Global Global Advanced Research Journal of Safety, Health and Physical Science (GARJSHPS) Vol. 1(1) pp. 001-006, December, 2012. Available online http://garj.org/garjshps/index.htm

Review

New reason for and the mechanisms of cardiac electrical instability. New reason hypertension.

Rustam G. Habchabov

Dagestan state medical academy, Russia 1, st. Lenina, Makhachkala city, 367000
E-mail: rustam033@gmail.com

Accepted 30 October, 2012

The reason for development of life-threatening arrhythmias leading to sudden and total death provoked by cardiac diseases is still unclear, even though cardiologic research in this regard is being carried all over the word. A damaging of the connective insulation cover of the conductive heart path accompanied by ectopic nodes oxidation may result in life-threatening arrhythmias. Such reason for it as the cardiac electrical instability hasn't been examined by anyone before. Speed of delivery of electricity from the heart to the nervous system and spread it throughout the body remains undervalued. Speed of delivery of electricity from the heart to the nervous system and spread it throughout the body remains undervalued. The slowdown of the magnetic induction (acceleration) of electricity from the heart to the central nervous system and its spread, resulting in a lack of speed and reduce electricity bioelectric processes of the body. This leads to activation of sympathic nervous system, followed by a cascade of pressor mechanisms and the development of essential hypertension.

Keywords: electric instability of heart; paroxysmal tachycardia; trembling; fibrillation; defibrillation; hypertension; nervous system; syndrome WPW-CLC.

INTRODUCTION

Cardiovascular diseases (CVDs) are the leading causes of disability and mortality of working people around the world, which leads to the need to find modern and effective methods of diagnosis, treatment and prevention of electrical instability of the heart (CEI).

At a young age patients with rheumatic disease, myocarditis, mitral valvular disease demonstrate flutter and fibrillation, while geriatric and older patients demonstrate it more often if they are diagnosed with ischemic heart disease (IHD), myocardial infraction (MI), chronic cardiac failure, mitral stenosis, hypertensive disease, cardiomyopathy, myocarditis, etc. (Murashko and Strutynskij 2000).

The condition specifying lethal arrhythmias is structural cardiac pathology (SCP) turning into an instable substrate as affected by various functional factors. Such structural changes conditioning the development of life-threatening arrhythmias (LTA) may be as follows: express hypertrophy, dilatation, cardiac aneurysm, myocardial necrosis and sclerosis, inflammation accompanied by myocardial tissue edema, etc. According to the data of many researchers, these changes constitute the anatomical substrate with various LTA development mechanisms (Buxton et al., 2001).

Many authors that conducted CEI research often concentrate only on myocardial necrotizing and cicatricial lesions (NCL) in IHD patients. Yet such authors ignore other patients with other CVDs that are exposed to total and sudden cardiac death (SCD), too, for no apparent reason.

It should be mentioned that practically all arrhythmias are easy to be analyzed, except for the reason and the

mechanism for flutter and fibrillation development. Moreover, the definition of atrial fibrillation is misleading because of the contradictions contained in electrocardiography (ECG) textbooks.

According to the definition contained in ECG textbooks (Murashko and Strutynskij 2000; Eagles 2004). Atrial and ventricular flutter is regular rhythmical movement of a potent impulse following one and the same path with simultaneous re-entry mechanism development (the reason and the method of such macro-re-entry mechanism being unclear to the researchers at that moment). Atrial and ventricular fibrillation is irregular excitation and contraction of some groups of muscle fibers, each of them being a sort of ectopic impulse site (such definition in ECG textbooks does not seem to be too correct for students and young scientists). Having read the definition and returning back to the "automatism function" chapter one may read that the automatism function is pertinent to Sinoatrial Node cells (SAN) and the conductive system of heart: atrioventricular AV connection, the connective system of atriums and ventricles, the contractive **myocardium** b**eing deprived of the automatism function** (Murashko and Strutynskij 2000; Eagles 2004). All this testifies to the fact that in the event of atrial fibrillation "**some groups of muscle fibers**" cannot be generated, even if the myocardium or cardiomyocytes change their properties. The "electric myocardial instability" is non-existent because the myocardium can only conduct electric impulses. Some may object that even though the myocardium is deprived of electrical activity, yet it may have a nonhomogeneity area associated with NCL that may prevent it from conducting electricity. Virtually, the electrical impulses avoid the area easily, even in the event of extensive MI, even if there are no serious problems of the cardiac conduction path (CCP). Atrial fibrillation itself is conditioned by multiple generations of ectopic focuses (EF) of a lesser power located in distal CCP and with the same number of colliding micro-re-entry waves. In fact, the myocardium cannot generate it and the ectopic focuses are not chaotically scattered along the myocardium, as some might believe. All EFs are placed along the CCP, as a string of beads, and have the same connective insulation cover (CIC) starting from the SAN. If this cover is intact, electric impulses cannot leave the myocardium. It's only Purkinje fibers that lack such cover and it's due to them that the myocardium is excitated.

First of all one should mention that CCPs are placed in the same manner as electric wires in walls of our houses are. They also have an insulation cover. Yet the wires located in walls are immovable and they almost do not wear out while a working myocardium with SCP causes them to wear out soon resulting in a block and/or damaging of CIC only.

In some cases of express SCP over-distension breaking or tearing of a CIC of one of the main proximal CCP may occur. It activates the nearest potent EF with flutter development. This causes a potent electrical impulse to reach the myocardium along the path of least resistance through such damaging and trigger the macro re-entry mechanism. This is how large F flutter waves are formed (besides, the same mechanism is described for Alzheimer disease when atherosclerosis damages the nerve's medullary sheath and nerve impulses diffuse into the surrounding tissue. Is CCP's atherosclerotic damaging also possible?).

What is the cause for potent EF activation with flutter development in the event of CIC damaging? If CICs are intact, CCPs are not subject to oxidation. Purkinje, the famous researcher, described transitional T-cells located between the conductive B-cells (Purkinje cells) and the myocardium. He thought that their main function was to conduct electric impulses but their main function is more likely to consist in forming an antioxidative barrier for the conductive B-cells. Myocardial intercellular fluid shall not get into CCP, so the two conductors having a completely different structure have to have an "adaptor" among them that would prevent them from oxidation. If we eliminate the T-cells, the conductivity of electric impulses between B-cells and the myocardium shall remain the same for some time but Purkinje fibres would be exposed to oxidation. With the course of time oxidation would produce irrevocable consequences. Nature did not provide for transitional T-cells for emergency cases. It results in rapid oxidation of the nearest proximal EF with concomitant flutter development. Further the oxidation processes shall reach other distal EF and flutter will gradually turn into fibrillation.

One may wonder if the proposed mechanism of flutter development is correct. The fact is that the most potent ectopy source in supraventricular paroxysmal tachycardia and atrial flutter is located in the atrium (atrial fibrillation cannot be accounted for; it is conditioned by multiple ectopy). Yet ECG demonstrates a different picture. Why? It may be explained by the fact that in supraventricular paroxysmal tachycardia an electric impulse has to overcome many obstacles passing through a number of small conductive paths and ectopic nodes within the atriums and it reaches the myocardium as a not-so-potent impulse to spin a macro-re-entry wave. In atrial flutter a potent electric impulse reaches the myocardium prematurely, through a CIC damaging, encountering no obstacles and spins a macro re-entry wave.

Such a mechanism can be compared with the syndrome WPW, only in this case, most of the waves of electrical excitation, tore through broken CIC covers less passed through CCP, and F-wave flutter - pre-excitation is a delta wave infarction. Such a powerful output of electrical excitation in the atrial myocardium contributes to more rapid reduction in the power voltage pathways and EF, which shortens the refractory period, which leads to a greater incidence of heart rate for atrial flutter than with supraventricular paroxysmal tachycardia. Accordingly, the termination of atrial flutter with the

restoration of sinus rhythm, the ECG will be celebrated broadened P wave (more than 0.13 sec.). P wave in this case may be, simply broad and or two-phase and two-humped, then a delta wave is present, but it will be merged with P tooth and becomes almost invisible. For example, I wrote about what was happening in the atria, but exactly the same process occurs in the ventricles.

Thus, we can say with confidence that flutter - a special type of arrhythmia, which is characteristic for the SCP with damage CIC. Without SCP is reduced activity of the brain and nervous system effects at SAN, which leads to his weakness and activation of EF primarily with the development of ventricular arrhythmia and tachycardia, which may progress to ventricular fibrillation, and this process will occur without oxidation of EF.

What is the cause of paroxysmal tachycardia? Damage CIC CCP upper atria and ventricles (pathways Wenckebach, Bachman and Torelli, transitional AV node and the bundle of His in the ventricles and legs) may not be so serious, and produced only cracks and pores. In this case, EF will only occasionally be oxidized, and not as often as when flutter when there is more serious damage, tear or rupture of CIC. Respectively, and no pre-excitation infarction in paroxysmal tachycardia will not be, because the pores and cracks in the CIC will not miss powerful electrical pulse. Just the same, often appearing pores or cracks to the development of paroxysmal tachycardia gradually rend CIC CCP faculty and a transition to flutter, but the source of ectopic, however, remains the same. That is - it is one and the same type of arrhythmia, but with a different mechanism of action.

In many cases flutter does not precede atrial fibrillation. It happens because in fibrillation CIC of mid-distal CCPs are to be damaged most frequently (atrial or ventricular Purkinje fibres and braches), especially in NCL. Multiple T-cells damaging is also possible and results in the oxidation of more than one less potent EFs and micro-re-entry mechanism's development.

We can say that for all heart disease, paroxysmal tachycardia (paroxysmal tachycardia, atrial flutter, atrial fibrillation) are associated with oxidative irritation EF. But even if, cardiac pathology is not diagnosed in the survey, especially among young people, except as the very presence of paroxysmal tachycardia, it may be due to disorders of other organs or exogenous intoxication effects on the myocardium, resulting in microscopic damage to the oxidation of CIC EF. Thus one may say that the reason for CEI development is the same for flutter and fibrillation.

Any sudden myocardial movements or lesions may trigger CIC CCP damaging in SCP: all types of tachycardia, extrasystoles, blocks, abrupt blood pressure (BP) increase, myocardial contractility increase (especially if accompanied with pathologically decreased contractility), NCL, etc.

As for the focalization, the most thin and easy-to-be-damaged ventricular segments are the right bundle and the left anterior bundle branch. Then main stem of the left branch, the Bundle of His and, finally, the right anterior bundle branch may be named in the descending order. Yet conductivity may be damaged in any segment or in a few segments simultaneously (Eagles 2004).

Development of other types of cardiac arrhythmias, most often associated with heart rate and power of electrical impulses, or with a slowing of conduction and excitability. Heart rate and the voltage in the conduction system is not unimportant role in the development of arrhythmias

1. Sympathetic stimulation SAN promotes super-powerful and of proceeds of electrical impulses to the EF.
2. The weak influence of the sympathetic and parasympathetic innervation increased SAN promotes ultra-low power and rare delivery of electrical pulses to the EF.

It is as if your house is not getting enough speed or heavy duty electric power, so you do not out of order all electrical, you are off the central network and has its own power generator. Likewise intensified EF, sometimes trying to kill poor performance of the central oscillator with arrhythmias (this effect can be called a defense mechanism of ectopic sites of unstable power electric pulses of SAN and conducting AV node). These arrhythmias often develop in patients with impaired functioning of the nervous system and without SCP.

Other main factor in arrhythmia development is associated with the affected contractility and excitation, most frequently in SCP patients:

1. Organic or functional weakness of the ectopic node (I, II, III block – by AV block type) that result in weak electric impulses arriving to the distal node and its eventual activation.
2. Over-distension of the conductive path (especially in places where ectopic nodes are located) in express hypertrophies, dilatations, etc. lead to the extension of conductivity which also enhances a delay in electric impulses and activates EF.
3. Myocardial inflammation slows down the conductivity and results in ectopic activity development.

The example may be as follows: a city has been growing for 20-30 years because its suburbs (myocardial hypertrophy, dilatation) were growing, too. If the public utility network is lacking in the new districts, the inhabitants of such districts would complain of the lack of electricity. The same is true for the heart. The central nervous system tries to increase the cardiac rate and the potency of electric impulses to compensate for it. Sometimes it is enough. If not, ectopic arrhythmia tries to compensate for such insufficiency.

In some cases electric cardioversion may stop atrial fluttering and fibrillation at their early stages (paroxysmal, persistent). Powerful SAN stimulation suppressing other generation centers takes place. For some time the oxidative influence on EF becomes not important and minute CIC damaging is regenerated after a while. High

doses of antyarrhytmic medicines at early stages of flutter and fibrillation are also capable of suppressing ectopic activity with gradual regeneration of minute CIC damaging and sinus rhythm restoration. It should be noted that the restoration of sinus rhythm does not depend on the size of damage CIC, but depends on the duration of the oxidation of EF and reverse the process.

Preventive use of repairers in SCP patients may prevent CIC CCP damaging, such repairers being: potassium orotate, ATP and solcoseryl. It goes without saying that basic treatment of patients with atrial flutter and fibrillation shall remain at the previous level– cardioversion and antyarrhytmic drugs. Though additional alkalizing medicines and repairers, with the exception of aspirin and other acid-containing substances, for the period of flutter and fibrillation treatment may contribute into restoring cardiac electrical stability.

To continue CIC and the conductive system's issue, let me add a few speculations of my own. Electric defibrillation discharges are considered to influence the heart directly, which leads to a spasm attack, lengthening the refractory period and the possible, the reopening of the SAN, but it's an error! In the course of defibrillation human anatomy prevents electric discharges from entering the myocardium and exiting from it because of the external epicardial and endocavitary endocardial cardiac layer that has CIC. Electric discharges are most likely to affect the heart in an indirect manner, through multiple nervous receptors. Electric impulses strive to reach the CNS and then brain activation concerning all its aspects, including the sympathic nervous system (SNS) of the heart, starting from and SAN and b-adrenoreceptors stimulation with catecholamines (adrenaline, noradrenaline) discharge. Nobody thought that external electric currents can also take nervous paths in the course of defibrillation.

Now I'm going to present a theory which would seem incredible to many scientists but it has a right to exist. It's still unknown where and how electric impulses are formed in the nervous system. Electric impulses seem more likely not to be generated by the nervous system but to be sent to it from all the nervous receptors, starting from the SAN and ending up with ventricular myocardium where the electric impulse comes to a dead end. Further electricity is conducted by the conductive (afferent) nerve fiber to various CNS divisions. One should understand that ECG and electroencephalogram (EEG) recording have nothing in common because they record excitation in different structures of the organism. Such circular relationship of the heart and the nervous system is more reasonable that two separate electric systems which would have inevitably come in conflict and lead to a short circuit in one and the same organism.

Proof that the electricity passes from the heart to the nervous system, may be a question that I ask all of cardiologists and of which no one thought. Where the missing part of the electricity, which is blocked in AV block? ventricles it does not pass and re-atrial excitation occurs.

Here is one more argument in favor of a single heart-nervous electric system: We (cardiologists) say that infants and adolescents is high heart rate, why? Because, for the growth and development of the young organism needs more electric energy that produces the heart!

Speed of delivery of electricity from the heart to the nervous system and spread it throughout the body remains undervalued. With age, the magnetic field weakens some people are likely to play a role in this, and genetic predisposition. The slowdown of the magnetic induction (acceleration) of electricity from the heart to the central nervous system (CNS) and its spread, resulting in a lack of speed and reduce electricity bioelectric processes of the body. This leads to activation of SNS, followed by a cascade of pressor mechanisms and the development of essential hypertension. Perhaps the creation of an artificial magnetic field in the transition zone between the heart and the brain at the level of the neck in the form of jewelry (magnetic chain), or another type of magnetic therapy, will contribute to the restoration of this field and the induction of electricity in patients with essential hypertension, neuro dystonia and tachyarrhythmias.

Passive work of the nervous system may be performed without electricity, as it happens during heart transplantation when a patient remains on artificial blood circulation for a few hours. EEG records passive work of the brain which should give an impulse for the transplanted heart to start. In some cases it's not enough and defibrillation is implemented.

Further we are going to analyze literature sources and main views of the authors and researchers with reference to the CEI issue. First of all one should mention that many authors write "electrical instability of myocardium" while in reality it's more correct to write "cardiac electrical instability".

Life-threatening LTA are caused by a combination of reasons predisposing to electrical instability of myocardium: a substrate (structural cardiac disease) modulating the dysfunction of the autonomous nervous system and LTA triggering factors. The morphologic substrate creating post-MI nonhomogeneity of impulse conductivity is a myocardial area adjoining the necrotized tissue formed by intertangled spots of healthy myocardial fibers and the connective tissue. In this place the impulse connective path is prolonged because the spots of connective tissue serve as barriers to the excitation wave and the conductivity is slowed down due to the affected parallel orientation of muscle fibers. Thus myocardial areas with delayed ventricular depolarization may be anatomic and physiological substrate for the re-entry – main mechanism of LTA development (Buziashvili et al., 2002; El-Sherif et al., 1981). ***The author of this study was close to understand the real situation but the***

***source of LTA development is not the borderline myocardium but CIC CCP necrotic damaging in the area with concomitant development of flutter and/or atrial fibrillation*.**

Following the results of the work of J.D. Kramer et al., long impulse spin path is not necessary a small diameter of myocardial tissue with its electrophysiological properties altered by acute myocardial ischemia or with a heterogeneous structure resulting from fibrous and necrotizing changes is enough to trigger the re-entry mechanism (Kramer et al., 1985).***Fibers of the conductive system with EF are more hypoxia-resistant and myocardial ischemia does not trigger the re-entry mechanism in them, while fibrous-necrotizing changes are capable of damaging CIC CCP and triggering arrhythmia with the re-entry mechanism.***

As a rule, in the course of the conducted study sudden ventricular tachycardia (VT) or ventricular fibrillation (VF) with the maintained ejection fraction was observed in patients with implanted cardioverters defibrillators; if the contractility is decreased before VT or VF attacks, normally gradual increase of ventricular ectopic activity is to be noted (Saeed et al., 2000). It goes without saying that cardiac failure is an essential arrhythmogenic factor and a risk marker of sudden arrhythmic death in IHD patients (Bigger et al,. 1984). A Cardiac aneurysm, post-infarction cicatricial changes and clinical manifestations of cardiac failure make the adverse outcome more probable. Left ventricular contractility decrease increases SCD risk not only in IHD but also in patients with other cardiac diseases (Gardner et al., 1989; Kaasik et al., 2001). Ejection fraction less than 40%, nonsustained ventricular tachycardia (VT) diagnosed by Holter monitoring and electrophysiological study in patients that had an acute MI in their medical history are remaining to be the main predictive markers of high SCD risk (Buxton et al., 2001). Such combination of two SCD risk factors as frequent ventricular arrhythmia and left ventricular dysfunction with an ejection fraction decrease <40% is especially unfavorable. According to the data of GISSI-2 research, a risk of sudden arrhythmic death in this case increases 16-fold (Gilyarov and Sulimov 2010; Maggioni et al., 1993).

Besides that factors that were mentioned above, other sudden death risk factors are known, autonomic imbalance of the heart with prevailing sympathetic activity, in particular. The most important markers of the state is a decrease of the cardiac rate variability (CRV) and also such factors as a continued prolongation of the QT interval dispersion and late ventricular potentials (LVP) (Nademanee et al., 1987; Podrid and Kowey 1996).

I can only add a few summarizing conclusion from the PhD thesis I defended with reference to CRV, QT interval dispersion and LVP. Initial degradation of CRV parameters in patients during the post-infarction period is associated with their anxious and depressive state after acute MI. Depression is diagnosed in 82% of patients in the post-infarction period (Osadchyy 2009). Such patients are afraid of death and are anxious for their health, they do not perceive their environment with joy. On the contrary, they become reserved and their SNS becomes more active than the parasympathetic nervous system (PNS). As positive psycho-emotional state of the patients is activated, they start to overcome depression and PNS functioning becomes stable, too. This results in the sinus rhythm variations. One should note that a positive psycho-emotional state of a post-MI patient may become negative and even stress condition. This, in turn, leads to SNS hyperarousal. Combined with atherosclerotic changes in coronary arteries, SNS hyperarousal may result in a spasm of coronary arteries and appearance of new necrotic lesions in the myocardium. So nervous control over SAH accomplished by the simultaneous participation from the sympathetic and parasympathetic nervous systems, and the method of investigation of CRV has no practical information.

LVP in the post-infarction period is improved less significantly than the QT interval dispersion parameters. This may be conditioned by the fact that LVP is more associated with the CCP block (His bundle branches and/or main stems of Purkinje fibres) that are completely blocked by the nonhomogeneous necrotized area and further cicatricial changes of the myocardium which make the electric impulse return and reach the myocardium through other CCPs.

As different from LVP, the regeneration processes of the QT interval are to a lesser extent associated with CCP pathological changes. In the acute MI, the QT interval dispersion is predominantly slowed down by necrosis and a reinfarction myocardial area having a nonhomogenious area which is 20-40% larger than in the cicatricial period. Further it improves re-polarization processes. In single cases of LVP improvement in the post-infarction period rare cases of the organism's capability to produce stem cells may be of importance. In such cases partial growth of new CCPs (bundle branch of the bundle of His or Purkinje fibres) is observed beyond the cicatricial area and results in the regenerated electric impulses conductivity and LVP elimination.

In conclusion I would like to mention that presently the knowledge about the conductive system formation is far from being complete. For example, progress in the study of additional conductive paths for electric impulses was made only due to ECG studies and is not probative. Nobody has ever seen these muscle bundles in a human body! To have a broad picture of all myocardial peculiarities one should know that there is a connective tissue frame between the atriums and the ventricles that prevents the ventricles from excitating together with the atriums. This frame has an innate opening defect of

various sizes. It's this defect that is ablated and not the additional conductivity path (Kent bundle). Pressure increase in an atrium or in a ventricle, or both, makes this defect open and electric impulses pass from atriums to ventricular myocardium from time to time (WPW syndrome). CLC syndrome is also characterized by a lack of additional conductivity path (James bundle). It's an innate periodic violation of AV delay by electrical impulses node with their accelerated conductivity. In such a case the AV node itself is ablated, and then it is partially cicatrized and slows down the conductivity. So, one may observe that additional conductivity paths between atriums and ventricles are absent. If they did exist, electrical impulses would be permanently passing though them, from birth to death, as far as CCP has no valves, while these pre-excitation syndromes may be transitory.

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