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RESEARCH ARTICLE

A New Approach to Measure Heart-Carotid Pulse Wave Velocity by Using an Ultrasound-Based Method

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Abstract: ***Introduction*:** Currently, regional or global assessments of vessel elasticity are performed by measuring pulse wave velocity (PWV) along a long arterial segment. However, this method of evaluating arterial stiffness is subject to bias due to several factors, including the difficulty of accurately measuring the arterial distance.

The aim of this research was: (a) to develop a non-invasive method for calculating pulse wave velocity in the heart–carotid pathway (hc-PWV) using electrocardiographic and ultrasonic images; (b) to measure hc-PWV in an adult hypertensive population using this new method and compare the results with values reported in the specialized literature; and (c) to perform a correlation analysis between hc-PWV and heart–femoral pulse wave velocity (hf-PWV) in a population of adult hypertensive subjects.

***Material and Methods*:** In this study, PWV was calculated using an image analysis technique developed in our laboratory. As an original technique, the theoretical background is first described, followed by its application in hypertensive volunteers. For each subject in the analysed population, the hc-PWV was calculated using the new technique, and the hc-PWV was measured using mechanotransducers.

***Results*:** in the analysed cohort of hypertensive patients, values of hc-PWV (8.57 ± 0.51 m/s) were similar to those obtained in the carotid-femoral pathway (8.57 ± 0.51 m/s versus 8.19 ± 1.27 m/s; PNS). Moreover, hc-PWV in our cohort of treated hypertensive patients (8.57 ± 0.51 m/s) was higher than that reported by other authors for healthy subjects in similar territories (4.9 ± 1.1 to 8.12 ± 3.54 m/s). Furthermore, the hc-PWV values in our cohort of treated hypertensive patients (8.57 ± 0.51 m/s) were lower than those reported in older subjects with systemic hypertension (11.56 ± 1.74 m/s). A significant correlation was observed between the hc-PWV and cf-PWV ($r=0.73$, $p <0.05$). A regression analysis was performed, yielding a slope of 0.2903.

***Discussion*:** This work showed a novel approach to measuring heart–carotid pulse wave velocity using ultrasound-based methods. In this initial exploration, our aim was not to evaluate or confirm a categorical result, but rather to highlight a trend toward a new methodology for calculating arterial stiffness, contrasted against previously validated standard methods.

***Conclusion*:** This study confirmed that hc-PWV can be calculated non-invasively using electrocardiographic and ultrasonic images. The calculated hc-PWV values were in the range of those reported in the literature.

Keywords: Pulse wave velocity, regional stiffness, heart carotid PWV.

1. INTRODUCTION

The association between arterial stiffness and well-known cardiovascular risk factors, such as age, dyslipidemia, hypertension, and diabetes mellitus, has been widely reported in extensive screening analyses and clinical settings [1, 2]. Measurement of arterial pulse wave velocity (PWV) and pulse wave analysis quantify vascular stiffness and provide prognostic information related to cardiovascular risk [3]. Carotid-femoral pulse wave velocity (cf-PWV) is considered an independent predictor of cardiovascular events in patients [4]. Moreover, longitudinal follow-up has shown that aortic PWV

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is a significant cardiovascular risk factor [5] and has been associated with all-cause mortality in a population with Type I Diabetes Mellitus [6].

As demonstrated, the structural constituents of the vascular wall are associated with variations in the viscoelastic properties of the arterial wall in both physiological and pathological conditions [7]. Altered extracellular matrix, involving collagen and elastin, has been identified as a factor in the development of preatherogenic damage [8]. In other words, the increase in stiffening is a consequence of age- and/or systemic disease-induced changes in the extracellular matrix. This increase in vascular rigidity seems to be more important in specific territories. For example, carotid stiffness is independently associated with incident cardiovascular morbidity and mortality [9].

Currently, a regional or global assessment of vessel elasticity is obtained by measuring PWV in a long arterial segment, allowing the detection of speed increases that correspond to lower vessel distensibility and, therefore, to higher vascular stiffness [10]. However, this modality for evaluating arterial stiffness is subject to bias influenced by several factors.

First, the distance between the carotid and femoral arteries cannot be measured [1, 11],

Second, short arterial stiffness cannot be measured using traditional methods [10],

Third, the arterial structures involved in the stiffness assessment are not uniform, and peripheral muscular vessels are stiffer than the elastic central arteries [12].

The most important bias in PWV calculation is the method used to measure the distance, a variable estimated from surface measurements that is only an approximation of the true value. As inaccuracies in distance measurements may cause significant errors in pulse wave calculation [5], new techniques have been developed to estimate the carotid-femoral length without direct surface measurements [13].

We consider these observations by the reviewer to be excellent, and they have been incorporated into the Introduction section of our manuscript to reflect and acknowledge the reviewer's valuable contribution.

Considering the above-mentioned reports, two factors must be resolved to obtain reliable measurements of arterial stiffness in clinical practice: the measurement of aortic path length along the carotid-femoral pathway and the elastic characterization of the carotid arteries [9, 13].

Therefore, a regional measurement technique for carotid–femoral pulse wave velocity could offer several advantages. First, it could avoid the challenge of estimating the carotid–femoral distance, where the aortic arch represents a significant source of error. Second, this technique could be independent of the measurement site, suggesting a potential independence from arterial geometry. Third, it could hold clinical relevance as a potential early indicator of vascular stiffness, central hemodynamics, or cardiovascular risk.

Thus, the aim of this research was:

a. To develop a non-invasive method to calculate the pulse wave velocity in the heart–carotid pathway using electrocardiographic and ultrasonic techniques.

b. To measure hc-PWV in an adult hypertensive population using a new non-invasive method and compare with values available in the specialized literature.

c. Correlation analysis between hc-PWV and cf-PWV in healthy subjects.

2. MATERIAL AND METHODS

In this research, pulse wave velocity was calculated using an image analysis technique developed in our laboratory. As an original technique, it was first described and applied in hypertensive volunteers. For each subject in the analysed population, the hc-PWV was calculated using the new technique, and the carotid-femoral pulse wave velocity was measured using mechanotransducers.

Heart carotid PWV calculated by Doppler image analysis - theoretical Background

The method described in this section aims to calculate regional pulse wave velocity noninvasively, without requiring any distance measurements on the body surface.

A Sonosite-Turbo Echograph, configured for a 75 mm/s sweep, enables the storage of a 12 sec video (AVI format) at 10 photographs per second. The stored data include images of the vessel, the electrocardiographic signal, and vascular Doppler flow, as shown in Fig. (**1**).

2.1. Image Sequence Processing

Sonosite-Turbo ultrasound system enables the acquisition of dynamic B-mode ultrasound images that include the arterial segment under study, with an overlaid ECG signal in the same image sequence. During post-processing, the QRS complex marks the onset of the systolic phase of the cardiac cycle, which should correspond to the beginning of systolic flow in the arterial signal. However, due to the finite speed of propagation of the pressure wave, there is a temporal delay between the QRS complex and the onset of systolic flow. If the spatial distance between the QRS complex and the base of the systolic flow is known—this can be determined based on the sweep speed of the ultrasound system—then dividing this distance by the time delay between the two signals yields the pulse wave velocity.

Once the Doppler flow image sequences and the patient's ECG signal were available, the transit time between the ECG R-peak and the blood flow was measured. To this end, the segmented images were converted into one-dimensional ECG and flow signals, which were then processed using temporal and/or frequency analysis to calculate the transit time. The reason for acquiring different heartbeats using a conventional method is that we can measure the transit times in each heartbeat, along with the corrected distance between the heart and the arterial point where we acquire the images. In this way,

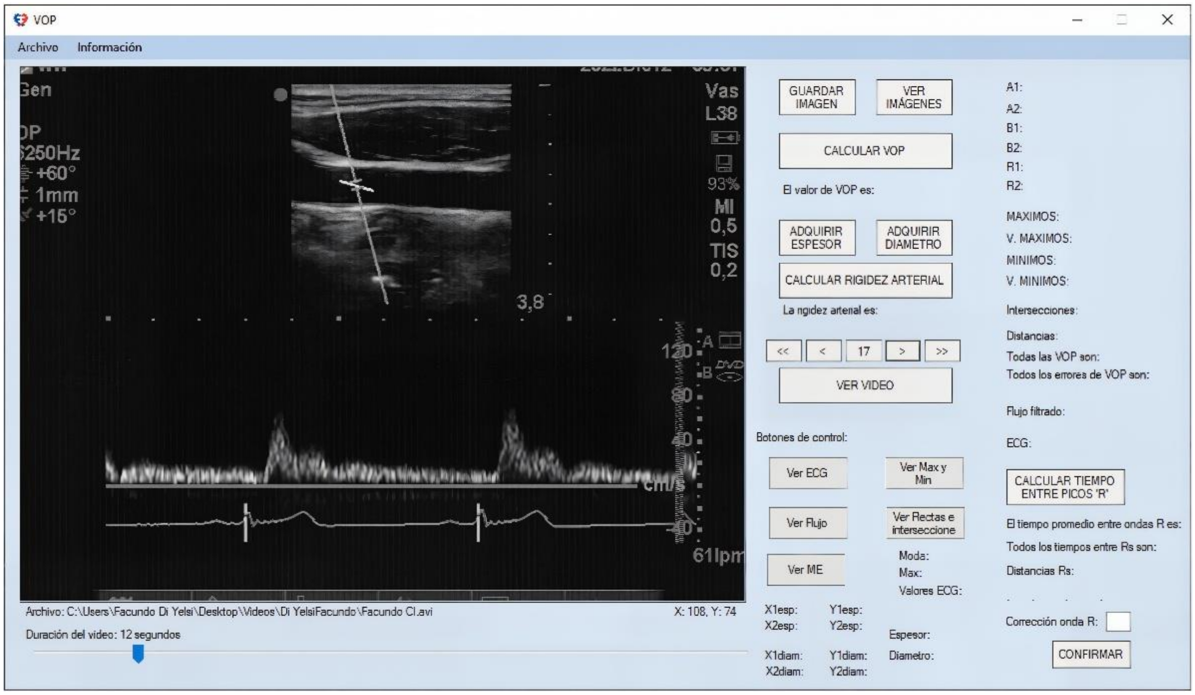

Fig. (1). Algorithm Screen for the time transit determination between arterial flow registration and ECG. (*A higher resolution / colour version of this figure is available in the electronic copy of the article*).

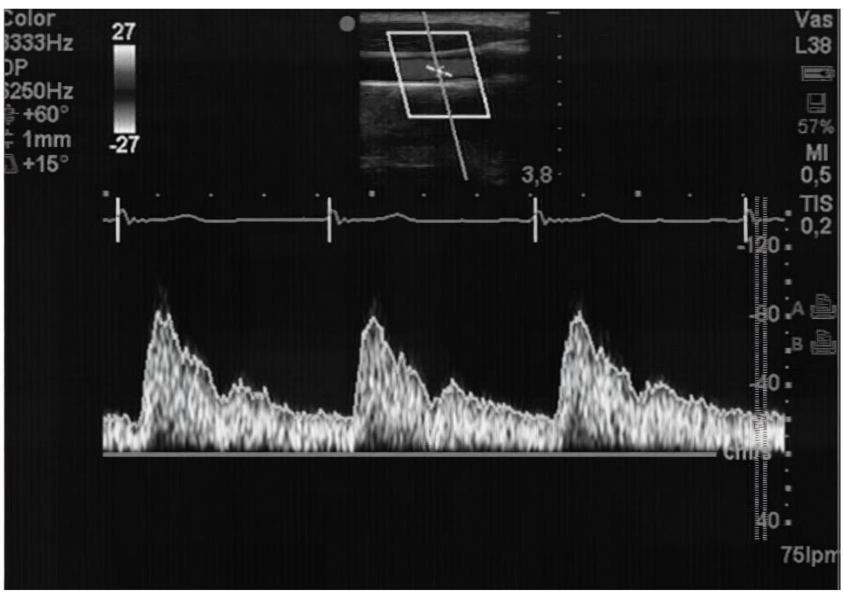

Fig. (2). ECG and Flow signals segmentation. (*A higher resolution / colour version of this figure is available in the electronic copy of the article*).

the conventional PWV can also be measured, and comparisons can be performed, as shown in Equation 1:

$$\text{VOP} = \text{mean} \pm \text{standard deviation} \quad (1)$$

Thus, variations in the unique PWV determination can be avoided.

It must be noted that the echographic Doppler sweeping speed must always be the same for all patients studied together with the temporal image resolution, that is, the image pixel distance. This is because, during the analysis, we work with samples that must be correlated with the Doppler temporal resolution.

2.2. Processing and Analysis of the Image Sequences

In Fig. (**2**), a segmentation (separation) of the arterial flow and electrocardiogram (ECG) signals can be observed. Once segmented, the curves must be tracked to identify the onset of systole in the flow signal and the negative peak of the S wave in the ECG signal.

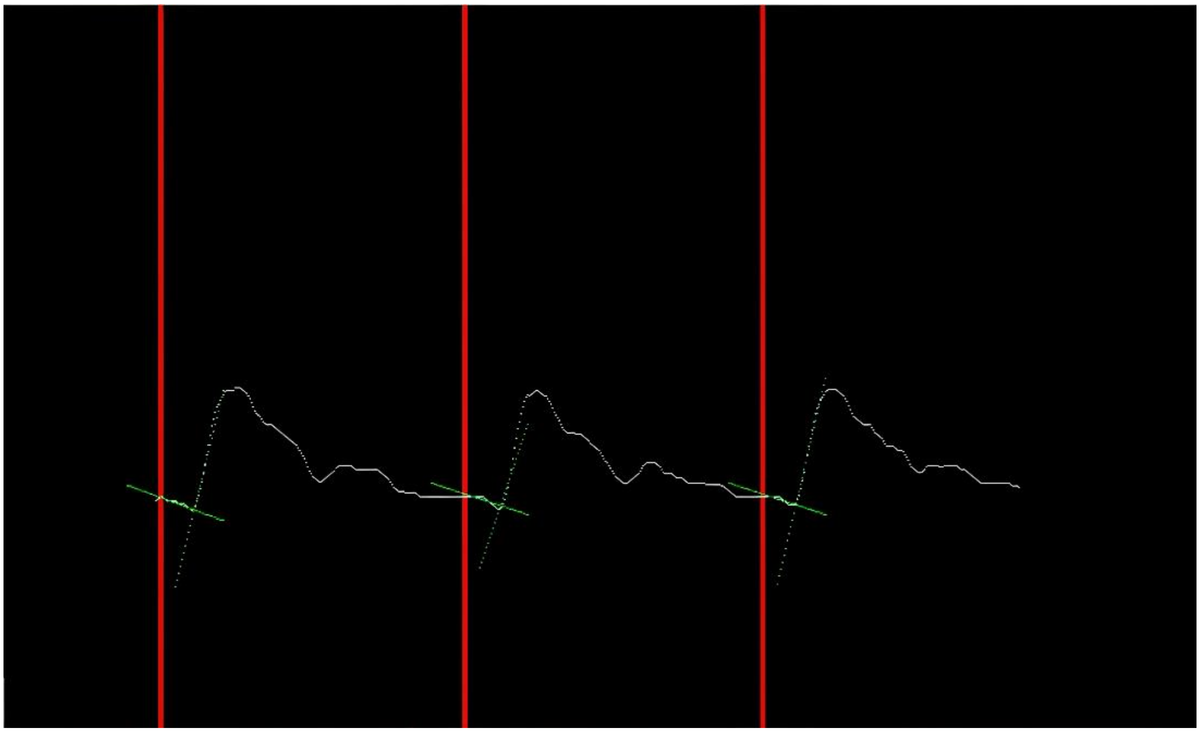

Fig. (3). ECG R peak and systolic start in the arterial flow register. (*A higher resolution / colour version of this figure is available in the electronic copy of the article*).

After completing segmentation of both the ECG and arterial flow signals, the next step is to perform linear interpolation of the arterial flow. This interpolation allows the construction of two lines by providing their parameters (slope and y-intercept), whose intersection is useful for determining the onset of systole.

Fig. (**3**) presents a unified representation in which vertical red lines indicate the positions of the R wave peaks in the ECG. Additionally, the flow signal contour is shown in white, and the resulting lines from the linear interpolation are displayed in green and blue.

Once the segmentation process was completed, the curves were followed to determine the systolic start of the flow signal and the negative ECG S-wave. The next step was to perform linear interpolation of the arterial flow. This yields two straight lines (the slope and ordinate of the origin. The intersection point represents the initial systolic point.

Once the coordinates corresponding to the onset of the systolic phase of the flow and the negative peak of the S wave in the ECG signal (in pixels) have been determined, their difference is computed as indicated in Equation 2

$$\tau_e = x_f - x_{ECG} \quad (2)$$

where τ_e is the spatial displacement (measured in pixels), x_f is the initial systole abscissa in the flow, and x_{ECG} is the ECG S-wave minimum abscissa.

Considering that the video signal from the ultrasound device has a specific sweep velocity (measured in meters per second or millimeters per second, depending on the manufacturer), and that the pixel distance in the ultrasound image corresponds to a certain time interval (one second, for example), we express the shape factor (F_S) as shown in Equation 3:

$$F_S = V_{sw} / pix_D \quad (3)$$

where V_{SW} is the sweep velocity (in meters per second), and pix_D is the pixel distance in the eco image (number of pixels in the image corresponding to one second).

Finally, using the interval (in pixels) from Equation 2 and the shape factor from Equation 3 (in meters per second per pixel), the pulse wave velocity (in meters per second) is obtained as expressed in Equation 4:

$$VOP = \tau_e \cdot F_S \quad (4)$$

It is extremely important to consider that this procedure must be performed for each registered heartbeat. Thus, the PWV value is the mean of the estimated velocities for each heartbeat, as shown in Equation 1.

2.3. Study Population

Based on inclusion and exclusion criteria, we defined an initial population of 16 participants (age range: 19–37 years; 60% male, 40% female). In all subjects included in this research pulse wave velocity measurements were carried out: a) employing a validated technique routinely used by our team using mechanotransducers which is described following, and b) using a new procedure that include the analysis of blood flow waves obtained with Doppler technique as is described above under the title "Heart carotid PWV calculated by Doppler image analysis - theoretical Background."

The analyzed cohort included asymptomatic ambulatory patients treated with antihypertensive therapy. All volunteers underwent a clinical interview as part of a non-invasive evaluation of atherosclerosis. Blood samples were obtained after 10–12 hours of fasting. The lipid profile, glycemia, and kidney functional parameters were determined for each volunteer. Anthropometric evaluation and clinical interviews allowed the assessment of exposure to cardiovascular risk factors.

In this study, the inclusion criteria were as follows: (1) arterial blood pressure at the time of examination ≤ 140/90 mm Hg; (2) glycaemia < 110 mg/dL; (3) total blood cholesterol levels < 200 mg/dL; and (4) normal serum triglyceride levels, defined as < 150 mg/dL. The same physician performed anthropometric assessment and blood sampling.

Blood pressure measurements were performed using an automatic sphygmomanometer (705IT; Omron Healthcare Inc.).

Antihypertensive therapy was successful in most patients (12 individuals). However, it was not effective in four patients, who were therefore excluded from the present study (congenital heart disease; obesity; obesity, hypertension, and smoker; sedentary and smoker).

2.4. Pulse Wave Velocity Measured by Mechanotransducers

All measurements were performed with the patient in a supine position in a quiet room with a stable temperature after 10 min of rest. PWV was measured in the carotid-femoral pathway using a technique previously developed by our team [14],[15] with a dedicated device (Arteriometer, Model V100; Oxitech, Buenos Aires, Argentina). This methodology includes the use of two high-fidelity silicon piezoresistive pressure sensors (Motorola MPX 2050, Motorola Inc., Corporate 1303 E. Algonquin Road, Schaumburg, Illinois 60196, USA) connected to an amplifier, which allows arterial pulse waves to be obtained. During data acquisition, both sensors were applied simultaneously at two sites along the same vascular pathway: the left carotid and femoral arteries.

Arterial pressure signals were acquired on a computer using specific software manufactured in our laboratory, which allows the calculation of the time delay between the two instantaneous pulse waves. This software runs on Windows and provides online digitized pressure-wave acquisition, including at least 10 cardiac cycles per continuous record. All records were continuously recorded and visualized on a computer screen. In the analyzed population, the same two physicians, one always using the pressure sensors and the other operating the computer, carried out the data acquisitions for all subjects included in this research. The operators monitored the pulse wave quality online, and the pressure signal acquisitions were repeated if necessary. Once the pressure signals were recorded, the software calculated the PWV online using manual measurement of the distance between sensors. In this study, the sensors were positioned in the (a) left carotid arteries and (b) left femoral arteries for cf-PWV evaluation.

For each subject, the mean and standard deviation of these pulse wave velocity measurements were calculated. The obtained mean value was considered the pulse wave velocity for each patient, and the standard deviation was used to ensure reliable measurement. If the measurement standard deviation exceeded 10 %, data acquisition was repeated.

After the pulse wave velocity calculation, the obtained value was corrected by multiplying by 0.8, according to international recommendations [16, 17].

2.5. Data Analysis

Once hc-PWV values were obtained, a comparative study was performed using data obtained from specialized literature (unpaired t-test). In the second step, a correlation study was conducted using cf-PWV values obtained with mechanotransducers and hc-PWV values calculated from Doppler image analysis [18], in accordance with previously reported international recommendations [19].

For all statistical analyses, $p < 0.05$ was considered the limit of statistical significance. Statistical analysis was performed using IBM-SPSS software (version 20.0).

2.6. Ethics Approval

This research was carried out in accordance with the ethical principles of the Declaration of Helsinki, the US Code of Federal Regulations (Part 46, Protection of Human Subjects), and the International Conference on Harmonization Guidelines for Good Clinical Practice. The study protocol was revised and approved by the Institutional Committee for Human Investigations of the Cardiology and Cardiovascular Surgery Institute of the Favaloro Foundation. The approval resolution was identified as DDI (1351) 2616 CBE 630/16. Written informed consent was obtained from each patient included in this study before enrollment.

3. RESULTS

In the analyzed cohort (n=16), four volunteers were excluded due to technical errors during data acquisition. hc-PWV values were obtained using the new method without any surface measurements of the heart-carotid distance. The values of hc-PWV obtained in this cohort of hypertensive patients (8.57 ± 0.51 m/s) were similar to those obtained in another elastic territory (carotid-femoral). As is shown in Table **1**, no statistical differences were observed in terms of PWV in the analysed elastic arteries segments: heart-carotid and carotid-femoral (8.57 ± 051 m/s versus 8.19 ± 1.27 m/s; PNS).

The hc-PWV values in our cohort of treated hypertensive patients (8.57 ± 0.51 m/s) were higher than those reported for healthy subjects in similar territories (4.9 ± 1.1 to 8.12 ± 3.54 m/s), as shown in Table **2** [20-24].

Furthermore, the hc-PWV values in our cohort of treated hypertensive patients (8.57 ± 0.51 m/s) were lower than those reported by Kawai and co-workers [20] in older subjects with systemic hypertension (11.56 ± 1.74 m/s). On the contrary,

Table 1. Regional Pulse Wave Velocity.

Patient #	cf-PWV	hc-PWVr
1	7.69	8.15
2	7.31	8.75
3	8.27	8.74
4	6.68	7.76
5	10.67	9.10
6	7.95	8.48
7	8.20	8.41
8	10.67	9.31
9	7.85	7.88
10	8.56	9.33
13	7.20	8.56
16	7.28	8.40
Mean	8.19	8.57
SD	1.27	0.51

Note: Carotid-femoral and regional heart-carotid pulse wave velocity (cf-PWV and hc-PWVr, respectively) measured in meters/second. Patients #11, #12, #14 and #15 were discarded. Paired t-test shows no differences ($p > 0.05$).

Table 2. Regional pulse wave velocities found in our research compared to previous reports

	Method	n	Age y. o.	PWV		
Present study	Ultrasound analysis hc #	12	19 – 37	8.57	±	0.51
Kawai *et al.* 2014 [20]	Applanation tonometry hc #	338	59 – 63	11.56	±	1.74
Huang *et al.* 2016 [21]	Ultrasound imaging AAo o	76	23 – 71	6.51	±	1.90
Loose *et al.* 2023 [24]	Three-dimensional MRI AAo +AA ●	38	20 – 80	8.12	±	3.54
Loose *et al.* 2023 [24]	Three-dimensional MRI AAo +AA ♦	38	20 – 80	8.00	±	3.58
Vulliémoz *et al.* 2002 [23]	Double-oblique slice MRI AAo ●+♦	13	26 – 48	4.9	±	1.1

Note: AAo: Ascending Aorta. AA: Aortic Arch. PWV: Pulse Wave Velocity meters/second (m/s). hc: heartcarotid. # hypertensive patients. o: aortic disease. ●: healthy females. ♦: healthy males. Unpaired t – test.

our results were lower than those reported by Huang *et al.* [21], in hypertensive patients (6.51 ± 1.90 m/s).

3.1. Correlation analysis between hc-PWV and cf-PWV

Except in two subjects, the hc-PWV values were higher than the cf-PWV values obtained in the same volunteer, as shown in Fig. (**4**). This is a tendency toward arterial stiffness observed in both elastic aortic territories explored in this research. A significant correlation was observed between the hc-PWV and cfPWV ($r=0.73$, $p<0.05$). A regression analysis was carried out, and the calculated slope was 0.2903, showing that each obtained value of hc-PWV was higher than the cf-PWV found in each subject of the analysed population.

Following, a Bland & Altman analysis was carried out [18], showing that the mean differences (invasive minus non-invasive central aortic pressure values) were within the 95% confidence interval and exhibited a normal distribution, as shown in Fig. (**5**).

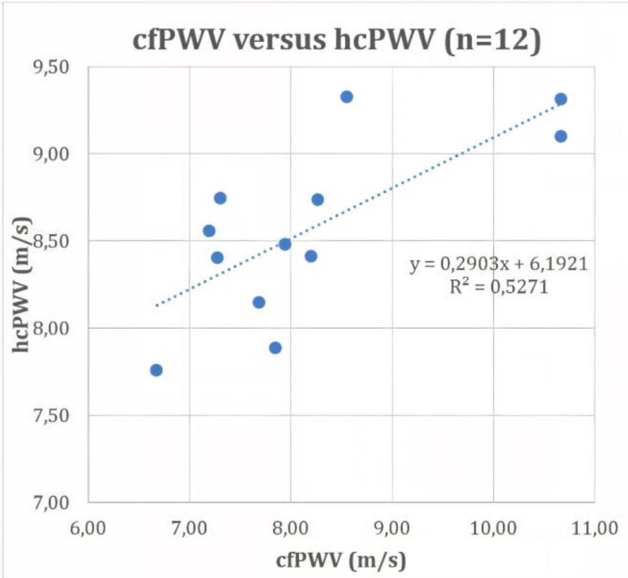

Fig. (4). Correlation plot in terms of pulse wave velocity in the carotid-femoral pathway (cf-PWV) versus heart-carotid pulse wave velocity (hc-PWV). Values are in meters per second (m/s). Slope = 0.2903. Correlation coefficient (R) was 0.726 (p<0.05). (*A higher resolution / colour version of this figure is available in the electronic copy of the article*).

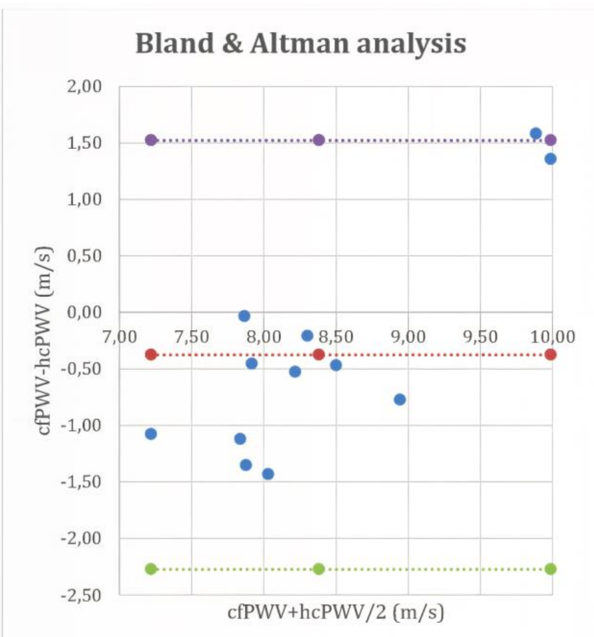

Fig. (5). Bland-Altman plot of differences between pulse wave velocities obtained in the carotid-femoral pathway (cf-PWV) and heart carotid pulse wave (hc-PWV). The mean differences for the entire population were within the 95% confidence interval. (*A higher resolution / colour version of this figure is available in the electronic copy of the article*).

4. DISCUSSION

In this study, a new technique to calculate the regional pulse wave velocity in elastic arteries was developed. As described above, the new method used well-known technologies, such as surface electrocardiogram and ultrasonic signals, to perform image analysis. In our laboratory, these techniques have been widely used over the last decade [15]. The proposed method for measuring regional pulse wave velocity does not require measurement of the distance on the body surface. As noted by many authors, the use of surface measurements in PWV calculations is a source of error that has persisted for decades; consequently, we developed a new method.

This work seeks a novel approach to measuring heart–carotid pulse wave velocity using ultrasound-based methods. In this initial exploration, our aim was not to evaluate or confirm a categorical result, but rather to highlight a trend toward a new methodology for calculating arterial stiffness, contrasted against previously validated standard methods.

The selection of the heart-carotid pathway as the first stage to explore the feasibility of the proposed method was because it is a territory made up of elastic arterial walls, which has already been examined by other authors. Measuring the distance between the left ventricle and carotid artery is not free from a certain degree of error, which is avoided by using the technique described here. In this investigation, two segments of the elastic arterial territory were compared (heart-carotid and carotid-femoral). Although they are not on the same path, similar comparisons have been made in the specialized literature on regional and local PWV. In fact, in a report by Simova et al., a comparative study was conducted between local PWV in the carotid arteries and PWV in the carotid-femoral pathway [22].

Measurement of the hc-PWV in this study showed a value of 8.57 ± 0.51 m/s; since the analyzed population included patients with hypertension, a certain degree of stiffening would be expected. Our population of hypertensive patients was younger than that studied by Kawai et al. [20], in which the hc-PWV value was 11.56 ± 1.74 m/s (higher than that obtained in our research). In healthy subjects, PWV values obtained by other authors in the ascending aorta [23] and the aortic arch [24] were lower than those calculated in younger hypertensive subjects, as shown in Table 2.

The correlation study between the hc-PWV values obtained with the proposed method and the cf-PWV values calculated using a validated technique showed a tendency that warrants further investigation, including a larger number of volunteers. See Figures 3 and 4.

In summary, our results indicate that by employing the imaging analysis proposed here, it is possible to evaluate hc-PWV, and the obtained values are in the range of those found in specialized literature in which similar populations were analyzed.

5. LIMITATIONS OF THIS STUDY

Because our attention was focused on developing a new method to calculate PWV without surface measurements, the number of subjects included in this study was limited. Moreover, the analyzed territory included only the pathway between the left ventricle and the carotid artery. Future studies that include a higher number of patients are necessary, and new arterial territories in which both the heart carotid and the heart femoral pathways should be included.

This new study involving a large patient cohort must be both reproducible and repeatable, for which the coefficient of variation is an excellent measure of variability in the results. However, this study aims to present a novel methodology for evaluating arterial stiffness. Therefore, our focus was on assessing the feasibility of the method rather than its repeatability and reproducibility. These aspects will be addressed in future studies with a larger sample size.

Moreover, the statistical power of our study could be enhanced by increasing the sample size, thereby reducing the rate of false positives and improving the reliability of comparisons. This improvement could be achieved not only by expanding the patient cohort but also by including a normotensive population to strengthen the methodology.

Finally, a potential source of error in our methodology may be attributed to a minimal temporal mismatch between the QRS complex and the onset of arterial systolic flow. Sonosite-Turbo can acquire dynamic B-mode images and images can indeed be used to synchronize vascular wall motion/flow with cardiac cycle events. The QRS marks ventricular depolarization, which leads to ventricular contraction and the onset of systole. However, the mechanical ejection (systolic flow onset) lags slightly behind the QRS because of an isovolumetric contraction time before the aortic valve opens. It is not the QRS itself, but rather the subsequent arterial upstroke, which is most closely aligned with pulse wave transmission. The finite velocity of the arterial pulse wave means there will be a delay between the central event (left ventricular ejection) and the peripheral detection of systolic upstroke, which is expected to be captured by pulse wave velocity (PWV). The “spatial distance between the QRS complex and the base of systolic flow” in the image is not the true anatomical distance between measurement sites. This implies the use of a correction factor close to 0.8 for the calculation of PWV, as indicated in reference [17]. However, future studies should investigate the appropriate correction factor, which we estimate to be greater than 0.95 due to the minimal millisecond delay associated with myocardial isovolumetric contraction.

CONCLUSION

This study demonstrates that it is possible to measure stiffness in the heart-carotid pathway by analyzing images generated with electrocardiographic and ultrasound signals. The values obtained are within the range reported in the literature. The elastic arterial pathway analyzed in this study showed that hc-PWV values obtained in the same hypertensive patients were correlated with cf-PWV measured in each patient using a validated technique.

AUTHORS’ CONTRIBUTIONS

It is hereby acknowledged that all authors have accepted responsibility for the manuscript's content and consented to its submission. They have meticulously reviewed all results and unanimously approved the final version of the manuscript.

LIST OF ABBREVIATIONS

PWV	=	Pulse Wave Velocity
hc-PWV	=	Heart–carotid Pathway
hf-PWV	=	Heart-femoral Pulse Wave Velocity
ECG	=	Electrocardiogram

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study protocol was revised and approved by the Institutional Committee for Human Investigations of the Cardiology and Cardiovascular Surgery Institute of the Favaloro Foundation (DDI (1351) 2616 CBE 630/16).

HUMAN AND ANIMAL RIGHTS

This research was carried out in accordance with the ethical principles of the Declaration of Helsinki, the US Code of Federal Regulations (Part 46, Protection of Human Subjects), and the International Conference on Harmonization Guidelines for Good Clinical Practice.

CONSENT FOR PUBLICATION

Written informed consent was obtained from each patient included in this study before enrollment.

AVAILABILITY OF DATA AND MATERIALS

All data generated or analyzed during this study are included in this published article.

FUNDING

None.

CONFLICT OF INTEREST

The author, Dr. Ramiro Sanchez, is the Co-EIC of CHR.

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