QT Peak Prolongation Is Not Associated with Left Ventricular Hypertrophy in Teenage Professional Football Players

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Objective: QT peak prolongation is associated with left ventricular hypertrophy (LVH) in patients with hypertension. This study tests the hypothesis that QT peak prolongation correlates with LV mass index in apparently healthy young football players.

Methods: QT peak and other ECG criteria for LVH were assessed in 117 male professional footballers (mean age 16.4 years $\pm$ SD 0.76). Their left ventricular mass index (LVMI) was assessed by transthoracic echocardiography. Heart rate-corrected QT peak (QTpc) interval was measured in lead I using Bazett's formula. Spearman (2-tailed) test and UNIANOVA was used to assess if there were correlations between QT peak and the various echocardiographic and ECG indices of LVH.

Results: Echocardiographic LVH, defined as $\text{LVMI} \geq 134 \text{ g/m}^2$, was seen in 79 (70.5%) subjects. ECG-defined LVH was present in 54 (50%) players by Sokolow-Lyon criteria, in 19 (16%) players by Romhilt Score, in 5 (4%) players by Cornell voltage criteria, and in 7 (6%) players by Cornell product $>2436 \text{ mm ms}$. There was no correlation between QT peak (QTpc) and LVMI on echocardiography (Spearman $r = 0.058$, 2-tailed $P = 0.54$). In addition, there was no relation between LVH and QTpc of lead I using any of the following ECG criteria: Sokolow-Lyon ($P = 0.6$), Romhilt ($P = 0.3$), Cornell voltage ($P = 0.8$), or Cornell product ($P = 0.6$).

Conclusion: QT peak interval, which is associated with pathological LVH in hypertensive patients and is a measure of risk of cardiac death, does not correlate with LVH characterized by myocyte hypertrophy in young apparently healthy professional footballers. **A.N.E. 2007;12(2):104-110**

QT peak; left ventricular hypertrophy; football players

The Framingham population study has identified left ventricular hypertrophy (LVH) as a powerful predictor of mortality.¹ Subjects with LVH had an especially high incidence of sudden death—up to 10 times of those without LVH. In essential hypertension, a reduction in left ventricular mass during treatment is a favorable prognostic marker that predicts a lesser risk for subsequent cardiovascular morbid events.²

A prolonged QT (end) interval (start of Q to end of T) and QT (end) dispersion is associated with cardiac death.^{3,4} QT (end) dispersion has also been shown to be associated with left ventricular mass index (LVMI) in hypertensive subjects⁵ and can be reduced by treatment.⁶ There is thus evidence of

a close link between increased QT (end) dispersion and LVH in hypertensive subjects.

However, despite the evidence supporting its use, QT dispersion is not routinely measured in clinical practice for two main reasons. Firstly, it is time consuming to work out the QT interval from all 12 leads of the ECG to measure QT dispersion. Secondly, the T end is not always easily identified. The T peak is easier to identify than the T end. For these reasons, the QT peak calculated from just one ECG lead would be a more convenient clinical index.

Long QT peak (measured from a single lead-lead I- of the ECG) has now been shown to be associated with LVH in two separate cohorts of patients;

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hypertensive subjects and patients who were referred for stress echocardiography.^{7,8} Some would argue that LVH has two components: myocyte hypertrophy, which is relatively innocuous, and myocardial fibrosis that is thought to be the pathological dysrhythmogenic component. In the two studies quoted above, one would expect LVH in those cohorts to be characterized by myocardial fibrosis and therefore, QT peak would be expected to be prolonged. What is not known however is whether QT peak would be prolonged in athletic LVH, which is thought to be predominantly due to myocyte hypertrophy. Therefore, in this study, we tested the hypothesis that QT peak measured from a single lead would correlate with LVMI in a large cohort of young professional football players.

A second aim of this study is to address the relative lack of data in the literature regarding normal values of QT peak. This study provided us with the unique opportunity to define normal ranges of QT peak for adolescent athletes.

METHODS

Young football players from six professional teams in England competing in the English Football League were routinely screened for unsuspected cardiovascular disease at The Cardiothoracic Centre in Liverpool. Over 32 months, players were screened and enrolled in the study. Their age, weight, and height were recorded. The body surface area (BSA) was calculated according to the formula: $BSA (m^2) = 0.0001 \times 71.84 \times \text{Weight (kg)}^{0.425} \times \text{Height (cm)}^{0.725}$.

Echocardiography

Transthoracic echocardiography was done using the Hewlett Packard SONOS 5500 ultrasound system with a 2.5 MHz transducer (Hewlett-Packard Inc, Andover, Massachusetts, USA).

LVMI estimations

Intraventricular septal thickness in end-diastole (IVSd), end-diastolic left ventricular internal dimension (LVIDd), and left ventricular posterior wall thickness in end-diastole (PWTd) were measured using M-mode echocardiography, obtained at the level of the tips of the mitral valve from parasternal views. LVMI was calculated using the American Society of Echocardiography (ASE) convention by the following equation:

$$LVMI = \{1.05 \times [(LVIDd + PWTd + IVSd)^3 - (LVIDd)^3]g\} / BSA.^{9,10}$$

LVH was defined as $LVMI \geq 134 \text{ g/m}^2$ in men.¹¹

Other echocardiographic criteria for LVH

Indexing LV mass to its allometric power (i.e. LV mass/height^{2.7}) represents an alternative approach and LVH would be present in adults if LVMI thus obtained was greater than 51 g/m^{2.712}. Whilst there is no consensus regarding cutoff value of LVMI in adolescent athletes, in pediatric practice, criteria for LVH can be defined as $LVMI > 38.6 \text{ g/m}^{2.713}$. Thus, UNIANOVA tests were performed to assess if QT peak was higher in subjects with LVH using these different echocardiographic criteria.

ECG Assessment

Resting 12-lead ECGs were obtained in all subjects in the supine position, using an analog system (Marquette Electronics, Milwaukee, Wisconsin, USA). The paper speed was 25 mm/s with a 10 mm/mV gain. One single observer blinded to all clinical details of the subjects except age/date of birth made the following measurements using a millimeter ruler and calipers.

QT peak measurements

QT peak was defined as the onset of QRS to the peak of the T wave. The T peak was defined as the point where the T wave had the maximum amplitude. This applied to inverted T waves too. If the T wave was biphasic, then the deflection with the higher amplitude was used. However, if the upward and the downward deflection were of equal amplitude, then the measurement would be omitted. The heart rate-corrected QT peak (QTpc) of lead I was calculated using Bazett's formula (QT peak/Square root of the preceding RR interval).¹⁴ Two consecutive readings were taken where possible and the mean QTpc was calculated. Heart rate (HR) was computed by the formula $HR = 60/RR$ interval.

QT end measurements

QT end (QT_e) was defined as the interval between the start of the Q wave and the end of the T wave in lead I. The T wave end was defined as the point when the T wave returned to the isoelectric line. If this point was not clearly defined,

then the reading would be omitted. If the T wave was followed by a U wave, then the nadir between the T and the U wave (i.e. the lowest point of the curve) would be taken as the point where the T wave ended. QT was measured from up to three ECG complexes and the mean was taken. To work out the heart rate-corrected QT interval (QT_{ec}), the preceding R-R interval was measured. QT_{ec} was calculated using Bazett's formula i.e. $QT_{ec} (ms) = QT (ms) / \sqrt{R-R \text{ interval (s)}}$.

Measurements of other ECG criteria for LVH

The classic Sokolow Lyon voltage criteria for LVH were defined as S in V1 + R in V5 or V6 > 3.5 mV.¹⁵ The Cornell Voltage criteria were defined as R in aVL plus S in V3. The Cornell Voltage is positive for LVH if it is ≥2.8 mV in male patients and 2 mV in female patients.¹⁶ The Cornell Product is a product of the Cornell Voltage and the QRS duration. If the Cornell Product is greater than or equal to 243.6 mV ms (2436 mm ms), then the Cornell product is positive for LVH.^{17,18}

Statistics

SPSS (SPSS Inc., Chicago, Illinois, USA) was used for statistical analysis. Spearman (2-tailed) test was used for nonparametric bivariate correlations between LVMI and QT_{pc}. UNIANOVA was used to assess correlations if the residual was normally distributed i.e. if the one-sample Kolmogorov-Smirnov test 2-tailed P > or equal to 0.05. The ability of long QT_{pc} to predict echocardiographic LVH was assessed using the Mantel-Haenszel Common Odds Ratio Estimate and Fisher's exact test, using 320 ms as cut-off value for QT peak (see below for explanation of choice of cutoff value).

Power Calculations

Power calculations were based on a small pilot study carried out on hypertensive subjects.⁷ Twenty-four out of 44 hypertensive subjects in that cohort had LVH (55%). Using long QT peak to detect LVH according to echocardiographic LVMI (type 1 error 0.05), we estimated that 90% power to detect 33.3% difference (odds ratio 5) requires 80 patients (40 in each group: <320 ms vs. ≥320 ms). In that cohort, 20 had prolonged QT peak defined above, and 24 had normal QT peak.

RESULTS

QT peak and other ECG criteria for LVH were assessed in 117 male professional footballers (age range 15–19, mean 16.4 years ± SD 0.76). Echocardiographic LVH was seen in 79 (70.5%) subjects. ECG-defined LVH was present in 59 (50.4%) players by Sokolow-Lyon criteria, and in 19 (16.2%) players by Romhilt Score.¹⁹ Only five players (4.3%) had LVH by Cornell voltage criteria and seven players (6%) by Cornell product >2436 mm ms.

The participants' mean heart rate was 64.8 beats per minute (± SD 11.9). QT_{pc} was not associated with LVH according to LVMI measurements using the ASE convention.

There was no correlation between QT peak interval in lead I and LVMI on echocardiography (Spearman $r = 0.058$, 2-tailed $P = 0.54$; Fig. 1). Hence, echocardiographic LVH in young athletes was not associated with prolongation of QT peak (Fisher's exact 2-tailed $P = 0.6$, Mantel-Haenszel Common Odds Ratio Estimate = 1.3, 95% CI 0.51–3.6, 2p = 0.55; Fig. 2). The QT_{pc} of subjects without LVH was 301 ± 3.8 ms (95% CI = 294–309 ms). QT_{pc} in those with LVH measured 306 ± 2.5 ms (95% CI = 301–311 ms). UNIANOVA $P = 0.33$ indicated no significant difference in QT_{pc} between those with LVH and those without LVH. The One-Sample Kolmogorov-Smirnov Test 2-tailed $P = 0.90$ and therefore confirmed the residual for QT_{pc} was normally distributed (Fig. 3).

Figure 1. There is no significant correlation between QT peak and left ventricular mass index (LVMI) in young professional footballers.

Figure 2. Echocardiographic LVH in teenage professional footballers was not associated with prolongation of QT peak.

The heart rate-corrected QT end (QTec) measured 403 ± 15.9 ms. No correlation was found between QTec and LVMI shown on echocardiography (Spearman $r = -0.025$, 2-tailed $P = 0.79$).

QTpc Was Not Associated with LVH According to other Echocardiographic Criteria

To be certain there was no correlation between LVMI and QT peak prolongation, we tested the correlation between QT peak and

LV mass indexed to its allometric power (i.e. LV mass/height^{2.7}). Spearman correlation between heart rate-corrected QT peak and LV mass indexed to its allometric power was again not statistically significant ($r = 0.13$, $2p = 0.19$). Subjects without LVH had QTpc of $302 \text{ ms} \pm 3.1$ (95% CI = 295–308 ms), and those with LVH had QTpc measuring $307 \text{ ms} \pm 2.7$ (95% CI = 301–312 ms); [UNIANOVA $P = 0.20$]. The One-Sample Kolmogorov-Smirnov Test 2-tailed $P > 0.05$, thus the residuals of QTpc of lead I were normally distributed.

Whilst there is no consensus regarding cutoff value of LVMI in adolescent athletes, criteria for LVH in pediatric practice can be defined as LVMI $> 38.6 \text{ g/m}^2$.⁷ Using the pediatric criteria, there is again no difference in QTpc between subjects with LVH and those without ($305 \pm 2.1 \text{ ms}$ vs $300 \pm 8.3 \text{ ms}$, UNIANOVA $P = 0.59$). The One-Sample Kolmogorov-Smirnov Test 2-tailed $P > 0.05$, which means the residuals of QTpc of lead I were normally distributed.

QTpc Was Not Associated with ECG LVH

Moreover, there was no relation between QTpc of lead I and LVH using any of the following ECG criteria: Sokolow-Lyon ($P = 0.6$), Romhilt ($P = 0.3$), Cornell product ($P = 0.6$), or Cornell voltage ($P = 0.8$). The standard Cornell Voltage criterion for LVH has high specificity but poor sensitivity at predicting LVH in teenage professional footballers (Fig. 4).

Figure 3. Residual for QTpc was normally distributed.

DISCUSSION

Sudden cardiac death in young athletes is a rare but recognized problem that often generates high profile publicity. Different screening methods, including ECG and echocardiography, to detect potential cardiovascular risks leading to sudden cardiac death have been used. The European Society of Cardiology has recently published a consensus statement suggesting a common European protocol of cardiovascular screening of young competitive athletes prior to the participation of sport activities. The authors of the report argue that using a 12-lead ECG as a screening tool helps in preventing sudden death.²⁰ Hence, it is important to study the significance of all potential pathological ECG changes in young athletes to improve the screening sensitivity. Athletes usually have training-induced LVH that

AUC=area under the curve

Cut-off values (Cornell Voltage,mV)-Greater Than or Equal To	Sensitivity	Specificity
1.4	73%	46%
1.7	53%	76%
2.5	14%	97%
2.8	6.3%	100%

Figure 4. ROC curve showing the relationship between Cornell voltage and echocardiographic LVH.

is believed to be physiological in nature^{21,22} and is different from the pathological LVH induced by hypertension or aortic stenosis. The current ECG criteria of LVH cannot differentiate between these two types of LVH.

Long QT dispersion is a feature of LVH in hypertensive patients, but it is not present in LVH induced by physical training.^{23,24} Long QT peak dispersion is also associated with LVH in patients with hypertension.²³ Measurement of QT dispersion is difficult and time consuming in clinical practice. Prolonged QT end of any lateral lead (I, avL, V5 and V6) is associated with death in patients who have suffered a stroke.²⁵ QT peak interval in a single lead is clearly easier to measure and appears to

be a more practical and rapid way to determine the presence of LVH in hypertensive patients.⁷

The present study is adequately powered to test the hypothesis that prolonged QT peak in lead I correlates with LVH in young professional footballers. Our study demonstrated no correlation between increased LVMI on echocardiography and both QT end and QT peak intervals. This differs from the results reported by Wong et al.,⁷ who found that QT peak was a sensitive indicator of LVH in patients with hypertension. The discrepancy in findings between the two studies might be explained by the hypothesis that QT peak is only prolonged in patients with pathological LVH whose hallmark is myocardial fibrosis, which is a dysrhythmogenic substrate. Thus, one would expect electrophysiological markers of cardiac risk, such as QT peak, to be prolonged in patients with pathological LVH, but not in physiological LVH that is characterized by myocyte hypertrophy. Both echocardiographic and ECG evidence of hypertension-induced LVH are associated with increased risk of mortality and cerebrovascular events.^{26,27}

It can be argued that exercise-induced cardiac hypertrophy displays features similar to mild compensated hypertrophy from other causes.²⁸ Following hypertrophy, action potential duration in midmyocardial and subepicardial myocytes is prolonged. This is contrary to the unchanged, or even shortening, action potential duration in subendocardial myocytes.^{29,30} This difference in action potential duration is thought to be proarrhythmic because of altered dispersion of repolarization and enhanced automaticity regardless of the cause of LVH.²⁸ However, sudden death related to LVH in young athletes is usually induced by an underlying undiagnosed cardiovascular disease such as hypertrophic cardiomyopathy and less commonly aortic stenosis i.e. the LVH is pathological. Those with no underlying cardiac disease have no such risk.²⁰

Therefore, the notion that LVH per se is proarrhythmic does not explain this difference in risk. Thus, the need still exist to uncover methods that could differentiate between the "risky" and the "not so risky" LVH. Further research is, therefore, required to confirm the role of novel electrocardiographic features such as QT peak prolongation that may be able to differentiate between pathological and physiological LVH.

Although most investigators would acknowledge that an indexation of left ventricular mass to body size is important, and most often this is done by

dividing by the body surface area, as in the present study, some would argue that this approach might misclassify obesity-induced LVH as normal. Therefore, indexing LV mass to its allometric power (i.e. LV mass/height^{2.7}) might be a preferred approach and LVH would be present in adults if LVMI thus obtained was greater than 51 g/m^{2.7}.³⁰ Thus to be certain there was no correlation between LVMI and QT peak prolongation, we tested the correlation between QT peak and LV mass indexed to its allometric power and found no evidence of QT peak prolongation in subjects with LVH using this echocardiographic criterion either.

In addition, it is recognized that LVMI cutoff values are different in adult and children. There is little data or consensus opinion regarding the precise cutoff value for LVMI in adolescents. In pediatric practice, criteria for LVH can be defined as LVMI > 38.6 g/m^{2.7}.³¹ Using the pediatric criteria, again there was no difference in QTpc between subjects with LVH and those without LVH (305 ± 2.1 ms vs 300 ± 8.3 ms).

It was suggested that the sensitivity of different ECG criteria of LVH was related to the gender of the patients, with the Cornell criteria to be superior for male patients, whereas the Sokolow-Lyon product criterion was superior for female patients.³¹ In the present study, Cornell Voltage has excellent specificity (100%) but poor sensitivity (6.3%) in detecting echocardiographic LVH in young male athletes. The poor sensitivity might be due to the fact that ECG LVH criteria only tend to detect individuals with an LVMI substantially above the normal range.³¹ Taken together, these findings suggest that voltage criteria of LVH are not generated merely because of LV wall thickening.

The pathological nature of that thickening might be important in determining the type of electrical changes that appear on the surface ECG.

Study limitations

We acknowledge that the main limitation of this study is the fact that the ECG measurements were all done manually. Digitized ECG measurement might be more accurate than manual measurement. Digitized ECG measurement, however, is not feasible in daily clinical practice. Thus, prior to the development of accurate and reproducible ECG analysis software that measures QT peak interval in lead I, manual measurements are important in the research setting as they reflect the actual measure-

ments that can be easily made in real-life clinical practice. Indeed, a recent study using manual measurements has confirmed a relationship between QT peak prolongation in lead I and LVH in patients who have suspected coronary ischemia.⁸ Secondly, it can be argued that cardiac magnetic resonance imaging (CMR) is better than echocardiography at assessing left ventricular mass. Certain conditions such as chronic obstructive pulmonary disease and obesity tend to make the echocardiographic images difficult or impossible to interpret. This problem may be less clinically relevant in young professional footballers. Unfortunately, cardiac MR is still not widely available in routine clinical practice in the UK.

Future research

A large long-term observational study is required to address the question whether footballers with long QT peak are at greater risk of sudden death. If the relationship between QT peak prolongation and sudden death risk is confirmed in adolescent athletes, then QT peak should be assessed prior to regarding the ECG as normal. More work will then be required to assess what cardiac abnormalities may be over-represented in patients with long QT peak, in addition to ensuring that their electrolytes (such as potassium, calcium, and magnesium) are normal.³²

The present study suggests that in adolescent athletes with no evidence of LVH, QT peak measures 301 ± 3.8 ms (95% CI = 294–309 ms). This provides a helpful reference range for QT peak, which is not well documented in the literature.

CONCLUSION

LVH is common amongst teenage footballers. However, there does not appear to be any correlation between LVH and QT peak in this cohort of teenage footballers. Our findings support the hypothesis that LVH in teenage footballers is likely to be due to myocyte hypertrophy rather than myocardial fibrosis, which is dysrhythmogenic and characterizes pathological LVH.

Competing interest: None

REFERENCES

1. Levy D, Garrison RJ, Savage DD, et al. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. *N Engl J Med* 1990;322:1561–1566.

2. Verdecchia P, Schillaci G, Borgioni C, et al. Prognostic significance of serial changes in left ventricular mass in essential hypertension. *Circulation* 1998;97:48-54.
3. Barr CS, Naas A, Freeman M, et al. QT dispersion and sudden unexpected death in chronic heart failure. *Lancet* 1994;343:327-329.
4. de Bruyne MC, Hoes AW, Kors JA, et al. QTc dispersion predicts cardiac mortality in the elderly: The Rotterdam Study. *Circulation* 1998;97:467-472.
5. Clarkson PB, Naas AA, McMahon A, et al. QT dispersion in essential hypertension. *QJM* 1995;88:327-332.
6. Lim PO, Nys M, Naas AA, et al. Irbesartan reduces QT dispersion in hypertensive individuals. *Hypertension* 1999;33:713-718.
7. Wong KYK, Lim PO, Wong SYS, et al. Does a prolonged QT peak identify left ventricular hypertrophy in hypertension? *Int J Cardiol* 2003;89:179-186.
8. Velavan P, Wong KY, Ball JB. Evaluation of a novel technique to detect left ventricular hypertrophy on the surface electrocardiogram- QT peak. *Circulation [Suppl]* 2004;110:III-580.
9. Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man. *Circulation* 1977;55:613-618.
10. Troy BL, Pomo J, Rackley CE. Measurement of left ventricular wall thickness and mass by echocardiography. *Circulation* 1972;45:602-611.
11. Verdecchia P, Schillaci G, Borgioni C, et al. Prognostic value of left ventricular mass and geometry in systemic hypertension with left ventricular hypertrophy. *Am J Cardiol* 1996;78:197-202.
12. Kahan T, Bergfeldt L. Left ventricular hypertrophy in hypertension: Its arrhythmogenic potential. *Heart* 2005;91:250-256.
13. de Simone G, Devereux RB, Daniels SR, et al. Effect of growth on variability of left ventricular mass: Assessment of allometric signals in adults and children and their capacity to predict cardiovascular risk. *J Am Coll Cardiol* 1995;25:1056-1062.
14. Bazett HC. An analysis of the time relations of electrocardiograms. *Heart* 1920;7:353-367.
15. Sokolow M, Lyon P. The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. *Am J Cardiol* 1949;37:161-168.
16. Casale PN, Devereux RB, Alonso DR, et al. Improved sex-specific criteria of left ventricular hypertrophy for clinical and computer interpretation of electrocardiograms: Validation with autopsy findings. *Circulation* 1987;75:565-572.
17. Molloy TJ, Okin PM, Devereux RB, et al. Electrocardiographic detection of left ventricular hypertrophy by the simple QRS voltage-duration product. *J Am Coll Cardiol* 1992;20:1180-1186.
18. Okin PM, Roman MJ, Devereux RB, et al. Electrocardiographic identification of increased left ventricular mass by simple voltage duration products. *J Am Coll Cardiol* 1995;25:417-423.
19. Romhilt DW, Estes EH. A point-score system for the ECG diagnosis of left ventricular hypertrophy. *Am Heart J* 1968;75:752-758.
20. Corrado D, Pelliccia A, Halvor Bjørnstad H, et al. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: Proposal for a common European protocol. *Eur Heart J* 2005;26:516-524.
21. Pluim BM, Lamb HJ, Kayser HW, et al. Functional and metabolic evaluation of the athlete's heart by magnetic resonance imaging and dobutamine stress magnetic resonance spectroscopy. *Circulation* 1998;97:666-672.
22. Kozakova M, Galetta F, Gregorini L, et al. Coronary vasodilator capacity and epicardial vessel remodeling in physiological and hypertensive hypertrophy. *Hypertension* 2000;36:343-349.
23. Perkiomaki JS, Ikaheimo MJ, Pikkujamsa SM, et al. Dispersion of the QT interval and autonomic modulation of heart rate in hypertensive men with and without left ventricular hypertrophy. *Hypertension* 1996;28:16-21.
24. Lonati LM, Magnaghi G, Bizzi C, et al. Patterns of QT dispersion in athletic and hypertensive left ventricular hypertrophy. *Ann Noninvasive Electrocardiol* 2004;9:252-256.
25. Wong KYK, MacWalter RS, Douglas D, et al. Long QTc predicts future cardiac death in stroke survivors. *Heart* 2003;89:377-381.
26. Verdecchia P, Porcellati C, Reboldi G, et al. Left ventricular hypertrophy as an independent predictor of acute cerebrovascular events in essential hypertension. *Circulation* 2001;104:2039-2044.
27. Sundstrom J, Lind L, Arnlov J, et al. Echocardiographic and electrocardiographic diagnoses of left ventricular hypertrophy predict mortality independently of each other in a population of elderly men. *Circulation* 2001;103:2346-2351.
28. Hart G. Exercise-induced cardiac hypertrophy: A substrate for sudden death in athletes? *Exp Physiol* 2003;88:639-644.
29. Shipsey SJ, Bryant SM, Hart G. Effects of hypertrophy on regional action potential characteristics in the rat left ventricle: A cellular basis for T-wave inversion? *Circulation* 1997;96:2061-2068.
30. Bryant SM, Shipsey SJ, Hart G. Regional differences in electrical and mechanical properties of myocytes from guinea-pig hearts with mild left ventricular hypertrophy. *Cardiovasc Res* 1997;35:315-323.
31. Alfakih K, Walters K, Jones T, et al. New gender-specific partition values for ECG criteria of left ventricular hypertrophy: Recalibration against cardiac MRI. *Hypertension* 2004;44:175-179.
32. Wong KY, McSwiggan S, Kennedy NS, et al. Spectrum of cardiac abnormalities associated with long QT in stroke survivors. *Heart* 2005;91:1306-1310.