

Cardiovascular tolerance of healthy elderly subjects to weight-lifting exercises

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ABSTRACT

BERMON, S., D. RAMA, and C. DOLISI. Cardiovascular tolerance of healthy elderly subjects to weight-lifting exercises. *Med. Sci. Sports Exerc.*, Vol. 32, No. 11, pp. 1845–1848, 2000. **Objective:** To evaluate the hemodynamic strain and the myocardial tolerance of weight-lifting exercises in healthy elderly subjects. **Methods:** Sixty-five healthy elderly subjects (32 men/33 women) aged 65–80, were studied. Weight-lifting exercises consisted of two sets of 12 repetitions at 12-repetition maximum (RM) and four sets of five repetitions at 5-RM for, horizontal leg press, seated chest press, and bilateral leg extension movements. Cardiovascular tolerance to weight-lifting exercises was evaluated both physiologically and biologically by measuring heart rate (HR) and blood pressures continuously during exercise, and cardiac troponin I (cTnI) blood concentration before and 6 h postexercise. Comparisons between resting and exercise or postexercise values were performed by a bilateral-paired *t*-test. A value of $P < 0.05$ was considered statistically significant. **Results:** No significant increase in cTnI circulating concentration was observed secondary to exercise (16.56 ± 2.23 vs 14.40 ± 1.96 ng·L⁻¹; mean \pm SEM). This was observed despite a significant ($P < 0.001$) exercise-induced increase in systolic (SAP) and diastolic arterial pressures (DAP) and HR. Highest values of SAP, DAP, and HR (223.6 ± 3.1 mm Hg, 139.6 ± 1.9 mm Hg, and 108 ± 2 min⁻¹, respectively) were measured during the horizontal leg press exercise. **Conclusion:** These data suggest that weight-lifting exercises can be conducted in healthy elderly subjects without clinical, electrical, and biological sign of myocardial ischemia, if appropriate selection criteria, and proper respiratory techniques during exercise are applied. **Key Words:** AGING, BLOOD PRESSURE, CARDIAC TROPONIN I, EXERCISE

It is well known that skeletal muscle size and strength decrease of approximately 1% per year after the fifth decade (1,10,15). These changes are mainly explained by a reduction in both muscle fiber size and number, an increase in type I/type II fiber area ratio (2,18), changes in the sarcoplasmic reticulum calcium kinetics (16), a reduction in the motor neurons number throughout life (26), and also by disuse (6). It has been suggested that once strength declines below a certain threshold level required for activities of daily living, loss of independence and reduced quality of life may occur (9,12,17).

Many studies have demonstrated that progressive resistance training exercises produce strength gains in older people (7,23). The amplitude of this adaptive response is partly explained by exercise type and intensity; a significant hypertrophy occurring for intensities superior to 50% of the one-repetition maximum (1-RM). Nevertheless, the highest hypertrophic response being observed secondary to high-intensity regimens such as 80% of 1-RM, several authors (23) have used this intensity in the design of strength training program for healthy elderly subjects.

On the other hand, there has been some concern about the safety of weight-lifting exercises in elderly subjects because of the potential of the isometric component of resistance exercise to provoke arrhythmias (3), wall motion abnormalities, and particularly high-pressor response (24). Owing to the fact that there is a close correlation between the pressure-rate product (13,21) and myocardial oxygen consumption, one can hypothesize that the functional overload of the cardiovascular system imposed during intense, repetitive muscle contractions could theoretically lead to an increased myocardial oxygen consumption. Moreover, elderly subjects undergoing weight-lifting exercises are deconditioned from inactivity and may suffer from known (8) or unrecognized cardiovascular diseases. This increased metabolic demand may lead to infra-clinical and even infra-electrical ischemia of the myocardium, which can only be detected with very sensitive biological assay that operate at the picomolar level. Thus, and although there has been no publication on the topic, measurements of cardiac troponin I (cTnI) concentration theoretically appear as a valuable and very sensitive tool to detect ischemia secondary to weight-lifting exercise in elderly humans.

Thus, the aim of the present study was to evaluate the hemodynamic strain and the myocardial tolerance to weight-lifting exercises, in healthy elderly subjects, using a new biological tool.

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METHODS

Subjects. A total of 65 elderly subjects (32 men and 33 women), ranging in age from 65 to 80 yr, were studied. All subjects were normotensive free of cardiorespiratory and neurological diseases and gave written informed consent to participate to the study. Volunteers underwent a multiphasic screening procedure that included a health history, physical examination, and resting electrocardiogram. They were sedentary (none had engaged in any kind of regular exercise for at least 3 yr before the experiment) and did not participate to any weight-lifting program. All were nonsmokers and nonalcohol drinkers, and none was using medication that could interfere with the study results (with special regard to beta-agonist, beta-blockers, other antihypertensive drugs, and nitrate preparations). This study met the requirements of the Local Standing Committee on Human Research.

1-RM determinations. Before the strength test, four low to medium resistance training sessions were conducted as an accommodation period so that all subjects could become familiar with the equipment and proper exercise techniques. At the end of this period, 1-RM for leg press, bilateral leg extension, and seated chest press were determined. The 1-RM determinations were performed at least 1 wk before the strength test.

Strength test. All subjects performed a standardized strength test, at the same time of day, on a Marcy Vertex II multi-station weight machine (Marcy Physical Fitness Products, Alhambra, CA). This strength test was preceded by a 15-min warm-up (10 min of cycling at 50 W and 5 min of calisthenics) period and consisted of two sets of 12 repetitions at 12-repetition maximum and four sets of five repetitions at 5-repetition maximum for horizontal leg press, seated chest press, and bilateral leg extension movements. The concentric and eccentric phases of exercise were performed in approximately 2 s each, and the rest interval between two sets was 2 min. For each repetition, subjects were instructed to avoid Valsalva maneuver. The standardized strength test started at 9.15 a.m. and lasted approximately 75 min.

Cardiac troponin I determinations. Blood samples were drawn from the antecubital vein using a 22-gauge latex catheter (Insyte[®], Becton Dickinson, Meylan, France) at 8:30 on fasted subjects and 6 h after the strength test. Serum blood sample were centrifuged at 1000 g at 4°C for 15 min; the resulting serum was immediately frozen in plastic Eppendorf tubes at -80°C for later analysis. cTnI concentrations were assessed by a standard assay (upper reference limit, 100 ng·L⁻¹) on an Access immunoassay system analyzer (Sanofi Diagnostic Pasteur, Marnes la Coquette, France) and by a new generation, highly sensitive immunoassay that has been extensively described elsewhere (20). Briefly, the solid phase of this assay is a polystyrene tube coated with 8E1 anti-cTnI monoclonal antibody (MAb). Revelation is performed with the peroxidase-labeled MAb 11E12. The samples and standards and the labeled tracer antibody are incubated in the coated tubes at room temperature. After washes, the enzymatic activity is revealed by

TABLE 1. Specific and total amounts of load lifted (mean ± SEM) during the strength test.

Bilateral Leg Press (daN)	Bilateral Leg Extension (daN)	Seated Chest Press (daN)	Total (daN)
2599 ± 110	1143 ± 55	987 ± 48	4729 ± 184

addition of a luminescent substrate. The generated signal is directly proportional to the concentration of cTnI available in the sample. All samples were run in duplicate, and the average value is reported. All measurements were performed blindly without knowledge of patients' data.

Blood pressures and heart rate measurements.

Systolic (SAP) and diastolic (DAP) arterial pressure were continuously and noninvasively measured (Finapres, Ohmeda 2300 NIBP monitor, Englewood, CA) and recorded, before and during the standardized strength test, using the plethysmograph method of the unloaded arterial wall (22). The measurement sites were the third finger of the non-dominant hand, or the second left toe, during lower limb exercises (i.e., horizontal leg press and bilateral leg extension) and upper limb exercise (seated chest press), respectively. Three ECG electrodes were placed to monitor and print heart rhythm and heart rate (HR) from the oscilloscope of a defibrillator (Physio-Control LifePak 9P, Redmond, CA). HR was also continuously measured and recorded, on a beat per beat basis, during the standardized strength test by using a Polar Vantage NV (Polar Electro Oy, Oulu, Finland) HR recorder. Before each set of exercise, three different investigators simultaneously started recordings of ECG, arterial pressures, and HR. Pressure traces from Finapres were then visually inspected for artifacts by the same investigator, and the highest arterial pressures (during exercise) and corresponding HR (measured on the ECG and checked on the Polar recordings) were kept for analysis.

Statistical analysis. Data are expressed as means with standard errors. Comparisons between resting and exercise (hemodynamic data) or postexercise (biological data) conditions were performed by using a paired, bilateral *t*-test. Pearson's formula was used to calculate the correlation coefficient between basal and postexercise concentrations for cTnI. A *P*-value less than 0.05 was considered to indicate statistical significance.

RESULTS

Preliminary statistical analysis (data not presented) showed no significant differences between men and women of the studied group for cardiovascular and biological parameters. Thus, male and female observations were amalgamated. Mean age, height, and body weight were 70.4 ± 0.4 yr, 164.6 ± 0.8 cm, and 68.9 ± 1.0 kg, respectively. The specific and total amounts of load lifted during the standardized strength test are presented in Table 1.

No history of chest pain, ECG abnormalities was reported among our subjects, during the study period. SAP, DAP, and HR values significantly increased (*P* < 0.001) during the strength test whatever the type of exercise considered (Table 2). Leg press exercise induced higher increase in SAP, HR

TABLE 2. Cardiovascular data recorded at rest and during the different exercises of the strength test.

	Bilateral Leg Press		Bilateral Leg Extension		Seated Chest Press	
	Rest	Exercise	Rest	Exercise	Rest	Exercise
SAP (mm Hg)	138.2 (1.6)	223.6* (3.1)	137.4 (1.7)	200.6 (2.9)	138.5 (1.7)	199.5 (2.7)
DAP (mm Hg)	78.2 (1.7)	139.6† (1.9)	78.4 (1.5)	127.4† (2.0)	78.3 (1.6)	123.4 (1.9)
HR (min ⁻¹)	69.3 (1.6)	107.5* (1.8)	70.4 (1.7)	105.5† (2.2)	69.9 (2.0)	102.1 (2.1)

Values are mean \pm SEM.

SAP, systolic arterial pressure; DAP, diastolic arterial pressure; HR, heart rate.

* Significantly different ($P < 0.01$) than values recorded during the seated chest press exercise.

† Significantly different ($P < 0.05$) than values recorded during the seated chest press exercise.

($P < 0.01$), and DAP ($P < 0.05$) than chest press. Similarly but to a lesser extent, bilateral leg extension induced higher increase in DAP and HR ($P < 0.05$) than chest press (Table 2).

Using the standard cTnI assay, no subject showed a postexercise concentration higher than the upper reference limit of 100 ng·L⁻¹. Neither standard nor ultrasensitive assay showed significant increase in circulating concentrations of cTnI (Table 3). Moreover, postexercise concentrations of cardiac troponin I, assessed by the ultrasensitive method, showed strong correlation ($r = 0.57$; $P < 0.001$) with concentrations at rest.

DISCUSSION

The main result in the present study was that an intense strength training session did not increase cTnI circulating concentrations. This was observed by using a standard assay and also a highly sensitive immunoassay that operate at the picomolar concentration range (lower limit of detection at 3 ng·L⁻¹). The recent availability of highly sensitive immunoassay for quantitative determination of the cardiac muscle isoform of the troponin I in human serum is very helpful in the field of exercise physiology. Indeed, specificity of CK-MB is sometimes insufficient in clinical and physiological situations mixing cardiac and skeletal muscle strains (4). The specificity of our monoclonal antibodies for the human cardiac isoform of troponin I is extremely high, and there is no detectable cross-reactivity (<0.01%) with the skeletal muscle isoforms of troponin I, even for concentrations over 200 μ g·L⁻¹ (4). Our basal and postexercise cTnI mean concentrations are slightly lower than the 20.4 \pm 3.2 ng·L⁻¹ measured by Missov et al. (20) using the same technique in 55 healthy blood donors (mean age: 47 yr).

This result, reported for the first time, is found despite elevated SAP, DAP, and HR values attesting to the high level of load (pressure-rate product) imposed to the myocardium. Indeed, some pressure-rate product results observed during the bilateral leg press exercise were superior to 26,000 mm Hg·min⁻¹, which is a value reported after 5 min of cycling exercise at 150–175 W in middle-aged men (14). This result

point out the fact that weight-lifting exercises, associated with a functional overload of the cardiovascular system and an increased myocardial oxygen consumption, can be conducted in healthy elderly subjects, without biological sign of ischemia even at the picomolar level.

Highest values of arterial pressure and HR were reached during the bilateral leg press, and to a lesser extent during the bilateral leg extension exercises. These results concur with those of Smolander et al. (25), who showed that in young and middle-aged men, the amplitude of the SAP, DAP, and HR responses is related to the amount of muscle mass involved during strength exercises and to the number of arteries occluded by the intramuscular mechanical compression (5). Nevertheless, the amplitudes of the SAP and DAP responses demonstrated by our older adults remained lower than the 320/250 mm Hg reported by MacDougall et al. (19) in young men performing double leg press to failure at 95% of 1-RM. This difference may be explained by the lower intensity of our protocol and by the fact that our elderly subjects were asked to exhale during the concentric phase of each strength movement. This last point is of importance because it has been shown that a simple Valsalva maneuver may increase SAP and DAP by 65 and 45 mm Hg, respectively, in a normotensive elderly subject (11). Performing weight-lifting exercise with an open glottis may significantly reduce intrathoracic and arterial pressures, leading to a reasonable level of cardiac work in these older adults.

The observed correlation between basal and postexercise cTnI values is an interesting finding. It attests to relatively steady concentrations of this parameter, for each subject, under physiological circumstances, and to a good tolerance to weight training as well. This fact could also reflect a natural equilibrium between cardiac myofibrilolysis and protein synthesis.

In conclusion, the present study, using the most accurate biological method actually available, showed that cardiovascular tolerance of elderly subjects to weight-lifting exercises is good, if appropriate selection criteria, and proper respiratory techniques are applied. Further studies are needed to test the usefulness of cTnI to monitor myocardial tolerance of clinically stable and aerobically trained cardiac patients to weight-lifting exercises.

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TABLE 3. Mean concentrations of cardiac troponin I (standard and ultra sensitive assays) before and six hours after the strength test.

	Baseline (Supine)	6 h Post-Exercise	P
Standard cardiac troponin I (ng·L ⁻¹)	22.86 \pm 1.08	21.45 \pm 1.53	0.40
Ultrasensitive cardiac troponin I (ng·L ⁻¹)	16.56 \pm 2.23	14.40 \pm 1.96	0.28

Values are mean \pm SEM.

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