THE SIGNIFICANCE OF ELECTROCARDIOGRAM IN CLINICAL PRACTICE AND SCIENTIFIC RESEARCH IN XXI CENTURY

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ABSTRACT

Currently, the electrocardiogram (ECG), in spite of being a method with more than 100 years of existence, remains as one of the three mainstays in cardiological clinical diagnosis; the two other being interrogatory and physical examination, and transthoracic echocardiogram.

In the field of cardiac arrhythmias, ECG is considered the gold standard.
In acute coronary syndromes, it is considered fundamental in therapeutic decision-making, since thrombolytic management depends on the electrocardiographic pattern.
In all the branches of cardiology, ECG has some usefulness. Thus, in the evaluation of cardiac chambers overloads, in spite of being of low sensitivity and high specificity, enables a diagnosis of associations such as branch and divisional blocks, and the concomitant presence of electrically inactive areas.
In Hypertrophic Cardiomyopathy, it has a relevant and essential role in Arrhythmogenic Right Ventricular Dysplasia.
In Latin America, the ECG pattern of chronic Chagasic Cardiomyopathy is characteristic and diagnostic.
In pericarditis, it may be useful for evolutionary diagnosis.
In congenital heart diseases, both acyanotic and cyanogenic, it may show typical features that support the diagnosis.
Also ECG could be very useful in different electrolytic studies, as well as in the intoxication effect by different types of drugs.
ECG could also be very useful in right enlargements, both acute (acute pulmonary embolism) and chronic (emphysema).
The ECG has shown to be very useful in the screening of the so-called primary electrical diseases.
The ECG allows detecting different types of artifacts, as the accidental exchange of electrodes, and shivering.

INTRODUCTION

Electrocardiology is the branch of cardiology devoted to the study of electric activity that originates in the heart.
The conventional 12-lead electrocardiogram is a linear, graphic recording, obtained from the body surface, of the electric activity generated by the heart and gathered by small metal plates, located in the body surface in 12 points pre-established by convention, known as leads. The device has an amplifier that magnifies the electric signs, and a galvanometer, which moves according to the activation and repolarization events of cardiomyocites.

VALUES AND LIMITATIONS OF THE ELECTROCARDIOGRAM

Until mid-70s, in the clinic-cardiological diagnosis, there was a basic armory made up by:

1) Clinical history and physical examination;
2) Chest roentgenogram, and
3) ECG.

At the beginning of the 80s, this trio substituted one of its pillars, remaining thus until today. The approach almost indispensable in the clinico-cardiological diagnosis in the 21st C, is the clinical history and physical examination, 12-lead ECG, and conventional transthoracic echocardiography.

ECG, with more than a century of existence, remains completely valid because it an economic method, easily reproducible, independent from the observer, and very useful for a wide variety of areas of cardiology.

From the conventional 12-lead ECG, several methodologies were derived, that help in the diagnosis and risk stratification. Table 1 shows the main methodologies derived from the conventional 12-lead ECG (Table 1).

Table 1

Derived Methodologies from conventional 12-Lead Electrocardiogram (ECG or EKG):

1) ECG with Modified Protocol[1]
2) ECG with accessory right parasternal chest leads[2].
3) ECG with high precordial leads[3].
4) ECG with posterolateral precordial leads[4].
5) Esophageal lead to the left atrium[5].
6) Vectorcardiogram (VCG) [6].
7) Stress Test, Exercise Testing, exercise test, exercise stress test treadmill exercise test, exercise electrocardiogram, graded exercise test or stress ECG[7].
8) Cardiopulmonary Metabolic Exercise Testing (CMET) or Exercise Metabolic Testing[8].
9) Ambulatory Electrocardiography Recorders or Long-Term Electrocardiographic Recording: Holter Monitoring and Memory-Looping Event Monitoring[9].
10) Signal-Averaged Electrocardiogram, signal average ECG (SAECG) or High-Resolution Electrocardiography[10].
11) Heart Rate Variability (HRV) or 24-hour Heart Rate Variability[11].
12) QT dispersion [12].
13) Microvolt T-Wave Alternans (MTWA) [13].
14) T_{peak} -T_{end} Interval[14].
15) Heart-rate turbulence (HRT) [15].
16) Deceleration capacity (DC) [16].
17) Body-Surface Potential Maps (BSPM) [17].
18) His Bundle Recording, His bundle Electrogram (HBE), His' bundle electrograph or His bundle electrography[18].

Note: Late potentials on signal-averaged ECG, baroreflex sensitivity, HRV, MTWA, T_{peak} -T_{end} Interval, HRT, DC, and QT dispersion, are used as noninvasive stratification markers in several conditions:

1) Myocardial infarction patients treated by thrombolysis;
2) Myocardial infarction patients treated by primary percutaneous transluminal coronary angioplasty (PTCA);
3) Cardiomyopathies;
4) Channelopathies.

What valuable information can ECG provide us with?

The method can suggest us variations regarding the ethnical group, biotype, age range, gender, chest shape, body mass.

It is considered gold standard in the evaluation of any type of arrhythmia, and considered irreplaceable in this aspect.

It is extremely important in making decisions in acute coronary syndromes. The clinical history and examination, the serial 12-lead electrocardiography, and measurement of biochemical markers of myocardial necrosis (troponin T, troponin I, and creatinine kinase MB) are the essential diagnostic tools in this scenario. The presence of ST elevation in 2 or more adjacent leads ≥ 2 mm in V1, V2, or V3 or ≥ 1 mm in other leads in a patient with typical chest pain of possible cardiac origin, mandates hospitalization and cardiac monitoring, in most cases the early administration of thrombolytic agents.

USEFULNESS OF ECG IN LEFT VENTRICULAR ENLARGEMENT EVALUATION

The advantages of ECG in left ventricular enlargement (LVE) diagnosis include: low cost, easy application in a great universe, high specificity (close to 99%), simple diagnostic criteria, possibility of identifying ischemia, necrosis, arrhythmias, and associated dromotropic disorders, regardless from the experience of the observer and the quality of the equipment.

The drawbacks of ECG for LVE diagnosis are low sensitivity (20% to 60%. Only 3% of the general population and 5% of hypertensive patients show LVE in ECG), low specificity to determine the enlargement modality, inverse ratio between sensitivity and specificity of ECG criteria for LVE: the greater the sensitivity, the lower the specificity and vice versa, and unfortunately, sensitivity and specificity are affected in concomitance of right ventricular enlargement, myocardial infarction(s), bundle branch block, and/or fascicular blocks, and by sue of drugs[19].

USEFULNESS AND CHARACTERISTICS OF ECG IN RIGHT VENTRICULAR ENLARGEMENT EVALUATION

The factors of ECG modifications in right ventricular enlargements (RVE) are conditioned by modifications in the positional orientation of the heart that the enlargement imposes (rotations), modality and severity of hemodynamic enlargement (systolic or pressure enlargement or diastolic, volume, or eccentric enlargement) and the region of the right ventricle (RV) predominantly enlarged. Thus, if the affected area is the *trabecular region* of the RV, the ECG modifications are observed in the V2 and V3 leads; if the affected portion of the RV is the

inferior right paraseptal region, the ECG modifications are observed in the V3 and V4 leads; and if the RV enlargement affects the *RV free wall*, the leads affected are from V1 through V4. If the affected region is the *basal, infundibular, right ventricular outflow tract (RVOT)*, the ECG modifications are observed in VR and the high right precordial leads ($V_{1\text{-}H}$-$V_{2\text{-}H}$). Finally, if the hypertrophied area of the RV is the right ventricular inflow tract (RVIT), the leads affected are V_4R, V_5R, and VF[20].

USEFULNESS OF ECG IN CARDIOMYOPATHIES DIAGNOSIS

In Hypertrophic Cardiomyopathy (HCM), the ECG is very useful, and it is altered in 90% of the cases. Left ventricular enlargement with strain pattern (QRS/T angle near 180º) and prominent R waves in intermediary precordial leads is observed in ≈80% of cases. In a 10%, very wide R waves in V1 and aVR are associated to deep and "clean" Q waves in V5 and V6, and/or inferior leads, by increase of the septal vector.

Left anterior fascicular blocks are present in ≈10% of cases. Complete LBBB pattern is observed after transvalvar myotomy/myomectomy surgery in more than 80% of cases. Complete RBBB: after absolute alcohol injection in the first septal perforating artery of the anterior descending artery in percutaneous transluminal septal ablation is the rule[21; 22;23].

Frequent and/or prolonged runs of nonsustained ventricular tachycardia, mainly in patients with alterations of conscience on Holter, paroxysmal atrial fibrillation events, and blood pressure decrease or inadequate increase during stress test, are considered risk factors associated to sudden cardiac death in HCM.

In hypertrophic apical cardiomyopathy (ApHCM), a specific variant of HCM, the ECG is irreplaceable when revealing the typical giant negative T waves (≥10 mm) in precordial ECG[24].

Sometimes, R-wave voltage and T-wave negativity progressively decreased in magnitude at serial ECGs.

Nonsustained or sustained VT in patients that developed apical aneurysm with normal coronary arteries are observed, and possible progressing to myocardial necrosis and aneurysm with abnormal Q waves, reflect the interrelation between upper anterior septal thickness and other regions of the left and right ventricles, and wider Q waves are associated with late enhancement.

In ApHCM, sustained cavity obliteration is an important pathophysiologic condition, as well as hypertrophy, ischemia, and prolonged QTc, which are considered jointly related to the development of aneurysm through interactions[25].

Arrhythmogenic Right Ventricular Dysplasia (ARVD)/Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Approximately 90% of patients carriers of ARVD show ECG anomalies. ARVD diagnosis may be excluded if ECG is normal 6 years after the VT episode[26].

A prolongation of QRS complex (110 ms) located in the right precordial leads (V1-V2) or in anteroseptal wall (V1-V3) in adult patients, in absence of CRBBB (prolonged S wave upstroke) from V1 to V3, 55 ms is the most prevalent characteristic of ECG (95% of cases) and are correlated with the severity of the disease and induction of VT in programmed ventricular stimulation (PVS). The QRS complex could be normal, with slightly increased duration in precordial leads from V1 to V3 (>110 ms) and with lower duration from V3 to V6. For this

reason, a protocol for ECG recording, when there is a suspicion of ARVC/D, has been proposed. Standard ECG with additional highly amplified and modified recording technique represents a single diagnostic test with high value in the clinical diagnosis of ARVD/C and should be used as a first line tool in noninvasive family screening.

The $^{\text{QRSD ratio of V1 + V2 + V3}}$ / $_{\text{QRSD V4 + V5 + V6}}$ > than 1.2 is found in 97% of the ARVC/D cases.

The QRS complex may show IRBBB in approximately 18% of cases and CRBBB in 15%.

Epsilon Wave: These are low-amplitude and short-duration waves located near the end of the QRS complex and the beginning of the ST segment (in the J point), visible from V1 to V3, and rarely in the leads of the frontal plane. Right precordial epsilon potentials were found in 23% in standard ECG and in 75% in highly amplified and modified recording technique[27].

Epsilon waves were present in 56.3% of patients with M-VT and LBBB, in 4.8% of patients with P-VT and in none of the patients with M-VT and RBBB[28]***.*** It is not pathognomonic but characteristic of the entity. Present in 30% of cases. The R' wave may be mistaken or be an epsilon wave. They correspond to late potentials that can be translated into RV delayed activation.

Epsilon waves are considered major criteria of diagnosis of ARVC/D. This condition is characterized by the presence of delayed potentials, which appear after the end of ventricular depolarization. As recorded by epicardial mapping this post-excitation phenomenon may also be demonstrated either by an intracavitary electrode or sometimes on an amplified ECG.

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T wave: Negative T waves from V_1 to V_2 or V_3 are very characteristic when present in children over 12 years old in the absence of RBBB***[29]***

The most relevant ECG features are inverted T wave from V_1 to V_3 leads and wider QRS complex (>120 ms) in V1 lead[30]***.***

T-wave inversions in V_1 through V_3 were observed in 85% of ARVC/D patients in the absence of RBBB compared with none in RVOT and normal controls, respectively[31]***.***

It often presents as T-wave inversion in the anterior leads of the electrocardiogram[32]. Chagas disease is a major cause of morbidity and mortality in Latin America. The association on ECG of complete right bundle branch block, left anterior fascicular block and polymorphic premature ventricular contractions are the typical ECG features of chronic Chagas disease cardiomyopathy. Impaired left ventricular function, class III/IV, cardiomegaly, and non-sustained ventricular tachycardia indicate a poor prognosis in patients with chronic Chagas disease[33].

USEFULNESS OF ECG IN PERICARDITIS

Altered in 90% of cases;

There are 4 stages or phases described, present in only 50% of cases:

FIRST PHASE: ST segment elevation (<5 mm) of superior concavity. It is observed only two hours before chest pain and it lasts for several days. ST segment changes are *EXTENSIVE*

AND NOT TOO INTENSE, normally noticeable in several leads simultaneously, excluding V1. Occasionally, reciprocal alterations are observed in aVR [34; 35].

SECOND PHASE: ST segment returns to baseline and flat T wave.

THIRD PHASE: inversion of T wave, with no formation of Q wave.

FOURTH PHASE: ECG normalization with gradual reversion of T wave inversion.

USEFULNESS OF ECG IN CONGENITAL HEAR DISEASES DIAGNOSIS

In congenital heart diseases, the ECG may be very useful.

Secundum Atrial Septal Defect (ASD)

The ECG is commonly used as a screening tool for diagnosis of the ostium secundum ASD; thus, the presence of an incomplete or complete right bundle branch pattern is observed in approximately 97% of cases. ECG criteria for right ventricular enlargement are present in just more than over half of young patients with large ASDs. Although ECG is more sensitive in younger patients, it is unreliable as a screen test for this lesion [36].

Ostium primum ASD or endocardial cushion defects

The ECG shows in the horizontal plane incomplete or complete RBBB pattern with signs that suggest right ventricular enlargement and/or biventricular enlargement. Thus, in the right precordial leads V3R and V1, we can see triphasic patterns of the rsR' or rSR' type with opposite ST and T, indicating not very high pressure in the pulmonary artery. In endocardial cushion defects, the right branch is congenitally longer; a fact responsible for the IRBBB or Advanced RBBB pattern. In fact, it is a false branch block, since the pattern is due to a delay in RV activation, because the stimulus must go through a longer trajectory. In more than 98% of the cases, extreme deviation of SAQRS is verified concomitantly in the left or right superior quadrants. The latter is more frequent in the total form and showing a pattern of counterclockwise rotation of QRS loop in the frontal plane of the LAFB type. Indeed, there is no true LAFB, but there is early activation of the LV postero-inferior wall by early onset too, of the postero-inferior fascicle of the His bundle, associated to a hypoplasia and extension greater than the antero-superior fascicle, which delays even more the activation of the antero-superior wall of the LV[37].

Tricuspid Atresia

The association of right atrial enlargement("Gamboa P wave"), eventually very high ("Himalayan P wave"[38]) left ventricular enlargement, extreme left axis deviation (LAFB like) observed in cyanotic infants is very suggestive of tricuspid atresia

Ebstein's Anomaly

A giant P wave, prolonged or short P-R interval, and bizarre QRS complex with low voltage right bundle branch block (RBBB) pattern and initial q wave in V1 suggest Ebstein's anomaly of the tricuspid valve. The absence of RBBB in patients with Ebstein's anomaly and recurrent tachycardia had 98% sensitivity and 92% specificity for the diagnosis of an accessory pathway[39].

Ventricular Septal Defect (VSD)

Wide isodiphasism in intermediary precordial leads suggestive of biventricular overload. This pattern is considered characteristic of VSD and entitled Katz-Wachtel signal[40].

An ECG pattern of myocardial infarction with Q waves of necrosis in infants is very suggestive of anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) or Bland-White-Garland syndrome[41].

Dextrocardia

A negative or minus-plus P wave in I, deep Q waves in I and VL and reverse progression: R wave of decreasing voltage from V2 to V5 is suggestive of true dextrocardia[42].

USEFULNESS OF ECG IN ELECTROLYTIC DISORDERS DIAGNOSIS

Hyperkalemia

Pointed with increased voltage, symmetrical and narrow based T waves ("desert tent" wave) are characteristic of slightly increased serum potassium levels (above 5.7 mEq/L). With high potassium levels (mean 8.4 mEq/l) there is frequent absence of P wave. The stimulus originates in the SA node, it is conducted to the AV node through internodal bundles and reaches the junction without depolarizing the atrial muscle (P wave is not recorded). With very high potassium levels, widening diffuse QRS complexes are observed[43], similar to left or right bundle branch block, associated to anterior or posterior fascicular block by extreme shift of SÂQRS in the frontal plane to left or right. Brugada-like pattern may be observed.

Hypopotassemia

Gradual ST segment depression $\geq$ 0.5 mm is observed in II or from V1 to V3, decrease of T wave amplitude (flat T wave) or possible T wave inversion, prominent U wave and QTc interval prolongation[44].

Hypercalcemia

Almost non existent ST segment with short Q-oTc, Q-aT intervals are observed. Q-oTc is the interval from Q wave onset to T wave onset corrected according to heart rate. Hypercalcemia-induced ST-segment elevation mimicking acute myocardial infarction[45].

Q-aT interval is the interval between QRS onset to T wave apex. Values below 270 ms are diagnostic of hypercalcemia[46].

Hypocalcemia

The major ECG manifestation is the QT interval prolongation at the expense of the increase in ST segment duration, with no modification of T wave. The phenomenon is observed in hypocalcemia and in hypothermia[47].

USEFULNESS OF ECG IN HYPOTHERMIA DIAGNOSIS

Typical ECG features in a hypothermal patient include sinus bradycardia, atrial fibrillation (present in ≈50% of cases), PR interval, QT and QTc intervals prolongation, different types of arrhythmias (both supraventricular and ventricular) and the appearance of a very characteristic extra wave, called J wave, sign of “camel hump”, hump-like deflection, injury potential, and the eponym Osborn wave [48], located between the end of the QRS complex and ST segment onset. J wave is characteristic of hypothermia; however, is not pathognomonic, since it may be observed in other conditions with normothermia.

UTILITY OF ECG IN DRUGS EFFECTS DIAGNOSIS

Digitalis effect and digitalis intoxication

The earliest modification of digitalis effect on ECG or “digitalis action” are prolonged PR interval, ST segment: shortening and superior convexity (“in spoon”) by shortening of phases 2 and 3 of action potential (AP), QT and QTc intervals shortening. Digitalis action is the main cause of acquired short QT interval. T wave flattening with apiculate form of terminal portion is observed in 10% of cases, as well as possible symmetrical inversion of T wave (pseudo-ischemic T wave), and prominent U wave.

The manifestations that suggest digitalis intoxication are sinus bradycardia, sinus arrest, SA outflow blocks, E.g.: Wenckebach, AV block of all degrees with predominance of first degree (conduction slowing and prolongation of refractory period in AV node), of outflow, AV dissociation: dominant suppression of pacemaker with passive escape of a low junctional focus or inappropriate acceleration of subsidiary pacemaker or more rarely, dissociation within the AV node proper and atrial tachycardia with variable AV conduction; the most common ones and almost pathognomonic: sudden appearance of atrial tachycardia during digitalis therapy in patient with AF, accelerated junctional rhythm or nonparoxysmal junctional tachycardia with frequent isorhythmic dissociation, isolated or bigeminal extrasystoles, fascicular VT, AIVR or accelerated idioventricular rhythm, bidirectional or bifascicular VT, ventricular flutter and slow VF.

The main electrocardiographic modifications with amiodarone are sinus bradycardia, atropine- and isoproterenol-resistant by decrease of sinus automatism, which acts directly on the automatic cells of the SA node; i.e. not mediated by b stimulus or cholinergic effect. Heart rate reduction when administered orally, is around 20-30%. The drug is contraindicated in the presence of SA node dysfunction and with heart rate < 50 bpm[49].

In rapid endovenous administration, it may cause reflex tachycardia by drop of blood pressure. It may cause SA block or sinus arrest. It may improve RR variability (HRV) parameters in post-AMI patients[50]. I It may cause bradycardia or tachycardia in cases of dysthyroidism secondary to use of the drug (present in 10-20% of cases)[51].

It may cause worsening of CHF symptoms when administered endovenously (2%). The drug administered in endovenous bolus is pretty safe and more effective than digoxin to control heart

rate and conversion to sinus rhythm, in patients with atrial fibrillation and high rate of ventricular response.

PR INTERVAL: it may prolong it in approximately 1/5 cases, mainly when high doses are used[52].

QRS DURATION: not significant. It may probably prolong it, particularly with high rates by the blocking effect of inactive channels of Na+, a fact that would account for depression in phase 0. This is the reason why high resolution ECG cannot be used in patients that take amiodarone, since the drug finally causes filtered QRS prolongation and appearance of potentials (LP) in patients with previously normal high resolution ECG**.**

T WAVE: it causes T wave broadening, flattening and notch. Ventricular repolarization alterations in ECG manifest approximately at the fourth day after the treatment starts orally, and persist for three weeks after interrupting the drug[53].

QT AND QTc INTERVAL: it causes marked and constant prolongation of these parameters[54]. This effect reaches its maximum only 10 weeks after the treatment starts[55]. In spite of prolonging the QT interval, it rarely triggers the feared Torsade de pointes, since it causes decrease of transmural dispersion in ventricular wall thickness due to its unequal effect on epi, endo, and M cells.

U WAVE: it may be prominent: > 1.5 mm with the effect of the drug.

The 12-lead electrocardiogram (ECG) has emerged as a popular bedside tool in the evaluation of acute tricyclic antidepressant (TCA) toxicity. Although the history and physical examination play a key role in the assessment of the patient with potential TCA poisoning, the presence or absence of features of TCA toxidrome are not sufficient to detect or rule out toxicity from this class of drugs. A variety of ECG findings occur with TCA toxicity. An ECG or rhythm strip, if available, should be checked during the prehospital assessment of a TCA overdose patient. A wide-complex arrhythmia with a QRS ≥ 100 ms is an indicator that the patient should be immediately stabilized, given sodium bicarbonate if there is a protocol for its use, and transported to an emergency department (Grade B of evidence). In a TCA overdose patient all ECG measurements are greater and remained abnormal for a significantly longer duration in those patients who develop seizures and/or ventricular arrhythmias. The conduction abnormalities seen in severe TCA toxicity vary widely in the time observed for resolution of these abnormalities and sometimes remain persistently abnormal[56].

The percentage of patients showing ECG changes and respiratory depression is higher when other drugs such as ethanol were ingested along with antidepressants than when only antidepressants were taken[57].

In acute poisoning with TCA are observed the following ECG parameters:

SINUS TACHYCARDIA: Heart rate (HR): of 100 or more is the most frequent ECG finding is sinus tachycardia (40.7%)[58]. Sinus tachycardia is due principally to anticholinergic effects[59] and inhibition of norepinephrine uptake by tricyclic antidepressants[60].

PQ INTERVAL PROLONGATION: The principal mechanism of toxicity is cardiac sodium channel blockade, which increases the duration of the cardiac action potential (AP) and refractory period and delays atrioventricular conduction.

RIGHTWARD SHIFT OF THE TERMINAL QRS 40 MS (T40) OF THE FRONTAL PLANE QRS COMPLEX VECTOR. QRS (T40) AXIS: Frequent right-axis deviation of 130 degrees to 270 degrees in the terminal 40-ms frontal plane QRS axis (T40-ms axis) of ECG. The sensitivity and specificity of the T40 axis were found to be only 29% and 83%, respectively[61]. T40-ms axis is a better indicator of tricyclic antidepressant toxicity than QRS interval prolongation[62].

PROLONGATION OF QRS COMPLEX DURATION: QRS duration ≥ 100 ms. is observed in ≈ 60% of cases[63]. TCA blocks sodium membrane channels with slowing of membrane depolarization, thus having quinidine like effects on the myocardium[64]. The electrophysiological effects that antidepressants exert on ion channels may affect the cardiac AP, lengthening both depolarization (QRS) and repolarization (ST-T) phases.

R WAVE IN VR Lead: Prominent R wave is observed in patients with significant tricyclic antidepressant poisoning[65].

ABNORMAL ST-TRACT ELEVATION IN THE RIGHT PRECORDIAL LEADS ASSOCIATED WITH MARKED QRS WIDENING (RIGHT BUNDLE BRANCH BLOCK AND LEFT ANTERIOR FASCICULAR BLOCK TYPE) [66]

NONSPECIFIC ST SEGMENT AND T WAVE CHANGES:

CORRECTED QT INTERVAL (QTC) PROLONGATION calculated with Bazett's (QTc(B)) and Hodges's (QTc(H)) formulas: in > 53% of cases. Inhibition of the human ether-à-go-go-related gene (HERG) potassium channel by tricyclic antidepressants is the main responsible for QTc prolongation.

BRUGADA LIKE REPOLARIZATION PATTERN after TCA ingestion is rare, and death or dysrhythmias did not occur. However, patients with Brugada like repolarization pattern are likely at increased risk for TCA-induced complications [67].

PSEUDO INFARCTION PATTERN[68].

ACUTE MYOCARDIAL INFARCTION[69].

CARDIAC ARRHYTHMIAS TENDENCY: Premature ventricular contractions, isolated, couplets and triplets, ventricular tachycardia and torsade des pointes (TdP) are described.[70]. Slowing repolarization can lead to early after depolarizations and TdP[71]. The prolongation of the QTc interval due to the inhibition of potassium channels, leads to the risk of developing TdP, which can result in sudden cardiac death (SCD). Amitriptyline and maprotiline are the TCA most likely to provoke TdPs which can evolve into ventricular fibrillation and SCD. Risk factors of family history of congenital long QT syndrome, age, female sex, metabolic and cardiovascular disease, metabolic inhibitors, hypokalemia, drug overdose, and co-prescription of drugs associated with QTc interval prolongation are found in cases of TdP associated with TCA. Clinicians should be cautious when prescribing TCA with other drugs, such as thioridazine, that are capable of prolonging the QT interval. [72].

USEFULNESS OF ECG IN ACUTE PULMONARY EMBOLISM[73;74;75].

ECG could be normal in acute pulmonary embolism (APE) (15%). When present, the electrocardiographic manifestations are usually early, transitory, and always a consequence of the following factors:

- Sudden RV systolic overload by pulmonary hypertension.

- Sudden positional changes of the heart: clockwise rotation in its longitudinal axis (dextrorotation) and verticalization by descent of diaphragm.
- Acute dilatation of the right atrium, which causes multifocal premature atrial contractions, acute atrial fibrillation, atrial flutter, etc.
- Hypoxia and subsequent ischemia, and myocardial lesion by coronary spasm, and appearance of arrhythmias: different degrees of sinoatrial blocks, nodal rhythm, AV dissociation, sinus arrest, etc.

ECG in APE is characterized by:

1) Being transitory, which justifies performing serial ECGs.
2) Sinus tachycardia.
3) Different types of supraventricular arrhythmias, especially, acute atrial fibrillation.
4) SAP rightward shift with apiculate P wave.
5) SAQRS rightward shift, or perpendicular to the frontal plane.
6) Deep Q waves only in DIII.
7) S waves in DI and aVL with depth >1.5 mm.
8) Low voltage of QRS complexes in limb leads (<5 mm).
9) Transitory incomplete or complete right bundle branch block (IRBBB or CRBBB).
10) Pseudo anterior or inferior infarction pattern.
11) McGinn White pattern: S1-Q3-T3. Deep S wave with duration of up to 30 ms in DI, deep Q, and late inverted T wave in DIII.
12) ST segment elevation or depression in right precordial leads.
13) Ischemic inverted T wave in right precordial leads.

USEFULNESS OF ECG IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

P wave presents increase of voltage (P-pulmonale): Marked by tall (>2.5 mm in the DII lead), narrow, peaked P wave, and with SAP shifted to the right, always at the right of +75º.

QRS shows a shift of the transition area to the left, with complexes of the rS or even QS type, from V1 to V6, which may resemble electrically inactive area in the anterior wall.

Most of ventricular depolarization forces are shifted backward and rightward (Type C or essential right ventricular enlargement). Schamrot reports the ECG "Lead I sign," pathognomonic of emphysema, and characterized by: SAP, SAQRS, and SAT near +90º [76].

USEFULNESS OF ECG IN CHANNELOPATIES OR ELECTRICAL HEART DISEASES

Purely electrical heart diseases are genetic or sporadic channelopathies responsible for a large number of sudden, unexpected deaths in otherwise healthy, young individuals in absence of any apparent demonstrable structural heart disease, even with invasive methods (except for biopsy), and not related to ischemia, electrolytic imbalances, and action of drugs[77;78].

CONGENITAL LONG QT SYNDROME[79; 80]

ECG is very important in the diagnosis of Congenital LQTS.

The main features are: low HR for the age, long QTc interval, T wave with notches, broad base, low or eventually giant, alternant polarity, prominent U wave and TdP tendency. ST segment prolongation is observed in the LQT3, variant caused by mutations of the SCN5A gene for the

sodium channel. A gain-of-function mutation causes persistent inward sodium current in phase 2 (plateau phase).

LQT1 is characterized by broad-based prolonged T waves. T duration is particularly long in the chromosome 11 genotype (lead II). T wave with low amplitude is observed in the LQT2 variant. There might be giant T waves; and notched T wave in three leads. T wave with notched appearance is typical of the LQT2 variant.

T wave Alternans is an arrhythmic marker.

Prominent or giant U wave is more evident from V4 to V6. There may be alternant U wave polarity during low HR. U wave increases its voltage during slow rates or after pauses[81].

QT intervals corrected for heart rate (QTc) longer than 450 ms in men and 460 ms in females are generally considered prolonged[82].

BRUGADA SYNDROME

BrS is characterized by a typical clinico-electrocardiographic pattern, and an increased risk of sudden cardiac death (SCD) as a result of very fast polymorphic ventricular tachyarrhythmia (PVT)/ventricular fibrillation (VF) in patients with an apparently structurally normal heart. Both repolarization and depolarization abnormalities in the walls of both ventricles, especially in the RVOT, constitute the hallmark of BrS mainly in those patients with ventricular arrhythmias[83-84-85].

First-degree AV block is observed in ≈ 50% of cases of BrS mainly in the presence of SCN5A mutation. Sometimes, incomplete right bundle branch block (IRBBB) or complete right bundle branch pattern (CRBBB) is observed in BrS. Frequently these patterns are atypical and characterized by absence of wide final S wave in left leads (DI, aVL, V_5 and V_6) and without broad terminal R wave in the aVR lead. The phenomenon produced the confusion of the names of “pseudo RBBB” and “RBBB like” [86]. True RBBB requires terminal wide S wave in left leads; I, VL, V_5 and V_6 and terminal broad R’ or R broad final wave in VR.

A typical ECG finding consists of ST-segment elevation in the right precordial leads (V_1-V_2) or anteroseptal wall (V_1-V_3) sensitive to the RVOT[87]. Since the first Consensus on BrS, three electrocardiographic patterns are acknowledged, called types 1, 2, and 3[88-89-90].

Only type 1 –much rarer- is diagnostic and characterized by presenting in the right precordial leads (V_1 and V_2) or in the antero-septal wall (V_1-V_3) of ECG, coved type or descending rectilinear oblique ST segment elevation ≥ 2 mm followed by T wave of negative polarity (configuration of ST-T in V1 and V2 due to a high take-off J point giving rise to a downsloping ST segment that is followed by a negative T wave).

Type 2 pattern displays in the right precordial leads (V_1 and V_2) or from V_1 through V_3, elevation of the J point and the initial portion of ST segment of ≥ 2 mm, and in the terminal portion ≥ 1 mm with saddleback appearance, followed by positive or biphasic T wave.

In type 3, the ST segment also has a saddleback or coved appearance, with elevation in J point and the onset of ST segment ≥ 2 mm and terminal portion ≤ 1 mm followed by positive T wave.

Types 2 and 3 are found as normal variants with a high rate, being included in the group of right end conduction delays by the fascicles of the right bundle branch of the His bundle[91].

Atrial arrhythmias have been reported in patients with BrS syndrome in ≈ 20% to 30% of patients[92].

A steep repolarization gradient in the epicardium but not the endocardium develops episodes of phase 2 re-entrant (P2R)-extrasystoles and subsequent self-terminating Polymorphic VT which might degenerate into VF by further depolarization and repolarization abnormalities. Conduction velocity restitution is shifted lower and APD restitution is more variable in VF cases than in Polymorphic VT cases in canine model of BrS[93]. The risk of arrhythmic events is higher in previously symptomatic patients and in those displaying a spontaneous type 1 ECG pattern. One third of patients having suffered from syncopal episodes or resuscitated from near-sudden death develops a new episode of VT/VF within two years. Recent publications have shown that IVF is often caused by premature ventricular contractions (PVCs) arising from the right ventricular outflow tract (RVOT) or the Purkinje system. Some cases are associated with unbalanced sympathetic cardiac innervation[94]. Some other cases are now believed to be a variant of the BrS. All VT/VF episodes are associated with ST-segment elevation and are initiated by short-coupled PVCs.

USEFULNESS FOR DETECTION IN ARTEFACTS

Artifact: Electrical activity displayed on graph paper that is superimposed on cardiac tracings, interfering with the interpretation of the rhythm; it may be caused by outside electrical sources, muscle tremors, patient movement; also called interference[95-96-97].

Main types of artifacts:

1) Misplacement of electrodes

Electrocardiographic (ECG) artifacts resulting from the misplacement of electrodes are frequent, difficult to detect, and can become of clinical importance. Errors in ECG performance do occur with an increasing frequency in an acute medical care setting. The frequency of ECG artifacts due to switched electrodes is 0.4% at the outpatient clinic and 4.0% at the intensive care unit[98].

> **(1.a) Limb electrode misconnection:** Pseudo dextrocardia by exchange of limb electrodes. All P, QRS, and T waves are negative in DI, but normal progression of QRS in precordial leads rules out this hypothesis, pointing out the exchange of arm electrodes.
>
> **(1.b) Precordial electrode misplacement:** Placement of electrode of V1 as V3 and vice-versa. Because in some devices they have the colors red and green respectively, the error could be made by a color-blind technician. This exchange originates R wave with increased voltage in V1[99-100].

Lead misplacement was suspected when one of the following morphological changes occurred:

- QRS axis deviation between ±180º and – 90º;
- Positive P wave in lead VR;
- Negative P waves in lead I and/or II, very low (<0.1 mV) amplitude in an isolated peripheral lead. Very low amplitude of the QRS complex in lead I, II, or III is observed for electrode misplacement in half of the cases[101].
- Abnormal R progression in precordial leads.

- Pseudo dextrocardia by exchange of limb electrodes. All P, QRS, and T waves are negative in DI, but normal progression of QRS in precordial leads rules out this hypothesis, pointing out the exchange of arm electrodes.

2) Great precordial electrode

Cause: use of conductor gel in the precordium belt.

Electrocardiographic alteration: ECG tracing equal from V1 through V6, that corresponds to the average of potentials in those leads.

3) Tremor

Cause: Parkinson disease and Parkinsonisms[102].

Electrocardiographic alteration: it resembles tachyarrhythmias as atrial flutter (pseudo-flutter) with irregularity sometimes constant and gross in the baseline, with a rate close to 300 oscillations per minute, caused by muscular tremor or even ventricular tachycardia. The placement of electrodes in the roots of limbs lessens the interference caused by myopotentials and it enables the true rhythm of the patient to manifest in ECG. Electrocardiographic artifacts simulating atrial flutter in patients undergoing continuous veno-venous hemodialysis are described. As end-stage renal disease and hemodialysis are associated with atrial tachyarrhythmias, physicians should be aware of the possibility of such artifacts induced by the dialysis equipment itself[103].

VT is mimicked when myopotentials originate oscillations of high frequency and great voltage. Additionally electrocardiographic artifacts mimicking VT could be observed during high-frequency oscillatory ventilation[104].

4) Electrical interference

Cause: inappropriate grounding.

Electrocardiographic alteration: oscillations in the baseline mimicking ventricular repolarization alterations or causing very bizarre ST/T alterations[96].

5) Artifact mimicking atrial tachycardia

ECG with sinus tachycardia. In prolonged DII tracing, the P wave becomes negative, but HR is not modified. P becomes negative, but HR does not change.

Limitations could be dangerous if we trust too much on a normal ECG, and neglect the clinical setting. The presence of a normal ECG does not rule out the presence of coronary artery disease; moreover, we should remember that there are infarctions without the presence of q wave, and without alterations of ventricular repolarization. The method has low sensitivity in the diagnosis of ventricular enlargements. A normal ECG does not imply the presence of a "life insurance". It is extremely important to be aware of the existence of false positives and false negatives.

In the release to practice sports, a normal ECG does not imply the absence of risk. The release without another, more refined test, like stress test or echocardiogram will depend on the clinical setting. We should always take into account the presence of factors that may influence on the

ECG, like: gender, weight, race, physical type, chest deformities (pectus excavatus, straight back, mastectomized), practice of competitive sports, hypothermia, hyperventilation, glucose injection, alcohol ingestion, etc[105-106].

The presence of artifacts: shaking, Parkinson, small children, accidental exchange of electrodes, bad standardization (average), inappropriate paper velocity.

The interpretation of ECGs should always be made considering the clinical setting.

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