NEW HYPOTHESIS
OF HEART FIBRILLATION AND DEFIBRILLATION

V.I. Ermoshkin
Research Centre “Resonance”, Moscow
e-mail: evlad48@list.ru

For a long time, more than 100 years, there is a problem of theoretical cardiology to prove true mechanisms and the true
causes of cardiac arrhythmias. The current problems of atrial and ventricular fibrillation, problem of sudden cardiac death
are of particular concern. There are also problems in the theory of defibrillation. In essence, we can say that at this time there
is no unified theory of ventricular fibrillation, there is no unified theory of defibrillation. There are many hypotheses, but
a unified theory has never been created. My proposed hypothesis, in my opinion, eliminates this drawback in medicine.

Key words: ventricular fibrillation, defibrillation, arrhythmia, sudden cardiac death.

Description of cardiac fibrillation mechanisms
and factors according to the current state of medi-
cine. According to clinical and experimental data it
is assumed that human ventricular fibrillation is
a chaotic, uncoordinated and ineffective contraction
of the individual groups of the ventricles muscle fibers
with a frequency of more than 250—300 times a mi-

ute. Still the ventricles are not able to contract syn-
chronously and the heart pumping function is actually
interrupted. Under this scenario sudden cardiac death
(SCD) is possible within 1—4 minutes if emergency
rescue measures are not taken. The clinical equivalent
of ventricular fibrillation (VF) is pulseless ventricular
tachycardia with apsychia and possible SCD.

At present time it is found that SCD cases are
mostly of arhythmogenic nature. In the vast majority
of cases (90%) the mechanisms underlying the SCD
development are VF and hemodynamically relevant
ventricular tachycardia.

It is worthy of note that the «picture» of the atrial
fibrillation (AF) is similar to that of VF, with the only
difference that AF cases are of a space-limited nature
with smaller impact on hemodynamics and less prob-
bility of death. Ventricular flutter and fibrillation
(VFLU and VF) can develop both on the background
of cardiac diseases and in association with various
noncardiac disorders.

Most commonly VFLU and VF result from long-
lasting hypertonia, heavy organic lesions of myocar-
dium associated with CAD, significant cardiac remo-
deling, myocardinfibrosis, poisoning.

Sometimes ventricular flutter and fibrillation occur
during cardiac surgery procedures such as coronogra-
phy, electrical cardioversion and defibrillation.

VF can occur in people with perfectly healthy
hearts, for instance, in case of drowning, electrocution
or sudden physical impact on breast in the heart
region.

And finally, a significant percent of the heart «mad-
ness» cases occur with no apparent cause. These are
cases of so called idiopathic VF. How can one explain
these cases? Here is the statement of this fact — the
fact of uncertainty that formed in cardiology. Bo-
eria L.A., Member of the Russian Academy of
Sciences and Russian Academy of Medical Sciences,

wrote on this topic in 2010 [1]: «It must be acknow-
egledged that with the majority of the ventricular
arrhythmia patients the disorder origin is still
beyond our understanding ... Premature extrasys-
toles (as «precursory symptoms» of VF) are recor-
ded during electrophysiological examination
(EPHE), but the reason of the premature activity is
unknown... We may conclude that idiopathic vent-
ricular fibrillation is a rare disease of unknown
origin».

In common sense it is clear that it is useless to
move forward and to be hopeful of success not having
explained the idiopathic VF cases and, unfortunately,
in recent decades it was proved in practice.
Nevertheless, in the situation of uncertainty the practical medicine arrives at the «logical» conclusion that there are many reasons and mechanisms of cardiac arrhythmia development (from extrasystole (EX) to VF). Treatment modalities are of great variety as well, every patient needs individual selection of surgical products and medicines, as well as individual methods.

The problem is a complicated one indeed. For the last 106 years (since the time of ECG invention) arrhythmia mechanism was never revealed. All attention of cardiologists and researches was focused on ECG, but ECG is not only a presentation of all-electric cardiac muscle activity. As a matter of fact ECG is an electric picture of the total activity: electrical, mechanical, chemical and neural activity!

Recently a few more «proven» and «experimentally-confirmed» theories of myocardial arrhythmia and fibrillations has been advanced in the world, but there was no theory unifying all types of arrhythmia. There is a belief, which is erroneous in my opinion, that every type of arrhythmia has its own mechanism of development. Meanwhile in the majority of countries mortality from cardiovascular diseases and from arrhythmia in particular is growing into an epidemic.

So, what results of the most advanced researches and what «theories» related to the cardiac arrhythmia are there currently in the medical science?

There is a great many scientific documents. Certainly, being the author of this article I cannot cover all hypotheses and scientific researches results for 100 years. I’ll try to rather briefly describe the main achievements in cardiology and arrhythmology for the last century and to synthesize a new unified arrhythmia theory based on this material: from trivial EX to VF and so on right up to asystole.

Let’s proceed from the premise that the heart excitation is the ability of the cardiac muscle cells and its conducting system to quick response to electrical, mechanical or chemical stimulation. These facts were known a great while ago, more than 100 years ago.

In my opinion it is necessary to introduce clarity to this statement. When setting the task to find the reasons for different types of the cardiac arrhythmia it should always give consideration both to the action of these three types of cardiac muscle stimulation separately and to their action in different combinations with each other.

Typically the cardiology states that arrhythmia and VF develop due to increase of cardiac conduction system (CCS) «automatism» and mechanism of macro and micro «reentry».

The so-called «reentry» is be regarded as a circular motion of electrical impulses. The length of the circle-wise path, in other words the «wave length» is the distance inside the myocardium the electrical impulse advances per one refractory period. In 1985 a group of authors headed by M. Allessie set forth the main principles fibrillation [2].

1. The excitation passes along the «main circle» or along random minor paths, i.e. using macro- and micro-reentry».

2. The velocity of fibrillatory excitation spread is from 0.2 to 0.5 m/s, i.e. far less than at excitation spread along the conducting system.

3. The excitation wave front can be of different length (from several mm to the length comparable to the size of atrium, i.e. several centimeters).

4. Each excitation wave exists only for a certain short time, the waves diverge, converge, disappear, in parallel generate new waves around inhomogeneities (necrotic zones), around refractoriness zones, around cavities.

5. For fibrillations development and maintaining the critical number of the excitation waves should be from 4 to 6 and more.

Besides, in most cases heart rhythm disorders are characterized by CCS «blockades», «delayed» or «concealed» conductions including those in «additional» paths. Most commonly ECG is used for arrhythmia diagnostics, including «Holter monitoring», electrophysiological examination (EPHE), blood pressure sensors and other devices.

Physician call the following factors as the most frequent direct causes of arrhythmia: hypertension, blood supply disturbances, diabetes mellitus, smoking,
alcohol, caffeine, stress, myocardial scarring after infarction (cardiosclerosis), scars after surgical interventions. Such scarring prevents electric impulse generation and/or interrupt the impulse spread in the cardiac muscle.

Arrhythmia is very often develops (from its simple forms of EX type and up to VF) due to ischemia [1; 3] or due to temporary electrolyte disorders. These disorders lead to multiple «blockades» of cardiac conduction system.

Such electrolytes as potassium, magnesium, sodium and calcium constitute the basis for electric impulse generation, maintaining and conduction in the heart. Too high or too low electrolytes concentration in the blood and in the heart cells affect the cardiac electrical activity and can cause arrhythmia.

Despite the fact that many types of arrhythmia are recorded with CAD the arrhythmia types most strongly associated with it are ventricular fibrillation and sudden cardiac death [1; 3—5]. Arteriolar constriction occurs till a part of the heart muscle dies (acute myocardial infarction) because of the lack of blood flow, for example, due to thrombosis. This may influence the electrical impulse propagation in the myocardium: electric excitation circles form at the boundary of the scar tissue.

Cardiac remodeling and cardiomyopathy become evident in primary stretching and thinning of the atria and ventricula walls, excessive thickening of the left ventricular walls or heart valves diseases.

Let's make the mini-summary of the academic researches. What was done incorrectly or what was not done at all?

Unfortunately, in the search for arrhythmia causes and mechanisms the MECHANICAL component of the myocardium excitation was completely «forgotten». Such «inattention» has stalled the develop-ment of a unified arrhythmia theory for many decades.

The results of the studies conducted by the research team led by the professor Kamkin A.G. stand apart. In my opinion, this very group has approached the solving of arrhythmia mystery at most. In fact, every-thing was ready for the synthesis of the arrhythmia unified theory more than 12 years ago. But a small step has not been done still. Judge for yourself.

Some fragments of Kamkin's theory are given below [6].

«The direct dependence of cardiomyocyte contraction from its excitation (action potential), i.e. excitation-contraction coupling, has been thoroughly examined already. However, there were clinical data giving the evidence of contraction-excitation feedback in myocardium — electrical processes modification under the influence of mechanical factors: myocardial stretching and (or) change in its contractile activity. Actually, such dependence was found Bainbridge F.A., an English cardiac physiologist, in 1915. He showed that the right atrial stretching causes cardiac rhythm acceleration in rats. O. Frank and E.H. Starling, Bainbridge's fellow countrymen, have achieved the increase of cardiac muscle contraction force by its stretching to a certain length. Based on this fact it could be concluded that the mechanical stimulation of cardiac tissue should cause a change in membrane potential in cells. Until recently, however, the impact of the feedback has neither been studied nor taken into account. In 1968 M.J. Lab, a physiologist from London, just put forward an assumption of such feedback, but he never proved it because of experimental methods imperfection.

To date a lot of clinical observations supporting this idea has been collected. For example, atrial arrhythmias — fibrillation, paroxysmal tachycardia — are well known. Such arrhythmias develop in the patients with acute atrial dilation or with gradual increase of their size. There were cases when mechanically induced arrhythmias occurred in the patients without these disorders by insertion of a catheter into the heart. Various cardiac rhythm disorders can also result from permanent mechanical overloads (for sportsmen as well) caused by arterial hypertension, congestive heart failure and chronic myocardial stretch. The relation-ship of mechanics and electricity is also confirmed by the mechanical stimulation of the heart that has been acknowledged in medicine over the years. Such
chest compressions is successfully used to recover the cardiac muscle contraction or to defibrillate the cardiac muscle.

Thus, clinical observations illustrate that heart chambers stretching, especially in case of heart pathology, leads to arrhythmias, but mechanical stimulation can also restore a normal cardiac rhythm, prevent fibrillation development. So, it is extremely important to understand which processes contribute to mechanoelectrical feedback”.

After special long-term investigations (1985—2002) prof. A.G. Kamkin and his team made a number of «discoveries» with far-reaching consequences.

«Thus, cardiomyocytes and fibroblasts (to an even greater degree) effectively convert a mechanical stimulation into electrical responses, and THE WORK OF THE FIRST CELLS IS MODULATED BY THE SECOND ONES» [6]. (Emphasis added by the author of the present analytical article. Here is the answer to the question about the nature of «reentry» and «ectopic focuses»!) «In a healthy heart cardiomyocytes stretching that leads to their membrane depolarization and fibroblasts stretching that causes a hyperpolarization are in equilibrium. But under pathological conditions the response to such mechanical stimulation is especially strong in both types of the cells, but in different ways. If the fibroblasts hyperpolarization value is higher than the value of cardiomyocytes depolarization the cardiac rhythm becomes slower and may even stop completely. And vice versa, if the latter one prevails it causes arrhythmia and fibrillation may develop.

The found interrelationship of these two types of cells and its effect on the cardiac function is of importance not only for fundamental science but also for practical medicine».

In my opinion the conclusions and the forecasts made by the authors of this theory are not complete enough. It seems to me that the effects achieved at short-term, fast, few milliseconds myocardium stretching-compression (for example, caused by pulse-wave impulses and / or mechanical impulses of any origin) were more important short-term solutions. No doubt, the issues of slow chronic myocardial stretch are also important, since in case of atrial and ventricular cardiomyopathy, in case of their fibrosis the myocardial becomes more sensitive to the stimulant of any origin: electrical, mechanical and chemical. Thus, this brings up the question of «protection» or additional «isolation» of the heart from hypertension, from mechanical impulses including those running through the vessels.

My hypothesis is that the initial types of arrhythmia may develop due to arterial pulse cycling through the veins to the atrium along the following route: aorta-arteries-arteriovenous anastomoses (AVA) — veins. And it is not critical whether the person is healthy or not, whether his heart and the whole cardiovascular system is healthy or unsound. Since it is MECHANICAL extraordinary pulses on the myocardium which can lead to EX and tachycardia development. Tachycardia always occurs (and disappears as well) instantly due to sudden resonance between the HR determined by the SA-node and one of the CVS natural frequencies [7, 8].

In such cases, the pathological pulse cyclically runs along the above mentioned vessels route and «generates» critical myocardial stimulation at venous entries or in the apex of the heart at equal intervals between beats with the HR two or three times higher than before the arrack — typically with the HR about 140 and 210 beats per minute, assuming that the average HR is 70 beats per minute (including the ventricular cells refractory period of 280—350 ms).

On the other hand, when arrhythmia of VF type has already occurred, regardless the underlying cause of the VF development, smaller mechanical impulses are usually continuously generated by the very tissue of the asynchronously beating heart, and these chaotic impulses are involved in the fibrillation episode maintenance.

SA-node maintains the ability to generate «correct» electric impulses and seemingly emits them from time to time. But the pulses generated by the SA-node cannot pass any significant distance along the conduction system as the entire conduction system is divided
into many sections that are either in refractory state or in the phase of readiness to excitation in accordance with the current random distribution of the myocardial tissue phases during VF. Exactly the same VF «attack» can be initiated in an «isolated heart» since the VF process is the function of the heart itself without a unified control through its own special electrical or mechanical effects. A heart cannot recover from the VF by itself since it is impossible to bring the whole myocardium its significant part of it to the same phase immediately. Most commonly such an attack caused by a haemodynamic compromise results in asystole and death due to complete exhaustion of residual nutrition of cardiomyocytes cell (it usually takes 1—4 minutes).

**Synthesis of a unified theory of arrhythmia.** At first glance it seems that the above mentioned scientific findings and hypotheses cannot be reduced to the only one. But it is not so. Judge for yourself. Let's make a certain analysis of arrhythmia causes and mechanisms adopted in modern cardiology.

The theory of «reentry» and «ectopic focus» with high automatism is not a formalized theory. It is a logically incomplete theory, but its extensive experimental data are certainly very useful for a more detailed description of the a unified theory of arrhythmia. Actually, this theory is a statement of electrical and chemical phenomena in the myocardium with different rhythm disorders observed during ECG and EPHE. The material is also useful due to its detailed description of factors, etiology and pathogenesis. In my opinion, the underlying cause of the arrhythmia and VF mechanism was not identified by cardiologists due to the fact that the MECHANICAL component of the arrhythmia was omitted.

The theory developed by the prof. Kamkin's group is the most feasible and promising, but it is also somewhat incomplete. In my opinion it is necessary to pay the closest attention to this theory describing arrhythmia causes and mechanism. All three components of the arrhythmia etiology, its electrical, mechanical and chemical nature of excitations, are reasonable. It is not clear why this theory (existing for 12 years) is still not demanded by academic science.

I suppose that my hypothesis about the arrhythmia caused by pathological pulse wave or by mechanical impulses of any other initiation supplements the most promising hypothesis. My hypothesis exists over 3 years. As it turned out later, my idea is an additional contribution to the unified theory that was lacked for the last few decades. In fact, my hypothesis makes a decisive supplement to the theory proposed by prof. Kamkin's group and extents it.

So, according to the new unified theory it must be assumed that «reentry» and «ectopic focuses» are in fact «mechanosensitive reentry» and «mechanosensitive ectopic focuses». This assumption has the following meaning: «ghost» arrhythmic electrical effects on the myocardium are secondary and invisible mechanical impulses and complex mechanical stress concentrations of myocardial tissue cardiomyocytes and fibroblasts are primary. According to [6] myocardium stretching relates to thousandths of mm, i.e. to the values of 4—12 microns, so it is impossible to fix it with eyes. As a result, when exceeding a certain threshold of compression intensity or stretching the cardiomyocytes and fibroblasts trigger a wave of cells depolarization through their mechanosensitive ion channels.

In other words, the function of cardiomyocytes can be modulated by fibroblasts and fibroblasts (that tend to grow in case of hypertension and CAD) are very good conductors of mechanical impulses including pulse waves. The more extensive the atherosclerosis the higher the conductivity, which is fully consistent with medical observations.

Some additional arguments for the hypotheses integration into one unified theory are given below.

1. In case of extrasystole and types of arrhythmias the conduction system «blockades» can be detected. They occur in case of interference of two or more excitation waves in the myocardium, for example, when a counterrunning earlier wave initiated by mechanical stimulation «encounters» the wave generated by the conductive system. Naturally, such blockade is usually a transient one, since no structural damages occur at the first stage of the arrhythmia development.
2. The status of cardiac ischemia with CAD or without it is specific state of the myocardium, when some parts of the cardiac muscle are satisfactorily supplied with blood, while others are not, when some coronary arteries have sufficient capacity, and some do not. This assumption has a right to exist, since if all the cells were equally supplied than in critical condition the size of the infarction zone would be equal to the size of the whole heart. But in practice it is not like that. The infarction zone and "myocardium parts with preinfarction syndrome" always have different sizes and locations depending on location and diameter of external and internal vessels, on extent of atherosclerosis and plaques. At a certain point irregular nutrition of myocardium and its CCS results in sharp increase of inhomogeneity of cardiomyocytes and conduction system electrophysiological characteristics, CCS blockade. It contributes to the development of myocardial electrical instability, which sometimes results in ventricular fibrillation, since electrical instability can automatically launch multiple independent cardiomyocytes excitation waves from different areas of the myocardium conduction system. Apparently, VF attack occurs because due to the numerous «blockades» the heart conduction system breaks into temporarily «independent» parts that automatically generate asynchronous bioelectric impulses of different frequency with the help of P-cells. Example: development of VF due to ischemia and hypoxia at drowning.

3. It should be born in mind that according to the «theory of elasticity» there are always stress concentrations around cavities and irregularities after mechanical waves propagation along elastic material. This is physics, it cannot be negated. Cardiac surgeon have the opportunity to see a «fanciful» picture of variable mechanical stresses during EPHE and RFA: micro re-entry of different sizes around venous entries, around necrosis areas, around scars and cicatrices.

4. It is proved and experimentally confirmed that the speed of arrhythmic excitation spread at VF is from 0.2 to 0.5 m/s. If the excitation propagated along real but «hidden» paths of the conduction system its speed would be from 1 to 4 m/s (established experimentally), i.e. 2—20 times faster. Hence, the mechanism of excitation propagation doesn't move along the conduction system but «form cell-to-cell», spreading in all directions.

So, we can state with certainty that in addition to all known myocardium disorders mechanical pulses also contribute to arrhythmia development and maintaining. It occurs due to mechanoelectrical feedback of specific cardiac ionic currents.

**Variants of cardiac rhythms disturbances**

A) If nothing but «correct» bioelectric impulses are delivered and conducted in the SA-node, and there is no cardiac ischemia, no spurious mechanical pulses, no electrolyte disorders, the myocardium works in sinus rhythm mode, as a rule there are no problems with hemodynamics by contrast with the variants described below.

B) If bioelectric impulses coming from the conducting system and high-amplitude mechanical impulses (pulse waves) coming from the CVS as a result of previous heart beats save some repeatability and regularity in time and space, there are the following types of arrhythmia: EX with constant «coupling» intervals, bigeminy, trigeminy, quadrigeminy and some others.

C) If bioelectric impulses are fully blocked by the work of mechanical impulses generated from one or several CVS «nodes» but the heart beats herewith save relative regularity, there are some arrhythmias like the ventricular N-mode tachycardia, «torsade de pointes» and others.

D) If normal bioelectric impulses to the extent of their passing from the SA-node to atriums are periodically blocked by the work of mechanical or electric impulses generated in the atriums due to their hyperesthesia in case of the hypertrophy, there are atrial fibrillations (AF) with narrow QRS but with non-uniform RR intervals at the ECG. (Herewith if spurious oscillations of atriums have regular or divisible HR component, then there are not the fibrillations but the flutters).

E) If normal bioelectric impulses are significantly blocked by the work of mechanical impulses generated from several CVS nodes or electrical impulses generated by the conductive system blocked in several places,
then «suddenly» the ventricular fibrillation attack occurs which leads to fully impaired blood circulation. As it follows from the experiments the quantity of individual excitation generation sources (electrical, mechanical and chemical ones) should be more than 4—6 for the occurrence of the VF attack. In rare case the VF attack can spontaneously pass to another arrhythmia regime or even to the sinus rhythm. But usually the VF attack ends by the asystole or the SCD (without resuscitation procedures).

F) Other variants of arrhythmia are also possible. So the VF attack occurs in the case of regular sinus rhythm failure when there is a disorder not lower than some degree in the rhythm management. Usually the disorder is made by some (4—6 or more) mechanical and electrical «individual» excitations having simultaneous impact on the myocardium. Such an event can take place both for a man (generally for all animals) with a healthy heart and for a man with some diseases, pathologies.

**New defibrillation hypothesis in terms of «mechanosensitive reentries»**. First of all let’s specify the defibrillation hypotheses some of which are discussed more than 100 years.

1. Hypothesis of cardiac muscle temporal paralysis.
2. Hypothesis of critical mass.
3. Hypothesis of upper sensitivity threshold.
4. Hypothesis of progressive depolarization.
5. Hypothesis of virtual electrodes.
6. Hypothesis of dispersion increase of cell refractory periods, etc. Inquiring reader can find information about them by himself. It’s true that there is still no plausible hypothesis of defibrillation. It was not created because there was also no rigorous and unified theory of the fibrillation.

If consider that A.G. Kamkin’s theory with my additions is realistic, than the defibrillation hypothesis intrinsically appears from it. Let’s name this new hypothesis as «The hypothesis of cells synchrony recovery of myocardium and its conductive system».

According to the unified fibrillation theory suggested by me one can assume that all the myocardium randomly divided into separated areas with «floating» borders between «the refractivity zones» and «the zones ready for depolarization» pulses in the asynchronous mode during the VF attack. Such operation mode is usually rather smooth but have some oscillations of wave amplitude. The low-amplitude or high-amplitude non-uniform fluctuation waves of the total potential of the myocardium cells polarization can be observed at the ECG at this time. The VF signature of each attack and each patient is its own, special, unreplicated one that is natural from the mathematic point of view because the sum of oscillations is random and corresponds to some «distribution law». The groups of myocardium cells prior to miss a beat and stop should almost fully exhaust their energy. Herewith it’s necessary to consider that not all the parts of myocardium were equally supplied with blood. So some part of cells evidently enters into the asystole state earlier and the other one enters later, thus these peculiarities are reflected at the ECG by the wave amplitude decrease up to full asystole. Based on the new general defibrillation theory, a heart with one or several zones of infarction, necrosis, with scares and plaques in coronary vessels can support the VF attack less time than the healthy heart, because the nonworking zones of infarction are the ballast, damper decelerating the mechanical oscillations for living, operating cells. Thus the VF process in the most cases is frequently the last, always different «signature» of the heart muscle prior to a fatal event. In my opinion it’s impossible to name such a state as any kind of arrhythmia. This is not correct; the VF is a natural dying. One can note also that the separated areas in equal oscillation phases can’t be too small or too big in their sizes. Their sizes though they are occasional can be defined by the velocity of mechanical impulses conduction over the myocardium and excitation speed of the mechanosensitive ion channels taking into account the random distribution zones of myocardium refractivity. It should also be taken into account that the mechanical impulse is able «unnoticeable or latently» to go though a relatively big zone of the refractivity without any marker and meeting with a zone ready of excitation to create a new excitation focus made of «nothing», giving rise to a consecutive wave of «re-
Cardiac conducting system and the very myocardium in the most percent of cases at the VF are potentially able to work, but there is no natural mechanism for spontaneous recovery of the proper myocardium operation. I think it’s statistically justifiable that 50% of separated areas of heart and its conducting system are in the refractory state at the VF at any time, the other 50% are in the state of readiness for excitation and «wait» for whether electric or external, saving, «volume», short-time impulse.

I consider that the feasible theory of defibrillation couldn’t occur prior to the proper theory of fibrillation. We had a possibility to observe that particular situation last more than 100 year from the time of the ECG invention. There were several hypotheses and «theories» of fibrillation and several hypotheses of defibrillation but they all had drawbacks and were not finally proved. Hardly anybody was agitated by such situation, though the mortality due to the CVD grew constantly or was at a rather high level.

I received the following terse reply from the institution of E.I. Chazov, the Academician of RAMS and RAN for my hypothesis of initial form of cardiac arrhythmia (extrasystole, trigeminy, quadrigeminy, some kinds of tachycardia) as a result of mechanical stress by the arterial pulse which goes cyclically through the AVA and veins to atriums.

«You are wrong. Different complicated electrophysiological processes realizing in the cardiac structures, described and proved by many experimental and clinical studies are in the basis of the arrhythmia. The hypothesis of author (i.e. mine, the author of this article) is negated by the development of paroxysmal cardiac rhythm in the isolated heart, surgically separated from the vascular system as well as the occurrence of paroxysmal tachycardia without pulse».

I’d like to explain my arguments briefly. It’s known that the ventricular fibrillations (VF) occur due to the presence of several, more than three sources of electric and mechanic myocardium excitations acting simultaneously with different rates. Such events can take place both in healthy and in sick heart. Electrical sources arise due to many blocks of conducting system, i.e. the conducting system is divided into many areas and each area automatically with different rates starts the electrical exciting impulses of cardiomyocytes. The blocks occur in their turn due to mechanical excitations of atriums and ventricles by the pulse mechanical wave, due to complicated dynamic picture of mechanical stresses in myocardium working in parallel with electrical impulses. Having started once, the disorder in cardiac muscle achieves the possible maximum level because the VF is supported by many random mechanical impulses produced by the very myocardium and random electrical impulses generated in different part of conducting system. The pulse wave coming prior to that condition over veins to atriums becomes irrelevant and disappears by itself due to inefficiency of the heart pumping function at the VF. The main thing is to launch the VF and then the alternating mechanical and electrical impulses are maintained by themselves till the residual supply in cells won’t be out. Thus the VF is the function of the very heart, its structure, with all defects, plaques, scars. And it doesn’t matter if it’s situated in its “working place” or is surgically isolated but located in the nutritional medium. The difference is only that the VF in the nutritional medium will exit longer because the cell “nourishing” isn’t stopped. In accordance with the VF generation mechanism suggested by me the fibrillations, flutterings and tachycardias can occur in the isolated heart but it’s unlikely that there will be bigeminyes, trigeminyes, quadrigeminyes because the latter kinds of arrhythmia under my hypothesis are generated by the impulse coming to the atriums in the strictly defined time after the cyclic run over the vessels.

The second critical inquiry of E.I. Chazov, the Academician was the question on paroxysmal ta-


cardia without pulse. In my opinion, this is situation when the continuous blood flow is interrupted due to its deficiency for a variety of reasons. Large veins collapse at the approach to the heart. All or some cardiac chambers also collapse with the last pumping.
This is not an «exotica». Such situation sometimes occurs and has its name in medicine — an «empty heart». How can we explain it? Let’s imagine that the heart is single-chambered as a ball. We make a hole in the ball and press the ball to the maximum. Then we release it. The ball will stay in cramped condition in the same way as the pressed «empty heart». There will generation of electrical impulses (according to the ECG) similar to the tachycardia, but the heart can’t be neither released, nor pressed because it’s squeezed to the maximum. There won’t be any pulse wave of course. When the heart tries to expand, the big negative pressure is generated inside the ventricles. It appears that the heart is restrained due to the macrophysical reasons and the micro ones. There is so called «electromechanical dissociation» according to the medicine terminology. (I don’t know if this term is appropriate). It’s obvious that in such cases the cardiopulmonary resuscitation should be urgently done, the volume of blood or its substitutes must be increased to fill the large veins with liquid. Is it possible that one should simply raise the lower part of patient body located horizontally? When veins and atriums are filled with blood again, the heart will recover its fairly proper operation.

I hope that I explained in a rather understandable way the physics of macro- and microprocesses which sometimes take place in the «isolated» and «empty» heart.

I’d like to emphasize that the mechanical constituent of the myocardium excitation was unfortunately not taken into account for a long time in the cardiology as a science. That’s why there was stagnation in the theoretical cardiology for many years.

Now let’s talk about the defibrillation with due consideration of the fibrillation model. If to use the fibrillation model suggested by me it’s evident that it’s impossible to make the myocardium synchronously working with its conducting system with a probability of 100% by one electric exposure.

From the other side after the first failed defibrillation by the electric impulse the repeated similar electric exposures with increasing impulse power (usually there is step-like power increase: 100, 200, 360 J) made after the necessary capacitor recharging during 3—10 sec and some pause of 30—60 sec required for massage activities will have not more than 50% chance of the favorable output under the modern methods. Actually if there is a delay of the emergency aid, the probability of successful cardiac muscle activation is decreasing with each new defibrillator discharge.

Step-like power increase of a defibrillator is indicated to eliminate theVF attack according to the up-to-date medicine recommendations.

Taking into account the suggested hypothesis to increase the possibility of the sinus rhythm restoration at the VF the following defibrillation process rework is required. There should be several external electric two-phase exposures. They are to be made one after another, for example, 2—5 consecutive exposures with rated intervals. Impulse power should be decreased (possible lower than 10—100 J), because the increased power in one isolated impulse can’t provide any advantage under the suggested defibrillation hypothesis. It’s disadvantageous and destroying the myocardium cells and its conducting system.

The probability of positive outcome is increasing with each new impulse if they are executed one after another according to the suggested method, i.e. emitting the pulse «burst». So it is not decreasing as with the common method. The fact is that 50% of cardiomyocytes recovering the phase synchronism after the first impulse will be excited synchronously in case of two defibrillations made one after another (let’s say in 400—600 ms). The new half of cells among the left 50% of them in the VF state will be recovered by the second impulse. As a result 75% of myocardium cells will recover their phase synchronism. The probability of patient life saving will be significantly increased.

87.5% of cardiomyocytes will recover the cell phase synchronism in case of three external electric exposures made one after one; if there four exposures the phase synchronism of up to 93.25% of cardiomyocytes will be restored; if there five exposures the phase synchronism of up to 96.65% of cardiomyocytes will be restored as well. Let’s compare the effectiveness of old and new methods: 50% and 97% means enormous difference! Such high percent of the cardio-
Myocyte synchronization (97%) will be probably enough to guarantee the patient saving from the VF attack. The impulses distribution of external electric exposure should be with the increasing interval between adjacent impulses. For example, the time series between five impulses can be the following: 333, 459, 585, 711 ms that corresponds to the rate equal to 180, 131, 102 and 85 beats/min. The given approximate number should be experimentally specified hereafter. One should start the exposures with minimum applicable interval between the adjacent impulses, because there are data that the dispersion of refractivity period of different myocardium cells is increased at the VF and the range of refractivity variation is in the limits of 280—330 ms.

The suggested recovery mode of all the myocardium contraction synchronism by the increase of interval between the external exposure impulses will be more effective, because such a mode won’t provide the big amount of spontaneous preexcitations of myocardium parts which have recovered their synchronism during the previous electric discharges. After the termination of the fifth impulse exposure, i.e. after 2.098 ms, the heart will smoothly transfer from the HR equal to 85 to the admissible HR controlled by the SA-node and equal to about 70—85 beats/min.

And now let’s come to the main presumption. It’s known that in case of the delay with the resuscitation procedures, i.e. after 4 minutes and more from the VF attack start, the chances to save a patient using the modern one-impulse defibrillation tend to zero. But if to take into account the suggested method using the «burst» of 5 (or more) defibrillation impulses, the chances for the life saving will increase. Let’s suppose that the defibrillation is applied at the end of 4 minutes and only 60% of cardiomyocytes from 100% are «able to react» in this time. This means that 40% of cells stopped due to insufficient nourishing, but they are still able to recover the operation, 30% of cells are in the refractivity state and 30% are ready for the depolarization. 30% of cells will be inphase after the first defibrillation impulse, 44% will be inphase after the second impulse, 55% — after the third impulse, 64% — after the fourth impulse, 71% — after the fifth impulse. Thus after the end of the fifth impulse termination, i.e. at the end of only 2.098 ms, the heart will most likely restore the sinus rhythm. 30% of cells according to the old method will start the inphase operation after one impulse of defibrillation and the anticipated success will be minimum one. As for the new five-impulse method, 71% of cells will operate in the inphase mode and this brings big hope for the success!

In the end it’s necessary to emphasize that the parameters of the burst signal are to be calibrated for the maximum permissible survival of a man got into trouble. This means that it’s necessary to execute the burst test cycles according to the power of one and all impulses, the ECG results «prior» and «after» the burst exposure on a patient with the VF attack. It appears that the burst variant of the VF attacks elimination can be recommended both for the defibrillators and personal cardiac pacemakers with the VF attacks tracking system.

As for the defibrillation of the AF attacks, the above mentioned reasoning are still actual with the only difference that the first impulse of the burst should be time-matched with the regular anticipated QRS and the required number and power of impulses should be evidently decreased.

Conclusions

New unified hypothesis of arrhythmia as well as the new hypothesis of fibrillation and defibrillation was suggested. It seems that more than 100-year theoretical stagnation in the cardiology comes to the end. It appears that the arguments to dispose the statement of L.A. Bokeria, the Academician, on the cause of the idiopathic ventricular fibrillation as the rare disease of unknown etiology were found. Also I managed to answer two main questions of E.I. Chasov, the Academician, addressed to me, the author of the arrhythmia hypothesis, to deny my hypothesis based on the role of mechanical excitements of extrasystoles by the impulse going to the atriums. These questions are the following.

A) Why does the arrhythmia develop in the isolated heart, i.e. surgically separated from the vascular
system if there is no arterial pulse under the problem statement conditions?

B) Why do the attacks of paroxysmal tachycardia without pulse occur in some cases?

Answers to these complicated questions can be found in the text of this hypothesis.

Eventually all main scientific fields described above were seamlessly integrated into a new unified hypothesis. Each part, viz. the clinical observations, «reentry» effect and «ectopic focuses» at EPHE, factors contributing to aetiology and pathogenesis of many kinds of arrhythmia up to ventricular and atrial fibrillation, role of mechanosensitive ion channels, role of pathologic pulse coming through the arteriovenous anastomoses to the atriums: all these researches and guesses provided the basis for a new theory. Discrepancies and disadvantages of main fields were almost eliminated. All this took place thanks to the new theory of «mechanoelectrical feedback» of professor A.G. Kamkin, thanks to the measurement of mechanical impulses of different sources, different intensity and structure which play a decisive role in the etiology and pathogenesis of cardiac arrhythmia. It developed that healthy people as well as sick persons can have the initial forms of arrhythmia and even SCD.

The discussions are necessary. Additional cycle of experimental works to confirm the received results is required as well. Thus there will be large amount of work and the new perspectives in control of cardiovascular diseases.

REFERENCES


