Initial ventilatory and circulatory responses to dynamic exercise are slowed in the elderly

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Ishida, Koji, Yasutake Sato, Keisho Katayama, and Miharu Miyamura. Initial ventilatory and circulatory responses to dynamic exercise are slowed in the elderly. J Appl Physiol 89: 1771–1777, 2000.—To elucidate the characteristics of ventilatory and circulatory responses at the onset of brief and light exercise in the elderly, 13 healthy, elderly men, aged 66.8 yr (mean), exerted bilateral leg extension-flexion movements for only 20 s with a weight around each ankle, with each weight being ~2.5% of their body mass. Similar movements were passively performed on the subjects by the experimenters. These results were compared with those of 13 healthy, young men (22.9 yr). Minute ventilation increased at the onset of voluntary exercise and passive movements in both groups but showed a slower increase in the elderly. Heart rate also increased in both groups but showed less change in the elderly. Mean blood pressure temporarily decreased in both groups but less in the elderly. The magnitude of relative change (gain) of heart rate in the elderly was significantly smaller than that in the young, whereas the increasing rate to reach one-half of the gain (response time) of ventilation in the elderly was significantly slower than that in the young. Similar tendencies were observed in the passive movements. It is concluded that the elderly show slower ventilatory response and attenuated circulatory response at the onset of dynamic voluntary exercise and passive movements.

With increasing age, ventilatory and circulatory responses to exercise should become less capable. For example, Shephard (25) reviewed that a longer time is required for ventilation, heart rate (HR), blood pressure (BP), and oxygen consumption (V̇O2) to attain equilibrium at any given workload with aging. Recently, some investigators maintained that the ventilatory and V̇O2 kinetics (time constant) during phase II, the exponential increase in these parameters from ~20 s after the start of exercise to the steady state, became longer in the elderly than in the young (1, 5). Nevertheless, little is known about the effect of aging on the “phase I” ventilatory response, which was the initial response to exercise within 20 s after exercise onset. Additionally, few investigators have examined HR and BP responses to exercise in the elderly immediately after the start of exercise. It appears that the initial ventilatory and circulatory responses to exercise should be very important in that these responses would affect the next phase of exercise. It is often the case that people become less active with advancing age; however, they sometimes have opportunities to move quickly for a short time (within a 20-s period), such as when crossing a street. If ventilation and HR do not increase during such brief exercise, heavy strain may occur after the cessation of the movement. Accordingly, inquiring into the ventilatory and circulatory responses to brief and light exercise in the elderly is required.

A firm definition of phase I has not been determined; therefore, the methods for analyzing phase I and the interpretation of the results have been conflicting. As for the problem of time (the length of phase I), some investigators (14, 31) who were interested in V̇O2 kinetics assumed that phase I lasted until the end-tidal CO2 partial pressure (PETCO2) began to increase, and others (4, 26) considered that phase I lasted until 15 or 20 s after the onset of exercise. As for the extent of the phase I response, Sietsema et al. (26) defined the phase I value as the plateau value or the value attained 20 s after the start of exercise, whereas we previously determined that the mean of the first two breaths was the extent of phase I (22). On the contrary, when we investigate cardiorespiratory responses to exercise with a step increase in workload, we should take into account both the extent of change of the response from rest to a steady state (gain) and the increasing rate of the response [response time (RT), or time constant]. A considerable number of investigators have previously observed the phase I ventilatory response only from the point of the gain. However, it is possible that the increasing rate of ventilation and circulation within 20 s of the start of exercise may be slowed in elderly people, similar to the phase II response. Despite this, few investigators have studied the speed of the response to brief exercise in the elderly.

Ventilatory and circulatory responses to exercise have been thought to be caused by central command and peripheral neural reflex mechanisms (16). To ex-
amine the effects of the peripheral neural reflex mechanism, passive movements are widely employed because the effect of central command would be reduced to the minimum (9, 23). It is possible that passive movements could lead to defining the factors that cause the different cardiorespiratory responses to exercise that have been observed in the elderly.

The purpose of the present study was, therefore, to clarify the characteristics of ventilatory and circulatory responses to brief and light voluntary exercise in the elderly by comparing these responses to those observed in the young and additionally, if any differences exist, to examine the mechanism that causes the different responses between the elderly and the young, especially by means of passive movements. We hypothesized that ventilatory and circulatory responses at the onset of dynamic exercise would be attenuated or slowed in the elderly.

METHODS

Subjects. Thirteen healthy men [age: 61–73 yr (mean: 66.8 ± 4.3 (SD) yr), height: 163.3 ± 4.0 cm, body mass: 58.9 ± 3.6 kg] volunteered to participate in the present study and were designated the elderly group (ELD). Another group of 13 healthy younger [YNG; age: 18–28 yr (22.9 ± 3.0 yr)] men served as the control group. Their mean height and body mass were 173.6 ± 6.0 cm and 66.2 ± 8.1 kg, respectively. These parameters showed significant differences between ELD and YNG. The proportion of body fat, obtained by a bioelectrical impedance device (model TBF-101, Tanita), was almost equal between ELD and YNG (16.5 ± 4.82 and 17.0 ± 4.26%, respectively), whereas fat-free mass in ELD (49.1 ± 3.72 kg) was significantly lower than that in YNG (54.7 ± 4.78 kg). The subjects were strictly screened before the experiment, and those that had a history of cardiopulmonary diseases, received medications that seriously affect circulatory and respiratory responses, were obese, and/or were current smokers were excluded. All subjects in this study were leading healthy and active lives. The subjects were informed of the experimental protocol and the possible risks involved in this study before giving written consent. Approval for the study was given by the ethics committee of the Research Center of Health, Physical Fitness and Sports at Nagoya University.

Exercise. During the experiment, subjects sat with their backs against an experimental chair with an electrogoniometer attached to the right knee joint. Two weight belts, each equivalent to ~2.5% of the subject’s body mass, were bound around both ankles. Subjects extended and relaxed both knee joints in an alternating pattern from ~110 to 20° in the anatomically flexed position. This voluntary exercise (VOL) began with the right limb, was signaled by the experimenter’s voice just before the start of the inspiratory periods, and continued for ~20 s at the rate of one leg extension and relaxation per second for each leg. The starting point was determined by the flow curve monitored on the oscilloscope. Subjects were instructed to keep the upper body as still as possible. Passive movements (PAS) were achieved by having the experimenter alternately pull ropes that were connected to the subject’s ankles, at the same pace, for the same length of time, and in the same posture as in VOL. All subjects were directed to relax and not resist the motion, and care was taken to immobilize the body as much as possible to avoid motion artifacts and muscle contractions.

Measurements. Tidal volume (VT), minute inspiratory ventilation (VI), expiratory time, and inspiratory time (TI) were measured with the breath-by-breath technique with a hot-wire flowmeter (model RF-H, Minato Ika-gaku) attached to a respiratory face mask. The dead space of the mask was ~100 ml. Respiratory frequency (f) was calculated from total respiratory time. Flow curve was monitored on a storage oscilloscope to ascertain the respiratory timing. End-tidal O2 partial pressure (PETO2) and PETCO2 were obtained by a rapid O2 and CO2 analyzer (model MG-360, Minato Ika-gaku). To determine the circulatory response, HR was calculated beat to beat from R spike using an electrocardiogram through a bioamplifier (model AB-621G, Nihon Kohden). BP was also measured noninvasively beat to beat by using the arterial tonometric method (model JENTOW EX, Colin). In short, the principle of arterial tonometry is that BP at the radial artery can be obtained by measuring the reaction forces produced by flattening the radial. Recently, this method has become preferred over the conventional finger photoplethysmographic method (Finapres), and the accuracy and reliability of BP measured by tonometry can be confirmed when compared with intra-arterial method (11, 24). A tonometric sensor was attached to the left wrist and placed on a padded platform at the level of the heart. The oscillometric measurements were carried out for accurate tonometric measurement before, and sometimes during, the main experiment. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were calculated by the tonometric signal, and mean blood pressure (MBP) was obtained from SBP and DBP. All signals were converted from analog to digital with an analog-to-digital converter board (model ADX-98H, Canopus) and a personal computer (model PC-9821Xa, NEC) at a sampling frequency of 100 Hz for 90 s, beginning and ending ~30 s before and after exercise. The digital data were stored in a hard disk unit and analyzed afterward by means of our own software on a personal computer.

Protocol. A preparatory experiment was carried out to familiarize the subjects with the experimental procedure and to check for abnormality of cardiorespiratory functions. The subjects practiced several movements so as not to respond suddenly to the motions and the experimenter’s voice. The main experiment was performed on a different day at least 2 h after the last meal. Each mode of brief exercise or movement was repeated for six bouts at ~3-min intervals, in a random order, for each subject to avoid order effects. Thereafter, to compare the differences of the extent of the workload between the two groups, the same exercises and movements were continued for 3 min one time, and the mean of the last 30-s value (STD; 3-min steady state value) of the parameters was analyzed. All parameters were measured in the same way as the brief exercise.

Data analysis and statistics. Breath-by-breath and beat-to-beat data were aligned with the onset of exercise and linearly interpolated between each breath or beat to yield a data point at each 1-s interval. Ensemble averaging was done across all six repetitions. To confirm whether the parameters had actually changed as a result of exercise compared with at rest, ANOVA and Dunnett’s t-test (post hoc) were carried out. Resting value (Rest) was the average of 20 s before brief exercise or movement. To compare differences between ELD and YNG, a Komogorov-Smirnov test was conducted to test for differences, and, if the difference was regular, then a two-sample t-test was done. If the difference was not regular, a nonparametric test (Mann-Whitney’s U-test) was performed. The comparison between ELD and YNG consisted of three analyses: 1) comparing Rest, STD, and the relative change obtained by dividing STD by Rest; 2) comparing the...
relative change of $V_i$ and HR during brief exercise normalized by Rest (0%) and STD (100%) in VOL and by Rest (0%) only in PAS, and comparing the relative change of MBP during brief exercise normalized by Rest (100%) in VOL and PAS; and 3) comparing the kinetics of $V_i$ and HR responses in detail. For the third analysis, we regarded the value of the relative change at 15 s after the start of exercise and movement as a gain (Gain), and the time for reaching one-half of the Gain as RT. For MBP response, we considered the minimal value of the relative change during exercise and movement as Gain and the time for reaching the minimal value as RT. These statistical analyses were calculated by computer software (SPSS 8.0, SPSS), and the level of significance was set at $P = 0.05$.

RESULTS

Figure 1 shows the absolute means ± SE of ventilatory responses 20 s before, during, and after VOL and PAS for ELD and YNG. $V_i$ in ELD increased slightly and gradually at the onset of VOL and reached a plateau within $\sim 15$ s. On the contrary, $V_i$ in YNG increased promptly and extensively at the onset of exercise, stopped increasing at $\sim 5$ s, and remained constant until the end of exercise. A similar tendency was observed in PAS. A significant increase ($P < 0.05$) of $V_i$ during exercise compared with Rest was detected in VOL and PAS from immediately after the start of exercise. Respiratory frequency increased quickly and greatly at the onset of exercise in both ELD and YNG. A significant increase ($P < 0.05$) of $f$ during exercise compared with Rest was observed from the onset of exercise without delay. $V_T$ in ELD slightly decreased temporarily after the onset of exercise and returned to the resting level at $\sim 10$ s and thereafter gradually increased, whereas $V_T$ in YNG gradually increased throughout the exercise. A similar tendency was found in PAS. $V_T/f_i$ showed the same change as $V_i$. $P_{ETCO_2}$ in ELD showed little change from Rest to 15 s after the onset of VOL (35.7 ± 3.7 vs. 35.6 ± 4.3 Torr) but began to increase gradually but slightly over the last 5 s (36.3 ± 3.4 Torr at 20 s). $P_{ETCO_2}$ in YNG decreased from Rest (36.8 ± 3.6 Torr) at $\sim 5$ s after VOL began (35.7 ± 3.8 Torr) and returned to the resting value at $\sim 15$ s (36.7 ± 3.9 Torr). $P_{ETCO_2}$ during PAS in both groups showed little change.

Figure 2 represents the cardiovascular responses in ELD and YNG. At rest, HR in ELD was lower than that seen in YNG, but there was no significant difference (Table 1). At the start of VOL, HR in ELD increased in an exponential fashion until $\sim 10$ s and thereafter showed a plateau. On the contrary, HR in YNG increased rapidly at the onset of VOL, increasing progressively until $\sim 15$ s and then slightly decreasing in an overshoot fashion. HR showed a significant increase ($P < 0.05$) during VOL compared with Rest from the beginning of exercise in both groups. HR during PAS demonstrated a similar but smaller change than VOL. HR during PAS was significantly ($P < 0.05$) larger than at Rest from $\sim 3$ s in both groups. After the exercise, HR in ELD decreased slower than that in YNG. Resting MBP, SBP, and DBP in ELD were significantly ($P < 0.05$) larger than those in YNG, but were within the normal ranges for each group. During VOL, these tendencies were retained. MBP, SBP, and DBP in both ELD and YNG began to decrease $\sim 5$ s after VOL began and reached a bottom value at $\sim 10$ s. After 10 s, all BP measurements increased gradually until the end of exercise. After exercise, they decreased.

![Fig. 1. Ventilatory responses before, during, and after voluntary exercise (VOL; left) and passive movements (PAS; right) for 20 s in elderly (ELD) and young (YNG) men (n = 13 for both groups). $V_i$, minute inspiratory ventilation; $V_T$, tidal volume; $f$, respiratory frequency. Values are means ± SE. Time 0 indicates exercise onset.](image-url)
and showed undershoot aspects before returning to the resting level. MBP, SBP, and DBP during PAS demonstrated similar, but smaller, changes.

Table 1 shows the means ± SD of $\dot{V}I$, HR, and MBP at rest during brief exercise and at steady state during 3-min continuous exercise.

<table>
<thead>
<tr>
<th></th>
<th>VOL Rest</th>
<th>VOL STD</th>
<th>Relative Change, %</th>
<th>PAS Rest</th>
<th>PAS STD</th>
<th>Relative Change, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\dot{V}I$, l/min</td>
<td>8.17 ± 1.76</td>
<td>17.28 ± 3.58$^*$</td>
<td>213.4 ± 20.9</td>
<td>7.43 ± 1.68</td>
<td>9.51 ± 1.82$^*$</td>
<td>129.8 ± 16.1</td>
</tr>
<tr>
<td>ELD</td>
<td>8.96 ± 0.93</td>
<td>20.95 ± 4.15</td>
<td>235.2 ± 47.9</td>
<td>8.24 ± 0.99</td>
<td>11.02 ± 1.70</td>
<td>134.5 ± 19.8</td>
</tr>
<tr>
<td>YNG</td>
<td>65.29 ± 7.03</td>
<td>83.74 ± 8.77</td>
<td>130.3 ± 11.3</td>
<td>64.04 ± 7.53</td>
<td>64.79 ± 6.94</td>
<td>101.4 ± 4.5</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>67.17 ± 9.25</td>
<td>87.04 ± 9.85</td>
<td>130.7 ± 15.3</td>
<td>66.56 ± 9.72</td>
<td>67.17 ± 10.53</td>
<td>101.0 ± 8.6</td>
</tr>
<tr>
<td>MBP, mmHg</td>
<td>96.26 ± 13.61$^*$</td>
<td>110.48 ± 14.88$^*$</td>
<td>115.6 ± 15.0</td>
<td>93.38 ± 10.22$^*$</td>
<td>102.75 ± 11.20$^*$</td>
<td>110.4 ± 9.7</td>
</tr>
<tr>
<td>ELD</td>
<td>81.27 ± 7.10</td>
<td>93.83 ± 8.85</td>
<td>116.5 ± 17.2</td>
<td>82.52 ± 8.26</td>
<td>89.71 ± 8.59</td>
<td>109.5 ± 13.5</td>
</tr>
<tr>
<td>YNG</td>
<td>81.27 ± 7.10</td>
<td>93.83 ± 8.85</td>
<td>116.5 ± 17.2</td>
<td>82.52 ± 8.26</td>
<td>89.71 ± 8.59</td>
<td>109.5 ± 13.5</td>
</tr>
</tbody>
</table>

Values are means ± SD. $\dot{V}I$, minute inspiratory ventilation; HR, heart rate; MBP, mean blood pressure; ELD, elderly subjects; YNG, younger subjects; VOL, voluntary exercise; PAS, passive movements; Rest, average of the resting value for 20 s before the brief exercise; STD, average of steady state value during last 30 s of 3-min continuous exercise; Relative change, STD/Rest × 100. * Significant difference between ELD and YNG ($P < 0.05$).

Figure 3 indicates the relative changes of $\dot{V}I$ and HR which were normalized by Rest (0%) and STD (100%) in VOL or by Rest (0%) only in PAS. Relative change of $\dot{V}I$ in ELD was significantly ($P < 0.05$) lower than that in YNG at the first half of exercise during both VOL and PAS. On the contrary, relative change of HR in ELD during both VOL and PAS was significantly ($P < 0.05$) lower than that in YNG throughout the exercise.

Figure 4 represents the kinetics of $\dot{V}I$, HR, and MBP responses. The Gain (15-s value) of $\dot{V}I$ in ELD during VOL and PAS (41.3 ± 20.8 and 29.0 ± 21.7%, respectively) was slightly lower than that in YNG (52.8 ± 12.3% VOL and 43.2 ± 19.6% PAS), but there were no significant differences. Gain of HR in ELD during VOL and PAS (57.0 ± 19.5 and 5.6 ± 3.5%, respectively) was significantly ($P < 0.05$) lower than Gain of HR in YNG (107.1 ± 48.9% VOL, 14.1 ± 9.6% PAS). RT of $\dot{V}I$ in ELD during VOL (4.6 ± 4.8 s) and PAS (5.7 ± 5.6 s) was significantly larger than RT in YNG (1.7 ± 1.4 s).
VOL and 1.2 ± 0.5 s PAS), although there were no significant differences in RT of HR between both groups. Gain of MBP, normalized by Rest (100%), in ELD during VOL (97.1 ± 3.6%) tended to be higher than that in YNG (94.6 ± 4.9%), and Gain of MBP in ELD during PAS (98.1 ± 2.7%) was significantly (P < 0.05) larger than that in YNG (95.1 ± 4.5%), indicating that ELD showed a smaller change than YNG. There were no significant differences in RT of MBP during VOL and PAS.

DISCUSSION

In this study, we attempted to elucidate the ventilatory and circulatory responses at the onset of dynamic exercise in the elderly. The results are as follows. 1) Whereas V̇I increased to a similar level at the latter part of exercise for 20 s in both ELD and YNG, the RT of V̇I in ELD was larger than that in YNG. 2) RT of HR was similar in both groups, but Gain was attenuated in ELD. 3) MBP decreased temporarily at the start of VOL in both groups, although the extent of decline was reduced in ELD. PAS produced a similar change. Consequently, the elderly should have slowed ventilatory response and attenuated circulatory response at the start of exercise.

Methodology. When comparing cardiorespiratory responses among subjects or different groups, the setting of the workload should be a matter of serious debate. Unfortunately, we did not measure maximal muscular strength of leg extension and maximal VO₂. However, the workload (2 weight belts, each equaling 2.5% of the subject’s body mass) per unit of fat-free mass, which is assumed to be well correlated with the muscle mass, did not show any significant difference between ELD and YNG (2.89 ± 0.17 and 2.97 ± 0.16%, respectively); thus the exerted force, i.e., the load to thigh muscles, would be relatively equal between the two groups. Judging from the STD exercise exerted in the same way as the brief exercise, there was no significant difference in relative changes of V̇I, HR, and MBP between ELD and YNG; therefore, the relative load to the respiratory and circulatory systems, compared with the resting values, would be almost identical between both groups. On the contrary, the relative demands estimated from peak value during maximal exercise may be greater for ELD because it is well accepted that maximal V̇I, HR, and VO₂ at maximal exercise decrease with aging (25). However, this should not weaken our findings, because the ELD did show slowed ventilatory and attenuated circulatory responses; nevertheless, the cardiorespiratory system must operate closer to the maximum in ELD than in YNG. Considering these points, it may safely be assumed that the workload used in the present study would not be a serious problem.

When we investigate the cardiorespiratory response to exercise with a step increase in work load, representing the response by means of the Gain and RT can bring us more detailed analyses of the response. We determined the 15-s value as Gain because 1) the lag time for circulation until the metabolites reached the carotid body was, in general, assumed to be ~15 s (2, 7); 2) in the present study, PETCO₂ in ELD began to increase after ~15 s compared with Rest; 3) average V̇I in ELD showed a plateau from ~15 s; and 4) HR demonstrated a peak value at ~15 s and decreased thereafter (overshoot). Indeed, time constant has been widely used in analyzing VO₂ kinetics, but our data were too limited to fit the exponential function, and, additionally, time constant cannot be calculated if the data...
demonstrate an overshoot (such as our data did). Thus we regarded the time when \( V_i \) and HR reached one-half of the Gain, or MBP reached the minimal value, as the index of the RT.

**Ventilatory response.** In the present study, there was no significant difference in Gain of \( V_i \) during exercise between ELD and YNG. This result indicates that the extent of \( V_i \) response at the start of exercise should be almost equal between the elderly and young. However, \( V_i \) in ELD showed a gradual increase and a longer RT than that of YNG. These observations provide strong evidence that the elderly should have a slower ventilatory response at the onset of exercise. This slowed \( V_i \) response was caused by the attenuated \( V_i \) response in ELD, especially for the first 10 s, despite the rapid increase in \( f \) in both groups. In addition, \( V_i/T_i \), which was assumed to be the index of the ventilatory drive (17), slowed more in ELD than in YNG and may indicate that ventilation in the elderly would be driven slowly at the onset of exercise.

The reason why the phase I response in ELD was slowed should be considered from two points: 1) neural inputs to the respiratory center and 2) output organs from the respiratory center. First, it is well known that the neurogenic drives for phase I are redundantly derived from central command from the motor cortex and/or hypothalamus and peripheral neural reflex (16). In the present study, PAS, which should be affected little by central command, produced similar results as VOL; thus it is assumed that peripheral neural reflex certainly plays a role in this slowed response in the elderly. It is possible that the sensitivity of the mechanoreceptors, which are presumed to be the primary sense organ of the peripheral reflex, might be reduced in the elderly. Previous investigators have observed degenerated peripheral sensory organs, i.e., decreased dynamic sensitivity of muscle spindle primary endings in aged rats (20) and deteriorated joint-position sense (proprioception) with increasing age in humans (27). Although it is also likely that the number of mechanoreceptors may decrease in the elderly, it was not clearly determined in the present study that the number and sensitivity of the mechanoreceptors and/or the afferent nerves were actually decreased in the ELD. Moreover, it is certain that the central nervous system degenerates with age, e.g., decreases in the number of frontal cortical synapses in those over the age of 60 yr (15), but it is not yet obvious how and to what extent the central nervous system components such as the motor cortex, hypothalamus, and respiratory center, in relation to phase I response, are affected by increasing age. Further investigation is required.

Concerning the output organs, Sparrow and Weiss (28) stated that many parameters of pulmonary function decline steadily with advancing age in humans, by virtue of the decrease in the strength of respiratory muscles, the stiffness of the chest wall, and the closure of small airways. We previously observed the attenuated phase I ventilatory response to light exercise during combined \( \beta \)-adrenergic and cholinergic blockades in normal adult subjects and suggested the possibility of the effect of airway dilation on the phase I response (8). Airway resistance decreases promptly at the onset of exercise, by virtue of rapid parasympathetic withdrawal (10), unlike in the elderly >60 yr old, who have attenuated parasympathetic nervous functions (30). However, it is doubtful that these respiratory dysfunctions would actually affect the ventilatory response of the elderly within a very short time at the onset of light exercise such as that used in the present study. Additional study is needed.

**HR response.** In the present study, Gain in HR was lower in ELD than that in YNG, although RT showed no significant difference between them. This was different from the ventilatory response. With regard to the input side of the circulatory center, it is assumed that the rapid increase in HR at the onset of exercise is derived from the central command and peripheral neural reflex redundantly, similar to ventilation (13, 18), allowing neural inputs to the circulatory center to affect the response. On the contrary, the output mechanism is quite different between respiration and circulation. Many previous investigators (3, 19) have revealed that a rapid increase in HR at the start of exercise is caused by a rapid withdrawal of the parasympathetic nervous system. As we have already mentioned, the activity of the parasympathetic nerves in the elderly is already reduced at rest and is not further inhibited by the onset of exercise; thus HR in the elderly is only able to be increased slightly (30). Furthermore, it is supposed that the functions of the heart as an effective organ become attenuated with increasing age. For example, myocardial contraction time is prolonged, and the filling rate of the left ventricle is reduced in healthy older individuals (12).

The notch phenomenon, which is the temporary HR overshoot, occurs not only at the start of light exercise with step load changes (6) but also at the start of passive movements (21). In the present study, most, but not all, YNG showed notch at the onset of VOL and PAS, although few ELD showed notch. The cause for the notch phenomenon was assumed to be derived from the unbalance of the sympathetic and parasympathetic nervous systems (6). The lack of notch in ELD may be due to the attenuated parasympathetic nervous system. Accordingly, weakened parasympathetic nervous system in the elderly would have some influence on lowered HR response. However, as we did not measure parasympathetic nerve activity, further research is required.

**BP response.** MBP temporarily and significantly decreased ~10 s after the onset of VOL compared with at rest and thereafter increased in both groups. The relative drop of MBP in ELD was nonsignificant but rather small compared with that in YNG during VOL and significantly smaller during PAS. Previous investigators (3, 29, 32) reported the transient MBP fall at the onset of dynamic exercise in normal adult subjects and suggested that the causes were due to decreases in pressure reflex and total peripheral resistance. Moreover, peripheral mechanoreceptors may have some influence on the response because a similar MBP drop.
occurred during PAS in the present study, and Nóbrenga et al. (23) suggested that mechanoreceptors activated by passive movements could cause the pressor response. The explanations for the attenuated MBP drop in ELD were attributed to the following: 1) lowered pressure reflex in the elderly, 2) difficulty in decreasing peripheral resistance because of reduced elasticity of blood vessels in the elderly (12, 25), and 3) attenuated neural inputs to the circulatory center, such as mechanoreceptors. More detailed investigation into BP response is necessary due to some of the limitations of our study; however, the initial fall of MBP in this study was not very important because the relative change of BP at the onset of exercise was much smaller than that of HR and ventilation.

In conclusion, ELD had slower ventilatory response, attenuated HR increase, and reduced temporal fall of BP at the onset of VOL and PAS compared with YNG. It is suggested that nervous functions, such as the peripheral neural reflex, play a role in these slowed and attenuated responses in the elderly.

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REFERENCES


