Quantification of Mechanical and Neural Components of Vagal Baroreflex in Humans

Brian E. Hunt, Lisamarie Fahy, William B. Farquhar, J. Andrew Taylor

Abstract—Traditionally, arterial baroreflex control of vagal neural outflow is quantified by heart period responses to falling and/or rising arterial pressures (ms/mm Hg). However, it is arterial pressure–dependent stretch of barosensory vessels that determines afferent baroreceptor responses, which, in turn, generate appropriate efferent cardiac vagal outflow. Thus, mechanical transduction of pressure into barosensory vessel stretch and neural transduction of stretch into vagal outflow are key steps in baroreflex regulation that determine the conventional integrated input-output relation. We developed a novel technique for direct estimation of gain in both mechanical and neural components of integrated cardiac vagal baroreflex control. Concurrent, beat-by-beat measures of arterial pressures (Finapres), carotid diameters (B-mode ultrasonography), and R-R intervals (ECG lead II) were made during bolus vasoactive drug infusions (modified Oxford technique) in 16 healthy humans. The systolic carotid diameter/pressure relationship ($r^2=0.79\pm0.008$, mean $\pm$SEM) provided a gain estimate of dynamic mechanical transduction of pressure into a baroreflex stimulus. The R-R interval/systolic diameter relationship ($r^2=0.77\pm0.009$) provided a gain estimate of afferent-efferent neural transduction of baroreflex stimulus into a vagal response. Variance between repeated measures for both estimates was no different than that for standard gain ($P>0.40$). Moreover, in these subjects, the simple product of the 2 estimates almost equaled standard baroreflex gain (ms/mm Hg = 0.98$x+2.27$; $r^2=0.93$, $P=0.001$). This technique provides reliable information on key baroreflex components not distinguished by standard assessments and gives insight to dynamic mechanical and neural events during acute changes in arterial pressure. (Hypertension. 2001;37:1362-1368.)

Key Words: arterial pressure ■ carotid arteries ■ ultrasonography

Depressed baroreflex sensitivity is associated with hypertension,1 heart disease,2 diabetes, and other pathophysiological states,3 attesting that appropriate baroreflex regulation of autonomic outflow is crucial to maintenance of cardiovascular health and homeostasis. Traditionally, cardiac-vagal baroreflex sensitivity or gain has been quantified by the magnitude of heart period responses to falling and/or rising arterial pressure (ie, $\Delta$ R-R interval/$\Delta$ systolic pressure).4 This estimate represents an integrated input-output relation and provides broad insight to baroreflex function. However, it has long been recognized that baroreceptors respond to deformation not pressure per se. Early in the last century, Sollman and Brown5 reported that stretch of the carotid artery produces bradycardia and hypotension in dogs. Similar reflex responses in humans derive from proportionalties between changes in carotid diameter and carotid nerve firing6 and between the R-R interval and the frequency of afferent carotid sinus nerve activation.7 Thus, mechanical transduction of arterial pressure into carotid stretch and neural transduction of carotid stretch into vagal outflow are the key steps in baroreflex regulation that determine the conventional integrated input-output relation.

Both mechanical transduction of pressure and neural transduction of stretch may be profoundly important in pathophysiological conditions. For example, greater carotid vascular stiffness in human hypertension1 would effectively desensitize baroreceptors and depress baroreflex autonomic regulation. Alternatively, in the absence of structural changes, cardiac vagal deficiencies, which can occur early in the progression of diabetes8 and may characterize orthostatic intolerance,3 would reduce neural transduction and blunt baroreflex responses to pressure decreases. However, elucidating either of these processes in humans is difficult. Static measures of basal arterial stiffness can be obtained and are associated with integrated baroreflex gain,1,9 yet they provide no information on dynamic vascular responses to rapid pressure changes10 that usually characterize baroreflex activation. Measures of the neural component of the baroreflex arc are exceedingly rare, because of the difficulty of obtaining recordings of afferent baroreceptor or efferent vagal nervous activity in humans. Nonetheless, the sparse information supports a close relation between baroreflex stimulus and vagal response. Changes in carotid diameter within the cardiac cycle produce proportionate firing of afferent barore-
ceptive fibers, direct stimulation of these afferents produces proportional R-R interval responses, and R-R interval changes proportionally with the rate of vagus nerve stimulation. These 3 observations suggest that the relation between barosensory vessel diameter and R-R interval may represent the afferent-efferent neural baroreflex arc in humans.

We set out to develop a technique for direct, minimally invasive estimation of both mechanical and neural components of the integrated cardiac-vagal baroreflex in humans. On the basis of the observations outlined above, we measured arterial pressures, common carotid diameters, and R-R intervals during a pharmacological arterial baroreflex stimulus to broaden our ability to assess baroreflex function. Our aim was to determine whether this novel approach of concurrent beat-by-beat measures provides reliable estimates of not only the standard integrated baroreflex gain but also the mechanical transduction of arterial pressure into barosensory vessel stretch and the neural transduction of barosensory vessel stretch into autonomic outflow, facets of baroreflex function difficult to ascertain in humans. Furthermore, we wanted to determine whether application of this technique to physiological and pathophysiological states characterized by alterations in baroreflex function would give unique insight to cardiovascular autonomic regulation in humans.

Methods

Eleven men and 5 women age 20 to 31 years were studied under supine resting conditions. All subjects were free of overt autonomic or cardiovascular disease and had a body mass index <26 kg/m² and resting brachial blood pressures <135/85 mm Hg. The Hebrew Rehabilitation Center for Aged Institutional Review Board approved the experimental protocol. The nature, purpose, and risks of the study were explained to each subject before written informed consent was obtained in accordance with institutional guidelines.

Protocol

Arterial Pressure and R-R Interval

Beat-by-beat arterial pressures from a finger photoplethysmograph (Finapres, Datex-Ohmeda) and a standard 3-lead ECG were recorded continuously. An oscillometric blood pressure system on the contralateral arm (Dinamap) was used to calibrate the Finapres against a standard brachial measure.

Carotid Ultrasonography

Longitudinal B-mode images of a common carotid artery, ~1 cm below the carotid bulb, were obtained using a high-resolution linear array transducer (7.5 MHz, Hewlett-Packard). The transducer was placed at 90° to the vessel so that the near and far wall interfaces were clearly discernible. Commercially available hardware (PCI Frame Grabber, Datax) and software (CVI Acquisition, Information Integrity) acquired 30 Hz images to the computer triggered from the R wave of the ECG. Fifteen consecutive carotid images were acquired with each trigger to approximate at least one third the cardiac cycle (ie, 300 ms of a 1500-ms R-R interval or 40-bpm heart rate) and, therefore, encompass both end-diastolic and peak-systolic carotid diameters.

Pharmacological Baroreflex Engagement

We used the modified Oxford technique, which involves a bolus injection of 100 µg of sodium nitroprusside followed in 60 seconds by a bolus of 150 µg of phenylephrine hydrochloride. Responses of a representative subject are shown in the top panels of Figure 1. This technique generally produces an initial ~15 mm Hg drop in arterial pressure followed by an ~15 mm Hg rise in pressure above resting supine levels. In this particular subject, arterial pressure only increased ~8 mm Hg above baseline, probably because of effective buffering by pronounced lengthening of the R-R interval. The advantage of the modified Oxford technique is that it produces a longer and greater pressure rise than the more commonly used Oxford technique (phenylephrine alone). Moreover, arterial baroreflex gain does not differ if pressure is raised from lower compared with resting levels; thus, acquisition of ultrasound images (ie, carotid diameters) once every ~2 seconds did not compromise the ability to make robust baroreflex gain estimates with the modified Oxford technique (see below, Baroreflex Gain Estimates). The drug injection sequence was repeated twice to assess test-retest reliability of baroreflex indices. Trials were separated by at least 15 minutes to allow full recovery to baseline. A total of 3 trials were necessary to obtain adequate image quality for at least 2 trials.

Data Analysis

Hemodynamic Data

Beat-by-beat arterial pressure and ECG waveforms were digitized to computer at 500 Hz for subsequent offline analysis with signal processing software (WINDAQ, Dataq). 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 the minimum of the beat-by-beat pressure waveform.
Carotid Diameter
We used image analysis software (CVI Analysis, Information Integrity) closely conforming to that described in detail previously. Briefly, several points in proximity to the edges of the near and far walls were selected. The software fit a spline that contained 100 points to each set of preliminary edge points. The direction locally perpendicular to each spline point was calculated, and the image was interpolated for 6 pixels in either direction perpendicular to the spline. The location along the interpolated line with the largest sum of first and second derivatives was then chosen as the best edge point. This process was repeated for each spline point along the 2 edges. Edge points, which represent weak edges (ie, points selected at edges with $<20\%$ of the maximum sum of the intensity derivatives of all edge points), were replaced by linear connections between the nearest strong edge points. The 2 sets of edge points extracted by the above procedure were modeled as a pair of parabolas, according to the user-selected preliminary edge points were reused for successive images and could be moved as a group to compensate for translational motion of the walls. The software fit a spline that contained 100 points to each set of preliminary edge points. The 2 sets of preliminary edge points were replaced by linear connections between the nearest strong edge points. The 2 sets of edge points extracted by the above procedure were modeled as a pair of parabolas, according to the user-selected preliminary edge points were reused for successive images and could be moved as a group to compensate for translational motion of the walls. The software fit a spline that contained 100 points to each set of preliminary edge points. The 2 sets of edge points extracted by the above procedure were modeled as a pair of parabolas, according to the user-selected preliminary edge points were reused for successive images and could be moved as a group to compensate for translational motion of the walls. The software fit a spline that contained 100 points to each set of preliminary edge points. The 2 sets of edge points extracted by the above procedure were modeled as a pair of parabolas, according to the user-selected preliminary edge points were reused for successive images and could be moved as a group to compensate for translational motion of the walls. The software fit a spline that contained 100 points to each set of preliminary edge points. The 2 sets of edge points extracted by the above procedure were modeled as a pair of parabolas, according to the user-selected preliminary edge points were reused for successive images and could be moved as a group to compensate for translational motion of the walls. The software fit a spline that contained 100 points to each set of preliminary edge points. The 2 sets of edge points extracted by the above procedure were modeled as a pair of parabolas, according to the user-selected preliminary edge points were reused for successive images and could be moved as a group to compensate for translational motion of the walls.

Association of Carotid Diameters and Hemodynamic Data
To minimize measurement error in carotid diameter estimation, we used a 3-diameter moving average (Figure 1, bottom panels). Custom software associated diameters to appropriate pressures and R-R intervals. Within a cardiac cycle, we associated systolic pressure with the largest carotid diameter and diastolic pressure with the smallest diameter before systole. We associated the R-R interval with systolic pressure and systolic diameter after appropriately accounting for baroreflex delays; pressure and diameter were associated with the concurrent R-R interval at heart periods $>800$ ms and the subsequent R-R interval at heart periods between 500 and 800 ms.

Results

Baroreflex Gain Estimates

Gain Calculations
Although heart rate is often used to characterize vagally mediated responses to baroreceptor input, we used the R-R interval, which is most linearly related to vagal outflow. We averaged systolic pressures, carotid diameters, and R-R intervals across 3 mm Hg systolic pressure increments, or bins, to account for variations related to ventilation and measurement error (Figure 2). This binning procedure not only increases the confidence in the derived relations among systolic pressure, systolic carotid diameter, and R-R interval, but it also reveals the sigmoid nature of these relations. By visual inspection, we excluded bins in the threshold and saturation regions of the pressure/R-R interval relation for all baroreflex analyses (Figure 2, open circles) to estimate the linear gain of mechanical (pressure/diameter), neural (R-R interval/diameter), and integrated (pressure/R-R interval) baroreflex responses. In our laboratory, visual determination of saturation and threshold regions are highly correlated to the use of the second derivative of the relation ($r^2=0.93$). The resulting linear correlation coefficients averaged 0.89±0.09 (range 0.74 to 0.98), 0.88±0.03 (range 0.68 to 0.97), and 0.95±0.02 (range 0.81 to 0.99) for the mechanical, neural, and integrated responses, demonstrating the close linear relations among systolic pressure, systolic carotid diameter, and R-R interval in these subjects.

Estimate Variance
A Bland-Altman plot of the 3 estimates of cardiac vagal baroreflex control is shown in Figure 3. Variability in repeated measures relative to the mean was comparable for each baroreflex component and the integrated baroreflex gain, with no systematic bias apparent across gains. The mean difference between trials was $0.06\times10^3$ mm/mm Hg for the mechanical component, $0.92\times10^3$ ms/mm for the neural component, and $0.32$ ms/mm Hg for integrated baroreflex gain. Furthermore, a Leven test for homogeneity of variance between individual trials indicated that measurement variability was no different among the 3 baroreflex gain estimates: $\sigma^2_{\text{mechanical}}=0.69\pm0.17$, $\sigma^2_{\text{neural}}=0.56\pm0.21$, and $\sigma^2_{\text{integrated}}=0.74\pm0.16$, ($P=0.67$).

Relations Among Estimates
Simultaneous measures of mechanical transduction of pressure into stretch near the carotid barosensory area (mm/mm Hg), neural transduction of stretch into vagal outflow (ms/mm), and the resulting integrated baroreflex...
gain (ms/mm Hg) are shown for 1 subject in Figure 4 (from raw data in Figure 1). This subject demonstrated a marked increase in heart period as systolic pressure increased from ~106 to 109 mm Hg (right panel), apparently because of nonlinearity in mechanical pressure transduction (left panel) rather than neural control (middle panel). In the group as a whole, although mechanical and neural gains were only modestly related to integrated cardiac-vagal baroreflex gain ($r = 0.53, P = 0.04$, and $r = 0.35, P = 0.18$), the simple product of the 2 gains was almost equivalent to the integrated gain (Figure 5). Thus, in these young healthy subjects, neither single component accounts for the integrated baroreflex gain; instead, mechanical and neural components act in concert to produce the integrated baroreflex response.

**Discussion**

Our approach to baroreflex assessment adds only a single variable to a standard pharmacological measure, yet provides unique insight to baroreflex function previously unobtainable in humans. Acquisition of beat-by-beat carotid diameters, arterial pressures, and heart periods during the modified Oxford technique allows evaluation of the mechanical and neural components that determine integrated cardiac-vagal baroreflex gain. Although the relations among these variables may contain important information on complex nonlinearities, they are highly reproducible for linear gain assessments, which are most commonly used to characterize autonomic cardiovascular control. As a result, the mechanical and neural baroreflex components can be estimated concomitant with the standard integrated relation via a single additional variable: carotid arterial diameter. Appropriate application of this technique may provide specific insight to alterations in autonomic control in diverse physiological and pathophysiological states.

**Technical Considerations**

The addition of beat-by-beat carotid diameters to arterial pressure and heart period requires acquisition, maintenance, and analysis of B-mode ultrasound images. Though baseline diameters were not different between trials (6.42 ± 0.14 vs. 6.41 ± 0.13; $P = 0.76$), indicating our image acquisition was reliable, we found that maintenance of image quality throughout the pharmacological intervention can be difficult;
necessitating 3 trials to consistently obtain 2 complete trials with adequate images for analysis in all subjects. Despite this difficulty, these data reliably characterize the entire range of barosensory vessel engagement during dynamic pressure changes that, to date, have not been described in humans. Other technologies for carotid diameter measurement are available.\textsuperscript{9,19} However, they are limited by an inability to estimate carotid diameters for more than a short duration, usually \(\approx 5\) to 10 seconds. (The duration of the pressure rise in our subjects averaged 76 seconds.) These processes reduce ultrasound information to only a small portion of the signal, producing diameter estimates with greater frequency resolution than B-mode imaging, yet no greater accuracy.\textsuperscript{15} It should be noted that our technique is affected by computer hardware limitations. As Figure 1 shows, after the initial few cardiac cycles, image sets were only acquired on approximately every other cardiac cycle because of the speed of the hard drive. Although we were not able to acquire carotid diameter data for every cardiac cycle for the current data, we acquired diameters for at least 60 cardiac cycles, encompassing the entire period of dynamic baroreceptor activation. Moreover, we have recently begun to use a high-speed hard drive and now acquire diameter data on nearly 100% of all cardiac cycles. Additionally, B-mode ultrasonography allows both real-time and post-hoc evaluation of data quality during a procedure that markedly alters the diameter of a highly elastic vessel. Pulsatile carotid diameter changes are \(\approx 15\%),\textsuperscript{9,20} and we observed an additional \(\approx 15\)% change in systolic diameter during bolus vasoactive drug infusion in our subjects. These large changes can easily alter the angle of ultrasound insonation and cause misestimation of diameter. However, B-mode imaging provides direct visual feedback to both ultrasound operator and image analyst to identify shifts that would create erroneous diameter changes. This provides a level of confidence in the data that compensates for the level of difficulty in its acquisition.

Our estimates of mechanical transmission, neural afferent-efferent activation, and integrated baroreflex gain were as reproducible, or more so, than other techniques. Reported coefficient of variations for cardiac-vagal baroreflex gain are \(\approx 29\)% for Valsalva’s maneuver;\textsuperscript{21} 40% for the spontaneous sequence method, and 52% for both frequency domain and Oxford techniques.\textsuperscript{22} We found a coefficient of variation of 23% for standard integrated baroreflex gain, slightly less than, but not significantly different from 32% for the mechanical component and 26% for the neural component. Although a third trial may have reduced the error of our estimates and may be necessary in individuals with lower baroreflex gain, 2 trials resulted in comparable variance without any systematic bias in each component and integrated gain. Thus, reliance on ultrasound-derived, beat-by-beat carotid diameters for our 2 baroreflex component estimates introduces no greater trial-to-trial variability to baroreflex gain assessments via the modified Oxford technique.

**Physiological Considerations**

We devised an approach to baroreflex physiology based on established findings that support a line of evidence for this technique. Arterial baroreceptive afferents are primarily responsive to pressure-related stretch.\textsuperscript{5,6} In their classic work, Kober and Arndt\textsuperscript{20} showed that changes in transmural arterial pressure were linearly related to changes in carotid diameter, suggesting carotid arterial pressure may be used as a surrogate for arterial diameter. However, significant deformation of barosensory areas in humans\textsuperscript{23,24} and altered afferent baroreceptor activity in dogs\textsuperscript{25} may occur in the absence of measurable arterial pressure changes. Therefore, pressure may not index the true arterial baroreceptor stimulus. In fact, the prevailing arterial pressure as well as efferent nervous activity appear to be determined by barosensory vessel diameter. In patients after endarterectomy, those with increased carotid sinus diameter had greater carotid sinus nerve activity and lower postoperative blood pressure.\textsuperscript{26} Thus, excising carotid tissue altered the relation between pressure and diameter but not that between diameter and nervous activity. Although the only human data demonstrate a simple proportionality between carotid sinus dimensions and integrated carotid sinus nerve activity within the cardiac cycle,\textsuperscript{6} animal data suggest a linear proportionality applies throughout broader pressure ranges.\textsuperscript{27,28} These data suggest that carotid diameter may serve as an appropriate index of the arterial baroreceptor stimulus and resulting efferent activity.

Afferent baroreceptor stimulation generates R-R interval responses that are proportional in humans\textsuperscript{7} and linear in cats.\textsuperscript{29} Although cardiac chronotropy can also be modulated by cardiovascular sympathetic afferents,\textsuperscript{30,31} rapid heart period responses to baroreflex engagement in humans is likely to be predominantly mediated through efferent vagal activity,\textsuperscript{2} because cardiac sympathetic activity tends to have longer lag time.\textsuperscript{32} Moreover, though acute and chronic cardiac sympathetic blockade may augment baroreflex sensitivity,\textsuperscript{33} baroreflex-mediated heart period slowing does not occur without intact cardiac parasympathetic activation.\textsuperscript{34,35} Thus, the R-R interval provides a commonly accepted estimate of the cardiac vagal efferent response to arterial baroreflex engagement.

Assessing each of the above steps in the baroreflex control of arterial pressure is difficult in the intact human. However, given that barosensory vessel stretch is a key determinant of reflex responses, we thought the simple addition of a diameter estimate would provide incisive information on baroreflex physiology. The standard relation of R-R interval to arterial pressure is a global estimate of the cardiac vagal limb of systemic control. We derived 2 additional relations: arterial pressure to carotid diameter and carotid diameter to R-R interval. The first provides information on the mechanical transduction of the controlled variable to the input variable for baroreflex engagement. The second provides a global index of the complex integration of baroreceptor afferent activation and CNS efferent outflow, resulting in cardiac slowing or acceleration. Thus, 2 distinct components that determine integrated baroreflex gain can be assessed.

It should be noted that a linear model is commonly used to estimate cardiac-vagal baroreflex gain. However, baroreflex-mediated R-R interval responses to arterial pressure changes demonstrate a clear sigmoid pattern; R-R interval responses are minimal below threshold and above saturation pressures and are maximal at some point within this range.\textsuperscript{36} Following
of baroreflex gain, the most important aspect of pressure
between threshold and saturation produces a robust estimate
Oxford testing. However, a linear model applied to the region
demonstrate the entire sigmoid range with the modified
previously that not all, nor all relations, will consistently
intriguing aspect of baroreflex physiology, we have shown
though using a sigmoid model would provide insight to this
mechanical and neural components of the reflex arc. Al-
likely the result of the interplay between nonlinear and linear
responses. This subject demonstrated a clear nonlinearity in
of events linking arterial pressure changes to R-R interval
not capture some aspects of the complex, nonlinear, cascade
of baroreflex pressure regulation. This work defined and tested an approach to baroreflex
and neural afferent-efferent activation were related strongly
to integrated baroreflex gain within subjects but only modestly among subjects. The simple product of the components
almost equaled the measured integrated gain for each trial.
This may not seem surprising, but each slope was determined
independently and thus no mathematical dependency existed.
Therefore, the strong relationship indicates that these 2
components encompass the primary determinants of arterial
baroreflex gain. There was ≈13% of the actual measured
integrated gain not accounted for, indicating there are other
aspects of baroreflex function not explained by these
measures. In the group as a whole, the neural component was not
significantly related to integrated gain, whereas the mechanical
component more strongly predicted integrated gain. Data
from Lage et al on basal measures of carotid compliance
show a similar relation to baroreflex gain in normotensive
and hypertensive adults. However, our laboratory and others
have reported that basal measures of carotid stiffness were
more strongly related to integrated baroreflex gain in young
adults (r = 0.78 to 0.82). This might be explained by the
fact that stiffness measures are not pressure dependent whereas both our mechanical component and compliance
measures are pressure dependent. However, our technique
provides information relevant to the dynamic, and possibly
more complex, mechanical transduction of pressure throughout the responsive range of the baroreflex.

**Clinical Implications**

Given our homogeneous study population, it may not be surprising that neither mechanical pressure transduction nor
neural baroreflex gain were strongly associated with integrated baroreflex gain. In young healthy adults without
arterial stiffening or autonomic neuropathy, it is quite possible that both mechanical and neural components play equally
important roles in baroreflex pressure regulation. However, insight to baroreflex function may be gained in populations
known to have altered vascular mechanics, neurological lesions, or depressed baroreflex function of unknown origin.
For example, coronary artery disease patients are known to have depressed integrated baroreflex gain that is presumed to
be parasympathetic in origin. However, preliminary data from our laboratory on 3 adults with coronary artery disease
shows the previously described decrement in integrated baroreflex gain compared with 4 age-matched healthy adults
(Figure 6), yet their neural baroreflex component is similar. This would suggest that autonomic neural function is intact
and that vascular structural change underlies blunted baroreflex function in coronary disease. However, the use in a
clinical setting would be dependent on establishing normative
data for each baroreflex component, which at present does not exist.

This work defined and tested an approach to baroreflex
physiology. Our findings, as well as the above preliminary data, demonstrate that important information on autonomic
physiology may be derived with appropriate application of
our technique. Although the standard description of baroreflex function from heart period and arterial pressure relations
provides broad insight to autonomic control, the simple
addition of a single key physiological variable may provide
more mechanistic insight. Assessment of the mechanical and
neural baroreflex components via our technique may provide
critical understanding of autonomic function in human health and disease.

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**Figure 6.** Differences in integrated baroreflex gain (left) and transduction gain (right) from preliminary data from healthy adults and coronary artery disease subjects.
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References
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