# SHORT PQ INTERVAL PLUS LONG QT INTERVAL ON SAME PERSON.

**Keywords**: Cycle cardiac. Short PR interval. Long QT interval. Tachycardia. Cardiac Rhythm.

**ABSTRACT**

For many authors (See References), there's a great relation between in altered duration of electrical cardiac system and the risk apparition of serious ventricular disturbances and even of sudden cardiac death. We're according with them. We have the conviction firm of the existence of more electric cardiac problems than haven't been published yet. This presentation could be its corroboration. OBJECTIVE: Show the existence of new electrocardiographic parameters to clinicians. METHODS: Evaluation of electrocardiographic parameters with measurement of its length, both manually as computerized way. RESULTS: A pattern of short PQ interval plus a long QT interval in the same person. CONCLUSIONS: Draw conclusions about a pattern, without previous support of bibliography would be a temerity and speculation. We just want to expose this clinical case. In none moment has been our intention make any type of conclusion; only has been show to doctors that these entities exist.

**INTRODUCTION**

Since more of 3000 year ago, we are studying the heart and in the two last centuries (XX and XXI), its electrical system. However, the last studies over its structure and there functions are related, almost all them, with entities already published (Variations on a same theme). This should not be necessarily so. Not only the entities as for example, short and long PQ interval or short and long QT interval (as well as its variations) are the only entities that have an own existence. They’re more than possible, the combinations between the values of duration of electrocardiographic intervals in same person. The Heart may have altered its conduction system in many more occasions that in the mentioned entities, because also are manifold its mathematical possibilities. It would be great errors no consider these criteria.

**METHODS and RESULTS**

The **methods** utilized for its valuation have been: Formulas of Bazzet, Fridericia and Framingham and the **measures** have been made with manual technique and digital technique (measure in pixel) in 12 leads. We have considered as normal values: PQ (or PR) interval equal or lesser 0.20 seconds and QT interval equal or lesser 0.45 seconds.

We have chosen, in order to simplify, the evaluation of values on orthogonal leads: D1, aVF, V2. These leads are more helpful for calculating the electrical axis of the heart. We also be have considered for this evaluation, the more equipotential derivations (lead); in this one case it is coincident with aVL and V2. Besides to the electrocardiograph, to this patient underwent a battery of tests: Holter, Ergometry, Analytical profiles, Echocardiogram. He was also tested for genetic study and electrophysiological study. In genetic studies there were not any specific alterations for gene of short PQ interval and nor for gene of long QT interval (alterations of channels of sodium or potassium).

In electrophysiological study there aren't any alterations of cardiac conduction after of blocking with smolol and posterior stimulation with adenosine. Why these normalities? We don't know yet, but they're already under actual study by our investigator team.

**Brief History**

He's a young man with 26 old-years; in your family history there are two sudden deaths of one possible cardiac origin (According to the doctors. Father and paternal uncle have died before 50 old years). He came to emergency department by one access palpitations and tachycardias not related to the physical effort (which appeared suddenly during physiological sleep). In a first moment, your electrocardiogram was considered as normal, but, with a detailed interpretation, we can see it as there are electric alterations than could be explain the tachycardias and a possible sudden cardiac death. (We think that these cardiac events should be studied in more profundity and more exhaustively than until to date).

From the point of view **Epidemiological**, we can expose, only, our clinical cases, position that when not having bibliographical support it is an impossible task in itself.

The Universe that we have at the present time, reaches the 32 clinical cases as the exposed here [Pertaining to 7 different families: North American 2 (2/7); Australian 1 (1/7); South American 2 (2/7); European 2 (2/7)]. The 100% of the cases are male and with age between the

12 and 40-old years. Absolutely all the patients have suffered, at least, three accesses of nocturnal tachycardia (inclusion criteria). In your familial antecedents, there are to the minus, one person with cardiac important symptomatology. As empirical treatment we have used a Beta Blocker (Sotalol).

Sotalol it's a very versatile drug, since it's structurally a beta blocker (antiarrhythmic class II) but it has also an action antiarrhythmic type I-A and has been classified on the antiarrhythmic of group class III).

In some case, it seems have action both in the auricle-ventricular node, as in myocardial cells and seems to reduce number of cardiological accesses per year. All our clinical cases (32/32) were a diagnosis ambulatory of psychosomatic alterations.

**DISCUSSION**

In illustration exposed here, we can see as there are alterations of electrical systole not published until to date: We are in front of an electrical cardiac entity: short PQ interval (lesser than 0.12 seconds) plus a long QT interval (greater than 0.450 seconds), into same person. And, however, the person hasn't had an important cardiac symptomatology**,** just slight symptoms.

If we give as true that the myocardium is more unstable and vulnerable when there are changes in the length of intervals (PQ and QT in this case. See references), it’s clear that this electrocardiogram meets those criteria. Therefore, the risk of serious heart rhythm disturbances (torsades de pointes, ventricular fibrillation, etc...) could be very high in this patient.

In this presentation, our questions have been:

- Is there a short PQ interval? Yes, there is.
- Is there a long QT interval? Yes, there is.
- Is there a high risk of events of lethal arrhythmias? Don't is known for us this problematic yet.

We’re then in front of a cardiac entity, that can have a high risk of serious arrhythmias, and therefore at high risk of sudden cardiac death.

**CONCLUSIONS**

It’s extremely difficult to draw **conclusions** without committing some sort of bias. Make some conclusion over this electrocardiographic pattern would be only make speculations because there aren't any antecedents as references. Our only intention with this exhibition has been to make public its existence. At no time has been to assert that there is a categorical relation, cause (electrocardiogram) - effect (symptoms).

If we give like certain all commentaries of cited authors (see references), we can to draw the following reflexions:

- Has this patient any risk of serious arrhythmias? We don't know yet. But it’s more than probable.
- Has this patient any risk of sudden cardiac death? We don't know yet. But it’s more than probable.

The only certain is than we’re before a pattern unknown and without any references and, therefore, is practically impossible say if may have risk of serious ventricular disturbances and/or sudden cardiac death.

**MANAGEMENT AND TREATMENT**

If since it is very difficult the unification of criteria among doctors, at this time, to decide what is the most effective treatment in the QT prolonged and in short PQ separately, when both are present in the same person (as the present case), such treatment can only be empirical, given his status unknown until today.

For some authors, the fundamental differences between QTL1 (KCNQ1 (KvLQT1) and QTL2 (KCNH2 -HERG) with QTL3 (SCN5A), besides the gene responsible, is the appearance of clinical and response to treatment with beta blockers. In QTL3, symptoms usually appear during sleep and are poorly controlled by the administration of beta-blockers. In clinical case presented, the symptoms are primarily nocturnal (tachycardias and palpitations). But, the most serious symptoms appear without relation with the physical effort.

Sotalol (a beta blocker), it appears that controls the duration of symptomatic phases in this patient.

There's a scheme Risk Stratification based on the genetic defect, the duration of the QTc and sex [8, 12]. The categories were divided into high-risk (50%: QTc> 500 ms, LQT1, LQT2, and men with LQT3), intermediate risk (30-40%: QTc <500 ms: LQT2 in women and men, and women with LQT3; QTc> 500 ms: in women with LQT3) and low risk (<30%: QTc <500 ms and men with LQT1 and LQT2). In our clinical case, could be between high and intermediate risk. There're authors who come to categorize a prolonged QT according to the morphology of the wave T.

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**NOTE BY AUTHORS**

The authors have thought it isn't necessary specify the numbers of references in the text. Only read, the references recommended. In they can see everyone assertions made for us.

Exception made in stratification cardiac risk**.**

**TABLE 1**

**In this table we can see the differences among cardiac entities with alteration in length of its intervals and its risk for suffer sudden cardiac death.**

**DIFFERENCES AMONG CARDIOLOGICAL ENTITIES**

| CARDIAC ENTITY | PQ interval (s) | QTc interval (s) | RISK SUDDEN CARDIAC DEATH |
|---|---|---|---|
| **W-P-W. Syndrome.** | SHORT. | NORMAL | YES. |
| **L-G-L. Syndrome.** | SHORT. | NORMAL | VARIABLE. |
| **MANHAIM Syndrome** | SHORT OR NORMAL. | NORMAL | VARIABLE. |
| **LONG QT Syndrome** | NORMAL. | LONG | YES. |
| **SHORT QT Syndrome** | NORMAL. | SHORT | YES |
| **SHORT PQ Syndrome** | SHORT | NORMAL | YES. |
| **SHORT PQ & QT (1)** | SHORT. | SHORT | UNKNOWN. |
| **SHORT PQ& LONG QT (2)** | SHORT. | LONG | UNKNOWN. |
| | | | |

**(1)** Breijo Márquez, FR. Decrease of electrical cardiac systole. Int J Cardiol 2008. 23; 126: e36-38.
**(2)** Actual illustration.

**FIGURE 1**.

Superior image. Octagonal leads (D1-aVF-V2)

Inferior image. Electrocardiogram complete (12-leads)