Electrocardiographic Markers of Sudden Cardiac Death (Including Left Ventricular Hypertrophy)

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KEYWORDS

• Arrhythmogenic right ventricular dysplasia/cardiomyopathy • Brugada syndrome
• Depolarization markers • Electrocardiogram • Left ventricular hypertrophy • Repolarization markers
• Sudden cardiac death

KEY POINTS

- The electrocardiogram provides significant information regarding the diagnosis and screening for patients at risk of sudden cardiac death.
- Left ventricular hypertrophy is an underestimated cause of sudden cardiac death that can easily be diagnosed by the electrocardiogram.
- The electrocardiogram also provides specific signs of inheritable cardiac disorders such as arrhythmogenic cardiomyopathy, hypertrophic cardiomyopathy, Brugada syndrome, and others.

MAIN CAUSES OF SUDDEN CARDIAC DEATH

The main cause of sudden cardiac death (SCD) is structural heart disease, mostly atherosclerotic heart disease, which represents approximately 85% of all cases (approximately 280,000 SCD per year in the United States). The remaining 15% is caused by cardiopathies without apparent structural heart disease (approximately 53,000 SCD per year). Besides structural heart disease, the other main cause of SCD is atherosclerotic heart disease, which includes coronary artery disease (CAD), followed by others such as nonischemic dilated cardiomyopathy/dilated cardiomyopathy, hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D), coronary artery anomalies, myocarditis, mitral valve prolapse, symptomatic moderate to severe calcified aortic stenosis (frequently bicuspid), congenital heart diseases before and after surgical correction, commotio cordis or cardiac concussion, and Wolf–Parkinson–White syndrome. Among the causes of SCD without apparent structural heart disease, channelopathies or "primary electrical" diseases stand out, such as Brugada syndrome and sudden unexplained nocturnal death syndrome. Both sudden

Conflict of Interest: None.
Disclosure: The authors do not report any conflict of interest regarding this work.
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Card Electrophysiol Clin 9 (2017) 605–629
http://dx.doi.org/10.1016/j.ccep.2017.07.011

unexplained nocturnal death syndrome and Brugada syndrome are phenotypically, genetically, and functionally the same disorder,[1] idiopathic ventricular fibrillation (VF), early repolarization syndrome/J wave syndrome, congenital or acquired long QT syndrome (LQTS), congenital short QT syndrome, catecholaminergic polymorphic ventricular tachycardia (VT), familial progressive cardiac conduction defect/disorder, or Lenègre disease.[2] Genetic screening and identification of the causal mutation are crucial for risk stratification and family counseling. Also, Lev's disease (acquired complete atrioventricular heart block due to idiopathic fibrosis and calcification of the electrical conduction system of the heart, most common in the elderly, and often described as senile degeneration of the conduction system), familial sick sinus syndrome, overlapping syndromes, short-coupled variant of Torsades de Pointes (TdP) with a normal QT interval or Leenhardt syndrome[3] with or without early repolarization on inferolateral leads,[4] sudden infant death syndrome, sudden unexpected death in infancy, and inborn errors of metabolism.[5]

More than 122 years after the discovery of the standard 12-lead electrocardiogram (ECG) by Willem Einthoven,[6] it remains the most common test that is used in the diagnostic armamentarium of the practicing clinician. Because SCD is a complex, multifactorial syndrome, its pathophysiology and triggers are poorly understood. Because SCD has a multifactorial risk profile, it stands to reason that using multiple risk markers, reflecting different facets of the heart's electrical activity, would convey more information than a single marker. At this time, no individual ECG finding has been found to be able to adequately stratify patients with regard to risk for SCD. However, one or more of these candidate surface ECG parameters may become useful components of future multifactorial risk stratification models.[7]

Currently, there is a trend of decreasing the incidence of fast VT/VF. At the same time, there is an increase in pulseless events (ie, cardiac arrest). More people in heart failure have asystole so defibrillation does not work. **Fig. 1** shows the causes of SCD and distribution of arrhythmias.

ELECTROCARDIOGRAPHIC MARKERS OF SUDDEN CARDIAC DEATH: ROLE OF THE ELECTROCARDIOGRAM AS A PART OF A RISK STRATIFICATION OF SUDDEN CARDIAC DEATH

Electrocardiographic depolarization and repolarization disorder and ECG markers of SCD

I. ECG markers of repolarization disorders in SCD
   1. The QT interval or electric systole
      - Prolonged QT/QT corrected QT (QT_c) interval
      - Short QT–QT_c interval
   2. Prolonged JT–corrected JT (JTc)
   3. Prolonged QT dispersion
   4. Inferolateral early repolarization syndrome/J wave syndrome
   5. Interval from the peak to the end of the T-wave ($T_{peak} - T_{end}$) or Tpe
   6. $T_{peak} - T_{end}$/QT ratio
   7. Macrowave alternans or T wave alternans
   8. Microwave alternans or T wave alternans

II. ECG markers of depolarization disorders in SCD
   1. Prolongation of QRS duration (QRS_d)
   2. QRS prolongation in right precordial leads (from V1 to V3).
   3. An S-wave ($\geq$0.1 mV and/or $\geq$40 ms) in lead I
   4. QRS dispersion
   5. Narrow and wide QRS fragmentation (fQRS and fQRS wide)
   6. Epsilon waves
   7. Presence of ventricular late potentials (LPs) using high-resolution or signal-averaged ECG

Electrocardiographic Markers of Repolarization Abnormalities in Sudden Cardiac Death

The QT interval or electric systole

The QT interval is the interval that extends between the first recognizable part of the QRS complex onset up to the last recognizable portion of the T wave (the latter may be hard to determine accurately). The end of T is defined as the return of the T wave to the T-P baseline. The QT interval represents the time between ventricular (electric) depolarization onset and (electric) repolarization offset (terminal part). Therefore, one should correct the QT interval according to the heart rate, the so-called QTc. Several mathematical formulas have been proposed. The most commonly used formula is the one proposed by Bazett in the 1920s.[8] Bazett's formula uses the QTc measurement divided by the square root of RR:

$$QTc = \frac{\text{Measured QT interval}}{\sqrt{RR}}$$

Bazett's formula correction of QT interval has been criticized because it tends to provide an inappropriately short QTc at low heart rates. Consequently, it is inappropriate for QTc measurements at higher rates. Several formulae have been proposed to correct the QT interval for the physiologic effect of heart rate changes (QTc), but none