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Root of knowledge in exercise physiology

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This article aims to give a personal view on the training that teachers must acquire in order to teach an organism to respond and adapt to exercise and training, respectively. It is believed that to achieve a high degree of competence in exercise physiology; texts and manuals of human physiology need to be studied in great depth. In my personal view, once teachers have acquired and consolidated knowledge in human physiology, logical reasoning will be sufficient to enable them to understand the two main objectives of exercise physiology (response and adaptation). From this view point, the role of the nervous system in the interrelationship between locomotion and the cardio-respiratory system was reviewed by the author. Based on the interrelation between locomotion and cardiorespiratory function, two integrators parameters were analyzed for endurance exercises: the maximum oxygen consumption and anaerobic threshold. The analysis is intended to express an honest, relevant knowledge for any teacher in exercise physiology. But mostly, the purpose of this article is for the reader to think carefully about the need to know the functioning of the body at rest (human physiology) and through logical reasoning to know how it responds to exercise. In short, this article, based on the experience of teaching human physiology for 30 years, is both thoughtful and provocative.

Key words: Human physiology, exercise physiology, response, adaptation, oxygen consumption, anaerobic threshold.

INTRODUCTION

When this brief review was carried out on the history of exercise physiology (Bassett, 2002; Hale, 2008; Johnson, 2014; Tipton, 1997; Wilmore, 2003), the following question was asked: does this discipline have its own entity? To answer this question, one must address two issues: 1) the objective of the study, this aspect of human physiology 2) the training that all professionals who are interested in this aspect of human physiology should have. Note, the study of the physiological function of human beings in a state of rest considered exercise physiology; while in reality, it should be considered as part of animal physiology.

Objective of the study of exercise physiology

Generally, this part of human physiology has addressed two important aspects: response adjustment and

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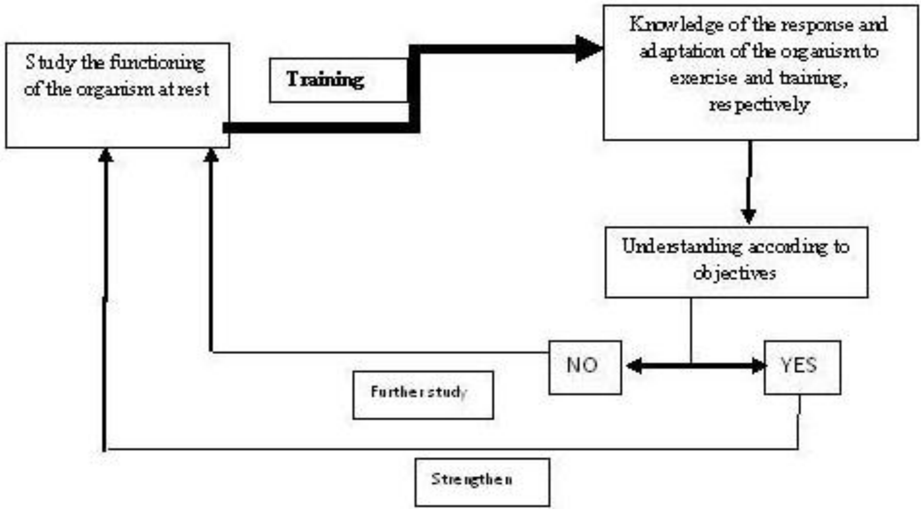


Figure 1. Simple scheme of the application of the knowledge of the body function at rest.

adaptation. Highly dynamic and moderately static sports (Mitchell et al., 2005) trigger a series of changes in the body that may develop efficiently even over long periods of time, as in all ultra-endurance tests. These changes may be temporary and disappear after exercise. These are what are known as 'responses' or 'adjustments'. However, when the changes remain over time, whether they are structural or functional changes or both, facilitating a better response to the same stimulus, they are known as adaptations. The result of biological adaptation is that the body responds better to the same stimulus. The simplest example of the difference between response and adaptation is the relationship between heart rate and exercise intensity. The function in these two variables is the same $(HR = a \cdot intensity + b)$ both for response and adaptation. The difference is that at submaximal intensity, when this organ has adapted, the heart rate is lower. Therefore, throughout history, all physiologists interested in exercise have tried to study these two major processes.

What training should exercise physiologists have?

As pointed out earlier, it seems obvious that an "exercise physiologist" should have an in-depth knowledge on how the organism functions in rest conditions and, if possible, extensive experience in animal models other than humans. This is the training that must be achieved. Archival Hill (Bassett, 2002), for example, had this training and all those who contributed to the knowledge in physiology with an interest in exercise. Such as those who were working in the Harvard laboratory (Johnson, 2014) in the United States or those who worked in different institutions in Scandinavian countries (Ottosson, 2010; Pernow, 1996).

Therefore, as indicated by Calderón (2012) in the diagram (Figure 1), depending on the level that needs to be achieved, it is sufficient to have an in-depth knowledge of physiology and use logical reasoning, which involves putting the organism in a stress situation, such as physical exercise. This understanding of exercise physiology is not normally shared by the vast majority of exercise physiologists, as demonstrated by the number of existing publications that are based on an understanding of self-identity (Costill et al., 2012; Hale, 2005; McArdle et al., 2006; Noble, 1986; Scott and Edward, 2001; Wilmore, 2003). The authors of these publications believe that it has an independent entity since it has a specific objective, which is knowing the response and adaptation of the organism, adding terms such as "applied" or "application", etc. to the generic name "exercise physiology". In line with Pasteur's idea ("*There are no such things as applied sciences, only applications of science")*, the exercise physiology is simultaneously the result, simple and complicated, of using reasoning on the basis of solid knowledge in relation to how the organism functions at rest.

Lastly, it is important to highlight how the process of adaptation to training is addressed. Normally, for any physiological variable studied during exercise, sedentary subjects are compared with trained subjects and never individuals with a similar degree of training. What could the reasons for this be? They are diverse and include: 1) that the instruments used are not accurate enough to determine small physiological variations, 2) that the differences in performance are multifactorial and not only physiological. Consequently, distinguishing between the champion, who systematically wins successive championships in which the best athletes of their

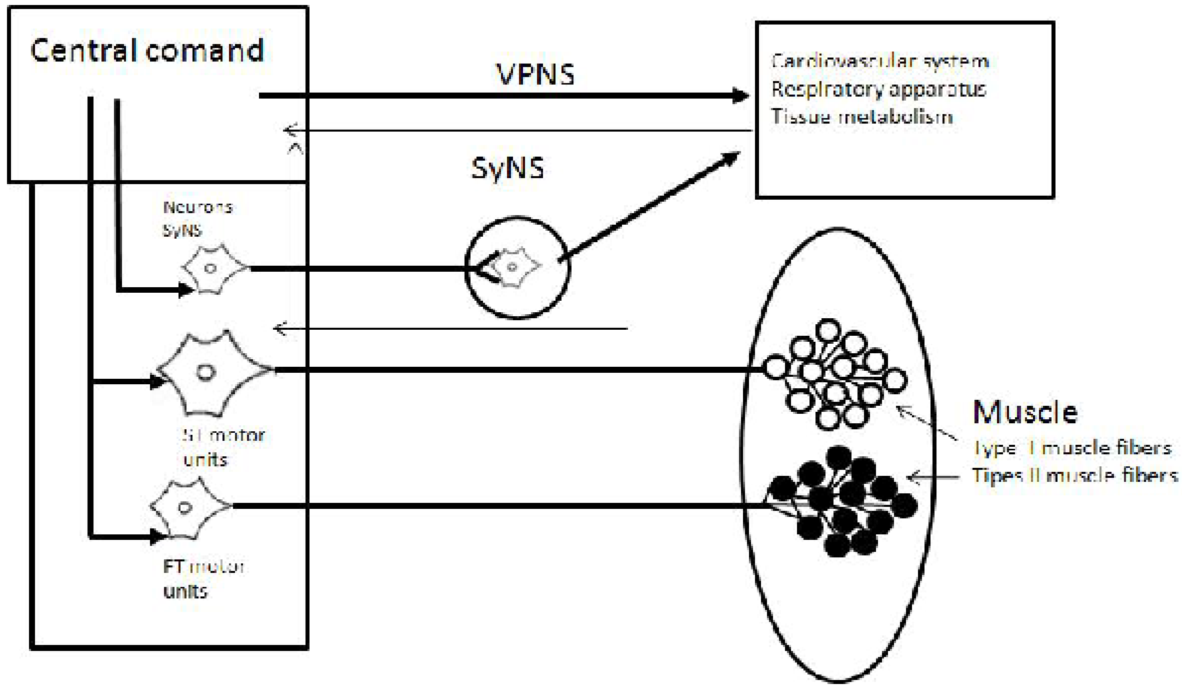


Figure 2. Simplified representation of the organization of the nervous system.

discipline compete, and the rest of the elite athletes is a complicated task to say the very least. We should therefore not be surprised that exercise physiology books only establish “extreme” differences (sedentary versus trained).

There are two alternatives for arguing the idea presented: 1) analyse using systems (cardiovascular, respiratory, etc.). The basis on which the knowledge of the response and adaptation of each participant to exercise and training is supported respectively, 2) analyse the integrated response and adaptation to exercise.

The first analysis option is unfeasible due to the nature of this article. Therefore, will proceed with the integrated view that constitutes the “physiologically most complete” exercise, that is classified as highly dynamic and moderately-low static (Mitchell et al., 2005). It should be pointed out that given the large number of references in the literature, the argument will be based on the review of texts and articles.

COMPRESSION OF THE INTEGRATED RESPONSE TO EXERCISE

Role of the nervous system

Minimal training in the anatomy and physiology of the Nervous System (NS) shows us the role that it plays during exercise. Although it is obvious, the function of the NS during exercise is absolute. The NS governs both the control of locomotion and the function of all the systems that allow oxygen and metabolic substrate supply, such as the cardiovascular and respiratory systems. Furthermore, the same simple anatomy and physiology knowledge of the NS shows the difficulty of analysing the response of the NS to exercise. Nevertheless, despite the difficulties indicated, below a speculative analysis will be carried out, certainly with a scientific basis and common sense, of how the NS participates in the control of movement and the new homeostasis situation that occurs, for example, in ultra-endurance events. The importance of the nervous system in the comprehension of the organism’s integrated response to exercise is illustrated in Figure 2. This simplified model determines that:

(1) The Somatic Nervous System (SNS) must sequentially activate the motor units in relation to the intensity of exercise, in line with Henneman’s principle (Henneman, 1981; Henneman, 1965), through descending motor pathways that establish synapses with the motor units of the muscles involved. That is, carrying out a basic motor pattern such as locomotion.

(2) The Autonomic Nervous System (ANS) must activate the neurons of the intermediolateral horn of the spinal cord while regulating the activity of the neurons located in different parts of the telencephalon. That is, activate the

thoracolumbar system (Sympathetic Nervous System = SyNS and decrease the activity of the craniocaudal system (Vegetative Parasympathetic Nervous System = VPNS).

(3) At the same time, the telencephalon is receiving various types of "afferent signals" that allow it to regulate the recruitment of motor units and the activity of the cardiovascular and respiratory systems in accordance with the intensity of the exercise.

It is easy to understand the lack of knowledge about all considered aspects. What seems obvious is the exquisite coordination that must occur between the two "parts" classically described in books: SNS and ANS. Section 5 (Locomotor and Respiration Interdependence) of chapter 11 of the guide by Rowell and Shepherd (1996) and more recently the review by Gossard et al. (2010) are sufficiently expressive. These articles establish the relationship between control of movement and autonomic control.

However, analysis of the figure leads to the many questions whose answers are unknown and that are analysed simply as:

1) What nervous centre or centres lead the somatic-autonomic coordination? In the terms of the diagram in the figure, one could also ask the question in the following manner: what part of the telencephalon "leads" the coordinated action? The abundance of articles is noticeable: when performing a search in medline with the following terms "central command AND exercise" there are 315 entries when the terms are found in any part of the text. As locomotion is extensive at any phylogenetic level of the brain, it seems logical for the central command to be a "functional expression" of the relationship between the limbic lobe and the diencephalon. In the review carried out by Dempsey et al. (1996) they establish a series of nervous centres involved in somatic-autonomic governance during stress reactions, such as exercise.

2) How is the recruitment of active motor units that are active during exercise, regulated or controlled? Much of the information available, being indirect, comes from the degree of glycogen depletion in the muscle fibres during exercise (Gollnick et al., 1974; Vøllestad and Blom, 1985). These studies confirm the principle established by Henneman (1965) and Henneman (1981) and subsequently confirmed by Burke (1981) in relation to the order of recruitment: ST units-FST units-FT units. It is extremely complex because the electrophysiological characteristics of the motor units that allow the order of recruitment to be explained (Burke, 1981), need to be considered in conjunction with the activity of the descending pathways in the group of anterior horn motorneurons.

3) How are the cardiovascular and respiratory functions regulated or controlled with the purpose of adapting them to the needs of the locomotor system? There is a considerable amount of knowledge about cardiovascular and respiratory control in a state of rest and, to a lesser extent, during exercise. For example, Figures 3 and 4, despite their simplicity, show the complexity of cardiovascular and respiratory control.

4) Lastly, how does the NS select the autonomous neurovegetative activity, guiding it towards the active systems and decreasing it in the seemingly inactive systems? The renal and digestive systems are the two great "ignored" systems in exercise physiology books. We believe that it is a mistake not to focus on these two systems. As in any exercise physiology textbook there are chapters on the response and adaptation of the cardiovascular, respiratory, endocrine systems and metabolism, there are specific chapters on the digestive system or the kidney only in a few books (Garrett and Kirkendall, 2000; Noble, 1986), clearly with a pathophysiological approach. Not even a monographic guide on exercise physiology published by the American Physiological Society (Rowell and Shepherd, 1996) addresses digestive and renal functions during exercise.

There may be two primary reasons for this. Firstly, it is considered that during exercise, these two systems are "silent", that is, they do not participate directly. The following remarks testify to this. The redistribution of cardiac output during exercise causes a percentage decrease in enteric and renal flow. Secondly, the experimental difficulties of assessing digestive and renal function at rest are accentuated during exercise. However, it is true that studies or reviews have been carried out (Castenfors, 1966, 1977; Hinchcliff et al., 1997; Luciani et al., 2010; McKeever, 1998; Poortmans, 1984; Poortmans and Vanderstraeten, 1994), for example on renal function in relation to exercise. However, these studies are aimed at explaining the pseudopathological manifestations that can occur as a result of exercise and not at explaining the physiological role.

During exercise, the activity of the NS causes a redistribution of cardiac output. Does the reduction of renal blood flow (RBF) affect renal function during exercise? To explain how renal function responds and what physiological mechanisms would allow to explain potential modifications in glomerular and tubular functions, it is necessary to have an in-depth knowledge of the control of these functions at rest and therefore study them.

In summary, to know the full extent of the inter-relationship between locomotion and the cardio-respiratory system upon exercising, it is necessary to extensively study neurophysiological control in a state of rest. This knowledge is the starting point for understanding, to the extent that scientific knowledge allows it, the integrated response of the organism to exercise.

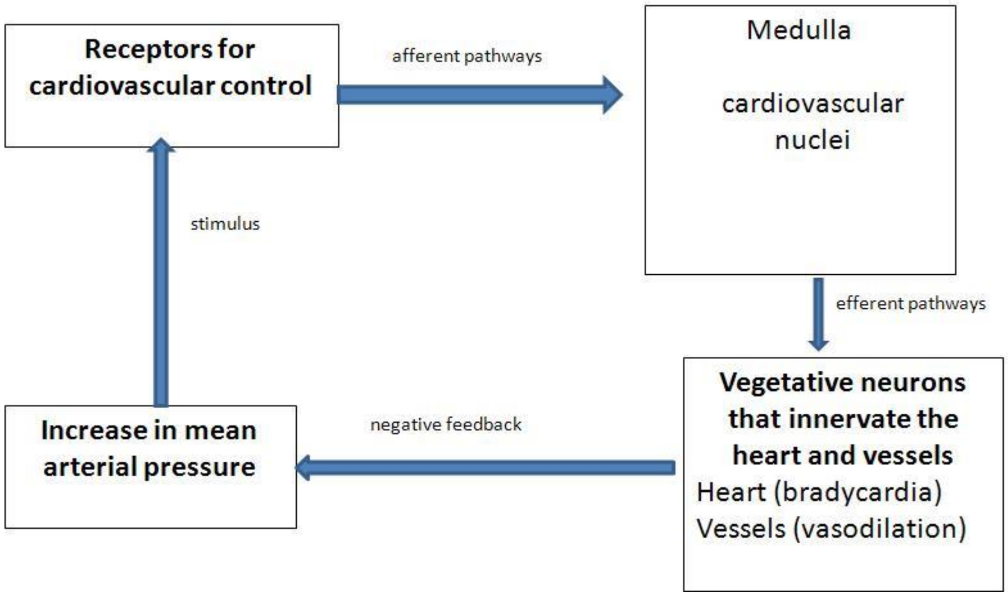


Figure 3. Feedback mechanism of the cardiovascular control.

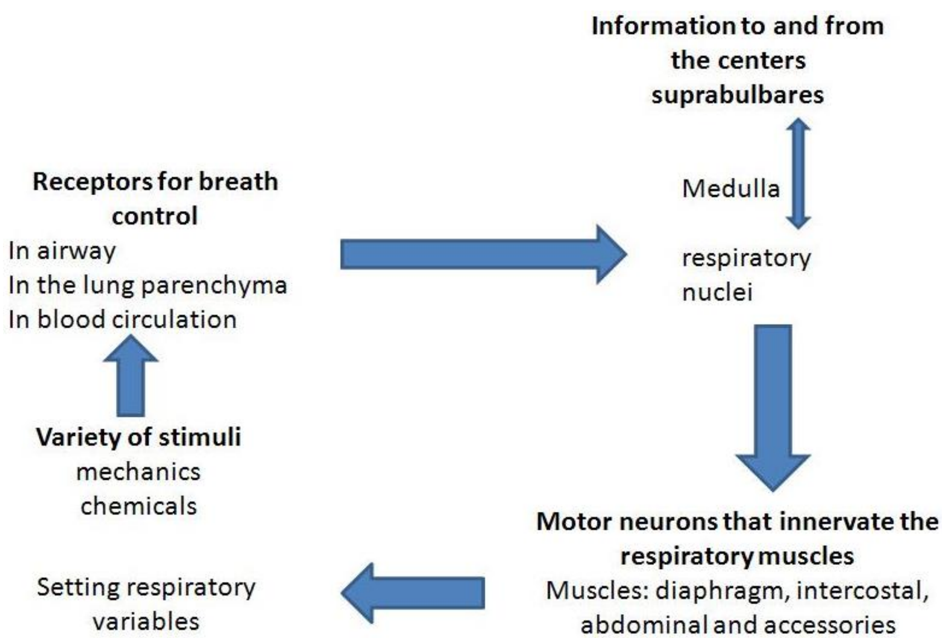


Figure 4. Feedback mechanism of the respiratory control.

Integrating parameters

The integrating vision allows one to know the parameters that are the result of the coordinated function between different physiological functions that allow the balance “offer” and “use” of energy. There are two integrating parameters: oxygen consumption (VO_2) and, simply stated, the anaerobic threshold (AT).

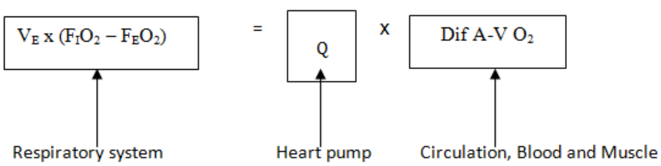
Oxygen consumption: central integrating parameter

To have an overall understanding of this integrating parameter, we can express VO_2 as follows. Removing

VO_2 from Fick's equation (Equation 1) to determine the mean cardiac output, assuming that V_I is equal to V_E (Equation 2), and equating the two equations (Equation 3), a good conceptual approximation of VO_2 is obtained.

$$Q = \frac{VO_2}{Dif\ a - v\ O_2}\ ;\ VO_2 = Q\ x\ Dif\ a - v\ O_2 \quad (1)$$

$$VO_2 = [(V]_I \times F_IO_2) - [(V]_E \times F_EO_2) = V_E\ (F_IO_2 - F_EO_2) \quad (2)$$



(3)

It can easily be deduced that VO_2 is a parameter that integrates the following physiological functions: 1) cardiovascular, 2) respiratory and 3) metabolic (muscle). The idea of the integration contributed by this parameter may be demonstrated by how it is used in the assessment of individuals who are healthy, trained and have a disease (Wasserman et al., 1999)

Anaerobic threshold (AT)

The term anaerobic threshold was coined by Wasserman and McIlroy (1964), when he used the respiratory exchange ratio (RER) to detect the start of anaerobic metabolism in individuals with heart disease whilst performing an exercise test. From the definition provided by Wasserman, there are a large number of studies that try to explain the relationship between the anaerobic threshold by respiratory exchange and the moment in which there is an increase in the concentration of lactate in the plasma. However, this relationship is difficult to explain physiologically, as is demonstrated by the relationship with other parameters as disparate as the composition of saliva, electromyographic activity, catecholamine concentration and even heart rate.

The interpretation given by Peinado et al. (2014) for this complex phenomenon suggest that the AT is the integrated result of the diversified response of the NS to a situation close to the maximum exercise limit. The NS activity would explain the variation in the response experienced by the respiratory system, the cardio-vascular system, certain glands and the muscle tissue. The problem of this understanding is that, in simple terms of NS functioning, the afferent information is unknown and nervous centres of the CNS are involved in the receiving and in giving corresponding orders for the PNS to proceed to such a diverse activation. It is only the result of the activity of the PNS, which is detected as:

(1) Increased ventilation
(2) An increase in catecholamine concentration
(3) Variations in the concentration of amylase and different ions in saliva
(4) Variations in the electromyographic pattern, which suggests greater activation of the FT motor units and as a result of an increase of lactate in plasma
(5) A variation in the HR/intensity relationship slope.

ADAPTATION OF THE ORGANISM TO RESISTANCE TRAINING

As complex as the physiological analysis of the integrated response to exercise with a high dynamic and low-moderate static component is, knowing the physiological mechanisms of the adaptation process is even more difficult. Below are three very specific examples, formulated as questions, which show us the complexity of knowing the mechanisms that explain the phenomenon of adaptation to training.

1) Does bradycardia following training and reported in all exercise physiology books, follow a modulation of the activity of the PyNS VPNS on the sinus node and/or an electrophysiological modification of the P cells mainly present in the sinus node?
2) Is the increased oxidative capacity of the skeletal muscle tissue following training due to an intrinsic adaptation phenomena and/or changes in the different motor units?
3) Is higher cardiac output following training a result of improved systolic ventricular function and/or diastolic ventricular function?

Despite that above mentioned, the physiological mechanisms that allow us to explain the training adaptations of the organism have been studied extensively. However, for the objective of this study, as well as for the response of the organism to exercise, the approach will be integrated, using the analysis of the most integrating physiological variable: oxygen consumption.

Adaptation of maximum oxygen consumption to training

When in Equations 1 and 2 the dependent variables have achieved their maximum levels, we obtain the maximum consumption of oxygen or equivalent terms (maximum aerobic capacity, peak oxygen consumption, maximal aerobic power etc.). Therefore, from an integrated physiological understanding, and through the analysis of

Equation 3, the maximum VO_2 would be the result of:

1) An improvement in the oxygen “uptake” capacity by the respiratory system, that is, increased alveolar ventilation (AV).
2) An improvement in the “pumping” (systolic ventricular function) and “filling” capacity (diastolic ventricular function) of the ventricles. It is necessary to consider that although both circulations are anatomically parallel, functionally they are in a series. Thus, improved ventricular function refers to both ventricles.
3) An improved capacity for ‘using’ oxygen, which, also in simple terms, means an improvement in enzyme capacity.
4) An improvement in the capacity of haemoglobin to extract oxygen, which will lead to a greater arteriovenous difference. This improved extraction capacity would be a result of an increase in the “supply” of blood, which in the most elementary of terms is an increase in the capillary surface and it has been indicated and as the objective of this study, this elementary analysis involves many difficulties that are clearly demonstrated. It is obvious that when starting from a low or very low VO_2 maximum level, its increase following a period of training may be the result of the adaptation of one or all of the factors indicated. However, when it is dealt with elite athletes, the variations of this parameter do not exist or cannot be explained from a physiological point of view, even though the athlete may experience improved performance. In terms of explaining the physiological factors that limit maximum VO_2, each researcher basically develops their opinion due to their interest in the research. In educational terms, the maximum VO_2 limiting factors are divided into central and peripheral.

Summary of the theories on the mechanisms of adaptation to training

As we have mentioned, it seems like a fantasy to address how adaptation occurs and, specifically, how the increase in the maximum VO_2 with training can be explained. Nevertheless, there are various theories proposed to explain the process of adapting to training. On the basis of theories relating to the adaptation of the organism to stress in general, the following theories have been applied to the process of training:

The ontogenetic theory, which assumes that each animal is equipped not only with a minimum volume for each organ necessary for survival, but that it has safety margins for potential contingencies. As such and as a result of greater physiological demand, there would be morphological “compensation” which would lead to an adapted function. However, this theory cannot be applied globally to the adaptation of the organism to training since not all organs adapt in the same way. Some tissues or organs increase their mass by creating new units, such as the thyroid gland or the ovaries. Other organs, however, only adapt by cell proliferation.

The functional theory proposes that any functional change occurring in an organ induces trophic stimulation. However, this theory cannot be absolutely applied to the phenomenon of adaptation to training. Firstly, morphological adaptation to a greater demand is limited and it is different depending on the organ or tissue. The intermittent or continuous function of the organ determines whether growth is mitotic or not. For example, most glands whose function is intermittent tend to grow by cellular division. Other organs or tissues, which function continuously, cannot adapt by cellular division, since an overproduction would clearly be harmful.

The overcompensation theory proposes that when the function of an organ decreases temporarily and subsequently when the demand for energy increases, there is a greater compensation than if the previous decrease of the function had not occurred. This theory was supported in results relating to concentrations of certain substrates (glycogen and phosphocreatine). When there is a depletion of muscle glycogen as a result of a high training load, it was found that during the recovery period the deposits of this fuel exceed previous levels.

The theory of degradation/synthesis establishes that there is a relationship between the degradation and synthesis processes, with the first being a stimulus for the second. There is scientific evidence that support it, such as in enzyme cell self-regulation or the mechanisms of neurohormonal regulation.

The abovementioned theories must be understood as complementary alternatives, not to be taken on their own, and in some cases as supplements. As such, for example, the phenomenon of overcompensation is closely linked to the increase in degradative activity. In any case, all of the theories allude to the fact that for there to be adaptation, there must be “chronification” of the stimulus, which is extended to the training process. However, the explanation of the “training effect” is closely related to the previous level of the organism and as such, it is relatively easy to understand how it occurs when the initial physical condition is low, but incredibly complex when the functional capacity is high, as occurs in elite sport.

Nervous system: the great ignored field of exercise physiology in an integrated explanation of the phenomena of adaptation to training

The main problem is that when physiological mechanisms are analysed, the central role that the NS plays is ignored. The NS is key to understanding not only neuromuscular adaptation (Gardiner, 2001), but also the adaptation of many other organs and tissues. Therefore, again with a simple knowledge of the organisation and function of the NS may result in one understanding how the adaptation phenomenon occurs. One of the organs that may explain the increase of the maximum VO_2 is the heart. The reason for focusing the analysis on this organ is that it is probably the most studied organ in the history of exercise physiology, mainly due to the application of technology to cardiac morph-functional assessment.

As previously stated in the introduction to this summary, bradycardia at rest and the decrease of the heart rate at submaximal intensities are one of the most thoroughly studied adaptation mechanisms. Starting from the idea that bradycardia originates from an increase in the activity of the VPNS greater vagal tone in vulgar terms, and it is analized in Figure 3, it is appreciated that it is difficult to know how it exactly occurs. Therefore, the only possibility that remains is that of asking questions that are difficult or even impossible to answer accurately.

1) Could post-training bradycardia consist of an adaptation of the pressure receptors located in the aortic arch and carotid sinus?
2) Could post-training bradycardia following an adaptation of the well-known bulbar nuclei be involved in cardiovascular control?
3) Given that one of the efferences of the baroreflex is directed towards the heart, can bradycardia be explained by an increase in its activity? That is, that which is generically known as an increase in the vagal tone.
4) Lastly, can the decrease in heart rate be explained by a variation in the response of P cells to levels of acetylcholine released by the VPNS nerve endings?

It is obvious that any person who has studied the baroreflex quite extensively will reply affirmatively to all of these questions, such that bradycardia is the result of a combination of the adaptation of: 1) receptors, 2) control nervous centres, 3) vagal efference 4) the response of the P cells of the sinus node. In any case, bradycardia is very complicated to explain. Lastly, the relationship between bradycardia and the morphological adaptation of the heart does not always have a reason to be established. The modifications of the heart with training, mainly echocardiography, have been extensively studied and summarised in review articles or articles with large populations (Maron, 1986; Pelliccia et al., 1999; Serratosa-Fernández, 1998)

CONCLUSIONS AND SUMMARY

The objective of this personal view article has been to highlight the root of exercise physiology. We understand that a very extensive knowledge about how an organism functions in a state of rest is the basis of exercise physiology. Greater knowledge of human physiology in better conditions would be required to understand the two main objectives of exercise physiology: 1) the response to exercise 2) the adaptation to training. Although it is obvious, it is necessary to highlight that without an in-depth knowledge of human physiology it is impossible to accurately address the two main objectives indicated.

Therefore, in order to achieve a high degree of competence in exercise physiology, one should extensively study the functioning of the organism at rest. Although there is a certain degree of overspecialisation, as inevitably comes with scientific knowledge, it is also valid. As such, for example, an expert in the response and adaptation of the heart should have an in-depth knowledge of ventricular function and determining factors. Lastly, it should be noted that losing sight of this understanding of exercise physiology is a mistake that may have serious consequences when training future exercise physiologists.

Conflict of Interests

The authors have not declared any conflict of interests.

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