



# Physical exercise and blood pressure control\*

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## ABSTRACT

Physical exercise causes a series of physiological responses resulting from autonomic and hemodynamic adaptation that affect the cardiovascular system. Many studies have demonstrated its beneficial effect on blood pressure. Bearing in mind that hypertension is a very prevalent condition causing high morbidity and mortality rate, the physical exercise plays an important role as a non-drug measure for its control or as an adjuvant to drug treatment.

## INTRODUCTION

Physical exercise is an activity presenting systematic repetitions of oriented movements feature with consequent increase on the oxygen intake due to muscular demand thus generating work<sup>(1)</sup>. The exercise represents a subgroup of physical activity designed with the objective of maintaining the physical conditioning<sup>(2)</sup>. It may also be defined as any muscular activity that generates strength and interrupts homeostasis<sup>(3)</sup>.

The physical exercise causes a series of physiological responses in the body systems, particularly in the cardiovascular system. With the objective of maintaining the cellular homeostasis in face of the increase on the metabolic demands, some mechanisms are set into action<sup>(4)</sup>. These mechanisms function under the form of reflex arches composed of receptors, afferent pathways, integrator centers, efferent pathways and effectors and many stages of these mechanisms have not yet been fully elucidated<sup>(5)</sup>.

## PHYSIOLOGICAL EFFECTS OF THE EXERCISE

The mechanisms responsible for the adaptations of the cardiovascular system to the exercise and the limitation indexes of the cardiovascular function are basic aspects related to the understanding of the adaptive functions. These mechanisms are multifactorial and allow the system to operate effectively at the most diverse situations. The physiological adaptations are performed from the metabolic demands in which information reach the cerebral trunk through the afferent pathways up to the bulbar reticular formation, where the central regulatory neurons are found<sup>(1)</sup>.

The physiological effects of the physical exercise may be classified into immediate acute, late acute and chronic. The acute effects, also called as responses, are those that occur in direct association with the exercise session; the immediate acute effects are those that occur in the peri and post-immediate physical exercise periods, with elevation on the cardiac frequency, pulmonary ventilation and sudoresis, while the late acute effects occur along the first 24 or 48 hours (many times up to 72 hours) after a physical

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exercise session, being identified in the slight reduction on the tensional levels, especially in hypertensive individuals, in the expansion of the plasmatic volume, in the improvement of the endothelial function<sup>(4,5)</sup> and in the action improvement and increase on the insulin sensitivity in the skeletal musculature<sup>(6-7)</sup>. Finally, the chronic effects, also called as adaptations resulting from the frequent and regular exposure to physical exercise sessions and represent morphofunctional aspects that distinguish an individual physically trained from an untrained individual, presenting as typical examples the rest relative bradycardia, the muscular hypertrophy, the physiological left ventricular hypertrophy and the increase on the maximal oxygen intake ( $\dot{V}O_2$  maximum)<sup>(4,5)</sup>. The exercise is also able to promote the angiogenesis by increasing the blood flow into the skeletal muscles and into the cardiac muscle<sup>(3,8)</sup>.

The regular practice of physical exercises promotes important autonomic and hemodynamic adaptations that will influence the cardiovascular system<sup>(7)</sup> with the objective of maintaining the cellular homeostasis in face of the increment of the metabolic demands. There are increases on the cardiac debt; redistribution on the blood flow and elevation on the circulatory perfusion into muscles in activity<sup>(4)</sup>. The systolic blood pressure (SBP) increases directly proportional to the increase on the cardiac debt. The diastolic blood pressure reflects the efficiency of the local vasodilator mechanism of the muscles in activity, which is as high as the local capillary density<sup>(1,9)</sup>. The vasodilatation of the skeletal muscle reduces the peripheral resistance to the blood flow and the sympathetically induced concomitant vasoconstriction occurring in non-exercised tissues compensates this vasodilatation. Hence, the total resistance to the blood flow drops significantly when the exercise starts, reaching a minimum value around 75% of the  $\dot{V}O_2$ max<sup>(3)</sup>. The tensional levels increase during physical exercise and during effort predominantly static, and intra-arterial pressure levels above 400/250 mmHg with no health damage have been verified in young and healthy individuals<sup>(4,10)</sup>.

In short, one may say that during an exercise period, the human body undergoes cardiovascular and respiratory adaptations in order to attend the increased demands of the active muscles and, as these adaptations are repeated, modifications in these muscles are verified, allowing the organism to improve its performance. Physiological and metabolic processes optimize the oxygen distribution throughout tissues in activity<sup>(11)</sup>. Therefore, the mechanisms that guide the post-physical training blood pressure drop are related to hemodynamic, humoral and neural factors<sup>(12)</sup>.

## PHYSICAL EXERCISE IN THE SYSTEMIC ARTERIAL HYPERTENSION TREATMENT

The systemic arterial hypertension represents one of the most important causes for cardiovascular morbidity in Brazil and affects from 15% to 20% of the adult population, also presenting significant prevalence in children and adolescents. Considered as one of the main risk factors for morbidity and cardiovascular mortality rate, it generates high social cost, once it is responsible for about 40% of the early retirement cases and work absenteeism in our envi-

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ronment<sup>(5,13)</sup>. The identification and treatment of patients with systemic arterial hypertension is a public health problem in Brazil.

The inactiveness is also important risk factor and the occurrence of a higher rate of cardiovascular events and a higher mortality rate in individuals with low physical conditioning level was already well established<sup>(14)</sup>. It is estimated that the inactiveness prevalence is of up to 56% in women and 37% in men considering the Brazilian urban population<sup>(15)</sup>.

Modifications on the life-style with the inclusion of physical exercises are recommended in the arterial hypertension treatment. Study involving 217 patients from both genders with ages ranging from 35 to 83 years showed that the adhesion of non-pharmacological measures, among which the practice of physical exercises, promoted perceptible effect on the reduction of the blood pressure levels<sup>(16)</sup>.

Randomized studies showed the undesirable effects of the pharmacological treatment in a subgroup of patients with systemic arterial hypertension, suggesting a change on the treatment approach of these individuals. The effect of the physical exercise on the rest levels of the blood pressure from light to moderate degree is especially important, once the hypertensive patient may reduce the dosage of his anti-hypertensive remedies or even to control his blood pressure without the adoption of pharmacological measures<sup>(7,15)</sup>. The trend of the early use of pharmacological measures has been replaced by non-pharmacological agents, among them, the aerobic physical exercise has been recommended for the treatment of the light systemic blood pressure hypertension<sup>(15)</sup>. The systemic arterial hypertension is a polygenic syndrome, being influenced by the genetic inheritance and only 75% of the hypertensive patients are responsive to physical training<sup>(7)</sup>.

Paffenbarger *et al.*<sup>(17)</sup>, in a 6-10 years follow up of 15.000 individuals graduated in Harvard, verified that those who practiced physical exercises regularly presented risk 35% lower of developing arterial hypertension when compared with inactive individuals.

In another study, Paffenbarger *et al.*<sup>(18)</sup>, followed 10,260 individuals graduated in Harvard for over than one decade and verified an inverse relation between the practice of physical activity and the risk of death due to all causes. When those who started sportive activity moderately intense (corresponding to an intensity of 4.5 or more metabolic equivalents) were compared with those who did not participated in this type of training, they observed a reduction of 23% in the death risk (confidence interval of 95%, 4% for 42%;  $p = 0.015$ ). The mortality rate decreased even among middle-aged individuals who increased physical activity through simple modifications in the occupational or recreational activities.

The search for an explanation for the effect of the exercise on the blood pressure of normal blood pressure individuals and especially hypertensive individuals has encouraged many researches in the last decades, and the reduction on the rest diastolic blood pressure after training is the topic most widely studied. The mechanisms that guide the post-physical training pressure drop are related to hemodynamic, humoral and neural factors<sup>(12)</sup>.

Among the hemodynamic factors, it was verified that both in rats spontaneously hypertensive and in humans, the practice of physical exercises reduces blood pressure due to the reduction on the cardiac debt, which is associated to the reduction on the cardiac frequency, once no alterations on the systolic volume were observed<sup>(12,19)</sup>. The drop on the systemic vascular resistance and hence on the blood pressure would be another alternative mechanism proposed in order to explain the post-exercise blood pressure drop<sup>(12,19,20)</sup>. A significant reduction on the pressure levels is obtained with low intensity training (50% of the peak oxygen intake). Thus, low intensity physical exercises reduce the blood pressure because they cause reduction on the cardiac debt, what may be explained due to the decrease on the rest cardiac frequency and the decrease on the sympathetic tonus in the heart as result of a

lower sympathetic intensification and higher vagal nerve removal<sup>(12,21,22)</sup>.

Some authors attribute the reduction on the blood pressure after the practice of physical exercises in hypertensive individuals to humoral alterations related to the production of vasoactive substances such as the atrial natriuretic peptide or the ouabaina-like, centrally modulated<sup>(12)</sup>. A significant improvement on the insulin sensibility also occurs<sup>(6,7,23)</sup>, besides the reduction of the plasmatic noradrenalin, suggesting reduction of the sympathetic nerve activity associated to the increase on the serum taurine and prostaglandin E, which inhibit the release of noradrenalin into the sympathetic nerve terminations and the reduction on the ouabaina-like factor, what would cause a noradrenalin recapture in the synaptic clefts<sup>(12)</sup>. This hypothesis is refused, once a reduction on the blood pressure can be demonstrated even before a reduction on the plasmatic noradrenalin levels is verified. Other authors report that the noradrenalin levels decrease with training only for hyperadrenergic individuals. A reduction on the plasmatic renin levels was also observed as well as an increase on the production of nitric acid<sup>(6,12,24,25)</sup>.

The aerobic training performed through exercises predominantly isotonic or dynamic generally does not change the rest systolic and diastolic blood pressure levels in normal blood pressure individuals, even though the average blood pressure may decrease in function of the lower basal cardiac frequency after physical training period<sup>(6)</sup>.

Grassi *et al.*<sup>(26)</sup> studied young normal blood pressure individuals and verified that after 10 weeks of physical exercise, besides the decrease on the systolic and diastolic blood pressure, a significant reduction on the sympathetic nerve activity was also observed (36%), fact not observed for the control group composed of those who did not perform physical exercises<sup>(12)</sup>.

With regard to the acute effect of the exercise on the 24-hour blood pressure curve in patients evaluated through the blood pressure ambulatorial monitoring (BPAM), Marceau *et al.* demonstrated that individuals trained at 50% and at 70% of the  $\dot{V}O_2$ max presented different pressure curve profiles; the individuals trained at 50% of the  $\dot{V}O_2$ max maintained reduction exclusively during the wake period, while the individuals trained at 70% of the  $\dot{V}O_2$ max maintained reduction even during the sleep period<sup>(27)</sup>.

Ishikawa *et al.*<sup>(28)</sup> studied 109 hypertensive individuals in stages I and II who performed mild exercise training for eight weeks in sports academies. They verified a significant reduction on the blood pressure in all of them, where the elder individuals presented lower reduction on the blood pressure levels if compared to the younger individuals. No influence of the genders on the results was verified.

More recently, Takata *et al.* submitted 207 individuals with essential hypertension of degrees 1 and 2 to a physical exercise program for eight weeks. The individuals were divided into five groups according to the exercise weekly duration and frequency (control group – inactive individuals, 30-60 minutes/week, 61-90, 91-120 and above 120 minutes/week). They verified that the rest diastolic blood pressure did not change for the control group; however, a significant reduction on the rest systolic and diastolic blood pressure was verified in the four groups submitted to the exercises. The reduction on the systolic blood pressure was higher for the group of 60-90 minutes/week, when compared to the group of 30-60 minutes/week. No higher reduction with the increase on the exercise volume was verified. The reduction on the diastolic blood pressure was not significantly different in the four groups. There was no obvious relation between the weekly exercise frequency and the magnitude of the decrease on the blood pressure cause by exercises<sup>(29)</sup>.

Hypertensive individuals maintain the most intense reduction on the blood pressure in the 24 hours after exercise<sup>(9)</sup>. Blood pressure evaluations in 25 and 45-minutes sessions after physical exercise at 50% of the maximal oxygen intake demonstrated more intense reductions after the 45-minutes sessions<sup>(30)</sup>. In this case, it

is possible that the blood pressure drop is due to the decrease on the peripheral vascular resistance<sup>(20,24,31)</sup>, and this fact may even be related to the vasodilatation in the active and inactive musculature caused by physical exercise as result of the accumulation of muscular metabolites (potassium, lactate and adenosine) or due to the dissipation of the heat produced by the physical exercise<sup>(30,32)</sup>. Alternatively, the increase on the blood flow may be a result of the reduction on the sympathetic tonus and the consequent increase on the peripheral vasodilatation<sup>(32)</sup>, which seems to be related to the increase on secretion of endogen opioids caused by exercise, presenting direct vasodilator effect<sup>(30)</sup>.

Cardiopulmonary and blood pressure receptors functional alterations such as the increase on its sensibility and the modification on its activation point and the recovery time may also contribute to the post-exercise vasodilator effect. The reduction on the alpha-adrenergic vasoconstrictor response verified in the recovery period – “down regulation” of the alpha-adrenergic receptors could also explain the higher post-exercise muscular blood flow. And yet, humoral factors such as adrenalin, the natriuretic atrial factors and the nitric oxide have been cited as factors involved in the post-exercise vasodilatation<sup>(9,30)</sup>.

Surprisingly, studies with rats spontaneously hypertensive showed that the decrease on the total peripheral vascular resistance was not the hemodynamic mechanism responsible for the decrease on the blood pressure after low intensity training, but rather a reduction on the cardiac debt<sup>(22)</sup>. In the study of Overton *et al.*<sup>(31)</sup>, blood flow measurements through Doppler in the iliac, renal and superior mesenteric arteries of rats genetically hypertensive trained at 60% and 70% of the  $\dot{V}O_2\text{max}$  showed no decrease on the regional vascular resistance in any of the arteries, suggesting that the reduction on the cardiac debt is the mechanism responsible.

## RECOMMENDATIONS FOR THE EXERCISE PRACTICE IN THE ARTERIAL HYPERTENSION

The Brazilian Cardiology Society recommends that hypertensive individuals start programs of regular physical exercise since submitted to previous clinical evaluation. The exercises must be of mild intensity, from three to six times a week in sessions of 30-60 minutes duration, performed with cardiac frequency between 60% and 80% of the maximum or between 50% and 70% of the  $\dot{V}O_2\text{max}$ <sup>(33)</sup>.

With slight modifications, these recommendations are in agreement with the recommendations of other international entities<sup>(34,35)</sup>.

## CONCLUSIONS

One concludes that the beneficial effects of physical exercises must be considered in the initial treatment of the hypertensive individual, aiming at avoiding or reducing the number and dosage of remedies. For inactive and hypertensive individuals, reductions clinically significant on the blood pressure may be obtained with the increase relatively small of physical activity, above the level usually employed for inactive individuals; besides, the exercise volume required to reduce blood pressure may be relatively small, being possible to be reached even by inactive individuals.

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