Exercise Therapy in Hypertensive Cardiovascular Disease

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Abstract

Hypertension is rare in the young, but its prevalence increases with age. Exercise contributes to the prevention of hypertension in normotensive subjects and to the control of blood pressure in hypertensive patients. The overall cardiovascular risk of the hypertensive patient does depend not only on blood pressure but also on the presence of other risk factors, target organ damage, and associated clinical conditions. The recommendations for preparticipation screening, sports participation, and follow-up depend on the overall risk profile of the individual patient. When antihypertensive treatment is required in addition to nonpharmacologic measures, calcium-channel blockers and blockers of the renin-angiotensin system are currently the drugs of choice for the patient who exercises. (Prog Cardiovasc Dis 2011;53:404-411) © 2011 Elsevier Inc. All rights reserved.

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Definition of hypertension and epidemiology

The diagnosis of hypertension (HTN) is based on multiple conventional blood pressure (BP) measurements, taken on separate occasions, in the sitting position, by use of a mercury sphygmomanometer or another calibrated device and is universally defined as a systolic BP of 140 mm Hg or higher, a diastolic BP of 90 mm Hg or higher, or both1-3 (Table 1). However, approximately 25% of patients with HTN by conventional measurements have a normal BP out of the office on 24-hour ambulatory monitoring or on home BP measurements—so-called white-coat or isolated clinic HTN.4,5 Young athletes with clinic HTN often have normal BP on ambulatory monitoring.6 On the other hand, patients may have masked or isolated ambulatory HTN, which is characterized by a normal BP in the office and an elevated BP out of the office.7 Twenty-four–hour ambulatory BP monitoring should be considered in cases of suspected white-coat HTN, considerable variability of office BP, marked discrepancy between BP measured in the office and at home, and in subjects with high office BP and low global cardiovascular (CV) risk.2 The threshold for the definition of HTN is 130/80 mm Hg for the average 24-hour BP. The threshold for daytime ambulatory BP and the self-measured BP at home is 135/85 mm Hg, whereas the cutoff is 120/70 mm Hg for nighttime BP.8

Systolic BP continues to increase throughout adult life, related to progressive arterial stiffening, whereas diastolic BP plateaus in the sixth decade of life and decreases thereafter, which explains the high prevalence of isolated systolic HTN in the elderly, defined as systolic BP 140 mm Hg or higher and diastolic BP less than 90 mm Hg.1,3 Typically, systolic BP is lower in women than in men younger than approximately 50 years, rises more steeply in women around menopause, and becomes higher in women than in men thereafter.

The prevalence of HTN in the population, defined as systolic BP 140 mm Hg or higher and/or diastolic BP 90 mm Hg or higher or being on antihypertensive treatment, is approximately 25%. When broken down by age and sex, the prevalence is approximately 15%, 30%, and 55% in men aged 18 to 39 years, 40 to 59 years, and 60 years or older, respectively, and about 5%, 30%, and 65% in

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HTN as a CV risk factor

Hypertension is associated with an increased incidence of all-cause and CV mortality, sudden death, stroke, coronary artery disease, heart failure, atrial fibrillation, peripheral arterial disease, and renal insufficiency. In the population at large, the relationship between CV complications and usual BP is linear on a logarithmic scale. In those older than 50 years, systolic BP is a more important risk factor than diastolic BP. The prognosis of white-coat HTN is better than that of sustained ambulatory HTN, and studies suggest that it is even similar to that of persons with true normal BP, whereas patients with masked HTN appear to have a worse outcome than true normotensives.

Despite conclusive evidence that antihypertensive therapy reduces the complications of HTN, only about half of all patients with HTN are under treatment; and only a fraction of these have normal BP. Systolic BP appears to be more difficult to control than diastolic BP, particularly in older patients.

### Etiology of HTN

Approximately 95% of hypertensive patients have essential or primary HTN, which is a multifactorial disease, resulting from an interaction between genetic and lifestyle/environmental factors including being overweight, high salt intake, excessive alcohol consumption, and physical inactivity. There is indeed evidence from cross-sectional epidemiological studies that physical inactivity is involved in the pathogenesis of HTN, and longitudinal observational studies found that physical activity (PA) or fitness are inversely related to the later development of HTN. Therefore, PA plays an important role in the prevention and control of high BP. Many studies have analyzed the relationship between PA and BP. In some studies, physical fitness was estimated from an exercise test; other studies used questionnaires and sometimes an interview relating to the subject’s PA at work, during leisure time, or both. It is accepted by most authors that the estimated PA by questionnaire and interview is a poor but, nevertheless, useful and possibly the best available tool. Furthermore, the relationships between results of physical fitness tests and PA pattern by interview or questionnaire are of a low order. In general, therefore, the methodology to estimate PA lacks accuracy. In addition, there are several confounding variables that might affect the relationship between PA and BP. Some of these such as age, weight, or indices of body fatness can be accounted for in the analysis. Others such as self-selection are hardly controllable. Indeed, “fit” subjects may have lower BP and choose a more active lifestyle, whereas subjects with higher BP may suffer from CV diseases (CVD), resulting in a lower degree of PA. Another problem is that the level of PA is low in most Western societies, which may hamper the finding of an association with BP. Although several studies did not observe significant independent relationships, others reported inverse relationships between BP and either habitual PA or physical fitness. The difference in BP between the most and the least in PA or fitness usually amounted to no more than about 5 mm Hg.

The main causes of secondary HTN involve renovascular, renal, and adrenal abnormalities. The role of ergogenic aids, which may increase BP, should be considered in the hypertensive sportsman or athlete. Athletes may be taking large doses of prohibited substances such as anabolic steroids, erythropoietin, stimulants, and so forth. The uncontrolled use of these agents has been associated with numerous side effects including HTN. In addition, the use of nonsteroidal anti-inflammatory drugs should be specifically considered.

### Table 1

Definitions and classification of clinic BP levels (mm Hg)

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Normal*</td>
<td>120-129</td>
<td>80-84</td>
</tr>
<tr>
<td>High normal*</td>
<td>130-139</td>
<td>85-89</td>
</tr>
<tr>
<td>Grade 1 HTN</td>
<td>140-159</td>
<td>90-99</td>
</tr>
<tr>
<td>Grade 2 HTN</td>
<td>160-179</td>
<td>100-109</td>
</tr>
<tr>
<td>Grade 3 HTN</td>
<td>≥180</td>
<td>≥110</td>
</tr>
<tr>
<td>Isolated systolic HTN</td>
<td>≥140</td>
<td>&lt;90</td>
</tr>
</tbody>
</table>

According to the recommendations of the European Society of Hypertension and the European Society of Cardiology.

* In the Joint National Committee 7 report, normal BP and high normal BP have been merged into the single category of pre-HTN; and grade 3 HTN is included in the category of grade 2 HTN.
because these compounds may increase BP and are commonly used in the athletic setting.\textsuperscript{18}

**Assessment of the severity of HTN and risk stratification**

The severity of HTN does depend not only on the BP level (Table 1) but also on the presence of other CV risk factors, organ damage, and CV and renal complications. Table 2 summarizes the classification based on the overall CV risk, as proposed by the European societies of HTN and cardiology.\textsuperscript{2} The terms low, moderate, high, and very high added risk, in comparison with healthy normotensives without risk factors, are calibrated to indicate an approximate absolute 10-year risk of CVD of less than 15\%, 15\% to 20\%, 20\% to 30\%, and greater than 30\%, respectively, according to the Framingham criteria or an approximate absolute risk of fatal CVD less than 4\%, 4\% to 5\%, 6\% to 8\%, and greater than 8\% according to the European systematic coronary risk evaluation (SCORE) system.\textsuperscript{19}

The risk stratification is based on the accumulated number of selected risk factors, on the presence of target organ damage, and/or on the presence of CV or renal disease, as outlined in Table 2. With regard to left ventricular hypertrophy (LVH), it should be noted that sports activity itself may induce LVH; the type of LVH and assessment of diastolic left ventricular function, speckle-tracking echocardiography, tissue Doppler imaging, and strain rate measurements may help to distinguish between hypertensive heart disease and athlete’s heart.\textsuperscript{20-25} Athlete’s heart typically shows maintained diastolic function and is, in general, considered a physiologic adaptation to training, in contrast to the LVH secondary to HTN. Hypertensive patients usually have concentric LVH, but eccentric LVH has also been described.\textsuperscript{26} Whether HTN in an athlete will trigger or accentuate the cardiac hypertrophy or athletic exercise in a person with LVH secondary to HTN will worsen the LVH is not known.

The importance of the risk stratification is that hypertensive patients at high or very high added risk should be treated promptly with antihypertensive drugs, whereas patients at low or moderate added risk are only treated when HTN persists despite lifestyle measures.

**Assessment of the risk associated with exercise**

Exercise-related sudden death at a younger age is mainly attributed to hypertrophic cardiomyopathy, anomalies of the coronary arteries, or arrhythmogenic right ventricular dysplasia\textsuperscript{23,27-29} and is unlikely to be related to HTN. On the other hand, coronary heart disease has been identified in approximately 75\% of victims of exercise-related sudden death above the age of 35 years.\textsuperscript{30} Whether elevated BP is a cause of exercise-related sudden death on its own is not known, but HTN is certainly a major risk factor for the development of coronary artery disease. In addition, HTN-induced LVH may cause life-threatening...

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**Table 2**

<table>
<thead>
<tr>
<th>Blood pressure (mm Hg)</th>
<th>Other Risk Factors, OD, or Disease</th>
<th>Normal (SBP 120-129 and/or DBP 80-84)</th>
<th>High Normal (SBP 130-139 and/or DBP 85-89)</th>
<th>Grade 1 HTN (SBP 140-159 and/or DBP 90-99)</th>
<th>Grade 2 HTN (SBP 160-179 and/or DBP 100-109)</th>
<th>Grade 3 HTN (SBP 180-199 and/or DBP 120-110)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No other risk factors*</td>
<td>Average risk\textsuperscript{1}</td>
<td>Average risk\textsuperscript{1}</td>
<td>Average risk\textsuperscript{1}</td>
<td>Low added risk\textsuperscript{2}</td>
<td>Moderate added risk\textsuperscript{2}</td>
<td>High added risk\textsuperscript{2}</td>
</tr>
<tr>
<td>1-2 risk factors*</td>
<td>Low added risk\textsuperscript{2}</td>
<td>Low added risk\textsuperscript{2}</td>
<td>Low added risk\textsuperscript{2}</td>
<td>Moderate added risk\textsuperscript{2}</td>
<td>Moderate added risk\textsuperscript{2}</td>
<td>High added risk\textsuperscript{2}</td>
</tr>
<tr>
<td>≥3 risk factors,* OD,\textsuperscript{1} MS, or diabetes</td>
<td>Moderate added risk\textsuperscript{2}</td>
<td>High added risk\textsuperscript{2}</td>
<td>High added risk\textsuperscript{2}</td>
<td>High added risk\textsuperscript{2}</td>
<td>Very high added risk\textsuperscript{2}</td>
<td>Very high added risk\textsuperscript{2}</td>
</tr>
<tr>
<td>Established CV or renal disease\textsuperscript{3}</td>
<td>Very high added risk\textsuperscript{2}</td>
<td>Very high added risk\textsuperscript{2}</td>
<td>Very high added risk\textsuperscript{2}</td>
<td>Very high added risk\textsuperscript{2}</td>
<td>Very high added risk\textsuperscript{2}</td>
<td>Very high added risk\textsuperscript{2}</td>
</tr>
</tbody>
</table>

*Risk factors used for stratification: level of BP and pulse pressure, sex and age (men >55 years and women >65 years), smoking, dyslipidemia (total cholesterol >190 mg/dL, LDL cholesterol >155 mg/dL or HDL cholesterol <40 mg/dL in men and <46 mg/dL in women, or triglycerides >150 mg/dL), fasting plasma glucose 100 to 125 mg/dL, abnormal glucose tolerance test, abdominal obesity (waist circumference: men ≥102 cm and women ≥88 cm), first-degree family history of premature CVD (men <55 years and women <65 years).

The terms low, moderate, high, and very high added risk, in comparison with healthy normotensives without risk factors (average risk), are calibrated to indicate an approximate absolute 10-year risk of CVD of less than 15\%, 15\% to 20\%, 20\% to 30\%, and greater than 30\%, respectively, according to the Framingham criteria, or an approximate absolute risk of fatal CVD less than 4\%, 4\% to 5\%, 6\% to 8\%, and greater than 8\% according to the European systematic coronary risk evaluation (SCORE) system.

Organ damage: HTN-induced LVH, ultrasound evidence of carotid wall thickening or atherosclerotic plaque, slight increase in plasma creatinine (men, 1.3-1.5 mg/dL; women, 1.2-1.4 mg/dL), estimated glomerular filtration rate (<60 mL/min per 1.73 m²), presence of microalbuminuria, carotid-femoral pulse wave velocity greater than 12 meters per second, and ankle/brachial BP index less than 0.9.

Includes cerebrovascular disease (stroke and transient ischemic attack), ischemic heart disease (myocardial infarction and angina), heart failure, peripheral vascular disease, renal disease (diabetic nephropathy, renal impairment, and proteinuria), and advanced retinopathy (hemorrhages, exudates, and papilloedema).

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*Abbreviations: SBP indicates systolic BP; DBP, diastolic BP; OD, organ damage; MS, metabolic syndrome.*
ventricular arrhythmias. It is likely that the risk associated with exercise can be derived from the overall risk stratification (Table 2). Therefore, the general approach of the hypertensive patient should also apply to the exercising patient.

**Diagnostic evaluation**

Diagnostic procedures are aimed at (1) establishing BP levels; (2) identifying secondary causes of HTN; (3) evaluating the overall CV risk by searching for other risk factors, target organ damage, and concomitant diseases or accompanying clinical conditions. Diagnostic procedures comprise a thorough individual and family history; physical examination, including repeated BP measurements according to established recommendations; and laboratory and instrumental investigations, of which some should be considered part of the routine approach in all subjects with high BP, some are recommended, and some are indicated only when suggested by the core examinations.

Routine tests for all hypertensive patients include the following: hemoglobin level and hematocrit; serum potassium, creatinine, and uric acid; estimated glomerular filtration rate; fasting plasma glucose; serum total, high-density lipoprotein (HDL), and low-density lipoprotein (LDL) cholesterol and triglycerides; urine analysis complemented by microalbuminuria dipstick test and sediment examination; and standard electrocardiography. Recommended tests include the following: echocardiography, carotid (or femoral) ultrasound, pulse wave velocity measurement, ankle-brachial BP ratio index, fundoscopy, quantitative proteinuria (if dipstick test positive), postprandial plasma glucose or glucose tolerance test (if fasting plasma glucose >100 mg/dL), and home and 24-hour BP monitoring. Extended evaluation may be necessary based on the findings from these investigations. The indication for exercise testing depends on the patient’s risk and on the sports characteristics (Table 3). In patients with HTN about to engage in intense or very intense dynamic and/or static exercise (intensity ≥60% of their maximum exercise capacity), a medically supervised peak or symptom-limited exercise test with electrocardiography and BP monitoring is warranted. In asymptomatic men or women with low or moderate added risk (Table 2), who engage in low-to-moderate PA (intensity <60% of maximum), there is generally no need for further testing beyond the routine evaluation. Asymptomatic individual patients with high or very high added risk may benefit from exercise testing before engaging in moderate-intensity exercise (40%-60% of maximum) but not for light or very light activity (<40% of maximum). Patients with exertional dyspnea, chest discomfort, or palpitations need further examination, which includes exercise testing, echocardiography, Holter monitoring, or combinations thereof. Echocardiography and exercise testing with electrocardiography and BP monitoring are considered routine tests in the competitive athlete with HTN.

A major problem with exercise testing in a population with low probability of coronary heart disease and in subjects with LVH is that most positive tests on electrocardiography are falsely positive. Stress myocardial scintigraphy or echocardiography and, ultimately, coronary angiography may be indicated in cases of doubt. There is currently insufficient evidence that the BP response to exercise should play a role in the recommendations for exercise in addition to BP at rest. However, subjects with an excessive rise of BP during exercise are more prone to develop HTN and should be followed up more closely. There is currently no generally accepted limit to stop an exercise test for high BP, but it is considered prudent to stop the test when systolic BP reaches approximately 240 mm Hg. Finally, physicians should be aware that elevated BP may impair exercise tolerance.

**Effects of exercise on BP**

**Dynamic exercise**

Systolic BP increases during acute dynamic exercise in proportion to the intensity of the effort. During longer term stable exercise, the BP tends to decrease after an initial increase of short duration. The increase is greater for systolic than for diastolic BP, which only slightly increases or even remains unchanged. For the same oxygen consumption, the rise is more pronounced in older subjects and when exercise is performed with smaller than with larger muscle groups. Acute exercise is usually followed by postexercise hypotension, which may last for several hours and is generally more pronounced and of longer duration in patients with HTN than in normotensive subjects.

Cross-sectional and longitudinal epidemiological studies indicate that physical inactivity and low fitness levels are associated with (a) higher BP levels and (b) increased

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**Table 3**

<table>
<thead>
<tr>
<th>Demands of Exercise*</th>
<th>Risk Category</th>
<th>Indication for exercise testing: yes or no</th>
</tr>
</thead>
<tbody>
<tr>
<td>Static and/or Dynamic</td>
<td>Low or Moderate</td>
<td>High or Very High†</td>
</tr>
<tr>
<td>Light (&lt;40% of maximum)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Moderate (40%-59% of maximum)</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>High (≥60% of maximum)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

* Demands of exercise refer to percentage of maximal exercise capacity or heart rate reserve.
† In case of an associated clinical condition, the recommendations for the specific condition should be followed.
incidence of HTN in the population. Meta-analyses of randomized controlled intervention studies concluded that regular dynamic endurance training at moderate intensity significantly reduces BP. A recent meta-analysis involved 72 trials and 105 study groups. After weighting for the number of participants, training induced significant net reductions of resting and daytime ambulatory BP of 3.0/2.4 and 3.3/3.5 mm Hg, respectively. The reduction of resting BP was more pronounced in the 30 hypertensive study groups (−6.9/−4.9) than in the others (−1.9/−1.6). Systemic vascular resistance decreased by 17%, plasma norepinephrine by 29%, and plasma renin activity by 20%. Body weight decreased by 1.2 kg, waist circumference by 2.8 cm, percent body fat by 1.4%, and the Homeostasis Model Assessment index of insulin resistance by 0.31 U; HDL cholesterol increased by 0.032 mmol·L⁻¹. Therefore, aerobic endurance training decreases BP through a reduction of vascular resistance, in which the sympathetic nervous system and the renin-angiotensin system appear to be involved, and favorably affects concomitant CV risk factors.

However, the optimal characteristics of the training program are still a matter of debate, particularly with regard to the intensity of exercise. We addressed this question, first, by analyzing relationships between exercise characteristics and BP response across randomized controlled trials by use of meta-analytic techniques; second, by examining the results from studies in which different training regimens have been applied; and, third, by performing a randomized cross-over trial in which participants trained at both lower and higher intensity.

In the meta-analysis of randomized controlled trials, interstudy differences in the changes in BP were not related to weekly frequency, time per session, or exercise intensity, which ranged from approximately 45% to 85%; these 3 characteristics combined explained less than 5% of the variance of the BP response. In studies that randomized participants to training programs with different intensities, the response of diastolic BP was not different according to training intensity. Some studies reported a greater reduction of systolic BP when intensity was about 40% than when participants exercised at about 70% of maximal exercise capacity, but this finding was not consistent, neither within nor between studies. In addition, it is of note that the training programs of several of these studies differed not only in intensity but also in other characteristics. The duration of the training sessions was often shorter in the high-intensity programs; the lighter exercise programs could be home-based and unsupervised, whereas the higher intensity exercises were usually supervised and performed in a specialized center. Finally, the type of exercises could differ among the groups.

Because it was not well known what exercise intensity is needed to obtain BP reductions in response to endurance training, Cornelissen et al compared the effect of training at lower and higher intensity on BP and, in addition, on other CV risk factors in 55-year-or-older sedentary men and women by use of a randomized cross-over design comprising 3 10-week periods. In the first and third period, participants exercised at, respectively, lower or higher intensity (33% or 66% of heart rate reserve) in random order, with a sedentary period in between. Training programs that comprised walking, jogging, cycling, and stepping were identical except for intensity and were performed 3 times 1 hour per week. Thirty-nine (18 men) of 48 randomized participants completed the study; age averaged 59 years. We observed that the change of aerobic power from baseline to the end of each period was more pronounced (P < .05) with higher intensity (+3.70 mL·kg⁻¹·min⁻¹; P < .001) than with lower intensity training (+2.31 mL·kg⁻¹·min⁻¹; P < .001). Systolic BP at rest and during submaximal exercise were reduced with both intensities (P < .01) by about 5 to 6 mm Hg, without significant differences in BP reduction between intensities. Only higher intensity training reduced weight (−1.09 kg; P < .001), body fat (−0.85%; P < .001), plasma triglycerides (−0.17 mmol/L; P < .05), and oxidized LDL (−5.92 U/L; P < .01). We concluded that, in 55-year-or-older sedentary men and women, endurance training for 3 times 1 hour per week at lower intensity increases fitness levels but to a lesser extent than does higher intensity training; that lower and higher intensity training reduce office and exercise systolic BP to a similar extent; and that only higher intensity training favorably affects body mass, body composition, and some blood lipids.

**Static exercise**

During acute static exercise, BP increases, which is more pronounced than with dynamic exercise, particularly with heavy static exercise at an intensity of greater than 40% to 50% of maximal voluntary contraction. In a meta-analysis of randomized controlled trials, “resistance” training at moderate intensity was found to reduce BP by 3.5/3.2 mm Hg. The meta-analysis included 9 studies designed to increase muscular strength, power, and/or endurance; and all but 1 study involved dynamic rather than purely static exercise. In fact, few sports are characterized by purely static efforts. However, only 3 trials in the meta-analysis reported on patients with HTN. In the mean time, the number of studies has substantially increased; and the BP-lowering effect of resistance training has recently been confirmed in a meta-analysis of 26 randomized controlled trials.

**Recommendations**

**General recommendations**

Physically active patients and athletes with HTN should be treated according to the general guidelines for the
management of HTN.1-3 Appropriate nonpharmacologic measures should be considered in all patients; these include moderate salt restriction, increase in fruit and vegetable intake, decrease in saturated and total fat intake, limitation of alcohol consumption to no more than 20- to 30-g ethanol per day for men and no more than 10- to 20-g ethanol per day for women, smoking cessation, and control of body weight. Antihypertensive drug therapy should be started promptly in patients at high or very high added risk for CV complications (Table 2). In patients at low or moderate added risk, drug treatment is only initiated when HTN would persist after several months or weeks despite appropriate lifestyle changes. The goal of antihypertensive therapy is to reduce BP to at least below 140/90 mm Hg and to lower values if tolerated in all hypertensive patients and to below 130/80 mm Hg in diabetic patients and other high or very high risk conditions, although the latter lower threshold has recently been disputed because of lack of hard evidence.43

Current evidence indicates that patients with white-coat HTN do not have to be treated with antihypertensive drugs, unless they would be at high or very high risk (Table 2); but regular follow-up and nonpharmacologic measures are recommended.1,2,13 Furthermore, subjects with normal BP at rest and exaggerated BP response to exercise should be followed up more closely.32,33

Choice of drugs

Several drug classes can be considered for the initiation of antihypertensive therapy: diuretics; β-blockers (BBs), calcium-channel blockers, angiotensin-converting enzyme inhibitors, and angiotensin II receptor blockers.1,2 However, diuretics and BBs are not recommended for first-line treatment in patients engaged in competitive or high-intensity endurance exercise.36,44,45 Diuretics impair exercise performance and capacity in the first weeks of treatment through a reduction in plasma volume, but exercise tolerance appears to be restored during longer term treatment; however, diuretics may cause electrolyte and fluid disturbances, effects that are not desirable in the endurance athlete. On the other hand, BBs reduce maximal aerobic power by on average 7% because of the reduction in maximal heart rate, which is not fully compensated by increases of maximal stroke volume, peripheral oxygen extraction, or both. Furthermore, the time that submaximal exercise can be sustained is reduced by approximately 20% by cardioselective BBs and by approximately 40% by nonselective BBs, most likely because of impaired lipolysis.36,46,47 There are indications that the BB nebivolol may not impair exercise performance.48 In addition, diuretics and BBs are on the doping list for some sports, in which weight loss or control of tremor is of paramount importance. Diuretics are also banned because they may be used to conceal the use of other doping agents, such as anabolic steroids, by diluting the urine samples. The hypertensive athlete who has to use a diuretic and/or a BB for therapeutic purposes should follow the “International Standard for Therapeutic Use Exceptions” of the World Anti-Doping Agency.18,45

Calcium-channel blockers and blockers of the renin-angiotensin system are currently the drugs of choice for the hypertensive endurance athlete46,49 and may be combined in case of insufficient BP control. However, the combination of an angiotensin-converting enzyme inhibitor and an angiotensin II receptor blocker is currently not advocated for the treatment of HTN because of lack of proof of benefit of the combination for BP control, and the combination may even cause harm.50 If a third drug is required, a low-dose, thiazide-like diuretic, possibly in combination with a potassium sparing agent, is recommended. There is no unequivocal evidence that antihypertensive agents would impair performance in resistance sports.

Table 4

Recommended for strenuous leisure-time PA and competitive sports participation in athletes with systemic HTN (and other risk factors) according to the CV risk profile

<table>
<thead>
<tr>
<th>Risk Category*</th>
<th>Evaluation</th>
<th>Criteria for Eligibility</th>
<th>Recommendations</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low added risk</td>
<td>History, PE, ECG, ET, Echo</td>
<td>Well-controlled BP</td>
<td>All sports</td>
<td>Yearly</td>
</tr>
<tr>
<td>Moderate added risk</td>
<td>History, PE, ECG, ET, Echo</td>
<td>Well-controlled BP and risk factors</td>
<td>All sports, with exclusion of high-static, high-dynamic sports (III C)</td>
<td>Yearly</td>
</tr>
<tr>
<td>High added risk</td>
<td>History, PE, ECG, ET, Echo</td>
<td>Well-controlled BP and risk factors</td>
<td>All sports, with exclusion of high static sports (III A-C)</td>
<td>Yearly</td>
</tr>
<tr>
<td>Very high added risk</td>
<td>History, PE, ECG, ET, Echo</td>
<td>Well-controlled BP and risk factors; no associated clinical conditions</td>
<td>Only low-moderate dynamic, low-static sports (I A-B)</td>
<td>6 mo</td>
</tr>
</tbody>
</table>

Abbreviations: PE indicates physical examination, including repeated blood pressure measurements according to guidelines; ECG, 12-lead electrocardiography; ET, exercise testing; Echo, echocardiography at rest.

* The terms low, moderate, high, and very high added risk, in comparison with healthy normotensives without risk factors, are calibrated to indicate an approximate absolute 10-year risk of CVD of less than 15%, 15% to 20%, 20% to 30%, and greater than 30%, respectively, according to the Framingham criteria, or an approximate absolute risk of fatal CVD less than 4%, 4% to 5%, 6% to 8%, and greater than 8% according to the European SCORE system.
Finally, it is of note that high-risk conditions may be compelling indications for the use of specific antihypertensive drug classes.  

**Recommendations for sports participation**

There is general agreement that exercise is a cornerstone therapy for the prevention, treatment, and control of HTN. Based upon current evidence, the following exercise prescription is recommended: frequency, on at least 5 and preferably all days of the week; intensity, moderate intensity; time, at least 30 minutes of PA per day; and type, primarily endurance PA, supplemented by resistance exercise.  

The exercising hypertensive patients should be assessed and treated according to the general guidelines for the management of HTN. In addition to the generally recommended tests, the indication for exercise testing depends on the patient’s risk and on the sports characteristics (Table 3).

Recommendations for participation in competitive sports in athletes with HTN are based on the results of the evaluation and on the risk stratification (Table 2) and with the understanding that the general recommendations for the management of HTN are observed, as described above and provided that the clinical condition is stable. Table 4 summarizes the recommendations with regard to competitive sports participation. The same recommendations may apply to patients who aim to engage in hard or very hard leisure-time sports activities to substantially enhance performance. However, most recreational physical activities are performed at low-to-moderate intensity. Dynamic sports activities are to be preferred, but also low-to-moderate resistance training is not harmful and may even contribute to BP control. In case of CV or renal complications, the recommendations are based on the type and severity of the associated clinical condition.

Finally, all exercising patients should be advised on exercise-related warning symptoms, such as chest pain or discomfort, abnormal dyspnea, dizziness, or malaise, which would necessitate consulting a qualified physician, and should be followed up at regular intervals, depending on the severity of HTN and the category of risk.

**Statement of Conflict of Interest**

The author declares that there is no conflict of interest.

**References**


