

Blood pressure response to moderate physical activity is increased in obesity

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ABSTRACT

Objective: To test the hypothesis that in young, normotensive obese subjects, physical activity at a fixed, moderate workload, causes a more pronounced hypertensive effect than in lean subjects.

Patients and methods: 24 subjects (12 with BMI >30 kg/m², 12 with BMI <25 kg/m²), underwent a moderate-intensity physical activity protocol (cycling at 100 W). Blood pressure and oxygen consumption were monitored continuously.

Results: In the obese subjects, physical activity caused a more pronounced increase in both systolic blood pressure (increase of 40.4 ± 15.3 mmHg vs 21.2 ± 10.2 mmHg in lean subjects; p=0.001) and diastolic blood pressure (17.5 ± 17.9 mmHg vs 3.2 ± 8.1 mmHg in lean subjects; p=0.02). In regression analyses, these differences were only partly explained by small differences in resting blood pressure.

Conclusion: Healthy obese subjects show an enhanced prohypertensive response of both systolic and diastolic blood pressure to moderate-intensity physical activity.

KEYWORDS

Blood pressure, exercise, obesity

INTRODUCTION

Epidemiological studies demonstrate a close association between obesity and increased blood pressure, and hypertension is considered to be a major contributor to cardiovascular risk in obese subjects.¹ The association between the degree of obesity and blood pressure appears to be linear and extends into the non-obese range of the body mass index (BMI).² Although the strength of the association between obesity and blood pressure varies among different racial and ethnic groups,³⁻⁶ estimates obtained from

the Framingham Heart Study suggest that overweight contributes to the aetiology of 'essential' hypertension in 75% of hypertensive males and 65% of hypertensive females.⁷ Hence, a sharp increase in the incidence of hypertension can be anticipated as a consequence of the unfolding obesity pandemic. Studies have identified multiple alterations in homeostatic mechanisms acting simultaneously to increase blood pressure. The main factors appear to be increased sympathetic nervous system activity in renal and peripheral soft tissues, and increased activity of the renin-angiotensin-aldosterone system.⁸ Even in young, normotensive obese subjects, subtle prohypertensive alterations of this kind can be observed already.⁸

An understudied aspect of obesity-related hypertension is the role of physical activity. Any degree of physical activity increases (mainly systolic) blood pressure.⁹ Given that several prohypertensive homeostatic alterations are present, even in young, normotensive obese subjects, physical activity could conceivably cause a more pronounced hypertensive effect in obese subjects. From a clinical perspective, this could be relevant, as it would imply that office blood pressure under resting conditions would underestimate the daily blood pressure load in obese individuals more than in lean individuals, particularly in physically active people. In addition, a recent study demonstrated that the increase in diastolic blood pressure during low-intensity exercise is an independent predictor of incident cardiovascular disease.¹⁰ Under experimental conditions of local exercise (i.e. handgrip test), a more pronounced blood pressure increase to physical stress tests has indeed been shown in obese children¹¹ but the results of similar studies in adults are more unequivocal.^{12,13} Previous experimental studies have been limited by the fact that physical stressors often did not resemble normal daily activities, such as walking, cycling, etc. Some studies using ambulatory blood pressure measurements combined with accelerometry have

observed a higher reactivity of blood pressure to regular daytime physical activity in obese subjects.^{14,15} However, subjects in these studies were relatively old, and many were hypertensive and/or used antihypertensive medication. Also, physical activity estimated by accelerometry may be an inaccurate reflection of actual workload, particularly in subjects with marked differences in BMI who engage in both weight-bearing and non-weight-bearing activities. Given the uncertainty as to whether obesity causes an enhanced hypertensive response to 'normal' physical activity, we studied, in normotensive subjects, the influence of obesity on blood pressure response to cycling; a non-weight-bearing, everyday type of physical activity. During cycling, the level of exercise was standardised at a moderate, fixed workload of 100 W. We hypothesised that, even at this matched workload level, obesity would be associated with an enhanced blood pressure increase.

METHODS

Study subjects

Subjects were recruited by advertisement in local newspapers and from university personnel. Subjects were eligible for inclusion if they were Caucasian, between 18 and 35 years old, and if they were either obese (BMI >30 kg/m²) or normal weight (BMI <25 kg/m²). Exclusion criteria were: hypertension (systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg), a history of smoking, use of liquorice products, history or any sign of cardiac, pulmonary, endocrine or renal disease, and use of any type of medication apart from oral contraceptives. Obese and lean subjects were matched for age, sex, and daily physical activity level, which we estimated in two different ways:

1. The NASA/Johnson Space Center Physical Activity Rating (PA-R)¹⁶
2. A simple, locally developed questionnaire on physical activity during commuting and leisure time (including sports) in metabolic equivalents (MET) hours per week.

Maximum oxygen consumption (VO₂) was calculated using the N-Ex BMI model,¹⁶ in order to be able to assess whether lean and obese subjects were active at the same level relative to their estimated VO_{2max} and VO_{2max}/kg body weight. Each participant gave written informed consent. The local ethics committee approved the study.

Protocol

All subjects were asked to refrain from strenuous exercise for 24 hours prior to the study. Food intake was restricted to a light meal four hours prior to testing, which was performed between 1 and 5 pm.

After arrival in the test room, there was a 30-minute resting and acclimatisation period. Subjects then took position on

a home trainer bicycle (Lode, Groningen, the Netherlands), wearing light clothes only. The test comprised five minutes adaptation (t=-10 to t=-5), five minutes of baseline recording (t=-5 to t=0) and ten minutes of cycling (t=0 to t=10) at a workload of 100 Watt.

During the test, heart rate (HR) and continuous analysis of blood pressure were monitored using beat-to-beat finger pulse wave analysis (Finapres Medical Systems, Amsterdam, the Netherlands). The hand was held at a fixed position (arm rest bar of bicycle) relative to the level of the heart. VO₂ was monitored by breathing through a mouth piece connected to a metabolic computer (Viasys, Los Angeles, USA)

Data analysis

Baseline SBP, DBP, HR and VO₂ was defined as the mean of measurements taken during the baseline recording period (t=-5 to t=0 minutes). DBP, HR and VO₂ turned out to be relatively stable during t=1 to t=10 minutes into the cycling phase. Therefore, the mean of measured values taken during this period was calculated to assess the changes between rest and physical activity. Mann-Whitney-U tests and general linear model analysis were performed to test for differences between the two groups. Multiple regression analysis was performed to adjust observed differences in blood pressure response between BMI categories for baseline blood pressure. Analyses were done with SPSS 16.0 for windows.

RESULTS

Baseline characteristics of included subjects are summarised in *table 1*. Both groups were well matched with respect to age, gender and estimated level of physical activity. As expected, baseline blood pressure was somewhat higher in the obese group. As expected, estimated absolute VO_{2max} was higher in obese, and VO_{2max}/kg was higher in lean subjects (*table 1*).

Table 1. Baseline characteristics

	Obese (12)	Lean (12)
Sex (male/female)	5/7	5/7
Age (years)	24.1 ± 4.6	24.0 ± 1.8
BMI (kg/m ²)	34.8 ± 2.6	21.8 ± 1.6
SBP (mmHg)	128 ± 11.7	120 ± 11.9
DBP (mmHg)	77 ± 8.5	70 ± 9.1
Heart rate (beats/min)	78 ± 7.1	72 ± 9.1
Daily activity (MET hrs/week)	22.1 ± 16.8	24.6 ± 16.9
Average PA-R rating (arbitrary units)	3.5 ± 1.1	3.7 ± 1.1
Predicted VO _{2max} (l/min)	3.37 ± 1.08	2.94 ± 0.63
Predicted VO _{2max} /kg (ml/kg/min)	32.4 ± 7.8	42.5 ± 7.4

Data are presented as mean ± SD. BMI = body mass index; SBP = systolic blood pressure; DBP = diastolic blood pressure; MET = metabolic equivalents, VO₂ = oxygen consumption; VO_{2max}/kg prediction by N-Ex BMI.¹⁶

In response to physical activity at the fixed 100 W workload, the increase in SBP and DBP was significantly more pronounced in obese vs lean subjects (table 2; figures 1 and 2). In the obese, SBP tended to decrease slightly after a maximum reached during t=2 to t=4 minutes, whereas SBP recovered almost completely in lean individuals (figure 1). The increase in SBP was significantly ($p < 0.02$) more pronounced in obese vs lean subjects at all time points after t=1 min (figure 1). DBP returned to values close to baseline values after eight minutes in lean subjects, but remained elevated in the obese (figure 2). The increase in DBP was significantly ($p < 0.02$) more pronounced in obese vs lean subjects during the full exercise period (figure 2). The activity-induced changes in heart rate were similar for both groups (table 2). Absolute VO_2 during cycling was higher ($p = 0.01$) in obese subjects, in contrast to VO_2/kg which was higher ($p = 0.01$) in lean subjects (table 2). Both groups were active at a comparable level relative to their predicted VO_{2max} (table 2).

Multiple regression analysis revealed that the difference in SBP response to exercise was not explained by baseline blood pressure differences. The increased DBP response in obese subjects was only partially explained by higher baseline DBP. After adjustment for baseline DBP, the difference in DBP response between obese and lean subjects was reduced from 14.3 to 9.5 mmHg. Additional analyses showed that the differences in DBP and SBP response to exercise were not explained by differences in baseline heart rate or heart rate increase during activity.

DISCUSSION

This study demonstrates that healthy obese subjects show an enhanced response of both SBP and DBP to a fixed, moderate level of physical activity. The physiological response to moderate physical activity consists of an increase in SBP and a stable or minor decrease

Table 2. Changes in haemodynamic parameters and oxygen consumption during physical activity (t=1 to t=10 minutes) in obese versus lean subjects

	Obese	Lean	P value
Δ SBP (mmHg)	40.4 ± 15.3	21.2 ± 10.2	0.001
Δ DBP (mmHg)	17.5 ± 17.9	3.2 ± 8.1	0.02
Δ HR (beats/min)	42.8 ± 19.2	36.9 ± 25.2	0.7
Δ VO_2 (l/min)	1.3 ± 0.3	1.2 ± 0.2	0.2
Peak VO_2 during test (l/min)	1.8 ± 0.5	1.5 ± 0.2	0.01
Peak VO_2/kg during test (ml/kg/min)	17.5 ± 3	22 ± 2.5	0.01
(peak VO_2/kg during test)/(VO_{2max}/kg)	0.56 ± 0.14	0.53 ± 0.13	0.55

Data are presented as mean ±SD. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; VO_2 = oxygen consumption; VO_{2max}/kg prediction by N-Ex BMI.[16] P values by Mann-Whitney test.

Figure 1. Time trends in systolic blood pressure response to moderate physical activity in lean and obese subjects

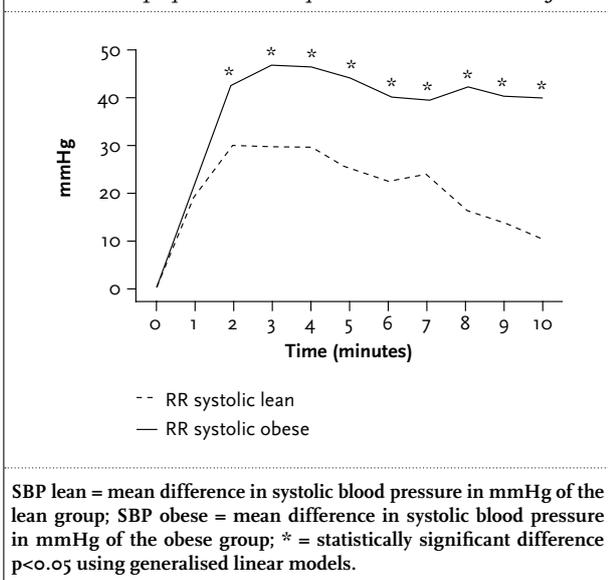
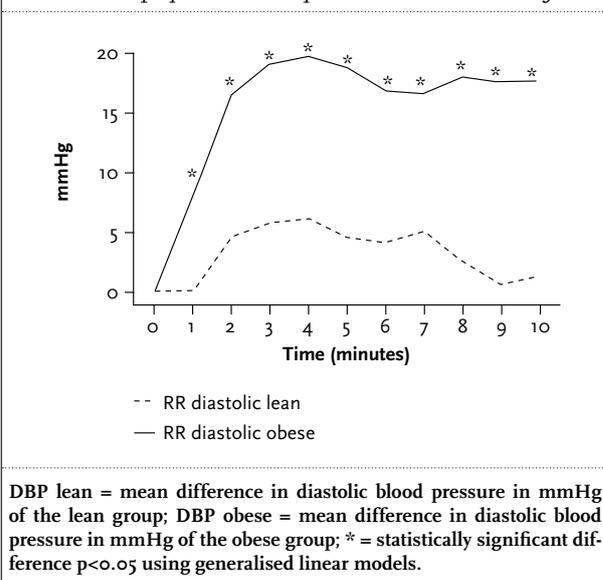


Figure 2. Time trends in diastolic blood pressure response to moderate physical activity in lean and obese subjects



in DBP.⁹ Several recent studies, using ambulatory blood pressure measurements combined with accelerometry, have shown a correlation between mean daytime blood pressure and daily physical activity level.^{14,15,17,18} Some studies have shown a more pronounced blood pressure increase to such daily activities in obese subjects,^{14,15} whereas others did not observe such a difference.¹⁸ Other factors that have been found to affect blood pressure response to physical activity include age and gender.¹⁸ However, in all these studies, the actual workload associated with physical activity was not measured, and it is possible that the more pronounced blood pressure response to physical activity as assessed by accelerometry in the obese was the result of a higher workload, for example during weight-bearing activities such as walking, climbing stairs, et cetera.

We did not study the underlying mechanism for the increased blood pressure response to exercise in obesity. As outlined in the introduction, a variety of prohypertensive alterations in blood pressure homeostasis are found in obese people, even when they are normotensive. Increased sympathetic nervous system activity and activation of the renin-angiotensin-aldosterone axis are amongst the mechanisms that act together to increase peripheral vascular resistance and promote sodium retention. Increased muscle neurovascular sympathetic tone has in fact been demonstrated to impair the physiological exercise-induced decrease in peripheral vascular resistance in obese children and adults.^{11,12} In addition to the disturbed neurovascular response to exercise, increased central arterial stiffness associated with obesity may play a role in the blood pressure response to physical activity.¹⁹ Previous studies suggested that increased cardiac output is unlikely to contribute to the difference in blood pressure response to physical and mental stressors²¹ although we did not find any studies actually measuring cardiac output in this context. It is unlikely that our findings would be explained by obese subjects being physically active at a higher relative level compared with the lean controls. Firstly we matched subjects for daily physical activity level, thus avoiding selection of inactive obese individuals. Secondly, the heart rate increase between both groups was similar. Finally, with respect to oxygen uptake, it is well established that VO_{2max} increases with BMI, but VO_{2max} /kg body weight is lower in obesity.²⁰ In our study, oxygen consumption relative to calculated maximum oxygen uptake was virtually identical in both groups.

Our study is limited by the relatively small number of included subjects, and thus requires confirmation in a larger study. Such studies should also focus on the possible role of the degree of obesity and the obesity phenotype (e.g. central vs peripheral obesity). In addition we have not physically determined VO_{2max} , but instead used a prediction model, which may be less precise. However, as outlined above, several additional arguments suggest that

exercise level was similar in both study groups. Finally, the increase in heart rate during exercise may increase finger blood pressure disproportionately, causing overestimation of the systemic blood pressure increase.²¹ However, this phenomenon has not been reported to be more outspoken in obese subjects, and mainly applies to systolic blood pressure, whereas diastolic blood pressure also increased more markedly in obesity. Statistical adjustment for heart rate differences did not affect our findings. Nonetheless, a definitive study using intra-arterial blood pressure measurement would reduce any theoretical element of confounding by the measurement technique.

Finally, although blood pressure changes were directionally similar in most lean and obese individuals, the degree of interindividual variation is substantial (see standard deviations *table 2*), precluding generalisation of our findings to all obese individuals.

CONCLUSIONS

Our findings have potential relevance for how we should interpret blood pressure measurements in obese subjects. An enhanced blood pressure increase in response to moderate physical activity in obese subjects would imply that resting blood pressure underestimates the daily blood pressure load more in obese than it does in lean people, particularly in circumstances of a high daily physical activity level. In such patients, ambulatory blood pressure monitoring during periods of daily physical activity may provide information relevant for cardiovascular risk stratification. This is supported by a recent study identifying diastolic blood pressure during low-intensity exercise as an independent predictor for incident cardiovascular disease.¹⁰ As weight loss is an effective means of resting blood pressure reduction²² and is associated with improvement of sympathetic muscle nerve overactivity,^{11,13} weight loss may well normalise the enhanced blood pressure response to physical activity in obese individuals.

REFERENCES

1. MacMahon S, Cutler J, Brittain E, Higgins M. Obesity and hypertension: epidemiological and clinical issues. *Eur Heart J*. 1987;8:57-70.
2. Jones DW, Kim JS, Andrew ME, Kim SJ, Hong YP. Body mass index and blood pressure in Korean men and women: the Korean National Blood Pressure Survey. *J Hypertens*. 1994;12:1433-7.
3. Doll S, Paccaud F, Bovet P, Burnier M, Wietlisbach V. Body mass index, abdominal adiposity and blood pressure: consistency of their association across developing and developed countries. *Int J Obes Relat Metab Disord*. 2002;26:48-57.
4. Dyer AR, Liu K, Walsh M, Kiefe C, Jacobs DR Jr, Bild DE. Ten-year incidence of elevated blood pressure and its predictors: the CARDIA Study Coronary Artery Risk Development in (Young) Adults. *J Hum Hypertens*. 1999;13:13-21.

5. Juhaeri Stevens J, Chambless LE, Tyroler HA, et al. Associations between weight gain and incident hypertension in a bi-ethnic cohort: the Atherosclerosis Risk in Communities Study. *Int J Obes Relat Metab Disord.* 2002;26:58-64
6. Okosun IS, Chandra KM, Choi S, Christman J, Dever GE, Prewitt TE. Hypertension and type 2 diabetes comorbidity in adults in the United States: risk of overall and regional adiposity. *Obes Res.* 2001;9:1-9.
7. Garrison RJ, Kannel WB, Stokes J 3rd, Castelli WP. Incidence and precursors of hypertension in young adults: the Framingham Off-spring Study. *Prev Med.* 1987;16:235-51.
8. Montani JP, Antic V, Yang Z, Dulloo A, Pathways from obesity to hypertension: from the perspective of a vicious triangle. *Int J Obes.* 2002;26(Suppl.2):S28-38.
9. Crawford MH, DiMarco JP, Walter JP. *Cardiology.* 2nd ed. Edinburgh, UK: Mosby; 2004.
10. Lewis GD, Gona P, Larson MG, et al. Exercise blood pressure and the risk of incident cardiovascular disease. *Am J Cardiol.* 2008;101:1614-20.
11. Ribeiro MM, Silva AG, Santos NS, et al. Diet and exercise training restore blood pressure and vasodilatory responses during physiological manoeuvres in obese children. *Circulation.* 2005;111:1915-23.
12. Kuniyoshi FH, Trombetta IC, Batalha LT, et al. Abnormal neurovascular control during sympathoexcitation in obesity. *Obesity Res.* 2003;11:1411-9.
13. Trombetta IC, Batalha LT, Rondon MU, et al. Weight loss improves neurovascular and muscle metaboreflex control in obesity *Am J Physiol.* 2003;285:H974-82.
14. Leary AC, Donnan PT, MacDonald TM, Murphy MB. The influence of physical activity on the variability of ambulatory blood pressure. *Am J Hypertens.* 2000;13:1067-73.
15. Cavelaars MN, Tulen JHM, van Bommel JH, ter Borg MJ, Mulder PGH, van den Meiracker AH. Determinants of ambulatory blood pressure response to physical activity. *J Hypertens.* 2002;20:2009-15.
16. Jackson AS, Blair SN, Mahar MT, Wier LT, Ross RM, Stuteville JE. Prediction of functional aerobic capacity without exercise testing. *Med Sci Sports Exerc.* 1990;22:863-70.
17. Stewart MJ, Brown H, Padfield PL. Can simultaneous ambulatory blood pressure and activity monitoring improve the definition of blood pressure? *Am J Hypertens.* 1993;6:S174-8.
18. Cavelaars M, Tulen JH, van Bommel JH, Mulder PG, van den Meiracker AH. Haemodynamic responses to physical activity and body posture during everyday life. *J Hypertens.* 2004;22:89-96.
19. Tounian P, Aggoun Y, Dubern B, et al. Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: a prospective study. *Lancet.* 2001;358:1400-4.
20. Goran M, Fields DA, Hunter GR, Herd SL, Weinsier RL. Total body fat does not influence maximal aerobic capacity. *Int J Obes Relat Metab Disord.* 2000;24:841-8.
21. Bos WJ, van den Meiracker AH, Wesseling KH, Schalekamp MA Effect of regional and systemic changes in vasomotor tone on finger pressure Amplification. *Hypertension.* 1995;26:315-20.
22. Mulrow CD, Chiquette E, Angel L, Summerbell C, Anagnostelis B, Grimm R. Dieting to reduce body weight for controlling hypertension in adults. *Cochrane Database Syst Rev.* 2000;(2):CD000484