The evolving science on sudden cardiac death–the marriage of left ventricular hypertrophy and QT dispersion

james ker[1]

[1]University of Pretoria Faculty of Health Sciences

October 23, 2024

Abstract

The first description of sudden cardiac death was made by Hippocrates in the 4'th century BC1. Such cases of sudden collapse and death has intrigued both the public and medical science for centuries and a practical definition is that sudden cardiac death is the unexpected and natural death from a cardiac cause within a short period of time, usually less than 1 hour from the onset of symptoms, in a person without any known prior condition1,2. Sudden cardiac death (SCD) is clearly the end-result of a wide variety of cardiac conditions—both congenital and acquired. However, the most common mechanism for the event of SCD is ventricular fibrillation1. However this is an evolving field of study and the recent study published by Stojanovic et al3 is of great importance as it links two well known risk factors for SCD—left ventricular hypertrophy (LVH) and QT-dispersion4. The finding by Stojanovic et al3 that septal thickness in both athletes and sedentary men are associated with increased QTd is concerning and future studies need to clarify if we need to keep the septum thin at all costs with more exercise for some and less for others.

The evolving science on sudden cardiac death—the marriage of left ventricular hypertrophy and QT-dispersion.

Professor James Ker

MBChB, MSc, DSc, PhD, LLM, MRCP, FRCP

Department of Internal Medicine

University of Pretoria

Pretoria

South Africa

Tel: +27128092810

E-mail: jker@wol.co.za

The first description of sudden cardiac death was made by Hippocrates in the 4'th century BC[1]. Such cases of sudden collapse and death has intrigued both the public and medical science for centuries and a practical definition is that sudden cardiac death is the unexpected and natural death from a cardiac cause within a short period of time, usually less than 1 hour from the onset of symptoms, in a person without any known prior condition[1,2]. Sudden cardiac death (SCD) is clearly the end-result of a wide variety of cardiac conditions—both congenital and acquired. However, the most common mechanism for the event of SCD is ventricular fibrillation[1].

Understandably, SCD can afflict both the athlete and the non-athlete and is the cause of 13-20% of all deaths in Western countries[2]. In athletes older than 35 years of age atherosclerotic coronary artery disease is the

most common cause of SCD, while primary cardiomyopathies and ion channelopathies are more commonly found in the young athlete with SCD[2]. However this is an evolving field of study and the recent study published by Stojanovic et al[3] is of great importance as it links two well known risk factors for SCD—left ventricular hypertrophy (LVH) and QT-dispersion[4]. The strong association between LVH and overall cardiovascular mortality first emerged from the Framingham heart study[4]. Initially, after this observation, several studies have confirmed the strong association between LVH and cardiovascular mortality, but the specific association with sudden cardiac death (SCD) came later with the Oregon Sudden Unexpected Death Study (Oregon SUDS)- one of the first to confirm the link between LVH and SCD[4,5]. The development of LVH creates various pathways to ventricular arrhythmogenesis, which include ventricular ectopy, in fact every additional millimetre of left ventricular wall thickness increases the risk of ventricular ectopy 2 to 3 fold[4]. LVH is the cause of significant cellular and interstitial remodelling of the myocardium which promotes ventricular arrhythmogenesis from both re-entry and triggered activity[4]. An increase in left ventricular mass (LVH) results in various myocardial alterations resulting in electrical remodelling with resultant prolonged QRS intervals, prolonged OT intervals, interstitial fibrosis with re-entry, sub-endocardial ischemia and an increased sensitivity to pro-arrhythmia due to an increase in left ventricular wall stress[4]. In fact, all forms of left ventricular hypertrophy, concentric, eccentric and even concentric remodelling without hypertrophy are all associated with an increased risk for sudden cardiac death[6].

The QT interval—the interval from the beginning of the QRS complex to the end of the T wave on the surface ECG—represents the period of global ventricular de-and repolarization[7]. Prolongation of this interval is strongly associated with an increased risk of lethal arrhythmia[7]. Regional differences in ventricular repolarization facilitate re-entry and is strongly associated with ventricular arrhythmias[7]. QT dispersion—the difference between the longest and shortest QT interval on the surface ECG—is indicative of such regional differences in ventricular repolarization and numerous publications support this association with lethal arrhythmia[7].Normal QT dispersion (QTd) is between 20-40 ms in normal individuals, with some authors proposing up to 65 ms[8].

It has been shown that LVH in patients with hypertension is associated with an increase in QT dispersion and that both these parameters can be normalised with anti-hypertensive therapy[9].

The finding by Stojanovic et al[3] that septal thickness in both athletes and sedentary men are associated with increased QTd is concerning and future studies need to clarify if we need to keep the septum thin at all costs with more exercise for some and less for others.