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Influence of population and exercise protocol characteristics on hemodynamic determinants of post-aerobic exercise hypotension

L.C. Brito, A.C.C. Queiroz and C.L.M. Forjaz

Laboratório de Hemodinâmica da Atividade Motora, Departamento de Biodinâmica do Movimento do Corpo Humano, Escola de Educação Física e Esporte, Universidade de São Paulo, São Paulo, SP, Brasil

Abstract

Due to differences in study populations and protocols, the hemodynamic determinants of post-aerobic exercise hypotension (PAEH) are controversial. This review analyzed the factors that might influence PAEH hemodynamic determinants, through a search on PubMed using the following key words: ''postexercise'' or ''post-exercise'' combined with ''hypotension'', ''blood pressure'', ''cardiac output'', and ''peripheral vascular resistance'', and ''aerobic exercise'' combined only with ''blood pressure''. Forty-seven studies were selected, and the following characteristics were analyzed: age, gender, training status, body mass index status, blood pressure status, exercise intensity, duration and mode (continuous or interval), time of day, and recovery position. Data analysis showed that *1*) most postexercise hypotension cases are due to a reduction in systemic vascular resistance; *2*) age, body mass index, and blood pressure status influence postexercise hemodynamics, favoring cardiac output decrease in elderly, overweight, and hypertensive subjects; *3*) gender and training status do not have an isolated influence; *4*) exercise duration, intensity, and mode also do not affect postexercise hemodynamics; *5*) time of day might have an influence, but more data are needed; and *6*) recovery in the supine position facilitates systemic vascular resistance decrease. In conclusion, many factors may influence postexercise hypotension hemodynamics, and future studies should directly address these specific influences because different combinations may explain the observed variability in postexercise hemodynamic studies.

Key words: Blood pressure; Exercise; Cardiac output; Peripheral vascular resistance

Introduction

Physical exercise, especially aerobic exercise, is recommended for reducing blood pressure (BP). A recent review (1) reported that a period of aerobic training (chronic effect) reduces systolic/diastolic BP in normotensive and hypertensive individuals, with greater reductions observed in hypertensive individuals (–1/–2 and –8/–5 mmHg, respectively). In addition, a significant reduction in BP has also been reported after a single session of aerobic exercise (acute affect) in normotensive and hypertensive individuals (–2/–3 and –9/–9 mmHg, respectively) (2), and this acute hypotensive effect has been called post-aerobic exercise hypotension (PAEH) (3).

PAEH is defined as a reduction in BP after a session of aerobic exercise in comparison with BP measured before exercise and/or measured on a control day without exercise (3). PAEH has been demonstrated as clinically relevant because it is significant in magnitude and is sustained for a prolonged period after exercise (3). Moreover, it has been reported in different populations, including individuals who are hypertensive (4-9), prehypertensive (10,11), and normotensive (5,12-30).

Many studies have investigated the hemodynamic determinants of PAEH. Some of them attribute PAEH to reduced cardiac output (CO) (6,9,12,15,19,21-24,30-34), while others report a peripheral vascular resistance (PVR) reduction (4,7,16-18,20-23,25-29,33-53). This controversy suggests that some other factors could influence these determinants.

The characteristics of the study population and protocols influence the magnitude and duration of PAEH (5,15,25). Therefore, these characteristics may also influence the hemodynamic determinants of PAEH, but

Correspondence: C.L.M. Forjaz, Av. Prof. Mello Moraes, 65, 05508-030 São Paulo, SP, Brasil. Fax: +55-11-3813-5921. E-mail: cforjaz@usp.br

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this question has not been systematically studied. A better understanding of these influences may provide a basis for a more effective exercise prescription aimed at BP reduction after exercise and may also facilitate comparisons among studies in the literature, thereby improving scientific knowledge in this field.

The purpose of this study was to analyze the existing data about the hemodynamic determinants of PAEH by investigating the possible influence of the characteristics of the population and the study protocol on these determinants.

Material and Methods

A literature search was performed for studies that measured CO and PVR after a session of aerobic exercise that produced PAEH. The search was conducted on PubMed with the following terms combined: ''postexercise'' or ''post-exercise'' combined with ''hypotension'', ''blood pressure'', ''cardiac output'', and ''peripheral vascular resistance'', and ''aerobic exercise'' combined only with ''blood pressure''. Studies published in English until February 2013 were included.

The characteristics of the population and protocols were extracted from the studies included in this review. With regard to the population, the following characteristics were analyzed: age (young: 18-39 years, middle-aged: 40-60 years, and elderly: >60 years old) (54); sex (male and female); training status (sedentary and trained in accordance with the authors' report); body mass index (normal BMI $\leq$25 kg/m^2 and excess body weight BMI $\geq$25 kg/m^2), and BP status (systolic/diastolic BP: normotensive <120/80 mmHg, prehypertensive 120 to 139 and/or 80 to 89 mmHg, and hypertensive $\geq$140/90 mmHg) (55). Regarding the experimental protocol, the following characteristics were analyzed: exercise duration (short: <30 min, medium: $\geq$30 min and <60 min, and long: $\geq$60 min), intensity (light: <50%, moderate: $\geq$50% and <80%, high: $\geq$80% VO_2peak), mode (continuous and interval), time of day when the exercise was performed (morning and/or afternoon), and body position adopted during measurements (sitting or supine). Two independent investigators performed the search and obtained the information from the studies. Disagreements were solved by consensus.

A descriptive analysis of the hemodynamic determinants (CO and PVR) of PAEH cited in the studies was conducted according to the different influences. In addition, a frequency comparison of the occurrence of CO and PVR reduction after exercise within the studies was performed for each influence using a chi-square test, in which $P \leq 0.05$.

Results

A total of 1216 studies were selected from the literature search. Of these, 439 did not investigate aerobic exercise, 274 did not investigate the acute effects of exercise, 421 did not measure hemodynamic determinants, and 39 did not observe PAEH. Thus, 43 studies reported the hemodynamic determinants of PAEH after aerobic exercise and were subsequently included in this review. Moreover, four studies were cited in references.

The main characteristics of the 47 studies are shown in Table 1 (studies with young subjects) and Table 2 (studies with middle-aged and elderly subjects). Table 3 shows the frequency of reduction of each hemodynamic determinant (CO and PVR) with regard to each studied factor of influence.

The 47 studies included in the analysis comprise 93 different experimental cases. PVR decreased after exercise in 64 (69%) cases, while CO decreased in 14 (15%) cases. In the other cases, none of the hemodynamic determinants decreased significantly.

Population characteristics

In terms of age, PVR decreased in 74% of cases involving young subjects; in middle-aged subjects, PVR decreased in 62% of cases; and PVR did not decrease in the elderly. However, CO reduced in 75% of cases for this group. The frequency of CO and PVR reduction was influenced by age ($P = 0.00$).

When considering sex, a reduction in PVR occurred in 89% of female cases and 60% of male cases, without any significant difference between genders.

When analyzing training status, PVR decreased in 62% and 69% of cases involving sedentary and trained subjects, respectively. These frequencies were not significantly different.

For subjects with normal BMI, PVR decreased in 75% of cases, while CO decreased in 13%. For subjects with excess body weight, PVR decreased in 44% of cases, whereas CO decreased in 28%. The frequencies of CO and PVR reduction were significantly different in terms of BMI status ($P = 0.05$).

Regarding BP status, PVR and CO decreased in 72% and 13% of normotensive subjects, respectively. For those who were prehypertensive, PVR decreased in 33% of cases, but CO did not change at any time. On the other hand, for those who were hypertensive, PVR decreased in 58% of cases, while CO was reduced in 42%. As such, there was a significant difference in the frequencies related to BP status ($P = 0.00$).

Exercise protocol characteristics

There was no significant difference between the frequencies of PVR and CO reductions after exercise in relation to exercise duration, intensity, or mode. PVR reduction was observed in 73%, 59%, and 80% of cases with short, medium, and long exercise durations, respectively. After light-, moderate-, and high-intensity exercises, decreased PVR was observed in 100%, 65%, and 77% of cases, respectively. Finally, a PVR decrease was reported in 70% of cases with continuous exercise and in

Table 1. Characteristics of the population and experimental protocol of the studies with young subjects that evaluated cardiac output (CO) and peripheral vascular resistance (PVR) after an aerobic exercise session that produced post-exercise hypotension.

Author	Age (years)	n-Sex	Train	BMI	BP	Duration	Intensity	Mod	Time	Pos	PVR	CO
Journeay (48)	24 ± 2	10F	T	N	NT	15 min (Sh)	70%VO_2peak (Mo)	C	–	Si	↓	↑
Journeay (47)	24 ± 2	10M	T	N	NT	15 min (Sh)	70%VO_2peak (Mo)	C	–	Si	↓	↑
Endo (30)	20-31	10M	SD	N	NT	60 min (Lo)	60%HRreserv (Mo)	C	–	Su	↑	↓
Lynn (19)	26 ± 7	14M	T	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	Mor	Su	↔	↓
Lockwood (17)	24 ± 4	11M	T	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	–	Su	↓	↔
Dujic (12)	22 ± 2	20M	T	N	NT	T.max (Sh)	T.Max (H)	C	Mor	Si	↔	↓
Teixeira (24)	26 ± 1	12M/11F	SD	N	NT	30 min (Me)	75%VO_2peak (Mo)	C	Af	Si	↑	↓
Halliwill (27)	21-28	7M/5F	SD	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	–	Su	↓	↑
Lockwood (18)	22 ± 2	7M/7F	T	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	–	Su	↓	↔
Halliwill (26)	22-27	5M/4F	T	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	–	Su	↓	↑
Piepoli (36)	31 ± 2	9M/1F	T	–	NT	T.max (Sh)	Max T (H)	C	–	Su	↓	↑
Piepoli (38)	24-34	4M/4F	SD	–	NT	T.max (Sh)	Max T (H)	C	–	Su	↓	↑
Isea (44)	Young	6M	–	–	NT	T.max (Sh)	Max T (H)	C	–	–	↓	↑
Halliwill (29)	21-33	4M/4F	–	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	–	Su	↓	↔
Rossow (56)	25 ± 3	10F	T	N	NT	60 min (Lo)	60%HRreserv (Mo)	C	–	Su	↓ ↓	↑
	25 ± 3	10F	T	N	NT	25 min (Sh)	100:60%VO_2peak (H)	I	–	Su	↓	↑ ↑
	26 ± 7	15M	T	N	NT	60 min (Lo)	60%HRreserv (Mo)	C	–	Su	↓	↑
	26 ± 7	15M	T	N	NT	25 min (Sh)	Max:60% (Mo)	I	–	Su	↓ ↓	↑ ↑
Journeay (51)	21 ± 1	7F	T	N	NT	15 min (Sh)	75%VO_2peak (Mo)	C	–	Si	↓ ↓	↔
	20 ± 1	7M	T	N	NT	15 min (Sh)	75%VO_2peak (Mo)	C	–	Si	↓	↔
Lynn (42)	24 ± 4	14F fol	SD	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	Mor	Su	↓	↑
	24 ± 4	14F ovu	SD	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	Mor	Su	↓	↑
	24 ± 4	14F lut	SD	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	Mor	Su	↓	↑
	23 ± 4	14M	SD	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	Mor	Su	↓	↑
Carter (34)	30 ± 2	8M	SD	N	NT	3 min (Sh)	60%HRmax (Mo)	C	Af	Si	↓	↔
	28 ± 3	11F	SD	N	NT	3 min (Sh)	60%HRmax (Mo)	C	Af	Si	↔	↓
McCord (50)	24 ± 5	7M	SD	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	Mor	Su	↓	↔
	22 ± 3	7M	T	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	Mor	Su	↑	↓
	26 ± 5	7F	SD	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	Mor	Su	↓	↔
	23 ± 3	7F	T	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	Mor	Su	↔	↔
Senitko (23)	24 ± 5	8M	SD	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	–	Si	↓	↑
	25 ± 5	8F	SD	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	–	Si	↓	↑
	26 ± 4	8F	T	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	–	Si	↓	↑
	28 ± 3	8M	T	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	–	Si	↔	↓
Izdebska (45)	26 ± 4	8M	–	OW	HT	20 min (Sh)	55%HRreserv (Mo)	C	–	Si	↓	↑
	25 ± 3	9M	–	OW	NT	20 min (Sh)	55%HRreserv (Mo)	C	–	Si	↔	↔
Hamer (15)	20 ± 2	6M	T	OW	NT	20 min (Sh)	75%HRreserv (Mo)	C	–	Su	↔	↓
	20 ± 1	10F	T	N	NT	20 min (Sh)	75%HRreserv (Mo)	C	–	Su	↓	↑

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Table 1. Continued.

Author	Age (years)	n-Sex	Train	BMI	BP	Duration	Intensity	Mod	Time	Pos	PVR	CO
Raine (21)	24 ± 1	7M	–	N	NT	T.max (Sh)	Max T (H)	C	–	Si	↓	↑
	24 ± 1	7M	–	N	NT	T.max (Sh)	Max T (H)	C	–	Su	↓	↑ ↑
Takahashi (33)	22 ± 2	7M	SD	N	NT	5 min (Sh)	80%VO_2peak (H)	C	–	Si	↑	↓
	22 ± 2	7M	SD	N	NT	5 min (Sh)	80%VO_2peak (H)	C	–	Su	↓	↑
Jones (53)	31 ± 7	6M	T	N	NT	30 min (Me)	60%VO_2peak (Mo)	C	Mor4h	Si	↔	↔
	31 ± 7	6M	T	N	NT	30 min (Me)	60%VO_2peak (Mo)	C	Mor6h	Si	↔	↔
	31 ± 7	6M	T	N	NT	30 min (Me)	60%VO_2peak (Mo)	C	Mor8h	Si	↔	↔
	31 ± 7	6M	T	N	NT	30 min (Me)	60%VO_2peak (Mo)	C	Mor10h	Si	↔	↔
	31 ± 7	6M	T	N	NT	30 min (Me)	60%VO_2peak (Mo)	C	Af4h	Si	↓	↔
	31 ± 7	6M	T	N	NT	30 min (Me)	60%VO_2peak (Mo)	C	Af6h	Si	↓	↔
	31 ± 7	6M	T	N	NT	30 min (Me)	60%VO_2peak (Mo)	C	Af8h	Si	↓	↔
	31 ± 7	6M	T	N	NT	30 min (Me)	60%VO_2peak (Mo)	C	Af 0h	Si	↔	↔
Jones (52)	26 ± 5	12M	T	N	NT	30 min (Me)	70%VO_2peak (Mo)	C	Mor	Si	↑	↑
	26 ± 5	12M	T	N	NT	30 min (Me)	70%VO_2peak (Mo)	C	Af	Si	↓	↑
Scott (22)	29 ± 2	10M	T	N	NT	60 min (Lo)	120:40%VO_2peak (H)	I	–	Su	↓	↑
	29 ± 2	10M	T	N	NT	50 min (Me)	10%<RCP (Mo)	C	–	Su	↓	↑
Jones (16)	28 ± 6	7M	T	N	NT	30 min (Me)	70%VO_2peak (Mo)	C	Mor	Si	↓	↑
	28 ± 6	7M	T	N	NT	30 min (Me)	40%VO_2peak (Li)	C	Mor	Si	↓	↑
	28 ± 6	7M	T	N	NT	50 min (Me)	40%VO_2peak (Li)	C	Mor	Si	↓	↑
Birch (46)	20 ± 1	15F cont	T	–	NT	30 min (Me)	60%VO_2max (Mo)	C	Mor	Su	↓	↑
	20 ± 1	15F wit	T	–	NT	30 min (Me)	60%VO_2max (Mo)	C	Mor	Su	↓	↑
Forjaz (28)	24 ± 1	23M/F	SD	N	NT	45 min (Me)	30%VO_2peak (Li)	C	Mor	Si	↓	↑
	24 ± 1	23M/F	SD	N	NT	45 min (Me)	50%VO_2peak (Mo)	C	Mor	Si	↓	↑
	24 ± 1	23M/F	SD	N	NT	45 min (Me)	75%VO_2peak (Mo)	C	Mor	Si	↓	↑
Floras (31)	Young	5M	–	–	HT	45 min (Me)	70%HRreserv (Mo)	C	–	–	↔	↓
	Young	5M	–	–	NT	45 min (Me)	70%HRreserv (Mo)	C	–	–	↔	↔
Esformes (49)	20 ± 1	8F fol	–	N	NT	30 min (Me)	80%T/lac (Mo)	C	–	Su	↓	↔
	20 ± 1	8F ovu	–	N	NT	30 min (Me)	80%T/lac (Mo)	C	–	Su	↓	↔
	20 ± 1	8F lut	–	N	NT	30 min (Me)	80%T/lac (Mo)	C	–	Su	↓	↔
McCord (20)	24 ± 3	5M/5F	SD	N	NT	60 min (Lo)	60%VO_2peak (Mo)	C	–	Su	↓ ↓	↑
Casiglia (37)	26 ± 6	10M	T	–	HT	61 ± 6 min (Lo)	Tan (Mo)	C	–	Si	↓	↔
	24 ± 6	8M	T	–	NT	61 ± 6 min (Lo)	Tan (Mo)	C	–	Si	↓	↔
Pescatello (40)	38 ± 2	7F	–	OW	HT	30 min (Me)	60%VO_2max (Mo)	C	Mor	Si	↓	↑
	34 ± 2	11F	–	OW	NT	30 min (Me)	60%VO_2max (Mo)	C	Mor	Si	↓	↑

M: males; F: females; fol: follicular phase; ovu: ovulatory follicular; lut: lutea mean phase; cont: contraceptive use; wit: without contraceptive; train: training status; Mod: mode; SD: sedentary; T: trained; BMI: body mass index status; N: normal; OW: overweight; BP: blood pressure status; NT: normotensive; HT: hypertensive; min: minute; Sh: short; Me: medium, Lo: long; Li: light; Mo: moderate; H: High; Max t: maximal test; Tan: anaerobic threshold; T/lac: lactate threshold; RCP: respiratory compensate point; HRmax: maximal heart rate; C: continuous; I: interval; Pos: position; Mor: morning; Af: afternoon; Si: sitting; Su: supine; ↓: decreased; ↑: increased; ↔: no change.

Table 2. Characteristics of the population and experimental protocol of the studies with middle-aged and elderly subjects that evaluated cardiac output (CO) and peripheral vascular resistance (PVR) after an aerobic exercise session that produced post-exercise hypotension.

Author	Age (years)	n-Sex	Train	BMI	BP	Duration	Intensity	Mod	Time	Pos	PVR	CO
Harvey (25)	54 ± 2	13F	–	OW	NT	45 min (Me)	60%HRmax (Mo)	C	Mor	Su	↓	↑
	28 ± 1	14F	–	N	NT	45 min (Me)	60%HRmax (Mo)	C	Mor	Su	↓	↑
Figueroa (41)	50 ± 1	8F	–	OW	NT/HT	20 min (Sh)	65%VO_2peak (Mo)	C	–	Su	↓	↔
	48 ± 2	12F	–	O	NT/HT	20 min (Sh)	65%VO_2peak (Mo)	C	–	Su	↓	↔
	49 ± 3	8F	–	N	NT/HT	20 min (Sh)	65%VO_2peak (Mo)	C	–	Su	↓ ↓	↔
Headley (39)	40 ± 7	10F bl	–	OW	Pre-HT	40 min (Me)	50%VO_2peak (Mo)	C	Mor	Si	↑	↔
	47 ± 11	10F wh	–	OW	Pre-HT	40 min (Me)	50%VO_2peak (Mo)	C	Mor	Si	↓	↔
Cleroux (4)	44 ± 2	11M/2F	–	N	HT	30 min (Me)	50%VO_2peak (Mo)	C	–	Su	↓	↑
	41 ± 2	7M/2F	–	N	NT	30 min (Me)	50%VO_2peak (Mo)	C	–	Su	↔	↑
Beaulieu (35)	43 ± 2	10M med	–	–	HT	30 min (Me)	50%VO_2peak (Mo)	C	Mor	Su	↓ ↓	↔
	43 ± 2	10M pla	–	–	HT	30 min (Me)	50%VO_2peak (Mo)	C	Mor	Su	↓	↔
New (43)	50 ± 8	9M	SD	OW	Pre-HT	30 min (Me)	75%VO_2peak (Mo)	C	–	Si	↓	↑
Lacombe (10)	57 ± 4	13M	SD	OW	Pre-HT	21 min (Sh)	60%VO_2peak (Mo)	C	Af	Si	↔	↔
	57 ± 4	13M	SD	OW	Pre-HT	20 min (Sh)	85:40% VO_2peak (H)	I	Af	Si	↔	↔
Liu (11)	45-60	8M/9F	SD	–	Pre-HT	30 min (Me)	65%VO_2peak (Mo)	C	Af	Si	↔	↔
Legramante (7)	49 ± 5	12M/3F	SD	N	HT	T.max (Sh)	Max T(H)	C	–	Su	↓	↔
Rueckert (32)	50 ± 2	1M/5F	–	OW	HT	45 min (Me)	60%HRreserv(Mo)	C	Mor	Si	↑	↓
Brandão Rondon (9)	68 ± 1	15M/9F	SD	OW	HT	45 min (Me)	50%VO_2peak (Mo)	C	Mor	Su	↔	↓
	68 ± 2	9M/9F	SD	OW	NT	45 min (Me)	50%VO_2peak (Mo)	C	Mor	Su	↔	↔
Hagberg (6)	64 ± 2	5M/4F	SD	OW	HT	45 min (Me)	50%VO_2max (Mo)	I	Mor	Si	↑	↓
	64 ± 3	7M/2F	SD	OW	HT	45 min (Me)	70%VO_2max (Mo)	I	Mor	Si	↑	↓

M: males; F: females; bl: black; wh: white; med: medicated; pla: placebo; SD: sedentary; BMI: body mass index; N: normal; OW: overweight; O: obesity; BP: blood pressure; NT: normotensive; HT: hypertensive; Pre-HT: pre-hypertensive; min: minute; Sh: short; Mod: mode; Me: medium; Mo: moderate; H: high; Max T: maximal test; C: continuous; I: interval; Mor: morning; Af: afternoon; Pos: position; Si: sitting; Su: supine; CO: cardiac output; SVR: systemic vascular resistance; ↓: decreased; ↑: increased; ↔: no change.

Table 3. Frequency of occurrence of cardiac output (CO) and peripheral vascular resistance (PVR) reduction after aerobic exercise in the different experimental conditions reported in the studies included in this review concerning the possible factors of influence.

	Conditions	↓ PVR	↓ CO	↔	↓ PVR (%)	↓ CO (%)	Chi-square	P
Age								
Young	73	54	10	9	74.0	13.4	16.71	0.00*
Middle-aged	16	10	1	5	62.5	6.3		
Elderly	4	0	3	1	0	75.0		
Sex								
Female	27	24	1	2	88.9	3.7	8.19	0.09
Male	45	27	8	10	60.0	17.8		
Training status								
Sedentary	29	18	7	4	62.1	24.1	1.51	0.47
Trained	39	27	5	7	69.2	12.8		
Body mass index status								
Normal	63	47	8	8	74.6	12.7	5.84	0.05*
Overweight	18	8	5	5	44.4	27.8		
Blood pressure status								
NT	72	52	9	11	72.2	12.5	19.13	0.00*
Pre-HT	6	2	0	4	33.3	0.0		
HT	12	7	5	0	58.3	41.7		
Exercise duration								
Short	26	19	4	3	73.1	15.4	6.34	0.18
Medium	42	25	6	11	59.5	14.3		
Long	25	20	4	1	80.0	16.0		
Exercise intensity								
Light	6	6	0	0	100.0	0.0	4.01	0.41
Moderate	74	48	12	14	64.9	16.2		
High	13	10	2	1	76.9	15.4		
Exercise mode								
Continuous	87	61	12	14	70.1	13.8	1.76	0.42
Interval	6	3	2	1	50.0	33.3		
Time of day								
Morning	36	21	7	8	58.3	19.4	0.92	0.63
Afternoon	11	5	2	4	45.5	18.2		
Position								
Sitting	45	26	8	11	57.8	18.2	7.18	0.03*
Supine	45	37	5	3	82.2	11.1		

NT: normotensive; Pre-HT: pre-hypertensive; HT: hypertensive; ↓: decreased; ↔: no change. *$P \leq 0.05$ (chi-square test).

50% of cases with interval exercise.

When exercise was performed in the morning and the afternoon, a PVR decrease was reported in 58% and 45% of cases, respectively, which are similar.

In regard to body position adopted at the time period of the measurement, PVR reduction was observed in 82% and 58% of cases of supine and sitting recovery, respectively, while CO decrease was reported in 18% and 11% of cases. These frequencies of occurrence were significantly different ($P = 0.03$).

Discussion

This review confirms the disseminated conception that, in most cases, PAEH is related to a reduction in PVR. However, this review also revealed that CO reduction was responsible for PAEH under many circumstances. Some factors related to the characteristics of the study protocols and population may influence the hemodynamic determinants of PAEH. In this sense, the current results suggest that a CO reduction after exercise is facilitated when elderly, hypertensive, and overweight subjects were studied and when recovery was performed in the sitting position.

Population characteristics

PAEH occurs at all ages, as confirmed by studies of individuals who are young (11,12,15-24,26-31,33-37,

40-42,45-53,56), middle-aged (4,7,10,25,32,39), and elderly (6,9). Aging leads to structural alterations in the cardiovascular system (57) that can influence PAEH hemodynamic determinants. The present findings suggest that postexercise PVR reduction occurs in young and middle-aged people (4,7,17,18,20,22,25-29,39-41,45,46, 48,49,51), while a reduction in CO is more evident in those who are elderly (6,9). We were only able to find one study that directly compared postexercise hemodynamics in different age groups (25). This study reported a reduction in PVR after exercise in young and middle-aged females (as observed in this review), but it did not assess elderly subjects (25). It is possible that an increase in arterial stiffness and PVR, as well as a decrease in endothelial function associated with aging (57), hinders postexercise PVR reduction after exercise, facilitating stroke volume and CO reduction in the elderly. Nevertheless, it is interesting to note that some studies reported postexercise CO reductions in young and middle-aged individuals (12,19,24,30-32), which suggests that other factors besides age also influence PAEH hemodynamic determinants.

At rest, females have lower PVR than males (58). Besides this difference, the results of this review did not suggest an influence of sex on post-aerobic exercise hemodynamics, although a P value of 0.09 was observed for the chi-square test. We identified five studies that directly compared sex effects on PAEH hemodynamic determinants. From those studies, two did not identify any difference between sexes (42,56), and two observed a CO reduction in males and a PVR reduction in females; however, these differences were only observed in trained subjects (23,50). The fifth study reported another pattern: a reduction in CO and PVR in females and males, respectively (34). Thus, the possible gender influence on CO and PVR behavior after exercise deserves further investigation.

Among other adaptations, aerobic training improves endothelial function, decreases PVR, and increases plasma volume (59). All of these changes may influence PAEH hemodynamic determinants. However, this review did not identify any influence related to training status *per se*. In fact, studies that directly compared postexercise CO and PVR responses in trained and sedentary individuals suggest that the influence of training status on PAEH hemodynamics may be gender dependent. These studies observed that training status did not affect female PAEH determinants, but CO decreased in trained males and PVR decreased in sedentary males after aerobic exercise (23,50). As in this review, training status and sex were independently considered, and their combined influence was not assessed.

Excess body weight is associated with cardiovascular alterations (60,61), which might affect PAEH hemodynamic determinants. In this review, although PVR reduction was the predominant determinant of PAEH in both normal subjects and individuals with excess weight, in the second group of subjects, CO reduction after exercise was observed in many cases (28%). Studies that directly compared postexercise hemodynamics in individuals with excess body weight and their normal weight peers reported controversial results. One study reported a CO reduction in overweight subjects and a PVR reduction in normal-weight subjects (15). Other studies reported PVR reductions in both groups (25,41). It is important to highlight that when overweight subjects were compared with obese subjects, PVR decreased for both (41). Thus, the effect of BMI status on PAEH determinants is not yet clear, but the presence of excess body weight might facilitate CO reduction.

Hypertension also promotes cardiovascular alterations, such as arterial stiffness, PVR increase, and endothelial dysfunction (57), which may pose a difficulty to postexercise vasodilation and PVR decrease. In accordance with the present review, when hypertensive subjects were studied, CO decreased after exercise in 42% of cases. To our knowledge, six studies directly compared responses after exercise in hypertensive and normotensive subjects, yet each revealed different results (4,9,31,37,40,45). In two studies, PVR decreased equally in both groups (37,40). In two others, only hypertensive individuals presented PVR reduction (4,45), and only hypertensive individuals showed CO reductions in two other studies (9,31). Thus, although there was no unanimity in these studies, they suggest that the presence of hypertension may facilitate postexercise CO reduction, which should be further addressed.

Exercise protocol characteristics

Regarding exercise characteristics, it is known that exercise duration, intensity, and mode affect PAEH magnitude and duration (14,28,62-64). However, the results of this review suggest that none of these factors affect PAEH hemodynamic determinants. These results are in agreement with previous studies that directly compared different exercise protocols. Two studies found similar postexercise hemodynamics regardless of exercise duration (16,56). Furthermore, six studies directly compared different exercise intensities (6,10,16,22,28,56); however, only the last study (56) reported a difference in postexercise hemodynamics, with PVR reduction occurring only after intense and not after moderate exercise in males, but not in females (56). Three studies directly compared continuous and interval exercise (10,22,56), but only the last study (56) reported a difference, with interval exercise producing a higher PVR reduction. Thus, exercise characteristics (duration, intensity, and mode) do not seem to influence PAEH hemodynamic determinants.

Circadian physiological alterations affect hemodynamic determinants (65,66). Previous studies that compared postexercise hemodynamics in the morning and afternoon suggest a decrease in PVR only after afternoon

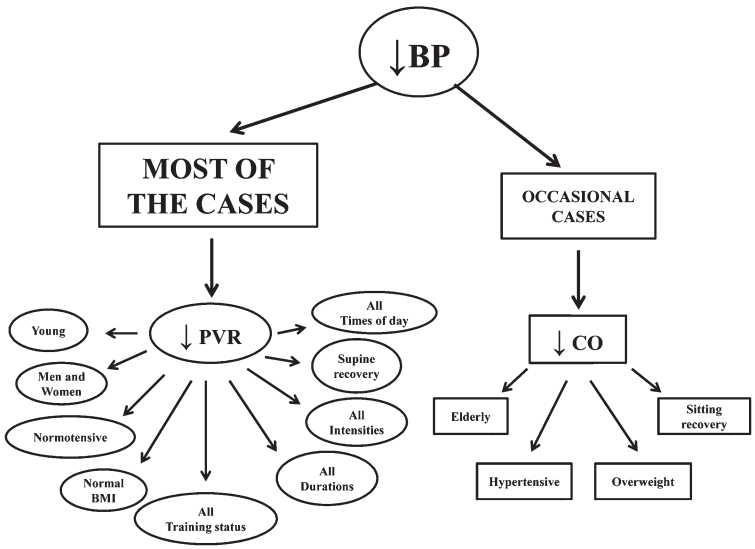

Figure 1. Schematic depicting the main findings of this review regarding the population and exercise protocol characteristics that may influence hemodynamic determinants (CO: cardiac output; PVR: peripheral vascular resistance) of post-aerobic exercise hypotension.

exercise (13,52,53). However, the results of this review did not identify any differences in PAEH hemodynamic determinants when exercise is performed in the morning and in the afternoon. As such, more data are needed.

Many PAEH studies have been performed with measurements taken in the supine position. This position facilitates hemodynamic assessment and avoids orthostatic stress, leading to a more specific analysis of exercise effects. However, the practical applicability is low for these measures because subjects do not tend to lie down after exercise. Thus, other studies performed hemodynamic evaluations in the sitting position. Orthostatic stress induced by the sitting position affects hemodynamic responses at rest (67) and may change hemodynamic behavior after exercise. Indeed, the present review suggests that the supine position facilitates PVR reduction, while the sitting position favors CO reduction after exercise. The same results were found in two studies that directly compared PAEH in these positions (21,33).

Possible influencing mechanisms

The mechanisms by which old age, hypertension status, increased BMI, and sitting position facilitate CO decrease and hamper PVR decrease after a single session of aerobic exercise are out of the scope of the present review. In addition, few studies investigated these possible mechanisms, and their results are controversial. Nevertheless, a hypothesis can be formulated and should be tested in the future. After a session of aerobic exercise, multiple hemodynamic responses were observed. Venous return decreases (9), leading to a decrease in stroke volume that favors CO reduction. Furthermore, it is known that venous return decrease deactivates the cardiopulmonary reflex, leading to an increase in peripheral sympathetic activity that increases PVR (68). In addition, a sympatholytic effect and a decrease in the alpha-adrenergic response to sympathetic stimuli have been reported after an aerobic exercise session (26), which may lead to a reduction in PVR. Moreover, it is supposed that some vasodilatory substances secreted by exercise, like nitric oxide, histamine, and prostaglandins, may contribute to maintaining vasodilation after exercise (69). However, the roles of some of these substances, such as nitric oxide (29) and prostaglandins (17), have been refuted in some studies. Finally, as BP decreases after exercise, the baroreflex is deactivated and, consequently, heart rate (HR) increases and peripheral sympathetic activity is stimulated (68). However, after a session of aerobic exercise, baroreflex sensitivity to HR control is blunted (70), and the set point of this reflex for peripheral adjustments is changed (26). Thus, these complex mechanisms interact after exercise, leading to decreased PVR in most circumstances and to decreased CO in some circumstances. It is possible that, as in elderly, hypertensive, and overweight subjects, sympathetic activity (57,71) and cardiopulmonary reflexes (68) are exacerbated while endothelial function (57,60) and baroreflex control (72) are impaired, PVR decrease after exercise is mitigated, and CO decrease is facilitated, which may explain the differences in PAEH suggested by this review. Once again, this is just a hypothesis that should be investigated in the future.

This review has some limitations. The search for articles was only carried out on PubMed. Although PubMed is the main health research database and includes better quality studies than other databases, the inclusion of other databases may have increased the number of studies. On the other hand, a strong aspect of this review is its novelty in presenting a quantitative

analysis of the frequencies of CO and PVR reduction after exercise with regard to each possible influence factor. We think this approach minimized the subjectivity of an analytic systematic review. Nevertheless, as a review, the results are only indicative of possible influence, and specific studies addressing the real effect of each influence should be conducted in the future.

In conclusion, the present literature review revealed that, in most cases, PAEH occurs following a decrease in PVR (Figure 1). However, CO reduction may be an important determinant of PAEH in some circumstances. The factors that might favor post-aerobic exercise CO reduction are advanced age, presence of excess body weight, presence of hypertension, and recovery in the sitting position. The review suggests that more studies are needed to directly address the influence of these factors. In addition, the different combinations of these factors may favor the reduction of CO or PVR, explaining the different results for PAEH determinants observed in the literature.

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