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Physical activity and exercise lower blood pressure in individuals with hypertension: narrative review of 27 RCTs

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ABSTRACT

Regular physical activity (PA) reduces the blood pressure (BP) of individuals with hypertension. The present review analysed the scientific evidence for the BP lowering effect of aerobic PA in 27 randomised controlled studies on individuals with hypertension, and shows that regular medium-to-high-intensity aerobic activity reduces the BP by a mean of 11/5 mm Hg (level of evidence, 3+). In addition, three randomised controlled trials (RCTs) on isometric (static) activity showed a BP reduction of similar magnitude in hypertensives; dynamic resistance training may show less effect, as shown in five available RCTs (level of evidence 2+). As both the prevalence of hypertension and physical inactivity are high and increasing in today's society, PA has a great role to play as a single (when indicated) or additive treatment for hypertension. Furthermore, as competitive athletes are getting older, it can be expected that more athletes at different competitive levels will have hypertension. Certain considerations must be applied regarding evaluation and treatment of hypertension in athletes. Eligibility for competitive sports may be affected if target organ damage (TOD) is present; however, an athlete with well-controlled BP, having no additional risk factors or TOD, is eligible for all sports.

INTRODUCTION

Regular physical activity (PA) prevents hypertension and is also a basic part of the treatment of hypertension. It may be part of the lifestyle treatment for low-risk patients or can be used in combination with pharmacological treatment for hypertensive patients with increased risk profile.[1] While most of the available studies show a blood pressure (BP) reducing effect from regular PA or an association with lower BP in physically active individuals, the scientific quality of many of these studies have been varying. This may be due to the small number of participants or the short follow-up. In addition, the intervention may be vaguely or not at all described, without delineating the type, intensity, frequency or duration (dosage) of exercise. In addition, earlier meta-analyses have included a mix of normotensives and prehypertensives. The present review, therefore, aims to discuss the scientific evidence for the BP lowering effect of PA, exclusively in patients with hypertension. This part of the paper (I) was based on (but completely rewritten) the latest FYSS chapter on 'physical activity and hypertension' (http://www.fyss.se), and on existing recommendations.[1]

In addition, in the context of sports medicine, hypertension is present in a growing number of athletes, at various levels. Hypertension in an athlete should be evaluated and treated specifically; this could be a possible barrier to active participation.[2] The second part (II) of the paper, reviews the existing recommendations and evaluation of hypertension in athletes.

Effects of PA on BP

Hypertension is an independent risk factor for cardiovascular disease, and it is considered to be the most important modifiable risk factor for cardiovascular morbidity and mortality.[3] The diagnosis should only be made with repeated measurements of >140 mm Hg systolic and/or 90 mm Hg diastolic BP, performed in a standardised manner with the patient in a sitting position, in accordance with current guidelines.[1] Ambulatory BP measurements (24 h BP) is increasingly used, both for diagnostics and for follow-up in the home setting, using a lower cut-off value for what is considered an abnormal BP-level, that is >120/75 at night.[1 4] The level of BP in itself is associated with cardiovascular complications but any concomitant additional risk factor increases the risk; consequently, the total risk profile of the patient should always be assessed.[1 3]

The great majority of patients with hypertension suffer from so called (s.c) essential hypertension (95%), which means that no single explanatory cause of the disease can be established.[1] Instead, essential hypertension is believed to be the result of a complex combination of genetic predisposition and lifestyle factors. These lifestyle factors include physical inactivity, aggravated alcohol intake, smoking and poor diet, associated with weight gain.[1] Specifically, physical inactivity may account for 5–13% of the risk for the development of hypertension.[5] The modern lifestyle, with widespread and decreasing physical activity in general, has the potential to worsen this situation, with the prevalence of hypertension being expected to reach 30% among adults worldwide by 2025.[6 7] Physical inactivity may increase even further in the future,[8] accentuating the problem of hypertension in society.

In contrast, about 5% of hypertensive individuals suffer from secondary hypertension that is caused by a variety of underlying defined diseases such as coarctatio aortae, renal artery stenosis, Cushings syndrome or pheochromocytoma.[1] Also medications could be a cause of increased BP, including non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, cyclosporine and/or

erythropoeitin.[1 5] These causes of hypertension must always be excluded in the initial evaluation of newly-diagnosed hypertensive patients.

The initial pathophysiological step towards established hypertension is increased cardiac output, being a product of the heart rate and stroke volume, in turn related to increased sympathetic activity. Later, secondary to vessel wall thickening and remodelling, the peripheral resistance increases causing more changes to manifest in the BP. Endothelial dysfunction, as well as accelerated atherosclerosis of the larger vessels, will take place secondary to prolonged hypertension and there may be left ventricular (LV) cardiac hypertrophy and evidence of kidney dysfunction. Eventually, various cardiovascular complications (stroke, myocardial infarction) may ensue in proportion to both the increased systolic and diastolic BP.[9]

Traditional treatment of hypertension is a combination of lifestyle modification and pharmacological treatment, when needed.[1] Reducing increased BP has been shown to reduce morbidity and mortality, even if these are small BP reductions.[9] Importantly, only a small fraction of people with known (some) or unknown (none) hypertension have reached target-BP levels[1 10 11] despite the multitude of available treatment options. Thus, lifestyle modifications, including increasing PA that is presently underutilised, have a potentially important role in BP treatment.

The role of PA in treatment of hypertension is dependent on both the level of BP and on the total risk profile of the individual; PA is the first-line treatment in hypertensive patients with low-medium cardiovascular risk.[1] In patients with higher risk profiles, PA is always recommended as additional treatment to pharmacological treatment.[1]

Acute effects of PA on BP

The acute effects of PA on BP is dependent on the type of activity performed, as well as on the intensity and level of resting BP (normotensive or hypertensive individual). During acute aerobic activity of sufficient intensity, the systolic pressure increases while the diastolic pressure is unchanged or increases marginally.[12] In the hypertensive patient, the BP response may be exaggerated[12] during aerobic activity. After completion of aerobic activity, the BP will decrease below resting BP, and this is called 'postexercise hypotension'.[13–15] The decrease in BP can be up to 10–20 mm Hg and typically persists for hours after training.[13–15]

During dynamic resistance training (concentric, excentric), for example, in weight-lifting, the BP typically increases more as compared to aerobic training, sometimes reaching very high levels. This is accompanied by an increase in peripheral resistance, but the heart rate and stroke volumes are less affected.[16 17]

Long-term effects of PA on BP

Regarding long-term effects of PA on BP levels, many studies have been performed, most of these show a BP reducing effect from PA or an association with lower BP in physically active individuals.[18–20]

However, the scientific quality of many available studies vary due to the small number of participants or short follow-up. In addition, the intervention may be vaguely or not at all described, with no proper delineating of the type, intensity, frequency or duration (dosage) of the exercise. Another important fact is that many previous studies and meta-analyses have included a mix of normotensives and prehypertensives. This will, of course, seriously affect the possibility of showing an effect of PA on hypertension.

Methods

For the current literature search, we started with the meta-analysis by Cornelissen and Smart,[18] by identifying the RCT's which included effects of PA on patients with hypertension up to 2011. We then performed an additional MEDLINE search in PubMed, EMBASE, Cochrane, AMED and CRD databases (from 1 January 2012 to 1 February 2014), as described in table 1. The above searches resulted in 2245 references (1382 from Pubmed). After manual removal of duplicates, 1764 references remained.

Applying our inclusion criteria of (1) Human studies, (2) Non-acute studies (>2 weeks), (3) Hypertensive patients included, (4) Intervention with defined type of PA used and (5) Randomised controlled studies (RCT)s only, we searched all 1764 titles and abstracts for relevant additional papers. Subsequently, we added a small number of papers (n=3) from the reference lists of included studies. Finally, 33 RCTs fulfilling the above criteria were identified, and these formed the basis for our conclusions regarding the BP lowering effects of various forms of PA in patients with hypertension.

We applied the GRADE system (http://www.gradeworkinggroup.org) of describing the level of scientific evidence for the effects of the different types of activity in the lowering of BP in hypertensives. The GRADE system defines four levels of scientific evidence, ranging from 1+ (lowest evidence) to 4+ (highest evidence).

Results

Aerobic PA

Regarding aerobic training, we included 27 RCTs, a total of 1480 participants (table 2). The included studies encompassed

Table 1 The search string and database yield for each search terms (PubMed)

Search#	Search term	Database yield (n)
#1	Blood pressure[mh] OR Hypertension[mh]	400 517
#2	Blood pressure*[tiab] OR Hypertens*[tiab] OR Bloodpressure*[tiab]	451 071
#3	#1 OR #2	606 770
#4	Physical education and training[mh] OR Resistance training[mh] OR Physical endurance[mh] OR Exercise [mh] OR Exercise therapy[mh] OR Sports[mh]	208 798
#5	Exercise*[tiab] OR Physical activity[tiab] OR Physically active[tiab] OR physical activities[tiab] OR (Training* [tiab] AND (aerobic*[tiab] OR strength*[tiab] OR physical[tiab] OR endurance[tiab] OR resistance[tiab]))	261 660
#6	#4 OR #5	367 526
#7	#6 AND #3	35 897
#8	Systematic[sb] OR systematic review[tiab] OR meta-analysis[tiab] OR meta-analyses[tiab] OR metaanalysis[tiab] OR metaanalyses[tiab] OR Meta-Analysis as Topic[mh] OR Meta-analysis[pt] OR Randomized controlled trial[pt] OR Controlled clinical trial[pt] OR Clinical Trials as topic[mh] OR Randomized controlled trials as topic[mh] OR Random allocation[mh] OR Random*[tiab] OR control[tiab]	275 017
#9	#7 AND #8	12 902
#10	((animals[mh]) NOT (animals[mh] AND humans[mh]))	3 857 919
#11	(Editorial[ptyp] OR Letter[ptyp] OR Comment[ptyp] OR case reports[ptyp]	2 781 326
#12	#9 NOT #10	12 075
#13	#12 NOT #11 Filters: Publication date from 2012/01/01; Danish; English; Norwegian; Swedish	1382

Table 2 RCTs with interventions of aerobic activity

Author	Year	Size (n)	Intervention(s) (n)	Control (n)	Sex	Activity	Intensity	Frequency	Duration (weeks)	Effect
Aerobic										
Blumenthal *et al*	1991	92	39/31	22	M/W	Walking/jogging or resistance training	70% VO_2max	35 min, 3×/w	16	ns
Cononie *et al*	1991	17	6/6	5	M/W	Walking/jogging or resistance training	75–85% VO_2max	35–45 min, 3×/w	26	Endurance: SBP ns, DBP 9 mm Hg Resistance: ns
de Plaen *et al*	1980	10	6	4	NA	Aerobic exercise	60–70% VO_2max	30 min, 3×/w	13	ns
Duncan *et al*	1985	56	44	12	M	Walking/jogging	70–80% max HR	60 min, 3×/w	16	SBP ns, DBP 9 mm Hg
Georgiades *et al*	2000	55	36	19	M/W	Walking/jogging and cycling	70–85% HRR	60 min, 3–4×/w	26	SBP 3, DBP 5 mm Hg
Hagberg *et al*	1989	31	11/10	10	M/W	Walking or walking/jogging/cycling	50% or 70–85% VO_2max	60 min, 3×/w	37	Low intensity: SBP 20, DBP 13 mm Hg Moderate: SBP ns, DBP 10 mm Hg
Kokkinos *et al*	1995	46	23	23	M	Cycling	74% max HR	45 min, 3×/w	32	SBP ns, DBP 5 mm Hg
Lamina	2010	357	112/140	105	M	Cycling (continuous or interval)	60–79% max HR	45–60 min 3×/w	8	Continuous: SBP 14, DBP 7 mm Hg Interval: SBP 16, DBP 4 mm Hg
Martin *et al*	1990	19	10	9	M	Walking/jogging/cycling	65–80 max HR	30 min, 4×/w	10	SBP ns, DBP 10 mm Hg
Molmen-Hansen	2011	88	31/28	29	M/W	Aerobic interval training or continuous training	>90% max HR or 70% max HR	3×/w	12	Interval: SBP 12, DBP 8 mm Hg Continuous: SBP 4, DBP 3 mm Hg
Sakai *et al*	1998	29	16	13	M/W	Cycling	40–60% VO_2max	60 min, 3×/w	4	SBP 11, DBP 7 mm Hg
Staffileno *et al*	2001	18	9	9	W	Aerobic exercise (eg, walking)	50–60% HRR	10 min x[3], 5×/w	8	SBP 8 mm Hg, DBP ns
Tanabe *et al*	1989	31	21	10	M/W	Cycling	40–60% VO_2max	60 min, 3×/w	10	SBP 15, DBP 7 mm Hg
Tanaka *et al*	1997	18	12	6	M/W	Swimming	60% HRR	60 min, 3×/w	10	SBP 6 mm Hg, DBP ns
Tsai, Chang *et al*	2002	23	12	11	NA	Walking/jogging	60–70% max HR	50 min, 3×/w	12	SBP 18, DBP 10 mm Hg
Tsai, Liu *et al*	2002	42	22	20	M/W	Walking/jogging	60–70% max HR	50 min, 3×/w	12	SBP 11, DBP 5 mm Hg
Tsai *et al*	2004	102	52	50	M/W	Walking/jogging	60–70% max HR	50 min, 3×/w	10	SBP 13, DBP 6 mm Hg
Tsuda *et al*	2003	16	8	8	M	Walking/jogging +calisthenics	40–60% VO_2max	30+30 min, 2×/w	26	SBP 10, DBP 6 mm Hg
Walking (no specified intensity)										
Lee *et al*	2007	202	102	100	M/W	Walking			26	SBP 15 mm Hg, DBP ns
Moreau *et al*	2001	24	15	9	W	Walking		3 km extra per day	24	SBP 11 mm Hg, DBP ns
Sohn *et al*	2007	19	8	10	M/W	Walking		30 min extra per day	26	ns
Dimeo *et al*	2012	50	24	26	M/W	Walking		3×/w	12	SBP 6, DBP 3 mm Hg
Higashi *et al*	1999	17	10	7	M/W	Walking		30 min 5–7×/w	12	SBP 7, DBP 4 mm Hg
Other forms of aerobic activity without specified intensity										
Arca *et al*	2013	52	19/19	14	W	Water/land aerobic training		50 min, 3×/w	12	Water: SBP 12 mm Hg, DBP ns. Land: SBP 12 mm Hg, DBP ns
Aweto	2012	38	23	15	M/W	Dance movement therapy		50 min, 2×/w	4	SBP 20, DBP 9 mm Hg
Guimaraes *et al*	2014	32	16	16	M/W	Water training (callisthenics +walking)		60 min, 3×/w	12	SBP 17, DBP 9 mm Hg
Krustrup *et al*	2013	33	22	11	M	Soccer		60 min, 2×/w	26	SBP 12, DBP 8 mm Hg

DBP, Diastolic blood pressure; ns, not significant; SBP, Systolic blood pressure.

those studies where the aerobic activity was defined in detail regarding intensity, frequency and duration.[21–38] In addition, we included studies on aerobic PA by hypertensive individuals, where the dose of activity was not possible to delineate, for example, a number of studies on walking where such detailed description of aerobic dose was not possible.[39–43] Also, a number of other RCT studies including unspecified aerobic activity, such as water training,[44 45] dance[46] and soccer,[47] were included in the analysis. In summary, the mean BP lowering effect of aerobic training in hypertensive patients, in the

included RCT studies, was 10.8/4.7 mm Hg, with 3+ level of evidence.

Most studies have included medium or high-intensity aerobic training (table 2), while a few studies have included both medium and high-intensity activity; these show similar BP reductions as that for aerobic activity overall. The duration of a session associated with the greatest effect was 40–60 min/session, and a frequency of at least three times/week had the largest BP lowering effect (table 2), also with 3+ level of evidence. Most of these interventions were performed on an ergometer cycle, thus making it possible to delineate the intensity of exercise.

However, other studies on specific aerobic activities showed significant results. Especially, walking has been studied in a number of RCTs; this PA shows significant results (table 2) and may perhaps be the most common form of aerobic activity studied. For example, 30 min extra walking/daily[41 43] or 3 km extra walking/day significantly reduced the BP in hypertensives.[40] Other forms of aerobic activity (water training, dance and soccer) also showed similar BP lowering effects as is defined for medium/high-intensity PA (in small RCTs), with a duration of 50–60 min and frequency of 2–3 times per week.[44–47]

Isometric (static) physical training

Regarding the effects of isometric (static) training, we identified three RCTs by considering the BP lowering effect of this training modality that included a total of 64 patients.[48–50] The mean BP lowering in these studies was 9.8/1.8 mm Hg, with 2+ level of evidence, as shown in table 3. The most common form of isometric exercise that is used in clinical practice is isometric handgrip (IHG) and isometric bilateral leg training. However, in the 3 studies,[48–50] only IHG was used. All included studies used 30% of maximal voluntary contraction, 4×2 min, performed three times/week.

Dynamic resistance training

Looking specifically at resistance training studies, we identified five RCTs with dynamic resistance training for patients with hypertension, a total of 148 patients.[21 22 51–53] No significant BP lowering effect could be seen (mean 0/0.5 mm Hg lowering), as shown in table 4. The included studies used various forms of resistance training,[21 22 51 53] including circuit training,[52] alone or in combination with aerobic training. Most studies showed non-significant or no BP lowering effect (table 4).

Mechanisms

Regular PA probably reduces BP by several possible mechanisms. Importantly, the effect of PA may be more significant in the early stages of hypertension, where the higher pressure is mainly due to an increased cardiac output and when the secondary adaptations and remodelling of the affected vessel walls is less established,[54] as described above.

Specifically, PA may reduce sympathetic activity/increase vagal tone, achieving a reduction in peripheral resistance. It is known that regular PA may reduce norepinephrine levels by about 30%,[55] and that these reductions may parallel the BP reductions.[1 24] Another BP lowering mechanism of PA is the release of vasodilating substances such as endorphins.[56] Reduced insulin resistance, secondary to PA, may also play a role.[57] The BP lowering effect could also be mediated by effects on kidney function,[58] for instance, reduction of the plasma-renin levels.[55] In addition, indirect positive effects on BP levels could also be achieved through effects on other risk factors, such as being overweight.

Hypertension in athletes

As outlined above, regular PA has been proven to have positive effects on both BP levels and the accompanying cardiac risk factors in individuals with hypertension. However, the existence of hypertension in the context of athletic activity throws specific challenges regarding treatment and eligibility for participation in elite sports.

The prevalence figures of hypertension in athletes varies greatly between studies, being 7% for male professional football (soccer) players[59] and 19% for collegiate (US) football players.[60] A recent systematic review[61] showed a prevalence of hypertension in the range of 0 to 45% for different populations, possibly attributable to various quality of methodology used in available studies. However, Berge *et al*[61] did not find any evidence for a *lower* BP in athletes, which may have been suspected. Importantly, as more and older athletes take part in various competitions, that is, master athletes, it can be expected that more athletes may have hypertension in the future. This is emphasised by the findings of the Swedish Lidingö-loppet, a 30 km cross-country race, showing that athletes have increased the running times during the years 1993–2007, which is a possible marker of decreased fitness, as well as that of being progressively older.[62] Furthermore, masked hypertension 'unmasked' by ambulatory BP recordings was common among 33% of middle-aged Swiss runners.[63] These findings emphasise the importance of including BP measurements in the cardiac screening of competitive athletes, as advocated by the European Association of Cardiac Prevention and Rehabilitation (EACPR).[64]

An association between the level of BP and increased LV mass/LV hypertrophy has been found.[61] It has also been suggested that exercise-induced cardiac adaptation may add to the adaptation secondary to hypertension, possibly having negative long-term effects.[65] There has also been shown to be an inverse association between the BP level and maximal exercise capacity (VO_2 max) in elite athletes.[66]

Table 3 RCTs with interventions of isometric resistance activity

Author	Year	Size (n)	Intervention(s) (n)	Control (n)	Sex	Activity	Intensity	Frequency	Duration (weeks)	Effect
Isometric resistance										
Badrov *et al*	2013	24	12	12	M/W	Isometric hand grip training	30% max voluntary contraction	4×2 min, 3×/w	10	SBP 8, DBP 5 mm Hg
Millar *et al*	2013	23	13	10	M/W	Isometric hand grip training	30% max voluntary contraction	4×2 min, 3×/w	8	SBP 5 mm Hg, DBP ns
Taylor *et al*	2003	17	9	8	M/W	Isometric hand grip training	30% max voluntary contraction	4×2 min, 3×/w	10	SBP 19 mm Hg, DBP ns

DBP, Diastolic blood pressure; ns, not significant; SBP, Systolic blood pressure.

Table 4 RCTs with interventions of dynamic resistance activity

Author	Year	Size (n)	Intervention(s) (n)	Control (n)	Sex	Activity	Frequency	Duration (weeks)	Effect
Dynamic resistance									
Blumenthal *et al*	1991	92	39/31	22	M/W	Walking/jogging or resistance training	35 min, 3×/w	16	ns
Cononie *et al*	1991	17	6/6	5	M/W	Walking/jogging or resistance training	35–45 min, 3×/w	26	Endurance: SBP ns, DBP 9 mmHg. Resistance: ns.
Elliot *et al*	2002	15	8	7	W	Resistance training	3×8 reps, 3×/w	8	ns
Harris *et al*	1987	26	10	16	M	Circuit weight training	3×/w	9	SBP ns, DBP 4 mm Hg
Stensvold *et al*	2010	43	11/11/10	11	M/W	Aerobic interval training, strength training or a combination	3×/w	12	ns

DBP, Diastolic blood pressure; ns, not significant; SBP, Systolic blood pressure.

The evaluation of an athlete with hypertension is outlined in box 1. Specifically, the existence of any underlying cardiovascular abnormality must be ruled out in an athlete with diagnosed hypertension. For example, an elevated BP in a young individual (athlete) must promptly lead to the exclusion of aortic coarctation, by using femoral pulse palpation as well as by measuring BP in the arms and legs. A thorough check of any use of anabolic steroids is recommended, as these have been shown to be associated with increased BP, possibly due to the sodium-retentive effects of steroids.[67]

Box 1 Evaluation of an athlete with hypertension

1. *Establishing the blood pressure (BP) levels*
   - Repeated BP-measurement, according to standards (for diagnosis)—including BP measurement in arms and legs.
2. *Identify/rule out secondary causes of hypertension and establish any target organ damage (TOD, eyes, heart, kidneys)*
   - Family history of hypertension, kidney disease and heart disease
   - Added physical examination: palpation of carotid and femoral pulses, auscultation of renal arteries and heart auscultation.
   - Laboratory tests (routine): haemaglobin, haematocrit, serum-potassium, creatinine, uric acid, plasma-glucose, serum-cholesterol, triglycerides and urine analysis.

   Recommended laboratory tests include: microalbuminuria and high-sensitive CRP.
   - Echocardiography
   - Resting-ECG
   - Maximal exercise test (competitive athletes)
   - Funduscopy
   - Further testing, when indicated by symptoms or examination-findings
3. *Risk stratification*
   - The total risk will be divided into low added risk, moderate, high or very high added risk as based on these three parameters:
   - the blood pressure level,
   - other existing risk factors,
   - the presence of TOD.
4. *Treatment*
   - Treatment according to the general guidelines for the management of hypertension (see text).
5. *Recommendations for strenuous physical activity/competitive sports*
   - Athletes with low added risk, including no TOD, with well-controlled BP, may compete in all sports.
   - For those with moderate, high or very high added risk, restrictions in sporting competition recommended does apply (Fagard 2005).

During exercise testing in athletes, for example, as part of determination of maximal aerobic capacity (Vo_2max), an exaggerated BP response may be encountered. An exaggerated BP response during exercise is associated with an increased risk of developing hypertension later in life, even when the resting BP levels are normal.[68] Thus, the finding of an exaggerated BP response should lead to regular follow-up of the BP.

The management of confirmed hypertension in athletes should follow the existing recommendations from the ESC.[2] The first step, as for all hypertensives, is to risk stratify, with the total risk of the individual being dependent not only on the actual BP level, but also on the existence of other cardiovascular risk factors and on any target organ damage (TOD).[2] Exclusion of TOD is especially important in athletes and includes opthalmological evaluation, kidney tests (creatinine, albuminuria) and ECG and echocardiography (LV hypertrophy). Regarding treatment, similar principles as that for all hypertensive patients are applied.[2] However, specific consideration must be taken regarding the choice of hypertensive medications, when these are indicated (see below).

Regarding eligibility for competitive sports, all sports are permitted for athletes with well-controlled BP (by lifestyle and/or medical treatment), who have low added risk including that no TOD can be shown.[2] In case of proven kidney, eye or heart TOD, elite activity may not be recommended.[2] In case of a BP >200/115, sporting activity is contraindicated and medical therapy should be instigated before any return to athletic activity may be reconsidered.[2]

Interaction with medical therapy

Both for the competitive athlete and the recreationally exercising individual with hypertension, careful consideration regarding appropriate medications must be taken. This is because some of the antihypertensive drugs may interact with physical performance, and thus be more or less suitable in association with exercise. This issue, however, is becoming less important as the latest and most commonly used BP medications are the least implicated substances in this regard.

However, β-blockers are still widely used in cardiovascular care, although no longer being first-line treatment for hypertension, in the absence of other cardiac disease. β-blockers are

problematic when taken in combination with exercise since these are associated with a lowering of maximal pulse during activity, up to 30 bpm.[12] The rate-pressure product is less increased during activity with β-blockers, compared to exercise using other antihypertensive medications,[69] leading to a lower maximal exercise capacity.[12] For the leisure-time active hypertensive patient, this may be of little importance. However, individuals with more demands on maximal performance (recreational or competitive) may have problems accepting β-blockers for their BP.

Diuretics are common secondary treatment for hypertension, and may lead to dehydration in exercising individuals, especially in warm temperatures, and may lead to hypokalaemia.[12]

In general, the most common medications for hypertension nowadays are one (or more) of the different vasodilating substances. Calcium channel-blockers may increase the risk of excessive vasodilation and accompanying hypotension, post exercise.[2] The ACE-inhibitors may have similar effects, especially in a warmer temperature, while effects of angiotensin-blockers (ARBs) have as yet not been studied. However, the same considerations as those recommended for ACE-inhibitors should apply. Today, the preferred antihypertensive medication used by exercising individuals, without any accompanying cardiovascular disease, is one of or a combination of the available vasodilators.[2] In addition, it is important to consider that diuretics and β-blockers (in some sports) are on WADAs list of prohibited drugs (http://www.wada-ama.org).

Follow-up

All patients with hypertension, regardless of sporting activity, should be regularly followed up. The initial total risk evaluation performed after diagnosis should be regularly updated, in addition to the regular BP check-ups. This risk evaluation should consist of controls of other cardiovascular risk factors, including the level of regular PA. In addition, any reversal of existing TOD should be assessed by regular kidney testing, as well as by ECG/echocardiography. Ambulatory 24 h BP may give additional information on the BP in the individual home setting. Adjustments of medication must be considered, as should the re-review of sports eligibility, when necessary.

The inclusion of a fitness test in the evaluation has several advantages. The individual fitness level gives predictive information[70] in addition to the regular risk score models, which normally do not include PA.[71] Awareness of the fitness level of the patient also aids in tailoring the individualised PA recommendations for treatment of hypertension. In addition, the fitness level may be used, in the follow-up of exercise recommendations, to evaluate the extent of lifestyle behavioural change achieved by the patient. Exercise recommendations may be constructed with the help of a specialised physiotherapist, especially if the patient also has a concomitant coronary artery disease. In addition to cardiovascular fitness, other functional tests evaluating strength, balance and mobility may give additional information on the health parameters through adoption of lifestyle change, especially in an older hypertensive patient.

Summary

Regular PA reduces the BP of individuals with hypertension. The present review analysed 27 randomised controlled studies on aerobic PA effects on hypertension, showing that regular medium-to-high-intensity aerobic activity may reduce the BP by 11/5 mm Hg. In addition, three RCTs on isometric (static) activity showed BP reduction of similar magnitude in hypertensives, while dynamic resistance training may show less effect, as shown in five available RCTs. This is important since the prevalence of both hypertension and physical inactivity are high and increasing in today's society. Thus, PA has a great role to play as a single or additive treatment for hypertension.

Furthermore, as athletes are getting older, it can be expected that more athletes at different competitive levels will have hypertension. Certain considerations must be applied regarding evaluation and treatment of hypertension in athletes. Eligibility for competitive sports may be affected if TOD is present. However, an athlete with well-controlled BP, having no additional risk factors or TOD, is eligible for all sports.

What are the findings?

- The novelty of the present review of the evidence for physical activity's effect on blood pressure is that only studies looking at hypertensive patients are included.
- Regular medium-to-high-intensity aerobic activity may lower the blood pressure in hypertensives by a mean of 11/5 mm Hg (grade of evidence, +++).
- Isometric (static) activity may also lower the blood pressure in the same patients to a similar degree, as based on less number of randomised controlled trials (grade of evidence, ++).
- As hypertension is closely associated with modern lifestyle, we can expect an increasing amount of hypertensive patients in the future.

How might it impact on clinical practice in the future?

- Physical activity has an important role not only in the prevention but also in the treatment of hypertension, with clinically relevant treatment effects.
- As participants at mass-gathering events are getting older and less fit, we can expect an increase of hypertension also in athletes.
- Eligibility for competitive sports may be affected if target organ damage is present; any medication has to be taken into account with regard to its effect on performance and/or doping.

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