**E-Journal of Cardiology 2012; Vol 1, No 2**

**www.e-journalofcardiology.com**

# New electrophysiological mechanisms of electrical instability of heart, defibrillation and hypertension.

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## Abstract

Reasons of development of life-threatening arrhythmias that lead to sudden and total death in cases of cardiovascular diseases are still unclear, though cardiologic researches of this problem are conducted throughout the world. Damage of connective tissue's insulation cover of the heart's conductive pathways with oxidation of ectopic nodes can lead to life-threatening arrhythmias. This damage has never been considered before as a cause of electrical instability of the heart.

Speed of conducting electricity from the heart to the nervous system and its distribution throughout the body remains underestimated. Slowdown of electric magnetic induction (acceleration) between the heart and the central nervous system as well as slowdown of its distribution results in insufficient speed of electric flow and reduction of bioelectric processes of the body. This leads to activation of the sympathetic nervous system with the subsequent cascade of pressor mechanisms and development of essential hypertension.

**Keywords:** electric instability of heart, paroxysmal tachycardia, flutter, fibrillation, defibrillation, hypertension, nervous system, syndrome WPW-CLC.

Cardiovascular diseases (CVDs) are major causes of disability and death in working-age population worldwide; that calls forth the need for modern effective methods of diagnostics, treatment and preventive measures for cardiac electrical instability (CEI).

In younger age flutter and fibrillation occur in patients with rheumatism, myocarditis, and mitral valvular disease; in elderly and old age it mostly occurs in patients with ischemic heart disease

(IHD), myocardial infarction (MI), chronic heart failure, mitral stenosis, hypertension, cardiomyopathy, myocarditis, etc. [14].

Structural pathology of the heart (SPH), which leads to development of unstable substrate due to the influence of different functional factors, determines the condition for occurrence of fatal arrhythmias. Structural changes resulting in development of life-threatening arrhythmias (LTA) are as follows: manifested hypertrophy, dilatation, cardiac aneurysm, necrotic and sclerotic myocardial processes, inflammation with myocardial tissue oedema, etc. According to the data of various researches, these changes comprise anatomic substrate with different mechanisms of LTA development [4].

Many authors of CEI studies focus only on research of necrotic-cicatricle processes (NCP) in myocardium at IHD. However, development of general sudden cardiac death (SCD) also occurs in patients with other CVDs, this fact is somehow ignored by these authors.

It should be noted that almost all issues involved in arrhythmias are easy for analysis, except for the reasons and development mechanism of flutter and fibrillation. The definition of atrial fibrillation is also confusing due to certain contradictions in definitions presented in various textbooks on electrocardiography (ECG).

According to such definitions [14.7], atrium and ventricle flutter is a regular rhythmic motion of a powerful excitation wave going along the same path with the development of re-entry (however, reasons and mechanism of macro-re-entry still remain unclear).

Atrium and ventricle fibrillation is random, chaotic excitation and contraction of separate groups of fibre muscles. Each of them, in fact, is a certain ectopic impulse centre (probably, this definition presented in textbooks on ECG is not entirely correct for students and young scientists).

Having read this definition and then going back to chapter “Automaticity function” one can read that automaticity is a function of sinoatrial node (SAN) cells and conducting system of the heart: atrioventricular (AV) block, conducting system of atriums and ventricles. However, contractile **myocardium does not have function of automaticity** [14.7]. This means that in case of fibrillation arrhythmia “separate groups of fibre muscles” cannot generate electrical impulses. Even if myocardium or cardiomyocytes change their properties and “electrical instability of myocardium” does not exist, the myocardium only conducts electrical impulses.

Some may argue that even if the myocardium does not have electrical activity, there might be a non-homogeneous area in the myocardium connected with NCP, which can interfere with conductivity of electricity. Electrical impulses can easily bypass this zone, even in case of extensive MI, if there is no serious damage to the conductive pathways of the heart (CPH). Fibrillation arrhythmia occurs due to the multiple formations of ectopic centres (EC) of less power, which are located in distal areas of CPH. This results in colliding of excitation waves and development of micro-re-entry mechanism. The myocardium cannot really generate electrical

impulses and ectopic nodes are not scattered chaotically on the myocardium as some people might think. All EC are located along CPH as beads on a thread and have unified connective tissue insulation cover (CTIC), which begins from SAN. If this cover is not damaged electric impulses cannot get to myocardium. Purkinje fibres are the only fibres that do not have such cover and excitation is conducted to myocardium via them [2].

It is necessary to note that CPH are laid in the same manner as electric cables are laid in the walls of our houses, moreover, they have insulation cover as well. However cables in the walls are motionless and they do not wear out much. CPH in a working myocardium with SPH may wear out quickly and cause blocking and/or damage of CTIC.

Excessive stretch may occur in case of manifested SPH. That leads to tearing or rupture of CTIC of one of the main proximal CPH. This causes oxidation and activation of the nearby powerful EC with development of flutter. Therefore, when a powerful electric impulse goes via such damaged area, most of the impulse untimely breaks through onto the myocardium along the path with the least resistance and large F waves of flutter are formed.

The matter is that if CTIC is not damaged, CPHs are not subjected to oxidation. Purkinje described transitional T-cells, which are located between conductive B-cells (they were later called Purkinje cells) and the myocardium [2]. He thought that their main function is to conduct electric impulse. However, their main function is to create an anti-oxidation barrier for conductive B-cells. Myocardial extracellular fluid should not get into CPH, especially during hypoxia and acidosis of tissues. There should be an adapter between two conductors of different structure which will not allow them to be oxidized. If to exclude T-cells from this link, conductivity of electric impulses between B-cells and the myocardium remains at the former level at any time, but Purkinje fibres and EC will be subjected to oxidation that will lead to development of acute CEI and in due course to consequences of chronic, irreversible oxidation. In case of a sudden event, transitional T-cells in the area of damaged CTIC are not stipulated by the nature. It leads to fast oxidation and irritation of proximal EC with development of flutter. Further oxidation process will reach others EC distal areas and flutter will gradually develop into fibrillation.

One may ask a question if the suggested mechanism of flutter development is true. The most potent source of ectopia at paroxysmal supraventricular tachycardia and atrial flutter is located in the atria (fibrillation arrhythmia does not count, as it is caused by multiple ectopia). However ECG reveals different picture. In the event of paroxysmal supraventricular tachycardia an electric impulse has to overcome obstacles to get through a lot of small conductive paths and ectopic nodes in atria. Then it reaches the myocardium as a less potent impulse so that to spin a macro re-entry wave. In case of atrial flutter a potent electric impulse reaches the myocardium without any obstacles via damaged CTIC and spins a macro re-entry wave.

This mechanism can be compared with WPW syndrome. Only in this case most of the electric excitation wave which has broken through the damaged CTIC covers a smaller wave which has

gone through CPH; F wave of flutter is a delta of the myocardium pre-excitation wave. Such potent output of electric excitation to the myocardium of atria assists faster decrease in capacity of electric pressure in conductive paths and EC that shortens the refractory period. It results in a higher heart rate (HR) in the case of atrium flutter than in the case of paroxysmal supraventricular tachycardia. Interruption of atrium flutter with restoration of the sinus rhythm will be marked on the ECG with a widened peak P (more than 0.13 seconds). The peak P may be simply expanded or two-phase and two-humped. There will be a delta wave, but it will merge with P peak and become almost unnoticeable.

I have described what happens in the atria as an example. However, exactly the same process occurs in the ventricles. Therefore, it is possible to tell with confidence that flutter is a special type arrhythmia, typical only for SPH with damaged CTIC. Without SPH the activity of the brain is decreased and SAN is affected by the nervous system. That results in its weakness and activation of EC of the ventricles with development of extrasystolia and tachycardia and further possibility of development of fibrillation; this process will occur without oxidation of EC.

What causes development of paroxysmal tachycardia?

Damage of CTIC CPH of the upper parts of atria and ventricles (conduction pathways of Wenckebach, Bachman and Thorel, transitional AV node, the bundle of His and its peduncles in the ventricles) may be not so serious and may result only in cracks and pores. In this case, EC will be oxidized only periodically, not as often as in the case of fibrillation when more serious damage such as tear or rupture of the CTIC occurs. Respectively there will be no pre-excitation of the myocardium in paroxysmal tachycardia because pores and cracks of the CTIC will not be able to conduct potent pre-excitation electrical impulse. Most often development of paroxysmal tachycardia will cause gradual development of pores or cracks into rupture of CTIC CPH which will result in flutter but the source of the ectopia remains the same. This is one and the same type of arrhythmia but with a different mechanism of action.

In most cases flutter does not precede fibrillation arrhythmia. This is due to the fact that CTIC of the media distal areas of CPH is damaged most of all in the case of fibrillation (Purkinje branches and fibres of atria and ventricles), especially at NCP, multiple damage of transitional T-cells is also possible. This results in oxidation of not one but several less potent ECs and development of micro re-entry Even if the cordial pathology is not diagnosed at examination, especially in young people, and there is nothing, except for paroxysmal tachycardia, it may be connected with pathology of other organs or exogenous intoxication influence on a myocardium that leads to microscopic damages of CTIC with oxidation of EC.

Any sudden movements or myocardial damages such as all types of tachycardia, extrasystolia, development of blockades, acute increase of blood pressure (BP), increase of myocardial contractility (especially in case of pathologically decreased contractility) and NCP can become a triggering factor for the damage of CTIC CPH at SPH. As for localization, the most subtle and easily damaged segments of the ventricles are the right bundle and the front branch of the left

bundle. Then in descending order of their sensitivity we can put the main branch of the left bundle, His' bundle, and finally the back branch of the left bundle. However, abnormality of conductivity can take place in any segment or multiple segments simultaneously [7].

In some cases flutter and fibrillation of atria can be interrupted at an early stage by conducting electric cardioversion (paroxysmal, persistent). Thus, there is a powerful stimulation of SAN which suppresses other centres of generation; oxidative effect on EC becomes insignificant for a while and a small damage of CTIC is gradually regenerated. The use of high doses of anti-arrhythmia drugs during the initial stages of flutter and fibrillation can suppress irritating activity of EC with restoration of sinus rhythm and gradual repair of small CTIC damages.

It is necessary to note that restoration of sinus rhythm depends not on the size of the CTIC damage but on the duration of EC irritation and the degree of reversibility of the oxidation process.

Preventive use of repairing drugs in patients with SPH, and especially in those with paroxysmal tachycardia can assist in prevention of more serious damage to CTIC CPH with development of trembling and fibrillation. However, the main treatment of patients with paroxysmal tachycardia, trembling and fibrillation should be of the same kind – electric pulse therapy, ablation of CTIC damages and anti-arrhythmia drugs.

Additional prescription of alkalizing, oxidation-restoration drugs and repairing preparations with exclusion of aspirin and other oxidizing means can assist in fast and stable restoration of the heart's electric stability.

To continue discussion of the subject regarding CTIC and conductive system I would like to add some more comments. It is considered, that during defibrillation procedure electric current goes directly to the heart which results in a myocardium's spasm and EC impulse, that leads to the prolonged refractory period and restoration of SAN work [6], but this is a mistake! The point is that electric discharge of defibrillation cannot enter the myocardium from the outside anatomically and electric impulse cannot come out of it because epicardial and intracavitary endocardial layer of the heart has CTIC. Most likely, electric discharges affect the heart; via multiple nerve receptors of the thorax electric impulses reach central nerve system. From there activation of brain goes in all directions, including the sympathetic nervous system (SNS) of the heart, SAN in particular, and stimulation of V-adrenoreceptors with the release of catecholamine (adrenaline, noradrenaline). For some reason nobody really thought that external electrical currents can also pass along the nerve pathways.

Now I am going to present a theory that might seem to be incredible to many scientists, but it has the right to exist. It is still unknown where and how electric impulses are generated in the nervous system. Most likely, electric impulses are formed not in the nervous system but they get there from the AV node immediately after excitation of ventricles. The pathways to atria are blocked and the electric impulse will not enter the ventricles until electricity is gone to the

nervous system. Therefore AV node does not conduct more than 180-220 electric impulses per minute performing the earthling function for the heart. Then electric energy is conducted along leading (esodic) nerve fibres to the central nerve system and from there to the whole nerve system. Of course, one must realize that there will be nothing in common in the records of ECG and electroencephalogram (EEG) except of the rhythm of excitations of certain EEG waves (fig. 1) because these methods reflect excitations of different structures of the body. This circular connection between the heart and the nervous system is more reasonable than the existence of two separate from each other electrical systems in one body that would have inevitably come across each other and would have led to short-circuits.

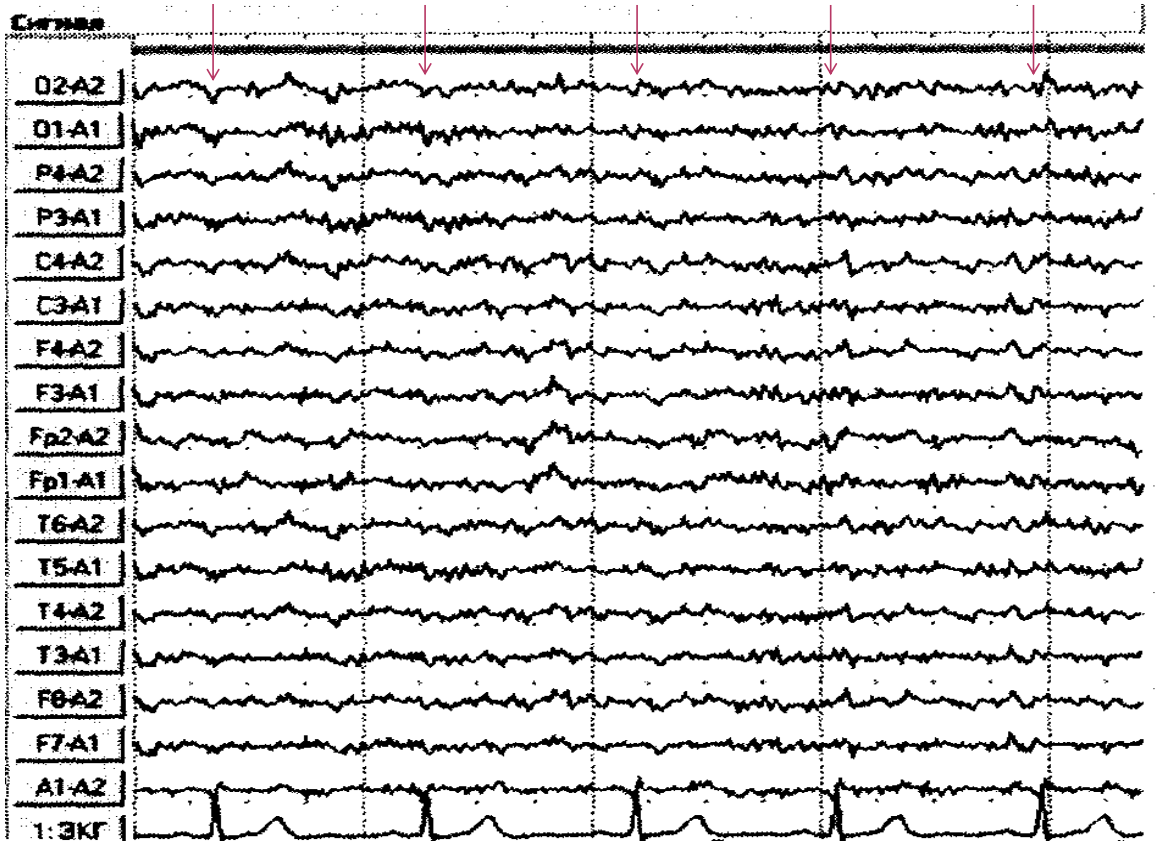


Figure 1. Red arrows above the EEG mark rhythmic cycles

which correlate with HR at the lower part of the ECG.

I am going to ask all cardiologists a question, the answer to which may serve as a proof that electricity goes from the heart to the nerve system (somehow nobody has thought about this question before): *w*hat happens to the part of electricity which is blocked at AV blockade as it does not reach ventricles and repeated excitation does not occur?

Speed of conducting electricity from the heart to the nervous system and its distribution throughout the body remains underestimated. With age, the magnetic field of some people is weakening; probably hereditary predisposition plays a certain role in this process. Slowdown of electric magnetic induction (acceleration) between the heart and the central nervous system and its distribution results in insufficient speed of electric flow and reduction of bioelectric processes in the body. This leads to the activation of the sympathetic nervous system, with the subsequent cascade of pressor mechanisms and development of essential hypertension. Probably creation of artificial magnetic field in the transit zone between the heart and the brain at the level of the neck in the form of a piece of jewellery (magnetic necklace) or other type of magnetic therapy would assist to restore this field and to induce electricity in patients with essential hypertension.

It should be noted that passive operation of the nervous system can be carried out without electricity as it happens in case of a heart transplant, when a patient is on an artificial circulation for a couple of hours. EEG is recording passive brain work, which should give an impulse for starting-up of the SAN of the implanted heart, although in some cases this does not happen and defibrillation procedure is carried out.

Review of the literature containing the main opinions of authors and researchers on CEI issue resulted in the following notes. First of all, it is necessary to mention that many authors write about electric instability of the myocardium thought the term “electric instability of heart” is more appropriate.

Combination of several causes which predispose occurrence of electrical instability of the myocardium is necessary for development of LTA: presence of the substrate (structural heart disease), modulating the dysfunction of the autonomic nervous system and triggering factors of LTA. The morphological substratum causing non-homogenous conduct of an impulse after MI is a borderline area of myocardium located next to necrosis tissue of the myocardium formed by viable myocardial fibres connected with each other and a connecting tissue. Impulse conducting pathway is prolonged here due to areas of connecting tissue which become obstacles for an excitation wave and the speed of conducting is slowed down due to the breach of the parallel orientation of muscle fibres. Thus, the area of infarction with delayed ventricular depolarization may be an anatomical and physiological substrate for re-entry – the primary mechanism of LTA development [5.8].

The results of research conducted by J.D. Kramer et al. give evidence that a long path of rotation pulse is not required; a small diameter of myocardial tissue with electrophysiological properties modified due to acute myocardial ischemia or heterogeneity of its structure as a result of fibro-necrotic changes is sufficient to start the re-entry mechanism [12].

In the study conducted in patients with implanted cardioverter-defibrillators, a sudden occurrence of ventricular tachycardia (VT) or ventricular fibrillation (VF) as a rule was observed in case of preserved fraction of ejection; gradual strengthening of ventricular ectopic activity is usually noted at reduced contractility prior to VT or VF attacks [17].

There is no doubt that the presence of heart failure is an important arrhythmia generating factor and risk marker of sudden arrhythmia death in patients with ischemic heart disease [3]. A heart aneurysm, post infarction cicatrices and clinical manifestations of cardiac insufficiency increases the likelihood of adverse outcome. Decreased left ventricular contractility increases the risk of SCD not only in case of IHD, but also in patients with other diseases of the heart [9.11]. Ejection fraction of less than 40%, unstable ventricular tachycardia (VT) at Holter monitoring and electrophysiological examination of patients who have had acute MI remain major predictive markers of a high SCD risk [4].

Combination of two SCD risk factors is particularly unfavourable -frequent ventricular extrasystole and dysfunction of left ventricle with decline of ejection fraction decline < 40%. According to the data of GISSI-2 study, the risk of sudden arrhythmia death in this case is 16 times higher [10.13].

Decrease in variability of the heart rhythm (VHR) is the major marker of a sudden death risk factor; such factors as increase of duration of QT interval's dispersion and presence of ventricle late potentials (VLP) are also important. [16].

As for VHR, QT interval dispersion and VLP, I would like to add some final conclusions from my dissertational thesis. Initial deterioration of VHR in patients in post infraction period is due to anxiety-depressive condition after acute MI. Depression is observed in 82% of patients in post infraction period [15]. Patients develop fear of death and anxiety for their health; they lack emotions of joyful perception of the world around them and become subdued which results in prevalence of CNS activity over parasympathetic nervous system (PSN). Activation of positive psycho-emotional state of patients is combined with overcoming depression and stabilized PSN functioning, resulting in variations of sinus rhythm. It should be noted that positive psycho-emotional state of the patient who has had MI may change to the negative state and even stress at any moment and this may lead to CNS hyperactivity, which combined with atherosclerotic changes in coronary arteries can result in spasm and occurrence of new necrotic areas in the myocardium. Therefore nervous regulation of SAN is more stable due to simultaneous participation of sympathetic and parasympathetic sections of autonomic nervous system, and VHR research method has no practical information [1].

VLP improves less significant in post infraction period than index of QT interval dispersion. This might be due to the fact that presence of VLP is connected with CPH blockade (branches of His bundle and/or major stems of Purkinje fibres) which are completely blocked by non-homogeneous necrotic area and later by cicatricial changes of myocardium that makes an electric impulse return and come out via other CPH to the myocardium [1].

Slowdown of dispersion of the QT interval in the acute stage of MI is more often connected with the presence of necrosis and re-infarction area of myocardium which has 20-40% more of non-homogeneous area than in the cicatrice period, which subsequently leads to the improvement of d repolarization processes. It is possible that in those few cases when indicators of PPL are

improving in post infarction period there may be a connection between these rare cases with the ability of the body to produce stem cells, which results in partial growth of new CPH (branch of His bundle or Purkinje fibres), bypassing the scar zone, resulting in the recovery of electric impulse conduction and elimination of PPL [1].

In conclusion, I should note that the present knowledge about the formation of the conducting system is incomplete. For example, progress in the researches of additional ways of conducting electrical impulses was only achieved thanks to the ECG and it is not a proof - nobody has ever seen muscle bundles in the human heart. In experiments on rabbits, researcher Kent watched some muscle bundles in the fibrous ring of the ventricles. Researchers Wolff, Parkinson and White whilst observing syndrome of pre-excitation on the ECG with delta wave, assumed its connection with Kent muscle bundles and called it by their names [2]. To have a complete picture of all myocardium features and electrophysiology of the heart, one should know that there is a connective tissue frame between atria and ventricles which does not allow ventricles to be stimulated together with atria. It is in this particular frame where a congenital opening defect of a various size is located; this is what is cauterized at ablation but not an additional conductive pathway (Kent's bundle). Increase of pressure in an atrium or a ventricle, or probably in both of them, causes this defect to open and electrical impulses are periodically conducted from the atria myocardium to the ventricles myocardium (WPW syndrome). Additional conductive pathway is also absent at CLC syndrome (James bundles), but there is a congenital periodic disruption of electric impulse' delays by the AV node. In this case the AV node itself is cauterized; it is partially cicatrized and slows down conductivity. Thus, it may be noted that there are no additional conductive pathways between the atria and ventricles and if they existed electrical impulses would have been conducted via them constantly, from birth to death, as there are no valves in muscle bundles and these pre-excitation syndromes are not permanent but temporary.

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(This article may be cited Habchabov RG. New electrophysiological mechanisms of electrical instability of heart, defibrillation and hypertension. *E-Journal of cardiology 2012; 1(2):17-27.)*