INVESTIGACIÓN CLÍNICA

Mechanical factors of cardiovascular risk in systemic arterial hypertension. A new sign of arterial rigidity

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Summary

Antecedent: By means of sphygmokinetocardiography (SKCG) we developed and arterial rigidity index (ARI) which measure the pulse wave aortic carotid reflexion time over the left ventricular ejection time (LVET). This index, together with the pulse wave velocity (PWV) and the pulse pressure (PP) are indicators of arterial stiffness. In this paper we measured these index in 27 systemic artery hypertension. Cases (group A, GA), with and without left ventricular hypertrophy (subgroups: A_1 SGA_1, n = 13, and A_2, SGA_2, n = 14), respectively, and 28 normotensive cases (group B, GB). **Protocol:** In two occasions: after 3 minutes of sitting position (SP) and after 3 minute of jogging in an upright position (UP), blood pressure, ARI, PP, PWV (aortic–hand finger distance/aorto–hand finger pulse time) and R–IV interval (electrocardiographic R wave–left early ventricular kinetocardiography deflexion) were measured. **Results:** Demography was similar in GA and GB. Systolic, diastolic and pulse pressure were significantly higher in GA *vs* GB. LVET (ms) was lower in GA *vs* GB in SP (268 ± 42 *vs* 274 ± 40, p < 0.001, respectively) and higher postexercise UP (280 ± 42 *vs* 244 ± 46, p < 0.001). PWV m/s were higher in SP in GA vs GB (9.8 ± 2.8 *vs* 7.4 ± 1.2, p < 0.001, respectively) and in UP (10.1 ± 1.9 *vs* 7.9 ± 9, p < 0.001, respectively). ARI was lower in UP in GA *vs* GB (0.48 ± 0.3 *vs* 0.80 ± 0.3, p < 0.003). Correlation index of PP *vs* SBP, *vs* DBP and *vs* PWV were significant in SP and in UP. Height had a significant correlation *vs* ARI in SP and UP (r = 0.60, p < 0.01, and r = 0.42, p

Resumen

FACTORES MECÁNICOS DE RIESGO CARDIOVASCULAR EN LA HIPERTENSIÓN ARTERIAL SISTÉMICA. UN NUEVO SIGNO DE RIGIDEZ ARTERIAL

Antecedentes: Mediante esfigmoquinetocardiografía (EQCG) se desarrolló un índice de rigidez arterial (IRA) que mide el tiempo aorto–onda de reflexión arterial sobre el período expulsivo. Este índice junto con la velocidad de la onda del pulso (VOP) y la presión del pulso (PP) son signos de rigidez arterial. Aquí medimos estos indicadores en 27 casos con hipertensión arterial (grupo A, GA) con y sin hipertrofia del ventrículo izquierdo: subgrupo A_1, SGA_1 de 13 casos y 14 individuos (subgrupo A_2, SGA_2) respectivamente y 28 casos normotensos (grupo B, GB). **Protocolo:** En 2 ocasiones: después de 3 minutos de posición sedente (PS) y después de 3 minutos de trote en posición ortostática (PO), se midió: la presión arterial (PA), el IRA, la VOP (distancia aorta–dedo–mano/tiempo de la onda del pulso aorta–dedo–mano) la PP y el intervalo R–IV (onda R del electrocardiograma –final de la deflexión ventricular temprana en el EQCG. **Resultados:** La demografía fue similar en ambos grupos. La PP, las presiones sistólica y diastólica fueron más altas en el GA *vs* el GB. El PE (ms) fue menor en el GA *vs* el GB en PS (268 ± 42 *vs* 274 ± 40, p < 0.001, respect.) y más alto en PO (280 ± 42 *vs* 244 ± 46, p < 0.001) en el GA *vs* GB. La VOP, m/s, fue más alta en PS en GA *vs* GB (9.8 ± 2.8 *vs* 7.4 ± 1.2, p < 0.001, respect.) y en PO (10.1 ± 1.9 *vs* 7.9 ± 9, p < 0.001, respect.). El IRA fue menor en PO en el GA *vs* GB (0.48 ± 0.3 *vs* 0.8 ± 0.3, p < 0.003). El índice de

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< 0.05, respectively). **Conclusion:** PWV is increased in GA *vs* GB patients. The ARI index is lower in GA *vs* GB cases in post exercise. PWV and PP showed a statistical significant correlation; height *vs* ARI had also a significant correlation: SKCG is a new method, that uses a not commercially instrument, which should have clinical application.

correlación de la PP *vs* PAS, PA y VOP tuvo significancia estadística en PS y en PO. La talla correlacionó con el IRA (r = 0.6, p < 0.01 en PS y r = 0.42, p < 0.05 en PO). **Conclusiones:** La VOP está aumentada y el IRA más bajo en el GA vs GB en PO (lo que indica mayor rigidez arterial). La VOP y la PP tuvieron correlación significativa con la talla al igual que esta última con el IRA. La EQCG es un método con aplicación clínica.
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Palabras clave: Mecánica arterial, Hipertensión arterial, Velocidad del pulso, Reflexión arterial.
Key words: Aarterial mechanicsm, Arterial Hypertension, Pulse velocity, Arterial reflexion.

Introduction

There is a dynamic and structural state of blood vessel hyperigidity in arterial systemic hypertension which increases the velocity of forward and retrograde arterial pulses,[1] and changes the circulatory reflection sites. This favors the early arrival of reflexive waves to the aortic root. The latter phenomenon alters the ventricular-aortic relationship, elevating the central systolic pressure and the arterial pulse pressure. It also boosts the telesystolic parietal stress of the left ventricle causing coronary circulation dysfunction.[2-4]

The deleterious effect of the above mentioned mechanical impairment is of epidemiological and clinical importance: population studies have revealed that increased pulse wave velocity (PWV) and increased arterial pulse pressure are risk factors for left ventricular hypertrophy (LVH),[5] total cardiovascular damage[6] and myocardial infarction.[7] Furthermore, transversal studies have demonstrated that these indicators are associated with brain hemorrhage,[8] cutaneous hypertensive microvascular changes,[9] renal failure-induced cardiovascular damage[10] and advanced age, to mention a few.[11]

Because pulse pressure can be easily measured, considerable interest has risen to apply this measurement in everyday clinical practice.[12] Numerous publications have shown that increased pulse pressure is a more potent cardiovascular risk indicator than systolic or diastolic blood pressures,[13] even though the latter have proven predictive value.[14] Moreover, the relationship between pulse pressure and arterial rigidity has been clearly established in experimental observations.[15,16] A clinical physiographic method called digital sphygmokinetocardiography was recently developed at our hospital.[1] This method is useful to

study important hemodynamic aspects of cardio-arterial function, as the following signals can be obtained from a patient in sitting position and in standing position after exercise: electrocardiography signals, finger photoplethysmography (f-PP), carotid oscillation pulse signals, as well as vibriograms generated by the contraction of the anterior and posterior left ventricle walls registered on the precordium and the subcostal abdominal aspect respectively.[1,17] Based on this method, we developed an arterial rigidity index (ARI) which measures the interference caused by the arterial reflection wave to left ventricle ejection time interval.[18] In the present study we analyze ARI, pulse wave velocity and pulse pressure, all indicators of arterial stiffness, to study hypertensive patients with and without left ventricular hypertrophy (LVH), as well as normotensive individual.

Objective

1) To analyze the correlation between the arterial rigidity index and pulse wave velocity as measured by sphygmokinetocardiography, as well as with systolic, diastolic and pulse pressures, in order to assess the relevance of these parameters in detecting arterial rigidity in everyday clinical practice.
2) To study the aortic-finger pulse wave velocity, the arterial reflection index and the electrocardiographic R–IV interval in hypertensive patients with and without LVH.

Methods

This clinical study was transversal, prospective, and analyzed age and gender matched cases and controls.

Subjects. We analyzed a total of 55 individuals belonging to two different groups: group A included 27 outpatients aged > 30 years and diag-
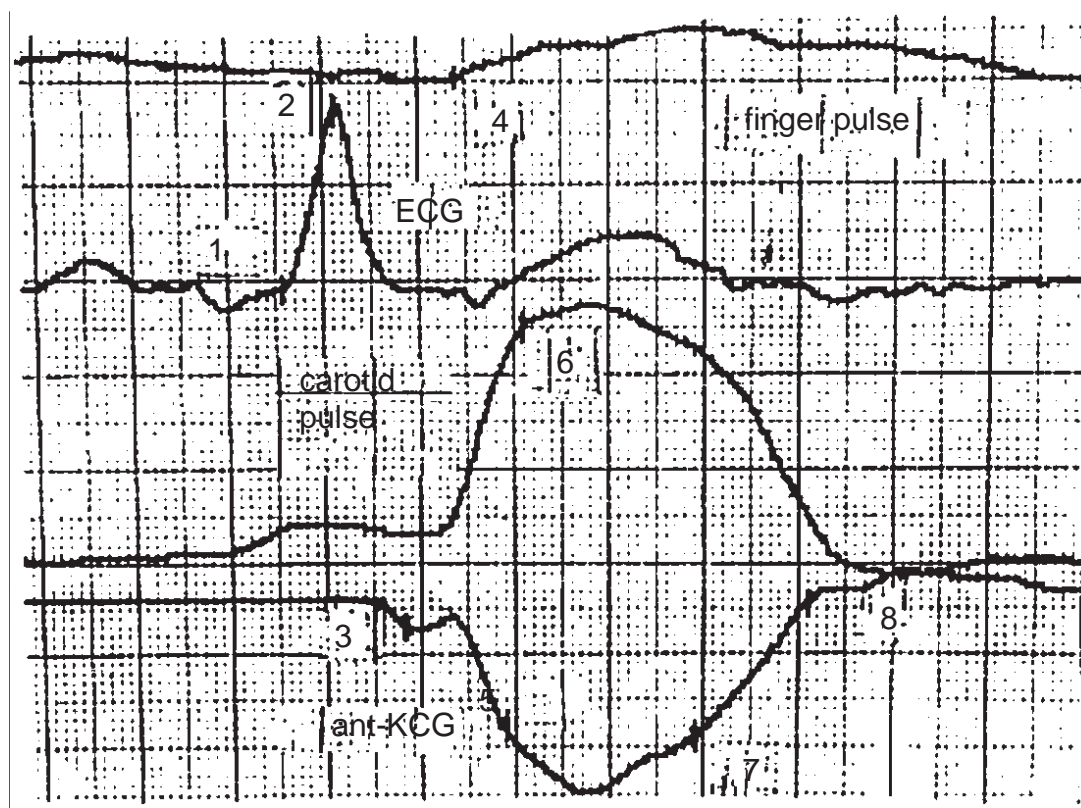
Fig. 1. Cardiocirculatory intervals measured by sphygmokinetocardiography. Points: 1) initiation of QRS complex. 2) Peak of R electrocardiographic wave. 3) Initiation of ventricular impulse deflexion. 4) Foot of the hand-finger pulse. 5) Final point of early ventricular impulse. 6) Carotid reflexion wave front. 7) Aortic valve closure. 8) End of ventricular relaxation. Intervals 1-2 pre–ejection time, 3-5 duration early ventricular impulse, 2-5 interval R-VI (VI = ventricular impulse), this interval include isovolumic interval 3-7, ejection time 3-4 aortic-hand finger time. The graph was run at 50 mm/seg; the amplitude was artificially magnified.

nosed with essential arterial hypertension, with no acute or chronic distressing diseases (particularly any type of organ failure), and who had not received any kind of pharmacological treatment at least 2 weeks before the study (8 cases had never been treated). All standard procedures to study hypertensive patients and a 12-lead ECG were performed. Blood pressure (BP) was measured by auscultatory sphygmomanometry on 3 different days, and individuals were diagnosed as hypertensive when the average blood pressure was higher than 140/90 mm Hg.[19] Group A individuals were further classified in two subgroups: SGA1 (13 cases) and SGA2 (14 cases) according to the presence or absence of left ventricle hypertrophy as assessed by the criteria of the Mexican School of Cardiology.[20] Group B included 28 government office employees who were normal on medical examination, and with an auscultatory BP lower than 140/90 mm Hg measured both in sitting and standing positions.

Individuals from both groups were matched according to age and gender.

Protocol. BP was measured between 10:00 AM and 4:00 PM in the sitting position by auscultatory sphygmomanometry. Afterwards, the sphygmokinetocardiographic study was performed using a device developed by us.[5,18] This device includes a personal computer (PC) with an analog to digital converting card with 10 bits resolution, allowing to record the DII-lead ECG, the photoplethysmographic pulse on the index finger, signals generated in pneumatic detectors to register the carotid oscillation pulse (COP) from a neck segment, as well as two vibration signs (kinetocardiograms, KCG) produced by the left ventricle and registered on the anterolateral aspect of the left hemithorax (ant-KCG) and on the subcostal region below (post-KCG). Signals were captured at a rate of 350 samples per second. MPX2052 Motorola pressure transducers were used to register two simultaneous signals

with two pneumatic detectors: The signals were then displayed on the PC monitor and saved to perform offline measurements.

Procedure. The following detectors were used: electrodes to register the ECG (DII), pulse photodetector placed on the index finger, and 3 pneumatic detectors named: no. 1 (D1), no. 2 (D2) and no. 3(D3) [4 x 12 cm rubber bags connected to the pressure transducer and to a manual air pump]. These detectors were held by inextensible cloth belts (6 cm width) positioned tightly and transversally surrounding the neck and or the thorax, as follow: no. 1 (D1) was placed on the neck from the midline to the left anterolateral side; D2 was placed on the anterolateral aspect of the thorax from the midclavicular line outwards, covering the 4th and 5th left intercostals spaces *(Fig. 2)*; and D3 was placed on the upper abdominal aspect (left subcostal) in parallel to detector no. 2. All detectors were connected to an electronic signal adequacy box and to the PC. With the patient sitting and in sustained inspiratory breathing, the pneumatic detectors were inflated to 50 mm Hg. We then simulta-

neously recorded the following electronic signals in 6 to 10 consecutive cardio-arterial cycles: the DII-lead ECG signal, the finger-hand photoplethysmographic pulse (FHP), the carotid oscillation pulse (COP) (detector 1), vibrations produced by the anterior left ventricle wall (ant-KCG) (detector 2), and the vibrations produced by the posterior left ventricle wall (post-KCG) transmitted through the diaphragm (detector 3). DII ECG lead, FHP and ant-KCG signals were obtained simultaneously, as well as the following combinations: COP + ant-KCG, and COP + post-KCG. Then patient stand for 3 minutes and a complete signal recordings and BP measurements were performed after jogging in a fixed position (60 steps per minute) for 3 minutes.

Measurements: All good quality signals were printed on millimetric paper. With the aid of the ECG, the finger-hand pulse or the carotid oscillation pulse, the following cardiologic or cardiocirculatory intervals were measured in triplicate *(Fig. 1)*: a) R-R electrocardiographic interval (R wave to R wave); b) ejection time (ET) measured by anterior sphygmokinetocardiogra-
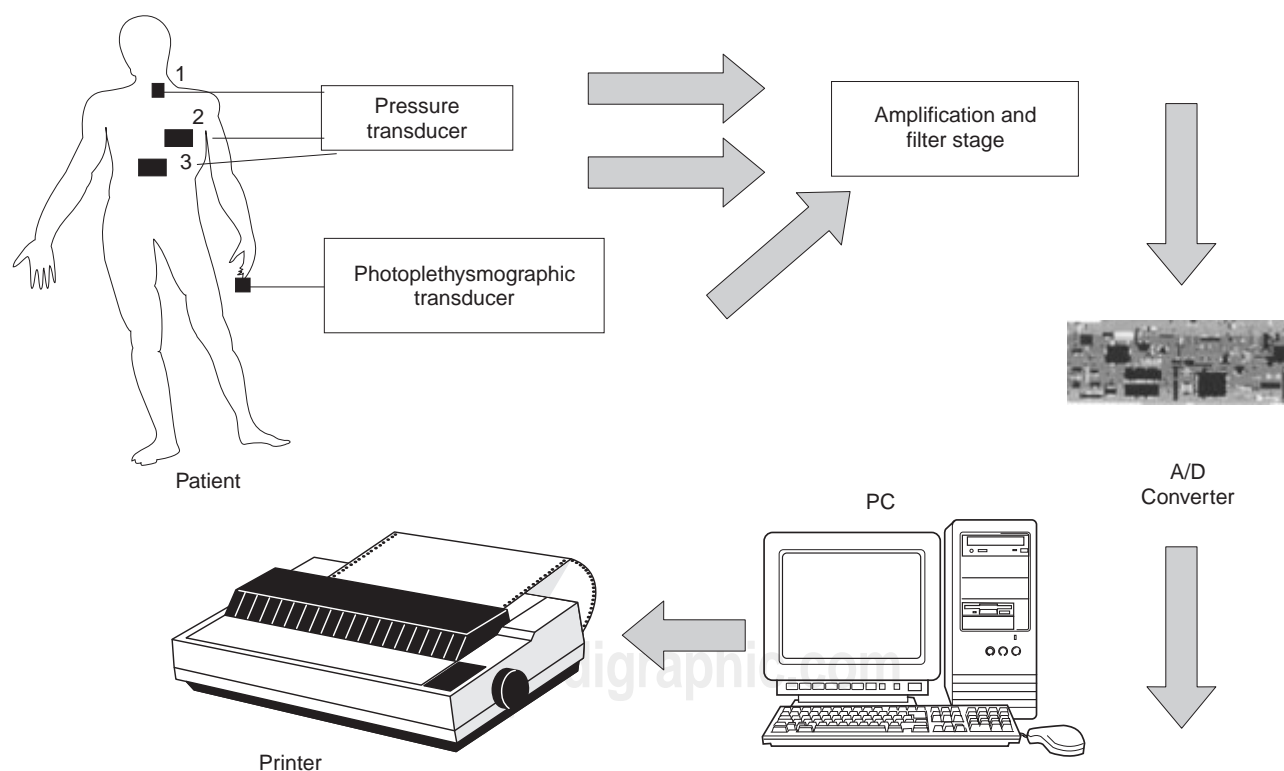
Fig. 2. Diagram showing the location of detectors (no. 1 in neck, no. 2 in antero-lateral aspect of left hemithorax, no. 3 subcostal region). Photoplethysmograph detector in finger.

phy, and defined as the interval between the initiation of the left ventricular impulse deflexion (VID) and the aortic valve closure (AVC); c) aortic-reflection time (ar-T) defined as the interval between the VID and the reflection wave front; d) aorta-finger hand time (A-FT), defined as the interval between the beginning of the ventricular impulse and the foot of the hand-finger pulse; and e) anterior and posterior R-VI time, defined as the interval between the ECG R-wave peak and the final point of the ventricular impulse on the anterior or the posterior KGCs respectively. Based on these measurements, we obtained the following indexes and intervals: a) arterial reflection index (ARI) estimated as ar-T/ET; b)anterior and posterior R-IV time; and c) pulse wave velocity (PWV), estimated by the following equation: PWV = d/A-FT, where d/ = the aorta-finger hand distance as measured with a metric tape, following the superficial projection of the arterial pathway (from the middle sternum to the distal index finger pulp),[17] and A-FT is the aorta-finger hand time.

The data used for the analysis were the mean of 3 measurements.

Statistical analysis

Continuous variables are expressed as the mean ± standard deviation, while discrete variables are reported as absolute or relative (percentage) frequencies. Differences among groups were compared using non-paired Student's t test for continuous variables, and the chi square test to compare proportions.[21] Statistical significance was considered when $p < 0.05$. The linear association between variables was assessed using Pearson's correlation coefficient.

Results

No differences in age (56.7 ± 9.98 *vs* 55.6 ± 11.77 years); sex ratio (male/female 13/15 *vs* 15/13), weight (74.6 ± 13.41 *vs* 76.0 ± 11.41 kg) and height (161 ± 9.81 *vs* 164 ± 9.33 cm) were observed between groups A and B respectively *(Table I)*. In these groups systolic, diastolic and pulse pressures (SBP, DBP and PP) were significantly higher in hypertensive patients both in the sitting position (159 ± 15.5, 92 ± 9.2 and 64.1 ± 10.3 mm Hg respectively) and in the standing position after exercise (187 ± 24.7, 105 ± 14.0 and 86 ± 19.3 mm Hg respectively) than in non-hypertensive subjects (SBP, DBP and PP were 118 ± 10.9, 73.9 ± 6.6 and 44.1 ± 10.8 mm Hg respectively in the sitting position, and 128 ± 11, 74.2 ± 7 and $.54 \pm 9.2$ mm Hg respectively after exercise in standing position). Mean PWV was significantly higher in group A (9.8 ± 2.8 m/s) than in group B (7.4 ± 1.2 m/s, $p < 0.001$), while the post-exercise arterial reflection index was significantly lower in group A (0.48 ± 0.3) than in group B (0.80 ± 0.3, $p < 0.001$) *(Table II)*.

Correlation indexes were measured in the total population. Positive correlations were found for PP and SBP in the sitting position ($r = 0.94$, $p < 0.001$); for PP and DBP in the sitting position ($r = 0.56$, $p < 0.005$), for PP and SBP after exercise ($r = 0.81$, $p < 0.0001$) and for PP and DBP after exercise ($r = 0.62$, $p < 0.005$). PP *vs* PWV and Height *vs* ARI also have a positive correlation index *(Table III)* in both positions *(Table 3)*.

Table I. Demography.

	GA	GB
Age (years)	56.7± 9.98	55.6 ±11.77
Sex ratio (M/F)	13 / 15	15 / 13
Weight (kgs)	74.6± 13.41	76.0 ±11.42
Height (cm)	161± 9.81*	164 ±9.33

* $p \leq 0.05$, GA = Hypertensive patient, GB, normotensive subjects

Table II. Sphygmokinetocardiographic data.

	Sitting position		Standing position after exercise	
	GA	GB	GA	GB
R-R (ms)	900 ± 130*	931 ± 147	843 ± 111	840 ± 134
SBP (mm Hg)	159 ± 15.5*	118 ± 10.9*	187 ± 24.7*	128 ± 11
DBP (mm Hg)	92.5 ± 9.2*	73.9 ± 6.6*	105 ± 14*	74.2 ± 7
PP (mm Hg)	64.1 ± 10.3*	44.1 ± 10.8*	86 ± 19.3*	54 ± 9.2
ET (ms)	268 ± 42*	274 ± 42*	280 ± 40*	244 ± 46
ARI	0.53 ± .27	0.58 ± 0.2	0.48 ± 0.3*	0.80 ± 0.3
PWV (m/s)	9.8 ± 2.8*	7.4 ± 1.2*	10.1 ± 1.9*	7.9 ± .9

* $p < 0.001$ GA *vs* GB   ** $p < 0.001$ GA *vs* GB
R-R, ECG; ET = ejection time; SBP = systolic blood pressure; BDP = diastolic blood pressure; PP = pulse pressure; ARI = arterial reflection index; PWV = pulse wave velocity. GA = Hypertensive patient,GB, normotensive subjects

Table III. Correlation indexes (estimated considering the total population, n = 55).

	Sitting position	Standing position after exercise
PP *vs* SBP	r = 0.94, p < 0.001	r = 0.81, p < 0.0001
PP *vs* DBP	r = 0.56, p < 0.005	r = 0.62, p < 0.005
PP *vs* PWV	r = 0.42, p < 0.05	r = 0.41, p < 0.05
Height *vs* ARI	r = 0.60, p < 0.01	r = 0.42, p < 0.05

Abbreviations as in Table II.

On comparing subgroups A1 (with LVH) *vs* A2 (without LVH), no significant differences were found on SBP in the sitting position (160 ± 15.2 *vs* 158 ± 16.4 mm Hg) and on pulse pressure (79 ± 18.2 *vs* 77 ± 15.3 mm Hg). Body height however, was significantly lower in hypertensive patients with LVH (157 ± 6.1 cm) as compared to those without LVH (164 ± 11.6 cm, p = 0.06) *(Table IV)*. In the sitting position, the ARI, PWV and R-IV time were not significantly different in subgroups A1 (0.45 ± 0.25, 9.1 ± 2.2m/s and 222 ± 35.5 ms respectively) and A2 (50 ± 0.3, 8.9 ± 3.2, and 229 ± 30.1, respectively). However, post-exercise measurements in the standing position revealed significant differences, as the ARI was significantly lower in SGA1 (0.45 ± 0.25) than in group SGA2 (0.62 ± 0.28, p < 0.001), while R-W time was significantly higher in group SGA1 (237 ± 32.4 *vs* 203 ± 32, p = 0.001).

Comments

The hemodynamic impairment occurring in systemic arterial hypertension has been until recently explained applying the law of Poiseuille, which assumes that blood pressure and blood flow are constants. However, circulatory flow is not laminar and has a pulsatile character that acts against the viscous resistance and simultaneously accelerates the blood flow mass.[22] For this reason, it is now considered that the arterial system represents a burden to left ventricle ejection and has two components: one is resistive, explained by the poiseullian circulatory model, and the other is pulsatile manifesting properties of unequal inertia and compliance heterogeneously distributed throughout the arterial tree.[23] Increased arterial rigidity in hypertension makes the pressure wave hyperpulsatile and alters the ventricle-artery relationship, leading to ventricular hypertrophy. Furthermore, it alters the viscoelastic properties of arteries favoring the development of arteriolosclerosis and aortosclerosis, which are both characteristic of this disease. Thus, the study

Table IV. Comparison of some measurements in hypertensive patients with and without left ventricular hypertrophy.

	SGA1 (with LVH)	SGA2 (without LVH)
Height (cm)	157± 6.1*	164±11.6
SBP (mm Hg) Sitting	160± 15.2	158±16.4
PP (mm Hg) Sitting	79± 18.2	77±15.3
ARI (SPE)	0.45± 0.25***	0.62±0.28
postR-VI (SPE)	237± 2.4***	203±3.2
PWV (m/s)	10.7±2.3**	9.6±1.4

* p = 0.06;** p = 0.094;*** p < 0.001
SPE: Standing post-exercise; other abbreviations as in Table II.

of pulsatile mechanics is particularly relevant in everyday clinical practice.[23]

Arterial Reflection Index. The arterial pulse wave has two components: 1) a forward or incident component which travels from the heart to the periphery, and 2) a retrograde or reflexive component that travels in the opposite direction. The latter component is the result of the addition of several reflection waves: some are generated in bifurcations near the heart, and others of variable magnitude are reflections originated at the arteriolar region of the arterial tree.[24] The pathogenic importance of these waves has been underscored in experimental studies where a hard, artificial aorta is inserted in dogs: blood flowing through this stiff aorta leads to an immediate increase of the pulse pressure, altered diastolic coronary flow[24] and boost the left ventricle parietal stress.[24] All these phenomena are associated with an early return of reflection waves. When the blood flows through the normal elastic aorta, all the above mentioned anomalies disappear.

At our hospital, we have developed a sphygmo-kinetocardiographic arterial reflection index, which measures the arrival time of arterial reflection waves in relation to the ejection time, and can disclose the interference of the retrograde flow on the forward flow at the aortic root. Moreover, this factor can distend the left ventricle during telesystole, and may cause ventricular extrasystolia by means of an electromechanical feedback phenomenon.[18]

In the present study, we found that the arterial reflection index was lower in hypertensive patients as compared to normal individuals *(Table II)*, indicating that early arterial reflection interferes to a greater extent with ventricular ejection. In addition, the ARI was significantly lower in hypertensive patients with LVH as compared to those without LVH, particularly after exercise.

These findings were expected, as the rigidity of the aorta and central arteries is characteristic in hypertensive individuals, particularly in those with LVH.[5] The ARI decrement after exercise is due to increased intrarterial pressure which distends the arterial wall, and according to the elastic module of the vessel, increases its rigidity. Numerous observations in the medical literature have associated the hypertensive state per se with the early return of reflection waves, especially when hypertension coexists with left ventricle hypertrophy, advanced age or renal failure.[10,26] These studies assess the effect of reflection waves by the so-called "augmentation index".[27] This index measures the increase in central systolic pressure caused by reflection waves using sophisticated technologies which rebuild the intraortic pulse from an arterial (carotid or subclavian) tonometric pulse with a mathematical transference function model.[28]

As mentioned above, the ARI measures the precocity of reflection arrivals in relation to ventricular ejection. In fact, it indicates the percentage of time that ejection is free of retrograde influence, and when expressed inversely, indicates the percentage of time the reflexive phenomenon interacts with ejection (i.e. an index = 0.30 indicates that mesosystolic outflow is interfered with during 70% of the ejection time).[18]

One of the limitations of ARI or augmentation index measurements is that both infer that carotid reflections are indicators of ascending aorta rigidity, even though the viscoelastic properties of both arterial segments are not necessarily the same. However, the morphology of the aortic pressure curve rebuilt by transference function is very similar to the carotid pressure curve. Thus, the study of the latter does not represent a relevant impediment.[29,30] Of course the time required for the carotid reflection to arrive to the aortic root reduces the accuracy of the temporality of events.

Pulse wave velocity (PWV). This parameter has been measured and analyzed since the last century, and is a well known indicator of arterial rigidity (because of the demonstrated inverse relationship between arterial compliance and arterial pulse wave velocity).[23] As a matter of fact, over the last years PWV has been underscored as a cardiovascular risk factor in arterial hypertension with or without LVH, and has been associated with diabetes mellitus, age, renal failure and atherosclerosis.[31-33] We corroborated once

again the statistical association between increased PWV and hypertension and that this parameters the ARI and the pulse pressure, are arterial stiffness indicators. In general terms, pulse wave velocity is clinically measured by registering the passing fronts of the wave on two superficial sites of an accessible arterial segment.[1,5] The sphygmokinetocardiograpy method developed at our department can register on the thorax the vibrations generated by left ventricle volume changes transmitted through the lung acting as a resonance box.[17,18] Transmission is better perceived during sustained inspiration, as the cardiac vibration signs are registered in the absence of respiratory sounds. In this manner, the aorta-finger hand pulse time can be measured in the same cardiocirculatory cycle: from the initiation of the pulse wave at the aortic root, inferred by the initiation of the ventricular impulse (ET initiation point on the ant-KCG, *Fig. 1*), until it reaches the foot of the hand-finger pulse.[17] This method is easy to perform, not only at rest but also in a standing position and after dynamic exercise. We have found no previous references in the medical literature describing this advantage for other procedures. This measurement provides interesting perspectives for clinical arterial mechanics studies.

R-VI time. Kinetocardiography has been used for more than 4 decades. It is a well known method used to measure systolic intervals, specially during the ejection period, and correlates well with the ejection fraction.[34,35] Our version of KCG has the technological advantage of digitalization, and in addition, our mechanical receptor remains properly positioned by means of an inextensible perithoracic belt. This allows to register signals from individuals in different physiological positions (lying, sitting, standing and after exercise). With our method, the systolic ventricular impulse is well defined and graphically expresses 2 components: one is fast, related to the initial ventricular contraction; and the other is slower, mainly expressing ventricular relaxation *(Fig. 1)*. The analysis of high fidelity left ventricle pressure curves and the apical kinetocardiogram in humans demonstrated that point "0" on the intraventricular pressure curve and the systolic ventricular impact on the apicogram are simultaneous, and that the outlines of both graphs overlap.[36,37] We thus considered that the R-VI time measures the duration of fast ventricular contractility. Experimentally, increased afterload leng-

thens the contractile duration of the left ventricle.[11] We observed here that the R-VI time registered by post-KCG after exercise was longer in hypertensive patients with LVH as compared to those without LVH *(Table IV)*, in accordance with the pathophysiologic assumption made above.

Pulse pressure. Pulse pressure is a well established cardiovascular damage indicator.[38] Information on this issue has increased considerably over the last years, and we now know that increased pulse pressure is associated with increased left ventricular mass[39], carotid atherosclerosis[40] and brain damage.[41] In some studies, pulse pressure has been identified as an independent risk factor of prognostic value for cardiovascular damage.[38]

The factor underlying this phenomenon is the loss of arterial compliance, which decreases arterial pulsatility cushioning and increases pulse pressure. We found here that pulse pressure was wider in hypertensive as compared to normal subjects, and that this parameter was more significant as an independent risk factor than systolic or diastolic blood pressures, confirming previous observations made elsewhere.[42-44] Thus, this parameter is a good indicator of arterial stiffness that can be applied in clinical practice. Unexpectedly, we found that PP was not significantly different in hypertensive patients with and without electrocardiographic evidence of LVH. We have no explanation for this observation, and will further analyze this issue in the future, studying a large number of cases.

Body Height. As expected,[45] we observed a correlation between short stature and the post-exercise arterial reflexion index *(Table III)*. In fact, height was significantly lower in hypertensive subjects with left ventricle hypertrophy as compared to those without LVH *(Table IV)*. Using other arterial stiffness indicators, Marchain et al. observed the same correlation in patients with renal failure[10] and suggested that this happens when renal failure onset occurs during body development leading to malnutrition and short stature. Further studies confirmed the inverse relationship between short stature and increased cardiovascular risk in all terminal renal failure patients.[46] Recently, this correlation was also observed in hypertensive non-uremic patients by studying pulsatile parameters indicative of impaired arterial hemodynamics.[45] As our findings are similar, we suggest that arterial reflection sites are closer to the heart in hypertensive subjects with short stature as compared to taller ones, favoring early arrival of reflection waves to the aortic root. Thus, short stature would be an independent risk factor, and when occurring in a hypertensive state would favor more severe cardiovascular damage. Perhaps this same pathogenic mechanism explains the association between cardiovascular risk (particularly coronary risk) and short stature reported since 1951.[47,48]

Conclusions

1. PWV is increased in sitting and orthostatic post-exercise positions in GA vs GB. Arterial reflexion index is reduced in the last position in systemic arterial hypertension as compared to normal individuals. This underscores arterial hyperigidity in hypertension, which is of particular pathogenic interest.

2. Pulse pressure shows a positive correlation with systolic blood pressure and to a lesser extent with diastolic blood pressure. PWV and PP showed a statistically significant correlation.

3. Early reflection waves correlate with short stature. This gives way to interesting pathophysiologic speculations.

4. Hypertensive patients with LVH had lower height, longer ventricular impulse and shorter post-exercise ARI as compared to those without LVH.

5. The method here described may have useful applications in everyday clinical practice. Specially it is convenient to study a large number of patients and verified changes of rigidity indexes with antihypertensive treatment.

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