate the dual conduction pattern of this structure. The two pathways have different electrophysiological properties: the *beta* pathway has a fast conduction pattern around 50 ms but has also a long effective refractory period. The *alfa* pathway has a slow conduction time around 300 ms associated with a short effective refractory period. The onset and the maintenance of the tachycardia need the circuit created by the two pathways with different conduction times and different refractory periods. There are two forms of tachycardia generated by this circuit: the "fast-slow" tachycardia and the most common "slow-fast" tachycardia (exclusively found in our patients). The way of initiation of the AVNRT usually happens by an atrial premature contraction which blocks the rapid pathway anterogradely. The premature excitation wave must depolarize the ventricle through the slow pathway. This change of pathway is evidenced on the surface ECG by a

brutal prolongation of the PR interval and on the endocavitary electrograms by a prolongation of the AH interval (9). Once arrived at the hisian end, the impulse propagates to the ventricle and simultaneously retrogrades to the atrial extremity of the AV node through the fast pathway found out of the refractory period. Then, the activation may transmit again anterograde through the slow pathway closing the circuit and initiating the tachycardia. This is the common form of the AVNRT, the "slow-fast" type. The "fast-slow" type AVNRT uses the slow pathway retrograde and the fast pathway anterogradely. In the five patients that were diagnosed with high SDR at EPS the mechanism was exclusively due to the high heart rate, greater than 250 /min (which in conditions of cathecolaminergic background tend to become even greater). The high heart rate produced hemodynamic collapse by cardiogenic hypo-diastolic shock and loss of consciousness requiring basic resuscitation maneuvers till the end of the tachycardia. Even if in the literature it is mentioned also another mechanism for SCD, respectively the degeneration of the AVNRT into VF, we did not identify any patient among the athletes included in the present study with this type of cardiac arrest. However, the cases described by other authors were patients who presented with aborted sudden death and had associated coronary artery disease - the coronary spasm vas inducible with ergonovine and initiated the tachycardia (*9)*.

Ventricular arrhythmias

Ventricular tachycardias. The ventricular tachycardia (VT) originating in the right ventricular outflow tract (RVOT) in the absence of clear structural heart disease is rather common and represents 10% of all VTs evaluated by the arrhythmia services (10,11). The nonsustained form is more frequent, representing between 60 and 92% in reported series (12). The episodes tended to increase in frequency and duration during exercise and emotional stress. The arrhythmogenic right ventricular dysplasia (ARVD) may be associated with recurrent, prolonged and multiple QRS morphology VTs during distinct episodes. In our study we experienced only 1 case of athlete with ARVD with multiple morphology VTs which required ICD (implantable cardioverter defibrillator). It has been observed that the T wave inversion in the anterior precordial leads extending to V3 in sinus rhythm along with incomplete or complete right bundle block may be associated with ARVD. Our findings revealed a majority of patients with SDR from this group diagnosed with fast RV TV and all had athletic heart restructured pattern. Among the 11 patients with VTs included in our study, we had 9 right ventricular VTs and 2 left ventricular VTs. One of the left ventricular VTs was an idiopathic polymorphic VT which is described in the literature as a sporadic or familial form frequently precipitated by catecholamine release during physical or emotional stress (13). The other one was an anterior focal monomorphicVT. Among the 9 patients with RV VTs, 6 patients had RVOT tachycardia and the other 3 had right ventricular triggering points, other than the RVOT and 1 patient had idiopathic polymorphic VT. The burst ventricular pacing performed during the EPS evidenced sustained fast VT with degeneration tendencies in all cases. In one of the 6 patients with RVOT, the tachycardia was due to the catecholamine–enhanced automaticity (mono-morphic VT) and in another one to reentry. The two declanchement mechanisms appears to be uncommon for the RVOT TV and were observed more frequently in TVs originating from other RV spots (13).

Some authors reported that endurance athletes with arrhythmias had a high prevalence of right ventricular structural and arrhythmic involvement (14). This affirmation is compatible with our findings. We performed the RFA in all 11 patients, with two recidives, one at 6 months; this recidive occurred in the case of an alpinist with ARVD. The ICD implantation was mandatory, with consequent interruption of the sports activities. The other recidive occurred after 3 days from RFA in a patient with the left polymorphic VT which necessitated further antiarrhythmic therapy. The other 7 patients resumed their athletic activity 1 month after the RFA with no early or late recidive.

Premature ventricular contractions

There were 4 p in our study population found with systematized right and left ventricular ectopies (two patients with couplets, 1 patient with triplets, 2 patients with bygeminisme). At the EPS during ventricular stimulation, we evidenced nonsustained fast monomorphic VT in 3 cases and sustained left monomorphic VT in 1 case. We defined non-sustained VT as the tachycardia under 30 second duration and self termination pattern. The sustained VT is the tachycardia above 30 seconds self terminating or electrically converted. The framing of the PVCs associated with non-sustained VTs in the group of life

threatening arrhythmias is based on multiple studies which showed a substantial increase in all-cause mortality and sudden death risk in young patients with hypertrophic cardiomyopathy. The SDR was demonstrated as independent of the frequency, duration, and the heart rate of non-sustained VT episodes (16).
The successful RFA was performed in all 4 cases with no recidive during one year of periodical reevaluation.

We propose an algorithm for the survivors of a SCD and for the patients at risk, even if they were asymptomatic until the EPS revealed the SDR.
The superiority of ICD implantation over antiarrhythmic drugs (amiodarone) was shown in the AVID trial (8). In these conditions, the ICD becomes the first line treatment for the patients with aborted death by ventricular fibrillation in whom ventricular arrhythmia can be induced at EPS and the ablation is unsuccessful.

VENTRICULAR TACHYCARDIAS	No. of patients	RFA	RECIDIVE
TOTAL VTs	11	11	2
TOTAL LEFT VTs	2	2	1
TOTAL RIGHT VTs	9	9	1
RVOT VTs	6	6	0
RV VTs	3	3	1

Figure 7**.** Types of ventricular tachycardia identified by the study's analyze**.**
VTs- ventricular tachycardia, RVOT VTs- right ventricular outflow tract tachycardia, RV VTs- right ventricular tachycardia.

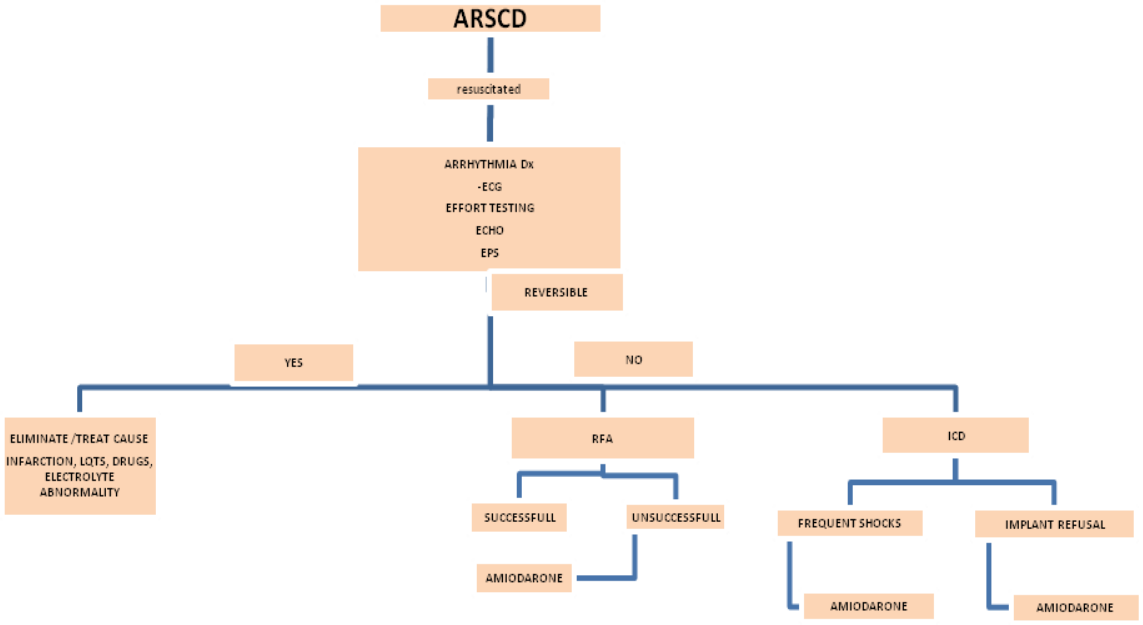

Figure 8. Treatment algorithm for patients with aborted arrhythmic sudden cardiac death
ARSCD-arrhythmic sudden cardiac death, Dx- diagnostic, ECG- electrocardiogram, Echo- echocardiography, EPS- electrophysiological study, LQTS- long QT syndrome; RFA- radiofrequency ablation, ICD- implantable cardioverter defibrillator.

Conclusions

The sudden death risk is much more spread than thought among the athletes. This is due to cardiac remodeling at one hand and to associated discrete pathologic conditions on the other hand.

This is the reason why a very detailed anamnesis, cardiac and general examination must be performed in this specific population. Any clinical suspicion of arrhythmia must be confirmed or denied before continuation of sports.

Even if there is no documented tachycardia but the symptoms are suggestive for arrhythmic events, further investigation by effort testing and EPS in selected cases must be done. The great and undesired impact of the sudden death justifies the recognition of silent or unsuspected substrate lying at the origin of life threatening arrhythmias in athletes. An arrhythmogenic substrate considered benign may become malign during time or in stressful conditions during competition. For that, we encourage the risk stratification by electrophysiological testing. We demonstrated at the EPS that various types of arrhythmia may be potential lethal in the context of an adrenergic background. This is the reason why a complex electrophysiological assessment and evaluation may be performed in the presence of any suspicion of arrhythmia. Our study revealed certain conditions associated with SCD, relatively common arrhythmias in fact, inherited or associated with the exercise induced heart remodeling or dysplasia.

The arrhythmias we found associated with SCD risk are: WPW syndrome with short AERP Kent bundle associated with AFIB; very fast AVROT produced by Kent bundle or concealed AP; very fast AVRNT produced by atrioventricular node duality; fast reentrant or focal VTs with risk of degeneration into VF at EPS; systematized PVCs with degeneration in fast sustained or non sustained VT at the EPS.

The two main mechanisms of death that we assessed in association with the above described arrhythmias are degeneration into VF or hypo diastolic shock with hemodynamic collapse due to very fast tachycardia.

Acknowledgments

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Abbreviations

AERP	anterograde effective refractory period
AFIB	atrial fibrillation
AP	accessory pathway
ARVD	arrhythmogenic right ventricular dysplasia
AT	atrial tachycardia
AVROT	atrioventricular reciprocating orthodromic tachycardia
AVRAT	atrioventricular reciprocating antidromic tachycardia
AVNRT	atrioventricular nodal reentrant tachycardia
ECG	electrocardiogram
EPS	electrophysiological study
ICD	implantable cardioverter defibrillator
PVC	premature ventricular contraction
RFA	radiofrequency ablation
RV	right ventricle
RVOT	right ventricular outflow tract
LV	left ventricle
SCD	sudden cardiac death
SDR	sudden death risk
VF	ventricular fibrillation
VT	ventricular tachycardia
WPW	Wolff-Parkinson-White (syndrome)

Corresponding author
Negru G. Alina
Victor Babes University of Medicine and Pharmacy Timisoara, Romania
E-mail: *alinanegru@umft.ro*
Phone: +40746239777

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