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REVIEW ARTICLE

Utility of the Exercise Electrocardiogram Testing in Sudden Cardiac Death Risk Stratification

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Background: Sudden cardiac death (SCD) remains a major public health problem. Current established criteria identifying those at risk of sudden arrhythmic death, and likely to benefit from implantable cardioverter defibrillators (ICDs), are neither sensitive nor specific. Exercise electrocardiogram (ECG) testing was traditionally used for information concerning patients' symptoms, exercise capacity, cardiovascular function, myocardial ischemia detection, and hemodynamic responses during activity in patients with hypertrophic cardiomyopathy.

Methods: We conducted a systematic review of MEDLINE on the utility of exercise ECG testing in SCD risk stratification.

Results: Exercise testing can unmask suspected primary electrical diseases in certain patients (catecholaminergic polymorphic ventricular tachycardia or concealed long QT syndrome) and can be effectively utilized to risk stratify patients at an increased (such as early repolarization syndrome and Brugada syndrome) or decreased risk of SCD, such as the loss of preexcitation on exercise testing in asymptomatic Wolff-Parkinson-White syndrome.

Conclusions: Exercise ECG testing helps in SCD risk stratification in patients with and without arrhythmogenic hereditary syndromes.

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electrocardiography; electrophysiology; exercise electrocardiogram testing; sudden cardiac arrest; sudden cardiac death; risk stratification

Sudden cardiac death (SCD) remains a major public health problem, accounting for up to 450,000 deaths annually in the United States and up to 15% of overall mortality.[1] The magnitude of SCD exceeds the total number of deaths from the acquired immunodeficiency syndrome (AIDS), breast cancer, lung cancer, and stroke annually.[2] Risk stratification for SCD remains a public health challenge and current established criteria identifying those at risk of sudden arrhythmic death, and likely to benefit from implantable cardioverter defibrillators (ICDs), are neither sensitive nor specific.[3,4] Only left ventricular ejection fraction is currently clinically used for ICD implantation as a primary prevention in patients without arrhythmogenic hereditary syndromes. Exercise electrocardiogram (ECG) testing was traditionally used for information concerning patients' symptoms, exercise capacity, cardiovascular function, myocardial ischemia detection, and hemodynamic responses during activity in patients with hypertrophic cardiomyopathy. This review will present studies that looked at various parameters in exercise ECG testing to help identify those at risk of SCD and will discuss the utility of the exercise ECG testing in SCD risk stratification in patients with and without arrhythmogenic hereditary syndromes.

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VENTRICULAR PREMATURE BEATS DURING EXERCISE ECG TESTING

In routine exercise, ECG testing of 2885 Framingham Heart offspring participants free of cardiovascular disease, 792 participants (27%) developed exercise ventricular premature beats (EVPBs) with a median value of 0.22 VPB per minute of exercise (1 EVPB for every 4.5 minutes of exercise).[5] Subjects were classified as infrequent EVPBs (VPBs at or below the median value) or frequent EVPBs (VPBs above median value).[5] The age-adjusted 15-year incidences of death were 4.21% (95% CI: 3.33–5.09) for the group with no EVPBs, 7.03% (95% CI: 4.62–9.44) for participants with infrequent EVPBs, and 8.27% (95% CI: 5.67–10.88) for individuals with frequent EVPBs.[5] All-cause adjusted mortality hazards ratios were 1.86 (95% CI: 1.24–2.79) for infrequent, and 1.71 (95% CI: 1.18–2.49) for frequent EVPBs versus no EVPBs.[5] The association of EVPBs to mortality risk was not influenced by VPB grade, presence of VPBs in the recovery period, left ventricular dysfunction, or an ischemic ST-segment response. Both infrequent and frequent EVPBs were associated with a 60%–80% increased risk of death from all causes.[5] Neither infrequent nor frequent EVPBs were related to the risk of coronary heart disease (CHD) events (recognized myocardial infarction, coronary insufficiency, or CHD death).[5]

In a study of 1460 patients with intermediate pretest probability of coronary artery disease (CAD), 10% developed exercise-induced ventricular arrhythmia (VA).[6] Using exercise echocardiography for cardiac death risk assessment, almost half of those with VA (48%) had abnormal echocardiographic findings (vs. 29% in group of VA-free patients) and greater prevalence of ischemia (39% vs. 22%).[6] In a similar study assessing exercise-induced VA in suspected CAD patients, Marieb et al. analyzed its prognostic significance in 383 patients undergoing both exercise thallium 201 stress test and cardiac catheterization.[7] Patients who developed VAs (162 patients, 42% of the study population), had greater incidence of redistribution and CAD.[7] These studies showed that exercise-induced VA in patients with suspected CAD is associated with greater risk of cardiac death and was a significant independent predictor factor.[6,7]

In addition, Gimeno et al. studied exercise-induced VA in hypertrophic cardiomyopathy. Exercise-induced VA was found to be associated with 2.82-fold increase risk of SCD or ICD discharge.[8]

A summary of the prognostic significance of VPBs during the exercise test and the recovery phase is presented in Table 1.

POSTEXERCISE HEART RATE RECOVERY AND POSTEXERCISE/RECOVERY VPBs

Decreased parasympathetic activity and abnormal vagal activation during recovery after exercise testing lead to an abnormal heart rate recovery (HRR) and the presence of severe ventricular ectopy during recovery. These markers of autonomic disturbance have been shown to be associated with a higher risk of all-cause mortality but not SCD.

Immediately after graded exercise, the heart rate (HR) normally falls in a biphasic manner, with an initial rapid decline occurring during the first 30 seconds to 1 minute of recovery.[9] HRR has been shown to be predictive of mortality; 1-minute HRR <18 beats per minute was shown to be associated with a markedly increased risk of all-cause death in the general population.[9] Abnormality in parasympathetic tone is postulated to play a major role in abnormal HRR.[9,10] In a study analyzing 5438 patients without history of heart failure or valvular disease undergoing stress echocardiography, 15% had an abnormal HRR (<18 beats per minute).[9] An abnormal HRR was shown to be independently predictive of death with a two-fold increase in all-cause mortality (adjusted hazard ratio (HR) = 2.1; 95% CI: 1.5–2.8; $P < 0.0001$) but not SCD.[9]

Another reflection of a decreased parasympathetic activity is the presence of ventricular ectopy during the recovery period of the exercise ECG stress test.[10] The occurrence of severe frequent VPBs during the first 5 minutes of recovery after exercise has been linked to all-cause mortality (but not SCD) in patients without and with heart failure and/or CAD.[10] In a study of 2132 heart failure patients with left systolic ejection fraction (LVEF) less than 35%, 290 patients (14%) had frequent ventricular ectopy in the recovery from treadmill exercise testing, of whom 140 (48%) had severe ventricular ectopy in recovery.[11] Frequent ventricular ectopy was defined as the presence of 7 or more ventricular premature beats/min, frequent ventricular couplets, ventricular bigeminy

Table 1. Prognostic Significance of VPBs during the Exercise Test and the Recovery Phase

Population	Variable	Prognosis	Study
General population	VPBs	VPBs during exercise cause 60%–80% increase in risk of death of all causes	Meibodi et al.[5]
		VPBs during 5-minute recovery increase all-cause mortality but not SCD	Goldberger et al.[10]
Intermediate pretest probability of CAD	Exercise-induced VA	Greater prevalence of segmental wall motion abnormalities and ischemia on echocardiography	Elhendy et al.[6]
		Greater incidence of redistribution on Thallium stress test and increased risk of SCD	Marieb et al.[7]
Hypertrophic cardiomyopathy	Exercise-induced VA	2.82-fold increase in SCD and ICD discharge	Gimeno et al.[8]
Heart failure	Ventricular ectopy during the recovery phase	Ventricular ectopy during recovery is associated with an increase in all cause mortality	O'Neill at al.[11]
SUDS family members	Inappropriate QT prolongation, dynamic Brugada pattern, ventricular ectopy during exercise testing	Anormal QT prolongation and dynamic Brugada pattern were diagnostic of long QT syndrome and BrS, respectively. In one third of patients with ventricular ectopy phenotypic cardiomyopathy or channelopathy was demonstrated on further investigations	Raju et al.[35]

or trigeminy, or any other form of ventricular tachycardia (either monomorphic or polymorphic) or ventricular fibrillation (VF).[11] Severe ventricular ectopy was defined as the presence of ventricular triplets, sustained or nonsustained ventricular tachycardia, ventricular flutter, polymorphic ventricular tachycardia, or VF.[11] The study showed that severe frequent ventricular ectopy in recovery was predictive of all-cause mortality (adjusted HR = 1.48; 95% CI: 1.10–1.97; P = 0.0089) after adjustment for baseline clinical and cardiovascular variables.[11] Blunted HR responses to exercise and abnormal hyperventilation in some heart failure patients have been shown to be prognostically important manifestations of autonomic dysfunction.[11]

In a large cohort study on more than 29,000 patients referred for exercise testing, frequent ventricular ectopy, defined as seven or more ventricular premature beats per minute, was evaluated during exercise and recovery.[12] Frequent ventricular ectopy during recovery was a single predictor of increased risk of death (HR = 1.6; 95% CI: 1.3–1.9; P < 0.001) and this was after adjustment of ventricular ectopy during exercise.[12]

CATECHOLAMINERGIC POLYMORPHIC VENTRICULAR TACHYCARDIA UNMASKED BY EXERCISE TESTING

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is an inherited arrhythmia syndrome with mutations in three causative genes identified: *RyR2*, which encodes the cardiac ryanodine receptor Ca^{2+} release channel; *CASQ2*, which encodes cardiac calsequestrin; and TRDN, which encodes triadin.[13–17] Exercise ECG testing is often the most useful procedure to establish the diagnosis of CPVT. In fact, exercise ECG testing unmasks the arrhythmia susceptibility of patients with CPVT with a progressive worsening of supraventricular and VAs that parallel the increase in workload during exercise ECG testing. Isolated supraventricular and ventricular ectopies usually start at a HR of 100–110 beats per minute. The complexity and frequency of arrhythmia progressively worsen from isolated premature ventricular beats to bigeminy, couplets, and to runs of ventricular and supraventricular tachycardias. If exercise is not promptly discontinued, ventricular

tachycardia may become faster and progressively more disorganized with potential degeneration into VF. These arrhythmias promptly recede during the recovery phase. A RyR2 mutation in the C-terminal channel-forming domain has an increased odds of nonsustained ventricular tachycardia compared with N-terminal domain. According to the 2013 Heart Rhythm Society (HRS)/European Heart Rhythm Association/Asia Pacific Heart Rhythm Society expert consensus, CPVT is diagnosed in the presence of a structurally normal heart, normal ECG, and unexplained exercise-induced bidirectional VT or polymorphic ventricular premature beats or VT in an individual <40 years of age. CPVT is diagnosed in family members of a CPVT index case with a normal heart who manifest exercise-induced premature ventricular contractions or bidirectional/polymorphic VT. CPVT can be diagnosed in the presence of a structurally normal heart and coronary arteries, normal ECG, and unexplained exercise-induced bidirectional VT or polymorphic ventricular premature beats or VT in an individual >40 years of age.[18]

Exercise ECG testing is also included as part of the evaluation of sudden cardiac arrest to assess for CPVT.[19] The first VT complex in CPVT most commonly originates in the right ventricular outflow tract. The American College of Cardiology/American Heart Association (AHA)/European Society of Cardiology guidelines currently support exercise ECG testing as a class I (level of evidence: B) indication, regardless of age, in patients with known or suspected exercise-induced ventricular, including catecholaminergic VT, to provoke the arrhythmia, achieve a diagnosis, and determine the patient's response to tachycardia.[20]

EXERCISE ECG TESTING IN RELATIVES OF PATIENTS WITH IDIOPATHIC VF, SUDDEN UNEXPLAINED DEATH SYNDROME, AND SUDDEN UNEXPECTED DEATH IN INFANCY

The HRS/European Heart Rhythm Association/Asian Pacific Heart Rhythm Society Expert Consensus currently support exercise ECG testing as a class I indication for the evaluation of first-degree relatives of all idiopathic VF and sudden unexplained death syndrome (SUDS) victims and the assessment of first-degree relatives with history of palpitations, arrhythmias, or syncope should be prioritized.[18] The HRS/EHRA/APHRS Expert Consensus currently support exercise ECG testing as a class IIa indication for the evaluation of first-degree relatives of sudden unexpected death in infancy (SUDI) with a family history of inherited heart disease or other SUDS or SUDI deaths and the assessment of first-degree relatives with history of arrhythmias or syncope should be prioritized.[18] The HRS/EHRA/APHRS Expert Consensus currently support exercise ECG testing as a class IIb indication for the evaluation of first-degree relatives of SUDI victims.[18]

EXERCISE TESTING IN THE RISK STRATIFICATION OF WPW FOR SCD

The prevalence of Wolff–Parkinson–White (WPW) syndrome is estimated to be 1–3 in 1000 individuals. Patients with WPW syndrome present with palpitations or presyncope caused by an atrioventricular reciprocating tachycardia or, less commonly, a primary atrial tachycardia. Rapid conduction of atrial fibrillation (AF) over the accessory pathway resulting in VF is rare but unfortunately may be the first manifestation of WPW syndrome.[21] The incidence of intermittent preexcitation in WPW using ambulatory monitoring was as high as 67%.[22] Exercise ECG testing has a useful role in unmasking intermittent preexcitation if intermittent preexcitation is not observed in ambulatory monitoring. In one study, abrupt disappearance of preexcitation during stress testing occurred in only 15% of a predominantly pediatric group of patients.[23] An abrupt loss of preexcitation during exercise stress test was a highly reliable noninvasive marker of nonrapid accessory pathway at baseline and a low risk for SCD.[23] According to the Pediatric and Congenital Electrophysiology Society (PACES)/Heart Rhythm Society (HRS)/American College of Cardiology Foundation (ACCF)/AHA/American Academy of Pediatrics (AAP)/Canadian Heart Rhythm Society (CHRS), an exercise stress test for young asymptomatic patient with WPW ECG pattern is a reasonable component of the evaluation if the ambulatory ECG exhibits persistent preexcitation (class IIa, levels of evidence b/c).[21] In patients with clear and abrupt loss of preexcitation at physiological

HRs, the accessory pathway properties pose a lower risk of sudden death. The 2012 PACES/HRS Expert Consensus Statement included an algorithm for managing WPW asymptomatic patients based on preexcitation and measurement of the shortest preexcited RR interval during induced AF.[21] Patients with asymptomatic WPW and with abrupt loss of preexcitation during exercise stress test do not need further electrophysiology testing and ablation procedure.

RECOVERY PHASE QTc SIGNIFICANCE IN THE DIAGNOSIS OF LONG QT SYNDROME

Long QT syndrome (LQTS) affects approximately 1 in 2,500 individuals and can present with syncope, seizures, and sudden death. Genetic testing for 13 LQTS susceptibility genes identifies the pathogenic substrate for about 80% of LQTS. At least one-third of genetically positive LQTS patients have a normal to borderline QTc measurement at rest (concealed LQTS). Many noninvasive tests are helpful in borderline LQTS such as standing ECG (looking for QT stunning [$QT_{standing} - QT_{supine}$]), exercise ECG testing (looking for QT hysteresis [$QT_{exercise} - QT_{recovery}$]) and epinephrine QT stress testing. These tests help unmask patients with concealed LQTS. Before exercising the patient with suspected LQTS and after a supine ECG is done to obtain a baseline QTc, it is important to do a standing ECG and assess the QTc. An abnormal response of QTc prolongation to HR upon standing that persists even after the HR slows back to baseline (QT stunning) helps in distinguishing patients with LQTS from healthy controls.[24]

In a study to determine diagnostic significance of peak exercise and recovery phase QTc parameters in patients with one of the three most common LQTS genotypes (LQT1, LQT2, or LQT3), 243 patients were genetically tested by Horner et al., 155 patients (64%) had LQT1–LQT3 associated mutations, whereas 88 (36%) had negative genetic tests.[21] Of the 155 patients with a positive genetic test, 82 had LQT1 (53%), 55 LQT2 (35%), and 18 LQT3 (12%). Fifty percent of the LQT1, 40% of the LQT2 (22/55), and 28% of the LQT3 (5/18) patients were concealed at rest, defined as resting QTc <460 milliseconds.[25] No significant difference between the resting QTc of the 88 patients with negative genetic tests (controls) and concealed LQT1, concealed LQT2, and concealed LQT3 patients. Patients with concealed LQTS, regardless of genotype, had paradoxical lengthening in QTc compared with the controls ($P < 0.0001$) during the first 5 minutes of recovery phase of the exercise electrogram testing, although the response was most pronounced in concealed LQT1. In patients with LQT1 and concealed LQT1, QTc lengthened significantly more than controls, LQT2, and LQT3 patients ($P < 0.0001$) during the first 5 minutes of recovery.[21] Exercise testing was also shown to unmask concealed LQTS, particularly concealed LQT1, even in the presence of beta-blockers that are known to obscure the LQT1 diagnostic profile during the epinephrine QT stress test.[25]

These findings by Horner et al. were similar to a previous study done by Chattha et al. on 60 patients separated into 3 groups: LQT1, LQT2, and a control group consisting of patients with the lowest probability of LQTS as determined by the Schwartz score and absence of LQTS mutations.[26] The QT interval increased as the RR interval increased in a linear manner in all three groups during the recovery phase.[26] QT adaptation was then studied in two phases: early recovery (134–100 bpm) and late recovery (100–67 bpm).[26] This study showed that QTc prolongation of LQT1 group peaked in early recovery, after which it gradually deceased into late recovery (80% LQT1 recorded their first recovery QTc measurement above 460 milliseconds).[26] LQT2 patients had QTc intervals that increased as the recovery period progressed. LQT1 subtype experiences the greatest QTc prolongation and cardiac events during heightened sympathetic states such as exercise and in early recovery phase while sympathetic hormones continue to exert their effect.[26] Again, patterns of QTc response were observed regardless of beta-blocker usage.

Exercise ECG testing has also been used in the evaluation of QTc prolongation in asymptomatic first-degree relatives of patients with LQTS. In a study of 69 asymptomatic first-degree relatives of genetically characterized LQT1 and LQT2 syndrome probands, 28 subjects had LQT1, 20 had LQT2, and 21 are noncarriers. QTc prolongation was seen in both LQT1 and LQT2 in early exercise, but at peak exercise, QTc prolongation is persistent in LQT1, whereas it normalizes in LQT2.[27] LQTS had exaggerated QTc prolongation during exercise

and recovery.[27] Late recovery (4 minutes after exercise) QTc prolongation was shown to be a sensitive and specific marker for LQT1 and LQT2, whereas QTc prolongation during early recovery is specific to LQT1.[27] Based on these findings, an algorithm was proposed for noninvasive risk stratification of first degree relatives of patients with LQTS. First, QTc at rest is evaluated; if overtly abnormal (>480 ms in women and >470 ms in men), patients are diagnosed as LQTS carriers with 100% specificity.[27] Patients with normal or borderline QTc prolongation at rest would undergo exercise test and QTc would be evaluated at 1-minute recovery and 4-minute recovery.[27] Abnormal QTc at 4-minute recovery means patients are probable LQTS carriers. In these subjects, 1-minute recovery is examined to differentiate between LQT1 and LQT2 (as LQT1 will have QTc>460 milliseconds whereas LQT2 carriers will have QTc <460 milliseconds).[27]

EARLY REPOLARIZATION RESPONSE TO EXERCISE AND RISK OF SCD

Early repolarization (ER), characterized by elevation of the QRS–ST junction (J point) in leads other than V_1–V_3 on ECG, had been suspected as a possible predicator for serious VAs and has emerged as a risk marker for SCD.[28,29] ER syndrome is diagnosed in the presence of J-point elevation $\geq$ 1 mm in $\geq$ 2 contiguous inferior and/or lateral leads of a standard 12-lead ECG in a patient resuscitated from otherwise unexplained VF/polymorphic VT. Exercise ECG testing can unmask high-risk patients with ER pattern who develop exercise-induced polymorphic VT.[30] Specifically, ER with horizontal/descending ST-segment morphology in the inferior and inferolateral leads was associated with idiopathic VF and conferred a 3-fold increased risk of arrhythmic death.[28] ER with rapidly ascending ST segment anterolaterally has been associated with athletic status.[28] Bastiaenen et al. screened 229 patients with history of aborted SCD, sustained symptomatic VA, unexplained syncope, and/or family history of sudden death but no definitive cardiac diagnosis following comprehensive clinical evaluation; 26 patients out of the 229 patients had baseline ER.[28] In this study, ER studied at baseline and peak exercise was stratified according to ST segment, territory, and degree of J-point elevation in 26 patients who underwent ajmaline provocation testing and 21 patients who were subjected to exercise tolerance testing (ETT). Exercise data were available for 21 of 26 patients with baseline ER. The pretest ECG demonstrated ER in only 18 of 21 of these (86%; 12 men).[28] Ajmaline provocation and ETT led to disappearance of all lateral ER and ER in all patients with rapidly ascending ST segment. In patients who had horizontal/descending ST segment at baseline, 40% ER persisted during ajmaline provocation and 75% ER persisted during ETT. Persistent inferior ER was observed in 44% of patients during ajmaline provocation and in 40% during ETT.[28] Patients with persistent ER during exercise were more likely to be symptomatic (due to aborted SCD, sustained symptomatic VA, and/or unexplained syncope) than patients with diminished ER during exercise and this were mainly due to unexplained syncope than those in whom ER diminished during exercise.[28]

This study is small and needs to be evaluated prospectively on a larger number of patients to assess the utility of exercise electrocardiography testing in ER syndrome.

AUGMENTED ST ELEVATION DURING RECOVERY FROM EXERCISE IN PATIENTS WITH BRUGADA SYNDROME

Brugada syndrome (BrS), defined by a distinct ECG pattern in precordial leads (V_1–V_3) presenting coved-type ST-segment elevation, is a marker of loss of function SCN5A sodium channel mutations that lead to SCD in middle-aged persons due to VF.[31] Mokimoto et al. studied 93 patients with BrS and 102 healthy control subjects during recovery phase from treadmill exercise testing.[32] Twenty-two patients had previous documented VF, 35 had history of syncope alone and 36 were asymptomatic among BrS patients. Significant augmentation of ST-segment elevation with coved pattern at early recovery phase was observed in 34 BrS patients (37%) and none in the control group.[32] ST-segment amplitude decreased at peak exercise and started to reascend at early recovery, and culminated at 3 minutes of recovery, in contrast to the other BrS patients and control group in which it decreased at peak exercise and gradually returned

to baseline.[32] During 76 ± 38 months follow up, 27% of BrS patients had cardiac events (SCD, VF, sustained ventricular tachyarrhythmias) and cardiac events were significantly higher in patients with augmented ST-segment elevation at early recovery (44% vs. 17%).[32] In addition, asymptomatic patients who had ST-segment augmentation at early recovery had a higher incidence of cardiac events than patients who did not.[32] Thus, exercise electrocardiographic testing assessing ST-segment augmentation at early recovery is useful in the risk stratification of BrS patients.

POSTEXERCISE RECOVERY OF THE SPATIAL QRS-T ANGLE AS A PREDICTOR OF SCD

Spatial divergence between the T wave vector and the QRS vector (Spatial QRS-T angle), a marker that considers both depolarization and repolarization abnormalities, has been shown to be a significant and independent predictor of CV death. Abnormal spatial QRS-T angle carries a six-fold elevated risk for SCD, and may be an even better predictor than T wave amplitude.[33]

Kenttä et al. studied the spatial relationship between the depolarization and repolarization wavefronts (total cosine R-to-T [TCRT]) on 1297 patients during the post–bicycle stress test (post-exercise) recovery phase in the Finnish Cardiovascular Study. Exercise-recovery hysteresis of TCRT was quantified by measuring the TCRT/HR loop area bounded by the exercise and first 3-minute postexercise recovery curves.[34] The HR-corrected TCRT/HR hysteresis was calculated by dividing the area with the HR decrement during the first 3 minutes of recovery.[34] During 45 ± 12 months follow up, 5.7% patients died, 2.6% were cardiac deaths and 1.9% were SCDs.[34] After adjustment for clinical risk factors, TCRT/HR loop area remained an independent predictor of cardiac death with hazard ratio 5.6 ($P < 0.007$) and SCD with hazard ratio 10.7 ($P < 0.024$).[34] Exercise-recovery hysteresis of TCRT might provide prognostic information on cardiac mortality and SCD. However, the parameter might be technically challenging to assess in patients.

CONCLUSIONS

Exercise testing can unmask suspected primary electrical diseases in certain patients (CPVT or concealed LQTS) and can be effectively utilized to risk stratify patients at an increased (such as ER syndrome and BrS) or decreased risk of SCD, such as the loss of preexcitation on exercise testing in asymptomatic WPW syndrome. Current guidelines support exercise ECG testing indication, regardless of age, in patients with known or suspected exercise-induced ventricular, including catecholaminergic VT, to provoke the arrhythmia, achieve a diagnosis, and determine the patient's response to tachycardia. Current guidelines support as well exercise ECG testing in first-degree relatives of patients with idiopathic VF and SUDS. The analysis of recovery phase QTc, repolarization dynamics, and augmentation of ST-elevation during recovery represent promising parameters for risk stratification of SCD but they need validation in other studies with a larger study population.

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