

*To my parents*  
Who Looked After Me  
In  
My Early Years

*To my professors*  
Who Taught Me  
& Still  
Teaching Me With No Limits

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*Review  
Of  
Literature*

*Introduction*  
*&*  
*Aim Of The Work*

# Introduction

Abnormalities of the electrocardiography do not necessarily indicate cardiac disease, much less coronary artery disease (*Marriott, H.J.L., et al., 1967*). When deviations from the normal, especially those affecting the ST segment and T waves, are encountered in the middle aged and elderly patients, they are glibly interpreted as "coronary insufficiency". Statistically, such inferences are no doubt often right, but the habit is a bad practice and is scientifically unsound (*Marriott, H.J.L. 1988*).

We must remember the following facts before attaching the cardiac or coronary label:

- 1- The range of normal is wide and its limits cannot be satisfactorily defined (*Simonson, E., 1956*). Changes well outside the accepted range are undoubtedly at times normal variants. Examples of this are the persistent "juvenile" pericordial pattern particular seen in healthy young blacks (*Littmann, D., 1946*). Unusual S-T elevation also especially common in blacks and often referred to as "early repolarization" (*Mirvis, D.M., 1982*).

It must be differentiated from acute myocardial infarction and pericarditis (*Ginzton, L.E., et al., 1982*)

The ST elevation of "early repolarization" may be resorted to the baseline with exercise or isoproterenol (*Morace, G., 1979*).

Other potential mimics include: ST-T depression "in suspended" hearts (*Evans, W., et al., 1957*), precordial T wave inversion during pregnancy, prolonged P-R intervals in occasional healthy hearts (*Manning, G.W., 1954*) and a right bundle branch block pattern in marathon runners, whose cardiovascular competence can hardly be questioned (*Beckner, G.L., et al., 1954*).

Striking T wave inversion is found in well-trained, top ranking athletes, such as professional bicyclists and marathon runners (*Hanne-Paparo, N., et al., 1971*).

ST-T wave changes, simulating those of ischaemic heart disease, have been reported in normal men with normal coronary arteriograms (*Taggart, B., 1979*). and about one third of normal men show 1 mm. ST depression and/or labile T wave inversion of up to 3 mm. during ambulatory monitoring (*Armstrong, W.F., 1982*).

In normal youths, suspicious T wave changes may be the result of increased sympatho-adrenal activity (*Atterhog, J.H., 1981*).

- 2- Numerous extracardiac factors can produce patterns similar to those seen in myocardial disease; without necessary impairing cardiac competence, many factors can cause changes in repolarization processes of the ventricles which are reflected in the electro-



cardiography in T wave or ST segment alterations. The T wave and, to a lesser extent, the S-T segment are unstable members that are easily upset by a great variety of major and minor provocations. Among the many stimulants that can affect them are eating (*Rochlin, I., et al., 1954*), drinking ice water (*Dowling, C.V., et al., 1951*), posture (*Scherf, D., et al., 1952*), hyperventilation (*Wasserburger, et al., 1956*), emotional disturbance such as startle reaction, fear or anxiety (*Mitchell, J.H., 1954*) and neurocirculatory asthenia (*Silverman, J.J., et al., 1951*). Numerous drugs can also induce ST-T changes such as digitalis, quinidine, procainamide, adrenaline, isopril, insulin. Extracardiac diseases such as electrolyte imbalances, the acute abdomen, shock, hiatus hernia, gall bladder disease, cerebrovascular accidents, psychosis and endocrine and metabolic disturbances were also reported (*Burch, G.E., et al., 1954*).

3. Cardiovascular diseases other than coronary artery disease may counterfeit the changes of ischaemic heart disease. Such mimics include myocarditis (*Palank, E.A., et al., 1979*), cardiomyopathy, hypertension and other diseases producing ventricular hypertrophy.
4. Finally, the heart may be the victim of a disease that is not primarily cardiac, such as pulmonary embolism, anaemia, hypothyroidism, myocarditis (*De La Chapelle, C.E., et al., 1954*) from infections (e.g., pneumonia, infectious mononucleosis), sarcoidosis, hemochromatosis,

primary amyloidosis, beri beri, scleroderma, disseminated muscular dystrophy and myasthenia gravis; all may produce changes in the tracing indicative of myocardial involvement and quite indistinguishable from some of the alterations resulting from coronary artery disease.

Numerous conditions can produce Q-waves that may be mistaken for those of myocardial infarction (*Proudfit, W.L., et al., 1985*):

1. Left ventricular hypertrophy.
2. Incomplete left bundle-branch block.
3. Hemiblock, anterior or posterior.
4. Myocardial replacement (neoplasm, sarcoid ...etc.).
5. Cardiomyopathy.
6. Wolff-parkinson-white syndrome.

Transient Q-waves may develop with coronary spasm of variant angina and even in occasional cases of pericarditis, (*Meller, J., 1975*) hypoglycaemia, hyperkalaemia, shock, acute pancreatitis and phosphorus poisoning.

A mere reversal of electrodes can simulate the Q-waves and ST segment displacement of acute myocardial infarction.

Therefore, in assessing the tracing that does not conform with our accepted standards, we should remember the whole array of common and uncommon possibilities and we should ask ourselves three questions:

1. Could this be normal variant?
2. Could these abnormalities be due to extracardiac factors; physiological or pathological?
3. Could these changes be due to heart disease other than coronary?

The danger of attributing changes of the 1st and 2nd category to heart disease is that the patient is branded as a cardiac.

The danger of labelling the third group as "coronary" is that the physician in charge of the case may thereby be blinded to the true nature of cardiac involvement and of the underlying primary disease.

We should often be content to state that the pattern is abnormal but non-specific. So, an abnormal tracing does not necessarily mean an abnormal heart (*Marriot, H.J.L., 1988*).

## *Aim of The Present Work*

The present study aims at assessing the electrocardiographic diagnosis of "early repolarization syndrome" in normal people and their families to determine:

- The prevalence of this normal variant (early repolarization syndrome) in families.
- Effect of isoproterenol on early repolarization syndrome.

# *Standards of Normal*

## *Normal standards*

### **Principles for determination of electrocardiographic normal standards:**

With the growth of clinical electrocardiography, reliable standards of normality have become increasingly important, but the volume of research on normal variability is very small as compared to that on disease and disorders. Further work on normal variability is essential (*Simonson, E., 1956*). Ernest Simonson summarized the principles for determination of electrocardiographic normal standards as follows:

1. The criteria of clinical health, used by various authors for selection of normal control groups have varied widely from highly selected groups as young aviators to non cardiac patients, even during terminal stages of the disease.
2. The population sample to which the standards refer should be precisely described, particular with regard to the items which have been shown to affect the electrocardiography (age, body weight, sex). Other items (race, geographical region, climate, occupation) should also be considered in the description of the groups, although the information of possible effects on the electrocardiogram is still scanty.
3. Normal standards derived from any particular group, although strictly valid only for this group, may have general application to other groups

although the application of "normal standards" even to apparently similar groups has still a certain margin of error.

4. The range in the standards for the combined groups will be much wider than for any of the single groups, resulting in a much poorer differentiation between normal and abnormal.
5. The normal limits should be given in terms of the percentage of normal population included.

Limits including 100 per cent of the normal population are not particular because a very large number of cardiac patients could also be included within such wide limits, resulting in a large percentage of false diagnosis of normality.

Arbitrary limits including 95-98 per cent of normal population have been shown to be practically workable. The probability that an electrocardiography items outside these range limits is due to pathology is significantly greater than the chance of a normal variation, but it is often not recognized that a small part (2-5%) of normal population will, by definition, exceed the limits.

6. The normal limits should be determined from evaluation of the frequency distribution.
7. In the normal electrocardiography standards for normal adults recommended by the criteria committee of the New York Heart

Association, many of the above principles have been ignored (*Simonson, E., 1956*).

#### **The electrocardiography in healthy people:**

Anatomic normality does not exclude functional changes (*Myers, G.B., et al., 1947*). Therefore, we need to consider the range of normality in the electrocardiography but of course we cannot escape from the fact that not all diseases cause symptoms or abnormal physical signs, and a subject who appears healthy may not be so.

In particular, individuals who present for screening may well have symptoms about which they have not consulted a doctor, so, it cannot be assumed that an electrocardiography obtained through a screening programme has come from healthy subject. The range of normality of the electrocardiography is therefore debatable; we have to consider the variations in the electrocardiography that we can expect to find in completely healthy people, and then we can think about the significance of more marked abnormality (*Hampton, J.R., 1992*).

Hampton summarized the acceptable variations in the normal electrocardiography as follows:

#### **The normal cardiac rhythm:**

Sinus rhythm is the only normal sustained rhythm. In young people the R-R interval is reduced (that is the heart rate is increased) by inspiration and this is called sinus arrhythmia. When sinus arrhythmia is