

INTRODUCTION

There is considerable evidence to suggest that QT interval is closely related to ventricular action potential and is a good non invasive measure of the repolarization segment (*Fronz et al., 1988; Zobel et al., 1995*).

Several reports have indicated that regional difference in static QT interval measurement from a surface 12 lead electrocardiogram (QT dispersion) may provide an indirect measure of underlying inhomogeneity of myocardial repolarization (*Perkiomaki et al., 1995; Pye et al., 1994*).

Furthermore an increase in dispersion of the QT interval has been reported to predict the occurrence of ventricular tachycardia in ischemic heart disease (IHD) and serves to identify patients at risk for life-threatening arrhythmia after a previous myocardial infarction (*Perkiomaki et al., 1995*).

Arrhythmia and sudden cardiac death in subject with IHD are dynamic events linked to physical activity (*Cobb and Weaver, 1986*).

It is thereby conceivable that exercise induced abnormalities in QT dispersion may facilitate the development of ventricular arrhythmia (*Konnel, 1992*).