Exercise Heart Rate Variability of Older Women in Relation to Level of Physical Activity

Sylvia Reland,1 Nathalie S. Ville,2 Sara Wong,3 Hervé Gauvrit,3 Gaëlle Kervio,1 and François Carré1

1Groupe de Recherche Cardiovasculaire, Unité de Biologie et Médecine du Sport, Rennes, France.
2Laboratoire de Physiologie et Biomécanique de l’Exercice Musculaire, UFR STAPS Université de Rennes II, France.
3Laboratoire du Traitement du Signal et de l’Image, INSERM Université de Rennes I, France.

The purpose of this study was to determine the effect of the level of physical activity in older women on heart rate (HR) response to its neural control at rest and during exercise by using heart rate variability (HRV) analysis. Electrocardiogram (ECG) was recorded in 3 (low, moderately, and highly) active groups of older women at rest and during submaximal exercise. Spectral HRV indexes were obtained from the ECG signal. At rest, highly active subjects have low HR without any alteration of HRV. During incremental submaximal exercise, parasympathetic modulations of HR decreased only in the highly active subjects (p < .01) without any alteration of HR, compared with the other groups. In older women, the effects of the level of physical activity on HR and HRV are dissociated. Quite a high level of physical training induces a higher sensitivity of sinus node response to the autonomic nervous system during exercise.

HEART rate (HR) is primarily controlled by autonomic nervous system input to the sinus node. Its responses to sympathetic and parasympathetic drive are explored by a useful noninvasive tool: heart rate variability (HRV) (1). HR is influenced by physiological factors such as cardiorespiratory fitness and aging (2). Indeed, young and older endurance-trained athletes generally present resting bradycardia concomitant with an alteration of autonomic balance illustrated by a high resting HRV (2). However there were some discrepancies concerning the aging process on resting HR in healthy sedentary people, which is decreased (3), increased (4), or remains unmodified (5). Generally, aging leads to a decrease in parasympathetic influence on the heart, which is manifested by a decrease in HRV (6). A low level of resting HRV has been identified as a risk marker for all causes of mortality (7). The incidence of clinically significant cardiovascular disease in women increases with age, particularly after menopause (8). As the postmenopausal women have both increased incidence of cardiac event and a low physical activity level (9), it is interesting to investigate the effects of physical activity levels on HR and HRV in older women.

HR increases linearly with exercise intensity, and heart rates at rest and at given levels of exercise are lower in both young and older exercise-trained subjects (5). The effects of physical training on parasympathetic and sympathetic control of heart rate are traditionally based on HRV studies conducted at rest or during postural change in older people (2,10,11). Physical exercise, particularly in young and middle-aged people, is used to study HRV during stress, which greatly modifies the cardiovascular system homeostasis and induces autonomic alterations (12–14). However, little is known about HRV in relation to physical activity level during exercise in older people, particularly in women.

Moreover, these studies lead to conflicting results, probably reflecting some differences in experimental protocols (15,16). All these data underscore the need to better understand the effects of physical activity levels on HR and HRV in older women during exercise. Therefore the aim of this study was to assess, in healthy postmenopausal women, the HR responses to its neural control. To this end, HRV analysis was used in accordance with different levels of physical activity, at rest, and during moderate-intensity physical exercise.

METHODS

Subjects

Thirty healthy postmenopausal women (aged 60 to 70 years) participated in the present investigation. The nature, purpose, and risks of the study were explained to each subject. Written informed consent was obtained from the subject, and ethics committee approval was received. The participants were all nonsmokers, normotensive, and free of cardiovascular disease by history and clinical examination, which included resting blood pressure measurement and electrocardiogram (ECG). None were taking cardioactive medication. Self-reported usual physical activity level was assessed using a physical activity questionnaire (17). A MET (Metabolic Equivalent Task) value was assigned to each activity. The number of hours spent in each activity per week was multiplied by the appropriate MET value and the subject body mass to obtain a value of energy expenditure (kcal/wk). The sum of the activity values was an estimation of weekly exercise energy expenditure. Three groups were then identified. Subjects reporting less than 1000 kcal/wk were classified as low active, which corresponds to the recommendations for minimal activity required (150 kcal/
day) to optimize health (18). This group (n = 12) was not engaged in any regular physical activity. Subjects reporting more than 2000 kcal/wk, which corresponds to a physical activity level above which there is a reduced age-adjusted relative risk of myocardial infarction, were considered highly active (19). They (n = 9) came from a cycle touring club and had cycled regularly (in excess of 80 kilometers per week) for more than 4 years. Subjects reporting physical activity levels of 1000–2000 kcal/wk were considered moderately active. This group (n = 9) had regularly practiced voluntary gymnastics for more than 4 years. Physiological capacity was assessed by a maximal oxygen uptake (VO₂max) measurement, a classical index of maximal cardiovascular function. The low active group VO₂max was lower than predicted (20). The moderately active VO₂max was 100%–120% of that predicted, while the highly active VO₂max was higher than 130% of predicted uptake. Body fat percentage was measured using the skinfold thickness method (21). Anthropometric and physiological data is described in Table 1.

**Experimental Procedure**

All subjects performed a graded maximal exercise test to measure VO₂max (ml/min/kg), HRmax (beat/min), maximal exercise power (Wmax, watts), and to reveal medical exclusion criteria for the study. The anaerobic threshold was calculated with Beaver’s method (20) by 2 technicians (N.S.V. and F.C.). The graded exercise test was performed on an ergocycle (ERG 900, Marquette Helilge, Milwaukee, WI), with continuous recording 12-lead ECG (Cardio System, Marquette Helilge) and breath-by-breath gas exchange analysis (Oxycon Delta, Jaeger, Hoechberg, Germany).

The subject sat quietly on the ergocycle for 2 minutes, connected to the gas analyzer. Then, after a 3-minute warming-up period at 20 watts, the work rate was increased 10 watts every minute until exhaustion. The mean VO₂ and HR as measured during the last 30 seconds of each stage were taken into account. Arterial blood pressure was automatically measured every 2 minutes. The exercise test was stopped when at least three classical criterions of VO₂ max were attained (22).

ECG recordings for HRV measurement were always performed 1 week after the maximal exercise test. Subjects were instructed to refrain from any excessive physical activity and ingesting caffeine or alcohol for at least 24 hours before testing. Trials were held in the morning, 3 hours after breakfast. Before measurement, subjects rested for 10 minutes in a supine position in a quiet room. All recordings were performed by the same technicians (S.R. and F.C.). Lighting was kept constant and noise levels were minimized during the test. The subjects performed 2 tests, including 1 familiarization test and 1 experimental test at intervals of 1 week. During the experimental test, a 3-lead ECG was recorded at rest, in a seated position on the ergocycle during a 6-minute period, and during dynamic exercise performed at 30% and 60% of individual Wmax, each over a period of 10 minutes. No attempt was made to influence breathing frequency or tidal volume.

**ECG Data Analysis**

The ECG was sampled at 1000 Hz with the PowerLab acquisition system (ADInstruments Pty. Ltd., Castle Hill, Australia) installed in a Macintosh computer (Power Mac, Cupertino, CA). The accuracy of the measurements in the present study was within 1 millisecond. The ECG recordings for the 6-minute long resting period and the last 6 minutes of all exercises were then analyzed. The detection of the QRS complex was conducted using Gritzali’s algorithm (23). The RR intervals sequence was defined by the procedure reported by Brugge and Andersen (24). Each RR interval was visually inspected for the 6-minute long resting period and the last 6 minutes after breakfast. Before measurement, subjects rested hours before testing. Trials were held in the morning, 3 hours after breakfast. Before measurement, subjects rested for 10 minutes in a supine position in a quiet room. All recordings were performed by the same technicians (S.R. and F.C.). Lighting was kept constant and noise levels were minimized during the test. The subjects performed 2 tests, including 1 familiarization test and 1 experimental test at intervals of 1 week. During the experimental test, a 3-lead ECG was recorded at rest, in a seated position on the ergocycle during a 6-minute period, and during dynamic exercise performed at 30% and 60% of individual Wmax, each over a period of 10 minutes. No attempt was made to influence breathing frequency or tidal volume.

**ECG Data Analysis**

The ECG was sampled at 1000 Hz with the PowerLab acquisition system (ADInstruments Pty. Ltd., Castle Hill, Australia) installed in a Macintosh computer (Power Mac, Cupertino, CA). The accuracy of the measurements in the present study was within 1 millisecond. The ECG recordings for the 6-minute long resting period and the last 6 minutes of all exercises were then analyzed. The detection of the QRS complex was conducted using Gritzali’s algorithm (23). The RR intervals sequence was defined by the procedure reported by Brugge and Andersen (24). Each RR interval was visually inspected for the 6-minute long resting period and the last 6 minutes after breakfast. Before measurement, subjects rested hours before testing. Trials were held in the morning, 3 hours after breakfast. Before measurement, subjects rested for 10 minutes in a supine position in a quiet room. All recordings were performed by the same technicians (S.R. and F.C.). Lighting was kept constant and noise levels were minimized during the test. The subjects performed 2 tests, including 1 familiarization test and 1 experimental test at intervals of 1 week. During the experimental test, a 3-lead ECG was recorded at rest, in a seated position on the ergocycle during a 6-minute period, and during dynamic exercise performed at 30% and 60% of individual Wmax, each over a period of 10 minutes. No attempt was made to influence breathing frequency or tidal volume.

**Table 1. Anthropometric and Physiological Data of Low, Moderately, and Highly Active Subjects**

<table>
<thead>
<tr>
<th></th>
<th>Low (n = 12)</th>
<th>Moderate (n = 9)</th>
<th>High (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>67.3 ± 1.2</td>
<td>68.8 ± 1.1</td>
<td>65.0 ± 1.3</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>57.8 ± 2.2</td>
<td>57.7 ± 2.5</td>
<td>57.2 ± 1.2</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>159.3 ± 1.4</td>
<td>157.6 ± 2.3</td>
<td>160.3 ± 1.0</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>22.7 ± 0.6</td>
<td>23.2 ± 0.7</td>
<td>22.2 ± 0.3</td>
</tr>
<tr>
<td>Body fat percentage (%)</td>
<td>33.5 ± 0.7</td>
<td>33.0 ± 0.8</td>
<td>32.9 ± 1.7</td>
</tr>
<tr>
<td>Activity questionnaire (kcal/wk)</td>
<td>707.0 ± 73.7</td>
<td>1496.1 ± 91.6*</td>
<td>3445.0 ± 409.3*</td>
</tr>
<tr>
<td>VO₂max (ml/min/kg)</td>
<td>19.2 ± 0.8</td>
<td>22.7 ± 0.7*</td>
<td>31.8 ± 1.4*</td>
</tr>
<tr>
<td>% predicted VO₂max</td>
<td>94.6 ± 2.1</td>
<td>115.4 ± 1.4</td>
<td>151.2 ± 5.8*</td>
</tr>
<tr>
<td>ATVO₂ (ml/min/kg)</td>
<td>12.3 ± 0.5</td>
<td>13.01 ± 0.6</td>
<td>19.1 ± 0.7*</td>
</tr>
<tr>
<td>HRmax (bpm)</td>
<td>151.1 ± 2.84</td>
<td>150.7 ± 4.81</td>
<td>164.6 ± 4.37*</td>
</tr>
<tr>
<td>Wmax (watts)</td>
<td>81.7 ± 3.8</td>
<td>95.6 ± 5.8*</td>
<td>137.8 ± 5.7*</td>
</tr>
</tbody>
</table>

* p < .05 between moderately and low active groups.
> p < .001.
§ p < .01.
* p < .05 between highly and moderately active groups.
/ p < .05 between highly and low active groups.
BMI = body mass index; VO₂max = maximal oxygen uptake; ATVO₂ = oxygen uptake at anaerobic threshold; HRmax = maximal heart rate; Wmax = maximal exercise power.
HEART RATE VARIABILITY OF OLDER WOMEN

HF and LF are expressed in absolute terms (ms\(^2\)). LF and HF are also expressed in normalized units (NU), which represent the relative value of each power component in proportion to the total power minus very low frequency (VLF) value (%). LF/HF is a marker of sympathovagal balance (25).

**Statistical Analysis**

Results were presented as mean ± standard error (SE) of the mean. A one-way analysis of variance (ANOVA) was performed to compare anthropometric and physiological data.

HRV parameters were systematically tested for normality. If the test was not passed, the natural log transformation was done before performing the statistical test.

The influence of physical activity levels on HRV was assessed during the experimental test. A two-way repeated measures ANOVA was performed on each variable (3 groups \(\times 3\) conditions). The 3 conditions for the test were the seated resting position, 30%, and 60% Wmax. The location of significant pair-wise differences was determined using a Tukey's post hoc procedure.

Pearson's correlation coefficient was used to assess the relationship of physical activity levels with HRV at anaerobic threshold (ATVO\(_2\)).

A \(p\) value \(\leq .05\) was considered significant. Statistical analyses were performed with Statistica software version 5.97 (StatSoft, Inc., France).

**RESULTS**

There were no significant differences in age, height, weight, body mass index, or body fat percentage between the 3 groups. The score for the physical activity questionnaire, Wmax, and VO\(_2\)max were the highest in the highly active group (\(p < .001\)), and higher in the moderately active group compared with the low active group (\(p < .05\)). ATVO\(_2\) and HRmax were significantly higher only in the highly active group (\(p < .001\) and \(p < .05\), respectively) (Table 1).

HRV components are shown in Tables 2 and 3. Resting RR was significantly longer in the highly active group versus the low active group (\(p < .05\)). RR decreased between rest and 30% Wmax, then decreased further at 60% Wmax in all groups (\(p < .001\)). There was no significant difference between groups concerning RR at 30% or at 60% Wmax (Table 2). Figure 1 illustrates RR alterations induced by exercise in a highly active subject.

Table 2. RR and Spectral Indexes of Heart Rate Variability in Low, Moderately, and Highly Active Groups

<table>
<thead>
<tr>
<th></th>
<th>Low (ms)</th>
<th>HF (ms(^2))</th>
<th>LF (ms(^2))</th>
<th>TP (ms(^2))</th>
<th>LF/HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>1</td>
<td>828 ± 25*</td>
<td>125.9 ± 50.4</td>
<td>642.5 ± 208.8</td>
<td>768.4 ± 255.1</td>
</tr>
<tr>
<td></td>
<td>1 vs 2</td>
<td>† †† ††† †††</td>
<td>† †† ††† †††</td>
<td>† †† ††† †††</td>
<td>† †† ††† †††</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>563 ± 16</td>
<td>7.9 ± 1.5</td>
<td>19.9 ± 4.8</td>
<td>27.7 ± 5.7</td>
</tr>
<tr>
<td></td>
<td>2 vs 3</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>456 ± 10</td>
<td>3.2 ± 0.7</td>
<td>2.7 ± 0.6</td>
<td>5.9 ± 0.9</td>
</tr>
<tr>
<td></td>
<td>1 vs 3</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
</tr>
<tr>
<td>Moderate</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>900 ± 42</td>
<td>151.6 ± 32.6</td>
<td>514.2 ± 147.2</td>
<td>655.8 ± 177.6</td>
</tr>
<tr>
<td></td>
<td>1 vs 2</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>642 ± 27</td>
<td>25.1 ± 8.4</td>
<td>51.0 ± 15.9</td>
<td>76.1 ± 19.5</td>
</tr>
<tr>
<td></td>
<td>2 vs 3</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>525 ± 17</td>
<td>8.4 ± 2.7</td>
<td>9.8 ± 3.4</td>
<td>18.2 ± 5.2</td>
</tr>
<tr>
<td></td>
<td>1 vs 3</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
</tr>
<tr>
<td>High</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>921 ± 42*</td>
<td>193.0 ± 59.9</td>
<td>524.4 ± 95.4</td>
<td>717.4 ± 150.5</td>
</tr>
<tr>
<td></td>
<td>1 vs 2</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>605 ± 11</td>
<td>12.9 ± 2.4</td>
<td>38.4 ± 8.2</td>
<td>51.3 ± 9.4</td>
</tr>
<tr>
<td></td>
<td>2 vs 3</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>472 ± 16</td>
<td>2.3 ± 1.8</td>
<td>3.1 ± 0.6</td>
<td>5.4 ± 0.9</td>
</tr>
<tr>
<td></td>
<td>1 vs 3</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
<td>† †† ††† ††††</td>
</tr>
</tbody>
</table>

**Notes:** Condition 1, at rest; Condition 2, during exercise at 30% Wmax; Condition 3, at 60% Wmax.

* \(p < .05\) between low and highly active groups.
† \(p < .01\) between rest and 30% Wmax.
‡ \(p < .001\).
§ \(p < .001\) between 30% and 60% Wmax.
| \(*\) \(p < .001\) between rest and 60% Wmax.
| \(p < .001\) between rest and 60% Wmax.

HF = high frequency; LF = low frequency; TP = total power; LF/HF = low frequency/high frequency.

At rest in a seated position, LF peak was centered at approximately 0.07 Hz, and HF peak was approximately 0.25 Hz. During exercise, LF peak was less sharp. However, no changes occurred in central frequency. On the contrary, HF became a well-defined peak and its central frequency increased (\(p < .01\)). Figure 2 illustrates the typical spectral data alterations obtained by exercise in the same highly active subject.

No significant difference was observed between groups at rest or during exercise, concerning all spectral HRV indexes (Tables 2 and 3).

During exercise, all the absolute spectral HRV indexes decreased from rest to 30% Wmax and from rest to 60% Wmax in the 3 groups (\(p < .001\)). From 30% to 60% Wmax, LF (\(p < .001\)) and TP (\(p < .01\)) decreased in the 3 groups while HF decreased only in the highly active one (\(p < .001\)).

Table 3. Indexes of Heart Rate Variability in Normalized Units in Low, Moderately, and Highly Active Groups

<table>
<thead>
<tr>
<th></th>
<th>Low</th>
<th>Moderate</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>LF NU (%)</td>
<td>83 ± 2*</td>
<td>68 ± 3†</td>
<td>47 ± 6</td>
</tr>
<tr>
<td>HF NU (%)</td>
<td>17 ± 2*</td>
<td>32 ± 3†</td>
<td>53 ± 6</td>
</tr>
</tbody>
</table>

**Notes:** Condition 1, at rest; Condition 2, during exercise at 30% Wmax; Condition 3, at 60% Wmax.

* \(p < .001\) between rest and 60% Wmax.
† \(p < .01\) between 30% and 60% Wmax.
LF = low frequency; HF = high frequency; NU = normalized units.
The LF/HF ratio (Table 2), between rest and both 30% Wmax and 60% Wmax, only decreased in the low active group ($p < .01$ and $p < .001$, respectively).

Concerning normalized spectral HRV values, between rest and 30% Wmax, no significant changes were observed in any group. From rest to 60% Wmax and from 30% to 60% Wmax, HF NU increased only in low active subjects ($p < .001$ and $p < .01$, respectively). Consequently, the results are inverted concerning LF NU.

No significant correlation was observed between resting HRV indexes and $\text{VO}_2\text{max}$ or AT$\text{VO}_2$. At 30% Wmax, LF and TP were positively correlated with $\text{VO}_2\text{max}$ ($r = .41$, $p < .05$, and $r = .38$, $p < .05$, respectively), and TP was correlated with AT$\text{VO}_2$ ($r = .38$, $p < .05$).

**DISCUSSION**

The main findings of the present study performed in postmenopausal women are that (a) the level of physical activity influences HR at rest, without any alteration in HRV indexes, and (b) at 30% and 60% Wmax, HR is not influenced by the level of physical activity, contrary to sinus node response to autonomic regulation.

It is widely assumed that regular exercise training induces a decrease in resting HR (2,5). This decreased HR can be observed both in young and middle-aged women (14). The origin of the training bradycardia is probably multifactorial, with a decrease of intrinsic heart rate (27) and/or an alteration of HRV (2,5). In women, resting HRV declines with aging in sedentary as in highly trained endurance populations (2). However, transversal study demonstrates that HRV is higher in trained women than in their sedentary peers, regardless of age (2). In older athletes, as in young women, training effects on HRV results mainly from an increased parasympathetic tone (2,5). LF seems also increased in endurance-trained young and older women (2,28), but the underlying mechanisms still need to be clarified. Our results confirm the impact of the level of physical activity on resting HR in older women. Nevertheless, no significant difference in the resting HRV indexes has been clearly demonstrated between our populations.
Moreover no correlation has been observed between VO\textsubscript{2}max and resting HRV indexes. In the present study, the highest level of physical activity is accompanied by the highest VO\textsubscript{2}max values. However, the physical activity level performed in the highly active group should not be sufficient to induce significant alterations to resting HRV. Thus, the lower resting heart rate in our highly active population may be due to a lower intrinsic heart rate as previously suggested (27).

Some stress conditions such as physical exercise alter HRV (12–14). Relatively little and inconsistent information is reported on the effect of the level of physical activity on HRV in older people during exercise. In our study and regardless of group, exercise decreased HF. Classically, the HF peak, which reflects respiratory sinus arrhythmia, corresponds to parasympathetic drive (25). This result confirms the withdrawal of the vagal outflow to the heart with increasing levels of dynamic exercise already shown by using selective parasympathetic blockade in healthy adult men (29). In low and moderately active subjects, HF decreased only between rest and 30% Wmax, with no further changes. However, in the highly active group, an alteration of sinus node response still persisted with the increase in exercise intensity between 30% Wmax and 60% Wmax, as shown by a further decrease in HF. Our results are in agreement with the study of Tulppo and colleagues (15) performed on young and older male subjects. Moreover, in young active males, although HF is reduced during exercise, it shows a further reduction after atropine, suggesting some degree of persistent vagal drive (30). The administration of atropine causes an increase in resting HR in young subjects, an effect that is blunted in the elderly (31). It suggests a decrease with aging of vagal influence on the heart (32). The gradual vagal withdrawal with exercise intensity observed in our highly active subjects supports the suggestion that regular physical activity compensates this aging process. We have found that quite a high level of physical activity is necessary to obtain a significant effect despite the well-known increase in sympathetic activity could be explained both by a sympathetic overstimulation of sinus node receptors, which prevents a variation of their response (34), and by the inhibition of the baroreflex control of HR (12). Concerning LF/HF ratio exercise time course, controversial results are presented in the literature (13, 26). We observed a decrease in this marker of sympathovagal balance (25) only in low active subjects during exercise. In the same way, we noted a weak but significant positive correlation between LF at 30% VO\textsubscript{2}max. Given their high resting LF/HF level (Table 2), these results suggest an early saturation of sinus node receptors, primarily of sympathetic nervous receptors, in this sedentary population.

LF and HF, expressed in NU, also emphasize the controlled and balanced behavior of the two branches of the autonomic nervous system (25). In our study, whatever the population, HF NU is maintained between rest and 30% Wmax and increased between rest and 60% Wmax in low active subjects. These results are in agreement with previous works performed on sedentary young and elderly people (12,26). At rest, the contribution of nonneural components in respiratory sinus arrhythmia is negligible. During exercise, an increasing proportion of respiratory sinus arrhythmia is due to nonneural mechanism (35). Thus during exercise, HF NU tends to overestimate the parasympathetic component. This increase in HF NU may be due partly to a mechanical stimulus linked to the increase in respiratory activity (35,36). This result is in accordance with a specific ventilatory mode in sedentary subjects, as previously suggested (16). Consequently, LF NU is maintained between rest and 30% Wmax and decreased between rest and 60% Wmax, which confirmed previous findings (12,26). This evolution, which is affected by the exercise intensity, could be explained by the remaining baroreflex responses only at the lower level (36).

Some potential limitations in our study should be considered. First, as the menstrual cycle can alter HRV indexes (37), only postmenopausal female subjects were included in this study. Some of the participants were receiving hormone replacement therapy. The role of this therapy in HRV is controversial (2,38), and our study cannot be used in that discussion. A similar ratio of women receiving hormone replacement therapy, however, was included in each group. Thus all groups would have been similarly affected by potential influences of hormone replacement therapy on HRV. Moreover, individual data of the subjects receiving hormonal therapy were dispersed within the group. Second, our study design cannot rule out the possibility of a genetics bias (39). Indeed, we cannot totally exclude a possible genetic effect on resting heart rate (i.e., intrinsic heart rate) (39). Third, in this study, we have chosen to compare the 3 groups at the same relative level of intensity. It would be interesting to investigate the
effects of training on HRV responses at the same absolute levels of exertion. At last, short data acquisition presents some limits. Indeed, the VLF band, which is defined as below 0.04 Hz and corresponds to the variations of thermoregulation and humoral factors (25), cannot be analyzed, whereas humoral factors are probably important to adapt heart rate during constant load exercise (40).

**Conclusion**
In postmenopausal women, the effects of the levels of physical activity on HR and HRV are dissociated. Quite a high level of physical training induces resting bradycardia without any changes in HRV indexes. In contrast, during exercise the assessment of HRV indexes appears to be more meaningful than at rest. Indeed, a sufficient level of physical training induces a higher sensitivity of sinus node response to exercise stress, which could play a role in thwarting cardiovascular accidents in older women.

**Acknowledgments**
We thank the medical and technical staff of the Centre Cardio-Pneumologique and of the Unité de Biologie et de Médecine du Sport (Rennes), the volunteers for their generous cooperation with our project, and David James for proofreading and rewriting the manuscript.

Address correspondence to Sylvia Reland, Unité de Biologie et Médecine du Sport, CHU Pontchaillou, 2 rue Henri Le Guilloux, 35000 Rennes, France. E-mail: sylvia_reland@yahoo.fr

**References**


Received December 9, 2002
Accepted March 31, 2003
Decision Editor: George E. Taffet, MD