Effects of Aging on Cardiorespiratory Responses to Brief and Intense Intermittent Exercise in Endurance-Trained Athletes

Karim Chamari,1,2 Said Ahmaidi,1 Jean Ayoub,2 Abdallah Merzouk,1 Costas Laparidis,3 Dominique Choquet,1,4 Jacques Mercier,2 and Christian Préfaut2

1Laboratoire de Recherches “APS et Conduites Motrices: Adaptations–Réadaptations,” Faculté des Sciences du Sport, Université de Picardie Jules Verne, Amiens Cedex, France.
2Laboratoire de Physiologie des Interactions, CHU A. de Villeneuve, Montpellier, Cedex 5, France.
3Department of Physical Education and Sports Science, Democritus University of Thrace, Komotini, Greece.
4Centre de Réadaptation Cardiaque, Hôpital de Corbie, France.

The aim of this study was to investigate the effects of aging on athletes’ cardiorespiratory responses to a brief intense intermittent effort, using the force-velocity test as an exercise model. Twelve young athletes (24.8 ± 1.3 years) and twelve master athletes (65.1 ± 1.2 years) with similar heights, body masses, and endurance training schedules participated in this study. They performed both a maximal graded exercise and the force-velocity tests. The force-velocity test consisted of the repetition of 6-second sprints against increasing braking forces with 5-minute recovery periods. None of the subjects presented abnormal electrocardiogram responses to the tests. During the force-velocity test, the heart rate magnitudes of response in all subjects were correlated to the corresponding sprint power output (p < .001), with higher values for the young athletes (p < .001). Both groups had similar systolic blood pressure peaks of response during the force-velocity test. Both groups had similar preexercise and end-of-recovery oxygen consumption (VO2), but the young athletes had higher peaks of response (p < .001). The VO2 magnitudes of response increased during the test (p < .01) in all subjects, with higher values for the young athletes (p < .001). There was a positive correlation between the VO2 magnitude of response and (1) the corresponding sprint power output (R = .58, p < .001) and (2) the corresponding number of sprint repetitions (R = .29, p < .02). The young athletes had higher end-of-recovery and peak carbon dioxide production (VCO2) responses than the master athletes (p < .001). Pulmonary ventilation (V̇E) peaks of response to the sprints were higher in the young athletes (p < .001). There was a positive relation between the V̇E and VCO2 peaks of response (R = .34, p < .001). In both groups the peak heart rate, VO2, VCO2, and V̇E values attained during the force-velocity test represented similar percentages of the maximal values reached at exhaustion of maximal graded exercise. These results showed that aging does not alter the percentage of the cardiorespiratory response to a brief intense intermittent exercise such as the force-velocity test. Moreover, the arterial blood pressure response is not significantly altered, whereas the vasodilatatory response is.

Numerous studies have investigated the effects of aging on the cardiorespiratory responses to maximal endurance exercise (1–4). Intermittent exercise, including repeated bouts of maximal efforts with varying work loads, constitutes the physical activity of a great number of subjects (5). Kavanaugh and Shepherd (6) suggested this type of exercise for elderly people. To our knowledge, few works have studied the responses of middle-aged adults to intense sprinting (7,8), and no study has yet investigated the effects of aging on the cardiovascular responses to intense intermittent exercise. The force-velocity test is a widely used laboratory test allowing the assessment of peak anaerobic power output (9–13). It consists of maximal intermittent exercise bouts of 6-second duration against increasing braking forces separated by 5-minute recovery periods, and it corresponds to the type of exercise used in some physical training sessions (5). When preceded by a warm-up (9), the force-velocity test is well tolerated by master athletes (12) and thus appears to be an appropriate model to obtain brief intense intermittent exercise in our population of subjects. The aim of this investigation was to study the effects of aging on the cardiorespiratory responses to a brief intense intermittent exercise test, by comparing the responses of young and master athletes to the force-velocity test.

Materials and Methods

Subjects

Twelve young and twelve master athletes participated in the study. The young athletes, aged 18 to 33 years (24.8 ± 1.3), and the master athletes, aged 59 to 72 years (65.1 ± 1.2), participated in regular endurance training. The young athletes competed regularly, whereas the master athletes were training for a cycling ride event (2700 km in 18 days, corresponding to ≈140 km day−1). The entire population of subjects reported training by both running and bicycling, 6–12 hours per week for 9–12 months per year, for at least the 5 years preceding the study. The anthropometric characteristics of the subjects are reported in Table 1. All subjects provided informed written consents before the study. Subjects had normal medical histories and physical examinations, and none were smokers. Their electrocardiographic responses...
during exercise and recovery of maximal graded exercise were normal. Criteria for exclusion from the study were as follows: symptoms of chest discomfort consistent with angina, cardiac arrhythmias more severe than occasional atrial ventricular premature contractions, flat or downsloping ST segment depression (0.1 mV and more), and an abnormal blood pressure response to exercise. Only one master athlete was excluded for severe cardiac arrhythmias during the initial phase of the maximal graded exercise, but twelve master athletes performed the entire study.

Protocol

All subjects were familiarized with the laboratory exercise tests. On arrival, they received standardized instructions as to the testing procedure. They underwent physical examinations and resting electrocardiograms (ECGs), and anthropometric characteristics were then taken. The subjects performed the maximal graded exercise followed by the force-velocity test. At least 2 days separated the exercise tests, and all the experiments were done in the afternoon (2–5 pm) at a laboratory temperature of approximately 20–22°C. The subjects were asked to abstain from physical exercise for 1 day before the experiment and from drinking caffeine beverages in the last 4 hours preceding the test.

Maximal Graded Exercise Test

This exercise test, which also allowed subject evaluation for clinical evidence of cardiovascular disease (14), was performed on a calibrated cycle ergometer (Monark 818 E, Varberg, Sweden). It consisted of a warm-up of 3 minutes at 30 W at a pedaling rate of 60 rpm, immediately followed by increments of 30 W min⁻¹. When the heart rate (HR) attained 80% of the predicted maximal theoretical HR (theo. HRmax), the exercise load was increased only by 15–10 W min⁻¹ in order to obtain a peak power as precise as possible. Oxygen consumption (VO₂) was considered maximal (VO₂max) if at least two of the following criteria were achieved: (1) a leveling off of VO₂ despite load increase, (2) a respiratory gas exchange ratio higher than 1.1, and (3) attainment of age theo. HRmax ± 5. All the subjects achieved physiological criteria for VO₂max. The power developed by the subjects while attaining VO₂max was assumed to be peak aerobic power (PAP). The peak HR attained at exhaustion of maximal graded exercise was considered as HRmax. Exhaustion occurred within 8–13 minutes of exercise for all subjects.

The Force-Velocity Exercise Test

Described by Péres and coworkers (15), this test is a widely used laboratory exercise test (9–13). This test, which allowed the measurement of cardiovascular responses to brief intense intermittent exercise, was performed on a cycle ergometer (Monark 818 E, Varberg, Sweden). An initial warm-up period (12) was performed by the subjects; it consisted of 4 minutes of pedaling at 30% of PAP with a short acceleration of 6 seconds at the end of each minute. The warm-up was immediately followed by 5 minutes of recovery before the test began. The force-velocity exercise test consisted of repetitive short maximal sprints against increasing braking forces. The duration of each sprint was fixed at 6 seconds, the maximum time it took for the vigorously motivated subject to attain maximal velocity (Vmax) for each sprint after the starting signal. The subjects were asked to breathe continuously during the sprints, that is, to avoid the Valsalva maneuver. The duration of each passive recovery period was fixed at 5 minutes. Subjects remained in a sitting position for the warm-up period, the sprints, and for all the recovery periods; their feet were fixed to the pedals with toe clips. The maximal velocity and force-velocity relationships were assessed during the test by an automatic system as described by Mercier and colleagues (10). The test began against a braking force of 2 kg for the young athletes and 1.5 kg for the master athletes. Thereafter, the braking force was increased by 2 kg for young athletes and 1.5 kg for master athletes, except at the end of the test, when pedaling frequency dropped below 130 rpm. Then the braking force was increased by 1 kg or 0.5 kg in order to obtain a peak power as precise as possible. For each sprint, power output was obtained by calculating the product of the braking force and corresponding Vmax. Given the linear force-velocity relationship, the power-force relationship is parabolic. Anaerobic peak power output was defined as the highest power output calculated for the different braking forces. It was assumed that the subject attained anaerobic power output if an additional load induced a power decrease. Depending on their physical fitness, the subjects performed five to seven sprints per session.

Measurement of the Cardiorespiratory Variables

Heart rate was determined from a three-lead ECG with 12 derivations, which was monitored continuously on a Quinton heart rate monitor (Q 3000, Seattle, WA). For maximal

### Table 1. Anthropometric Characteristics, % Theo. VO₂max and % Theo. HRmax, and PPO

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Age (y)</th>
<th>Body Mass (kg)</th>
<th>Height (cm)</th>
<th>% Theo. HRmax</th>
<th>% Theo. VO₂max</th>
<th>PO (A) (W)</th>
<th>PO (B) (W)</th>
<th>PPO (C) (W)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young athletes (n = 12)</td>
<td>24.8</td>
<td>73.5</td>
<td>177.3</td>
<td>102.4</td>
<td>128.2</td>
<td>406</td>
<td>827</td>
<td>1089</td>
</tr>
<tr>
<td>SEM</td>
<td>1.3</td>
<td>2.8</td>
<td>2.2</td>
<td>1.1</td>
<td>5.4</td>
<td>17</td>
<td>38</td>
<td>40</td>
</tr>
<tr>
<td>Master athletes (n = 12)</td>
<td>65.1</td>
<td>74.5</td>
<td>173.0</td>
<td>96.3</td>
<td>123.0</td>
<td>244</td>
<td>480</td>
<td>624</td>
</tr>
<tr>
<td>SEM</td>
<td>1.2</td>
<td>2.6</td>
<td>1.7</td>
<td>3.1</td>
<td>6.8</td>
<td>6</td>
<td>28</td>
<td>33</td>
</tr>
</tbody>
</table>

Notes: % theo. HRmax, percentage of theoretical maximal heart rate; % theo. VO₂max, percentage of theoretical maximal oxygen uptake; PO, sprint power output; A, B, and C, sprints representing 18–33%, 50–66%, and 100%, respectively, of the braking force corresponding to PPO; PPO, anaerobic peak power output measured during the force-velocity test; SEM, standard error of the mean; NS, nonsignificant.

* p < .001.
graded exercise, HR was averaged over the last 20 seconds of every minute. However, during the force-velocity test, HR was monitored at rest with subjects sitting in a chair, on the cycle ergometer in preexercise conditions, and during the successive sprints and recovery periods. Because of its relative stability, HR was averaged over 30 seconds at rest, at preexercise, and at the end of each recovery period (from 4 minutes, 15 seconds to 4 minutes, 45 seconds). However, in order to assess accurately the peak HR response to the sprints, HR was averaged over 5 seconds; the highest value was considered as the peak HR (11).

Arterial blood pressure (ABP) with its two components, systolic blood pressure (SBP) and diastolic blood pressure (DBP), was measured by an automatic blood pressure monitor, which correlates a subject’s ECG-R waves with the Korotkof signals received from microphones placed in the blood pressure cuff on the right arm of the subject (Quinton 410, Seattle, WA). SBP and DBP were measured at rest, at preexercise, and at 30 seconds, 1 minute, 2 minutes, and at 4 minutes, 30 seconds of each recovery period (11). During rest and preexercise, several measurements were taken until two similar values (±5 mm Hg) were obtained (16). The two values were then averaged, and the mean was considered to be the rest and preexercise value. Every automatic measurement with the blood pressure monitor took ~15 seconds to complete; thus, in order to obtain an ABP measure at a given time in the recovery period, the measurement was started 15 seconds in advance.

Measurement of the ventilatory variables was done by connecting the subjects to a breath-by-breath automated metabolic system (CPX, Medical Graphics, St. Paul, MN). This system allowed continuous measurement of pulmonary gas exchanges and ventilation during preexercise, exercise, and recovery of both tests. Prior to each test, the gas analyzers were calibrated with gases of known concentrations. The subjects were connected to the CPX system, breathing through a 100-ml dead-space low-resistance valve. VO₂, carbon dioxide production (VCO₂), and the ventilatory variables were averaged on eight respiratory cycles (11, 17). This sliding technique provides a value at each respiratory cycle that is averaged on the concerned cycle and the seven preceding ones. Peak VO₂ and peak VCO₂ were followed separately, and the ventilatory variables were noted with respect to peak VCO₂ (17).

Expression of the results.—During maximal graded exercise, VO₂, VCO₂, and ventilatory variables were averaged over the last 20 seconds of every minute. However, for the force-velocity test, only three of the sprints and the respective recovery periods were studied because of the numerous and unequal number of bouts of exercise performed by the subjects. The first sprint (A) ranged from 18% to 33%, the second one (B) from 50% to 66%, and the third sprint (C) corresponded (100%) to the braking force against which the subjects attained their anaerobic peak power output (11).

Calculations.—Mean arterial blood pressure (MBP) was calculated by the following formula, in which values are expressed in mm Hg millimeters of mercury (16, 18):

$$\text{MBP} = \text{DBP} + \frac{1}{3}(\text{SBP} - \text{DBP})$$

The percentage of the total peripheral resistance decrease (% TPRD) was calculated from the MBP and HR by using the following formula (18):

$$\%\text{TPRD} = \frac{[(\text{MBP}/\text{HR})_2 - (\text{MBP}/\text{HR})_1] \times 100}{(\text{MBP}/\text{HR})_1}$$

with 1 = rest, and 2 = preexercise or recovery.

HR values corresponding to MBP were averaged over 5 seconds. The % TPRD was calculated at preexercise, and at 30 seconds, 1 minute, 2 minutes, and 4 minutes, 30 seconds of each recovery period. HR, SBP, DBP, and % TPRD magnitudes of response to each sprint were calculated from the difference of the presprint value preceding every bout of exercise and the peak or dip of the following response (16).

The maximal theoretical percentages of HR and VO₂max (% theo. HRmax and % theo. VO₂max, respectively) were calculated for the two groups according to Jones and colleagues (19).

Statistics

Values are expressed as mean ± the standard error of the mean (m ± SEM). The comparison of the two groups’ anthropometric characteristics and the cardiorespiratory variables measured at exhaustion of maximal graded exercise were performed by using an unpaired Student’s t test. In order to study the time course of the cardiorespiratory responses during the force-velocity test, a two-way analysis of variance (ANOVA) was done (F₁, level of load, and F₂, age group). An ANOVA was also conducted on the sprint power outputs and cardiorespiratory magnitudes of response to the sprints. When the ANOVA F ratio interaction (F₁ × F₂) was significant, the analysis was then completed by a contrast test to determine the location of the differences. The relationships between the cardiorespiratory magnitudes of response to the sprints and the corresponding sprint power outputs and number of sprint repetitions were calculated by linear regression coefficients. These relationships were calculated for the entire population of subjects. Statistical significance was fixed at p < .05.

Results

Regarding the anthropometric characteristics of the two groups (Table 1), young athletes and master athletes had similar heights and body masses. Moreover, at exhaustion of the maximal graded exercise, both groups had similar % theo. HRmax and % theo. VO₂max, respectively. Power outputs measured during sprints A, B, and C increased significantly with rising braking force (p < .001). The young athletes had significantly higher power outputs than master athletes for sprints A, B, and C (p < .001). However, these three sprints represented similar percentages of the braking force corresponding to the anaerobic peak power output for the two groups.

ECG and HR Responses

None of the subjects presented ECG abnormalities in response to the force-velocity test. At preexercise and at the end of the first sprint recovery, the two groups of subjects had similar HR values (Figure 1). At the end of the recovery
periods for sprints B and C, as well as for the three peaks of response, the young athletes had higher HRs than master athletes (interaction $F$ ratio $= 92.6; p < .001$). The HR peaks of response to the sprints represented similar % HRmax in both groups. Young athletes’ HR magnitudes of response to the sprints were significantly higher than those of master athletes ($p < .001$).

**Arterial Blood Pressure**

The young athletes and master athletes had similar SBPs at preexercise, rest, and during the force-velocity test (Figure 2). The young athletes had lower DBPs at 30 seconds of recovery of sprint B and at 1 minute of recovery of sprints B and C (interaction $F$ ratio $= 17.15; p < .01$). The young athletes had smaller relative increases in SBP and greater reductions in DBP response to sprints B and C than master athletes ($p < .001$ and $p < .01$, respectively).

**Total Peripheral Resistance Decrease**

The young athletes had significantly greater decreases in total peripheral resistance than master athletes (Figure 3) at preexercise and during the force-velocity test (interaction $F$ ratio $= 33.64; p < .001$). Young athletes and master athletes had similar TPRD magnitudes of response to the sprints.

**Oxygen Consumption**

In all subjects, the end-of-recovery VO$_2$ values were similar to the preexercise values, and both groups of subjects had similar preexercise and end-of-recovery values. The VO$_2$ peaks of response to the sprints increased significantly during the test, as the sprint C peak of response was higher than that of A (interaction $F$ ratio $= 91.07; p < .001$) in all subjects (Figure 4), and the young athletes’ peaks of response to the sprints were higher than those of master athletes ($p < .001$). The VO$_2$ peaks of response to the sprints represented similar % VO$_2$max in both groups. The VO$_2$ magnitudes of response increased during the test ($p < .01$) in all subjects and were always higher for the young athletes with respect to master athletes ($p < .001$). There was a positive relation between the VO$_2$ magnitude of response and (1) the corresponding sprint power output ($R = .58, p < .001$) and (2) the corresponding number of sprint repetitions ($R = .29, p < .02$).

**Carbon Dioxide Production**

The end-of-recovery VCO$_2$ values were similar to the preexercise values in master athletes, but they increased significantly in young athletes (interaction $F$ ratio $= 81.03; p < .001$; see Figure 5). The VCO$_2$ peaks of response to the sprints were stable during the test. The young athletes always had higher VCO$_2$ peaks of responses than master athletes ($p < .001$). The VCO$_2$ peaks of response to the sprints represented similar % VCO$_2$max in both groups. The VCO$_2$
magnitudes of response were stable during the test in all subjects, and they were always higher for the young athletes \((p < .001)\).

**Pulmonary Ventilation**

The end-of-recovery \(V_E\) values were similar to the preexercise values for both groups (Figure 6). The \(V_E\) peaks of response to the sprints increased during the test, as the sprint \(C\) peak of response was higher than that of \(A\) (interaction \(F\) ratio = 45.42; \(p < .001\)) in all subjects, with higher peaks in young athletes with respect to master athletes \((p < .001)\). The \(V_E\) peaks of response to the sprints represented similar \% \(V_{E\text{max}}\) in both groups. The \(V_E\) magnitudes of response were stable during the test and were always higher for the young athletes \((p < .001)\). There was a positive relation between the \(V_E\) and \(V_{CO_2}\) peaks of response \((R = .84, p < .001)\).

**DISCUSSION**

This study showed the following in response to a brief intense intermittent exercise: (1) none of the subjects presented ECG abnormalities in response to the force-velocity test; (2) the young athletes had higher cardiorespiratory peaks and magnitudes of response to the sprints than master athletes, but similar peaks of response when these were expressed as percentages of measured maximal values; and (3) the young athletes had lower total peripheral resistance than master athletes, at preexercise and during the force-velocity test.

It is widely acknowledged that the age-related decline in maximal physical performance is the consequence of many factors; these include the impairment of different functions as a result of the aging process per se, a progressive reduction in physical activity, health status, body mass, and hereditary factors, which are difficult to control (20). In order to try to isolate the effects of aging on the exercise cardiovascular responses, the present cross-sectional study was conducted on two groups of lean healthy young and master athletes who observed a similar endurance training schedule. The two groups of subjects presented similar percentages of maximal theoretical \(VO_2\max\) in their respective age categories (19); it was thus assumed that either young athletes and master athletes had similar relative fitness levels (3) or that they underwent the same potential bias as did the population of Jones and colleagues (19). Nevertheless, the possibility of an effect of the numbers of years of training cannot be ruled out, as the master athletes had been training for a longer time than the young athletes because of their age. All subjects were asked to exert maximal effort for both exercise tests. The motivation in the two age groups was considered to be comparable, because for the maximal graded exercise they had similar percentages of theoretical maximal heart rates in their respective age categories (3).

The % TPRD was calculated by using a formula in which it was assumed that the stroke volume was constant when the measurements of ABP and HR were made. Thus, the results presented for this variable have to be interpreted with care. Indeed, the possibility of erroneous values cannot be ruled out if stroke volume is altered during sprint recoveries.
The force-velocity test was preceded by a warm-up (9, 12). As pointed out by Barnard and colleagues (7), a warm-up period is useful for decreasing the eventual abnormal ECG responses that may occur in response to sudden brief intense exercise. Indeed, none of the subjects presented any abnormal ECG responses that would have impeded testing. Furthermore, none of the subjects was injured or reported any pain during the experiment. However, such intense exercise has to be used cautiously with at-risk populations, for example, untrained elderly people with higher risks of injuries or cardiac events (21).

The HR peak of response to the first force-velocity sprint was significantly higher in young athletes than in master athletes. This is in accordance with the results on continuous exercise, where, for a similar relative submaximal work load, the young athletes had higher HRs than master athletes (22). Seals and colleagues (23) reported that the reduced HR response with aging is probably due to both less withdrawal of cardiac vagal tone and diminished beta-adrenergic responsiveness. When expressed as percentage of HRmax, the HR peaks of response to the sprints represented similar values in both groups. This is in accordance with the effects of physical fitness on these responses (11), and it indicates that aging does not alter the percentage of HR response to a given sprint intensity.

The young and master athletes had similar peak SBP responses. This is in accordance with the results of Hagberg and coworkers (22) on submaximal aerobic exercise. Furthermore, the lower magnitudes of SBP response with increasing load in young athletes is in accordance with the results on maximal graded exercise in which a greater increase in SBP is found in older subjects (24). The age-related stiffening of the arterial tree may explain the finding of increased SBP magnitudes of response in master athletes despite the higher magnitudes of HR response in young athletes (25). The greater DBP decreases in young athletes may also be explained by the lower vascular resistance in these subjects (26). The young athletes had greater decreases in TPRD than master athletes; this was essentially caused by the decrease that occurred in preexercise conditions. A higher peripheral resistance was also found in master athletes during maximal graded exercise (27). This can be explained by the age-induced elevation in peripheral vascular resistance at a given exercise intensity. These age-related alterations may be attributed to decreased volume distensibility of central vessels, an associated partial obliteration of peripheral vascular beds with a resultant increase in peripheral resistance, and a relatively small decline in flow (27).

The VO2 peaks of response to the sprints increased, as did the VO2 magnitudes of response, which were correlated to the corresponding sprint power outputs and number of sprint repetitions during the force-velocity test. This is in accordance with the responses of young trained and un-
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trained subjects to the force-velocity test (11), and it suggests that aging does not alter the VO₂ linearity to such exercise; that is, VO₂ response is quantitatively related to the amount of work done (28,29). The higher young athletes’ VO₂ peaks of response were probably due to the higher sprint power outputs, as VO₂ closely parallels the exercise work load (29), and to their higher absolute VO₂max (11, 30). Moreover, the VO₂ peaks of response were presumably related to the rapid change in pulmonary blood flow by the “cardiodynamic” response (31), as the young athletes had higher HR responses than master athletes. Both groups had similar end-of-recovery VO₂. This is in contrast with the observations of Chick and coworkers (3) of a slowed VO₂ recovery in aged subjects. However, as noted by these authors, at the end of a relatively long recovery period no difference is found between young and aged subjects.

The VO₂ peaks and magnitudes of response to the sprints did not increase during the test. This confirms in the elderly the results of trained and untrained young subjects to the force-velocity test, indicating that VO₂ linearity (29) cannot be assumed during brief intense intermittent exercise (11). The young athletes had higher VO₂ peaks of response to the sprints. The VO₂ peak of response can be attributed to the “cardiodynamic” effect (31) and to the excess VO₂’s reflecting the amount of CO₂ eliminated to compensate for the acid-base balance (2). The higher HR response of the young athletes can explain the higher VO₂ peak of response, but the hypothesis of an already increased lactatemia probably explains only a relatively small part of the response. The VO₂ peak response occurs in the first seconds of recovery, whereas it was shown that venous blood lactate is significantly increased later during the force-velocity test sprint recoveries (10,13). Both groups of subjects had similar relative VO₂ peaks of response to the sprints. This is in accordance with the fact that both groups had similar relative HR peaks of response to the sprints. The end-of-recovery VO₂ values were similar to the preexercise values in master athletes, but they increased significantly in young athletes. This finding is in agreement with previous results in young athletes (11), in whom the VO₂ rise may be attributed to the buffering of H+ from lactic acid in the muscle (2), as glycolysis is already activated in 6-second short intensive exercise (5,10,13). The absence of end-of-recovery VO₂ increase in master athletes may reflect lower lactatemia in these subjects, as it was shown that maximal exercise venous blood lactate is reduced in elderly people, probably as a result of a reduced sensitivity to the sympathetic activity (32).

Ventilation was followed with respect to VO₂ because it has been established that VO₂ leads V E (17). Indeed, V E and VO₂ were well correlated in response to the force-velocity test. The stability of the V E magnitudes of response during the test confirms in the elderly the findings in young subjects that ventilation linearity (29) cannot be assumed during brief intense intermittent exercise. The young athletes had higher V E peaks of response to the sprints. The V E response to exercise is multifactorial (33); thus, the V E peaks are probably the sum of the following three phenomena: (1) the cardiodynamic response, as the initial rise in V E roughly parallels changes in HR (17,28); (2) the induced catecholamine rise (17,33), as the increase in catecholamines in response to the force-velocity sprints was shown (5) in 6-second intermittent exercise; and (3) the above-mentioned increases in CO₂ and H⁺ production (17). The higher V E peaks of the young athletes could be due to the higher HR response. There is the possibility of a higher catecholamine production in young athletes, as suggested by the finding that aging is associated with the attenuation of adrenergic stimulation to exercise (3). Lastly, the higher V E peaks of response may be explained by the higher VCO₂ of the young athletes and/or the hypothetized higher lactatemia that may have contributed to the tendency of an increased end-of-recovery V E in young athletes (3). The finding of a similar relative V E response to the force-velocity test in young and master athletes is not in accordance with the findings of Brischetto and colleagues (1). However, these authors have also showed a contrasting phenomenon of a blunted V E response in elderly people to hypercapnia. Moreover, McConnell and Davies (4) suggested that the above-mentioned higher V E in the elderly was the consequence of a lower mechanical efficiency and a higher metabolic requirement. However, the present study of intense intermittent exercise is different from the above-mentioned studies, and both groups of subjects pedaled against similar relative work loads. Moreover, it was shown by McConnell and Davies (4) that young and elderly subjects had similar mechanical efficiencies at high work loads.

In summary, this study showed that in response to a brief intense intermittent exercise with increasing load, none of the endurance-trained subjects presented ECG abnormalities. The young athletes had higher peaks and magnitudes of cardiorespiratory responses; however, when these were expressed as percentages of the maximal values, the young and master athletes had similar responses. Thus, the percentage of the HR and ventilatory response to brief intense intermittent exercise is not altered by aging.

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Address correspondence to Professeur S. Ahmaidi, Faculté des Sciences du Sport, Allée P. Grousset, Campus Universitaire Le Baill, 80025, Amiens Cedex, France. E-mail: Said.Ahmaidi@ca.u-picardie.fr

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