Effect of Bicycling on the Baroreflex Regulation of Pulse Interval

By J. D. Bristow, E. B. Brown, Jr., D. J. C. Cunningham, M. G. Howson, E. Strange Petersen, T. G. Pickering, and P. Sleight

ABSTRACT

The reflex control of pulse interval during erect bicycle exercise was studied in nine normal subjects aged 18 to 28, breathing an oxygen-rich mixture. Rises in the directly measured arterial pressure were produced by intravenous injections of phenylephrine, and systolic pressure was correlated with the following pulse interval. The slope of the systolic pressure-pulse interval relation was used to express reflex sensitivity. With increasing exercise, the reflex sensitivity decreased progressively so that at a level of exercise which produced a heart rate of 150/min there was no reflex cardiac slowing in response to a provoked rise of pressure. The reduction in reflex sensitivity was studied during the changes from rest to exercise and vice versa. Reflex sensitivity increased within 5 seconds of the onset of exercise.

KEY WORDS

arterial pressure  baroreceptors  phenylephrine

The inverse relation between heart rate and arterial blood pressure was recognized by Marey (1) more than a century ago, and its underlying reflex mechanism has been well described (2). With the onset of muscular exercise, however, both heart rate and arterial pressure rise, indicating that the relation changes. The change could be a resetting of the reflex at a new level appropriate to the exercise, or a suppression of the reflex, or both. Two previous groups of workers (3, 4) have investigated the reflex during exercise; both concluded that it was still effective, thus agreeing with the statement of Heymans and Neil (2): "It is a common mistake to claim that the baroreceptors are less active in exercise. . . ."

A quantitative method for expressing baroreflex sensitivity in man has been described (5): a rise of systolic pressure produced by the alpha-adrenergic drug phenylephrine, which does not affect heart rate directly (6), is related beat by beat to the reflex lengthening of pulse interval. The present experiments were undertaken to examine changes in this reflex immediately after the onset and the end of exercise and also during the steady state; three preliminary reports have appeared (7-9). Our results, but not our conclusions, are in agreement with those of the previous workers.

Methods

Brachial artery pressure was recorded via an intraarterial polythene cannula (i.d. 1 mm) connected to a strain gauge transducer. The tip of the cannula lay at the termination of the subclavian artery. Pulse interval was measured from the arterial pressure record, which was taken on paper moving at 25 mm/sec, on magnetic tape, or on both. A second cannula was inserted in an antecubital vein, through which repeated sudden injections of phenylephrine were given. All but one of the subjects (who breathed air) breathed an oxygen-rich mixture ($P_{\text{O}_2} = 200$...
Comparison of two methods of expressing baroreflex sensitivity. Horizontal axis, as change of pulse interval per unit change of systolic pressure, vertical axis, as change of pulse interval per unit change of mean pressure. The results are pooled data from four subjects. Each point represents one injection of phenylephrine; injections were made both at rest and during exercise. The diagonal line is the line of identity.

EXPERIMENTAL PROCEDURE

In the experiments on steady-state exercise and the transients between rest and exercise, the subjects were four males and one female, aged 20 to 28 years. One of the males was in exceptionally good physical training. All subjects were asked to avoid caffeine and smoking the night before each experiment, as such substances might affect circulatory function.

The reflex sensitivity was calculated by plotting the pulse interval of each beat against the systolic pressure of the preceding beat, starting from the end of the injection and proceeding to the peak systolic pressure. We have treated the relation as linear and have expressed reflex sensitivity as the slope (regression coefficient) of the line. In the early experiments (rest and steady-state exercise), inspiratory pulses were eliminated from the calculation to lessen the effects of sinus arrhythmia. Later analysis, however, did not show significantly worse correlations when all pulses were included; regression coefficients were, on average, 9% lower. The method is described in more detail elsewhere (5).

In the later experiments (graded exercise), systolic, mean, pulse, and diastolic pressures and pulse interval were obtained by computer from the magnetic tape recordings.

For the experiments on graded exercise the regressions of pulse interval on pressure were calculated using both systolic and mean pressures. The slopes and correlation coefficients at rest and during exercise were virtually identical (Fig. 1). Diastolic pressure was nearly as well correlated with pulse interval, but pulse pressure was little changed by the drug and so gave very poor correlations.

In the experiments on steady-state exercise and the transients between rest and exercise, the subjects were four males and one female, aged 20 to 25 years. One of the males was in exceptionally good physical training. All subjects were asked to avoid caffeine and smoking the night before each experiment, as such substances might affect circulatory function.

Two or three injections of phenylephrine were given while the subject was resting on the bicycle, the effects of the drug on pressure and heart rate are very brief when given intravenously (Pickering et al., unpublished observations), so injections could be given at 3-minute intervals.

The dose was adjusted to give a systolic pressure rise of about 20 mm Hg; larger doses were needed to produce the pressure rise during exercise (up to 180 \( \mu \)g) than at rest (50 to 100 \( \mu \)g). The subjects did not know when the injections were being given.

The transient changes at the beginning of exercise were studied six to eight times in each experiment, using one level of exercise (Table 1). The flywheel of the ergometer was set in motion by an electric motor, a quiet command to start was given by an experimenter standing behind the subject, and the drive on the flywheel was disconnected. Phenylephrine or saline was injected either 5 seconds before or 5 seconds after the command. The subject stopped exercising a minute later and the procedure was repeated after 2 minutes of rest. The effects of such short bouts of exercise are quickly reversible (20).

There followed a period of continuous exercise at a work load of 57 to 60 watts (oxygen consumption about 1 liter/min). After at least 7 minutes of pedaling, three injections of phenylephrine and one or two injections of saline were given at 3-minute intervals. Study of the transient changes at the end of exercise followed the same pattern as those at the beginning (steps for 1 minute, pedal for 2, stop for 1 . . .). Finally, three injections of phenylephrine were given between the second and tenth minute of recovery.

In two other male subjects, steady exercise continued at a single load for a period long enough for 26 and 10 phenylephrine injections, respectively, spaced 3 minutes apart.

The second series of experiments was devoted to a study of the effects of several grades of steady bicycle exercise in one female and three male subjects aged 18 to 21. After three to five determinations of the reflex sensitivity at rest, the
subjects exercised at 12.5, 25, 50, 101, and (the men only) 151 watts, in ascending and then
descending order of work intensity, with no
breaks between the levels. At each work intensity,
three phenylephrine injections were given at 3-
minute intervals after a steady heart rate and
blood pressure had been reached (4 to 6
minutes); each work load therefore lasted for
about 10 to 12 minutes. Three further injections
were given during recovery.

Results

REST AND STEADY-STATE EXERCISE

Figure 2 shows the effects of a single
injection of phenylephrine at rest and during
exercise. The relation between pulse interval
and systolic pressure is effectively linear in
both states. It is evident that during exercise
the sensitivity of the reflex (slope of the line)
have been greatly reduced; the setting of the
reflex has also changed in the sense that the
points representing the preinjection values of
pulse interval and systolic pressure in exercise
do not lie on the line for rest (Fig. 2). A more
elaborate but more precise definition of
baroreflex setting has been given previously
(11, 12).

The mean results of the first seven experi-
ments (those at a single work load) are shown
subject by subject in Table 1; graphs illustrat-
ing the extreme examples (for the athlete,
subject 4, and the girl, subject 1) have already
been published (7).

In each experiment, all the exercise lines
had slopes which were numerically lower than
any slope determined for rest or recovery; in
other words, reflex sensitivity as we define it
was reduced in exercise. The athlete showed
the smallest change in both slope and heart
rate, the girl showed the largest, and the other
subjects showed intermediate changes; per-
haps this variation reflects the varying degrees
of physical fitness.

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rate, the girl showed the largest, and the other
subjects showed intermediate changes; per-
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of physical fitness.

Inspection of Table 1 shows that the more
the diminution of pulse interval (i.e., the
greater the exercise tachycardia), the greater
was the depression of reflex sensitivity as
expressed by slope. The correlation between
these variables for exercise is high and very

\[ r = 0.82 \]

\[ r = 0.82 \]

\[ r = 0.82 \]

\[ r = 0.82 \]

\[ r = 0.82 \]
Table 1

Mean Steady-State Values of Pulse Interval and Systolic Pressure before Drug Injection (Io, and Po) and of Slope of I-P Relation after Drug Injection.

| Subject | Condition | Io (msec) | Po (mm Hg) | Slope (AL/AP) | p
<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>Rest</td>
<td>631</td>
<td>90</td>
<td>16.0</td>
<td>3</td>
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<tr>
<td></td>
<td>Exercise</td>
<td>454</td>
<td>120</td>
<td>1.5</td>
<td>3</td>
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<tr>
<td></td>
<td>Recovery</td>
<td>552</td>
<td>87</td>
<td>11.6</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>Rest</td>
<td>732</td>
<td>137</td>
<td>25.7</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Exercise</td>
<td>511</td>
<td>154</td>
<td>7.0</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Recovery</td>
<td>654</td>
<td>157</td>
<td>13.7</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>Rest</td>
<td>406</td>
<td>556</td>
<td>2.1</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Exercise</td>
<td>435</td>
<td>954</td>
<td>773 ± 33</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Recovery</td>
<td>529</td>
<td>145</td>
<td>12.4</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>Rest</td>
<td>540</td>
<td>135</td>
<td>20.5</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Exercise</td>
<td>774</td>
<td>121</td>
<td>18.2</td>
<td>2</td>
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<tr>
<td></td>
<td>Recovery</td>
<td>669</td>
<td>131</td>
<td>19.5</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>Rest</td>
<td>858</td>
<td>118</td>
<td>13.8</td>
<td>3</td>
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<tr>
<td></td>
<td>Exercise</td>
<td>760</td>
<td>855</td>
<td>6.1</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Recovery</td>
<td>555</td>
<td>120</td>
<td>12.7</td>
<td>3</td>
</tr>
<tr>
<td>6</td>
<td>Rest</td>
<td>837</td>
<td>131</td>
<td>16.5</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Exercise</td>
<td>837 ± 14</td>
<td>142 ± 6</td>
<td>16.1 ± 2.4</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Recovery</td>
<td>846</td>
<td>147</td>
<td>15.9</td>
<td>1</td>
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<tr>
<td>7</td>
<td>Rest</td>
<td>922</td>
<td>50</td>
<td>11.2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Exercise</td>
<td>723 ± 22</td>
<td>116 ± 3</td>
<td>7.4 ± 3.5</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Recovery</td>
<td>628</td>
<td>106</td>
<td>8.9 ± 5</td>
<td>5</td>
</tr>
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</table>

*Girl. fAthlete. N = number of injections. Standard deviations of repeated determinations.

Table 2

Correlations of Reflex Sensitivity (AL/AP) with Initial Pulse Interval (Io), Initial Systolic Pressure (Po), and with the Ratio of Io to Po.

<table>
<thead>
<tr>
<th>Group</th>
<th>Correlation Coefficient</th>
<th>p</th>
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<tbody>
<tr>
<td>Rest</td>
<td>0.502</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Exercise</td>
<td>0.925</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Subgroups are those in Table 1.
Mean Steady-State Values of I, Po, and Slope of I-P Relation after Drug Injection at Rest and during Work of Varying Intensities

<table>
<thead>
<tr>
<th>Condition</th>
<th>I</th>
<th>Po</th>
<th>Slope (ΔI/ΔP)</th>
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</thead>
<tbody>
<tr>
<td>Rest</td>
<td>1.3</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Work (50)</td>
<td>13</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Recovery</td>
<td>25</td>
<td>69</td>
<td></td>
</tr>
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<tr>
<th>Subject</th>
<th>I</th>
<th>Po</th>
<th>Slope</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>489</td>
<td>92</td>
<td>21.8</td>
</tr>
<tr>
<td>9</td>
<td>840</td>
<td>820</td>
<td>716</td>
</tr>
<tr>
<td>10</td>
<td>650</td>
<td>580</td>
<td>526</td>
</tr>
<tr>
<td>11</td>
<td>583</td>
<td>510</td>
<td>441</td>
</tr>
</tbody>
</table>

N = number of injections.

Mean Steady-State Values of I, Po, and Slope of I-P Relation after Drug Injection at Rest and during Work of Varying Intensities

<table>
<thead>
<tr>
<th>Condition</th>
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<td>583</td>
<td>510</td>
<td>441</td>
</tr>
</tbody>
</table>

The same tendency was seen in all four subjects.

In a single individual, heart rate and work load are closely correlated (13), and we would expect, therefore, that pulse interval and slope, both of which diminish with work load, should themselves be positively correlated. This is found to be the case (Fig. 3, right). Of course it is not possible for the relations between all these pairs of variables to be linear. Nevertheless, the linear correlation coefficient between slope and preinjection pulse interval is higher than for slope and work load in three out of four subjects (Table 4). This correlates with the observation that when the same work load was used at different stages of an experiment, the values of slope were often different, following the changes of pulse interval rather than of work load (Table 3).

TRANSITIONS BETWEEN REST, ONE LEVEL OF EXERCISE, AND RECOVERY

One of the requirements of the phenylephrine method of analyzing the baroreflex is that there should be a steady baseline level of both systolic pressure and pulse interval, so that when the pressure is raised by the drug, the resulting changes of interval are produced only by the reflex. Since both variables are changing during the transients at the beginning and end of exercise, it is necessary to distinguish those changes occurring as part of the transition process from those due to the drug. The procedure by which this was done was as follows. For each subject two or three injections of saline and four to six of phenylephrine were carried out at the start ("on-transient") of exercise; the phenylephrine injections were done just before or just after the start so that the pressure rise came at different times. Then the records were analyzed in successive 5-second periods, taking zero time as the start of exercise, and proceeding for 30 seconds. Figure 4 shows these changes plotted as the average results for each type of injection during the transitions between the steady states of rest, work, and recovery.
BAROREFLEX DURING EXERCISE IN MAN

FIGURE 3

Left: Each line is the regression line for one injection of phenylephrine; the positions of the symbols show the preinjection values of pulse interval and systolic pressure. The work load was increased in stages from 13 to 151 watts. Right: Same data plotted in a different way. Each point represents one injection. For each injection the slope of the regression line (dA/dP) has been plotted against the preinjection pulse interval (I). The line is the regression line for this relation. Subject 8. Open diamonds = resting supine; other symbols as in Figure 2.

FIGURE 4

Changes of systolic pressure (top panels) and pulse interval (bottom panels) in subject 5 for the transition periods at the beginning and end of exercise. The steady state values are shown by +. Solid lines, saline injections; broken lines, phenylephrine injected just before the transition (first arrow); dotted lines, phenylephrine injected just after the transition (second arrow).
exercise, and recovery for one subject. For the un-transient it can be seen that the systolic pressure rose the same amount for all three types of injection (early and late phenylephrine, and saline) until about 10 or 15 seconds, when the early phenylephrine injections produced a further rise relative to the line for saline. This rise was associated with a moderate bradycardia. The rise due to the late phenylephrine injection occurred about 10 seconds later, and at this stage the bradycardia was less pronounced. The phenylephrine injections at the "off" produced about the same rise of systolic pressure (Fig. 4, right), but the associated bradycardia was greater than at the corresponding stage at the "on." Therefore, Figure 5 shows, the results of both early and late phenylephrine and the saline injections were pooled for each 5-second period in each subject, and plots of pulse interval against systolic pressure (for expiratory beats only) were made. In the lower panels shown in the figure (10 to 30 seconds after the start) the phenylephrine points are separated from the saline ones, and there was therefore sufficient spread in the values for the regression of pulse interval on systolic pressure (calculated for all the points) to reach significance \( P < 0.05 \). In the first two periods, however, the phenylephrine has not yet produced any change, so there is no separation of the phenylephrine points from the saline ones, and we can say nothing about the sensitivity of the reflex. The procedure for analyzing the "off-transients" at the end of exercise was the same. The regression lines in Figure 5 show that by 10 or 25 seconds the slope is already beginning to flatten, and by 30 seconds the full depression found in steady-state exercise has occurred. The changes at the end of exercise are equally rapid.

The percent changes of pulse interval, systolic pressure, and reflex sensitivity for both sets of transients are summarized for four of the five subjects in Figure 6 (the athlete showed such small changes at this level of exercise that his transients were not worth analyzing, and neither were the pressure

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**FIGURE 5**

Same data as Figure 4 for the transient at the beginning of exercise. Pulse interval plotted against systolic pressure for the pooled data of each 5-second period in succession. + = saline; solid circles = phenylephrine injected before start; open circles = phenylephrine injected after start. The thin lines show the steady state values for rest and exercise, and the thick lines the regression lines during the transients. The numbers by each line are the slopes. Phenylephrine points occurring after the peak of pressure are omitted.
BAROREFLEX DURING EXERCISE IN MAN

On transients

Off transients

Seconds after start

20 30

Seconds after stop

Changes of pulse interval (top panels), systolic pressure (middle panels), and reflex sensitivity (bottom panels) occurring at the beginning and end of exercise for four subjects, each denoted by a different symbol. The lines are the average changes. Only significant values of reflex sensitivity are plotted.

Discussion

The main conclusion from these results is that the change in the relation between pulse interval and arterial pressure which occurs during exercise takes the form of a loss of baroreflex sensitivity and also a resetting of the reflex, when setting is defined as previously (11). A recent study of the effects of electrical stimulation of the carotid sinus nerve in man (14) showed that changes of heart rate were slightly smaller during exercise than at rest, although in both cases they were too closely linked to pulse interval than to systolic pressure, just as in the steady state (Fig. 3, right).
small for the differences to be significant. Our conclusions are, however, at variance with those of the other two groups who have tackled the problem, and the reasons for the discrepancy require examination. Bevegard and Shepherd (3) stimulated the carotid baroreceptors by applying suction to a rigid box enclosing the neck and measured the changes in mean arterial pressure and heart rate. They reported similar changes in rate during rest and exercise (Robinson et al. (4) raised the pressure with sustained infusions of phenylephrine, but they too expressed the response in terms of mean pressure and heart rate. Furthermore, the hardest level of exercise studied by both groups was comparatively mild (heart rate < 125/min). Examination of our data together with those of these two studies shows that the discrepancy lies in the analysis rather than in the results.

The results reported here are expressed in terms of the systolic pressure in the subclavian artery, and it may be objected that the waveform at this site is an imperfect index of the waveform at the carotid sinus or aortic receptors. Furthermore, the baroreceptors are known to respond to rate of change of pressure and to the frequency of distension (15) as well as to the steady level of pressure. With the drug-induced transient rises of pressure, the systolic, diastolic, and mean pressures rise together, and so there is little change in pulse pressure; it seems, therefore, that the method reveals primarily the response of the system to changes in mean pressure rather than to dynamic components of the pressure wave. In keeping with this is the finding that pulse interval is correlated about equally well with systolic and mean pressures, and much better with both of these than with pulse pressure. It seems that the difference in interpretation is thus not due to our use of systolic rather than mean arterial pressure.

The other difference is that their (3, 4) results are expressed in terms of heart rate, whereas we have used pulse interval. The plot of pulse interval against systolic pressure is effectively linear and the plot of its reciprocal, heart rate, against pressure is hyperbolic. Valid comparisons only be made between the slopes of straight lines, but comparison of the "slopes" of hyperbolas are generally invalid. When the heart rate-pressure plot is used, the subjects studied by these authors together with our own first five experiments show a decrease of reflex sensitivity in seven cases, no change in one, and an increase in four. When the pulse interval-pressure plot is used, all three groups' data show a diminution of reflex sensitivity. Furthermore, in hard exercise, when pulse interval falls below 400 msec (heart rate > 150/min), reflex sensitivity, however expressed, approaches zero. The apparent discrepancy thus depends on how the results are analyzed and also on the level to which exercise is taken. There are also theoretical reasons for expressing the effects of autonomic activity on the heart as change of interval rather than its reciprocal (12).

During steady-state exercise, there is a close relation between the slopes of the reflex lines and the preinjection pulse interval such that, at a value of pulse interval of about 400 msec, the reflex is wholly abolished. This relation is not just an artifact of the method of analysis of the transient changes of blood pressure which we have used because steady-state experiments (3, 4), inasmuch as they are comparable, give the same results when plotted in this way. The same relation does not necessarily apply during the transition between rest and exercise, and recovery, for reflex sensitivity appeared to change even more quickly than pulse interval (Fig. 6). Pulse interval is itself closely linked to the intensity of exercise (whether expressed as oxygen consumption or external work rate); work rate too is correlated with the degree of suppression of baroreflex sensitivity, but usually not as closely as is pulse interval (Table 4).

We do not know the reasons for the diminution of sensitivity of the cardiac component of the baroreflex during exercise. At the receptor level, all the effective stimuli to the baroreceptors are probably increased, although the aortic pressure may be little changed (16). It is possible that a given
BAROREFLEX DURING EXERCISE IN MAN

The baroreflex is less effective when superimposed on a high background level of stimulation than on a low one, if the baroreceptors operate on a Weber-Fechner basis (17). Smyth et al. (5, their Fig. 6), however, assessed the baroreflex sensitivity in sleeping subjects at naturally occurring arterial pressures and also at pressure elevated by 10 to 15 mm Hg by steady infusion of angiotensin; the high reflex sensitivity associated with sleep was not lessened by raising the arterial pressure, and hence also the background stimulation of the receptors. Similarly, in Figure 3 (left) of the present paper, it may be seen that the steady background arterial pressure during recumbency was higher than during rest sitting on the bicycle; nevertheless, as in the sleeping subjects, the slopes were higher during recumbency than during rest sitting up, being related to pulse interval rather than to systolic pressure.

In the cat (18) and dog (19, 20), increasing the background level of sympathetic stimulation has little effect on the response of pulse interval to vagal stimulation, and it is therefore unlikely that interaction between vagus and sympathetic activity at the sinoatrial node could account for more than a small part of the diminution of response reported here.

Within the central nervous system it is possible that the great increase in general afferent activity (e.g., from working muscles) is sufficient to reduce the central effects of the baroreceptor on the cardiovascular center; the reverse phenomenon may account for the increase in reflex sensitivity seen in sleep (5). Further modulation may be provided by changes in the complex of chemical stimuli (12) such as occur in steady-state exercise.

Whatever the mechanism may be, baroreflex sensitivity is another variable in the circulatory system that demonstrably is closely adjusted to the intensity of exercise. More specific central nervous effects on cardiovascular responses have also been described in animals (21, 22) and in man (23).

References
Effect of Bicycling on the Baroreflex Regulation of Pulse Interval
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