ECG - Checklists for Sports Cardiology Evaluation

(Löllgen, Houston, 2018)

Cardiomyopathies

- HCM, NC CMP, DCM
- ARVD

Marfansyndrome

- Electrical diseases
- Long-QT Syndrom
- BrugadaSyndrom
- CPVT

Why Checklists: Young Males ECG: Six percent with abnormal ECG Reduce Sudden death !! (Seattle Criteria)

	Nbr of Detections	Percentage [%]
Total number of analysed resting ECGs	274'468	100.00
Abnormal ECG finding	17'765	6.47
T-wave inversion	1'193	0.43
ST segment depression	3'080	1.12
Pathologic Q waves	5'947	2.17
Complete left bundle branch block	92	0.03
Intraventricular conduction delay	385	0.14
Left axis deviation	2'800	1.02
Left atrial enlargement	1'709	0.62
Right ventricular hypertrophy pattern	991	0.36
Ventricular pre-excitation	71	0.03
Long QT interval	1'412	0.51
Short QT interval	6	0.00
Brugada-like ECG pattern	0	0.00
Profound sinus bradycardia	0	0.00
Atrial tachyarrhythmias	1'599	0.58
Premature ventricular contractions	416	0.15
Ventricular arrhythmias	126	0.05
Other abnormality	162	0.06

Table 2 Absolute and relative numbers of Seattle Criteria detections.

(N= 440000,Swiss Army) (Abaecherli et al.,2014)

Goals of Examination:

Detection of Occult Diseases (Priori,2013,2015)

>>> Structural („electrical“) abnormalities (Priori,2014)

- **Ion channel diseases**: long and short QT-syndrome, Brugada-syndrome, early repolarisation
- **Preexcitation Syndrom** (WPW) with intermitt.atrial tachycadia and esp. atrial fibrillation),
- **CPVT** Catecholaminergic polymorphic ventricular tachykardia
- Progressive **cardiac conduction disease**, unexplained cardiac arrest (idiopathic VF)
- **Unexplained cardiac arrest:** Sudden unexplained death syndrom and sudden unexplained death in infancy

- **>>> Acquired diseases : most causes in elderly**

  Coronary artery disease, arterial hypertension, drugs (TdP), myocarditis, contusio cordis, concussion

Structural Abnormalities of the Heart

> **Structural abnormalities (Heart muscle)**

- **Cardiomyopathies:** hypertrophic *, with or without outflow obstruction, dilatative, non- compaction cm, arrhythmogen (ARVD), **(„Moges)**
- Marfan-Syndrome,
- Abnormal findings more in Afro-Americans and in Basketball players

(e.g. Marfan-Syndrom) (ECG abnormal in 3.7 %,pot. lethal 0.3%, (Asif, BJSM 2014)

CAD, Coronary abnormalities

*n =750000 with HCM in US, diagnosed in 100000 of them,

mostly non-invasive imaging (Maron, NEJM, Aug. 2018)

„Electrical“ causes of cardiac problems in athletes

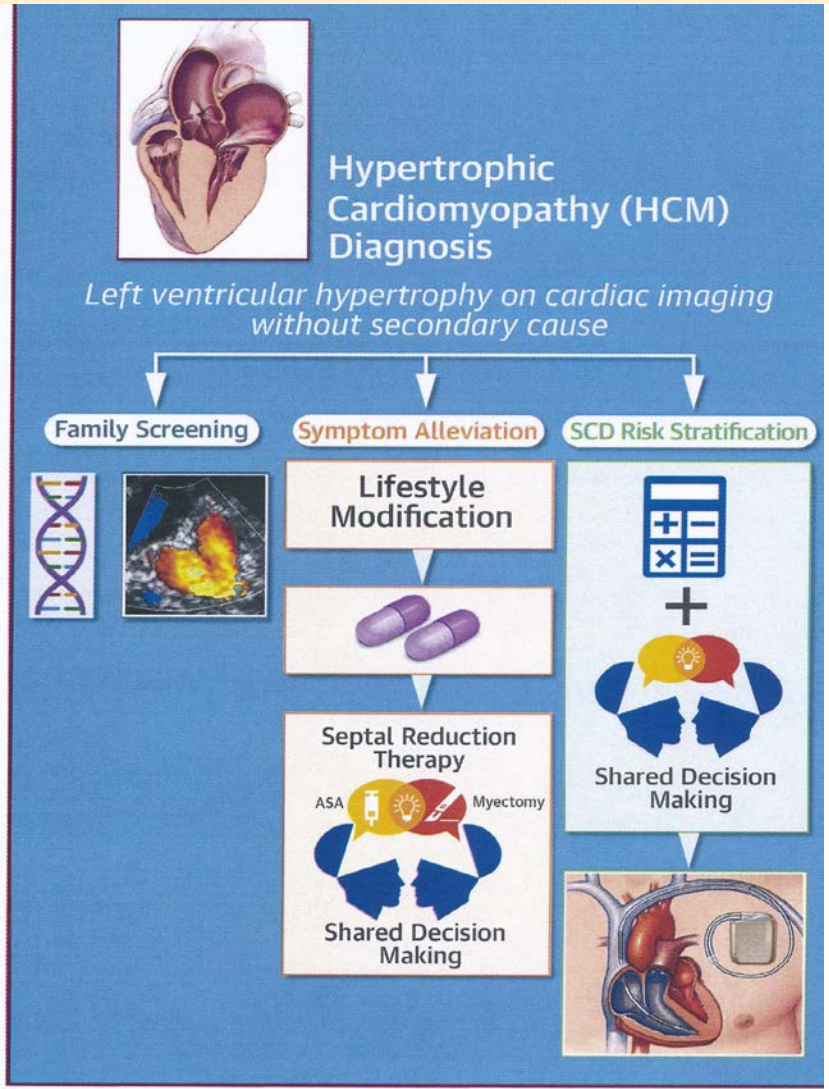
Ion- Chanel diseases or congenital arrhythmogenic diseases

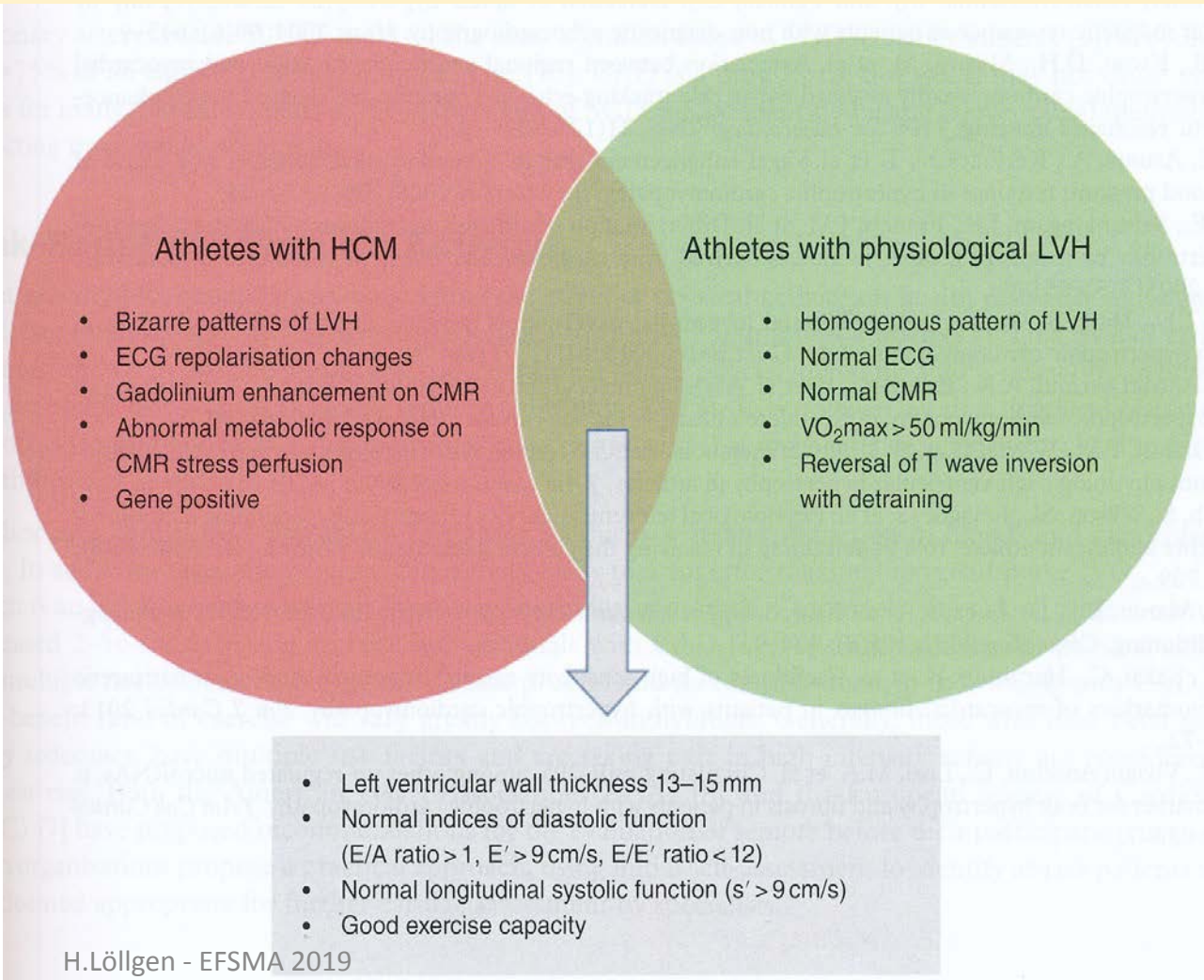
- Long - QT-Syndrome (LQT1 – 8) :
  congenital, acquired (drugs)
  (z.B. Jervell-Lange-Nielsen-Syndrom) (JLNS 1 – 2)
- Short QT-Syndrome (SQTS 1 -3)
  Brugada –Syndrome (BrS 1 – 3)
- Catecholaminergic polymorph ventricular tachycardia
  (CPVT 1- 2)

Conduction disturbancies

HCM Diagnosis

Geske et al., JACC HF,2018





HCM : Checklist

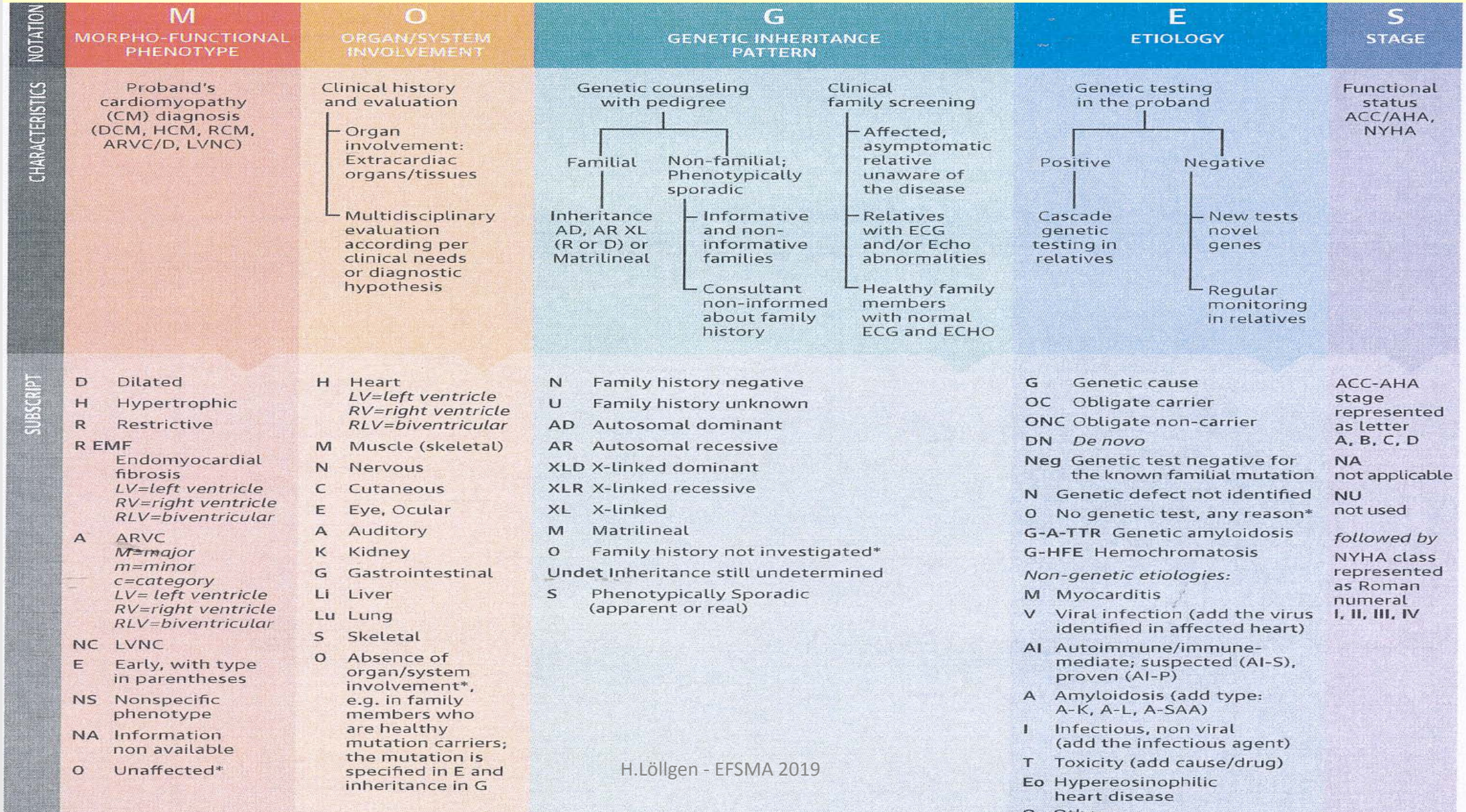
Prinz et al.,Dtsch Ärztebl Int 2011

Algorithm to identify at-risk HCM patients for implantation with an ICD 25

First degree risk factors	Definition
Positive family history of sudden cardiac death	Cases with SCD <45 years
Recurrent syncope	≥ 2 incidents
LVH	≥ 30 mm at any site in the LV
Abnormal blood pressure response during exercise	Increase <20 mm hg or fall >20 mm Hg after transient increase
Non-sustained VT in Holter ECG	≥ 3 consecutive QRS complexes with a heart rate of ≥ 120 bpm.
Second degree risk factors	
Atrial fibrillations/atrial flutter LA dilatation High LVOT gradient at rest Evidence of myocardial ischemia during exercise	Any form, provided cannot be eliminated >45 mm (in m-mode ECG) >80 mm Hg (CW Doppler)
Early manifestation of HCM Myocardial bridging near the LAD Marked fibrosis in cardiac MRI	<30 years of age in younger patients (<45 years) fibrosis of ≥ 2 segments in a 17-segment model of the LV

SCD, sudden cardiac death; LVH, left ventricular hypertrophy; LA, left atrium; LV, left ventricle; LVOT, left ventricular outflow tract; VT, ventricular tachycardia; LAD, left anterior descending artery

Moge(s) Nosology System for Classifying CM Patients (Arbustini, 2016)



ECG in Athletes vs. HOCM

Rachel.E.Bent et al., JACC 2015

TABLE 1 Optimal Electrocardiography Characteristics

		Hypertrophic Cardiomyopathy		Athlete		Multivariate Regression	
	Points	% With Characteristic	% With Isolated Characteristic	% With Characteristic	% With Isolated Characteristic	Odds Ratio	p Value
TWI <0 mV in V_4, V_5, V_6	1	37.9	8.3	0.80	0.71	87.1	<0.001
TWI < -0.5 mV in V_4, V_5, V_6 (major)	2	12.1	1.3	0	0		
QTc >480 ms	1	14.6	1.7	0.44	0.44	3.9	0.41
Q-wave >40 ms in V_5 or aVF	1	6.3	2.1	0.18	0.18	4.6	0.40
ST-segment depression <-0.05 mV V_5 or V_6	1	33.3	4.2	0.27	0.18	13.4	0.033
ST-segment depression <-0.1 mV V_5 or V_6 (major)	2	15.4	0.83	0	0		
Left atrial abnormality	1	14.2	2.5	1.2	1.2	4.2	0.13
Left axis deviation	1	16.3	5.0	0.80	0.80	22.9	0.003
Right axis deviation	1	4.2	2.5	0.36	0.36	17.0	0.025
QRS duration >140 ms	1	12.5	2.5	0.089	0.089	9.3	0.18
Left bundle branch block (major)	2	5.9	0.83	0	0		

The multivariate regression model was adjusted for age, sex, ethnicity, heart rate, and all of the other electrocardiography characteristics in the first column.

TWI = T-wave inversion.

In support „of the digital ECG“ in athletic screening“

Checklist: Criteria for ARVD: Major and Minor

S.Priori et al.,ESC Guideline, 2015

Diagnostic criteria[edit]

There is no pathognomonic feature of ARVD. The diagnosis of ARVD is based on a combination of major and minor criteria. To make a diagnosis of ARVD requires either 2 major criteria *or* 1 major and 2 minor criteria *or* 4 minor criteria.

Major criteria

- Right ventricular dysfunction
- Severe dilatation and reduction of RV ejection fraction with little or no LV impairment
- Localized RV aneurysms
- Severe segmental dilatation of the RV
- Tissue characterization
- Fibrofatty replacement of myocardium on endomyocardial biopsy
- Conduction abnormalities
- Epsilon waves in $V_1 - V_3$
- Localized prolongation (>110 ms) of QRS in $V_1 - V_3$
- Family history
- Familial disease confirmed on autopsy or surgery

Minor criteria

- Right ventricular dysfunction
- Mild global RV dilatation and/or reduced ejection fraction with normal LV.
- Mild segmental dilatation of the RV
- Regional RV hypokinesis
- Tissue characterization
- Conduction abnormalities
- Inverted T waves in V_2 and V_3 in an individual over 12 years old, in the absence of a right bundle branch block (RBBB)
- Late potentials on signal averaged EKG.
- Ventricular tachycardia with a left bundle branch block (LBBB) morphology
- Frequent PVCs (> 1000 PVCs / 24 hours)
- Family history
- Family history of sudden cardiac death before age 35
- Family history of ARVD

In Detail : Task Force Criteria for ARVD

Major and Minor Criteria

(NEJM,2017, D'Ascenzi et al., JACC,2018)

Table 1. International Task Force Criteria for the Diagnosis of Arrhythmogenic Right Ventricular Cardiomyopathy.*

Category	Major Criteria	Minor Criteria
Global or regional dysfunction and structural alteration†		
On two-dimensional echocardiography	Regional RV akinesia, dyskinesia, or aneurysm and one of the following (end diastole): PLAX RVOT ≥32 mm (≥19 mm per square meter when corrected for body-surface area), PSAX RVOT ≥36 mm (≥21 mm per square meter when corrected for body-surface area), or fractional area change of ≤33%	Regional RV akinesia or dyskinesia and one of the following (end diastole): PLAX RVOT 29 to <32 mm (16 to <19 mm per square meter when corrected for body-surface area), PSAX RVOT 32 to <36 mm (18 to <21 mm per square meter when corrected for body-surface area), or fractional area change of 34 to 40%
On MRI	Regional RV akinesia or dyskinesia or dyssynchronous RV contraction and one of the following: ratio of RV end-diastolic volume to body-surface area ≥110 ml per square meter (male patients) or ≥100 ml per square meter (female patients), or RV ejection fraction ≤40%	Regional RV akinesia or dyskinesia or dyssynchronous RV contraction and one of the following: ratio of RV end-diastolic volume to body-surface area 100 to <110 ml per square meter (male patients) or 90 to <100 ml per square meter (female patients), or RV ejection fraction 41 to 45%
On RV angiography	Regional RV akinesia, dyskinesia, or aneurysm	
Tissue characterization	<60% residual myocytes on morphometric analysis (or <50%, if estimated) and fibrous replacement of the RV free-wall myocardium, with or without fatty replacement of tissue, in at least one endomyocardial-biopsy sample	60 to 75% residual myocytes, on morphometric analysis (or 50 to 65%, if estimated) and fibrous replacement of the RV free-wall myocardium, with or without fatty replacement of tissue, in at least one endomyocardial-biopsy sample
Repolarization abnormalities	Inverted T waves in right precordial leads (V_1, V_2, and V_3) or beyond in patients older than 14 yr of age (in the absence of complete right bundle-branch block, QRS ≥120 msec)	Inverted T waves in leads V_1 and V_2 in patients older than 14 yr of age (in the absence of complete right bundle-branch block) or in V_4, V_5, or V_6; inverted T waves in leads V_1, V_2, V_3, and V_4 in patients older than 14 yr of age (in the presence of complete right bundle-branch block)
Depolarization and conduction abnormalities	Epsilon wave (reproducible low-amplitude signals from end of QRS complex to onset of the T wave) in the right precordial leads (V_1, V_2, and V_3)	Late potentials on signal-averaged ECG in at least one of three parameters in the absence of a QRS complex duration of ≥110 msec on the standard ECG; filtered QRS complex duration, ≥114 msec; duration of terminal QRS complex <40 μV (low-amplitude signal duration), ≥38 msec; root-mean-square voltage of terminal 40 msec, ≤20 μV; terminal activation duration of QRS complex, ≥55 msec, measured from the nadir of the S wave to the end of the QRS complex, including R′, in V_1, V_2, or V_3, in the absence of complete right bundle-branch block
Arrhythmias	Nonsustained or sustained ventricular tachycardia with a left bundle-branch block and superior axis pattern (negative or indeterminate QRS complex in leads II, III, and aVF and positive QRS complex in lead aVL)	Nonsustained or sustained ventricular tachycardia of RV outflow configuration with a left bundle-branch block and inferior axis pattern (positive QRS complex in leads II, III, and aVF and negative QRS complex in lead aVL) or unknown axis, or >500 ventricular extrasystoles per 24 hr (on Holter monitoring)
Family history	ARVC confirmed in a first-degree relative who meets current task-force criteria, ARVC confirmed pathologically at autopsy or surgery in a first-degree relative, or identification of a pathogenic mutation categorized as associated or probably associated with ARVC in the patient under evaluation‡	History of ARVC in a first-degree relative in whom it is not possible or practical to determine whether current task-force criteria are met, premature sudden death (at <35 yr of age) due to suspected ARVC in a first-degree relative, or ARVC confirmed pathologically or by current task-force criteria in a second-degree relative

* The table is adapted from Marcus et al.[49] The diagnosis of arrhythmogenic right ventricular cardiomyopathy (ARVC) is considered to be definite if the patient meets two major criteria, one major and two minor criteria, or four minor criteria from different categories; the diagnosis is considered to be borderline if the patient meets one major and one minor criteria or three minor criteria from different categories, and the diagnosis is classified as possible if the patient meets one major or two minor criteria from different categories. ECG denotes electrocardiogram, PLAX parasternal long-axis view, PSAX parasternal short-axis view, RV right ventricular, and RVOT RV outflow tract.

† Hypokinesia is not included in this or subsequent definitions of RV regional wall-motion abnormalities for the proposed modified criteria.

‡ A pathogenic mutation is a DNA alteration associated with ARVC that alters or is expected to alter the encoded protein, is unobserved or rare in a large, non-ARVC control population, and either alters or is predicted to alter the structure or function of the protein or has shown linkage to the disease phenotype in a conclusive pedigree (i.e., a pedigree providing conclusive evidence of a mendelian inheritance of the disease phenotype).

ARVD: Clinical Indicators … Differentiation between Athletic Remodeling and ARVD (Zaidi et al.,JACC 2015)