(*Circulation.* 1996;93:1527-1532.)

Articles

Fundamental Relations Between Short-term RR Interval and Arterial Pressure Oscillations in Humans

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Abstract

Background One of the principal explanations for respiratory sinus arrhythmia is that it reflects arterial baroreflex buffering of respiration-induced arterial pressure fluctuations. If this explanation is correct, then elimination of RR interval fluctuations should increase respiratory arterial pressure fluctuations.

Methods and Results We measured RR interval and arterial pressure fluctuations during normal sinus rhythm and fixed-rate atrial pacing at 17.2±1.8 (SEM) beats per minute greater than the sinus rate in 16 healthy men and 4 healthy women, 20 to 34 years of age. Measurements were made during controlled-frequency breathing (15 breaths per minute or 0.25 Hz) with subjects in the supine and 40° head-up tilt positions. We characterized RR interval and arterial pressure variabilities in low-frequency (0.05 to 0.15 Hz) and respiratory-frequency (0.20 to 0.30 Hz) ranges with fast Fourier transform power spectra and used cross-spectral analysis to determine the phase relation between the two signals. As expected, cardiac

pacing eliminated beat-to-beat RR interval variability. Against expectations, however, cardiac pacing in the supine position significantly reduced arterial pressure oscillations in the respiratory frequency (systolic, 6.8±1.8 to 2.9±0.6 mm Hg^2/Hz, *P*=.017). In contrast, cardiac pacing in the 40° tilt position increased arterial pressure variability (systolic, 8.0±1.8 to 10.8±2.6, *P*=.027). Cross-spectral analysis showed that 40° tilt shifted the phase relation between systolic pressure and RR interval at the respiratory frequency from positive to negative (9±7° versus -17±11°, *P*=.04); that is, in the supine position, RR interval changes appeared to lead arterial pressure changes, and in the upright position, RR interval changes appeared to follow arterial pressure changes.

Conclusions These results demonstrate that respiratory sinus arrhythmia can actually contribute to respiratory arterial pressure fluctuations. Therefore, respiratory sinus arrhythmia does not represent simple baroreflex buffering of arterial pressure.

Key Words: waves • nervous system, autonomic • reflex • physiology • Fourier analysis

Introduction

One of the principal explanations for respiratory RR interval fluctuations, or respiratory sinus arrhythmia, is that they represent baroreflex buffering of arterial pressure changes induced by the mechanical effects of breathing on intrathoracic pressures, venous return to the heart, and ventricular preload and afterload.[1] [2] [3] [4] [5] [6] [7] Prospective testing of this hypothesis has been difficult in humans. The most obvious way to discern the relation between respiratory RR interval and arterial pressure fluctuations is to obtain measurements with and without RR interval fluctuations. Investigators have fixed RR interval in two ways: they have given large atropine doses to healthy subjects[3] [4] [6] [8] [9] [10] or paced the heart in patients with cardiac pacemakers.[8] Neither approach is entirely satisfactory. Cholinergic blockade with atropine not only stabilizes the RR interval but also increases arterial pressure[3] [4] [6] [8] [10] ; pressure elevations may affect arterial compliance[11] and thereby change the magnitude of arterial pressure variability, independent of RR interval changes. Therefore, this physiology may be studied more simply by fixed-rate cardiac pacing. However, patients with pacemakers usually have heart disease, which may alter the relation between arterial pressure and RR interval. The only available data on cardiac pacing in healthy hearts are derived from dogs and suggest that respiratory sinus arrhythmia generates respiratory-frequency fluctuations in arterial pressure.[12] [13] Thus, the fundamental relation between RR interval and arterial pressure oscillations in humans remains uncertain.

We circumvented the problems with human research simply by studying healthy volunteers with and without atrial pacing with an esophageal electrode. Our atrial pacing paradigm during controlled-frequency breathing allowed us to characterize arterial pressure oscillations in the presence and absence of respiratory sinus arrhythmia without affecting average arterial pressures. We tested the hypothesis that if heart rate variability represents baroreflex buffering of arterial pressure, abolition of heart rate variability should augment arterial

pressure variability. Our study yields opposite results and suggests that respiratory sinus arrhythmia contributes importantly to arterial pressure fluctuations in supine humans.

Methods

Top
Abstract
Introduction
Methods
Results
Discussion
References

Subjects

We studied 16 healthy men and 4 healthy women, 20 to 34 years of age. None were on medications, all were nonsmokers, and all had average levels of physical activity. This study was approved by the institutional review boards for human experimentation of the Hunter Holmes McGuire Department of Veterans Affairs Medical Center and the Medical College of Virginia. All subjects gave their written informed consent to participate.

Measurements and Protocol

We measured RR intervals from lead II of the ECG. We estimated beat-to-beat arterial pressure 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.[14] We also measured brachial arterial pressure with an oscillometric device (Dinamap, Critikon) placed on the right arm. A bellows around the subject's upper abdomen was connected to a strain-gauge pressure transducer to indicate respiratory excursions.

We paced the heart at a constant rate with transesophageal electrical stimuli sufficient to override normal sinus rhythm consistently. A bipolar electrode (TAPSUL or TAPCATH, Arzco) was introduced either orally or nasally and positioned in the esophagus behind the left atrium. The location of the electrode was considered adequate when the atrial electrogram voltage was equal to or greater than the QRS voltage and when a satisfactory paced rhythm was maintained. Transesophageal atrial pacing is sometimes associated with significant discomfort; adequate pacing without undue discomfort at stimuli <20 mA was achieved in 70% of the trials (see below).

Measurements were made during 7 minutes of controlled-frequency breathing (15 breaths per minute, 0.25 Hz) with normal sinus and paced rhythm. Cardiac pacing was set at a rate that minimized the number of breakthrough sinus beats (17.2±1.8 beats per minute [mean±SEM] greater than sinus rate). Measurements were made with subjects in both the supine and 40° passive head-up tilt positions. Paced rhythm was not achieved in all subjects in both positions; data were obtained on a total of 14 subjects in the supine position and 14 subjects in the 40° head-up tilt position.

Data Analysis and Statistics

The ECG, respiration, and beat-to-beat arterial pressure waveforms were recorded on FM tape and subsequently digitized at 1000 samples per second for off-line analysis with signal processing software (CODAS, Dataq Instruments; DADiSP, DSP Development Corp). The recording speed and digitizing rate of these data allowed accurate measurement of RR intervals to the nearest millisecond.

Consistent cardiac pacing was difficult to achieve; therefore, the 4 minutes with the highest ratio of paced to sinus rhythm RR intervals were extracted from the 7 minutes of pacing for data analysis. On average, normal sinus rhythm accounted for only 3% of all cardiac intervals evaluated (range, 0% to 12%), and the occurrence of normal sinus rhythm during pacing did not have a consistent, time-dependent pattern in any subject. The corresponding 4-minute period was extracted from the 7 minutes of normal sinus rhythm for comparison.

The means and SDs for the RR interval and systolic and diastolic pressures were calculated from the beat-to-beat values. Frequency domain analysis of variability was performed on beat-to-beat RR intervals, beat-to-beat systolic and diastolic pressures, and the respiratory signal. A power spectrum analysis technique based on the Welch algorithm of averaging periodograms was used.[15] The 240-second time series of beat-to-beat RR interval and systolic pressure were interpolated at 4 Hz to obtain equidistant time intervals and then divided into three equal overlapping segments. Each segment was 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 0.0042 Hz. The areas under power spectra in the low and respiratory frequencies (defined as 0.05 to 0.15 and 0.20 to 0.30 Hz) were integrated and used for statistical comparisons.

Coherence and phase between systolic pressure and RR interval variabilities during normal sinus rhythm were assessed by cross-spectral analysis based on models previously described.[5] [16] When the coherence function exceeds 0.5 (range, 0 to 1) at any frequency, the phase function provides a statistically reliable estimate of the time relations between the two signals. A negative phase suggests that changes in input (systolic pressure) precede changes in output (RR interval); a positive phase suggests the converse.[16] We interpreted a negative phase as suggestive of a baroreflex link between RR interval and systolic pressure because of known baroreflex latencies; pressure changes provoke RR interval changes with latencies as short as 0.24 second.[17]

The results from a 2x2 ANOVA of the data from those subjects who could be paced in both the supine and 40° tilt positions (n=8) did not differ from the results of paired and unpaired Student's *t* tests of all subjects' data (n=14). Therefore, we used Student's paired *t* test to assess the effects of cardiac pacing and Student's unpaired *t* test to assess the effects of 40° tilt. A value of P=.05 was considered significant. All values are given as mean±SEM.

Results

Top
Abstract
Introduction
Methods
Results
Discussion
References

Breathing frequency was well controlled for all conditions in all subjects: respiratory power was always centered near 0.25 Hz and concentrated within 0.20 to 0.30 Hz. During normal sinus rhythm, RR interval and all indexes of RR interval variability were less in the 40° tilt than in the supine position (the Table). Forty-degree tilt did not affect average arterial pressures or respiratory-frequency arterial pressure variability; however, 40° tilt did increase low-frequency arterial pressure variability. Fig 1 is a tracing from a representative subject. As expected, RR interval was less, with essentially no beat-to-beat variability during cardiac

pacing. Average data from all subjects are depicted in Figs 2 through 4 and are given in the Table. Cardiac pacing in both the supine and 40° tilt positions had no effect on average arterial pressures. However, cardiac pacing had significant effects on arterial pressure oscillations in the respiratory frequency (Fig 2). In the supine position, elimination of sinus arrhythmia decreased respiratory-frequency systolic and diastolic (not shown) pressure variabilities by 44% and 31% (both $P<.05$). In contrast, in the 40° tilt position, elimination of sinus arrhythmia increased respiratory-frequency arterial pressure variabilities. Systolic variability increased by 40%, and diastolic variability increased by >100% (both $P<.05$). In the supine position, elimination of RR interval fluctuations did not alter low-frequency arterial pressure variabilities. In the 40° tilt position, elimination of RR interval fluctuations did not alter low-frequency systolic pressure variability but almost doubled low-frequency diastolic pressure variability.

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Table 1. Average RR Intervals, Arterial Pressures, and Indexes of Variability During Sinus and Paced Cardiac Rhythm With Patients in the Supine and 40° Tilt Positions

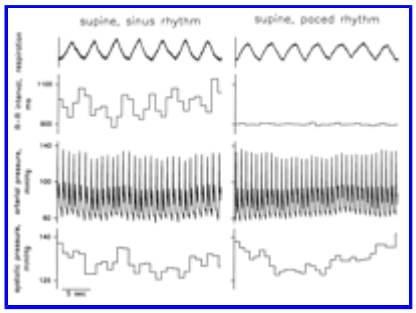

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Figure 1. Experimental record from one supine subject during sinus and paced rhythm with controlled-frequency respiration.

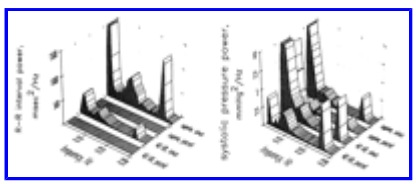

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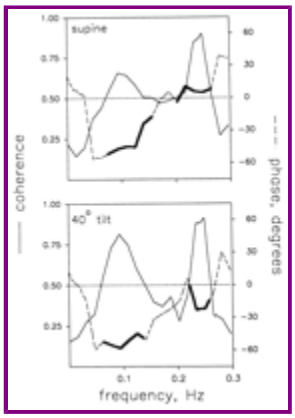
Figure 2. Average RR interval and systolic pressure power spectra during sinus and paced rhythm with a patient in the supine and 40° tilt positions.

Figure 3. Average coherence (solid lines) and phase (dotted lines) relations between systolic pressure and RR interval variabilities with subjects in the supine and 40° tilt positions.



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The phase lines are darkened where coherence exceeds 0.5.

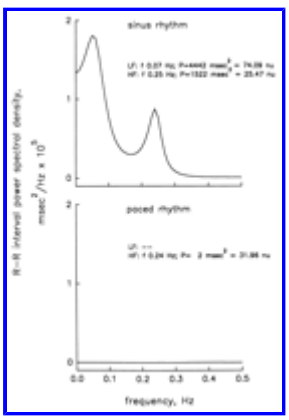

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Figure 4. Autoregressive power spectra of RR interval during sinus and paced rhythm from the subject in Fig 1. Autoregressive-derived values for center frequency (f), absolute power (P), and normalized units (nu) are shown for low-frequency (LF) and respiratory-frequency (HF) RR interval variability.

Cross-spectral analysis provided further insight to the position-dependent relation between arterial pressure and RR interval variabilities during normal sinus rhythm (Fig 3). There was significant coherence between systolic pressure and RR interval variabilities in both low and respiratory frequencies in both positions (average coherence >0.72). The phase of the systolic pressure–RR interval relation in the low-frequency range was not different in the supine and 40° tilt positions (-44±8° versus -48±4°). However, the phase of the systolic pressure–RR interval relation in the respiratory frequency was positive in the supine position but negative in the 40° tilt position (9±7 versus -17±11, *P*=.04). These results suggest that systolic pressure variations in the low frequency precede RR interval variations, regardless of whether subjects are supine or tilted. In contrast, the phase estimates suggest that systolic pressure variations in the respiratory frequency follow those in RR interval when subjects are supine but precede those in RR interval when subjects are tilted.

Discussion

Top
Abstract
Introduction
Methods
Results
Discussion
References

Our data provide unique insight to fundamental relations between RR interval and arterial pressure oscillations in humans. The effects of cardiac pacing indicate that RR interval fluctuations subserve a conventional baroreflex role in buffering arterial pressure fluctuations only when mechanical influences on arterial pressure (eg, respiration, central blood volume) are greater than those in healthy supine humans.

These results clearly demonstrate that RR interval fluctuations at the respiratory and low frequencies do not dampen, and in fact can augment, arterial pressure fluctuations in supine humans. Respiratory-frequency systolic pressure variations follow RR interval variations, and respiratory arterial pressure variability is decreased by fixed-rate atrial pacing. Although low-frequency systolic pressure variations precede RR interval variations, these pressure oscillations are unaffected by fixed-rate atrial pacing. In contrast, both respiratory-frequency and low-frequency RR interval fluctuations dampen arterial pressure fluctuations in tilted humans. Systolic pressure variations in the respiratory and low frequencies precede those in RR interval, and arterial pressure variability is increased by fixed-rate atrial pacing. These data suggest that body position in part determines the mechanisms for the linkage between arterial pressure and RR interval.

Respiratory-Frequency Oscillations

Respiration-synchronous fluctuations in intrathoracic pressure provoke fluctuations in stroke volume,[2] which contribute to respiratory-frequency arterial pressure variability in humans.[1 3 4] Presumably, this arterial pressure fluctuation provokes parallel changes in arterial baroreceptor activity and increases and decreases in cardiac vagal outflow, resulting in respiratory sinus arrhythmia.[5 7 10 16] By this reasoning, respiratory sinus arrhythmia arises from a baroreflex mechanism that should counteract stroke volume fluctuations and reduce arterial pressure fluctuations.[3]

Investigation of this hypothesis in healthy humans has relied on muscarinic blockade to reduce RR interval variability.[3 4 6 8 9 10] However, muscarinic blockade also increases arterial pressure[3 4 6 8 10] and decreases beat-to-beat sympathetic vasomotor outflow,[18] both of which affect arterial compliance.[11] Altered arterial compliance may explain the increased arterial pressure variability with muscarinic blockade in humans.[3 9 10] Fixed-rate atrial pacing, a simpler way to prevent RR interval variability, reduces respiratory-frequency arterial pressure variability in dogs.[12 13] We found that fixed-rate atrial pacing reduces respiratory-frequency arterial pressure oscillations in supine humans. In contrast, we found that fixed-rate atrial pacing increases arterial pressure oscillations in tilted subjects. These findings may be explained by the position-dependent phase relations between systolic pressure and RR interval at the respiratory frequency. The phase relation in the supine position was positive, suggesting that systolic pressure oscillations followed those in RR interval. Conversely, the phase in the tilted position was negative, suggesting that systolic pressure oscillations preceded those in RR interval. A similar phase shift in the systolic pressure–RR interval relation with orthostatic stress was described previously.[19] A phase shift

induced by orthostasis may indicate engagement of the arterial baroreflex; reductions in effective blood volume reduce aortic baroreceptive areas,[20] resulting in arterial baroreceptor engagement. This suggests that respiratory sinus arrhythmia buffers respiratory arterial pressure oscillations in upright humans but augments arterial pressure oscillations in supine humans.

This interpretation of our data agrees with the closed-loop models of systolic pressure variability formulated by Saul et al[6] and Turjanmaa et al.[21] These investigators proposed that the contribution of RR interval variability to respiratory variance in arterial pressure was greater than the contribution of pressure to interval. Furthermore, Saul et al[6] suggested that the mechanical influences of respiration on arterial pressure are greater in the upright than the supine position and in the upright position alter RR interval through the baroreflex. Our data, considered with proposed models of respiratory-frequency cardiovascular variability, underscore the mutable nature of the links between respiration, arterial pressure, and RR interval.

Low-Frequency Oscillations

Arterial pressure Mayer waves, occurring at an interval of about 10 seconds or 0.10 Hz in humans, are presumed to result from rhythmic, sympathetic vasomotor activity.[1] [22] [23] [24] RR interval oscillations at this same frequency are mediated by both cardiac sympathetic and cardiac vagal outflows[12] and are thought to represent arterial baroreflex responses to pressure oscillations.[10] [12] [16] Our data demonstrate that in supine humans, low-frequency RR interval oscillations follow but do not dampen arterial pressure oscillations; elimination of RR interval variability does not increase low-frequency arterial pressure oscillations. However, when vascular sympathetic outflow was increased by 40° tilt,[25] elimination of low-frequency RR interval variability augmented the diastolic pressure Mayer waves. This effect may not have been seen in systolic pressure because of the greater dampening of changes in RR interval on diastolic pressure; in reaction to a higher systolic pressure, a longer RR interval provides a longer diastolic runoff time, resulting in a lower diastolic pressure.[16] Therefore, elimination of this dampening effect by cardiac pacing markedly enhanced low-frequency diastolic pressure variability but had minimal effect on systolic variability. Similar to the results from respiratory-frequency arterial pressure variability, these data indicate that low-frequency RR interval oscillations buffer low-frequency arterial pressure oscillations only in upright humans. Our results in supine humans fit best with the hypothesis that low-frequency arterial pressure oscillations result from a latency in baroreflex-induced changes in vascular sympathetic outflow—in other words, a pressure-pressure feedback loop.[26] Our data do not exclude an arterial baroreflex link between low-frequency systolic pressure and RR interval oscillations; however, we demonstrated such a link only when sympathetic outflow was augmented.

Study Limitations

Although we found a high coherence between RR interval and systolic pressure fluctuations at both the low and respiratory frequencies, phase estimates provide only gross indexes of the time relation between two signals. In a respiratory-frequency cycle of 4 seconds, a positive phase relation could indicate that the RR interval output precedes the systolic pressure input with a feed-forward delay between 0 and 2 seconds or that the RR interval output follows the

systolic pressure input with a feedback delay between 2 and 4 seconds. We interpreted a positive phase relation at the respiratory frequency as RR interval leading systolic pressure within 2 seconds. The alternative, that systolic pressure leads RR interval within 2 to 4 seconds, cannot be explained on the basis of known baroreflex latencies; the vagal baroreceptor–cardiac reflex latency is less than 1 second.[17] However, it may be that our estimates of phase, which are not entirely consistent with either feedback or feed-forward delays indicate that baroreflex and mechanical links between RR interval and systolic pressure are inconstant and that the dominant influence determines the phase relation. Nonetheless, our findings of a positive phase in the supine position and a negative phase in the 40° tilt position are consistent with the effects of cardiac pacing on respiratory arterial pressure oscillations.

Study Implications

These data underscore theoretical limitations of time and frequency domain measures of baroreflex function derived from spontaneous, parallel changes in arterial pressure and RR interval. Short-term changes in systolic pressure and RR interval have been proposed to be cause-and-effect events linked through the baroreflex.[27] [28] The majority of these short-term changes are concentrated within a narrow frequency range (≈0.04 to 0.3 Hz), which prominently includes both low- and respiratory-frequency oscillations.[29] However, our data in supine humans minimize a direct baroreflex buffering role for these short-term RR interval oscillations. Alternatively, the close relation between the amplitudes of arterial pressure and RR interval variabilities may simply reflect the simultaneous effect of respiration on cardiac vagal outflow[30] and arterial pressure,[2] possibly explaining the high baroreflex sensitivity derived from beat-to-beat changes.[31] Although measures of spontaneous baroreflex function may be appropriate in some conditions, our cardiac pacing data bring into question measures of baroreflex function derived from either spectral or beat-to-beat analyses in supine humans.

These data also underscore an important point for quantifying cardiovascular variability with power spectral analysis. Appropriate techniques to quantify cardiovascular variability continue to be a topic of debate.[32] [33] [34] Dividing the power of the respiratory- and low-frequency components by the total power across all frequencies (ie, normalizing) has been proposed as the most relevant measure of variability.[35] Fig 4 shows the result of analyzing RR interval variability data in a representative subject (see Fig 1) according to the suggested autoregressive technique.[35] Our results are significantly altered by this analysis; monotonic cardiac pacing in the supine position does not alter respiratory sinus arrhythmia measured in normalized units (73±9 versus 77±6, P=.734). Only the absolute measure of variability reflected the elimination of heart rate oscillations by cardiac pacing; normalizing power at the respiratory frequency to total power artificially increased the remaining small component. It may be argued that normalized units and absolute units yield complementary information; one cannot be considered without taking the other into account. Yet, even in response to a more physiological condition, 40° tilt, we found a striking divergence in the results from the two autoregressive measures. Absolute low-frequency RR interval power decreased by 30%, whereas normalized power increased by 21% from supine to tilt. Thus, results from both artificial manipulation of cardiovascular variability by cardiac pacing and the normal physiological response to tilt demonstrate that these two measures may not be complementary

and explicitly emphasize the need to quantify frequency-specific oscillations with absolute values derived from power spectral analysis.

Conclusions

Our results challenge the concept that short-term fluctuations in RR interval are linked inextricably to those in arterial pressure through the arterial baroreflex. We found that elimination of respiratory sinus arrhythmia diminished respiratory-frequency arterial pressure fluctuations and therefore do not buffer these arterial pressure fluctuations in supine humans. We conclude that respiratory sinus arrhythmia may be mediated by the baroreflex only when the mechanical effects of respiration on arterial pressure are greater than those in supine humans.

Acknowledgments

This research was supported by the National Institute on Aging (grant AG05636-01), the NHLBI (grant HL-22296), the Department of Veterans Affairs, and the National Aeronautics and Space Administration (grants NAS9-16046 and NAG9-412). We thank Jeffery B. Hoag for his technical assistance in completing these studies.

Received July 11, 1995; revision received October 26, 1995; accepted November 5, 1995.

References

Top
Abstract
Introduction
Methods
Results
Discussion
References

1. Dornhorst AC, Howard P, Leathart GL. Respiratory variations in blood pressure. *Circulation.* 1952;6:553-558. [Medline] [Order article via Infotrieve]
2. Innes JA, De Cort SC, Kox W, Guz A. Within-breath modulation of left ventricular function during normal breathing and positive-pressure ventilation in man. *J Physiol.* 1993;460:487-502. [Abstract]
3. Toska K, Eriksen M. Respiration-synchronous fluctuations in stroke volume, heart rate and arterial pressure in humans. *J Physiol.* 1993;472:501-512. [Abstract]
4. Triedman JK, Saul JP. Blood pressure modulation by central venous pressure and respiration. *Circulation.* 1994;89:169-179. [Abstract]
5. DeBoer RW, Karemaker JM, Strackee J. Relationships between short-term blood-pressure fluctuations and heart-rate variability in resting subjects, II: a simple model. *Med Biol Eng Comput.* 1985;23:359-364. [Medline] [Order article via Infotrieve]
6. Saul JP, Berger RD, Albrecht P, Stein SP, Chen MH, Cohen RJ. Transfer function analysis of the circulation: unique insights into cardiovascular regulation. *Am J Physiol.* 1991;261:H1231-H1245. [Abstract/Free Full Text]
7. Guzzetti S, Cogliati C, Broggi C, Carozzi C, Caldiroli D, Lombardi F, Malliani A. Influences of neural mechanisms on heart period and arterial pressure variabilities in quadriplegic patients. *Am J Physiol.* 1994;266:H1112-H1120. [Abstract/Free Full Text]
8. Kitney RI, Fulton T, McDonald AH, Linkens DA. Transient interactions between blood pressure, respiration, and heart rate in man. *Biomed Eng.* 1985;7:212-222.
9. Elghozi JL, Laude D, Girard A. Effects of respiration on blood pressure and heart rate variability in humans. *Clin Exp Pharmacol Physiol.* 1991;18:735-742. [Medline] [Order article via Infotrieve]
10. Scheffer GJ, TenVoorde BJ, Karemaker JM, Ros HH. Effects of epidural analgesia and

atropine on heart rate and blood pressure variability: implications for the interpretation of beat-to-beat fluctuations. *Eur J Anaesthesiol.* 1994;11:75-80. [Medline] [Order article via Infotrieve]

11. Boutouryie P, Lacolley P, Girerd X, Beck l, Safar M, Laurent S. Sympathetic activation decreases medium-sized arterial compliance in humans. *Am J Physiol.* 1994;267:H1368-H1376. [Abstract/Free Full Text]
12. Akselrod S, Gordon D, Madwed JB, Snidman NC, Shannon DC, Cohen RJ. Hemodynamic regulation: investigation by spectral analysis. *Am J Physiol.* 1985;249:H867-H875. [Abstract/Free Full Text]
13. Wang M, Evans JM, Hassebrook L, Fischer C, Knapp CF. Frequency response characteristics and spectral patterns of arterial pressure in heart paced dogs. *FASEB J.* 1993;7:A1175. Abstract.
14. Omboni S, Parati G, Frattola A, Mutti E, Di Rienzo M, Castiglioni P, Mancia G. Spectral and sequence analysis of finger blood pressure variability: comparison with analysis of intra-arterial recordings. *Hypertension.* 1993;22:26-33. [Abstract]
15. Welch PD. The use of fast Fourier transform for the estimation of power spectra: a method based on time averaging over short, modified periodograms. *IEEE Trans Audio Electroaccoust.* 1967;15:70-73.
16. DeBoer RW, Karemaker JM, Strackee J. Hemodynamic fluctuations and baroreflex sensitivity in humans: a beat-to-beat model. *Am J Physiol.* 1987;253:H680-H689. [Abstract/Free Full Text]
17. Eckberg DL. Temporal response patterns of the human sinus node to brief carotid baroreceptor stimuli. *J Physiol.* 1976;258:769-782. [Abstract]
18. Porter TR, Eckberg DL, Fritsch JM, Rea RF, Beightol LA, Schmedtje JF, Mohanty PK. Autonomic pathophysiology in heart failure patients: sympathetic-cholinergic interrelations. *J Clin Invest.* 1990;85:1362-1371. [Medline] [Order article via Infotrieve]
19. Blaber AP, Yamamoto Y, Hughson RL. Change in phase relationship between SBP and R-R interval during lower body negative pressure. *Am J Physiol.* 1995;37:H1688-H1693.
20. Taylor JA, Halliwill JR, Brown TE, Hayano J, Eckberg DL. `Non-hypotensive' hypovolaemia reduces ascending aortic dimensions in humans. *J Physiol.* 1995;483:289-298. [Abstract]
21. Turjanmaa V, Kalli S, Sydnmaa M, Uusitalo A. Short-term variability of systolic blood pressure and heart rate in normotensive subjects. *Clin Physiol.* 1990;10:389-401. [Medline] [Order article via Infotrieve]
22. Guyton AC, Batson HM, Smith CM, Armstrong GG. Method for studying competence of the body's blood pressure regulatory mechanisms and effect of pressoreceptor denervation. *Am J Physiol.* 1951;164:360-368.
23. Hyndman BW, Kitney RI, Sayers BM. Spontaneous rhythms in physiological control systems. *Nature.* 1971;233:339-341. [Medline] [Order article via Infotrieve]
24. Preiss G, Polosa C. Patterns of sympathetic neuron activity associated with Mayer waves. *Am J Physiol.* 1974;226:724-730. [Medline] [Order article via Infotrieve]
25. Tsunoda S, Shindo K, Shiozawa Z, Mano T. Comparison between muscle sympathetic nerve activity and calf vascular resistance with head-up tilting in humans. *J Auton Nerv Syst.* 1991;33:277-281. [Medline] [Order article via Infotrieve]
26. Baselli G, Cerutti S, Civardi S, Malliani A, Pagani M. Cardiovascular variability signals: towards the identification of a closed-loop model of the neural mechanisms. *IEEE Trans Biomed Eng.* 1988;35:1033-1046. [Medline] [Order article via Infotrieve]
27. Bertinieri G, di Rienzo M, Cavallazzi A, Ferrari AU, Pedotti A, Mancia G. A new approach to the analysis of the arterial baroreflex. *J Hypertens.* 1985;3:S79-S81.
28. Robbe HWJ, Mulder LJM, Rudel H, Langewitz WA, Veldman JBP, Mulder G. Assessment of baroreceptor reflex sensitivity by means of spectral analysis. *Hypertension.* 1987;10:538-543. [Abstract]
29. Kitney RI, Rompelman O. *The Study of Heart Rate Variability*. Oxford, UK: Clarendon

Press; 1980.
30. Katona PG, Jih F. Respiratory sinus arrhythmia: noninvasive measure of parasympathetic cardiac control. *J Appl Physiol.* 1975;39:801-805. [Abstract/Free Full Text]
31. Frankel RA, Metting PJ, Britton SL. Evaluation of spontaneous baroreflex sensitivity in conscious dog. *J Physiol.* 1993;462:31-45. [Abstract]
32. Furlan R, Pagani M, Malliani A. Effects of exercise training on control of heart rate. *Cardiovasc Res.* 1993;27:2286-2287. [Medline] [Order article via Infotrieve]
33. Pagani M, Lombardi F, Malliani A. Heart rate variability: disagreement on the markers of sympathetic and parasympathetic activities. *J Am Coll Cardiol.* 1993;22:951-954. [Medline] [Order article via Infotrieve]
34. Stauss HM, Persson PB. Power spectral analysis of heart rate and blood pressure: markers for autonomic balance or indicators of baroreflex control? *Clin Sci.* 1995;88:1-2. [Medline] [Order article via Infotrieve]
35. Montano N, Ruscone TG, Porta A, Lombardi F, Pagani M, Malliani A. Power spectrum analysis of heart rate variability to assess the changes in sympathovagal balance during graded orthostatic tilt. *Circulation.* 1994;90:1826-1831.[Abstract]

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J. Am. Coll. Cardiol., October 4, 2005; 46(7): 1314 - 1321.

[Abstract] [Full Text] [PDF]

Am. J. Physiol: Heart and Circulatory Physiology

A. Kamiya, J. Hayano, T. Kawada, D. Michikami, K. Yamamoto, H. Ariumi, S. Shimizu, K. Uemura, T. Miyamoto, T. Aiba, K. Sunagawa, and M. Sugimachi

Low-frequency oscillation of sympathetic nerve activity decreases during development of tilt-induced syncope preceding sympathetic withdrawal and bradycardia

Am J Physiol Heart Circ Physiol, October 1, 2005; 289(4): H1758 - H1769.

[Abstract] [Full Text] [PDF]

Journal of Applied Physiology

K. Okazaki, K.-i. Iwasaki, A. Prasad, M. D. Palmer, E. R. Martini, Q. Fu, A. Arbab-Zadeh, R. Zhang, and B. D. Levine

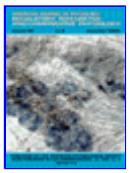
Dose-response relationship of endurance training for autonomic circulatory control in healthy seniors

J Appl Physiol, September 1, 2005; 99(3): 1041 - 1049.

[Abstract] [Full Text] [PDF]

Experimental Physiology HNMF

G. Gulli, V. E. Claydon, V. L. Cooper, and R. Hainsworth
R-R interval-blood pressure interaction in subjects with different tolerances to orthostatic stress
Exp. Physiol., May 1, 2005; 90(3): 367 - 375.
[Abstract] [Full Text] [PDF]

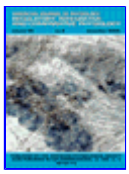


Am. J. Physiol: Regulatory, Integrative and Comparative Physiology HNMF

P. E. Hammer and J. P. Saul
Resonance in a mathematical model of baroreflex control: arterial blood pressure waves accompanying postural stress
Am J Physiol Regulatory Integrative Comp Physiol, June 1, 2005; 288(6): R1637 - R1648.
[Abstract] [Full Text] [PDF]

Am. J. Physiol: Heart and Circulatory Physiology HNMF

G. Nollo, L. Faes, A. Porta, R. Antolini, and F. Ravelli
Exploring directionality in spontaneous heart period and systolic pressure variability interactions in humans: implications in the evaluation of baroreflex gain
Am J Physiol Heart Circ Physiol, April 1, 2005; 288(4): H1777 - H1785.
[Abstract] [Full Text] [PDF]



Am. J. Physiol: Regulatory, Integrative and Comparative Physiology HNMF

D. D. O'Leary, J. K. Shoemaker, M. R. Edwards, and R. L. Hughson
Spontaneous beat-by-beat fluctuations of total peripheral and cerebrovascular resistance in response to tilt
Am J Physiol Regulatory Integrative Comp Physiol, September 1, 2004; 287(3): R670 - R679.
[Abstract] [Full Text] [PDF]

Journal of Applied Physiology HNMF

I. Fietze, D. Romberg, M. Glos, S. Endres, H. Theres, C. Witt, and V. K. Somers
Effects of positive-pressure ventilation on the spontaneous baroreflex in healthy subjects
J Appl Physiol, March 1, 2004; 96(3): 1155 - 1160.
[Abstract] [Full Text] [PDF]

Journal of the American College of Cardiology HNMF

P. Kaushal and J. A. Taylor
Inter-relations among declines in arterial distensibility, baroreflex function and respiratory sinus arrhythmia
J. Am. Coll. Cardiol., May 1, 2002; 39(9): 1524 - 1530.
[Abstract] [Full Text] [PDF]

Hypertension HNMF

R. D. Lipman, J. K. Salisbury, and J. A. Taylor
Spontaneous Indices Are Inconsistent With Arterial Baroreflex Gain

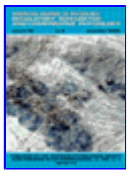
Hypertension, October 1, 2003; 42(4): 481 - 487.
[Abstract] [Full Text] [PDF]

The Journal of Physiology HINARI

D. L. Eckberg
The human respiratory gate
J. Physiol., April 15, 2003; 548(2): 339 - 352.
[Abstract] [Full Text] [PDF]

HEART ONLINE and EDUCATION IN HEART HINARI

J O Schwab, G Eichner, H Schmitt, S Weber, M Coch, and B Waldecker
The relative contribution of the sinus and AV node to heart rate variability
Heart, March 1, 2003; 89(3): 337 - 338.
[Full Text] [PDF]



Am. J. Physiol: Regulatory, Integrative and Comparative Physiology HINARI

B. R. Baldridge, D. E. Burgess, E. E. Zimmerman, J. J. Carroll, A. G. Sprinkle, R. O. Speakman, S.-G. Li, D. R. Brown, R. F. Taylor, S. Dworkin, and D. C. Randall
Heart rate-arterial blood pressure relationship in conscious rat before vs. after spinal cord transection
Am J Physiol Regulatory Integrative Comp Physiol, September 1, 2002; 283(3): R748 - 756.
[Abstract] [Full Text] [PDF]

Am. J. Physiol: Heart and Circulatory Physiology HINARI

G. Nollo, L. Faes, A. Porta, B. Pellegrini, F. Ravelli, M. Del Greco, M. Disertori, and R. Antolini
Evidence of unbalanced regulatory mechanism of heart rate and systolic pressure after acute myocardial infarction
Am J Physiol Heart Circ Physiol, September 1, 2002; 283(3): H1200 - 1207.
[Abstract] [Full Text] [PDF]

The Journal of Physiology HINARI

R. Zhang, K. Iwasaki, J. H. Zuckerman, K. Behbehani, C. G. Crandall, and B. D. Levine
Mechanism of blood pressure and R-R variability: insights from ganglion blockade in humans
J. Physiol., August 15, 2002; 543(1): 337 - 348.
[Abstract] [Full Text] [PDF]

The Journal of Physiology HINARI

M. A. Cohen and J. A. Taylor
Short-term cardiovascular oscillations in man: measuring and modelling the physiologies
J. Physiol., August 1, 2002; 542(3): 669 - 683.

[Abstract] [Full Text] [PDF]

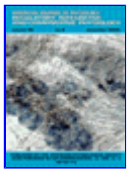
Circulation HOME

K. Sakata, H. Kumagai, M. Osaka, T. Onami, T. Matsuura, M. Imai, and T. Saruta

Potentiated Sympathetic Nervous and Renin-Angiotensin Systems Reduce Nonlinear Correlation Between Sympathetic Activity and Blood Pressure in Conscious Spontaneously Hypertensive Rats

Circulation, July 30, 2002; 106(5): 620 - 625.

[Abstract] [Full Text] [PDF]



Am. J. Physiol: Regulatory, Integrative and Comparative Physiology HOME

H.-K. Liu, S.-J. Guild, J. V. Ringwood, C. J. Barrett, B. L. Leonard, S.-K. Nguang, M. A. Navakatikyan, and S. C. Malpas

Dynamic baroreflex control of blood pressure: influence of the heart vs. peripheral resistance

Am J Physiol Regulatory Integrative Comp Physiol, August 1, 2002; 283 (2): R533 - 542.

[Abstract] [Full Text] [PDF]

Am. J. Physiol: Heart and Circulatory Physiology HOME

S. D. Beske, G. E. Alvarez, T. P. Ballard, and K. P. Davy

Reduced cardiovagal baroreflex gain in visceral obesity: implications for the metabolic syndrome

Am J Physiol Heart Circ Physiol, February 1, 2002; 282(2): H630 - 635.

[Abstract] [Full Text] [PDF]

Am. J. Physiol: Heart and Circulatory Physiology HOME

S. C. Malpas

Neural influences on cardiovascular variability: possibilities and pitfalls

Am J Physiol Heart Circ Physiol, January 1, 2002; 282(1): H6 - 20.

[Abstract] [Full Text] [PDF]

Am. J. Physiol: Heart and Circulatory Physiology HOME

D. W. Wray, K. J. Formes, M. S. Weiss, A. H. O-Yurvati, P. B. Raven, R. Zhang, and X. Shi

Vagal cardiac function and arterial blood pressure stability

Am J Physiol Heart Circ Physiol, November 1, 2001; 281(5): H1870 - 1880.

[Abstract] [Full Text] [PDF]

The Journal of Physiology HOME

M. Elstad, K. Toska, K. H. Chon, E. A. Raeder, and R. J. Cohen

Respiratory sinus arrhythmia: opposite effects on systolic and mean arterial pressure in supine humans

J. Physiol., October 1, 2001; 536(1): 251 - 259.

[Abstract] [Full Text] [PDF]

PSYCHOSOMATIC MEDICINE

H. Schachinger, M. Weinbacher, A. Kiss, R. Ritz, and W. Langewitz
Cardiovascular Indices of Peripheral and Central Sympathetic Activation
Psychosom Med, September 1, 2001; 63(5): 788 - 796.
[Abstract] [Full Text] [PDF]

PSYCHOSOMATIC MEDICINE

R. P. Sloan, P. A. Shapiro, E. Bagiella, M. M. Myers, and J. M. Gorman
Cardiac Autonomic Control Buffers Blood Pressure Variability Responses to Challenge: A Psychophysiologic Model of Coronary Artery Disease
Psychosom Med, January 1, 1999; 61(1): 58 - 68.
[Abstract] [Full Text] [PDF]

Am. J. Physiol: Heart and Circulatory Physiology

L. J. Badra, W. H. Cooke, J. B. Hoag, A. A. Crossman, T. A. Kuusela, K. U. O. Tahvanainen, and D. L. Eckberg
Respiratory modulation of human autonomic rhythms
Am J Physiol Heart Circ Physiol, June 1, 2001; 280(6): H2674 - 2688.
[Abstract] [Full Text]

Journal of Applied Physiology

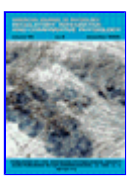
J. W. Hamner, R. J. Morin, J. L. Rudolph, and J. A. Taylor
Inconsistent link between low-frequency oscillations: R-R interval responses to augmented Mayer waves
J Appl Physiol, April 1, 2001; 90(4): 1559 - 1564.
[Abstract] [Full Text]

Circulation

J. M. Legramante, G. Raimondi, M. Massaro, and F. Iellamo
Positive and Negative Feedback Mechanisms in the Neural Regulation of Cardiovascular Function in Healthy and Spinal Cord-Injured Humans
Circulation, March 6, 2001; 103(9): 1250 - 1255.
[Abstract] [Full Text] [PDF]

Am. J. Physiol: Heart and Circulatory Physiology

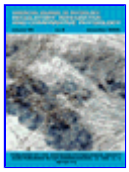
R. Zhang, K. Behbehani, C. G. Crandall, J. H. Zuckerman, and B. D. Levine
Dynamic regulation of heart rate during acute hypotension: new insight into baroreflex function
Am J Physiol Heart Circ Physiol, January 1, 2001; 280(1): H407 - 419.
[Abstract] [Full Text]



Am. J. Physiol: Regulatory, Integrative and Comparative Physiology

K.-I. Iwasaki, R. Zhang, J. H. Zuckerman, J. A. Pawelczyk, and B. D. Levine
Effect of head-down-tilt bed rest and hypovolemia on dynamic regulation of heart rate and blood pressure

Am J Physiol Regulatory Integrative Comp Physiol, December 1, 2000; 279(6): R2189 - 2199.
[Abstract] [Full Text]



Am. J. Physiol: Regulatory, Integrative and Comparative Physiology HINARI

A. Just, J. Faulhaber, and H. Ehmke
Autonomic cardiovascular control in conscious mice
Am J Physiol Regulatory Integrative Comp Physiol, December 1, 2000; 279(6): R2214 - 2221.
[Abstract] [Full Text]

Am. J. Physiol: Heart and Circulatory Physiology HINARI

C. G. Crandall, R. Zhang, and B. D. Levine
Effects of whole body heating on dynamic baroreflex regulation of heart rate in humans
Am J Physiol Heart Circ Physiol, November 1, 2000; 279(5): H2486 - 2492.
[Abstract] [Full Text]

Am. J. Physiol: Heart and Circulatory Physiology HINARI

A. Porta, G. Baselli, O. Rimoldi, A. Malliani, and M. Pagani
Assessing baroreflex gain from spontaneous variability in conscious dogs: role of causality and respiration
Am J Physiol Heart Circ Physiol, November 1, 2000; 279(5): H2558 - 2567.
[Abstract] [Full Text]

Am. J. Physiol: Heart and Circulatory Physiology HINARI

D. Chemla, E. Aptecar, J.-L. Hébert, C. Coirault, D. Loisance, Y. Lecarpentier, and A. Nitenberg
Short-term variability of pulse pressure and systolic and diastolic time in heart transplant recipients
Am J Physiol Heart Circ Physiol, July 1, 2000; 279(1): H122 - 129.
[Abstract] [Full Text]

Hypertension HINARI

J. P. Fauvel, C. Cerutti, P. Quelin, M. Laville, M. P. Gustin, C. Z. Paultre, and M. Ducher
Mental Stress-Induced Increase in Blood Pressure Is Not Related to Baroreflex Sensitivity in Middle-Aged Healthy Men
Hypertension, April 1, 2000; 35(4): 887 - 891.
[Abstract] [Full Text] [PDF]

HEART ONLINE and EDUCATION IN HEART HINARI

J M Serrador, H C Finlayson, and R L Hughson
Physical activity is a major contributor to the ultra low frequency components of heart rate variability
Heart, December 1, 1999; 82(6): 9e - 9.
[Abstract] [Full Text]

Hypertension HOME

M. V. Pitzalis, F. Massari, C. Forleo, A. Fioretti, R. Colombo, C. Balducci, F. Mastropasqua, and P. Rizzon

Respiratory Systolic Pressure Variability During Atrial Fibrillation and Sinus Rhythm

Hypertension, November 1, 1999; 34(5): 1060 - 1065.

[Abstract] [Full Text] [PDF]

Circulation HOME

J. A. Taylor, C. W. Myers, N. Montano, C. Cogliati, A. Porta, M. Pagani, A. Malliani, K. Narkiewicz, F. M. Abboud, and V. K. Somers

Mathematical Treatment of Autonomic Oscillations - 2 • Response

Circulation, October 12, 1999; 100(15): e64 - 64.

[Full Text] [PDF]

The Journal of Physiology HOME

J. M. Karemaker

Autonomic integration: the physiological basis of cardiovascular variability

J. Physiol., June 1, 1999; 517(2): 316 - 316.

[Abstract] [Full Text]

The Journal of Physiology HOME

W. H. Cooke, J. B. Hoag, A. A. Crossman, T. A. Kuusela, K. U. O. Tahvanainen, and D. L. Eckberg

Human responses to upright tilt: a window on central autonomic integration

J. Physiol., June 1, 1999; 517(2): 617 - 628.

[Abstract] [Full Text]

Circulation HOME

D. P. Francis, L. C. Davies, M. Piepoli, M. Rauchhaus, P. Ponikowski, and A. J. S. Coats

Origin of Oscillatory Kinetics of Respiratory Gas Exchange in Chronic Heart Failure

Circulation, September 7, 1999; 100(10): 1065 - 1070.

[Abstract] [Full Text] [PDF]

Circulation HOME

J. M. Legramante, G. Raimondi, M. Massaro, S. Cassarino, G. Peruzzi, and F. Iellamo

Investigating Feed-Forward Neural Regulation of Circulation From Analysis of Spontaneous Arterial Pressure and Heart Rate Fluctuations

Circulation, April 6, 1999; 99(13): 1760 - 1766.

[Abstract] [Full Text] [PDF]

Am. J. Physiol: Heart and Circulatory Physiology HOME

T. Laitinen, J. Hartikainen, L. Niskanen, G. Geelen, and E. Länsimies

Sympathovagal balance is major determinant of short-term blood

pressure variability in healthy subjects
Am J Physiol Heart Circ Physiol, April 1, 1999; 276(4): H1245 - 1252.
[Abstract] [Full Text]

Circulation HOME

M. Piepoli, P. Sleight, S. Leuzzi, F. Valle, G. Spadacini, C. Passino, J. Johnston, and L. Bernardi
Origin of Respiratory Sinus Arrhythmia in Conscious Humans : An Important Role for Arterial Carotid Baroreceptors
Circulation, April 1, 1997; 95(7): 1813 - 1821.
[Abstract] [Full Text]

Circulation HOME

M. Malik and D. L Eckberg
Sympathovagal Balance: A Critical Appraisal • Response
Circulation, December 8, 1998; 98(23): 2643 - 2644.
[Full Text] [PDF]

Circulation HOME

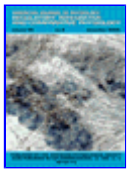
J. A. Taylor, D. L. Carr, C. W. Myers, and D. L. Eckberg
Mechanisms Underlying Very-Low-Frequency RR-Interval Oscillations in Humans
Circulation, August 11, 1998; 98(6): 547 - 555.
[Abstract] [Full Text] [PDF]

Circulation HOME

R. L. Cooley, N. Montano, C. Cogliati, P. van de Borne, W. Richenbacher, R. Oren, and V. K. Somers
Evidence for a Central Origin of the Low-Frequency Oscillation in RR-Interval Variability
Circulation, August 11, 1998; 98(6): 556 - 561.
[Abstract] [Full Text] [PDF]

Circulation HOME

M. V. Pitzalis, F. Mastropasqua, A. Passantino, F. Massari, L. Ligurgo, C. Forleo, C. Balducci, F. Lombardi, and P. Rizzon
Comparison Between Noninvasive Indices of Baroreceptor Sensitivity and the Phenylephrine Method in Post–Myocardial Infarction Patients
Circulation, April 14, 1998; 97(14): 1362 - 1367.
[Abstract] [Full Text] [PDF]



Am. J. Physiol: Regulatory, Integrative and Comparative Physiology HOME

P. B. Persson
Spectrum analysis of cardiovascular time series
Am J Physiol Regulatory Integrative Comp Physiol, October 1, 1997; 273 (4): R1201 - 1210.
[Abstract] [Full Text]

Am. J. Physiol: Heart and Circulatory Physiology HOME

J. A. Taylor, T. D. Williams, D. R. Seals, and K. P. Davy
Low-frequency arterial pressure fluctuations do not reflect sympathetic outflow: gender and age differences
Am J Physiol Heart Circ Physiol, April 1, 1998; 274(4): H1194 - 1201.
[Abstract] [Full Text]

Am. J. Physiol: Heart and Circulatory Physiology HOME

R. A. Henry, I-L. Lu, L. A. Beightol, and D. L. Eckberg
Interactions between CO2 chemoreflexes and arterial baroreflexes
Am J Physiol Heart Circ Physiol, June 1, 1998; 274(6): H2177 - 2187.
[Abstract] [Full Text]

Journal of Applied Physiology HOME

L. A. Lipsitz, R. Morin, M. Gagnon, D. Kiely, and A. Medina
Vasomotor instability preceding tilt-induced syncope: does respiration play a role?
J Appl Physiol, August 1, 1997; 83(2): 383 - 390.
[Abstract] [Full Text]

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