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Chapter 5

# Exercise for Hypertension

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**Abstract**

High blood pressure (HBP) does not cause discomfort but still, you need to take care of and treat it. Otherwise, over time, it can damage the heart, kidneys, brain, and eyes. Lifestyle changes are essential in HBP, and physical activity is a parameter of great influence. In order to achieve the benefits derived from physical activity, it must be adequately prescribed, an aspect that will be developed in this chapter. The first section addresses the physiopathology of hypertension, with special interest in the pathological mechanisms that may induce hypertension, devices for monitoring blood pressure (BP), and an overview of the particularities that present hypertension in the presence of other pathologies and over the life span. Second section focuses on exercise prescription for hypertensive people, exploring each type of exercise that has been proved to be effective. We discuss for each type of exercise, the benefits, mechanisms involved in these benefits, the appropriate dose of exercise, and other methodological considerations including risk management issues. We conclude with a clinical case study. A detailed exercise training program will be developed for this particular case study in order to try to bridge the gap between theory and practice.

**Keywords:** primary hypertension, blood pressure, resistance training, aerobic exercise, lifestyle

## 1. Introduction

High blood pressure (HBP) is the greatest public health problem worldwide because of its high prevalence and because it is associated with increased cardiovascular and renal complications. In the year 2000, hypertension in the adult population stood about 26.4%. Projected calculations for 2025 estimate the prevalence at 29.0%. In absolute terms worldwide, this

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increase will go from 972 million hypertensives in 2000 to 1,560,000,000 in 2025, representing a raise of approximately 60% [1].

Hypertension is the most prevalent cardiovascular disease (CVD), affecting 20–50% of the population in developed countries. Its prevalence increases with age, especially after 50 years old when it affects more than 50% of the population. All segments of the population (pregnant women, elderly, people with diabetes, etc.) are susceptible to developing a pathological elevation of blood pressure (BP). Hypertension is usually diagnosed when systolic blood pressure (SBP) is ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg, sustained constantly. However, the dividing line between normal blood pressure and hypertension is arbitrary and actually artificial. The value of BP is the result of the interaction of genetic and environmental factors (e.g., *polygenic inheritance, salt intake, smoking, stress, diet, etc.*).

A high BP has been identified as a risk factor for coronary artery disease (CAD), heart failure (HF), stroke, and peripheral arterial insufficiency. Hypertension is associated with earlier changes in organ systems, such as left ventricular (LV) hypertrophy, renal failure, retinopathy, and vascular dementia, which are grouped under the term "target organ damage" (TOD). Furthermore, hypertensive patients have a higher incidence of cardiovascular comorbidities (e.g., *diabetes, insulin resistance, dyslipidemia, etc.*) compared to the general population.

Currently, HBP represents a major challenge to public health as it is frequently associated with sudden death due to the silent nature of the condition. The first step is to secure a firm diagnosis. The European League of Hypertension recommends the use of an ambulatory BP measurement (monitoring ambulatory blood pressure (MAPA)), which is a device for automated blood pressure measurement repeatedly for 24 h. This monitoring test provides a more accurate picture of the circadian pattern of blood pressure changes over an average day and night. Recently, there has been a marked interest in this circadian pattern and the variability of BP because having an abnormal BP pattern has been related to increased cardiovascular risk and other health problems. In particular, night blood pressure seems to be the best predictor of cardiovascular events and the degree of development of cardiovascular disease. The MAPA can also differentiate permanent hypertension and hypertension secondary related to stress (white-coat effect). Scientific evidence regarding the effectiveness of the MAPA has mainly emerged from the CARDIORISC project in Spain, which includes the largest global database available with more than 40,000 patients [2].

Inadequate treatment of hypertension increases drastically the potential damage on the main target organs (*e.g., heart, brain, kidney, retina, etc.*). There are several nonpharmacological strategies for preventing and treating hypertension, such as the DASH diet and physical activity. Recent evidence suggests that higher physical activity levels are associated not only with lower diurnal BP levels but also with lower levels of night BP, which represents a healthier circadian pattern and thus a reduced cardiovascular comorbidity. There is a considerable amount of scientific evidence, including meta-analysis, supporting the antihypertensive effects of different types of exercise for blood pressure, such as continuous aerobic training. However, the appropriate type, intensity, time, and frequency of exercise remain unclear. In parallel, other types of exercise such as isometric strength training or high-intensity aerobic training (HIT) are gaining attention in the literature, showing very promising results. In order to achieve

and maximize the benefits of exercise safely, professionals should have updated guidelines for prescribing exercise for hypertension. These guidelines should be based on scientific evidence and provide precise practical information about how to design, implement, and control a training program.

## 2. Pathological mechanisms

### 2.1. Etiological factors

#### *2.1.1. Genetics*

From a clinical standpoint, the influence of genetics in hypertension is determined by a familial aggregation, so that the prevalence increases among relatives of first degree. Besides this clinical observation, knowledge of the genes involved in the development of hypertension is scarce. It seems clear that genetic determinants can be modified by environmental ones, so that the blood pressure or the resulting phenotype depends on the interaction of both factors. Genotypic and phenotypic research will be of great importance in the future as it may be able to help develop personalized treatments and prevention programs based on these findings [3–6].

#### *2.1.2. Environmental factors*

The main factors related to the development of hypertension are linked to the progress and changes in lifestyle and diet. The mechanisms by which these environmental determinants increase blood pressure and promote the development of cardiovascular disease are not fully clarified but it seems that a high caloric intake and decreased energy expenditure produce sympathetic hyperactivity [3–6].

#### *2.1.3. Possible pathological mechanisms*

*1. Sympathetic overactivity*. It is characterized by an imbalance between sympathetic and parasympathetic activity. Sympathetic activation may be caused by a direct stimulus by chronic stress, high caloric intake, obesity, and physical inactivity among others. 2. *Renin-angiotensin system (RAS)*. The renin-angiotensin system is undoubtedly mainly responsible for the development of vascular disease and one of the main focuses of therapeutic care. Angiotensin II is the main effector system with specific receptors at different levels that promote vasoconstriction and fluid retention, the latter thanks to the stimulation of adrenal aldosterone secretion as well as inflammatory, proaggregatory, and prothrombotic reactions. 3. *Dysfunction and endothelial injury*. The endothelium plays a fundamental role in vascular pathophysiology alterations observed in hypertension. Cardiovascular complications include both dysfunction and damage in the endothelial cell layer. In the latter case, the most plausible hypothesis is the inability of predisposed individuals to repair endothelial cell damage that occurs in normal circumstances, mainly due to a decrease in endothelial progenitor cells, primarily responsible for these repair processes. Regarding functional alterations, these consist of an imbalance

between the production of vasodilator and anti-inflammatory substances including nitric oxide and the production of pro-inflammatory and primarily endothelin vasoconstrictors. 4. *Structural changes in the arteries*. Three types of changes in arteries are described in hypertension: the presence of capillary rarefaction, hypertrophy of the middle layer of resistance arteries, and stiffness of large arteries [3–6].

## 3. Blood pressure monitoring (*random vs MAPA*)

The diagnosis of hypertension and the therapeutic decisions derived from this diagnosis require the highest reliability possible. The difficulties in measuring the blood pressure of a subject are derived from its variability, associated mainly with his/her physical and mental activity, limitations in the accuracy of an indirect measure (with the observer as a major source of inaccuracy), and sometimes the alert reaction produced in a person to blood pressure monitoring, which in some cases can be of clinical relevance (white-coat phenomenon).

Measuring blood pressure in a clinical setting: the reference technique of measuring blood pressure has been classically performed in the clinical setting by the auscultatory technique with the mercury sphygmomanometer, and most recently by semiautomatic and validated self-measurement devices. Monitoring ambulatory blood pressure provides real value of great importance. MAPA keeps 24-h Holter records of the blood pressure of a person. The patient may be classified with respect to the values of blood pressure in four exclusive conditions: real normotensive, sustained hypertension, white-coat hypertension, and masked hypertension. Because of its advantages, MAPA is increasingly considered a necessary element in the basic assessment of hypertensive patients, with the recommendation of some clinical guidelines to use it for confirmation of diagnosis, previous to making therapeutic decisions [3–6].

## 4. Morbidity of hypertension

The main clinical guidelines for the diagnosis and treatment of hypertension advise for the evaluation, to the possible extent, of the existence of subclinical organ damage. The reason for this is the consideration of organ damage as an intermediate stage in the development of cardiovascular disease, with importance in the stratification of absolute cardiovascular risk, and therefore the need for a different therapeutic approach. Multiple components of subclinical organ damage in heart, renal, and cerebral level have been described [3–6].

### 4.1. Main consequences

In the **heart**, hypertension causes left ventricular hypertrophy, which is the first step for developing hypertensive heart disease.

The silent damage produced in the **kidney** should be assessed in all hypertensive patients at diagnosis. Furthermore, at least annually, the evaluation should proceed by determining serum creatinine, renal blood flow, as well as the microalbuminuria.

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The evaluation of **vascular injury** is more complex and its relationship with prognosis is less evident. In particular, in regard to the modifications induced in the treatment, there is no universal recommendation for vascular assessment in hypertensive patients although there is evidence about the usefulness of the ankle arm index. The ankle arm index is an indicator of the existence of peripheral arterial disease and can be altered in apparently asymptomatic patients.

## 5. Drugs and pharmacology associated

The aim of an antihypertensive therapy is twofold. On one hand, reducing mortality and cardiovascular morbidity associated with increased pressure and, on the other, avoiding the progression and regression of subclinical visceral organ damage. Treatment should be initiated immediately in all patients with hypertension grade III (SBP of >180; DBP of >110), regardless of the presence of other conditions, and for any other hypertensive patients in the presence of diabetes, target organ damage, or chronic kidney disease established by cardiovascular disease. Although there is no clear consensus in the clinical guidelines, there is a general indication for starting treatment in elderly individuals at values above 150 mmHg 90 [3–6].

### 5.1. Diuretics

Diuretics are first-line drugs, and there is more evidence available of its protective capacity than with other drugs. There are three different subgroups of diuretics: thiazide, loop diuretics of Henle, and potassium-sparing, although only the former has clearly demonstrated a clear benefit in cardiovascular pressure in hypertensive patients.

### 5.2. Calcium antagonists

There are three main groups of calcium-channel blockers. Their mechanism of action consists of the inhibition of dependent calcium channels in the membrane potential blocking the entry of calcium into the cell. The biggest drawback of these drugs is the frequent occurrence of side effects. In nearly a third of these patients, side effects are derived from cutaneous vasodilation and are primarily manifested by ankle edema. Calcium antagonists constitute one of the best options for a combined therapy. They have beneficial and synergistic effects when combined with RAS blockers (angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs)) and in the case of dihydropyridines, with beta blockers. In both cases, an additional antihypertensive benefit is achieved. Furthermore, the metabolic side effects of diuretics are avoided.

### 5.3. Inhibitors of the converting enzyme of angiotensin

The mechanism of action consists of the inhibition of angiotensin II formation from angiotensin I. ACEIs also cause a decrease in aldosterone secretion induced by angiotensin II and inhibit the breakdown of bradykinin to increase levels of the mentioned vasodilator peptide. They are currently considered a first step for drug treatment in hypertension, and have demonstrated

their ability to prevent cardiovascular events in hypertensive patients. Combined therapies with thiazide and loop diuretics are especially effective as they prevent the formation of angiotensin II-induced activation of renin secretion. One of its biggest advantages is that they are effective and safe in associated diseases, prolong survival, and reduce complications of patients with infarction and ventricular dysfunction. Likewise, these are required drugs in the treatment of heart failure and chronic renal failure with proteinuria. Finally, they are especially indicated in hypertension associated to diabetes mellitus due to its ability to prevent micro- and macrovascular coronary disease and nephropathy progression of secondary complications. Side effects are mainly the appearance in some patients of refractory therapeutically nonproductive dry cough. There have also been reported isolated cases of angioedema, which appears with the first doses, probably reflecting a phenomenon of hypersensitivity.

### 5.4. Antagonists angiotensin II receptor

These drugs reduce the inhibition of the renin-angiotensin system by the specific antagonism of angiotensin I and II. The advantage compared to ACE inhibitors is that it is better tolerated. A unique feature is a major effect on stroke prevention.

## 6. Hypertension in different biological conditions

Treatment of hypertension in special situations deserves special consideration because of its high prevalence and its added cardiovascular risk [3–6]:

### 6.1. Elderly

In hypertensive patients over 80 years, treatment should start from equal or higher levels of 160/90 and with the goal of achieving values of 150/90. From this recommendation are excluded hypertensive patients with great fragility, high levels of dependence, or debilitating diseases in which treatment decisions should be individualized.

### 6.2. Diabetes mellitus

It is a situation of high cardiovascular risk. In addition, diabetic nephropathy is a serious health problem that can be the leading cause for a need of dialysis and kidney transplant. Prognosis of diabetic patients depends on many factors besides glycemic control, among which figures blood pressure. The basic objectives in this case are the reduction of blood pressure to levels below 140/90, and strict metabolic control of the glycosylated hemoglobin to levels lower than 7%, in addition to low-density lipoprotein (LDL) cholesterol levels below 100 mg. The drug treatment for diabetic patients should be based on a serotonin-releasing agent (SRA) blocking agent, especially because of its renal protective effect, although with most patients it will be necessary to use two or more associated drugs. In this case, the groups to combine would be calcium-channel blockers and diuretics.

### 6.3. Ischemic heart disease

The objective of the control in these cases is to achieve figures lower than 140/90. Regardless of age, beta-blockers are the drugs of choice in patients with hypertension and angina. Calcium antagonists are an alternative in case of contraindication or intolerance to beta-blockers. It has also been found that spironolactone associated with beta-blockers reduces mortality in heart failure. There are studies supporting the indication of ACE inhibitors or ARBs in all patients with ischemic heart disease.

### 6.4. Cerebrovascular disease

Primary prevention constitutes one of the main expressions of the effectiveness of antihypertensive treatment control. Objectives are set here at values below 140/90 mmHg. Stroke especially affects the elderly hypertensive and depends markedly on the figures of control of the blood pressure. In clinical studies, it has been observed that diuretics and calcium antagonists have a special preventive capacity.

### 6.5. Kidney disease

Hypertensive patients with albuminuria kidney disease or reduction of renal filtrate below 30 ml/min should be considered at high risk for injury in the target organ.

### 6.6. Resistant hypertension

It is defined as the inability to achieve the desirable blood pressure despite modifications in lifestyle, the use of a diuretic, and at least two other blood pressure medicines. The prevalence is between 10 and 15%. Diagnosis requires confirmation by ambulatory monitoring (MAPA), since even a third of these patients present a white-coat phenomenon. Many patients will need the administration of a fourth type of drug. A small group of patients maintains a high blood pressure despite complex therapeutic regimens often with up to six or seven drugs and by ruling out other causes responsible for resistance to treatment. Recently, some nonpharmacological therapeutic techniques have been developed that have been proved effective in reducing blood pressure in these uncontrolled patients, such as renal sympathetic denervation, stimulation of carotid baroreceptors, or implantation of an arteriovenous fistula.

## 7. Exercise for hypertension

Guidelines for the management of hypertension recommend lifestyle modifications, in particular, a combination of diet and exercise. Nonpharmacological strategies are aimed to change the lifestyle and should be instituted in hypertensive and pre-hypertensive patients. Supervised exercise, the DASH diet, and weight reduction, combined with pharmacological therapy, are effective treatments for normalization of blood pressure. This statement is based on the knowledge that exercise is a landmark in primary prevention and coadjuvant in the treatment of hypertension [7]. Exercise is considered as a polypill and, thus, is necessary to

know the optimal dosage (posology) in order to obtain maximal benefits [8]. Specifically, the response of blood pressure to exercise aims to ensure adequate blood flow to active muscles. BP depends on cardiac output (Q), which varies depending on myocardial contractility, heart rate (HR), blood volume, and peripheral vascular resistance. During the development of training, changes occur at the cardiovascular level, including increased systolic blood pressure, diastolic blood pressure, and mean arterial pressure (MAP) [9, 10].

The possible mechanisms of the antihypertensive effects of exercise are not fully clear but may include [7] (a) reduction in sympathetical-induced vasoconstriction and reduced catecholamine segregation; (b) increased insulin sensitivity; (c) anti-inflammatory effects; and (d) vascular structural adaptations.

Different international organizations such as the American Heart Association, American College of Cardiology, American College of Sports Medicine, European Society of Hypertension, European Society of Cardiology, or the Canadian Hypertension Education Program have developed their exercise prescription guidelines for adults with hypertension (for a review, see the original statements or Pescatello et al. [11]). Because the ideal dose is still unknown, the emergence of new modalities of exercise, in this chapter updated exercise strategies for managing and preventing hypertension, is presented. The modalities of exercise that have shown a relatively stronger supporting evidence are aerobic exercise (Class I, level of evidence A), dynamic resistance exercise (Class IIA, level of evidence B), and isometric handgrip (Class IIB, level of evidence C) [3].

### 7.1. Cardiovascular exercise

Low aerobic fitness has been proved to be a strong predictor for future cardiovascular disease and all-cause mortality in people with hypertension [12]. Not surprisingly therefore, there still seems to be a consensus that aerobic exercise should be prescribed as the primary type of exercise for the prevention, treatment, and control of hypertension [11]. Aerobic training can be performed either at continuous intensity or by short intervals of high-intensity exercise.

#### *7.1.1. Continuous aerobic*

The most extensive evidence with endurance training for hypertension is available for the continuous modality, understood as exercise of a constant sustainable intensity that is carried out over a given period of time, usually long.

##### *7.1.1.1. Benefits of continuous aerobic training for hypertension*

An acute session of continuous aerobic exercise can lower BP during the post-exercise period, known as the post-exercise hypotension (PEH). PEH is considered a prolonged decrease in resting blood pressure in the minutes and hours (up to 24 h) after an acute bout of exercise [13]. This decrease is more pronounced in hypertensive patients [14] and has also been shown in very old adults [15].

Chronic exercise results in more sustained reductions in BP, known as the exercise-training response [16]. According to different reviews and meta-analysis, aerobic exercise reduces BP

in 5–7 mmHg [17]. However, it should also be noted that there is a considerable heterogeneity of the individual response to exercise training, attributed to environmental and genetic factors. About 20–25% of hypertensive patients could be nonrespondents to exercise [18].

#### *7.1.1.2. Mechanisms of benefits*

PEH is mediated by both central and peripheral factors. The fall in blood pressure seen after exercise seems to be caused by a reduced signal transduction from sympathetic nerve activation into vasoconstriction, and also local vasodilatator mechanisms. Recent evidence suggests that muscle afferents may play a major role in this response [19].

Chronic aerobic exercise in hypertensive patients may lead to a reduction in left ventricular mass [16], although results are not always conclusive. Decreased oxidative stress and inflammation levels, as well as an improved autonomic function, have been described as key mechanisms that contribute to the physiological benefits obtained with exercise. However, further research is needed to elucidate the exact mechanisms involved in the adaptations to chronic aerobic exercise [16].

#### *7.1.1.3. Dose of exercise*

When it comes to prescribe physical activity, the dose-response question is a very relevant issue. It refers to the relationship between different doses of physical activity and changes in a defined health parameter. The total physiological load is calculated for continuous endurance training as the product of the intensity, duration, and frequency of the exercise. There is still an ongoing debate about the optimal training dose, frequency, duration, and intensity of exercise for hypertensive patients.

In regard to intensity, few studies have actually compared the effects of different intensities of endurance exercise in blood pressure. Cornelissen et al. [20], using a randomized crossover design, compared the effects of two different programs of endurance training in BP, HR, and heart rate variability (HRV) in a group of 36 healthy sedentary older adults (mean age: 59 years, range: 55–71 years) with SBP of ≥120 mmHg or DBP of ≥80 mmHg. The study was composed of three 10-week periods. In the first and third period, the participants exercised at 33 or 66% of HR reserve (3 days per week, 50 min per session) in random order, with a sedentary period in between. The results of this study showed that endurance training at both lower and higher intensities reduced SBP significantly ($P < 0.05$) at rest, before exercise, during exercise, and during recovery to a similar extent. The effect of training on HR was more pronounced ($P < 0.05$) with higher intensity, whereas HRV was unaffected by the intervention.

However, more recent evidence suggests that moderate to high intensities produce higher BP reductions than low-intensity endurance training (<40% of heart rate reserve or <55% of maximum heart rate) [17].

On the other hand, the most appropriate duration and frequency of the exercise programs is not fully clear either, but most evidence is in line with the American College on Sports Medicine's recommendations of performing aerobic exercise most, preferably all, days of the week. The duration of each exercise session should range from 30 to 60 min [11].

In parallel, Cornelissen and Smart [17] concluded, after conducting a systematic review and meta-analysis, that exercise programs longer than 210 min per week produced the smallest reductions in BP compared to shorter programs, but probably due to the lower intensity of longer programs.

*7.1.2. High-intensity interval training*

There is increasing evidence showing that high-intensity training (85–100% of peak oxygen uptake [$VO_{2peak}$]) in hypertensive patients may elicit higher CVD benefits, including lowering blood pressure, compared to low- and moderate-intensity training [20, 21]. High-intensity interval training (HITT) is the most common form of high-intensity training, and consists of short bouts of high-intensity exercise interspersed with resting periods of similar, lower, or longer duration.

*7.1.2.1. Benefits of HIIT for hypertension*

Repeated bouts of high-intensity interval exercise (>85 $VO_{2max}$) have been shown to induce similar PEH responses to those obtained with continuous modalities of endurance training performed at lower intensities (~60 to 65% $VO_{2max}$), both when matched for exercise volume [22], and when compared to lower volume but performed at maximal intensity [23].

A 12-week HIIT program (85–90% of $VO_{2max}$) lowered blood pressure, improved cardiac function and aerobic capacity, and reduced the mean HR, with superior effects than continuous aerobic training performed at a lower intensity (60% of $VO_{2max}$) [21].

*7.1.2.2. Mechanisms of benefits*

The mechanisms of the benefits elicited by HIIT programs are probably similar to the ones described in the continuous modality. However, higher intensities elicit exponentially greater increases in sympathetic activity, so it is possible that it may produce higher autonomic adaptations. It has also been suggested that the larger shear stress provided by high-intensity exercise compared to moderate intensity programs further enhances the endothelial function, which plays a major role in the regulation of vasomotor tone and the development of atherosclerosis, and it has also been shown to be a strong predictor of CV risk in hypertensive patients [21–25].

*7.1.2.3. Dose of exercisee*

When it comes to HIIT training, the most important variables to consider are the duration of the short bouts of high-intensity exercise as well as the duration of the resting periods. More research is needed for establishing the optimal ratio of exercise and rest in people with hypertension, with protocols ranging from 4 × 4-min interval at 85–90% of $VO_{2max}$ with a 3-min active pause between bouts [21] to two sets of 10 min composed of repeated phases of 15 s at 100% interspersed by 15 s of passive recovery, with another 4 min of passive recovery between sets [25].

In patients with coronary artery disease and heart failure, it has been suggested that short bouts of high-intensity exercise (100% of maximal aerobic power) such as 15 s for people with CAD and 30 s for people with HF (with passive recovery phases of the same duration between bouts, ratio 1:1) are optimal for obtaining benefits and improving adherence [26]. It is possible that these combinations are also adequate for people with hypertension as shown by Sosner et al. [25], but more research is needed in order to establish the most appropriate training regime.

### 7.2. Resistance exercise: dynamic resistance training

#### *7.2.1. Benefits of dynamic resistance training for hypertension*

Resistance exercise is increasingly recommended because studies have shown a reduced systolic blood pressure of 1.8 and 3.3 mmHg in diastolic blood pressure with this type of training [17].

#### *7.2.2. Mechanisms of benefits*

Initially, SBP and DBP rise because of the exercise pressor reflex to the cardiovascular center in the medulla from proprioceptors (mechanoreceptors and metaboreceptors) in active muscles. Arterial pressure rises to overcome the resistance to muscle perfusion caused by elevated intramuscular pressure interrupting arterial blood flow.

A hypotensive response has been reported several hours after exercise, caused by reduced norepinephrine levels and thus by the inhibition of sympathetic activity, a reduction in circulating angiotensin II, adenosine, and endothelin levels and their receptors in the central nervous system leading to decreased pulmonary vascular resistance (PVR), and increased baroreflex sensitivity. Hypotension is also triggered by the vasodilator effect of prostaglandins and nitric oxide.

#### *7.2.3. Dose of exercise*

As a general recommendation, Pescatello et al. [11] recommend to carry out a strength-training program of moderate intensity (i.e., 60–80% of 1 RM) consisting of eight to 10 exercises targeting the major muscle groups (performing multisets of 10–12 reps), and using different means of resistance training. This training should ideally be repeated 2–3 days per week.

Usually, it has been recommended to perform strength training dynamically, perhaps to avoid an increase in blood pressure. This effect is possibly due to a mechanical compression, causing increased peripheral resistance. Such resistance is related to blood viscosity and the length and radius of the vessel during muscle contraction in exercised muscle groups. This compression phenomenon that obstructs blood flow is associated with a reflex vasoconstriction in unexercised body areas that elevate BP. Moreover, when holding the breath while performing strength training (usually when the load is very high), an increase in the intrathoracic pressure caused by what is known as the Valsalva maneuver (MV) occurs [27]. Fleck et al. [28] observed that the SBP, DBP, and the double product (DP) increase during the last repetitions (one to three)

of the series leading to volitional and muscular failure, regardless of the intensity. It appears that both, the prolongation of effort and the degree of reduction in execution speed (increased muscle static component) as the last few repetitions approach, cause an increase in BP regardless of the intensity, especially when performing sets to failure. In the same way, Wilborn et al. [10] demonstrated that the BP is significantly higher after 85% of 1 RM than 65% of that 1 RM. They found no significant differences in the response of SBP between exercising at 65 and 85% of 1 RM. The results of this study suggest that reasons other than mechanical compression and Valsalva maneuver may be the cause of the increase in BP due to the fact that 1 RM caused minor responses in BP. The study concluded that the longer duration of exercise at 65% of 1 RM is most likely to cause the largest increase in SBP and HR at this intensity.

### 7.3. Resistance exercise: isometric resistance training

Over the past 5 years, emerging evidence supports the potential use of isometric training for hypertension. Nevertheless, the meta-analysis of Cornelissen and Smart [17] highlights that isometric exercise shows the greater reductions in systolic and diastolic blood pressure, 10.9 and 6.2 mmHg, respectively, compared to other types of training. In a recent meta-analysis, Inder et al. [29], using a meta-analysis, have shown that isometric exercise will be more efficient in reduction on hypertension in males of >45 years old, using arms exercises unilaterally over 8 weeks of training.

In regard to the optimal dose for this type of training, Millar et al. [30] have reported that effective interventions should be characterized by:

- 4 × 2 min (1–4 min rest) of 20–50% of the maximal voluntary contraction.
- 3–4 days/week during at least 4–10 weeks.
- Being able to be carried out by lower limb exercises (i.e., leg press or squat) or upper limb (i.e., handgrip).

Finally, it should be noted that there is emerging evidence that shows significant improvements in blood pressure after physical-training programs with vascular occlusion superimposed, although the mechanisms of action that could support these improvements are currently unknown and therefore it is very difficult to establish appropriate recommendations for this type of exercise [31].

Some aspects that should be considered in connection with the exercise in people affected by hypertension are as follows:

- Progression should be gradual, avoiding large increases, especially in the intensity variable.
- Do not begin an exercise program if the person presents values of arterial pressure of 180/105 mmHg or higher.
- Monitor blood pressure during (or at the end of a set) the exercise by the average of two consecutive measurements spaced by 30 s.
- Use a cooldown phase of at least 5–10 min, in order to avoid an excessive post-exercise hypotension effect.

- If the response to exercise is hypotensive, that is, an inability to increase blood pressure despite increased demands for physical exercise, usually less than 20–30 mmHg, exercise must stop. This phenomenon may reflect the need for adjustment of the antihypertensive treatment.
- Monitor the pressor effect of an exercise at a given intensity. This factor is calculated by multiplying the systolic blood pressure for heart rate registered after exercise to a specific intensity.
- Provide a sufficient recovery time, at least 30 s, to return to baseline conditions of the cardiovascular system.
- Avoid exercise in which the head level is below the hips (declined exercises).
- Avoid high number of repetitions and muscular failure in resistance training.

## 8. Case study

**Age:** 55 years **Sex:** Male

**BP:** 155/110 mmHg. **Medication:** Treated with diuretics for 3 months.

**Physical activity levels:** Sedentary.

**Body mass index (BMI):** 33 kg/m$^2$.

**Patient history:** No familiar antecedents of cardiovascular accident.

The proposed exercise would be summarized as follows:

| | | Aerobic exercise | Isometric exercise |
|---|---|---|---|
| Initial stage | Fisrt week | **Walking**<br>At least 3 days/week<br>Accumulated 3 set of 10 minutes<br>Low-moderate intensity (40-60 % HR reserve) | **Handgrip exercise**<br>2 days/week<br>2 sets x 1 minute (20-30% 1RM) |
| | Second week | **Walking**<br>At least 5 days/week<br>Acumulated 3 set of 10 minutes<br>Low-moderate intensity (40-60 % HR reserve) | **Handgrip exercise**<br>2 days/week<br>2 sets x 2 minute (20-30% 1RM)<br>**Squat**<br>2 days/week<br>2 sets x 1 minute (20-30% 1RM) |

| | | Aerobic exercise | Isometric exercise | Dynamic resistance training |
|---|---|---|---|---|
| Progression stage | Third week | **Walking or cycling**<br>2 days / week 30 minutes<br>Low-moderate intensity (40-60 % HR reserve)<br><br>3 days/week<br>Accumulated 3 set of 10 minutes<br>Low-moderate intensity (40-60 % HR reserve) | **Handgrip exercise**<br>3 days/week<br>2 sets x 1 minute (20-30% 1RM)<br><br>**Squat**<br>2 days/week<br>3 sets x 1 minute (20-30% 1RM) | **Core exercises**<br>1.Superman<br>2.Lateral bridge<br>3.Modified curl-up<br>2-3 sets x 10-20 seconds<br>**Upper limb exercises**<br>4.Chest Press<br>5.Row machine<br>**Lower limb exercises**<br>6.Hamstrings machine<br>7.Calves machine<br>8.Abductor machine<br>2-3 sets x 10 – 12 reps (15) (40-50% 1RM) |
| | Fith week | **Walking or cycling**<br>2 days / week 30 minutes<br>Low-moderate intensity (40-60 % HR reserve)<br><br>2 days / week 40 minutes<br>Low-moderate intensity (40-60 % HR reserve)<br><br>1 days/week<br>Accumulated 3 set of 10 minutes<br>Moderate-high intensity (60-80 % HR reserve) | **Handgrip exercise**<br>3 days/week<br>2 sets x 2 minute (20-30% 1RM)<br><br>**Squat**<br>2 days/week<br>4 sets x 1 minute (20-30% 1RM) | **Core exercises**<br>1.Superman<br>2Lateral bridge<br>3.Modified curl-up<br>2-3 sets x 20-30 seconds<br>**Upper limb exercises**<br>4.Chest Press<br>5.Row machine<br>**Lower limb exercises**<br>6.Leg press<br>7.Hamstrings machine<br>8.Calves machine<br>9.Abductor machine<br>2-3 sets x 10 – 12 reps (15) (50-60% 1RM) |

| | | Aerobic exercise | Isometric exercise | Dynamic resistance training |
|---|---|---|---|---|
| Progression stage | Sixth week | **Walking or cycling**<br>2 days / week 40 minutes<br>Low-moderate intensity (40-60 % HR reserve)<br>2 days / week 50 minutes<br>Low-moderate intensity (40-60 % HR reserve)<br>1 days/week<br>Acumulated 3 set of 10 minutes<br>Moderate-high intensity (60-80 % HR reserve) | **Handgrip exercise**<br>4 days/week<br>3 sets x 1 minute (20-30% 1RM)<br>**Squat**<br>2 days/week<br>3 sets x 2 minute (20-30% 1RM) | **Core exercises**<br>1.Superman<br>2Lateral bridge<br>3.Modified curl-up<br>2-3 sets x 30-40 seconds<br>**Upper limb exercises**<br>4.Chest Press<br>5.Row machine<br>6.Shoulder press<br>**Lower limb exercises**<br>7.Lunge<br>8.Hamstrings machine<br>9.Calves machine<br>10.Abductor machine<br>2-3 sets x 10 – 12 reps (15) (60-70% 1RM) |
| | Tenth week | **Walking or cycling or jogging or swimming**<br>2 days / week 50 minutes<br>Low-moderate intensity (40-60 % HR reserve)<br>2 days / week 60 minutes<br>Low-moderate intensity (40-60 % HR reserve)<br>1 days/week<br>10-30 minutes<br>High Intensity Exercise<br>High intensity (>80 % HR reserve) | **Handgrip exercise**<br>4 days/week<br>4 sets x 1 minute (20-30% 1RM)<br>**Squat**<br>2 days/week<br>4 sets x 2 minute (20-30% 1RM) | **Core exercises**<br>1.Superman<br>2.Lateral bridge<br>3.Modified curl-up<br>2-3 sets x 40-50 seconds<br>**Upper limb exercises**<br>4.Chest Press<br>5.Row machine<br>6.Shoulder press<br>7.Triceps machine<br>**Lower limb exercises**<br>8.Lunge<br>9.Hamstrings machine<br>10.Calves machine<br>2-3 sets x 10 – 12 reps (15) (60-80% 1RM) |

| | | Aerobic exercise | Isometric exercise | Dynamic resistance training |
|---|---|---|---|---|
| Maintain stage | >Eleventh week | **Walking or cycling or jogging or swimming**<br>At least 4 days / week 60 minutes Low-moderate intensity (40-60 % HR reserve)<br>1 days/week Acumulated 3 set of 10 minutes Moderate-high intensity (60-80 % HR reserve) | **Handgrip exercise**<br>4 days/week<br>4 sets x 2 minute<br>(20-30% 1RM)<br>**Squat**<br>2 days/week<br>4 sets x 2 minute<br>(20-50% 1RM) | **Core exercises**<br>1.Superman<br>2.Lateral bridge<br>3.Modified curl-up<br>2-3 sets x 50-60 seconds<br>**Upper limb exercises**<br>4.Chest Press<br>5.Row machine<br>6.Shoulder press<br>7.Triceps machine<br>8.Biceps machine<br>**Lower limb exercises**<br>9.Lunge<br>10.Deadlift (without Valsalva maneuver)<br>2-3 sets x 10 – 12 reps (15)<br>(60-80% 1RM) |

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