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Heterogeneity of the arterial tree in essential hypertension: a noninvasive study of the terminal aorta and the common carotid artery

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Summary:

The diameter and the stiffness of the distal portion of the abdominal aorta and of the common carotid artery were evaluated noninvasively in 72 subjects with mild to moderate hypertension by comparing 39 normotensive controls of same age and sex distribution. For each arterial segment, pulsatile changes of diameter were determined by a newly described echotracking technique and pulse pressure was evaluated by applanation tonometry and oscillometric methods. In both territories, distensibility of hypertensive subjects was significantly reduced but compliance was reduced only at the aortic level. Diastolic diameter was increased at the carotid but not at the aortic site. Multiple regression analysis indicated that whereas compliance and distensibility were strongly influenced by BP in the terminal aorta, age was the predominant factor influencing carotid arterial stiffness. Following 30 days of converting enzyme inhibition, the decrease of BP was associated with a normalisation of aortic compliance and distensibility whereas no change was observed at the site of the carotid artery. The study provides evidence that the response of the arteries to the changes of BP differ in the various parts of the arterial tree. This factor may influence the drug effect produced by chronic converting enzyme inhibition.

Keywords: *hypertension, arterial system, antihypertensive therapy*

Introduction

The heterogeneity of the arterial system has been recognised for 30 years on the basis of several observations (review in ref. 1). First, the elastic central arteries have important histomorphometric differences compared with the peripheral muscular arteries. Second, in hypertension the pulse pressure signal which causes the arterial systolic-diastolic distension, varies along the arterial tree and augments substantially from central to peripheral arteries. Finally, arteries, as they get smaller, have an increasing dependence of extrinsic tone on calcium in the extracellular space and an increased variability in the neurotransmitters involved.[2] In past years, such differential aspects of the arterial system have been poorly investigated in terms of clinical management and therapeutic approach. However, following antihypertensive drug treatment, it has been observed[3] that whereas the incidence of stroke is considerably reduced there is less influence on the incidence of ischaemic heart disease.

Recent studies on arterial stiffness have suggested that in hypertensive subjects the rigidity of peripheral arteries, such as the radial artery, is much more influenced by BP than by age whereas central arteries, like the carotid artery and the thoracic aorta, are less influenced by BP but much more by the ageing process.[4] Such observations clearly indicate that the arteries respond very differently in each particular compartment of the vascular system and that this response may be influenced selectively, either by the ageing process, by hypertension or by a combination of both factors.

The purpose of the present study was: (1) to evaluate in normotensive and hypertensive subjects, the diameter and the stiffness of two different arteries: the common carotid artery and the terminal aorta, and (2) to determine for each of them the specific influence of age and BP. The latter question is analysed before and following BP reduction caused by a one month treatment with the converting enzyme inhibitor, Ramipril.

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Patients and methods

Patients

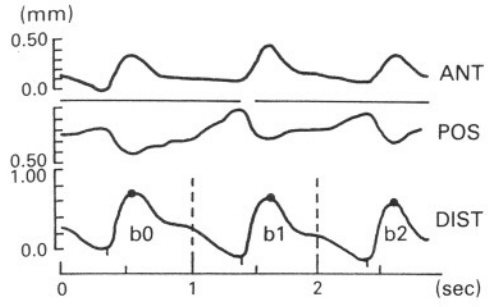
One hundred and eleven subjects (70 men and 41 women) were selected from the Hypertension Centre, Broussais Hospital, Paris. Of these subjects, 72 had mild to moderate essential hypertension and 39 had no past history of high BP and were considered normotensive subjects. Criteria for selection have been extensively published.[5,14] Mean ± SEM age was 47 ± 2 years (range 23–71 years). Weight and height were 71 ± 2 kg and 169 ± 1 cm, respectively. In all hypertensive patients, treatments were discontinued at least 21 days before the study and DBP (by conventional sphymomanometry) remained above 90 mmHg throughout this ambulatory washout period. Patients had no signs, symptoms or history of cardiac, renal or cerebrovascular accident or major diseases other than hypertension. On the basis of conventional echo-Doppler methods, no stenosis of > 30% of the lumen area or atheromatous plaque of the common, internal carotid, or iliofemoral arteries was noticed. The evaluation of velocities with a continuous Doppler device confirmed the absence of any significant lesion of the studied segments. Thus, although all of the included patients had normal arteries from the clinical and ultrasonic points of view, this study could not accurately evaluate the thickness of the arterial wall and it is possible that wall thickening was more pronounced in some of the studied segments than others. Plasma glucose, total cholesterol and high density lipoprotein cholesterol were constantly within the normal range. Twenty subjects smoked. None of the 111 subject used antidiabetic or hypolipidaemic medication. Informed consent was obtained from each subject after a detailed description of the procedure. The protocol was approved by INSERM (Institut National de la Santé et de la Recherche Médicale, France). Each subject was investigated in a controlled environment of 22 ± 1 °C. After 20 minutes of rest in the supine position, SBP and DBP and heart rate were determined every two minutes by an oscillometric recorder (model 845, Dinamap, Critikon, Tampa, FL) positioned on the right arm. The same parameters were also measured with a mercury sphygmomanometer on the right arm.[5] In this latter case, mean arterial pressure (MAP) was estimated by the formula MAP = DBP + (SBP-DBP)/3, where DBP and SBP are diastolic and systolic BP, respectively. Then, arterial measurements were performed at the sites of the right common carotid artery and the terminal aorta. Diameter changes were studied exactly at the same (carotid or aortic) point for repeat determinations. A marker was used to localise the site of the arterial segment.

Systolic-diastolic variations of arterial diameter[5–7]

The vessel wall motion of the arteries was measured by using an original pulsed ultrasound echotracking system based on Doppler shift. The operating frequency of the device was 5 MHz. The details of this method have been described elsewhere.[6] Briefly, this system enables the transcutaneous assessment of the displacement of the arterial wall during the cardiac cycle and hence the time-dependent changes in arterial diameter relative to its initial diameter at the start of the cardiac cycle. The availability of the electrocardiogram (ECG) trigger facilitates the detection of the peak distension of the artery relative to its initial diameter. The lowest and highest values within 300 ms after the occurrence of the ECG trigger are taken as the minimum and maximum values of the distension waveform, respectively. This procedure can detect the diastolic and systolic peak diameters in both common carotid artery (CCA) and terminal aorta. Measurements were performed for the CCA at 2 cm below the carotid bifurcation and for the aorta at 3 cm above the aortic bifurcation. Based on the two-dimensional B-mode image, a M-line perpendicular to the artery was selected. The radiofrequency signal of three to eight cardiac cycles was recorded, digitised and temporarily stored in a large memory. Two sample volumes, selected under cursor control, were positioned on the anterior and posterior arterial walls. To overcome the possibility that nearby structures generating prominent echoes may have temporarily entered the selected sample volumes, thus obscuring the vessel wall signal, a tracking system was developed that allowed the vessel walls to be tracked by the sample volumes. Then the displacement of the arterial wall was obtained by processing the Doppler signals originating from the two selected sample volumes. A typical displacement waveform of the anterior and posterior walls of the common carotid artery is shown in Figure 1: the successive values of the stroke change in diameter during systole (Ds-Dd), the end-diastolic diameter (Dd) and the relative stroke change in diameter ([Ds-Dd]/Dd) were computed from the recording.

With this procedure, the side of measurement did not influence the values of arterial dimensional data. For instance, in 17 normotensive subjects, no significant difference was observed between measurements performed on the left and the right common carotid arteries (7.0 ± 0.3 vs. 7.0 ± 0.2 mm, 0.45 ± 0.03 vs. 0.44 ± 0.02 mm and 6.6 ± 0.5% vs. 5.4 ± 0.4% for Dd, Ds-Dd and [Ds-Dd]/Dd, respectively).

The repeatability of the carotid artery measurement was studied in five normotensive subjects.[7] The coefficient of variation (standard deviation expressed as a percentage of the mean of several successive measurements) was used for this purpose. First, repeatability was assessed during the recording of three to eight successive cardiac cycles as shown in Figure 1. The mean ± SEM coefficients of variation determined under these conditions were 1.0 ± 0.3%, 6.0 ± 0.1% and 6.0 ± 0.1% for Dd, Ds-Dd and (Ds-Dd)/Dd, respectively. Second, repeatability was assessed during 12 measurements performed by each of two observers over a 90 minute period in five subjects. Each measurement of the 12 was the mean of three to eight values



Initial diameter: 8.78 mm Data from: DAS

Beat	DIST (μm)	DIAM (mm)	DIST (%)	RR-INT (ms)	RISE-TIME (ms)
0	606	8.59	7.06	1057	92
1	661	8.47	7.81	1037	95
2	634	8.47	7.48		89
Mean	634	8.51	7.45	1037	92
STDEV	28	0.07	0.37	0	3

Figure 1 Typical example of the pulsatile changes of the common carotid artery diameter obtained with the echotracking technique. ANT = anterior wall; POS = posterior wall; dist = systolic-diastolic diameter changes; DIAM = diameter b0; b1 and b2 = beats 0, 1 and 2, respectively; RR-INT = RR interval; DAS = data analysis.

corresponding to three to eight cardiac cycles. Under these conditions, the mean intraobserver coefficient of variation was $3.0 \pm 0.1\%$, $8.0 \pm 0.1\%$, and $10.0 \pm 0.1\%$ for Dd, Ds-Dd and (Ds(Dd)/Dd, respectively.

The repeatability of the terminal aorta measurements was studied in seven normotensive subjects following the same procedure. Inter-observer coefficients of variations were $1.5 \pm 0.4\%$, $7.8 \pm 0.3\%$ and $8.2 \pm 0.2\%$ for Dd, Ds-Dd and (Ds-Dd)/Dd, respectively. Intra-observer coefficients of variation were $4.6 \pm 0.3\%$, $10.5 \pm 0.2\%$ and $12.0 \pm 0.3\%$, respectively.

Systolic-diastolic variations of BP[5,8,9]

To improve the accuracy of noninvasive recording of the arterial pressure wave contour at the site of the carotid artery, we used a pencil-type probe incorporating a high-fidelity strain-gauge transducer (Millar Instruments, Houston, TX). The transducer has a small pressure-sensitive area (0.5×1.0 mm) with a frequency response of > 2 kHz that is coplanar with a larger area (7 mm diameter) of flat surface that is in contact with the skin overlying the pulse. The instrument uses the principle of applanation tonometry as it is used in ocular tonometry for registration of intraocular pressure. In principle, flattening (applanation) of a curved surface that is subject to internal pressure allows direct measurement of the pulse pressure within the structure. The use and accuracy of the tonometer were previously studied and validated on the exposed canine femoral artery and percutaneously on the human radial artery.[8] In addition, in 16 subjects undergoing catheterisation, BP was measured simultaneously by two methods: invasively at the site of the aortic arch and noninvasively at the site of the common carotid artery. A significant positive correlation ($r = 0.93$, $P < 0.0001$) was observed with a slope equal to 1.05 and an intercept that was not significantly different from zero (0.4 mmHg).[5] In another study in 105 subjects, we measured brachial pulse pressure by conventional sphygmomanometry and radial pulse pressure by applanation tonometry. The two parameters were strongly correlated: $r = 0.97$, slope, 0.98, intercept, 1.4 mmHg. Because the tonometer transducer is small relative to the size of the artery, the positioning of the transducer over the site of the artery was found to be an important consideration for clinical investigation. Difficulties arising from these measurements and the way to correct them have been described in detail elsewhere.[5] Intra-observer variability of the measurement was $4.7 \pm 2.5\%$ and inter-observer variability was $6.1 \pm 3.5\%$. Such levels of reproducibility can be achieved after four to six weeks use of the probes.

For the pulse pressure measurement at the site of the terminal aorta, brachial pulse pressure measurements by Dinamap was used. The differences in the pressure contour in the aorta and the brachial artery are well known because of the pressure wave reflections[1] and it is possible that the values of aortic pulse pressure may be slightly underestimated by this procedure. However, we have previously shown[10] that brachial pulse pressure gives an acceptable approximation of intra-aortic pulse pressure because the square root of the brachial-aortic pulse wave velocity ratio does not differ greatly in hypertensive and normotensive subjects and is close to one. On the other hand, using invasive studies, Imura *et al.*[11] have clearly shown that pulse pressure measured at the site of the brachial artery and of the terminal portion of the abdominal aorta are practically identical in terms of statistical significance.

Estimation of arterial compliance and distensibility[1,5,6]

The distension of an artery (change in diameter) during a cardiac cycle depends on the elastic characteristics of the vessel wall (and the surrounding tissues) and the local pulse pressure. This relation can be expressed as $C = dV/dP$, where C represents compliance, dV the systolic-diastolic changes of the volume of an arterial segment and dP the pulse pressure (systolic minus diastolic BP). Assuming that the increase in volume is caused only by the distension of the artery (and not by elongation), the cross-sectional compliance CC (compliance per unit of length) can be expressed as $CC = dA/dP$, where A is the arterial cross-sectional area and dA the systolic-diastolic change in cross-sectional area in a cylindrical artery. To consider the effect of the distension on the stretching of the arterial wall, we also used the distensibility coefficient DC (distensibility per unit of length), defined as $DC = (dA/A)/dP$. Therefore, the quantity DC represents the strain of the arterial wall for a

given pulse pressure and pertains to the mechanical loading of the artery during a cardiac cycle. In the present study, CC and DC were calculated from nonsimultaneous measurements of diameter and pressure on the same arterial segment and, therefore, are presented as estimations rather than as direct measurements.

Therapeutic design

A one month administration of the converting enzyme inhibitor, Ramipril, was performed in 10 of the hypertensive subjects. The drug was given orally at the dosage of 5 mg once daily, which is known to produce a significant antihypertensive effect of about 15–20 mmHg.[12] In these subjects two haemodynamic studies were performed, the first before the beginning of the treatment and the second after 30 days of active treatment, three hours after the last drug administration.

Statistical analysis

All values are given as mean ± SEM. Comparison of arterial parameters and effect of treatment were done using an analysis of variance (ANOVA) followed by a paired Student's *t*-test. The influence of age, sex and BP on elastic arterial properties were tested with single or multiple regression analysis. The level of significance was $P < 0.05$.

Results

Table I shows the mean values of systolic, diastolic, mean and pulse pressure recorded with an automatic device at the site of the brachial artery.

Mean values of haemodynamic parameters (normotensive versus hypertensive patients)

At the site of the carotid artery, pulse pressure and diastolic diameter were higher ($P < 0.01$) in the hypertensive than in the normotensive group (Table II). The former had a reduced distensibility ($P < 0.001$) but a nearly normal compliance. Pulsatile changes of arterial diameter were within the normal range in hypertensive subjects.

At the site of the terminal aorta, pulse pressure was increased ($P < 0.001$) but diastolic diameter was unmodified in hypertensive subjects (Table III).

Table I Brachial blood pressure measurements

Parameter (mmHg) (Dinamap apparatus)	*Normotensives*	*Hypertensives*
Systolic blood pressure	129 ± 2	162 ± 2***
Diastolic blood pressure	74 ± 2	96 ± 1***
Mean blood pressure	93 ± 1	118 ± 1***
Pulse pressure	54 ± 2	66 ± 1***

Values are mean ± 1 standard error of the mean
****P < 0.001*

Table II Carotid artery parameters

	Normotensives	*Hypertensives*
Carotid pulse pressure (tonometer) (mmHg)	47 ± 3	58 ± 2**
Dd (mm)	6.6 ± 0.2	7.2 ± 0.1**
Ds-Dd (μm)	407 ± 27	378 ± 18
(Ds-Dd)/Dd (%)	6.3 ± 0.5	5.3 ± 0.2
Distensibility ($KPa^{-1} \times 10^{-3}$)	24.7 ± 3.0	17.1 ± 1.2***
Compliance ($m^2.KPa^{-1} \times 10^{-7}$)	7.8 ± 0.7	6.5 ± 0.6

Values are mean ± SEM
Dd = diastolic diameter; Ds = systolic diameter
***P < 0.01, ***P < 0.001, normotensives versus hypertensives*

Table III Arterial parameters of the terminal aorta

	Normotensives	*Hypertensives*
Pulse pressure (Dinamap) (mmHg)	54 ± 2	66 ± 1***
Dd (mm)	15.4 ± 0.5	16.2 ± 0.4
Ds-Dd (μm)	675 ± 77	472 ± 42**
(Ds-Dd)/Dd (%)	4.5 ± 0.5	3.0 ± 0.3**
Distensibility ($KPa^{-1} \times 10^{-3}$)	14 ± 1.7	7.6 ± 0.8***
Compliance ($m^2.KPa^{-1} \times 10^{-7}$)	24.8 ± 3.2	15.3 ± 1.4***

Values are mean ± SEM
For abbreviations see Table II
***P < 0.01, ***P < 0.001 for normotensive versus hypertensive subjects*

Pulsatile changes of diameter were significantly reduced ($P < 0.01$). Both distensibility and compliance were significantly reduced ($P < 0.001$) compared with normotensive controls.

Relations of carotid artery and terminal aorta parameters in the overall population (normotensives + hypertensives

At the site of the carotid artery, multiple regression analysis indicated that: (1) diastolic diameter was positively correlated with age ($P < 0.00001$), mean arterial pressure ($P < 0.0001$) and height ($P < 0.03$), each of them representing respectively 13.4, 9.2 and 2.4 of total variance, (2) distensibility was negatively correlated with age ($P < 0.000001$) and mean arterial pressure ($P < 0.000001$) with a respective proportion of 40% and 15.7% of total variance, and (3) compliance was negatively correlated with age ($P < 0.00001$) and mean arterial pressure ($P < 0.04$), each of them representing 22.8 and 2.7, respectively, of total variance.

At the site of the terminal aorta, quite different results were observed. Diastolic diameter was positively correlated with age ($P < 0.001$; 17.5% of total variance), height ($P < 0.005$; 13.6% of total variance) and mean arterial pressure ($P < 0.03$; 6.9% of total variance). Distensibility showed a strong negative correlation with mean arterial pressure ($P < 0.00001$; 37.5% of total variance) but was not significantly influenced by age. A similar pattern was observed for compliance and mean blood pressure ($P < 0.005$; 16.6% of total variance).

Effect of short-term converting enzyme inhibition

Ramipril produced a significant decrease in mean arterial pressure from 118 ± 1.4 mmHg to 100 ± 2.3 mmHg ($P < 0.001$) without modification of the heart rate (Figure 2). There was no drug effect on the carotid artery, involving both pulse pressure and arterial parameters. For the terminal aorta, whereas diastolic diameter did not change, pulse pressure decreased (52 ± 3 vs. 61 ± 3 mmHg; $P < 0.05$) and pulsatile changes in diameter increased ($P < 0.01$) from 3.1 ± 0.5 to 5.2 ± 0.6%. Compliance and distensibility increased significantly ($P < 0.001$; $P < 0.01$).

Discussion

The present investigation was designed to evaluate the arterial parameters of the terminal aorta by comparison with those of the common carotid artery in a group of 111 normotensive and hyper-

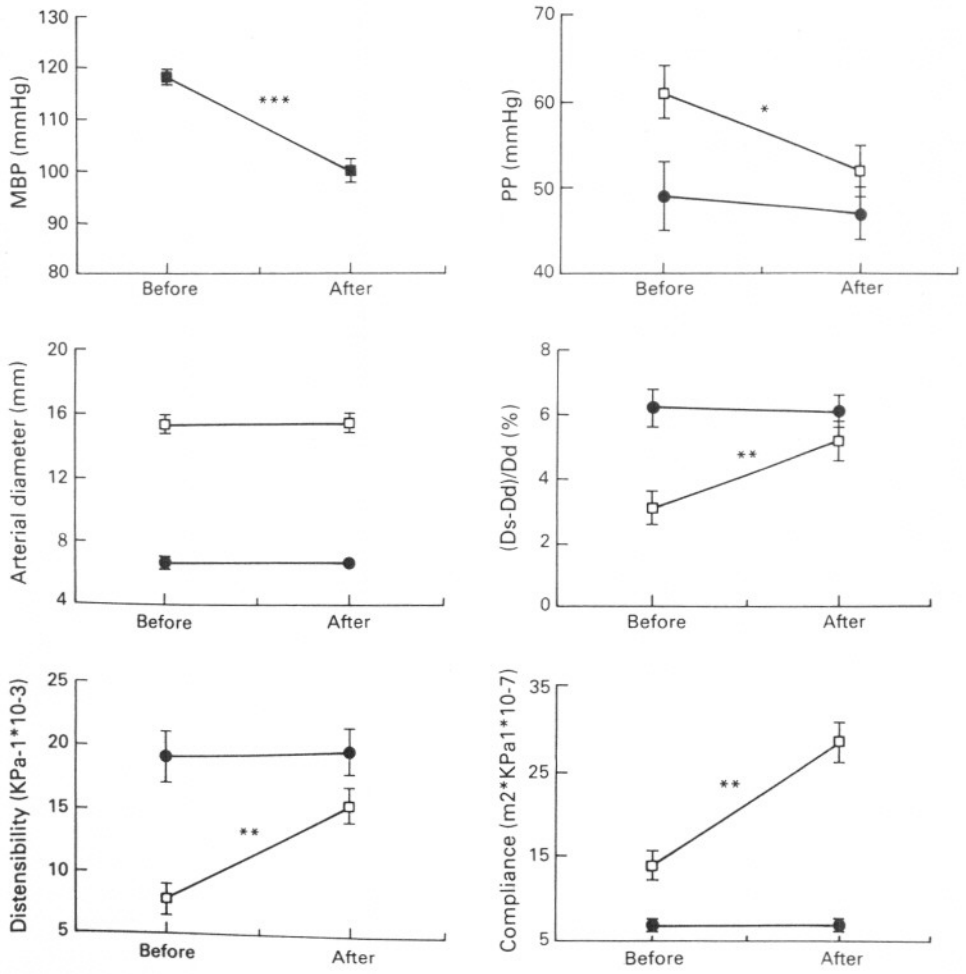


Figure 2 Arterial parameters before and after one month of ramipril 5 mg treatment. Aorta □; carotid ●.

tensive subjects with two specific characteristics. First, on the basis of clinical and biological investigations, and particularly on the basis of thorough echo Doppler determinations, all these subjects were shown to have no significant atherosclerotic complications. Second, the aim of the design was to study two peripheral large arteries for the same level of mean arterial pressure, a parameter that is known to be nearly the same in all parts of the arterial tree.[1,13] The three major conclusions of the study were: (1) in hypertensive patients the pulsatile component of BP was increased in both carotid artery and terminal aorta compared with normotensive subjects, (2) carotid artery elastic properties were mainly influenced by age whereas aortic stiffness was mainly determined by BP levels, and (3) BP reduction following converting enzyme inhibition improved the aortic but not the carotid artery stiffness.

It is well established that the contour of the aortic pressure alters as it travels toward the peripheral arteries.[1,9,13] Whereas the mean BP is only slightly lower in peripheral than in central arteries, the pulse pressure has a greater amplitude. In the normal human, the amplification of pulse pressure between the aorta and brachial artery averages 18–31% and that between the initial aorta and radial arteries's 46%.[1,9] The modifications of pulse pressure between central and peripheral arteries depend principally on nonuniform arterial elasticity of arteries and on peripheral wave reflections. Direct measurement of carotid pulse pressure by applanation tonometry revealed that in humans the alteration in the pulsatile component of BP is underestimated by the measurements of brachial artery sphygmomanometric BP. Indeed, the brachial artery pulse pressure is higher than that of the carotid artery, as extensively shown by invasive studies[2] and confirmed by this one. Similarly, pulse pressure at the terminal portion of the abdominal aorta is higher than carotid pulse pressure but statistically in the same extent as brachial pulse pressure, as noted by Imura *et al.*[11] Finally, in the present investigation, it appears that the mechanical signal causing the pulsatile change of arterial diameter was higher at the site of the terminal aorta than at the site of the carotid artery and this difference was more pronounced in the hypertensive than in the normotensive population.

In recent years, several devices have been described to transcutaneously measure arterial diameter and wall motion by tracking the echo signals from both the anterior and posterior arterial walls.[5–7,11,14,15] These instruments are characterised by sufficient linearity, dynamic range and tracking speed even when the signal-to-noise ratio of the original signal is not high. The device used in the present study was substantially improved with the help of the Doppler shift to evaluate well motion, thus permitting a high degree of reproducibility, as extensively published.[6,15] In this respect, the values of the aortic and carotid arterial parameters in normotensive controls were quite similar to those previously reported.[5–7,11,14,15] In contrast, the parameters of the terminal aorta in subjects with hypertension were the first reported in the literature using such noninvasive technics.

Whereas pulse pressure was higher in the hypertensive subjects, absolute and relative pulsatile changes in diameter were decreased in the aorta by 30% while remaining unchanged at the site of the carotid artery. This result shows that in hypertension there is a decreased capacity of the arterial wall to respond to the increased pulsatile changes of pressure and that the aorta is more affected than the carotid artery. This alteration is expressed by a striking decrease in distensibility at the site of the terminal aorta (about 50% compared with normotensive subjects) and, in a lesser extent, at the site of the carotid artery.

Interestingly, the compliance of the carotid artery was not significantly reduced in the hypertensive subjects whereas this parameter was significantly modified at the aortic site. This difference is principally due to the increase in the diameter of the hypertensive carotid artery. Since arterial compliance is directly related to arterial distensibility and cross-sectional area, we can say that the increase in carotid artery diameter counteracts the decreased carotid distensibility in hypertensive subjects. On the contrary, at the site of the aorta compliance is substantially reduced since the arterial cross-sectional area remains unchanged, despite a 50% decrease in arterial distensibility. We can, therefore, suggest that the aorta is much more affected than the carotid artery by hypertension.

In the present study, the elastic properties of the carotid artery were mainly influenced by ageing. A striking decrease in carotid artery distensibility with age was observed. Similar results have been reported by other authors[1,11] and by our group.[4,5,7] On the contrary, the terminal aorta elastic properties were much less influenced by ageing. Such differences between the two arterial segments can be tentatively explained by their structure. Histomorphometric data indicate that the carotid is a rather elastic artery whereas the terminal aorta is a more muscular artery. In the later, the absence of increase in aortic diameter in the presence of hypertension suggests an active response of the hypertensive aortic wall and an increase in smooth muscle tone. On the other hand, age related alterations might modify preferentially the mechanical properties of an elastic artery as the carotid artery. Nevertheless, the results of the multiple regression analysis reported in this study cannot really demonstrate a cause-to-effect relationship between arterial stiffness on one hand and age and BP on the other. For that reason, the response of the two arterial parameters to converting enzyme inhibition is important to consider in the present investigation.

Converting enzyme inhibitors are able not only to decrease BP but also to increase arterial diameter.[16] In humans, the increase was observed principally at the site of the brachial artery but not of the carotid artery. The present design was the first allowing one to compare, for the same mean BP reduction, the effects on two different territories: the carotid artery and the terminal aorta. As previously reported,[16] arterial diameter and stiffness were not

modified at the carotid level suggesting that, in predominantly elastic arteries, the changes produced by converting enzyme inhibition are offset by the concomitant decrease in BP. On the contrary, at the level of the terminal aorta, compliance and distensibility increased markedly following converting enzyme inhibition and even reached the normotensive levels. This finding suggests a specific effect on aortic vasomotor tone, in addition to BP reduction. Taken together, these data corroborate those of the multiple regression analysis, pointing to a predominant influence of BP on aortic stiffness.

In conclusion, the present study has shown that the factors modifying the elastic properties of large arteries may differ from one territory to the other. BP seems to be, by far, the main factor for terminal aorta stiffness whereas age is the principal factor for carotid artery rigidity. Such findings emphasise the different behaviour of the muscular and elastic artery following antihypertensive drug treatment.

Appendix

The indices of arterial stiffness described in this study are commonly accepted in the literature[1] and previously described for the echotraching technique that was used in this paper.[5–7,14,15] Other parameters might be described. We give here the correspondance with the present indices, accepting the model of a cylindrical artery:

1. The elastic modulus of volume distensibility of the artery per unit length of lumen is dP/(dA/A), which is the inverse of that we call 'distensibility coefficient' (DC).
2. The elastic circumferential strain (dA/2A), which is represented here by the relative change in diameter [(Ds-Dd)/Dd], may be deduced from the product between distensibility × pulse pressure (Tables II and III).
3. The diametric distension (dD) or the total distension per pulse (dA), which are represented here by (Ds-Dd) or may be deduced from the product: compliance × pulse pressure (Tables II and III).

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