Validation of a Method for Noninvasive Measurement of Central Arterial Pressure

Tali Sharir, Alon Marmor, Chih-Tai Ting, Jaw-Wen Chen, Cheng-Pen Liu, Mau-Song Chang, Frank C.P. Yin, and David A. Kass

The goal of this study was to validate a newly improved noninvasive method for calibrated measurement of the ascending portion of the central arterial pressure wave in humans. Noninvasive pressure waveforms were generated by measuring the time delay between the R wave of the electrocardiogram and onset of brachial artery flow (by Doppler) during computer-controlled upper arm cuff deflation. This delay shortens with falling cuff pressure (becoming near constant at and below diastolic pressure), so that a plot of pressure versus time delay yields the ascending portion of the arterial waveform. These waveforms were compared with simultaneous invasive ascending aortic pressures in 57 adult patients (31 by fluid manometer [group A] and 26 by catheter-tipped micromanometer [group B]) during routine cardiac catheterization. Patient age ranged from 26 to 77 years. Eighty percent of group A patients and 40% of group B had coronary artery disease. Noninvasive systolic and diastolic pressures were very similar to invasive values in both groups ($P_{\text{nl}}=0.98 \cdot P_{\text{nl, r}}=0.99, p<0.0001$). Instantaneous pressure differences between waveforms were also similar in both groups, averaging between 4.5 and 5.5 mm Hg. Micromanometer and noninvasive pressure data were also obtained before and after intravenous nitroglycerin (n=5) and isometric handgrip (n=8) and demonstrated good agreement. A potential application of these pressures is for estimating maximal ventricular power to assess systolic function. This was tested using invasive pressure-volume data from four patients under a variety of conditions (exercise, pacing, etc.). Maximal power based on the noninvasive pressures was nearly identical to the fully invasive values. Thus, the new noninvasive pressure technique provides accurate measures of central systolic and diastolic pressures, provides good approximations to the ascending portion of central arterial pressure wave in many subjects, and can be applied to accurately measure maximal ventricular power. (Hypertension 1993;21:74–82)

**KEY WORDS** • blood pressure • hemodynamics • ventricular function • hypertension, essential • blood pressure determination

There is growing interest in the quantitative and descriptive analysis of the arterial pressure waveform. In addition to the recognized importance of accurate central arterial systolic and diastolic pressure measurement, the pulse waveform itself and calibrated pressures between these end points can provide valuable information regarding vascular and ventricular function. Examples of quantitative hemodynamic applications include the combining of noninvasive pressure with echocardiographic dimensions or flow data to assess systolic stress/volume ratios or maximal left ventricular power. This requires calibrated central arterial pressures beyond merely peak and trough, limiting the utility of standard cuff techniques. Although applanation tonometry can provide useful waveform information, it is semiquantitative in that it requires independent calibration.

An alternative noninvasive approach for obtaining calibrated central arterial pressures was reported by Marmor et al in 1987. The method involved measuring the time delay between the R wave of the electrocardiogram (ECG) and the brachial pulse during gradual deflation of an arm cuff. The delay shortened with declining cuff pressure, enabling pressure–time data for the ascending limb of the arterial pressure wave to be estimated. This earlier study, using preliminary methodology, reported results from a small group of middle-aged subjects. The instrumentation has since undergone substantial modification, with the use of a Doppler sensor to detect the appearance of brachial artery flow and new and sophisticated computer algorithms for real-time signal processing and automated pressure wave analysis and timing.

The primary goal of the present study was to test the accuracy of this newly modified and further developed system in a much larger adult population, including data before and after loading interventions. A potentially useful application of these pressures is for noninvasive systolic function assessment by estimation of maximal ventricular power. Therefore, the present study also
examined the validity of this application by comparing simultaneous power estimates based on the noninvasive arterial versus invasive ventricular pressures.

Methods

Study Population

The study group for invasive versus noninvasive pressure comparisons was composed of 57 adult human patients referred for routine cardiac catheterization. Thirty-one patients were studied at the Johns Hopkins Medical Institutions, with invasive pressure measured by a fluid-filled catheter system. A second group of 26 patients was studied at the Veterans General Hospital, Taipei, Taiwan, using a catheter-tipped micromanometer for invasive pressure measurement. Table 1 summarizes general clinical characteristics of each patient group. Patient ages in the two groups ranged from 26 to 77 years. Rest left ventricular function varied among patients, with ejection fractions as low as 20% to as high as 80%. In a subset of the micromanometer-comparison patients, data were also obtained before and after intravenous nitroglycerin (n=5) and isometric handgrip exercise (n=8).

To test the accuracy of maximal left ventricular power derived from noninvasive pressure, we studied four additional patients. These patients underwent routine coronary angiography, ventriculography, and right heart catheterization. In addition, pressure-volume loops were measured by a conductance catheter–micromanometer technique, as previously described in detail.9–11 Simultaneous noninvasive arterial pressures were also obtained. Informed consent was provided by all patients, and the protocols were approved by the Joint Committee on Clinical Investigation of The Johns Hopkins Medical Institutions and the Veterans Hospital in Taipei, Taiwan.

Noninvasive Pressure Wave Determination

Noninvasive arterial pressure was measured with a newly designed computer-controlled device (Cardiospec 2000, SRD Medical, Shorashim, Israel). This device consists of three components: 1) a sphygmomanometric arm cuff attached to an air pressure unit, 2) a Doppler transducer applied to the arm at the antecubital space over the brachial artery, and 3) an ECG monitoring system. All three elements are controlled by an 80286 processor–based computer. ECG and Doppler flow signals are amplified and real time filtered in time and frequency domains to minimize motion and muscle artifacts and to optimize the consistency of upstroke identification. This is important to assure the reliability and stability of the measurements.

For measurement of pressures, the cuff is automatically inflated until intracuff pressure exceeds peak systolic blood pressure and the Doppler signal at the brachial artery disappears. As the cuff pressure declines (computer-controlled deflation at 2 or 3 mm Hg per beat), brachial flow returns, and the onset of flow is detected for each beat and referenced to the time from the preceding R wave. The time delay is composed of three elements: 1) the pre-ejection period, from the peak R wave to the opening of the aortic valve; 2) the propagation time of the pressure wave from the heart to brachial artery; and 3) the time required for arterial pressure to exceed the intracuff pressure and thus allow flow to be sensed by the Doppler probe.

Both the first and second time delays are essentially independent of cuff pressure. This is because inflation of the cuff only trivially alters proximal arterial distending pressure, and thus, isovolumic contraction time and pulse wave velocity are minimally altered. This lack of cuff inflation effect on central aortic pressure was confirmed in six patients. Inflation altered pressure by a mean of only 1.02 mm Hg (maximum, 2 mm Hg; minimum, −0.23 mm Hg) over a cardiac cycle. The last delay, in contrast, shortens with declining cuff pressure. Once cuff pressure falls below diastolic pressure, this delay is little further changed and primarily equals the pulse propagation time plus pre-ejection period. Plotting cuff pressure against the corresponding time delay from multiple successive cardiac cycles generates the ascending arterial pressure waveform (Figure 1). Real-time computer graphics display ECG and Doppler flow signals, R wave and flow onset times, cuff pressure, and the pressure–time points of the arterial wave during cuff deflation. This provides visual confirmation of the adequacy and stability of the ECG and Doppler signals and consistency of the upstroke triggers throughout the measurement period.

Although data are plotted as pressure varying with time, they are obtained with time being a function of declining cuff pressure. Thus, the data are first sorted to be monotonically increasing with respect to time. Systolic pressure is the maximal pressure value, usually the point with the longest time delay. Diastolic pressure is taken at the inflection point of the noninvasive pressure curve. This is calculated by finding a tangent to the waveform anchored at pressure=0, time (t)=0.8 times the mean time delay for the pressure=0 data points of the run (dashed line in Figure 1). This method avoids the need for derivative analysis, which can be complicated by signal noise. Once systolic and diastolic end points are determined, the noninvasive pressure data are phase shifted so that the onset of ejection (diastolic pressure) occurs at t=0 (see also Figure 1).

Invasive Pressure Determination

Invasive ascending aortic pressure was measured by either fluid-filled pigtail catheter (n=31) or micromanometer (n=26). The frequency characteristics of the fluid-filled system (transducer plus 110-cm catheter filled with saline) are provided in Figure 2, using a

<table>
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<tr>
<th>Table 1. Characteristics of the Two Study Groups</th>
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<tr>
<td>Method of invasive pressure measurement</td>
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<tr>
<td>---------------------------------------------</td>
</tr>
<tr>
<td>Fluid-filled</td>
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<td>Micromanometer</td>
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CAD, coronary artery disease; LV, left ventricle.
simultaneous micromanometer pressure response as a standard. This system had a flat response to 6 Hz, with minimal amplitude amplification and negligible phase delay up to 10 Hz. Six harmonics are generally adequate to describe most of the characteristics of the arterial waveform. These data were digitized into 200 equi-spaced points, and results of five sequential cycles were averaged to yield a single invasive pressure waveform. The time coordinates were converted into milliseconds and adjusted with an automated algorithm so that $t=0$ occurred at the pressure upstroke to synchronize with the noninvasive pressures. Micromanometer pressures were digitized at 200 or 250 Hz, and a similar number of beats were digitally averaged to yield the comparison waveform.

For comparison between invasive and noninvasive systolic and diastolic pressures, one to four noninvasive estimates were averaged and results were compared to simultaneous invasive values. Instantaneous pressure differences between invasive and noninvasive ascending waveforms ($\Delta P$) were determined by interpolating the invasive waveform to assess $\Delta P$ at each of the noninvasive time points. Data were then averaged over equi-spaced bins to generate a mean difference profile.

In a subset of group B patients, data were also compared before and after two loading interventions (isometric handgrip and nitroglycerin). Automated synchronization and analysis of the respective invasive and noninvasive waveforms were handled identically to the baseline data described above.

Comparison of Noninvasive and Invasive Pressure for Power Assessment

A potentially valuable application of the new noninvasive pressures is for measurement of ventricular contractile function assessed by maximal power ($PWR_{max}$). We have previously shown in animals that central aortic pressure can substitute for left ventricular pressure to assess $PWR_{max}$. To further test if $PWR_{max}$ could be accurately determined using the new noninvasive arterial pressures in humans, we compared $PWR_{max}$ obtained from the product of noninvasive pressure and flow to that determined from fully invasive measurements in four additional patients. After routine right and left heart catheterization, an 8F conductance catheter (Webster Labs, Baldwin Park, Calif.) was advanced to the left ventricular apex and connected to a stimulator/processor unit (VCU, Cardiac Pacemakers Inc., St. Paul, Minn.) for left ventricular volume measurement. Left ventricular pressure was obtained by a 2F micromanometer (SPC-320, Millar Instruments Inc., Houston, Tex.) advanced down the lumen of the conductance catheter. Micromanometer pressure was zero-balanced to air and calibrated to a fluid-filled transducer. The noninvasive pressure cuff was placed on the left arm, and simultaneous noninvasive central arterial pressures were obtained. Data were obtained at base-

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**Figure 1.** Graph shows example of noninvasive arterial pressure measurement. As pressure in an arm cuff is reduced, the time delay between the R wave of the electrocardiogram and the onset of brachial artery flow (sensed by a Doppler probe) shortens. Plotting cuff pressure versus time delay for each beat results in raw data points shown by open triangles. Peak systolic and diastolic pressures are automatically determined (large diamonds). For the latter, this is done by placing a tangent to the waveform (dashed line) and finding the intersection point. Pressure-time data are then phase shifted (small diamonds) so that $t=0$ at the diastolic pressure point (onset of aortic valve opening).

**Figure 2.** Line graphs show frequency response of fluid-filled system. Top panel: Amplitude; bottom panel: phase, on a semilogarithmic scale. Amplitude and phase are referenced to a simultaneous micromanometer signal. The fluid system had a flat frequency response to 6 Hz, with only slight amplitude magnification and phase delay between 6 and 10 Hz. This is adequate for the present analysis.
line, during isotonic exercise (handgrip) and atrial pacing (90–150 beats per minute), and after intravenous infusion of esmolol or dobutamine (total of 18 different measurements, approximately four or five per patient).

Invasive pressure/volume data were digitized at 200 Hz. Because left ventricular power is the instantaneous product of pressure and flow, a flow signal was required. This was determined from the time derivative of the volume catheter signal. Catheter volumes were calibrated to a four-term Fourier series. This was analytically differentiated to yield flow (dV/dt). For invasive power measurement (PWR<sub>inv</sub>), micromanometer pressure and dV/dt signals were multiplied, and the maximal value of this product was PWR<sub>inv</sub>:

\[
PWR_{\text{inv}}(W) = [P_LV(\text{mm Hg}) \cdot dV/dt(\text{ml/sec})]_{\text{max}} \cdot 1.33 \cdot 10^{-4}
\]

Substitution of noninvasive pressures for P<sub>LV</sub> in this equation was performed using linear interpolation of the data to obtain 100 equispaced points in time from diastolic to systolic pressure. This result was smoothed with a five-point sliding filter (Hanning). The noninvasive data were synchronized with the flow signal and multiplied by flow to yield instantaneous power and PWR<sub>inv</sub>.

**Analysis and Statistics**

Noninvasive versus invasive systolic and diastolic pressures and PWR<sub>inv</sub> estimates were compared by linear least-squares regression. Instantaneous pressure differences between noninvasive and invasive waveforms were combined from all patients by first normalizing each data set to the respective invasive pressure time scale, so that it spanned t=0 at the onset of ejection (diastolic pressure) to t=1.0 at peak systolic pressure. As noted above, these data were bin averaged at approximately a 10% time increment (from t=0 → 1) to determine a mean difference profile.

**Results**

**Noninvasive Versus Invasive Pressure**

Systolic and diastolic noninvasive pressures were very similar to corresponding invasive ascending aortic values (Figure 3). Table 2 provides regression results for systolic, diastolic, and combined pressure analyses. Data sets obtained by fluid-filled catheter and micromanometer both demonstrated similar excellent agreement, with slopes not significantly different from one and intercepts not significantly different from zero for all comparisons. Regression slopes assuming a zero axis intercept are also provided in Table 2. For combined data, this yielded a slope of 0.98–0.99, with a correlation coefficient (r) of 0.98 and a standard error of the estimate (SEE) of 6.1 mm Hg.

Figure 4 displays several examples from patients with varying clinical histories, ages, and ambient pressures (from upper left moving clockwise, ages are 59, 75, 26, and 29 years, respectively). The two top panels are from the fluid-filled catheter comparison group and the bottom two from the micromanometer group. In each instance, the invasive ascending aortic pressure upstrokes were reasonably well traced by the noninvasive measurements both in shape and magnitude. This response was observed in many of the patients, particularly those older than 55 years.

Noninvasive pressure waveforms did not always follow the invasive curves as well as displayed in Figure 4. Figure 5 displays two worst-case examples. The top panel displays an example from the fluid-filled catheter group in which good pressure agreement was observed in the early upstroke, but there were disparities after a prominent inflection point. This pattern was observed in <18% of patients, generally those with an early and prolonged plateau in the pressure waveform. Despite these waveform differences, absolute systolic and diastolic noninvasive pressures remained consistent with invasive values. The bottom panel of Figure 5 shows an example from the micromanometer group. This patient

**TABLE 2. Regression Parameters for Systolic and Diastolic Invasive (x) Versus Noninvasive (y) Pressure Comparisons and Combined Data From the Two Study Groups**

<table>
<thead>
<tr>
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<th>Fluid-filled group (n=31)</th>
<th>Micromanometer group (n=26)</th>
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<td></td>
<td>Slope*        Intercept</td>
<td>r</td>
</tr>
<tr>
<td>Systolic</td>
<td>0.96 (0.98)   2.8</td>
<td>0.97</td>
</tr>
<tr>
<td>Diastolic</td>
<td>0.86 (0.99)   9.9</td>
<td>0.87</td>
</tr>
<tr>
<td>Both</td>
<td>0.96 (0.98)   3.1</td>
<td>0.99</td>
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SEE, standard error of the estimate. All regressions are highly significant at p<0.0001.

*Regression slopes in parentheses are provided for line passing through the origin.
had severe aortic regurgitation, and the noninvasive waveform deviated during the midportion of the wave. This is an extreme example of such pressure deviation, but a small degree of overestimation was not that infrequent. These patterns may result from inaccuracies in the precise timing of the ECG and flow upstrokes, as well as real differences in upper brachial versus central aortic pressures due to wave reflections. They tended to occur more commonly in younger subjects.

Figure 6 shows mean instantaneous difference analyses between synchronized noninvasive and invasive pressures for both fluid-filled and micromanometer data groups. Overall, there was good agreement. The mean difference was +5.5±7.7 mm Hg for the fluid-filled data (n = 1,051) and +4.7±7.1 mm Hg for the micromanometer data (n = 693). Despite different clinical and methodological aspects of the two patient groups, pressure difference trends were nearly the same. Errors averaged between 0 and 8 mm Hg, with the largest discrepancies occurring during early-to-mid ejection. This is likely due to the rapid pressure rise during this period. The micromanometer data displayed somewhat less error during the initial 20% of ejection as compared with the fluid-filled data, which may in part reflect the frequency response limitations of the latter. Errors at the very onset of ejection and toward peak systole were less, consistent with the high correlation of diastolic and systolic end points displayed in Figure 3.

**Nitroglycerin and Handgrip Pressure Comparisons**

Figure 7 displays an example of micromanometer and noninvasive pressure data obtained before and after intravenous nitroglycerin in one patient (top panel) and isometric handgrip in another (bottom panel). Changes in central aortic systolic and diastolic pressures were both well estimated by the noninvasive method, and the waveforms reasonably followed the respective invasive pressure curves. Group mean results for systolic and diastolic pressure changes from both interventions are summarized in Table 3. Mean invasive systolic and diastolic pressures fell by -21.3±10.1 and -1.9±3.5 mm Hg, respectively, with nitroglycerin and rose by +23.3±7.7 and +13.2±7.6 mm Hg with isometric handgrip. Corresponding noninvasive estimates of these changes were statistically similar (Table 3).

Figure 8 displays systolic and diastolic pressures for each patient before and after a given intervention (connected by line segments). The group data fell along the line of identity, with an average linear regression relation given by Pressure\(_{\text{invasive}}\) = 0.96 · Pressure\(_{\text{invasive}}\) + 3.6, \(r=0.98, p<0.0001\). Thus, in addition to providing good rest estimates, the noninvasive system also reasonably tracked changes in central arterial pressures after load-interventions.

**PWR\(_{\text{max}}\): Invasive Pressure Versus Noninvasive Pressure Determination**

The reasonable accuracy of the noninvasive pressure measurements suggested that these pressures could be used to estimate maximal left ventricular power in human patients. This application was examined in four additional patients. Figure 9 displays an example of left ventricular power determined from invasive left ventricular micromanometer pressure versus noninvasive cuff pressure. The top panel shows a digitized volume signal (squares) with the four-term Fourier fit (solid line) and the analytical derivative of this fit, which is volume flow (dashed line). The middle panel displays micromanometer (ventricular) and noninvasive (arterial) pressure, and the bottom panel shows the two power curves resulting from the product of each respective pressure with flow. Both invasive (PWR\(_{\text{max}}\)) and noninvasive (PWR\(_{\text{max}}\)) maximal power measurements had nearly identical values in both magnitude and timing. Note that PWR\(_{\text{max}}\) occurred before peak systolic pressure in this example, so that a simple product of \(P\) and maximal flow would overestimate PWR\(_{\text{max}}\). The magnitude of overestimation varied 2–30% in these patients, being greatest in those with enhanced wave reflections and wide pulse pressures.

Figure 10 displays PWR\(_{\text{max}}\) and time to PWR\(_{\text{max}}\) (ttPWR\(_{\text{max}}\), measured from the peak R wave of the
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FIGURE 5. Graphs show two worst-case examples in which absolute values of noninvasive diastolic and systolic pressures were very close to the invasive measurements, but the waveform patterns differed, particularly after an early plateau-inflation point in the aortic pressure. Top panel: Data from a 37-year-old with normal left ventricular function and coronary arteries (fluid-filled catheter group). Bottom panel: Data from a 59-year-old with severe aortic regurgitation (micromanometer group). Real pressure discrepancies from reflected waves as well as methodological limitations likely contributed to such disparities. Such marked disparities occurred in fewer than 20% of patients.

Discussion

The major goal of this study was to evaluate the accuracy of a newly developed and improved method for noninvasive measurement of calibrated central ascending arterial pressure in humans. Comparisons between invasive and noninvasive systolic and diastolic pressures as well as instantaneous pressures generally revealed excellent agreement. This was confirmed in two entirely independent patient populations, with studies performed in separate laboratories and using different invasive measurement techniques. Furthermore, relative changes in pressure induced by nitroglycerin and handgrip exercise were also well estimated by the noninvasive system. As a new application of these data, we also assessed whether noninvasive arterial pressures could substitute for invasive ventricular pressure to determine maximal ventricular power. The results revealed excellent agreement in both amplitude and timing under varying conditions. Thus, the new technique should prove a useful addition to noninvasive methods for assessing arterial and ventricular function.

Pressure Measurement

Similar to approaches that rely on brachial artery flow at the antecubital fossa, the present technique likely reflects pressures in the proximal brachial artery (above the cuff occlusion point). In older patients, such as those typically presenting with cardiovascular disease, pressure amplification between the ascending aorta and upper brachial artery is less marked, and these pressures correlate well. This is not always true. In very young individuals with compliant vasculatures and in patients with rapid heart rates or marked peripheral vasoconstriction, the brachial pressure waveform becomes less representative of more central aortic press-

ECG) for all 18 different conditions from the four patients. Estimates based on the noninvasive pressures were virtually identical to the corresponding invasive value. Linear regression analysis yielded the following relations: $P_{WR_{max}} = 0.99 \cdot P_{WR_{max}} - 0.2$, $r = 0.97$, $SEE = 0.72 \text{ W}$; and $tPWR_{max} = 0.91 \cdot tPWR_{max} + 19.5$, $r = 0.98$, $SEE = 10.8 \text{ msec}$, both not significantly different from the line of identity.

FIGURE 6. Graph shows instantaneous pressure discrepancies ($\Delta P$) between noninvasive (NonInv) and invasive (Inv) pressures. Each patient’s data was normalized in time so that the onset of ejection was at $t=0$ and the time of invasive pressure peak systole was at $t=1$. Data were then bin averaged to display mean±SD difference trend. Results for fluid-filled and micromanometer study groups are both shown. Data are very similar between groups, despite differences in age, clinical makeup, and invasive measurement techniques. Absolute pressure discrepancy varied about a mean of between 4.7 and 5.5 mm Hg. Maximal mean discrepancy was approximately 8 mm Hg in both data sets.
Invasive and noninvasive pressures were generally observed in a younger subset of the present study, appearing as a phase advance in the noninvasive data after a plateau in the central pressure waveform (e.g., Table 3). Indeed, larger pressure disparities were generally observed in a younger subset of the present study, appearing as a phase advance in the noninvasive data after a plateau in the central pressure waveform (e.g., Table 3). Invasive and noninvasive results were statistically similar.

Several issues should be noted. Despite some waveform discrepancies, absolute systolic and diastolic pressure values generally displayed excellent agreement across all age groups. This argues against significant pressure amplification effects, which can be observed at distal arterial sites. Such amplification is less common in older patients but can be enhanced during exercise. This latter potential limitation will require further study. Second, although pressure cuff techniques can overestimate systolic pressure in the presence of highly reduced vascular compressibility ("pseudohypertension"), this was rarely observed in the present study. This is intriguing, as many patients were beyond their 60th decile and had cardiovascular disease. It is possible that the use of the Doppler sensor as opposed to traditional audible frequencies to assess flow improved both the sensitivity and specificity of systolic and diastolic pressure estimation.

Some distinctions should be made between the new method and previously reported applanation tonometry techniques. The tonometer transducer provides a continuous high-frequency content waveform yielding beat-to-beat information over the full cardiac cycle. However, it is uncalibrated and requires careful steady positioning, because varying pressure on the transducer can markedly alter the waveform. The new method yields data only during the ascending pressure phase and is derived from many sequential cycles, taking between 15 and 30 seconds to obtain. This requires steady-state conditions, and substantial variability in

Figure 7. Graphs show comparison of invasive and noninvasive pressures before and after intravenous nitroglycerin (NG) (top panel) in one patient and isometric handgrip (bottom panel) in another. Invasive data are by micromanometer. The sets of pressures tracked each other well with both maneuvers.

Figure 8. Graph shows group data for handgrip and nitroglycerin intervention comparison. Systolic and diastolic pressures before and after each intervention are shown connected by lines. Paired data from all 13 patients are shown (eight with handgrip, five with nitroglycerin). Dashed line is the linear regression of all data and had a slope of 0.96, intercept of 3.6 mm Hg, and r of 0.98, similar to the line of identity.
heart rate from frequent ectopy, sinus arrhythmia, or respiratory effects will adversely affect the data. Given the duration for acquisition, high-frequency fluctuations are less likely to be faithfully recorded, which limits its use for waveform analysis. Its major strength, however, is that it provides non-user-dependent calibrated pressures that are quite accurate for systolic and diastolic values and that reasonably approximate the initial rising phase of the waveform. For the specific application of assessing left ventricular power, these aspects are important. The new method may also be useful in combination with tonometer data by providing calibration pressures.

Application to Noninvasive Power Assessment

Ventricular power has been previously proposed as a systolic function measurement by several groups. Until recently, however, the loading sensitivity and potential for noninvasive assessment was unclear. We recently reported that PWR_max can be accurately determined from the central aortic pressure–flow product and, when adjusted for chamber volume, yields a fairly load-insensitive contractile function index. The major advantage of this index over existing ones derives from its potential for noninvasive assessment from steady-state data. Because the major goal of the present study was to evaluate the noninvasive pressure measurements, we did not examine a fully noninvasive power estimate (i.e., validating noninvasive flow as well). The principal test was whether the noninvasive arterial pressures could substitute for micromanometer ventricular pressure and still accurately estimate PWR_max. The results showed excellent agreement.

PWR_max is an early ejection index, occurring within the initial 25–30% of ejected volume. As a result, only the pressure upstroke of the arterial wave is needed.
Moreover, deviation of noninvasive pressures from true central aortic pressure, such as in Figure 5, will have little influence on $PWR_{mx}$ determination, because this occurs shortly after flow deceleration, when reflected waves first return to the central aorta.\(^8\) As noted above, even patients with waveform disparities late in the pressure upstroke demonstrated agreement up to the inflection point. Thus, while posing a limitation for pressure waveform analysis, such discrepancies are less relevant to $PWR_{mx}$ determination. This is further supported by the data shown in Figures 10A and 10B, which include varying mean pressures, pulse width, heart rate, and other factors that can alter the arterial waveform. Despite this, the correlation between $PWR_{mx}$ values was excellent.

**Limitations**

Several limitations to this study should be noted. Although the comparisons between noninvasive and invasive central aortic pressures were generally good, the data do reflect a selective subset of all potential human subjects. Specifically, study patients had been referred for cardiac catheterization, many but not all had coronary disease, and 70% were older than 50 years. Therefore, one should be cautious in extrapolating these data, particularly to very young patients with no cardiac or peripheral vascular disease. Also, the present data were obtained with patients in a recumbent position at rest, and results from standing or upright positions or during aerobic exercise could differ. Future studies comparing central, brachial, and carotid pressures at rest and during exercise should help address these issues.

**Conclusion**

We have presented a new method for determining a calibrated ascending portion of the central arterial waveform. The results show excellent agreement for systolic and diastolic pressures and often very good agreement for the waveform itself, particularly in middle-aged adult patients with cardiovascular disease. Furthermore, these pressures can be used to accurately assess maximal ventricular power in humans. The new methodology is very promising for studies in which noninvasive quantitation of blood pressure is desired. By using a Doppler sensor to discern the onset of brachial artery flow, pressure assessment may be more reliable in patients in whom transmission of audible frequencies is limited (i.e., obese or hypertensive patients). Combining these pressures with one of several techniques for noninvasive flow measurement (e.g., Doppler or nuclear ventriculography) should enable accurate entirely noninvasive determination of maximal ventricular power\(^22,23\) in humans. In animals, $PWR_{mx}$ in conjunction with chamber volume data provides an index of ventricular contractile performance with minimal load dependence.\(^8\) Ongoing studies in humans with normal and diseased ventricles should define the clinical utility of a $PWR_{mx}$ index.

**References**

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