

Review

Effect of exercise on blood pressure control in hypertensive patients

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Several large epidemiological studies have reported an inverse relationship between blood pressure and physical activity. However, longitudinal intervention studies are more appropriate for assessing the effects of physical activity. We performed meta-analyses of randomized controlled trials involving dynamic aerobic endurance training or resistance training. The meta-analysis on endurance training involved 72 trials and 105 study groups. After weighting for the number of trained participants, training induced significant net reductions in resting and daytime ambulatory blood pressure of, respectively, 3.0/2.4 mmHg ($P < 0.001$) and 3.3/3.5 mmHg ($P < 0.01$). The reduction in resting blood pressure was more pronounced in the 30 hypertensive study groups ($-6.9/-4.9$) than in the others ($-1.9/-1.6$; $P < 0.001$ for all). Systemic vascular resistance decreased by 7.1% ($P < 0.05$), plasma norepinephrine by 29% ($P < 0.001$), and plasma renin activity by 20% ($P < 0.05$). Body weight decreased by 1.2 kg ($P < 0.001$), waist circumference by 2.8 cm ($P < 0.001$), percentage body fat by 1.4% ($P < 0.001$) and the homeostasis model assessment index of insulin resistance by 0.31 units ($P < 0.01$); high-density lipoprotein cholesterol increased by 0.032 mmol/l ($P < 0.05$). Resistance training has been less well studied. A meta-analysis of nine randomized controlled trials (12 study groups) on mostly dynamic resistance training revealed a weighted net reduction in blood pressure of 3.2 ($P=0.10$)/3.5 ($P < 0.01$) mmHg associated with exercise. Endurance training decreases blood pressure through a reduction in systemic vascular resistance, in which the sympathetic nervous system and the renin-angiotensin system appear to be involved, and favourably affects concomitant cardiovascular risk factors. The few available data suggest that resistance training can reduce blood pressure. Exercise is a cornerstone therapy for the prevention, treatment and control of hypertension. *Eur J Cardiovasc Prev Rehabil* 14:12-17 © 2007 The European Society of Cardiology

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Introduction

There is overwhelming evidence from epidemiological prospective follow-up studies indicating that physical activity, assessed by questionnaire or interview, and physical fitness, measured on exercise testing, are inversely related to the incidence of cardiovascular disease and mortality [1-3]. The benefits of moderately vigorous activity [4] and of greater fitness [5,6] have also been shown in hypertensive patients. It is currently not known whether the beneficial effect of physical activity derives from the influence of physical activity itself or results from its effects on other risk factors or pathophy-

siological mechanisms, or all of these. There is evidence from randomized controlled trials and observational studies that exercise has favourable effects on blood pressure, glucose homeostasis, blood lipids, body fatness, smoking behaviour and endothelial function, among other factors [3].

With regard to blood pressure, several large epidemiological studies, which allowed for age and anthropometric characteristics, have reported an inverse relationship between blood pressure and either habitual physical activity assessed by questionnaire or interview or measured physical fitness [7]. In addition, exercise and fitness were inversely related to the later development of hypertension [8]. The population attributable risk percentage of physical inactivity for hypertension was

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estimated at 5–13% [9]. Not all epidemiological studies support this view, however, but the low level of physical activity in western societies may have hampered the detection of such a relationship. Moreover, in studies that found a significant association, the difference in blood pressure between the most and the least physically active subjects usually amounted to no more than 5 mmHg after adjustment for confounding variables, such as age, anthropometric characteristics and lifestyle factors.

It remains difficult, however, to ascribe differences in blood pressure or in the incidence of hypertension within a population to differences in levels of physical activity or fitness, because of confounding factors that cannot be accounted for, such as self selection, or because of the limitations of statistical adjustment for confounders that have been taken into consideration. Randomized controlled trials are therefore of paramount importance to assess the effects of exercise on blood pressure. Physical exercise can be divided into two broad categories, namely dynamic aerobic endurance training and resistance training [10]. We recently performed meta-analyses of randomized controlled trials on the effects of each type of exercise on blood pressure, and, if available, on blood pressure-regulating mechanisms and cardiovascular risk factors [11–13]. The database used for the meta-analysis contains articles published up to December 2003, and the following criteria were applied with regard to their acceptability: randomized controlled trials of at least 4 weeks' duration concerning normotensive or hypertensive subjects, or both, in whom cardiovascular diseases were reasonably well excluded; random allocation to intervention groups and control groups or control phases in the case of crossover designs; full publication in a peer-reviewed journal; and the absence of confounding by some other intervention during the intervention of interest. Finally, studies were accepted only when the actual blood pressures for the intervention and the control groups or phases, or the pressure changes during the intervention and control periods, were available. With regard to the statistical analysis, meta-analyses consisted of analyses of pooled data with study groups as the units of analysis, with weighting for the number of trained participants in each group. The net effects of physical training were assessed by weighted pooled analyses of the changes in the intervention groups, adjusted for control data. Results are reported as weighted means and 95% confidence limits (95% CL). Finally, weighted meta-regression analysis was applied to assess whether variations in the results were related to variations in study group or training characteristics.

Dynamic aerobic endurance training

Dynamic aerobic endurance exercise refers to training programmes that involve large muscle groups in dynamic activities, designed specifically to increase aerobic

endurance performance [10]. Previous meta-analyses on the effects of this type of exercise on blood pressure, including the most recent ones [14–17], reported on resting blood pressure only. Our aim was to perform a comprehensive meta-analysis, including resting, ambulatory and exercise blood pressure, blood pressure-regulating mechanisms and concomitant cardiovascular risk factors [12,18].

The meta-analysis [12] involved 72 trials, 105 study groups and 3936 participants. The average age of the study groups ranged from 21 to 83 years (median 47), and 57% of the participants were men. The study duration varied from 4 to 52 weeks (median 16). The average training frequency ranged from one to 7 days per week (median 3), and average intensity was between 30 and 87.5% of heart rate reserve (median 65). Each training session lasted from 15 to 63 min (median 40) after the exclusion of warm-up and cool-down activities, and involved mainly walking, jogging, running and cycling. Peak oxygen uptake increased by 4.0 (95% CL 3.5; 4.5) ml/min per kilogram from a baseline value of 31.1 ml/min per kilogram and heart rate decreased by 4.8 (3.9; 5.7) beats per minute (bpm) from 73 bpm at baseline.

Blood pressure

Table 1 summarizes the results on blood pressure. The overall reduction in resting blood pressure averaged 3.0/2.4 mmHg, after weighting for the number of trained participants and adjustment for the control observations. A number of limitations should be considered, namely that participants were aware of their allocation to control or intervention in training studies, and several important scientific criteria were not always observed, such as regular follow-up of the control subjects, attention to possible changes in other lifestyle factors, and blinded or automated blood pressure measurements. However, the fact that net daytime ambulatory blood pressure was reduced to a similar extent as conventional blood pressure

Table 1 Baseline data for the training group and weighted net changes in resting blood pressure, and daytime and night-time ambulatory blood pressure in response to dynamic aerobic endurance training

Blood pressure (mmHg)	N	Baseline	Net change (95% CL)	P value
Resting blood pressure				
Systolic	104	128.1	-3.0 (-4.0; -2.0)	< 0.001
Diastolic	102	81.6	-2.4 (-3.1; -1.7)	< 0.001
Daytime blood pressure^a				
Systolic	11	134.8	-3.3 (-5.8; -0.9)	< 0.01
Diastolic	11	85.6	-3.5 (-5.2; -1.9)	< 0.01
Night-time blood pressure				
Systolic	6	116.4	-0.6 (-2.8; +1.6)	NS
Diastolic	6	71.4	-1.0 (-2.5; +0.5)	NS

CL, confidence limits; N, number of study groups in which the variable was reported. Values are means, weighted for the number of trained participants. ^aIncludes 24-h blood pressure in two trials that only reported 24-h blood pressure.

in the overall analysis ($-3.3/-3.5$ mmHg; Table 1) supports the blood pressure-lowering effect of endurance training. It is noteworthy that training does not affect night-time blood pressure. In addition, a number of studies reported on blood pressure measured during exercise testing [18]. In eight randomized controlled trials blood pressure was measured during cycle ergometer exercise at a median work load of 100 W (range 60–140). Blood pressure was assessed during treadmill exercise at an energy expenditure of approximately four metabolic equivalents in two other studies. Pretraining exercise systolic blood pressure (SBP) averaged 180 mmHg and heart rate 124 bpm. The weighted net training-mediated decrease in SBP and heart rate were significant, corresponding to 7.0 mmHg and 6.0 bpm, respectively.

When the study groups were divided according to the average baseline blood pressure, the change of resting blood pressure was more pronounced in the 30 hypertensive study groups [-6.9 ($-9.1; -4.6$)/ -4.9 ($-6.5; -3.3$) mmHg] than in the 72 normotensive groups [-2.0 ($-3.0; -0.9$)/ -1.6 ($-2.3; -1.0$) mmHg]. The suggestion that the response of blood pressure is more pronounced in the hypertensive individuals is corroborated by the results of seven studies in which normotensive and hypertensive individuals followed the same training programme [19]. In each of the studies the blood pressure change was greater in the hypertensive patients than in the normotensive individuals. The weighted averages were -13 ($-15; -11$)/ -8 ($-10; -6$) mmHg in the hypertensive and -3 ($-7; +0.5$)/ -2 ($-5; +1$) mmHg in the normotensive individuals.

There were no significant relationships between the training-induced net changes in resting blood pressure and the age at baseline. Blood pressure decreased by 3.8 (1.8; 5.8)/2.0 (0.6; 3.5) mmHg in the 31 study groups with an average age of less than 40 years, by 2.3 (1.1; 3.6)/2.1 (1.2; 2.9) mmHg when the age was between 40 and 59 years, and by 5.4 (2.8; 8.1)/3.3 (1.8; 4.8) mmHg in those aged 60 years and older. In addition, the change in blood pressure was not related to baseline body mass index [20] and there was no evidence that the response was different in men and women.

Finally, we analysed whether the training characteristics affected the results, and found no significant effect of training frequency, intensity and mode, and of the time per session on the blood pressure response, despite the wide variation in these characteristics [14]. Nevertheless, the blood pressure decrease appeared to be somewhat more pronounced with greater increases in peak oxygen uptake ($r = 0.24$, $P < 0.05$ for SBP; $r = 0.40$, $P < 0.001$ for diastolic blood pressure). Finally, the blood pressure reduction became smaller with a longer total study

duration ($P < 0.05$), possibly related to a loss of compliance.

Mechanisms of the changes in blood pressure

Haemodynamic measurements were reported in 17 study groups. The decrease in mean blood pressure by 4.3 (2.1; 6.4)% was based on a 7.1 (1.4; 14)% reduction in systemic vascular resistance with unchanged cardiac output. The 9.3 (6; 13)% decrease in heart rate was counterbalanced by a 15.4 (7.5; 23.5)% increase in stroke volume. These results are compatible with the generally accepted effect of aerobic endurance training on resting haemodynamics [21]. A decrease in the activity of the autonomic nervous system is most likely involved in the training-induced reduction in blood pressure and systemic vascular resistance, as evidenced by the lower levels of plasma norepinephrine [-29 ($-40; -18$)%; $N = 18$] and of plasma renin activity [-20 ($-35; -5$)%; $N = 10$] in the fit state when compared with untrained values [12]. The lack of an effect on blood pressure during sleep, when sympathetic activity is low, is compatible with a role for the sympathetic nervous system in the hypotensive effect of endurance training. The reduction in insulin resistance may also have contributed to the favourable effect on blood pressure. An improvement in endothelial function is another potentially important mechanism, but the available data are few and not suitable for meta-analysis [12].

Cardiovascular risk factors

As shown in Table 2 exercise training induced a significant net reduction in weight, but metaregression analysis showed that the changes in weight did not explain the differences in blood pressure response among study groups [20]. We also noted significant reductions in body fat and abdominal visceral fat in response to training. Further beneficial effects were the increase in high-density lipoprotein cholesterol and decreases in triglycerides, glucose, insulin and the homeostasis model assessment index of insulin resistance. These findings are compatible with an overall improvement in cardiovascular risk. However, it is difficult to quantify exactly the overall risk reduction associated with all observed changes, but the findings are compatible with the evidence from epidemiological prospective follow-up studies that physical activity and fitness are inversely related to the incidence of cardiovascular disease and mortality [1–3,22]; the benefits of moderately vigorous activity and of greater fitness have also been shown in hypertensive patients [4–6].

Resistance training

Resistance training refers to training programmes that involve strength, weight, static or isometric exercises designed specifically to increase muscular strength, power and endurance [10]. We identified only nine

Table 2 Baseline data for the training group and weighted net changes of anthropometric characteristics and cardiovascular risk factors in response to dynamic aerobic endurance training

Variable	N	Baseline	Net change (95% CL)	P value
Weight (kg)	80	75.2	-1.2 (-1.5; -0.9)	< 0.001
Body fat (%)	30	30.9	-1.4 (-1.8; -1.0)	< 0.001
Waist circumference (cm)	9	91.3	-2.8 (-4.0; -1.7)	< 0.001
Waist-to-hip ratio	8	0.90	-0.0092 (-0.018; -0.0004)	< 0.05
Cholesterol (mmol/l)				
Total	31	5.5	-0.040 (-0.13; +0.045)	NS
HDL	38	1.4	+0.032 (+0.005; +0.059)	< 0.05
LDL	30	3.6	-0.078 (-0.30; +0.15)	NS
Triglycerides (mmol/l)	39	1.4	-0.11 (-0.24; +0.0095)	0.07
Glucose (mmol/l)	18	5.0	-0.15 (-0.20; -0.11)	< 0.001
Insulin (IU/l)	19	11.6	-1.4 (-2.2; -0.53)	< 0.005
HOMA index	14	2.1	-0.31; (-0.53; -0.094)	< 0.01

CL, Confidence limits; HDL, high-density lipoprotein; HOMA, homeostasis model assessment; LDL, low-density lipoprotein; N, number of study groups in which the variable was reported. Values are reported as means, weighted for the number of trained participants.

randomized controlled trials on the effects of resistance training on blood pressure, involving 12 study groups and 341 participants [13]. The average age of the study groups ranged from 20 to 72 years (median 69) and 61% of the participants were men. Study durations varied from 6 to 26 weeks (median 14). Training frequency amounted to 2 and 3-weekly sessions in two and 10 study groups, respectively, and intensity ranged from 30 to 90% of one repetition maximum (median 70). The duration of each session is difficult to assess from the reported data. The number of different exercises performed ranged from one to 14, whereas the number of sets for each type of exercise ranged from one to four. The number of repetitions per set ranged from one to 25. The mode of training was purely static in one study, and comprised dynamic muscular exercises with variable resistance in the others. The training programme resulted in an overall weighted net change in SBP of 3.2 mmHg ($P = 0.10$) and in diastolic blood pressure of 3.5 mmHg ($P < 0.01$; Table 3). However, based on the average pretraining blood pressure, only three trials were conducted in hypertensive patients, so that no reliable conclusions can be drawn for those patients, in whom more studies are needed.

It is important to consider the training regimens in those studies. The change in blood pressure cannot be attributed to pure static training. In all but one study, most exercises were dynamic and, therefore, involved movements of the arms, legs or trunk, or both. Furthermore, the training intensity was not always high and ranged from 30 to 60% of one repetition maximum in half of the study groups and from 70 to 80% of one repetition maximum in the others. It is noteworthy that aerobic power increased by 10.5% in the six study groups in which it was measured, which would not be expected from static training. This suggests that the kind of resistance exercises used in most protocols comprised an aerobic component to some extent. It should also be mentioned that there is no evidence that strength training leads to an increase in blood pressure.

Table 3 Baseline data for the training group and weighted net changes in response to resistance training

Variable	Baseline		Net change		P value
	N	Mean	N	Mean (95% CL)	
Blood pressure (mmHg)					
Systolic	12	131.0	12	-3.2 (-7.1 to +0.7)	0.10
Diastolic	12	81.1	12	-3.5 (-6.1 to -0.9)	< 0.01
VO _{2max} (ml/min per kg)	9	24.7	6	+2.6 (+0.3 to +4.8)	< 0.05
Heart rate (bpm)	10	70.7	8	+1.0 (-1.7 to +3.7)	NS
Weight (kg)	8	76.4	4	+0.33 (-2.7 to +3.4)	NS
Body fat (%)	6	30.1	4	-0.94 (-1.6 to -0.25)	< 0.01

CL, Confidence limits; N, number of study groups in which the variable was reported; VO_{2max}, maximal oxygen uptake. Values are reported as means, weighted for the number of trained participants.

Few studies have addressed the underlying mechanisms responsible for the reduction in blood pressure in response to resistance training. Vanhoof *et al.* [23] could not observe any change in sympathetic tone, assessed by power spectral analysis of heart rate variability, and Coconie *et al.* [24] found no change in plasma epinephrine and norepinephrine levels at rest after training. The lack of a change in heart rate in our meta-analysis (Table 3) could support the absence of a change in sympathetic activity.

Conclusion

Our results indicate that aerobic endurance training decreases blood pressure through a reduction in systemic vascular resistance in which the sympathetic nervous system and the renin-angiotensin system appear to be involved, and favourably affects cardiovascular risk factors. The effect on blood pressure is more pronounced in hypertensive than in normotensive individuals. Therefore, exercise contributes to the control of blood pressure in hypertensive patients and is likely to contribute to the prevention of hypertension in normotensive subjects, which is compatible with observational studies that concluded that exercise and fitness are inversely related to the later development of hypertension [8]. Although

there are fewer data on resistance training, the data suggest that resistance training of moderate intensity is able to reduce blood pressure. Various approaches have been used to increase physical activity and maintain adherence. The effectiveness of these methods has been reviewed by Kahn *et al.* [25].

Our results support the recommendation that exercise is a cornerstone therapy for the prevention, treatment and control of hypertension. Based upon the current evidence the following exercise prescription is recommended: frequency: on most, preferably all days of the week; intensity: moderate intensity; time: at least 30 min of physical activity per day; and type: primarily endurance physical activity, supplemented by resistance exercise [18,26]. The exercising hypertensive patients should be assessed and treated according to the general guidelines for the management of hypertension [27]. In addition to the generally recommended tests, the indication for exercise testing depends on the patient's risk and on the sports characteristics (Table 4) [25–28]. In patients with hypertension about to engage in hard or very hard exercise (intensity $\geq 60\%$ of maximum), a medically supervised peak or symptom-limited exercise test with electrocardiogram and blood pressure monitoring is warranted. In asymptomatic men or women with low or moderate added risk, who engage in light-to-moderate dynamic physical activity (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 dyspnoea, chest discomfort or palpitations need further examination, which includes exercise testing, echocardiography, Holter monitoring, or combinations thereof.

With regard to treatment, appropriate non-pharmacological measures should be considered in all patients, that is moderate salt restriction, an increase in fruit and vegetable intake, a decrease in saturated and total fat intake, limitation of alcohol consumption to no more than 20–30 g ethanol per day for men and no more than 10–20 g ethanol per day for women, smoking cessation

Table 4 Indications for exercise testing for sports participation in patients with hypertension

Demands of exercise	Risk category [26]	
	Low or moderate	High or very high ^a
Static or dynamic		
Light ($< 40\%$ of max)	No	No
Moderate (40–59% of max)	No	Yes
High ($\geq 60\%$ of max)	Yes	Yes

^aIn the case of an associated clinical condition, the recommendations for the specific condition should be observed [26].

and control of body weight. Antihypertensive drug therapy should be started promptly in patients at high or very high added risk of cardiovascular complications. In patients at moderate added risk, drug treatment is only initiated when hypertension persists after several months despite appropriate lifestyle changes. Drug treatment is currently not considered mandatory in patients at low added risk. The goal of antihypertensive therapy is to reduce blood pressure to at least below 140/90 mmHg and to lower values if tolerated in all hypertensive patients, and to below 130/80 mmHg in individuals with diabetes. Current evidence indicates that patients with white-coat hypertension do not have to be treated with antihypertensive drugs, unless they are at high or very high risk, but regular follow-up and non-pharmacological measures are recommended. Also individuals with normal blood pressure at rest and an exaggerated blood pressure response to exercise should be followed up more closely. Several drug classes can be considered for the initiation of antihypertensive therapy: diuretics; beta-blockers; calcium antagonists; angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers. However, diuretics and beta-blockers are not recommended for first-line treatment in the exercising hypertensive patient because they may impair exercise performance [19]. Finally, all exercising patients should be advised on exercise-related warning symptoms, such as chest pain or discomfort, abnormal dyspnoea, dizziness or malaise, which would necessitate consulting a qualified physician.

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References

- Powell KE, Thompson PD, Caspersen CJ, Kendrick JS. Physical activity and the incidence of coronary heart disease. *Ann Rev Public Health* 1987; **8**:253–287.
- Berlin JA, Colditz GA. A meta-analysis of physical activity in the prevention of coronary heart disease. *Am J Epidemiol* 1990; **132**:12–28.
- Fagard RH. Physical exercise and coronary artery disease. *Acta Cardiol* 2002; **57**:91–100.
- Shaper AG, Wannamethee G, Walker M. Physical activity, hypertension and risk of heart attack in men without evidence of ischaemic heart disease. *J Hum Hypertens* 1994; **8**:3–10.
- Pardaens K, Reybrouck T, Thijs L, Fagard R. Prognostic significance of peak oxygen uptake in hypertension. *Med Sci Sports Exerc* 1996; **28**:794–800.
- Blair SN, Kohl HW III, Barlow CE, Gibbons LW. Physical fitness and all-cause mortality in hypertensive men. *Ann Med* 1991; **23**:307–312.
- Fagard RH, Cornelissen V. Physical activity, exercise, fitness and blood pressure. In: Battagay EJ, Lip GYH, Bakris GL, editors. *Hypertension, principles and practice*. Boca Raton: Taylor and Francis Books; 2005. pp. 195–206.
- Fagard RH. Physical activity, physical fitness and the incidence of hypertension. *J Hypertens* 2005; **23**:265–267.
- Geleijnse JM, Grobbee DE, Kok FJ. Impact of dietary and lifestyle factors on the prevalence of hypertension in western populations. *J Hum Hypertens* 2005; **19** (Suppl 3s):S1–S4.
- Howley ET. Type of activity: resistance, aerobic and leisure versus occupational physical activity. *Med Sci Sports Exerc* 2001; **33** (6 Suppl):S364–S369.
- Fagard RH, Staessen JA, Thijs L. Advantages and disadvantages of the meta-analysis approach. *J Hypertens* 1996; **14** (Suppl 2):S9–S13.

- 12 Cornelissen VA, Fagard RH. Effects of endurance training on blood pressure, blood pressure regulating mechanisms and cardiovascular risk factors. *Hypertension* 2005; **46**:667–675.
- 13 Cornelissen VA, Fagard RH. Effect of resistance training on resting blood pressure: a meta-analysis of randomized controlled trials. *J Hypertens* 2005; **23**:251–259.
- 14 Fagard RH. Exercise characteristics and the blood pressure response to dynamic physical training. *Med Sci Sports Exerc* 2001; **33** (Suppl): S484–S492.
- 15 Kelley GA, Kelley KA, Tran ZV. Aerobic exercise and resting blood pressure: a meta-analytic review of randomised, controlled trials. *Prev Cardiol* 2001; **4**:73–80.
- 16 Whelton SP, Chin A, Xin X, He J. Effect of aerobic exercise on blood pressure: a meta-analysis of randomized controlled trials. *Ann Intern Med* 2002; **136**:493–503.
- 17 Dickinson HO, Mason JM, Nicolson DJ, Campbell F, Beyer FR, Cook JV, et al. Lifestyle interventions to reduce raised blood pressure: a systematic review of randomized controlled trials. *J Hypertens* 2006; **24**: 215–233.
- 18 Pescatello LS, Franklin BA, Fagard R, Farquhar WB, Kelley GA, Ray CA. Exercise and hypertension: American College of Sports Medicine, Position Stand. *Med Sci Sports Exerc* 2004; **36**:533–552.
- 19 Fagard R, Amery A. Physical exercise in hypertension. In: Laragh JH, Brenner BM, editors. *Hypertension: pathophysiology, diagnosis and management*, 2nd ed. New York: Raven Press Ltd.; 1995. pp. 2269–2681.
- 20 Fagard RH. Physical activity in the prevention and treatment of hypertension in the obese. *Med Sci Sports Exerc* 1999; **31** (Suppl):S624–S630.
- 21 Rowell LB. Human cardiovascular adjustments to exercise and thermal stress. *Physiol Rev* 1974; **54**:75–159.
- 22 Thompson PD, Buchner D, Pina IL, Balady GJ, Williams MA, Marcus BH, et al. American Heart Association Council on Clinical Cardiology Subcommittee on Exercise, Rehabilitation and Prevention, American Heart Association Council on Nutrition, Physical Activity and Metabolism Subcommittee on Physical Activity. Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease: a statement from the Council on Clinical Cardiology Subcommittee on Exercise, Rehabilitation and Prevention and the Council on Nutrition, Physical Activity and Metabolism Subcommittee on Physical Activity. *Circulation* 2003; **107**:3109–3116.
- 23 Van Hoof R, Macor F, Lijnen P, Staessen J, Thijs L, Vanhees L, et al. Effect of strength training on blood pressure measured in various conditions in sedentary men. *J Sports Med* 1996; **17**:415–422.
- 24 Coconie CC, Graves JE, Pollock ML, Phillips MI, Summers C, Hagberg JA. Effect of exercise training on blood pressure in 70- to 90-yr-old men and women. *Med Sci Sports Exerc* 1991; **23**:505–511.
- 25 Kahn EB, Ramsey LT, Brownson RC, Heath GW, Howze EH, Powell KE, et al. The effectiveness of interventions to increase physical activity. A systematic review. *Am J Prev Med* 2002; **22**:73–107.
- 26 Fagard RH, Björnstad HH, Borjesson M, Carré F, Deligiannis A, Vanhees L. ESC Study Group on Sports Cardiology Recommendations for participation in leisure-time physical activities and competitive sports for patients with hypertension. *Eur J Cardiovasc Prev Rehab* 2005; **12**:326–331.
- 27 Guidelines Committee. 2003 European Society of Hypertension, European Society of Cardiology guidelines for the management of arterial hypertension. *J Hypertens* 2003; **21**:1011–1053.
- 28 Pelliccia A, Fagard R, Björnstad HH, Anastassakis A, Arbustini E, Assanelli D, et al. Recommendations for competitive sports participation in athletes with cardiovascular disease. *Eur Heart J* 2005; **26**:1422–1445.