

THE HYPOTHESIS OF CARDIAC ARRHYTHMIAS

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ABSTRACT

Background. Cardiovascular diseases(CVDs) are the main causes of death in all countries. Majority of these deaths occur due to arrhythmias. The aim of this review to attempt to propose new hypothesis regarding the pathogenesis of extrasystoles and pathological tachycardia.

Methods. Internet search and discussion with experts: Frolov V.M., Shirokov E.A., Singh R.B. et al.

Results. The extrasystoles and tachycardia occur in some people due to the pulse propagation through abnormal contour of vessels: arteries - natural shunts - veins. Thus, sometimes arterial pulse jumps through shunts on veins of the internal organs. In this case, pathological pulse arrives to left or right atriums. The atriums, as opposed to veins, have mechanoelectrical properties and are capable of transforming mechanical impulse into electrical impulse. The electrical impulse especially at hypersensitive foci can generate atrial extrasystoles i.e. premature heartbeat. In a ratio of 1:2 or 1:3 pulse rate from the "pacemaker" and the frequency of the contour of vessels may suddenly produce "mechanical resonance" between the two systems, leading to pathological tachycardia. It is possible, that the risk of premature beats and tachycardia decreases, if there is suppression of mechanical impulse (abnormal heartbeat) on the approach to the atria. This serves to apply scars on the hollow and / or pulmonary veins, but not the atria or ventricles. It can be expected that in the near future, we will find this and other methods for suppressing abnormal heartbeat.

Conclusions. Atrium appears to have mechanoelectrical properties and are capable of transforming mechanical impulse into the electrical one. The electrical impulse, especially at hypersensitive ectopic center(s), can generate atrial extrasystole i.e. premature heart beat and supraventricular tachycardia.

Key Words. Arrhythmias, premature heartbeat, pathological tachycardia.

Introduction- According to the World Health Organization, presumably in 2030 at about 23.6 million people worldwide will be dead from cardiovascular diseases, mainly heart disease, arrhythmias and stroke, which are projected to remain the major causes of death.

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Why from CVD, and particularly from arrhythmias, dies so many people, often young, in the prime of their creative capabilities and without heart disease? Maybe there is not medicine looks for the cause of arrhythmias and abnormal tachycardia? The proposed hypothesis

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is an attempt to approach the solving of arrhythmias problem from the opposite side.

The essence of the following hypothesis is the following. It is assumed that the nature of U wave on the ECG, the nature of extrasystoles (ES) - is a "bioelectric response" of the sensitive heart tissue on a run (or into a series of circular runs) of the arterial pulse on specific vessels of the internal organs "heart-arterynatural shunts-vein-heart "and excessive mechanical momentary pressure in the muscle atrium or ventriculars. Excessive mechanical pressure that is brought by abnormal pulse, by direct piezoelectric effect [1], or, in other words, mechanoelectrical feedback heart [2] is converted into alternating electrical potential. Is it because of single monomorphic ES intervals "clutch" is almost equal to each other, just as are equal intervals between the ES for short and long "runs" of paroxysmal tachycardia? And they are equal because the pulse transit time by the same vessels in adjacent sweeps at ECG the same, and change interval time can not be rapidly changed. Is it because complexes generated by "ectopic" momentum expanded the and deformed, in contrast to sinus complexes ORS? Apparently, the ORS complexes expanded because of the area of critical operation of the mechanical momentum in the atria or the ventricles is not concentrated in one point, but extended, and the sequence of contractions of the atria and ventricles is disturbed and becomes not optimal.

"Narrow" QRS complex may be only after generation of pulses by the "point" SA and AV nodes of healthy person pacemaker. More often, the focus area of the mechanical impulse (abnormal heartbeat) at the output of the pulmonary veins is a certain area on the back wall of the left atrium, it is in this area is most often formed "ectopic hearth", generating an alternating electrical potential and trigger abrupt ES.

Enigma of «U wave» and «third tone» on the phonocardiogram (ECG) To understand the idea of the hypothesis, it is necessary to pay attention to the data obtained on phonocardiograms synchronized with the electrocardiogram. It turns out that the so-called "third tone" on phonocardiogram always follows the little-known U wave on the ECG [9], but on this fact medicine pays no attention. So far, it is believed that the U wave has little clinical significance, moreover, it is small and is detected in less than 50% of cases.

"Third tone" was "lucky" more: for people over 40 years the tone is usually very low energy, medical researchers internally added to the pathological, the nature of this tone is still being debated, put forward several hypotheses, but no final conclusion has not been done . We note only that the place of origin U wave and "third tone" according to the medical research match - a top in the left atrium of the heart. I think it's a coincidence is not accidental: the cause of these two phenomena, apparently, is a pathological pulse, passed in a closed circuit of blood vessels and caused fluctuations in electrical potential on the atria and, with some natural delay then forced to mechanical vibrations of the atria. In addition, accidental U wave and the "third tone" does not have a permanent place of deployment on ECG, observations show that they, for some unknown reason, periodically displaced relative to the P and T waves according to some rule.

Mechanoelectrical effect in the heart

Thus, we can assume that the ES – is a natural mechanoelectrical response of atrials and ventriculars to an external mechanical impact, particularly if the heart muscles due to hypertension or due to increased loads sports hypertrophied and if hypertrophied, the thus more sensitive.

For example, the loss of momentum in the patient, competent doctor hit his hand on the patient's sternum is trying to run an artificial ES and then, with luck, the right heart rate. Another example: in the experience irritation of the myocardium by "bristle", followed by the generation of ES - see blog cardiologist Mary

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Skryabina. In addition, a recent case: when struck on the chest hockey puck from Omsk A. Cherepanov (or after body checking in hockey) accompanying ES apparently caused the crash of the pacemaker, have atrial fibrillation, and the young man died.

At autopsy it was found that hypertrophied heart hockey player weighed 495 at the normal of 290 grams.

On the other hand, in the absence of cardiac hypertrophy, i.e. normal, the sensitivity of the tissue beneath and run a ES is more complex.

This is because of the the absence of hypertension and atherosclerosis, shunts, connecting in case of excessive rise in blood pressure, arterial and venous pools are closed, pulsing action is too high at the mouths of the veins should not be, arterial pulse wave for the benefit of circulation in the capillaries and should not reach fades in the interstitial fluid bodies and the mouths of the veins.

"Vulnerable points" on the ECG

This hypothesis has been suggested that abnormal paroxysmal tachycardia occurs, when the time interval between two sinus beats, QQ on the interval on the electrocardiogram, fits exactly two, even three or more periods of occurrence of U wave. At this time, heart rate (HR) is usually equal to 70, or a little more beats per minute, so to identify the U wave, as well as the P and T waves on the ECG is difficult, and sometimes impossible due to their interference.

The assumption made above, we can say, is confirmed by experimental data, namely, in the methodical development for practitioners. For example, in paper [3] it is said, that the systole of the patient (the interval QT), must be strictly less than half the interval QQ. Medical evidence shows that the lengthening of the QT interval more then normal may be tachycardia and sudden cardiac death. Deadly arrhythmias also due to shortening of the interval QT.

Interestingly, the plausible explanation, what is the mechanism of occurrence of fatal

tachycardia at elongation and shortening of the QT interval medicine does not, only they say that allegedly genetics is guilty.

According to the long-term observations, "safe" QT interval should be between 0.30 to 0.42 seconds, depending on heart rate during tachycardia previous session.

But this fact does not neglect medicine. This hypothesis suggests that, in some cases, abnormal heart rate, and with it the U wave, located on the right slope of the wave T, exactly falls into the "point of failure", equal to half of the interval QQ, and causes pathological tachycardia.

If the wave U is to the right or left of vulnerable point, then there are only single ES when exactly in the middle, the ES and possibly subsequent attack of tachycardia. So what happens when a mysterious coincidence, when the tooth U is in the middle of the interval QQ?

After all, the modern world cardiology during past few decades of years has been tirelessly deciphering the origin of ES and tachycardia, but the results so far, there are only statistics and observations, but this statistic is terrible.

Perhaps a surprise to most physicians, the answer is: at this time THERE IS NOT DRAMATICALLY MORE THAN Α MECHANICAL RESONANCE [4,5] frequencies of the "pacemaker" and one of the natural frequencies of a particular circuit vessels "heart-artery-shunts-vien-heart", heart rate changes abruptly 2 or 3 times, i.e. typical (average) heart rate of 70 beats sharp jumps in the range of 140 (\pm 10) or 210 (\pm 10) beats per minute. It can be assumed that in the range of 140 beats in pathological changes in heart rate QT prolongation over 0.42 seconds, and in the range of 210 beats per minute when shortening of the QT interval less than 0.30 seconds. The balance of this: if the most likely heart rate of 70 beats per minute, ie, at the interval OO equal to 0.86 sec, fits exactly two of the QT interval by 0.43 seconds and exactly three of the QT interval by 0.287 seconds!

Thus practical medicine brilliantly confirms the hypothesis proposed!

Apparently, SAFE LENGTH OF THE QT INTERVAL SHOULD BE BETWEEN 33.3% AND 50% OF THE LENGTH OF THE INTERVAL QQ, CLOSER TO ITS MOST OPTIMAL VALUE IN 41.7%, THIS OPTION WILL BE CORRECT FOR ELDERLY, AND FOR YOUNG PEOPLE, AND FOR THE BABIES.

Fortunately, the attack of pathological tachycardia may not occur if the heart rate at the mouths of the veins, and with it the U wave and the "third" heart sound is too small, ie, below a certain threshold, which is often happened in young people, and in the people leading a healthy lifestyle with moderate and regular physical activity. Besides for young people threshold for create ES above, because cardio vascular system (CVS) is a younger, a range of in vascular tone is changes more, atherosclerosis if only slightly, the CVS less sensitive to mechanical shocks the heart.

As a rule, after the attack of tachycardia, cycled part of the circulatory system, ie a specific vascular circuit can go to the AUTO GENERATION OF OSCILLATIONS, when part of the mechanical energy pulsing waves from the output of the system gets its input, ie, on atrial, and thus supports fluctuations CVS few seconds, hours or days, blocking signals pacemaker.

This man on the eve of the attack pathological tachycardia may be at rest and did not anticipate what awaits him in a minute.

The output of the RESONANCE BETWEEN THE TWO SYSTEMS is also a quick and unexpected for the patient, this seems to be due to a critical reduction of the amplitude of the mechanical momentum in the mouth of the veins, or because of a change for some reason, the integrated speed run along the contour of the pathological heart vessels, etc. is due to changes in vascular tone, may also be due to the "fatigue", and relax blood vessels. We note in particular that the entrance to the resonance and after leaving the resonance, it is often possible to observe the ECG solitary or paired ES that leave "traces" of his "guilt" in the pathological tachycardia - this is the time of critical change the resonance condition. Events with the occurrence of ES and "running" pathological tachycardia occasionally occur in healthy people, but more often in the development of hypertension, degenerative disc disease and atherosclerosis, loss of elasticity of the walls of blood vessels and, as a consequence, increase the amplitude and pulse wave velocity via the vessels.

One can only assume that the ES and runs of tachycardia does not have consequences for the health of the myocardium - they seem to speed up the process of "aging" of the heart. With intervening years DUE TO INCREASED VASCULAR STIFFNESS amplitude and pulse rate is increased, which leads to displacement of the U wave to the left along the time axis and overlay it on the T wave, or even to the segment ST. In a healthy person without hypertension and atherosclerosis tooth U on ECG either not fixed, because, natural shunts that connect arteries and veins are in their infancy and pulse can not jump on the veins and cause ES, or fixed with difficulty, because vessels young and flexible, mechanical wave propagation on a "soft" vessels quickly fades, U wave amplitude is small, ie subthreshold.

We also recognize, that the proposed hypothesis has some deficiencies, it still can not explain, for example, the reason for such allodromy bigemia, trigeminy; can not explain ventricular tachycardia of the "pirouette" cause of elevation segment ST.

It is likely that such complex arrhythmias due to the fact that vascular resonating circuits of varying thickness, may be several in one patient. Perhaps, in some circumstances there is a continual management of the heart with the help of "pacemaker" and using autogeneration by abnormal heart beats of the pulse. We can only say that in this area new researches are needed.

Prediction of Lang G.F., academic of the USSR Academy of Medical Sciences

In my view, the possibility of central nervous system in the regulation of heart rate upon the occurrence of unpredictable resonance pacemaker and one of the natural frequencies of vessels pathological tachycardia in in atherosclerosis is too small. At this time, the CNS is "stupored", because it has increased rigidity of blood vessels, particularly of elderly patients, in their tone adjustment range and hence is limited to, the range of movement of the speed control pulse to and offset receptacles U wave.

However, it will be understandable, if you take my HYPOTHESIS ABOUT THE RESONANCE OF THE HEART RATE OF THE PACEMAKER AND THE NATURAL FREQUENCY OF THE CIRCUIT OF BLOOD VESSELS.

On the other hand, the heart rate generated by pacemaker, depends on many factors and is always is regulated, changed, "supervised by CNS", including stress loads, or hormonal activity, for example, depending on the phase of respiration : one on breath, on exhale - the other, etc.

Another observation in favor of the hypothesis: the rate of pathological tachycardia deficit does not happen, but it happens when arrhythmia or tachycardia, controlled by the central nervous system.

Many years ago Academic of Medical Sciences of the USSR, Lang G.F. (1875-1948) drew attention to the strange persistence of pathological tachycardia with heart rate, in contrast to heart rate setting by pacemaker. It was Lang G.F., that spoke in support of the extra cardiac effects on heart rate in pathological tachycardia (PT), i.e. he pointed out that the problem is somewhere out of the heart. But what did he said, few people listened.

The search for the causes of arrhythmias last 30-40 years, and they were mainly in the

heart of genes, intercellular and intracellular levels, but without significant breakthroughs (successes) - the statistics of deaths from arrhythmias in the last decades confirms this fact.

Radiofrequency ablation of the heart, some doubts

If this hypothesis about the nature of ES and PT, which was discussed from 2011, is confirmed, it is clear that in the future the ablation of the atria and ventricles will be perceived as a misunderstanding.

There will be more simple and more effective methods of suppression of pathological mechanical pulses. The current task - to confirm or refute the hypothesis. It is in this I need help! Help is needed in order to plan a new electrical and mechanical and physiological studies in one of the international medical universities, and confirm or refute the hypothesis. "On balance" there are millions of lives, millions of human hopes. This would be very difficult, because all surgical cardiology over the past 20-30 years, "focused" on operations such as radiofrequency ablation (RFA) in the atria and ventricles to the medical direction is extremely spent a lot of money. It seems that, cutting, cauterizing or freezing "ectopic centres" in the atrium at the mouths of the veins, surgeons, apparently not realizing it until the end, solved two problems, both problems are not solved until the end:

a) formed scars on the atria to the reflection and scattering of the mechanical wave (pulse),

b) reduced the area of sensitive material of artrium that is capable of piezoelectric effect, ie, capacity to convert mechanical or tissue tension in the electric potential.

Why all the attention of surgeons are focused on the mouths of veins? Why ectopic centers are most often located at mouths of veins? I think, all because of the fact that pulse runs cyclically along the contour of the vessels and can get to the atrium only through the mouth of the cavity or pulmonary vein. The main minus of the present accepted in many 6

operations such as RFA is the foolowing: such surgery is very painful, has lack of efficiency, people are afraid to "burn" sometimes completely healthy heart, besides atrium and ventricle injured by surgeon as a result of RFA irretrievably lost many of its properties, inherent from birth. As a result, the resourse of the heart is finished faster, bringing ischemia, heart attacks and death. (Just as a heart the resource engine will be terminated, if it has earlier ignition. Everyone should understand that extasistoles for CVS - are the same preignition).

I reported about this hypothesis at an international medical conference in the People's Friendship University (Moscow) in November 2012. Several articles (hypotheses) for cardiology [6,7] were published, I have written a book [8], but so far there is no any official positive response.

It is believed that the present theory of «re-entry» is not true. It is true only that the wave of electrical excitation, to be called "rotor", runs cyclically via the back wall of the left atrium, but this wave is secondary, the primary factor is the mechanical pulse wave that repeatedly jumps from veins to the atrium. The prevalence of CVD has already exceeded all reasonable limits! People, are you sad?

I am ready to cooperate, we should act altogether, and not blindly copy "advanced technology" electro physiological studies and radiofrequency ablation (RFA).

If the hypothesis is correct, the elimination of "ectopic hearth" can be made easier and in quite another way, and to put the scars on our hearts will not be necessary. May be it will be a beginning of a new era...

Proposal for cardiac surgeons

Based on the above hypothesis, I suggest that patients with ES, jogs tachycardia and some other arrhythmias scarring, scars on the hollow and / or pulmonary veins shoul be away as far as possible and as appropriate, from the atria. According to my hypothesis, abnormal heartbeat (mechanical wave), for example, can get into the left atrium via the route: heart aorta - pulmonary artery of long systemic circulation - shunts (anastomoses in the lungs, the transition to the small circle) - pulmonary veins - left atrium. There are other ways that should be found and neutralized. It is assumed that after the application of scars on the veins (or even on the some of the arteries), heart pulse will be reflected from these artificial discontinuities on the vessels, partly will go back and dissipate, so the likelihood of getting heart for atrial reduced to decrease the risk of ES and arrhythmia. After spending proactively such simple operations on vessels of a certain part of young people or children, apparently, you can not be afraid for their lives, even if patients have syndromes of elongated or shortened intervals QT. The length of the QT interval should be between 33% up to 50% of the length of the interval QQ, the optimum length should be 41.7%. It is obvious that to solve these issues specialized studies and experiments are necessary.

Conclusions

1) This hypothesis is completely new and yet more plausible than its predecessors. The proposed hypothesis can explain many of the observed phenomena and the facts, associated with the occurrence of extrasystoles and pathological tachycardia due to the passage of a pathological pulse on atrials.

2) This hypothesis can easily explain safe range of the changes for adults in QT interval from 0.30 to 0.42 sec. When extending this range, tachycardia in the range of 140 punches is possible in excessive shortening - in the range of 210 beats per minute.

3) In favor of this hypothesis is the fact that the relationship between «U wave» ECG and "third tone" on the phonocardiogram emerging at the top of the left atrium. Observations show that the third tone should always take some time for the U wave. Modern medicine classifies both phenomena supposedly as pathological, that finally becomes apparent.

4) Pathological tachycardia has a hard consistency of the repetition period, ie QQ contiguous intervals on the ECG are changed very little. In addition, lack (deficit) of pulse in such a tachycardia does not happen. This suggests that the generation of PT heart beats is not carried out by the central nervous system and the "pacemaker". This phenomenon occurs when the CVS parameter values reaches to some specific values, and is called the mechanical resonance frequencies of the "pacemaker" and the frequency of one of the circuits vessels of internal organs.

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List of abbreviations

CVD - cardiovascular disease, ECG - electrocardiogram, ES - extrasystole, SA - the sinoatrial node, AB - the atrioventricular node, HR - heart rate, CVS - cardio vascular system, CNS - central nervous system, EPS - Electrical physiological studies, RFA - radiofrequency ablation,

- Peoples' Friendship University in

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