

Mechanisms of high heart rate variability: a fresh look

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Abstract

Consideration is being given herein to some mechanisms of high heart rate variability (high HRV), which cannot be attributed to sports exercise loading. The mechanism responsible for high HRV is explained as that resulted from the continuous performance (opening and closure) of arteriovenous anastomoses in different organs and systems in a human organism. An assessment of this phenomenon is given herein from the point of view of a practicing physician who treats regularly patients with already established clinical diagnoses and those without an established nosological profile according to International Statistical Classification of Diseases and Related Health Problems 10th Revision.

Keywords

Heart rate variability, Arteriovenous anastomoses, Circulatory shunting, Organ insufficiency, Organopathy

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Introduction

An early detection of abnormalities in the performance of blood vessels to prevent further serious irreversible injury can be treated as a really life-saving matter. And this is just the early diagnostics which is capable of solving all the problems.

It is well known that cardiovascular diseases cause most deaths as compared with other diseases [1]. Therefore, an identification of conditions along with the relevant anatomical & physiological features in a human organism, which may contribute to

or provoke an initiation of cardiovascular diseases, is actually the most critical and significant problem to be considered by clinical experts in practice. In this connection, it should be mentioned that one of the most promising tendencies of analytical tools, used for detection of possible causes of cardiovascular diseases, is an assessment of cases of high heart rate variability in a human organism [2-9]. When assessing HRV in healthy individuals, usually the meaningful interpretable deviations from the normal HRV should not exceed 20

to 30%. Moreover, according to classical cardiology, even the 10 % deviation from the normal HRV must be treated as not acceptable in general. Following the conventional way, possible HRV deviations beyond the said limits shall not subject to any assessment and have to be interpreted with certain provisions and stipulations. To treat extreme HRV abnormalities, the special term “entropy chaos” has been introduced. But, unfortunately, this term essentially is capable only to capture the observed status, without furnishing any reasonable explanations thereof. The question is: when we are detecting high HRV levels, what processes in an organism are evidenced by them? Do they indicate a pathology case or do they exhibit an adaptation reaction performed by an organism? And if it is just the adaptation reaction or response, what significance should be assigned thereto? One of the ideas outlined herein is to present an original study, covering the above mentioned problem cases.

Aims

The author hereof considers in his study possible mechanisms of high HRV which cannot be attributed to regular intense physical exercise loading.

Materials and methods

If we wish to gain a more penetrating insight into the above mentioned process, we should dwell on physiology of the cardiovascular system performance. Below given are some generally accepted classical definitions related to physiology of the cardiovascular system which are not subject to any critical review.

Cardiac output (CO) is the amount of blood the heart pumps through the circulatory system into the aorta

in a minute, and it includes the total blood volume flowing via the vessel bed per minute. The cardiac output is the most important indicator of hemodynamics in an organism.

Venous return (VR) is the flow of blood back to the right atrium of the heart per minute. Venous return must equal cardiac output (CO).

If for some reason venous return increases or changes, showing steadily fluctuations in the amount of blood entering the right atrium, the relevant mechanisms of the cardiac regulation should control the process in order to provide a balance between cardiac output and venous return. It is conventional to assume that the heart always attempts to pursue this goal in its performance. By this means, cardiac output is controlled in accord to actual venous return. When it is stated that cardiac output is regulated according to venous return, it means, that the heart does not play a leading part in the cardiac output control.

But what determines the nature of venous return that is decisive for cardiac output control? First, it should be said there are a lot of prime factors which influence peripheral circulation and which provide blood supply from veins to the heart, but not only the prime factors play a crucial role in the circulation control in an organism. Taking into account some specific features of the design of the cardiovascular system is of great importance. The design of the cardiovascular system provides for a possibility that a certain amount of blood, leaving the arterial system, is directly delivered to the venous system, so that the capillary bed is bypassed. This is precisely the mechanism that is central to the new theory of arrhythmia (NTA) originated by Vladimir I. Ermoshkin [10-12]. The

bypasses as alternative pathways used for blood delivery are called arterio-venous anastomoses (AV anastomoses, or AVA).

It should be noted that every AV anastomosis has its own regulating mechanisms which operate in such a way that blood is either discharged or undischarged to the venous system. If we take into account that the circulatory system is in possession of more than one anastomosis, considering the fact that each of them under operation meets its individual pre-specified requirements, it means that not constant, but variable amounts of blood are received by the right atrium in the course of time. In doing so, the heart seeks how to properly balance cardiac output (CO) and venous return (VR).

What mechanisms are involved in order to provide the required CO-VR balance?

Our heart contains its own “built-in” mechanism capable of automatically pumping the required amount of blood to the right atrium from the veins. It is the well-known Frank-Starling mechanism (also referred to as the Frank-Starling law) [13-16]. According to the Frank-Starling law, an increase in the blood amount supplied to the heart stretches the ventricles, and, in its turn, it increases the force of contractions, so that a greater volume of blood, as compared with the previous condition, leaves the heart for greater circulation. By this means, the full amount of blood supplied to the heart is automatically delivered to the aorta, without any delay, and circulates again and again throughout the blood vessel system.

Another important factor in this case is an increase in the heart rate as a response to the actual myocardium wall tension. So, the tension of the

wall of the right atrium in the sinus node area produces an immediate effect on the capability of pacemaking cells to generate excitation and leads to an elevation of the heart rate. Besides, stretching of the right atrium causes an atrial reflex response which is referred to as the Bainbridge reflex [17-21]. Pulses from the right ventricle are delivered to the blood vessel control center in the medulla oblongata, and then the pulses travel via the sympathetic and the vagus nerves to the heart to initiate an increase in the heart rate [22-24]. These mechanisms are activated, when venous return increases from one blood amount, entering the right ventricle, to another [25-28].

When each individual blood amount, entering the right ventricle, is differing from another due to involvement of the AV anastomoses, which open and undertake the bypass function, at the initial stages the heart is capable of correcting the process by adjusting the heart rate variability.

In such a manner, under the normal conditions, when no stress factors are available and when the AV anastomoses are not in operation, cardiac output is fully controlled by the peripheral mechanisms, which determine the actual amount of venous return. A cardiointervalogram will demonstrate all this as an adequate variability which reflects the regulatory mechanisms of the known members in the heart rhythm control system.

Results

In our practice, we have detected a number of patients who demonstrated high HRV. Our examination and observation cohort covered 25 patient cases with identified high heart

rate variability. A typical example of a high HRV, which is not attributed to sports training activities, is given herein below. Symptoms reported by the high HRV patients in all the cases were extremely varying and nonspecific, so that it was impossible to fit the observed symptoms in the nosological classification. As a rule, analyzing such cases, a doctor in primary health care makes his diagnosis as follows: vegetative vascular dystonia. But our cardiometric examination of patient Ya. clearly demonstrated that actually the reported symptoms and conditions should be attributed to the performance of the opened AV anastomoses (see Figure 1).

The high HRV patterns in the same female patient are exhibited in Figure 2 and Table 1 herein below.

The non-typicality index (NTI) (see Table 1) bears witness to failure in the performance of the adaptation mechanisms in the organism of the female patient in question. At the same time, her NTI reflects an absolutely non-typical age- and sex-related pattern, departing from that ty-



Figure 1. Cardiometric markers of opening of AV anastomoses.

Legend: ABA – opening of AV anastomoses.

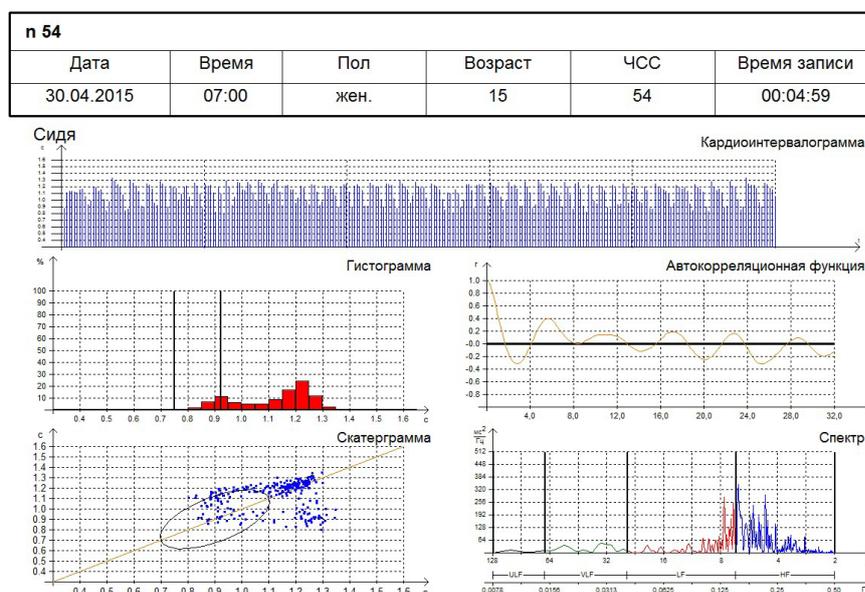


Figure 2. High HRV patterns

Table 1. General assessment of regulatory systems (position: sitting)

Characteristics of heart rhythm regulatory system	Particular diagnostic findings	Assessment, score	Deviation from the mode
A. Total effect of regulation	Pronounced bradycardia	- 2	- 2
B. Functions of automatism action	Pronounced arrhythmia	- 2	- 2
C. Vegetative homeostasis	Pronounced dominance of parasympathetic nervous system	- 2	- 2
D. Vasomotor (vascular) center	Normal activity of medullary cardiac and vasomotor center	0	- 1
E. Sympathetic medullary cardiac & vascular center	Pronounced weakness in activity of medullary cardiac & vascular center performance	- 2	- 2
Indicator of regulatory system activity (IRSA)			

pical in the cohort population under examination. A large body of completed studies based on monitoring and examinations of patients of this sort is sufficient to allow definite conclusions on the real cause of the high HRV phenomenon, which is not related to heavy sports training loads. It should be added that such examinations are supported by an assessment of failures in the adaptation mechanism performance, with use of special hardware & software system. Actually, a high HRV exceeding 20 – 30% is the marker of the adaptation-adjusting reaction to compensate any fluctuations in the blood amounts, entering the right atrium, by changing the heart rate in order to restore the proper balance between venous return and cardiac output. A pathognomonic sign of the particular pattern of the cardiointervalogram (CIG) is the absence of a clear-cut wave character on the CIG chart that corresponds to respiratory movements.

Conclusion

From what has been said it can be assumed that high heart rate variability may be judged to be the marker of opening of arteriovenous anastomoses within the blood vessel system in an organism. This suggestion is also supported by the fact that there is the cardiometric syndrome by Yermoshkin - Lukyanchenko which apparently shows the performance of the AV anastomoses according to the specific shape of a Rheogram (Figure 1 herein) [10-12]. This concept is essential for practice since high HRV indicating the AV anastomoses in operation should be interpreted as a warning sign for possible development of various types of extrasystoles, arrhythmias and sudden cardiac

death syndrome. For patients of this sort required should be further careful clinical examinations like pulse wave analysis in order to exclude the presence of pathology of cardiovascular character. As a rule, the high HRV patients show a considerably labile performance of the cardiovascular system that is accompanied by various clinical signs (unconsciousness, vertigo, sudden cardiac death events). Our concept of an explanation of the mechanism responsible for high HRV suggests further questions as listed below:

1. What treatment & prevention measures should be taken for high HRV patients in order to improve their ability to withstand stress factors?
2. What features of clinical progression of various diseases might be expected in patients of this type?
3. Should any prophylactic medical examinations be provided for the above patients on a regular basis, with due consideration of their anatomical & physiological peculiarities of the cardiovascular system?

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Anatomy and physiology of the sinoatrial node: modern concepts

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Deciding to discuss the above topic is determined due to a number of reasons. Firstly, the currently used ablation techniques make possible to reach the area of the sinoatrial (SA) node that required an accurate description of the node localization and possible ways of carrying out ablation procedures. Elucidation of SA node anatomical features in a human will allow solving the problem of the multifocal activation of atria and, as a sequence, the ablation of intranodal reentrant tachycardia. Secondly, over the last decade some disputes on the cardiac pacemaking mechanisms do not end in many international journals. Probably it is high time to submit this issue for discussion by our Russian national community of electrophysiology experts.

Recent reports by Lakatta E.G. and Maltsev V.A. have significantly improved the model according to the Di Francesco's membrane theory. Finally, the latest studies demonstrate that beating rate is controlled, in part, by local Ca²⁺ releases (LCRs) that creates a Rhythmic Ca²⁺ Clock in cardiac pacemaker cells.

Thus, it is necessary to enumerate some stages in the history of the detection of the sinoatrial node and the relevant studies on its performance. The first models of the SA structure: the pacemaker cells located in the SA node and the cluster-type structure of the said SA node. The two types of the pacemaker cells distribution in the nodal area: the model of gradual transition from typical nodal to atrial cells separated by an area of transitional cells versus the mosaic-type model of distribution of the nodal cells. Introduction of the definition of the transition area cells. Methods of the SA area identification. Electrophysiological features of the pacemaker cells and the atrial myocardium and capabilities of their interaction. Normal conduction pathways: how many of them are available, where do they located and how far do they extend? Intranodal reentrant tachycardia. A sequential alteration of the normal conducting pathways as a possible cause of pacemaker migration. An example of such combination based on the Holter monitoring recording. Currents responsible for control of the spontaneous diastolic depolarization. The Di Francesco's membrane theory: the leading role in potential-dependent If current. Development of another competitive pacemaking theory. Is it possible to combine the "membrane" and "the Rhythmic Ca²⁺ Clock" within the framework of a new model? Some prospects for artificial pacemaking cells.