

Wave Intensity Analysis: a Novel Non-Invasive Method for Determining Arterial Wave Transmission

A Zambanini, AW Khir, S M Byrd, K H Parker, S A McG Thom, A D Hughes

Imperial College, London, UK

Abstract

Wave intensity analysis is a novel technique for assessing wavelet transmission in the cardiovascular system. Using this tool, we have developed non-invasive techniques to study wave transmission in both central & peripheral arteries in man. The aim of this study was to determine the reproducibility of various haemodynamic measures in the carotid, brachial and radial arteries.

12 treated hypertensive men underwent applanation tonometry and pulsed Doppler ultrasound studies of the carotid, brachial and radial arteries on 2 occasions. Coefficients of variation for the local wave speed, cardiac compression wave intensity and main reflected wave intensity ranged between 3.7-6.6%, 8.2-11.4% and 12.5-19.6% respectively.

We conclude that non-invasive methods used for wave intensity analysis are reproducible & provide additional information regarding the complex phenomenon of arterial wave transmission in man.

1. Introduction

The maintenance of blood flow and pressure gradients in the arterial circulation is mediated through complex haemodynamic interactions resulting from energy transfer between the heart and arteries [1]. The heart functions primarily to transfer hydraulic energy to organs and tissues, providing the energy in the form of individual forward travelling waves which initiate flow and maintain pressure in the arteries [2]. Sites of impedance mismatch, such as bifurcations and high resistance vessels, result in reflection of some wave energy back towards the heart. This leads to interactions between forward and backward traveling waves resulting in complex patterns of blood flow and pressure augmentation at different points in the circulation. Analysis of forward and backward traveling wave intensity or energy can provide important information regarding dynamic cardiac and arterial function [3,4].

The study of arterial waves has classically been performed using vascular impedance theory, which assumes that there is a linear relationship between pressure and flow in a one-dimensional system at steady

state [5]. An alternative approach is to utilize the method of characteristics, which assumes neither periodicity nor linearity but does assume waves to be one-dimensional in character [6]. This has resulted in the development of wave intensity analysis (WIA) as a novel mathematical tool to describe arterial wave power. Wave intensity, defined as power per unit area, can be calculated from changes in pressure and flow velocity and local wave speed can also be determined. Furthermore, WIA allows the separation of forward and backward traveling waves and, by studying concomitant changes in pressure, these can be further classified as compression or expansion wavelets [7].

To date, most of the published studies using WIA to study arterial waves, have acquired simultaneous pressure and flow data using invasive methods [3,4,6-8], focusing on cardiac or central aortic haemodynamics rather than studying waves in peripheral arteries. However, with the advent of applanation tonometry and pulsed wave Doppler, it is now possible to measure both arterial pressure [2,9,10] and flow velocity [2,11] non-invasively & reproducibly in superficial peripheral arteries. The purpose of this paper is to describe a novel non-invasive method utilizing WIA to study wave intensity in the common carotid, brachial and radial arteries in man.

2. Methods

2.1. Participants

This study was approved by the St. Mary's Hospital Research Ethics Committee and all subjects who participated in this study gave written informed consent. All subjects were part of the Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT) and were receiving antihypertensive treatment [12].

2.2. Study procedures

Subjects were examined while lying supine on a couch in a quiet darkened room. Room temperature was controlled at 23-25 °C and all subjects were rested for 10 minutes before any measurements were recorded. Brachial artery blood pressure was measured in the left arm using a validated semi-automated blood pressure monitor (Omron HEM-705CP, Hoofddorp, The

Netherlands) [13] and an appropriately sized arm cuff. At least three blood pressure measurements were recorded over the course of the study and these were used to calculate average systolic (SBP) and diastolic (DBP) blood pressure. Mean arterial blood pressure (MAP) was calculated as: $MAP = DBP + (SBP-DBP)/3$ [14].

2.2.1. Applanation tonometry

Applanation tonometry, which was originally developed for the measurement of intra-ocular pressure, can also be used to record peripheral arterial pressure waveforms using a high fidelity strain-gauge tipped pencil probe [15]. During applanation, the superior wall of the artery is flattened by the probe tip; the force acting on the transducer at this point represents intra-arterial pressure [15].

The subject's head rested on a single pillow with the neck slightly extended to allow adequate palpation of the right carotid artery. In the case of the brachial and radial arteries, the right elbow and wrist were supported in an extended position using a pillow. The area of maximum pulsation was identified and the tonometer (SPT 301, Millar instruments Inc., Houston, USA) was then applied to this site. The signal was amplified (TCB-500 pre-amplifier, Millar Instruments Inc.) and digitized at 200Hz using a personal computer (PC) and specialized software (DasyLab, Dasytec, Mönchengladbach, Germany). A concomitant single lead electrocardiogram (ECG) trace was also acquired. Simultaneous display of the pressure waveform and ECG trace to the PC monitor allowed the operator to determine whether these were acceptable. Criteria used to determine acceptability included the presence of a stable baseline (taking into account respiratory variation) and ensuring that the pressure waveform was of maximum amplitude. Once these criteria were met, approximately 30 to 40 seconds of data were saved to the PC hard disc.

2.2.2. Flow velocity measurement

Once the pressure waveforms had been acquired, the right common carotid (CCA), brachial and radial arteries, were imaged using a 7.5-10 MHz linear array transducer and HDI5000 ultrasound machine (ATL-Philips, Bothell, USA). The subject's head and right arm remained in the same position as that used for tonometry. After identifying the carotid bulb and bifurcation using B-mode imaging, a 1 mm sample volume was placed in the center of the CCA lumen at a distance of 2 cm proximal to the bulb. The brachial artery was identified immediately before the bifurcation into the radial and ulnar arteries and a 1 mm sample volume placed in the centre of the distal 1-2cm of the brachial artery. The optimal site for acquisition of velocity data from the radial artery was identified using 2 criteria. The first was that adequate filling of the artery occurred using color Doppler and the second was that a

1mm sample volume could be placed in the lumen of the artery such that the length of the sample volume lay parallel to the walls of the artery. At all sites, pulsed wave Doppler ultrasound at a frequency of 6MHz was used to insonate the arterial lumen whilst ensuring that the ultrasound beam was positioned at 60° incident to the direction of central luminal blood flow [16].

The pulsed Doppler velocity waveform and simultaneous single lead ECG trace were acquired continuously for 20 cardiac cycles at a frequency of 200Hz and then recorded digitally to a PC using specialist software (HDIlab, ATL). Within HDIlab, it was possible to determine the peak Doppler velocities which were saved for further analysis.

2.3. Reproducibility studies

All subjects underwent the two sets of measurements at all 3 sites separated by an interval of 10 minutes during which they remained supine and undisturbed.

2.4. Ensemble averaging

Further processing of the pressure and flow velocity data occurred within the Matlab programming environment (MathWorks, Natick, USA). Six contiguous pressure waveforms were selected and then separated using the peak R wave of each ECG complex as a marker for the beginning and end of each cycle. The waveforms were ensemble averaged after each was aligned for the first 30-50 ms of the rapid rise in pressure. In order to achieve a single carotid pressure waveform (P), the ensemble averaged pressure data were calibrated according to the force required to achieve satisfactory applanation and MAP using a previously described algorithm [17]. Brachial and radial pressure waveforms were calibrated to the measured brachial SBP and DBP.

Peak flow velocity waveforms were also modified using an algorithm tested in a string phantom (unpublished data) in order to correct for the overestimations of peak velocity, acceleration and deceleration that may occur with some ultrasound systems. Six contiguous modified flow velocity waveforms were selected and then separated using the peak R wave of each ECG complex as a marker for the beginning and end of each cycle. The waveforms were ensemble averaged after each was aligned such that the first 30-50 ms of the rapid rise in flow velocity lay at the same point in time. A single ensemble averaged flow velocity waveform was generated (U).

2.5. Wave intensity analysis

The mathematical methods of WIA have been well described [6,18]. The change in pressure (dP) and flow velocity (dU) in each sampling period were used to calculate net wave intensity (dI). In order to determine the forward and backward wave intensities, local wave

speed was calculated using a pressure-flow velocity loop [19].

Compression waves were defined as occurring during positive changes in pressure and expansion waves during negative changes.

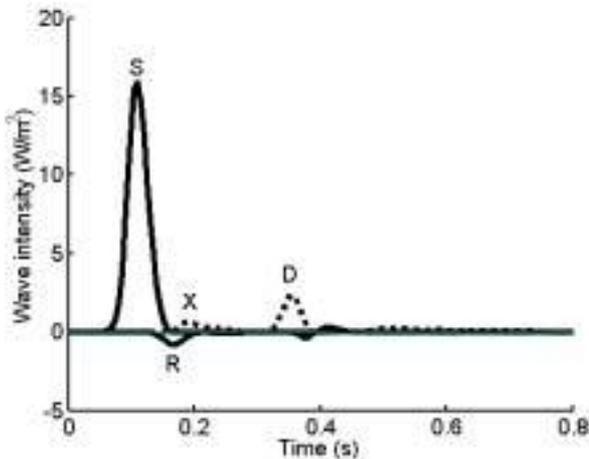


Figure 1: Wave intensities at the right CCA. Forward travelling waves have positive values while backward travelling waves are negative. Compression waves are in bold lines and expansion waves are in dotted lines.

2.6. Statistics

The main forward and backward wave intensities were identified as shown in Figure 1. Wave speed (c) and the intensity of the main cardiac compression wave (S), the reflected compression wave (R), the mid-systolic (X) and end-systolic expansion waves (D) were calculated. Reproducibility for these parameters between the 2 studies was expressed using the mean difference (\pm SD) between the studies and coefficients of variation.

3. Results

All data are expressed as mean (\pm SD). 12 treated male hypertensive subjects, mean age 63 (\pm 9) years, participated in the study. Over an average of 16 (\pm 3) months, 7 men had taken a combination of atenolol / bendroflumethiazide while the other 5 had taken amlodipine / perindopril. Average brachial blood pressure was 128 (\pm 12) / 82 (\pm 8) mmHg.

3.1. Reproducibility results

The mean values for wave speed and intensities are shown in Table 1. Mean differences for each parameter at each of the 3 sites are shown in Table 2. Coefficients of variation for each parameter at each of the 3 sites were as follows: CCA { $c = 6.6\%$, $S = 11.4\%$, $R = 19.6\%$, $X = 31.6\%$ and $D = 11.9\%$ }, Brachial { $c = 3.7\%$, $S = 8.2\%$, $R = 12.5\%$, $X = 15.9\%$ and $D = 9.1\%$ }, Radial { $c = 4.5\%$, $S = 10.7\%$, $R = 15.5\%$, $X = 26.0\%$ and $D = 19.4\%$ }.

	CCA	Brachial	Radial
c	7.5 (2.4)	9.3 (2.3)	11.6 (5.0)
S	13.78 (7.27)	19.67 (10.19)	13.29 (6.02)
R	1.39 (0.80)	2.48 (1.10)	3.65 (2.33)
X	0.46 (0.36)	2.11 (1.49)	1.91 (1.41)
D	2.85 (1.30)	2.14 (1.02)	1.29 (0.63)

Table 1: Mean values for wave speed and wave intensities at 3 arteries. Data expressed as mean (\pm SD), units are m/s for wave speed and W/m^2 for wave intensities.

	CCA	Brachial	Radial
c	0.10 (0.72)	-0.12 (0.51)	-0.28 (0.66)
S	1.59 (1.93)	0.12 (2.85)	-0.70 (2.38)
R	0.04 (0.40)	0.11 (0.56)	-0.32 (0.73)
X	0.12 (0.12)	0.08 (0.54)	-0.29 (0.85)
D	0.18 (0.75)	0.07 (0.45)	-0.13 (0.42)

Table 2: Mean differences for wave speed and wave intensities for 2 studies performed at 3 arteries. Data expressed as mean difference (\pm SD), units are m/s for wave speed and W/m^2 for wave intensities.

4. Conclusions

Previous work in the human ascending aorta in patients of similar age to our study [8], showed a pattern of wave intensities resembling those we obtained in the CCA. Additionally, the magnitude of the S and R waves in the aorta and CCA were similar. We can be confident therefore, that our non-invasive methods provide reasonable estimates of wave intensity.

CCA wave speed was lower than that in the muscular brachial and radial arteries. Brachial and radial artery compliance is largely unaffected in hypertensive patients who have stiff carotid arteries [20]. Despite having hypertension and multiple risk factors for coronary heart disease, the patients we studied had a 'normal' pattern of arterial stiffness. This may be explained by the duration of blood pressure control and treatment.

Although the pattern of wave intensities was similar in the CCA, brachial and radial arteries, the magnitude of both wave speed and intensities differed between the arteries. These results are similar to those from our previous work [21] that suggested that the mid-systolic expansion wave (X) was larger in magnitude in the muscular radial and brachial arteries compared with elastic CCA.

The X wave probably represents a re-reflection phenomenon. A putative mechanism is that the X wave, seen at the radial artery, is generated following proximal reflection of the R wave at the brachial bifurcation. Although R is a compression wave, the cross-sectional area of the brachial artery at the bifurcation is greater than that of the radial artery, and the net result of

reflection is the generation of an expansion wave travelling distally down the radial artery. The effect of the X wave is to reduce pressure and flow during mid-systole. This is important as the D wave, which is cardiac generated, appears to be attenuated as it travels distally into the muscular arteries.

A reflection coefficient can be determined by measuring the amount of reflection of the S wave from downstream sites using the ratio of R:S wave intensities. In our study, the reflection coefficient differed between the 3 sites and at the radial artery, the coefficient had a value approximately twice that at the CCA. These data suggest that wave transmission is non-uniform through the arterial system and that important differences between elastic and muscular arteries exist.

We have described the measurement reproducibility of wave speed and intensities in treated hypertensive subjects. Our results suggest that there was little variability for the largest waves, in the measures of wave speed or intensity. However, the X wave had the highest coefficients of variation perhaps due to the greater variability of flow velocity during mid-systole. Additionally, the X wave was typically the smallest wave present and small differences in the magnitude of the wave intensity are likely to have resulted in large coefficients of variation.

Wave intensity analysis using non-invasively acquired data is an easily applicable and reproducible method for clinical studies. Future work may provide additional information regarding ventriculoarterial interactions in both health and disease.

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Address for correspondence:

Dr. Andrew Zambanini
 Department of Clinical Pharmacology,
 10th Floor QEOM,
 St. Mary's Hospital,
 London, W2 1NY,
 United Kingdom

a.zambanini@ic.ac.uk