Low-frequency arterial pressure fluctuations do not reflect sympathetic outflow: gender and age differences

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1 Hebrew Rehabilitation Center for Aged, Beth Israel Deaconess Medical Center, and Harvard Medical School Division on Aging, Boston, Massachusetts 02167; 2 Department of Kinesiology, University of Colorado, Boulder 80309; and Department of Medicine (Cardiology and Geriatric Medicine), University of Colorado Health Sciences Center, Denver, Colorado 80262

Taylor, J. Andrew, Todd D. Williams, Douglas R. Seals, and Kevin P. Davy. Low-frequency arterial pressure fluctuations do not reflect sympathetic outflow: gender and age differences. Am. J. Physiol. 274 (Heart Circ. Physiol. 43): H1194–H1201, 1998.—Low-frequency arterial pressure oscillations (Mayer waves) have been proposed as an index of vascular sympathetic outflow. However, cross-sectional differences in these pressure oscillations may not reflect different levels of sympathetic nervous outflow in humans. Three groups of healthy subjects with characteristically different sympathetic nervous outflow were studied: young females (n = 10, 18–28 yr), young males (n = 11, 18–29 yr), and older males (n = 13, 60–72 yr). Average R-R interval, arterial pressures, and systolic pressure variability at the Mayer wave frequency (0.05–0.15 Hz) did not differ among the three groups. Diastolic pressure Mayer wave variability was similar in young females vs. young males (39 ± 10 vs. 34 ± 5 mmHg<sup>2</sup>) and lower in older males vs. young males (14 ± 2 vs. 24 mmHg<sup>2</sup>; P < 0.05). In contrast, muscle sympathetic activity was lowest in young females (892 ± 249 total activity/min) and highest in older males (3,616 ± 528 total activity/min; both P < 0.05 vs. young males: 2,505 ± 285 total activity/min). Across the three groups, arterial pressure Mayer wave variability did not correlate with any index of sympathetic activity. Our results demonstrate that arterial pressure Mayer wave amplitude is not a surrogate measure of vascular sympathetic outflow.

autonomic nervous system; power spectral analysis; heart rate variability

THE ORIGINAL OBSERVATIONS of arterial pressure waves at a frequency lower than the respiratory rate indicated that this oscillation may arise from an inherent rhythmicity in cardiovascular vasomotor centers (11, 14, 28). Subsequent research refined this hypothesis, demonstrating that 10-s arterial pressure Mayer waves can be produced by a central (37) or baroreceptor (15)-mediated sympathetic oscillation coupled with a delay in the α-adrenergic vasoconstrictor response (25). Although most postulated mechanisms necessitate a vascular sympathetic effector for Mayer wave generation, they do not require proportionality between vascular sympathetic tone and Mayer wave amplitude. Despite this, differences or alterations in low-frequency arterial pressure variability under conditions that prominently change autonomic tone in humans have been presumed to represent proportional changes in vascular sympathetic outflow (12, 24, 27, 38), although the proportionality between low-frequency arterial pressure variability and directly measured sympathetic nervous activity is unclear (33). Findings in animals suggest that Mayer wave amplitude is not proportional to the level of mean sympathetic nervous outflow (46) but that it is related to the level of sympathetic oscillations (36).

We hypothesized that mean sympathetic activity and Mayer wave amplitude are not correlated but that the levels of Mayer wave frequency oscillations in arterial pressure and sympathetic activity are correlated. To elucidate whether cross-sectional differences in Mayer wave amplitude represent any aspect of vascular sympathetic activity, we assessed sympathetic activity and Mayer wave amplitude in three groups of healthy subjects with characteristically different levels of muscle sympathetic outflow. Young females exhibit less basal sympathetic nerve activity than young males, who in turn exhibit less basal sympathetic nerve activity than older males (30). Frequency domain analysis of arterial pressures and directly measured muscle sympathetic nerve activity afforded insight to Mayer wave amplitude and its relation to average sympathetic nervous outflow and to sympathetic nervous oscillations. Our results suggest that cross-sectional differences in Mayer wave amplitude do not provide insight to differences in vascular sympathetic outflow.

METHODS

Subjects. We assessed data from 10 young females [aged 18–28 yr; mean, 22 ± 4 (SE) yr], 11 young males [18–29 yr; mean, 24 ± 4 (SE) yr], and 13 older males [60–72 yr; mean, 65 ± 4 (SE) yr]. Subjects were not on medication, were not obese (body mass index range 17.9–28.7 for all subjects), and were not smokers. All older subjects were free of any signs or symptoms of overt coronary heart disease based on medical history and resting and maximal exercise electrocardiograms. The study protocol was approved by the Institutional Review Boards of the University of Colorado and the Hebrew Rehabilitation Center for Aged. All subjects gave verbal and written informed consent.

Protocol and measurements. Lead II of the electrocardiogram, beat-to-beat arterial pressures, respiratory excursions, and muscle sympathetic nerve activity were recorded continuously during 5 min of controlled frequency breathing (15 breaths/min, 0.25 Hz) with subjects in the supine position. Figure 1 shows representative tracings from a young female, young male, and older male.

Beat-to-beat arterial pressure was estimated from the middle phalanx of the middle finger of the left hand with a photoplethysmograph (Finapres, model 2300, Ohmeda). This device has been validated for power spectral analysis of arterial pressure variability (32). Brachial arterial pressure also was measured with an oscilometric device (Dynamap, Critikon) or an auscultatory sphygmomanometer on the right arm immediately before the 5-min measurement period. These pressure readings were used to confirm the Finapres-
derived arterial pressures. Respiratory excursions were measured by inductive plethysmography (Respitrace, Ambulatory Monitoring). Multiunit postganglionic muscle sympathetic nerve activity was measured via a tungsten microelectrode inserted into the right peroneal nerve near the fibular head, as previously described (49). The raw nerve signal was amplified, rectified, and integrated before recording (Nerve Traffic Analyzer, model 662c-3, University of Iowa Biomedical Engineering). Recordings of muscle sympathetic nerve activity were confirmed by the relation of nervous activity to the cardiac cycle and to respiratory activity.

Data analysis and statistics. The electrocardiogram, respiration, beat-to-beat arterial pressure, and muscle sympathetic activity waveforms were digitized at 500 samples/s for off-line analysis with signal-processing software (CODAS, Dataq Instruments; DADiSP, DSP Development). R-R intervals, arterial pressures, and muscle sympathetic activity corresponding to nonsinus beats were eliminated; no subject demonstrated more than two nonsinus beats during the 5-min recording period. R-R intervals were derived from the time difference between marks placed on the peak of the R waves. Systolic and diastolic pressures were derived from the maximum and minimum of the beat-to-beat pressure waveform.

Average sympathetic outflow and sympathetic oscillations were calculated with the sympathetic neurogram standardized for burst height. The largest sympathetic burst occurring during the measurement period was assigned a value of 1,000 arbitrary integration units (aiu) for each subject; all other bursts were calibrated against that standard (49). This procedure eliminates the confound of electrode position on burst height; the voltage amplitude of multifiber sympathetic activity is critically dependent on the position of the electrode relative to the fibers. This is an important consideration for spectral analysis of sympathetic oscillations; differences in spectral power of uncalibrated recordings could result merely from closer electrode placement. Thus normalization of burst amplitude allows group comparisons for both total activity and oscillation amplitude of sympathetic outflow. Subsequently, muscle sympathetic nerve activity was quantified by custom-made programs designed to identify sympathetic bursts above baseline noise with the appropriate delay from the R wave of the electrocardiogram (~1.3 s; Ref. 10). Only bursts with a signal-to-noise ratio >2:1 were included for analysis, and noise artifacts were identified and manually edited from the analysis. For calculation of average muscle sympathetic activity, the area under each burst was measured, and the average burst area was derived for each subject. Average muscle sympathetic nerve activity was calculated as a function of time both as bursts per minute and as total activity (average burst area × bursts/min).

Average R-R interval and systolic and diastolic pressures were calculated from beat-to-beat values for each subject. Frequency domain analysis of variability was performed on beat-to-beat R-R intervals and beat-to-beat arterial pressures and on the respiratory and muscle sympathetic nerve activity waveforms. A power spectrum analysis technique based on the Welch algorithm of averaging periodograms was used (50). To obtain equidistant time intervals, the 300-s time series of beat-to-beat R-R intervals and arterial pressures were interpolated at 5 Hz, and the time series of respiratory and muscle sympathetic nerve activity waveforms were decimated to 5 Hz. Each time series was divided into five equal overlapping segments, detrended, Hanning filtered, and fast Fourier transformed to its frequency representation squared. The periodograms were averaged to produce the spectrum estimate. This method yielded a frequency resolution of 0.001 Hz. The areas under the power spectra in the Mayer wave and respiratory frequencies (defined as 0.05–0.15 and 0.20–0.30 Hz) were integrated and used for statistical comparisons. Relative (normalized) units were not used for comparisons of cardiovascular variabilities, since normalization produces unreliable estimates of power (47) and artificially generates an antipodal relation between the two primary oscillations (i.e., as one increases, the other must decrease; Ref. 9).

Coherence between fluctuations in muscle sympathetic nerve activity and arterial pressure was assessed by cross-spectral analysis based on models previously described (7, 8). For our coherence estimates, values exceeding 0.5 (range 0–1) represent a statistically reliable relation between the

![Fig. 1. Raw data from a representative young female, young male, and older male during paced breathing at 0.25 Hz.](http://ajpheart.physiology.org/Downloadedfromhttp://ajpheart.physiology.org/)
variabilities in the two signals at $P = 0.10$ (50). The coherence between fluctuations in muscle sympathetic nerve activity and both systolic and diastolic pressure Mayer waves were examined.

Group differences in time domain variables (e.g., average R-R interval, average arterial pressures) were assessed by Student's unpaired $t$-test. Group differences in variability data were assessed by Student's unpaired $t$-test after log transformation to achieve normal distribution and equal variance. Spearman rank-order correlations were used to assess the association between arterial pressure Mayer wave amplitude and average sympathetic activity and sympathetic nerve oscillations. A significance level of $P < 0.05$ was used. Data are presented as means $\pm$ SE.

RESULTS

Subjects controlled respiration very well throughout the 5-min measurement period; the majority of respiratory power was between 0.20 and 0.30 Hz in all subjects ($93.5 \pm 0.7\%$). Average R-R interval did not differ among the three groups (Table 1). There were no gender-related differences in R-R interval variability at either respiratory or low frequencies (Table 2). However, R-R interval variability in the respiratory and low frequencies was 87 and 83% lower in older males vs. young males ($P < 0.05$).

Average arterial pressures were not different in the three groups. There were marked gender- and age-related differences in average muscle sympathetic nerve activity calculated as either total activity per minute or bursts per minute. Total sympathetic nerve activity in the young females was only 36% of the activity in the young males whose sympathetic activity was, in turn, 31% lower than the activity in the older males (all $P < 0.05$; Table 1). There were no gender- or age-related differences in respiratory frequency variability in either systolic or diastolic pressure. However, both the young females and older males demonstrated less respiratory frequency variability in muscle sympathetic activity than the young males ($P < 0.05$; Table 2).

The striking gender- and age-related differences in average sympathetic outflow were not paralleled by similar differences in Mayer wave amplitude (Fig. 2). In fact, neither systolic nor diastolic pressure Mayer wave amplitude was different in the young females and males. Moreover, although an age-related difference in Mayer wave amplitude was apparent, it was not analogous to that in average sympathetic activity; despite greater sympathetic outflow, the older males had 60% less diastolic pressure variability in the low frequency than the young males ($P < 0.05$). Differences in the amplitude of sympathetic oscillations at the Mayer wave frequency were also inconsistent with group differences in Mayer wave amplitude. Both young females and older males had lower sympathetic oscillations at the Mayer wave frequency than the young males (both $P < 0.05$; Fig. 2).

Neither systolic nor diastolic Mayer wave amplitude was predictive of average sympathetic activity (Fig. 3); across all subjects and within each group, the correlation coefficient did not exceed 0.37 (all $P > 0.20$). There was also no relation between the sympathetic low-frequency oscillations and average sympathetic outflow (all $r < 0.40$). However, the Mayer wave oscillations in arterial pressure and sympathetic outflow were related both within individual subjects and within two of the three subject groups. The within-subject analogy to the correlation statistic, coherence, indicated a significant relation between Mayer wave oscillations in arterial pressure and sympathetic outflow in $>90\%$ of the subjects. Coherence between systolic pressure and sympathetic activity averaged 0.60 in all subjects with only 5 of 34 subjects below 0.50, and that between diastolic pressure and sympathetic activity averaged 0.61 with 4 of 34 subjects below 0.50. The subjects without significant coherence were equally distributed across the three groups. Thus a strong frequency domain relation between arterial pressure and sympathetic activity at the Mayer wave frequency existed within a majority of

### Table 1. Mean values for R-R interval, arterial pressures, and muscle sympathetic nerve activity during 5-min paced breathing at 0.25 Hz in 3 groups

<table>
<thead>
<tr>
<th></th>
<th>Young female</th>
<th>Young male</th>
<th>Older male</th>
</tr>
</thead>
<tbody>
<tr>
<td>R-R Interval, ms</td>
<td>892 ± 39</td>
<td>984 ± 32</td>
<td>1,058 ± 44</td>
</tr>
<tr>
<td>Systolic Pressure, mmHg</td>
<td>123 ± 5</td>
<td>129 ± 5</td>
<td>133 ± 6</td>
</tr>
<tr>
<td>Diastolic Pressure, mmHg</td>
<td>68 ± 4</td>
<td>74 ± 4</td>
<td>69 ± 3</td>
</tr>
<tr>
<td>Sympathetic Nerve Activity</td>
<td>893 ± 250*</td>
<td>2,505 ± 286†</td>
<td>3,616 ± 529†</td>
</tr>
</tbody>
</table>

Values are means $\pm$ SE. *$P < 0.05$ vs. young males. †$P < 0.05$ vs. young females.

### Table 2. Low and respiratory frequency variability in R-R interval, arterial pressures, and muscle sympathetic nerve activity during 5-min paced breathing at 0.25 Hz in 3 groups

<table>
<thead>
<tr>
<th></th>
<th>Low frequency</th>
<th>Respiratory frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>R-R Interval, ms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Young female</td>
<td>10,164 ± 2,495</td>
<td>11,894 ± 3,667</td>
</tr>
<tr>
<td>Young male</td>
<td>14,422 ± 3,083</td>
<td>13,823 ± 1,788</td>
</tr>
<tr>
<td>Older male</td>
<td>2,471 ± 468†</td>
<td>1,908 ± 1,102†</td>
</tr>
<tr>
<td>Systolic Pressure, mmHg</td>
<td>76 ± 14</td>
<td>22 ± 6</td>
</tr>
<tr>
<td>Diastolic Pressure, mmHg</td>
<td>39 ± 10</td>
<td>4 ± 1</td>
</tr>
<tr>
<td>Sympathetic Activity, aiu⁰</td>
<td>12,421 ± 2,082*</td>
<td>13,431 ± 2,710*</td>
</tr>
</tbody>
</table>

Values are means $\pm$ SE; aiu, arbitrary integration unit. *$P < 0.05$ vs. young males. †$P < 0.05$ vs. young females.
subjects. Despite this, only the male subject groups demonstrated any correlation between the amplitudes of sympathetic and arterial pressure oscillations (Fig. 4). Interestingly, systolic pressure Mayer waves were negatively related to sympathetic oscillations in the young group, whereas arterial pressure Mayer waves were positively related in the older group (Fig. 4, and diastolic $r = 0.62$, $P < 0.05$). However, these correlations indicate that only 34–40% of the variability in Mayer wave amplitude can be accounted for by sympathetic Mayer wave oscillations. Thus arterial pressure Mayer waves did not reflect sympathetic outflow reliably across groups and only marginally reflected sympathetic oscillations in the two male groups.

**DISCUSSION**

These results indicate that arterial pressure Mayer wave amplitude does not provide a surrogate measure for vascular sympathetic outflow. There was no proportionality between arterial pressure Mayer wave amplitude and average sympathetic activity across three groups with markedly different sympathetic outflow.

Although a modest relation between Mayer waves and sympathetic oscillations was evident in the young and older males, it was highly inconsistent between these two groups. Even broad examination of gender- and age-related differences does not provide convincing evidence for exclusive sympathetic mediation of arterial pressure Mayer waves. Lower sympathetic nerve activity and lower sympathetic oscillations at the Mayer wave frequency did not correspond to smaller arterial pressure Mayer waves in the young females. In contrast, higher sympathetic nerve activity and lower sympathetic oscillations at the Mayer wave frequency did correspond to smaller arterial pressure Mayer waves in the older males. The juxtaposition of these findings underscores the fundamental complexity of cardiovascular variabilities and their limitations as convenient markers of autonomic outflow.

**Significance of Mayer wave oscillations.** Although respiratory frequency arterial pressure variability derives, in part, from mechanical effects of respiratory sinus arrhythmia, arterial pressure Mayer waves do not originate from heart rate oscillations in the low
frequency (31, 47). Mayer waves are most apparent in response to sympathoexcitatory stimuli (26), and coherence between arterial pressure and sympathetic activity at the Mayer wave frequency has been shown in both the present and previous studies (21, 33, 36). Thus a reasonable conclusion is that waxing and waning vascular resistance because of sympathetic neural oscillations generates arterial pressure Mayer waves (1, 8, 11, 14, 15, 25, 37). This construct underlies two premises for the use of Mayer wave amplitude as an index of autonomic function. Because a sympathetic effector has been presumed a prerequisite for arterial pressure Mayer waves (16) and some studies have found that α-receptor blockade (4, 20) or spinal cord injury (18) eliminates Mayer waves, low-frequency arterial pressure variability has been identified as an index of vascular sympathetic outflow (12, 24, 27, 33, 38). Furthermore, because the arterial baroreflex may be a key component for sympathetic oscillations (5, 8, 15, 25) and some data suggest that Mayer waves are dependent on intact arterial baroreflex function (3, 45), it has been proposed that Mayer wave amplitude is proportional to baroreflex gain (8, 45). Although both are based on reasonable evidence, neither premise accounts for countervailing influences on Mayer wave amplitude.

We found that mean levels of vascular sympathetic outflow and Mayer wave amplitude are not proportional. These findings indicate that low-frequency arterial pressure variability should not be identified as an index of vascular sympathetic outflow. Indeed, the exact relation between mean sympathetic outflow and Mayer wave amplitude is unclear. Although acute sympathetic ganglionic blockade reduces low-frequency arterial pressure variability in both humans (43) and rats (3), some patients with high spinal transection demonstrate clearly identifiable Mayer waves (16, 22), and rats after chemical sympathectomy demonstrate increased low-frequency arterial pressure variability (6). Thus it appears that Mayer waves can be generated by mechanisms other than sympathetically mediated vasoconstriction, such as autochthonous vascular smooth muscle contractions (44).

We did not find that arterial pressure Mayer wave amplitude was correlated with sympathetic oscillations at the Mayer wave frequency in all subjects. This finding brings into question the idea that arterial pressure Mayer waves are reflective of baroreflex gain (8, 45). If the classical concept of arterial baroreflex gain (42) is extrapolated to interindividual differences in cardiovascular variabilities, then oscillations in arterial pressure input should be comparable to oscillations in baroreflex-mediated sympathetic output. Although this relation did not maintain across all subjects, it did maintain within most subjects. The high coherence between low-frequency sympathetic and arterial pressure oscillations in most subjects may denote a baroreflex link. However, our data indicate that similar sympathetic outflow at the Mayer wave frequency may generate different levels of arterial pressure oscillations; our young females and older males had striking differences in Mayer wave amplitude with similar low-frequency sympathetic oscillations. Thus our results suggest that Mayer wave amplitude is influenced importantly by factors other than baroreflex gain.

Our findings support the observations of Stauss et al. (46). They found no relations between arterial pressure Mayer waves and either the mean level or the Mayer wave oscillations of splanchnic sympathetic outflow in rats. However, our findings are in contrast to the only previous data in humans. Pagani et al. (33) found very high significance levels ($P = 0.00001$–0.04) of regressions between arterial pressure Mayer waves, average sympathetic nervous outflow, and sympathetic nervous oscillations in young males. Nonetheless, the significance levels did not relate to a high predictive power; calculated correlation coefficients for their data show values that do not exceed 0.46. Thus the only previous data in humans support our findings of no relation between arterial pressure Mayer waves and average sympathetic outflow and only a limited rela-
tion between arterial pressure Mayer waves and sympathetic oscillations.

Gender- and age-related differences. There is some previous data on gender- and age-related differences in arterial pressure Mayer waves. There is one report of lower systolic pressure variability in women compared with men (41), yet we found no gender-related difference in our young subjects. A previous comparison of healthy older and young subjects found an age-related reduction in both systolic and diastolic Mayer wave amplitude (48). Although we found systolic Mayer wave amplitude was \( \geq 20\% \) lower, only diastolic Mayer wave amplitude was statistically lower in the older compared with the young men. Disagreement between the present and previous findings may be because of methodological differences; gender- and age-related differences in the previous studies were only evident during spontaneous breathing. Breathing frequency has profound impact on arterial pressure variability, in part through changes in respiratory sinus arrhythmia (23). Furthermore, respiratory-related heart rate variability augments systolic more than diastolic pressure (47). Thus previously reported gender- and age-related differences in systolic Mayer wave amplitude may be merely because of slow breathing frequencies in female and older subjects.

The lack of intergroup correlations and the discrepant intragroup relations between sympathetic outflow and arterial pressure Mayer waves indicate that no single mechanism explains gender- or age-related differences. However, there may be factors that alter vascular responsiveness to sympathetic activity and that underlie general differences in the relations among arterial pressure Mayer waves, mean sympathetic activity, and sympathetic oscillation. Gender-related differences could derive from the effects of estrogen on vascular smooth muscle (13). Estrogen enhances flow-dependent nitric oxide release (40), which has been shown to diminish arterial pressure oscillations (29, 35). However, our findings of similar oscillations in young females and males are contrary to this effect. Gender- and age-related differences in levels of and sensitivity to angiotensin (19) could define the relations between arterial pressure oscillations and sympathetic outflow; angiotensin is a powerful vasoconstrictive that may alter the vascular responsiveness to adrenergic stimulation. However, acute angiotensin blockade does not alter arterial pressure Mayer waves in young males and females (2), although it may have more significant effects on Mayer waves in older humans. Reduced sensitivity of \( \alpha \)-receptors on vascular smooth muscle with age has been reported (17), which would decrease the arterial pressure response to sympathetic activity, yet we found a direct relation between the amplitude of sympathetic outflow oscillations and arterial pressure Mayer waves in our older males. The structural conduit for Mayer waves is the arterial vasculature; thus arterial stiffness may be a prime component of differences in the relation between sympathetic outflow oscillations and Mayer wave amplitude. Although there are no analogous gender-related data, vascular stiffness does increase profoundly with age (39). Although this may explain lower Mayer waves in older men, it is likely that the striking age-related difference in the relation between Mayer wave oscillations in arterial pressure and sympathetic outflow is multifactorial.

Study limitations. Our young females were not examined at a consistent point in the menstrual cycle; thus the lack of correlation between arterial pressure and sympathetic oscillations in this group may have been secondary to variable hormonal status. Nonetheless, this supports our contention that large intersubject variability precludes the use of arterial pressure Mayer waves as a simple marker for vascular sympathetic oscillation. Although these data elucidate cross-sectional differences in arterial pressure and sympathetic activity, Mayer wave oscillations, they do not inform the proportionality between sympathetic outflow and Mayer waves within individuals. It is unknown if Mayer wave relations are maintained under changing conditions within subjects. If these relations are stable within subjects, Mayer waves could be a valid comparator for cardiovascular autonomic control. However, this seems improbable; present findings of marked subject-to-subject variability and previous findings of the mutable links between cardiovascular variabilities (47) indicate that Mayer waves likely have myriad effectors and are not only secondary to baroreflex-mediated sympathetic oscillation.

Conclusions. Understanding arterial pressure variability may have clinical application, since the magnitude of variability is related to morbid events including cardiac ischemia and heart failure (34). However, our results demonstrate that Mayer wave amplitude does not reflect mean sympathetic activity. Moreover, the relations we found between Mayer wave frequency oscillations in arterial pressure and sympathetic activity were evident only within specific groups and were inconsistent between those groups. Thus it appears that Mayer waves may be generated and influenced by a myriad of factors other than sympathetically mediated vasoconstriction and/or baroreflex gain. Our data underscore the fundamental complexity of cardiovascular variabilities and their limitations as convenient markers of autonomic outflow.

This research was supported by the American Federation for Aging Research; National Institute on Aging Grants AG-14226-01, AG-06537, and AG-00687; and the Colorado American Heart Association. Address for reprint requests: J. A. Taylor, Laboratory for Cardiovascular Research, HRCA Research and Training Institute, 1200 Centre St., Boston, MA 02131.

Received 10 July 1997; accepted in final form 17 December 1997.

REFERENCES


3. Cerutti, C., C. Barres, and C. Paultre. Baroreflex modulation of blood pressure and heart rate variabilities in rats: assessment...


25. Mayer waves and sympathetic outflow


29. Mayer, S. Studien zur Physiologie des Herzens und der Blut-
gefässe: 5. Abhandlung: Uber spontane Blutdruckschwank-


33. Pagani, M., N. Montano, A. Porta, A. Malliani, F. M. Ab-


35. Peresson, P. A. J. E. Baumann, H. Ehmke, B. Nafz, U. Wittmann, and H. R. Kirchheim. Phasic and 24-h blood pressure control by endothelin-derivated relaxing factor in con-


40. Rosselli, M., B. Ithumh, P. J. Keller, E. K. J. Jackson, and R. K. Dubey. Circulating nitric oxide (nitrite/nitrate) levels in postmenopausal women substituted with 17β-estradiol and nor-

41. Ryan, S. M., L. A. Lipsitz, and A. L. Goldberger. Importance of gender in spectral analysis of blood pressure dynamics (Ab-


43. Scheffler, G. J., B. J., J. M. Karemaker, and H. H. Ros. Effects of epidual analgesia and atropine on heart rate and


