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Radial artery compliance in patients with peripheral vascular disease

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Abstract: Compliance in largely central arteries of patients with peripheral vascular disease (PVD) has been reported to be reduced. However, the arterial tree is an inhomogeneous system, and there remains uncertainty about whether the peripheral arteries (e.g. the medium-sized muscular radial artery) undergo a similar change to the central arteries. The aim of this study was to investigate the radial artery elasticity in 19 patients with PVD compared with 18 normal subjects of comparable age and sex. Using a noninvasive high-resolution echo-tracking device coupled to a photoplethysmograph (Finapres system) allowing simultaneous arterial diameter and finger blood pressure monitoring, we measured the radial artery compliance by determining the diameter–pressure, compliance–pressure and distensibility–pressure curves. The results showed that the diameter of the radial artery was similar in the two groups, but that the compliance and distensibility were not further reduced in patients with PVD than in the normal controls at 100 mmHg and for a common blood pressure range. The present studies demonstrate that in patients with PVD the radial arterial compliance is not reduced, which indicates that the change in arterial elasticity is not identical. The potential mechanisms involved in this change in radial artery compliance are discussed.

Key words: atherosclerosis, arterial blood pressure, arterial compliance, peripheral vascular disease, radial artery, ultrasonography

Introduction

Compliance is an important property of the arterial system. It is one measure of the physical function of the wall of the vasculature. Arterial compliance was found to be abnormal in several vascular diseases.[1–3] Previous studies[3–6] demonstrated that, in patients with peripheral vascular disease (PVD), largely central artery compliance was reduced. However, the arterial tree is not a homogeneous system. Major differences exist in the structure and the function of various arteries.[9–13] It is not clear whether, in patients with PVD, a decrease in central artery compliance is also accompanied by a similar change in the peripheral arteries.

The recent development of a noninvasive, high-resolution, echo-tracking device provides an approach for detecting the compliance of the radial artery, a medium-sized muscular vessel.[14,15] By using an ultrasound probe and a photoplethysmograph to measure the arterial diameter and pressure simultaneously, it is possible to relate the arterial diameter change to a given change in pressure and then calculate arterial cross-sectional compliance, as previously described.[16,17] No data are available about the change in radial arterial compliance in patients with PVD. Therefore, the present study was performed to investigate the radial artery compliance of patients with PVD compared with normal subjects of the comparable age and sex.

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Methods

Study population

Eighteen normal subjects aged 41–68 years (13 men, 5 women; mean age 60 ± 1.8 years) and 19 patients with PVD aged 43–70 years (17 men, 2 women; mean age 63 ± 1.9 years) entered this study. The normal subjects had no evidence of diabetes mellitus, hypertension or other cardiovascular abnormalities. The ankle–arm pressure ratio with the Doppler method[18,19] was constantly greater than 1.0 (range 1.14–1.33), excluding the peripheral arterial disease.

The diagnosis for the patients with PVD was based on a thorough clinical examination, including the determination of ankle and arm systolic pressures. The ankle–arm pressure ratio was constantly lower than 0.81 (range 0.8–0.57), confirming the presence of PVD. None of the 19 patients had clinical symptoms and signs of hypertension, heart failure, coronary insufficiency or neurological disease.

No patients and control subjects had taken any drugs for at least 2 weeks before starting this study. The clinical characteristics of both groups are presented Table 1. This study was approved by the institutional ethics committee.

Measurement of radial artery compliance

The compliance of an artery is defined as an increase in volume for a given pressure increase. Since this change in arterial volume is mainly due to the change in arterial cross-sectional area, compliance can also be defined as the change in arterial cross-sectional area.[14,15] In the present study an A-mode ultrasonic echo-tracking device (NIUS02; SMH, Bienne, Switzerland), which is linked to a commercially available photoplethysmograph (Finapres, Ohmeda, Zug, Switzerland) allowing simultaneous arterial pressure

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Table 1 Clinical characteristics of controls and patients with PVD

Variables	Controls (n = 18)	PVD (n = 19)
Age (years)	60.5 ± 7.5	63.5 ± 8.4
Sex ratio (m/f)	13/5	17/2
Body mass index (kg/m^2)	25.3 ± 2.5	24.5 ± 2.6
Systolic blood pressure (mmHg)	125.6 ± 10.4	135.7 ± 9.2*
Diastolic blood pressure (mmHg)	80.9 ± 8.6	80.0 ± 6.1
Mean arterial pressure (mmHg)	96.2 ± 7.9	98.1 ± 5.4
Pulse pressure (mmHg)	43.6 ± 9.5	54.7 ± 7.6**
Heart rate (bpm)	62.0 ± 6.6	63.5 ± 8.3
Mean ankle/arm systemic blood pressure ratio	1.34 ± 0.08	0.75 ± 0.02**
Smokers (no.)	3	8
Cholesterol (mmol/l)	5.7 ± 0.8	5.8 ± 0.7
Triglyceride (mmol/l)	1.5 ± 0.12	1.5 ± 0.13

Values are mean ± sd; *$p < 0.05$; **$p < 0.01$ vs controls.

monitoring, was employed to measure the radial arterial diameter and finger pressure changes.[14] The lumen cross-sectional area (LCSA) of the radial artery was measured by using the arctangent model of Langewouters et al,[16] which is based on the following formula:

$$LCSA = \alpha \left[\frac{\pi}{2} + \tan^{-1} \frac{(P - \beta)}{\gamma} \right] \quad (1)$$

where P is the arterial internal pressure, and α, β and γ are three optimal parameters describing the spatial position of the diameter–pressure curve. From this formula, local arterial cross-sectional compliance can be calculated as follows:[17]

$$C = \frac{\Delta LCSA}{\Delta P} \quad (2)$$

Because of the nonlinearity of the cross-section pressure curve, compliance reduces as blood pressure increases. Therefore, we established the compliance–pressure over the common blood pressure range. Using equation 1, the local arterial cross-sectional compliance can be calculated as follows:

$$C = \frac{\alpha}{\gamma} \times \frac{1}{1 + [(P - \beta)/\gamma]^2} \quad (3)$$

and expressed as millimeters squared per millimeter of mercury for blood pressure from the diastolic to systolic range (compliance–pressure curve). The arterial cross-sectional distensibility is the inverse of the Peterson elastic modulus.[20] It is calculated by $D = 1/LCSA \times \Delta LCSA/\Delta P$ over the blood pressure range from diastole to systole (distensibility–pressure curve).

The investigation was performed in a controlled environment kept at 23 °C after the participants had resting for at least 15 min in a supine position. The measurement was always performed at the same time by a single operator. Briefly, the probe was placed perpendicularly over the radial artery axis in its largest cross-sectional dimension, 2 cm above the major skin fold of the wrist. After a switch to A mode, the backscattered echoes from the inner anterior and posterior walls were visualized on an oscilloscope, and the related high-radio-frequency signals were picked up by an electronic tracer directed to a computer acquisition system that was automatically programmed to analyse these signals. Each patient was recorded for 10 min, and the average of three 5 s recordings of radial artery diameter and blood pressure taken at 3 min intervals was used. All the readings were obtained on the right side, similar to Hayoz et al,[21] with the arm extended and fixed comfortably on a splint. A gel was applied between the extremity of the probe and the skin surface. Caution was used not to exert any pressure on the skin. Blood pressure was measured continuously throughout the study using a servoplethysmomanometer connected to the NIUS02. This apparatus was fitted to the right middle finger. Figure 1 shows the original recording of a simultaneous arterial diameter and finger pressure.

The coefficient of variation (CV) of radial artery diameter measurement was performed by the same operator at 10-minute intervals in eight subjects (four control individuals and four PVD patients), according to the formula CV = standard deviation (Δ)/mean (Δ) × 100, where Δ is the absolute difference. The CV was 2.9%, similar to a previous report.[15]

Data are presented as mean ± s.d. Student's t-test for unpaired observations was used to compare the difference between two means. For the statistical evalution of the diameter–, compliance– and distensibility–pressure curves we used split-plot ANOVA (repeated measures) and Tukey HSD tests for multiple comparison. A p value < 0.05 was considered significant.

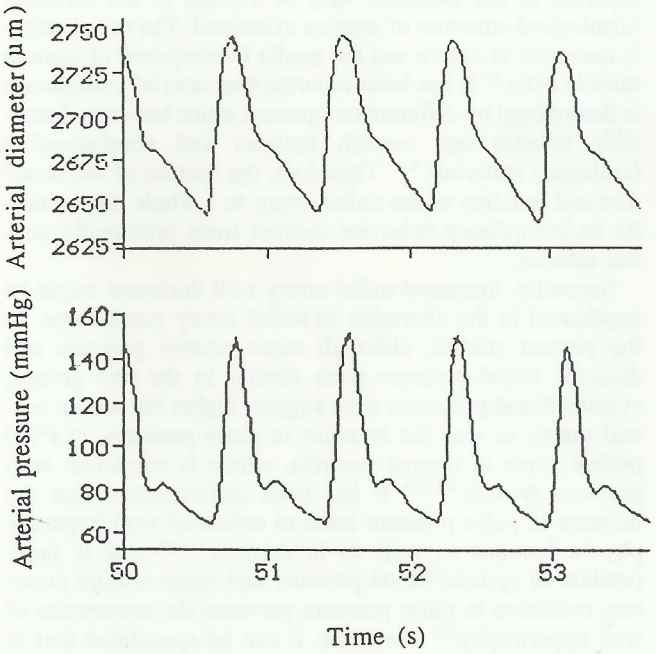

Figure 1 Original recording of simultaneously taken radial artery diameter (upper panel) and finger arterial pressure (lower panel)

Results

Table 1 shows the clinical characteristics of the patients with PVD and the normal subjects. Age, sex, body mass index, diastolic blood pressure, mean arterial pressure, heart rate and blood lipids were comparable in the two groups; the ratio of the ankle and arm systolic blood pressure was significantly lower; systolic blood pressure and pulse pressure were higher in the patients with PVD compared with the controls.

Figure 2 shows (a) a small progressive increase in radial artery diameter, (b) a marked progressive reduction in radial artery compliance and (c) distensibility in both PVD patients and normal subjects as blood pressure increased from diastolic to systolic pressure values. Over a similar blood pressure range, radial artery diameters were similar in the two groups, but compliance and distensibility were not further reduced in patients with PVD than in normal subjects. Table 2 shows the alterations in radial artery parameters at an isobaric pressure of 100 mmHg

Discussion

The major findings of the present study are that the diameter of the radial artery is similar in the two groups, and that compliance and distensibility of the radial artery are not reduced any more in patients with PVD than in the normal subjects. Our present findings of radial artery compliance measurement of the patients with PVD were contrary to previous studies of the central artery compliance,[3–6] which demonstrated a reduction. The data reported here did not permit us to clarify the mechanism behind this phenomenon; however, several possibilities can be discussed.

First, one possible explanation for the discrepancy between the present observation and those previously reported in the literature may be related to the different histological structure of arteries examined. The radial artery is muscular in nature and the media is composed of smooth muscle cells.[20] It has been reported that arterial compliance is determined by different component ratios between distensible (elastin and smooth muscle) and nondistensible (collagen) materials.[9,22] Therefore, the feature of the structure and function of the radial artery as a whole may underlie its compliance behavior distinct from proximally central arteries.

Secondly, increased radial artery wall thickness might be implicated in the alteration in radial artery compliance. In the present studies, although mean arterial pressure and diastolic blood pressure were similar in the two groups, systolic blood pressures were slightly higher but within normal range, as was the increase in pulse pressure, in PVD patients than in normal controls, which is consistent with previous reports.[6,23,24] It has been demonstrated that the increase in pulse pressure leads to enhanced wall hypertrophy in humans as well as in animals,[25–29] and is independent of systolic blood pressure and mean arterial pressure; reduction in pulse pressure prevents the occurrence of wall hypertrophy.[29] Therefore, it can be speculated that in patients with PVD the adaptation of the vasculature to the increased intravascular pulse pressure probably includes not only an increased thickness of the vascular wall, but also

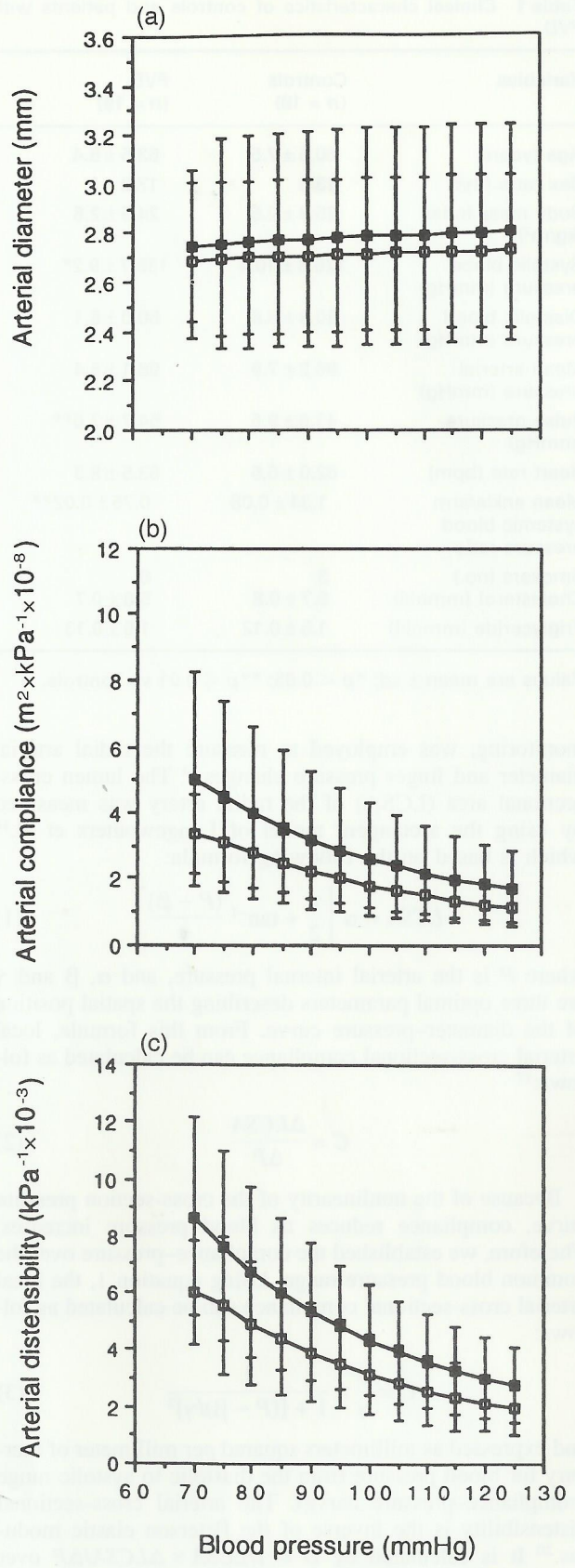

Figure 2 (a) Diameter–pressure relationships, (b) compliance–pressure relationships and (c) distensibility–pressure relationships in control (□) and PVD (■) subjects

Table 2 Parameters of the radial artery at 100 mmHg

Variables	Controls (n = 18)	PVD (n = 19)
Diastolic diameter (mm)	2.71 ± 0.41	2.78 ± 0.57
Distensibility ($kP_a^{-1} \times 10^{-3}$)	3.16 ± 1.17	4.34 ± 1.54**
Cross-sectional compliance ($m^2 \times kP_a^{-1} \times 10^{-8}$)	1.80 ± 0.68	2.69 ± 1.58**

Values are mean ± sd; **$p < 0.01$ vs controls.

a reduction in the elastic modulus of the wall materials, which may explain the findings of the present studies. This is evidenced by several recent studies[21,30,31] that radial artery compliance was not reduced in hypertensives, despite increased wall thickness. More recently Baumbach[32] demonstrated that, in Sprague-Dawley rats, hypertrophy in cerebral arterioles induced by increased pulse pressure is associated with enhanced arterial distensibility. Our present studies, taken together with previous observations, strongly indicate that hypertrophy of the arterial wall is, at least in the radial artery, not necessarily accompanied by reduced arterial elasticity, a mechanism that is compensatory for increased arterial pressure.

Thirdly, an increase in radial artery compliance could be partly compensatory for the decrease in largely central artery compliance. Large arteries act as a reservoir storing blood during cardiac ejection and releasing it during diastole, enabling the pulsatile cardiac output to be converted to a steady flow in the capillaries. Patel et al[33] reported that extensibility of the systemic arteries decreased from the ascending aorta to the iliac or femoral artery and increased again in peripheral arteries (e.g. the forearm artery) giving rise to a U-shaped pattern. Therefore, when decreased central artery compliance was present, an increase in peripheral arterial compliance can diminish the compliance gradient between central and peripheral arterial sites, participating in the cardiovascular hemodynamic homeostatic control.

Finally, it should be emphasized that the present findings are subject to the following limitations. First, we implied that the increased thickness with a reduced elastic modulus of the radial artery involves compliance alteration in patients with PVD, but there was lack of direct data correlated to the arterial compliance. Secondly, blood pressure was measured at the site of the finger and not the radial artery, thus leading to potential errors due to different pressure curves at these sites; generally, however, these errors should be similar in the two groups and hence should not affect the comparative data. Moreover, previous observations have demonstrated that this finger blood pressure measurement can provide accurate arterial blood pressure values compared with intra-arterial measurement.[34]

In summary, the current study indicates that in patients with PVD the radial artery compliance is, contrary to the reduction in largely central arterial compliance, not further reduced, suggesting that the alteration in systemic arterial compliance is inhomogeneous. The mechanism underlying this alteration in radial artery compliance is not yet completely understood. Further study is under investigation in our laboratory to clarify the relationships between structure and elastic properties of the radial artery wall related to hemodynamic changes.

References

1 O'Rourke M. Mechanical principles in arterial disease. *Hypertension* 1995; **26**: 2–9.
2 Laurent S. Arterial wall hypertrophy and stiffness in essential hypertension. *Hypertension* 1995; **26**: 355–62.
3 Levenson JA, Simon AC, Fiessinger JN, Safar ME, London GM, Housset EM. Systemic arterial compliance in patients with arteriosclerosis obliterans of the lower limbs: observations on the effect of intravenous propranolol. *Arteriosclerosis* 1982; **2**: 266–71.
4 Levenosn JA, Simon AC, Maarek BE, Gitelman RJ, Fiessinger JN, Safar ME. Regional compliance of brachial artery and saline infusion in patients with arteriosclerosis obliterans. *Arteriosclerosis* 1985; **5**: 80–87.
5 Safar ME, Laurent ST, Asmar RE, Safavian A, London GM. Systolic hypertension in patients with arteriosclerosis obliterans of the lower limbs. *Angiology* 1987; **38**: 287–95.
6 Safar ME. Arteriosclerosis obliterans of the lower limbs as a model of peripheral vascular disease with hypertension. *J Cardiovasc Pharmacol* 1991; **18** (suppl 4): S73–S77.
7 Joseph M, Hasson JE, Warnock DF, Warnock DF, L'italine GJ, Abbott WM. Non-invasive measurement of nonlinear arterial elasticity. *Am J Physiol* 1986; **250**: H181–H188.
8 O'Rourke M. Arterial stiffness, systolic blood pressure and logical treatment of arterial hypertension. *Hypertension* 1990; **15**: 339–47.
9 Learoyd BM, Taylor MG. Alteration with age in the viscoelastic properties of human arterial walls. *Circ Res* 1966; **XVIII**: 278–92.
10 Simon AC, Levenson J, Bouthire J, Safar ME, Avolio AP. Evidence of early degenerative changes in large arteries in human essential hypertension. *Hypertension* 1985; **7**: 675–80.
11 Heagerty AM, Aalkjær C, Bund SJ, Korsgaard N, Mulvany MJ. Small artery structure in hypertension: dual processes of remodeling and growth. *Hypertension* 1993; **21**: 391–97.
12 Reneman RS, Merode TV, Brands PJ, Hoeks APG. Inhomogeneities in arterial wall properties under normal and pathological condition. *J Hypertension* 1992; **10** (suppl 6): S35–S39.
13 Peterson LH, Jensen RE, Parnell J. Mechanical properties of arteries in vivo. *Circ Res* 1960; **8**: 662–39.
14 Tardy Y, Meister F, Perret F, Brunner HR, Arditi M. Non-invasive estimate of the mechanical properties of peripheral arteries from ultrasonic and photoplethysmographic measurements. *Clin Phys Physiol Meas* 1991; **12**: 39–54.
15 Girerd X, Mourad JJ, Copie X et al. Noninvasive detection of an increased vascular mass in untreated hypertensive patients. *Am J Hypertension* 1994; **7**: 1076–84.
16 Langewouters GJ, Zwart A, Busse R, Wesseling KH. Pressure diameter relationship of segments of human finger arteries. *Clin Phys Physiol Meas* 1988; **7**: 43–45.
17 Dobrin PB. Vascular mechanics. In: Sheperd JT, Abboud FM eds. *Handbook of physiology, Section 2: the cardiovascular system*, Volume III: *Peripheral circulation and organ blood flow*. Baltimore, MD: Williams & Wilkins, 1983: 65–102.
18 Barness RW. Noninvasive diagnostic assessment of peripheral vascular disease. *Circulation* 1991; **83** (suppl 1): 20–27.
19 Nicolaides AN. Assessment of leg ischemia. *BMJ* 1991; **303**: 1323–26.
20 Girerd X, Mourad JJ, Acar C et al. Noninvasive measurement of medium-sized artery intimal-medium thickness in humans: in vivo validation. *J Vasc Res* 1994; **31**: 114–20.
21 Hayoz D, Rutschmann B, Perret F et al. Conduit artery compliance and distensibility are not necessarily reduced in hypertension. *Hypertension* 1992; **20**: 1–6.
22 Son JAM, Smedts F, Vincent JG, Lier HJJ, Kubat K. Comparative

anatomic studies of various arterial conduits for myocardial revascularization. *J Thorac Cardiovasc Surg* 1990; **99**: 703–707.
23 Safar ME, Toto-Moukouo JJ, Asmar RE, Laurent S. Increased pulse pressure in patients with atherosclerosis obliterans of the lower limbs. *Atherosclerosis* 1987; **7**: 232–37.
24 Safar ME, Laurent S, Asmar RE, Safavian A, London GM. Systolic hypertension in patients with atherosclerosis obliterans of the lower limbs. *Angiology* 1987; **38**: 287–95.
25 James MA, Watt PAC, Potter JF, Thurston H, Swales JD. Pulse pressure and resistance artery structure in the elderly. *Hypertension* 1995; **26**: 301–306.
26 Christerense KL. Reducing pulse pressure in hypertension may normalize small artery structure. *Hypertension* 1991; **18**: 722–27.
27 Baumbach GL. Is pulse pressure a stimulus for altered vascular structure in chronic hypertension? *Hypertension* 1991; **18**: 728–29.
28 Hajdu MA, Heistad DD, Baumbach GL. Effect of antihypertensive treatment on mechanics of cerebral arterioles in rats. *Hypertension* 1990; **17**: 308–16.
29 Baumbach GL, Siems JE, Heistad DD. Effects of local reduction in pressure on distensibility and composition of cerebral arterioles. *Circ Res* 1991; **68**: 339–51.
30 Laurent S, Hayoz D, Trazzi S et al. Isobaric compliance of the radial artery is increased in patients with essential hypertension. *J Hypertens* 1993; **11**: 89–98.
31 Girerd X, Mourad JJ, Copie X et al. Noninvasive detection of an increased vascular mass in untreated hypertensive patients. *Am J Hypertens* 1994; **7**: 1076–84.
32 Baumbach GL. Effects of increased pulse pressure on cerebral arterioles? *Hypertension* 1996; **27**: 159–67.
33 Patel DJ, Coleman BR, Carpentier RG et al. Clinical relevance of distal arterial compliance? *J Hypertens* 1992; **10** (suppl 6): S15–S17.
34 Parati G, Casadei R, Groppelli A, Di Rienzo M, Mancia G. Comparison of finger and intraarterial blood pressure monitoring at rest and during laboratory testing. *Hypertension* 1989; **13**: 647–55.